The Obesity Epidemic and Food Addiction: Clinical Similarities to Drug Dependence

Jeffrey L. Fortuna, Dr.P.H.*

Abstract — As of 2010 nearly 70% of adult Americans were overweight or obese. Specifically, 35.7% of adult Americans are obese, and this is the highest level of obesity in the recorded history of the United States. A number of environmental factors, most notably the number of fast food outlets, have contributed to the obesity epidemic as well as to the binge prone dynamic. There is evidence that bingeing on sugar-dense, palatable foods increases extracellular dopamine in the striatum and thereby possesses addictive potential. Moreover, elevated blood glucose levels catalyze the absorption of tryptophan through the large neutral amino acid (LNAA) complex and its subsequent conversion into the mood-elevating chemical serotonin. There appear to be several biological and psychological similarities between food addiction and drug dependence including craving and loss of control. Nonetheless there is at least one apparent difference: acute tryptophan depletion does not appear to induce a relapse in recovering drug-dependent individuals, although it may induce dysphoria. In some individuals, palatable foods have palliative properties and can be viewed as a form of self medication. This article will examine environmental factors that have contributed to the obesity epidemic, and will compare the clinical similarities and differences of food addiction and drug dependence.

Keywords — acute tryptophan depletion, binge eating, fast food, orbitofrontal cortex, palliative, striatum

Overweight and obesity are now at an epidemic level in the United States. As of 2010, the Centers for Disease Control estimated that 69.9% of Americans were overweight or obese (Ogden et al. 2012). There are numerous forces, including biological, genetic, psychological, and environmental causes contributing to this epidemic. In addition, there are many clinical, psychological, and biological similarities between food dependence and drug dependence. This article will compare and contrast these similarities and differences, and provide insight into various environmental factors that play into the binge-prone dynamics of excess food and/or drug intake.

THE SKYROCKETING OBESITY EPIDEMIC

As of 2012, the United States had the highest obesity rates in its history. The CDC did not collect data on obesity and overweight until 1958, some 54 years ago (CDC/NCHS 2011). The obesity rate has steadily and remarkably increased over that time span. In 2010, 35.7% of adult Americans were obese with a BMI of 30 or higher (Ogden et al. 2012). In sharp contrast, only 4.8% of adults were obese in 1959. In addition to the 35.7% who are obese, another 34.2% are overweight (Flegal et al. 2010). More generally, the National Health and Nutrition Education Survey (NHANES; CDC/NCHS 2011) has reported that as a country we have gone from
only 13% of our population being overweight or obese in 1959 to 69.9% being overweight or obese as of 2010. This is more than a fivefold increase in the last fifty years. In addition there is a concurrent epidemic of type 2 diabetes in the United States. More specifically, the CDC noted that less than one million Americans had diabetes in 1959. In sharp contrast, more than 26 million Americans had diabetes as of 2010 (CDC/NCHS 2011). This is more than a twenty-eight-fold increase in the past 50 years: a clear epidemic.

There are many factors and forces that have contributed to these epidemics. They include the emergence of fast food establishments, much greater portion size, the availability of soda and other so-called “junk food,” insufficient physical activity, and insufficient intake of Omega 3 fatty acids in comparison to the more ubiquitous Omega 6 fatty acids. This article will examine the dramatic increase in fast food restaurants, and the impact of specific foods (e.g., soda) in the obesity epidemic. In addition, the question of the addictive properties of highly palatable (sugar rich and/or fat rich) foods will be detailed. Finally, comparisons between drug dependence and food “addiction” will be analyzed.

One of the most important questions that needs to be addressed is: how has this happened? There are many answers to this complex issue. Before we can solve the problem we need to understand the evolution of the obesity epidemic. It is important to understand the past, a time in this country when epidemic obesity did not exist. A healthier future, in large measure, is underlied by a thorough understanding of the past. What specifically has changed?

**THE FAST FOOD EPIDEMIC: TROUBLING TRENDS**

Clearly, a major cause in the obesity epidemic is the overwhelming number of fast food restaurants, and more importantly, how often individuals frequent them. Fast food restaurants are a relatively new phenomenon. They became prominent in the 1950s, with the opening of the first McDonalds in San Bernardino, California in 1957. So this is an industry that began less than 55 years ago.

Numbers quite often can demonstrate the enormity of the problem. In 1958, there were an estimated 600 fast food establishments in the US. By 1970, there were an estimated 30,000 fast food restaurants, a phenomenal increase. By 1980 the number had more than quadrupled to 140,000 fast food restaurants (Paeratakul et al. 2003). As of 2010, there were an estimated 222,000 fast food restaurants in the United States alone. Not only are fast food outlets ubiquitous in the U.S., but many of them are international corporations. So in less than 55 years the number of fast food restaurants has grown exponentially, and so coincidentally has the obesity epidemic.

