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Common Phenotype in Patients with Both Food and Substance Dependence: Case Reports

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Abstract

The understanding that genes play a significant role in reward dependence and associated behavioral and drug addictions is highlighted in the emergence of Reward Deficiency Syndrome (RDS). Here we show two case reports that unequivocally indicate the definite commonality between food and drug addiction. These human cases not atypically raise the question as to how to treat these two seemingly diverse addictions. We suggest that research directed in an attempt to induce natural activation of dopaminergic reward circuitry as a form of common therapy may indeed be parsimonious.

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Conflict of Interest Kenneth Blum, PhD, holds a number of US and foreign patents related to diagnosis and treatment of RDS, which has been exclusively licensed to LifeGen, Inc. Lederach, PA. With Dominion Diagnostics, LLC, North Kingstown, Rhode Island along with LifeGen, Inc., they are actively involved in the commercial development of GARS. John Giordano is also a partner in LifeGen, Inc. There are no other conflicts of interest and all authors read and approved the manuscript.

Keywords

Common phenotypy; Dopaminergic function; Reward Deficiency Syndrome (RDS); Food addiction; Substance Use Disorder (SUD)

Introduction

The concept of food addiction has been pioneered by Gold's team [1]. They have extensively published in this area of neuroscience and psychiatry [2]. It is well-known that both psychoactive substances and glucose induce a preferential neuronal release of dopamine following acute ingestion or administration involving common mechanisms [3]. In 1996, Blum et al. [4] published an article describing Reward Deficiency Syndrome (RDS) suggesting a commonality between substance dependence (drugs etc.) and behavioral addiction (food etc.) involving common dopaminergic polymorphic genetic antecedents [5]. Moreover, RDS is considered to be an umbrella term to describe common genetic antecedents of multiple impulsive, compulsive and addictive behaviors [6]. Moreover, neural circuits implicated in drug conditioning, craving and relapse overlap extensively with those involved in natural reward and reinforcement like food [7].

Exposure to drug-related cues in human addicts results in drug craving and localized activation of central circuits that are known to mediate cue-induced reinstatement of drug-seeking behavior in animal models of relapse [8]. It has been shown that similar regional activation patterns occur in humans in response to cues associated with foods [9]. Furthermore, drug- and food-related cues not only activate common neuroanatomical regions but also result in similar activity-regulated gene expression through mRNA within these shared areas [10].

Cues predictive of food availability are powerful modulators of appetite as well as food-seeking and ingestive behaviors including binge eating [11]. Corwin et al. [12] proposed that the upregulation of a number of early genes in unique patterns within cortico striatal, thalamic, and hypothalamic networks suggests that food cues are capable of powerfully altering neuronal processing in areas mediating the integration of emotion, cognition, arousal, and the regulation of energy balance.

The dopaminergic, enkephalinergic, and fos gene expressions are important regulatory genetic pathways for food and cocaine craving behaviors as well as other second messenger gene polymorphisms [13,14]. Individuals possessing a paucity of serotonergic and/or dopaminergic receptors and an increased rate of synaptic dopamine catabolism, due to high catabolic genotype of the COMT gene, are predisposed to self-medicating any substance or behavior that will activate dopamine release including alcohol, opiates, psychostimulants, nicotine, glucose, gambling, sex, and even excessive internet gaming, among others [15]. While there is no one standard therapeutic modality to treat RDS we believe following required clinical trials a long term dopaminergic activation approach will ultimately lead to a common safe and effective modality to treat aberrant food and drug craving behaviors.

The purpose of this article is to present two case reports showing that these commonality theories as proposed by Avena and Gold [16] and by Blum et al. [17] typically appear in the clinical world and treatment of both food and drug addiction deserve intensive investigation.

Case Study Showing Commonality of Food and Drug Addiction

Case 1

Client is a 42 year old Caucasian female with a history of food addiction since childhood. She began eating an increased amount of sugar and flour prior to 5 years old. She stated, “Since my father worked late at night, I was lonely only to find myself going to food to comfort me”.

Client’s height is 5′4 and her present weight is 185 lbs. Her highest weight was 254 lbs. (age 33) in 2003. At that time she had the gastric bypass surgery. Her lowest weight was 145 lbs. (age 34) in 2004, one year post surgery. She was still addicted to sugar and flour products and experienced the “dumping effect” when ingesting sugar. She began to consume drugs, i.e. cocaine, inhalants, marijuana, crystal meth, nicotine and alcohol at that time. In 2007, her father died and her alcohol intake had increased to the point of being out of control leading her to drug treatment. She began to gain her weight leading her to the present weight at 185. She went into detox at Palm Partners. She was consuming an enormous amount of sugar and flour while in detox. She was admitted G and G Holistic Recovery Program on 12/24/2012. For the past week she has developed edema. On 1/7/2013 the MD placed her on Lasix to decrease the edema. In addition, client complains of having cravings for alcohol on a daily basis for the past week.

