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Review

Overlap of food addiction and substance use disorders definitions: Analysis of animal and human studies

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ABSTRACT

Food has both homeostatic and hedonic components, which makes it a potent natural reward. Food related reward could therefore promote an escalation of intake and trigger symptoms associated to withdrawal, suggesting a behavioral parallel with substance abuse. Animal and human theoretical models of food reward and addiction have emerged, raising further interrogations on the validity of a bond between Substance Use Disorders, as clinically categorized in the DSM 5, and food reward. These models propose that highly palatable food items, rich in sugar and/or fat, are overly stimulating to the brain's reward pathways. Moreover, studies have also investigated the possibility of causal link between food reward and the contemporary obesity epidemic, with obesity being potentiated and maintained due to this overwhelming food reward. Although natural rewards are a hot topic in the definition and categorization of Substance Use Disorders, proofs of concept and definite evidence are still inconclusive. This review focuses on available results from experimental studies in animal and human models exploring the concept of food addiction, in an effort to determine if it depicts a specific phenotype and if there is truly a neurobiological similarity between food addiction and Substance Use Disorders. It describes results from sugar, fat and sweet-fat bingeing in rodent models, and behavioral and neurobiological assessments in different human populations. Although pieces of behavioral and neurobiological evidence supporting a food addiction phenotype in animals and humans are interesting, it seems premature to conclude on its validity.

1. Introduction

Substance use disorders (SUDs), commonly referred to addiction, have been extensively studied in the last decades and several lines of evidence suggest it consists in a neuroadaptative pathology. Accordingly, addiction is the behavioral end result of pharmacological overstimulation and usurpation of neural mechanisms of reward, motivated learning and memory. Although substances like alcohol, cocaine and nicotine are widely popular and central to the study of addiction and SUDs, growing interest is emerging from compulsive activities that are not clearly characterized as SUDs such as shopping, internet, sexual behaviors and compulsive overeating (Davis and Carter, 2009; Halpern et al., 2013; Liang et al., 2006). Moreover, a substantial body of research now supports a similar view of the neurobiological extent of natural rewards and their influence in the development of addictive disorders. Natural rewards such as sexual behavior, social interactions and food are intrinsically essential for survival but also comprise hedonic characteristics, and may engage similar brain pathways as drugs of abuse. Indeed, they can modulate and induce neuroplasticity in the reward pathways, similarly to traditional drugs of abuse (Olsen, 2011; Pitchers et al., 2010; Weatherford et al., 1990).

Food has both homeostatic and hedonic components, which makes it a potent natural reward and conditioning stimulus (Rada et al., 2010; Volkow et al., 2011). In clinical settings, the complex entanglement between compulsive behavior, eating disorders and obesity has drawn the attention towards similarities with SUDs. In fact, sources have proposed the idea of defining obesity as a brain disorder, a pathology of neuroadaptation (Kenny, 2011a, 2011b; Shafat et al., 2009). Theoretical models support the idea of a food addiction (FA), in which highly palatable foods have an abuse potential similar to classic drugs, can overly stimulate the reward

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pathways and, importantly, may lead to human or animal obesity. FA has gained a certain popularity recently, as more and more importance is given to social obesogenic factors, such as the overwhelming availability of highly palatable, calorie-dense, sweet and fat food items. For example, *Biological Psychiatry* has dedicated its May 2013 special issue to FA and its theoretical relationship with drug addictions. Animal (Heyne et al., 2009; Sampey et al., 2011) and human (Berner et al., 2012; Finlayson et al., 2007) theoretical models of FA and overeating have emerged, raising further interrogations on the validity of the issue.

Concerns and controversy arise with 1) the definition of FA as a SUD, as various lines of results in the past decade have investigated this concept under different names and clinical criteria and with 2) its level of relevance and causality in the development and maintenance of obesity. This review article presents experimental and preclinical data presently available in the literature as to whether or not FA should scientifically be comprised in and/or is consistent with the definition of SUDs. We will discuss this literature in the context of the recent definition of SUD in the DSM-5 (APA, 2013). Specifically, we will review data on three animal models of FA and relevant results in human studies.

