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A commentary on the associations among 'food addiction', binge eating disorder, and obesity: Overlapping conditions with idiosyncratic clinical features

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ABSTRACT

This commentary discusses the evidence linking patterns of compulsive overeating, such as binge eating and grazing, with a putative psychopathological condition known commonly as 'food addiction'. It also addresses their distinctiveness as independent – albeit overlapping – clinical entities. Discussions focus largely on their respective clinical features and neuropsychobiological associations. Despite semantic issues about the appropriateness of the food-addiction label, there is accumulating evidence that some vulnerable individuals display addictive symptoms in relation to their consumption of certain highly rewarding foods. It is also argued in this paper that despite a positive relationship between obesity and addictive tendencies towards food, it is over-inclusive to model *obesity* as an addiction disorder, especially given the multi-faceted etiology and current pervasiveness of weight gain worldwide.

1. Introduction

When it published the 5th Edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) in 2013, the American Psychiatric Association (APA) formally recognized, for the first time, the existence of behavioral addictions in its chapter on *Substance-Related and Addictive Disorders*. Although Gambling Disorder is currently the only condition listed in that category, we are told that other 'excessive behaviors' related to eating, exercise, shopping, and sex had also been considered for inclusion. Nevertheless, at the time of publication, none was believed to have sufficient research-based validation as a mental-health problem (Potenza, 2014).

Importantly, however, the view that some individuals can become "addicted" to food has been widely accepted by the general public for decades, as indicated, for example, by the founding in 1960 of *Overeaters Anonymous,* and in 1987 of *Food Addicts Anonymous* (Davis, 2014). Both are self-help programs grounded in the 12-step approach first used in the 1930's to treat alcoholism. A testament to the popularity of these, and similar, organizations, is the thousands of regular meetings currently held around the world. It is also notable that the addictive potential of highly palatable foods, like chocolate, was described as early as 1890 in one of the original addiction-medicine journals (see Davis & Carter, 2014 for a fuller description of this early commentary). There it was argued that the regular consumption of strongly palatable substances could foster a condition where "*there is an intense and irresistible craving set up for such food*" (Clouston, 1890; pg. 207).

In the past decade, scientific study of the 'food-addiction' construct has grown rapidly as seen by the exponential increase in peer-reviewed scientific and clinical journal publications (Gearhardt, Davis, Kuschner, & Brownell, 2011). Although this putative condition has not yet been formally accepted into the lexicon of psychiatry, as noted above, an operationally useful diagnostic tool for research purposes was developed in 2009 (Gearhardt, Corbin, & Brownell, 2009). The *Yale Food Addiction Scale* (YFAS) is conceptually elegant in design by providing a direct parallel to the criteria used to classify substance dependence in the DSM-IV (APA, 1994) where "food(s)" is substituted for the word "substance" in the items of the scale. To date, a substantial body of clinical and bio-behavioral research using the YFAS supports the view that in certain cases, it is most appropriate to conceptualize compulsive overeating as a *bona fide* addiction disorder (see Davis, 2013; Hoebel, Avena, Bocarsly, & Rada, 2009; Munn-Chernoff & Baker, 2016; Shriner & Gold, 2014). Such research has also added to our understanding of risk factors for, and correlates of, addictive tendencies towards food – in particular, those which are hyper-

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palatable, ultra-processed, and high in sugar, fat, and salt.[1]

Notwithstanding supportive evidence for the food-addictive construct, some critics have pointed to difficulties in the human condition of identifying valid indicators of substance-dependence criteria such as tolerance and withdrawal (e.g. Ziauddeen & Fletcher, 2013). Interestingly, the symptom heterogeneity across all substance-use disorders has been acknowledged in the current (2013) version 5 of the DSM where each specific substance has been listed as its own disorder, and diagnosis only requires a relatively small number (variable depending on severity) of the 11 presented symptoms. Moreover, for certain substances (e.g. inhalants and hallucinogens) withdrawal is not even included as a diagnostic criterion.

Regrettably, some reviewers have also tended to obscure the evidence in this field by conflating the food-addiction concept with binge eating disorder (BED) and/or obesity (e.g. Volkow, Wang, Tomasi, & Baler, 2013; Ziauddeen & Fletcher, 2013). In other words – and perhaps as a result of their considerable overlap – the distinctiveness of these clinical classifications has often been overlooked. Evidence of genetic and neurophysiological similarities underlying substance addiction, binge eating disorder, and obesity (Carlier, Marshe, Cmorejova, Davis, & Muller, 2015; Frank, 2015) may too have contributed to the conceptual fusion of these overeating phenotypes.