Case 2

Here is a case report of a 44 year old Caucasian client that has had food addiction issues as far back as she can remember, resulting in the gastric bypass surgery. In addition, during high school began drinking alcohol excessively and at 40 years old began using opiates to the point of losing her Nursing license.

This Patient began overeating at 3 years old. Even though she was thin, she always ate extra food containing sugar and flour. She consistently overate and reports she was addicted to sugar and flour products for all these years. During high school years, she was a baton twirler cheerleader and had to be weighed in, therefore began purging (self-induced vomiting) and became bulimic to control her weight. She would binge and purge 1-2 times daily for 3 years. Client remembers her weight being 150-160 lbs. in high school. Post high school she stopped binge purge behaviors since she did not need to weigh in anymore, however her alcohol intake increased. While in college she would hide alcohol and sneak drinks. She married and got pregnant and stopped drinking and then she increased binge behaviors. Her weight increased as she overate while pregnant. She was 23 years old, first pregnancy – pre pregnancy weighed 150 lbs., age 27, second pregnancy – pre pregnancy weighed 180 lbs. and age 31, third pregnancy – pre pregnancy weighed 250 lbs. It is apparent how the progression of the overeating and how her weight inclined with each pregnancy.

At 32 years old, her weight was at a high of 300 lbs. She then went for the gastric bypass surgery. At that time, since she could not eat the enormous amounts of food, due to the gastric surgery and having a small pouch, she returned to drinking alcohol. At the beginning, she would get so drunk with so little alcohol, as a result of the surgery, and within a year she could drink two 2 liter bottles of wine or a fifth of vodka, in one sitting, until a black out. At the age of 33, her weight was down (at her lowest) at 165 lbs.

She experienced a divorce and the alcohol kept increasing. She then remarried and had 2 miscarriages at the age of 40, with her second husband. Between ages 40 – 43 she would steal pills (opiates) from work until getting caught and losing her Nursing license. At that time the food increased, as she stretched her pouch from the gastric bypass surgery and

continued to drink until her 3rd DUI (Driving Under the Influence) landed her in a treatment center.

Her family history consists of both parents being overweight. In addition, her father was an alcoholic and divorced the mother when client was 10 years of age. Father was a Vietnam Vet and died of a brain aneurysm at the age of 46. Mother never did drugs, except for the "food" as she also overate. Her mother is currently at age 74, height 5′4″ and weighs 220 lbs.

It is evident there is a genetic component of addiction. It is apparent that this Patient was so fully addicted to sugar and when she could not eat the sugar she switched to the alcohol (which metabolizes into sugar, as well as the flour). Since the gastric bypass surgery limited her to eat the amounts of food to feed the Reward System she had no choice but to consume alcohol and then opiates resulting in losing her Nursing license and 3 DUIs (Driving Under the Influences). In addition, the traumas she suffered (i.e. parents' divorce at age 10, her divorce, 2 miscarriages, etc.) resulted in the diagnosis Post Traumatic Stress Disorder coupled with the RDS.

Conclusion

Dysregulation of Dopamine function is important in reward processes leading to addictive behavior such as self-administration of opioids and other drugs of abuse including nicotine, alcohol and food. Dopamine and closely related neurotransmitters are also involved in a broadly distributed neural network that regulates eating behavior, affecting both homeostatic and hedonic mechanisms. In this sense, dopaminergic and opioidergic mechanisms are particularly implicated in the modulation of highly palatable foods, and opioid antagonists attenuate both addictive drug taking and appetite for palatable food. Thus, craving for palatable food could be considered as a form of dopamine-opioid-related addiction. While there are five dopaminergic receptors the D1 and D2 have been extensively researched and strongly implicated in reward.

Similarly, there are three main families of opioid receptors (μ, κ, and δ) of which μ-receptors are most strongly implicated in reward. The cases showing common phenotypy between food and drug addiction suggest a common therapeutic target [18]. Thus administration of selective μ-agonists into the NAcc of rodents induces feeding even in satiated animals, while administration of μ-antagonists reduces food intake. Similarly dopaminergic agonists reduce appetite [19] while dopamine anatgonists especially at D2 loci increase ingestive behavior [20]. Pharmacological studies also suggest a role for κ- and δ-opioid receptors. Preliminary data from transgenic knockout models suggests that mice lacking some of these receptors (both dopamine and opioids) are resistant to high-fat diet-induced obesity. Have we hatched the common phenotypy egg and should we consider common treatment for these two seemingly diverse substances?

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