1.1. Historical perspective of animal and human FA research

Although FA is a relatively novel concept, clinicians and scientists have addressed the close relationship between SUDs and feeding behavior for more than 25 years, in animal models as well as in humans (Fig. 1). For instance, Spiegel and colleagues have investigated the effect of naltrexone, an opioid antagonist frequently used to decrease cravings in alcohol use disorder, on food intake in obese males. They found that administration of naltrexone significantly reduced appetite but did not induce weight loss (Spiegel et al., 1987). The clinical effectiveness of naltrexone has also been investigated in eating disorders. Jonas and Gold have found that administration of naltrexone in bulimic patients significantly decreased binge eating. Moreover, their work has also paved the way in the investigation of the role of endogenous opiate peptides in binge-eating (Jonas and Gold, 1988a, 1988b). Thus, the compulsive component in binge eating has drawn important comparisons with drug abuse in early works.

Conversely, in early research based on animal models, Hoebel and colleagues have studied dysregulation of feeding behavior and body weight following perturbations of noradrenergic and serotoninergic systems (Ahlskog and Hoebel, 1973; Breisch et al., 1976). Food and drug rewards were also investigated in a subsequent study on dopamine systems, in which cocaine- and amphetamine-induced dopamine release was compared to food-rewarded behavior (Hernandez and Hoebel, 1988). Of interest, the terms "addiction to food" were proposed in this early article, marking a certain separation of binge-eating from eating disorders. Seminal works from Hoebel, Avena, Bocarsly and colleagues have followed, directly focused on food reward and its addictive potential (Avena and Hoebel, 2003a; Colantuoni et al., 2001; Rada et al., 2005). Several of these papers have favored the emergence of animal sugar, fat and sweet-fat bingeing models, which are described in the following sections.

1.2. Food addiction in the context of the DSM-5

Addiction is a broad term used in neurosciences, psychology and psychiatry, encompassing the definitions of dependence and substance abuse. As defined in the DSM-5, SUD is a maladaptive pattern of substance use leading to clinically significant impairment or distress, and is represented by cognitive, behavioral and physiological symptoms (DMS-5). Criteria for SUD diagnosis include tolerance, withdrawal symptoms, considerable time and energy spent on finding, using and recovering from the substance abuse, unsuccessful attempts to quit using, continued and compulsive usage despite negative and destructive consequences. Despite recognizing non-substance-related disorders related to the use of other rewards (i.e. gambling), the DSM-5 does not categorize similar disorders related to natural rewards as behavioral addictions or SUDs. A new category of "Addiction and Related Behavior" was proposed by the American Psychological Association before the publication of DSM-5, including behavioral addictions and addictions to natural rewards (Johnson and Kenny, 2010; Olsen, 2011). Similarly to SUDs, FA is currently characterized with food craving, risk of relapse, withdrawal symptoms and tolerance.

Food cravings are a hallmark of FA and are defined as an intense desire to consume a particular food that is difficult to resist and are differentiated from hunger by the fact that the consumption of the desired food will only compensate the craving whereas consumption of various types of food will alleviate hunger (Martin et al., 2011). Therefore, food cravings are not initiated by caloric restriction, dieting or food deprivation, but are susceptible to conditioning, similarly to aversion to certain types of food. Food craving is a

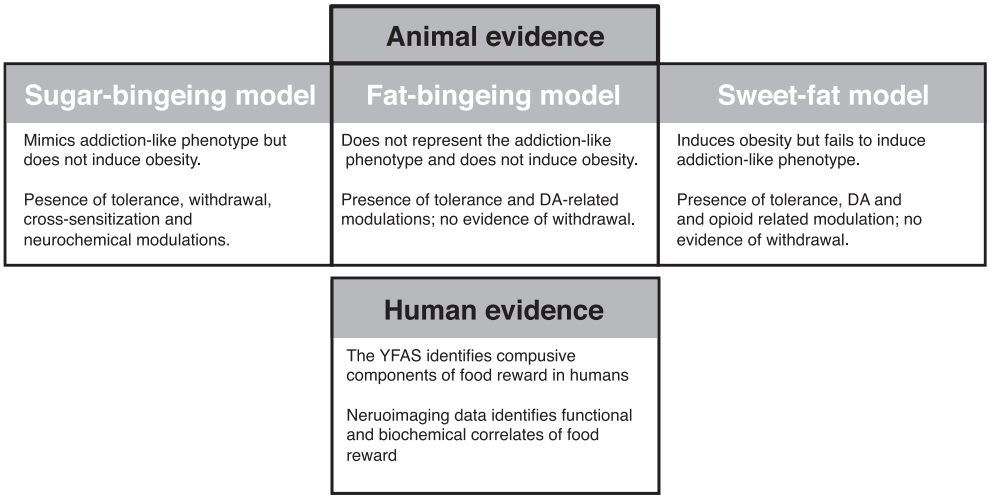

Fig. 1. Schematic of the animal and human models of food reward.