The purpose of the current commentary is to extend the discussion about whether so-called 'food addiction' has the properties of a legitimate clinical disorder worthy of belonging in the DSM-5's *Substance-Related and Addictive Disorders category*; importantly also, to present arguments for the distinctiveness of food addiction from general overeating, and to consider its identity as a possible sub-type of obesity. In this regard, it is becoming clear that the experience of 'loss-of-control' (LOC) over food consumption can reflect a clinically significant eating disturbance *irrespective* of the amount of food that is eaten (Latner, Mond, Kelly, Haynes, & Hay, 2014). Indeed, several studies indicate that LOC is a stronger predictor of comorbid psychopathology, emotional distress, and psychosocial impairment than the quantity of consumption, or the daily pattern of food intake (see Latner et al., 2014). It will be argued therefore that binge eating is neither a necessary nor a sufficient component of food addiction, and that other forms of compulsive intake may also characterize this putative disorder.

2. Classifications and causality

While we may appreciate the singularity among overeating conditions in theory, their unique qualities are often ignored in scientific studies, especially when we employ indirect measures in the formation of operational definitions. Quintessentially, **obesity** is a mutable physical state characterized by higher levels of adipose tissue than are deemed healthy, but which can change relatively rapidly as a result of alterations in energy input or energy expenditure. Moreover, it is a condition that can only be assessed by proxy in the living organism - typically by a height-weight ratio known as body mass index (BMI: weight [kg]/height[m^2]).

In 2013, the *American Medical Association* stated that obesity was a "disease". While this regulatory body has no legal authority in the matter, their opinion does wield some influence, and has engendered considerable discussion with strong viewpoints on either side of the debate (Dawson, 2003; Kelly, 2013; Laville, 2012). It is no longer a moot point, however, that obesity has a major association with several disease states, which can themselves lead to further morbidities (e.g. Martin-Rodriguez, Guillen-Grima, Marti, & Brugos-Larumbe, 2015). In many Western countries, there has been an exponential increase in rates of obesity from the 1980's onwards (Flegal, Kruszon-Moran, Carroll, Fryar, & Ogden, 2016). Currently about 1/3 of the adult population in many parts of the world meets the criterion for obesity. Indeed, according to the World Health Organization, the global prevalence of overweight/obesity has doubled since 1980 – an astonishing occurrence given that such a dramatic change has taken place in a little more than one generation. Whether obesity is called a "disease" or not is largely a matter of semantics. There is little doubt that obesity is a condition with multiple causes, which must, prominently, include dramatic changes in our food environment during the same time period as its most prominent increase. In addition, causal factors tend to function in the form of interactions between our inherited biology and our physical surroundings (Albuquerque, Stice, Rodriguez-Lopez, Manco, & Nobrega, 2015; Davis, 2013).

Perhaps a more difficult issue to tackle is how BED and food addiction converge and diverge, both clinically and concerning aetiology, given that both conditions mutually reflect a compulsive overeating psychopathology – typically of foods that are hyper-palatable and calorically-dense. Indeed the two labels are frequently used synonymously. One clear difference is that BED has been conceptualized as *a psycho-behavioral* pathology. In other words, the diagnostic criteria for this disorder, according to the DSM-5, include only behavioral and psychological symptoms such as the frequency of binge episodes, the amount of food consumed, and their occurrence in the absence of hunger. In addition, these episodes are typically marked by feelings of loss of control, and a guilty, disgusted, and depressed mood state. BED also appears to be largely a *culture-bound syndrome* since its clinical emergence only occurred prominently in the early 1990's during a period marked by the most rapid rises in obesity rates ever recorded. In recent decades, several individual differences have been identified as risk factors, which seem to render some individuals more susceptible to the pronounced, relatively recent, and dramatic changes in our food environment (Davis, 2013; 2015; Giuliano & Cottone, 2015; Smink, van Hoeken, & Hoek, 2012).

By contrast**, addiction disorders** (substance-related or behavioral) are viewed as a state of developing pathophysiology fostered by excessive activation of brain reward circuitry, and inferred by symptoms like tolerance, withdrawal, and strong cravings – all of which have an established biological basis (Le Moal & Koob, 2007; Volkow, Koob, & McLellan, 2016). Some have therefore claimed that addiction is essentially a "brain disease" (e.g. Leshner, 2001; Wise, 2000) – or more recently, "an acquired disease of the brain" (Volkow et al., 2016) – with various behavioral markers, a primer of which is its increasingly compulsive nature. In addition, there is a particularly insidious and downwardly-spiraling aspect of addictions – viz. that central features of the disorder such as excessive use or engagement, in turn contribute to other defining characteristics of the condition like its escalating loss of control and the tendency towards chronic relapse. In other words, addiction disorders can be viewed as self-perpetuating clinical phenomena.

It is true that the brain-disease model of addiction has received some criticism (e.g. Heim, 2014), but while it has been challenged on various counts, it has not been successfully or entirely refuted. In fact, the past few decades have provided increasing evidence for the substance-induced neurobiological processes that underpin the compulsivity, inflexibility, and negative emotional states associated with addiction (Volkow & Koob, 2015; Volkow et al., 2016). Indeed, it is not necessary to negate the value of the disease model to accept that other influences are also essential to the acquisition of an addiction including those related to moral, cultural, psychological, and environments factors. The development of an addiction is

[1] Throughout the paper, use of the term 'food addiction' in the context of human research will refer to classification by the YFAS criteria, unless otherwise specified.