Some of the statistics regarding the number of Americans who consume fast food are rather staggering. For example, fast food restaurants serve more than 50 million Americans every day (Wells & Buzby 2008). In addition, 42% of heavy consumers of fast food restaurants eat at fast food outlets 12 times or more per month (Pomeranz & Brownell 2008). As unbelievable as it may seem, U.S. consumers spent over 215 billion dollars on fast food in 2008. One study noted that despite the present economic recession, on average Americans spend 48% of their personal food budget on restaurant food (National Restaurant Association 2006). This is especially true of fast food restaurants or quick service restaurants. The eight largest fast food chains have an estimated 140,000 establishments throughout the United States.

In my clinical practice alone I have noted that over 80% of my adolescent clients eat at fast food outlets at least three times per week. It seems to be the casual gathering place or “hang out” for many adolescents, particularly after school. Whereas in the past there may have been an occasional visit to a fast food outlet for adolescents, nowadays it seems to have become rather routine. Not only this, but a number of studies have documented that children and adults eat nearly twice as many calories at restaurants, particularly fast food restaurants (Pomeranz & Brownell 2008). In addition, one study conducted at the Rudd Center at Yale University noted that out of 3,039 meal options that a child or adolescent could order at any of the “big eight” fast food restaurants, only 12 met/meet the nutritional standards of the National Academy of Science and the USDA (Rudd Center for Food Policy and Obesity 2010). This is striking and clearly a failing grade for the fast food industry.

The dramatic increase in fast food restaurants is not the only cause of the obesity epidemic, but it is one definitive cause. Location is also a factor; there are a disproportionately higher number of fast food restaurants and quick service establishments in poor urban and rural areas in every major city in the United States. Moreover, several studies have noted that a disproportionate number of fast food restaurants are located within 500 yards of a school (Davis & Carpenter 2009; Chriqui et al. 2008). This alone represents a public health problem and a genuine temptation for children and adolescents. New zoning laws in local communities can be effectively used to restrict the proximity of fast food outlets to schools.

**THE SODA CONUNDRUM: SOME STAGGERING NUMBERS**

Soda, which is essentially sugar water, is included in the overwhelming majority of fast food meals. What many Americans don’t know is that on average a 12 ounce can of soda contains 11 teaspoons of sugar. Each teaspoon is
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had previously noted that alcohol primes the release of endorphins from the arcuate nucleus in the hypothalamus, thereby kindling or priming the release of dopamine in the nucleus accumbens.

So, in some individuals, highly palatable foods trigger endorphin and dopamine release in a manner that is quite similar to many drugs of abuse. Those individuals are at greater risk to develop a wide range of eating disorders, including obesity. Likewise, and in a similar manner, other individuals prime endorphin when they drink or ingest a small amount of a drug or alcohol. Nonetheless, with the induction of tolerance, a small amount of a drug no longer induces a euphoric response in the striatum.

Ifland and colleagues (2009) have proposed that a possible explanation for overeating is that processed foods with high concentrations of refined sugar and fat are addictive substances, based upon their euphoric neural effects. Animal studies have also noted that the ingestion of sugar rich foods can prime the release of endorphin (Fortuna 2010). For example, Avena and colleagues (2006) documented that rats with intermittent access to sugar will drink sucrose solution in a binge-like manner that stimulates the release of dopamine within the nucleus accumbens. Moreover rats that are food deprived and then given 12 hour access to a sugar solution demonstrate bingeing behavior. Specifically, the rats consume copious amounts of food in the first hour (Colantuoni et al. 2001).

Of equal interest, a separate study found that when rats binged on sugar, the amount of dopamine release was proportional to the sucrose concentration, not the volume of sucrose consumption (Hajnal, Smith & Norgren 2004). The implication may be obvious: sugar-dense foods such as pop tarts, candy, or soda may indeed prime more dopamine.

**HYPER PALATABLE FOODS PRIME ENDOPHIN AND DOPAMINE**

It has been known for some time that sugar primes endorphin as well as dopamine in the nucleus accumbens. One study referred to the location of this effect, induced by chocolate, as the hedonic “hot spot” (Berridge & Pecina 2005) within the outer shell of the nucleus accumbens. Moreover, that research helped to confirm that opioid circuits in the nucleus accumbens were central to sweet preference in animal models. Some research (Snoek et al. 2004) has suggested that sweet tasting, sucrose-dense foods, when compared with fat dense foods, may consistently produce more endorphin release in both obese and bulimic individuals. Moreover, Drexnowski (1995) noted that highly palatable, high fat-sweets cause the release of endogenous opiates in reward pathways in specific individuals. Similarly, Gianolackis and colleagues (1996)
(ATD) will also trigger depression in many individuals who have had an episode of depression in the past or others with dysthymia. It is unknown if ATD will trigger a relapse to alcohol or other drug consumption in recovering individuals.

Similarity 1: Craving Similarities

There are some unmistakable clinical similarities between food addiction and substance dependence. One notable similarity is that showing an image or video to a substance dependent or food “addicted” individual can activate many of the identical neural pathways in the brain. For example, Gearhardt and colleagues (2011) studied a set of both obese and lean women, who were assessed as “food addicts” using the Yale Food Addiction Scale. When shown an image of a chocolate milkshake, there was notable activation in the anterior cingulate gyrus and amygdala of the women who were diagnosed as food addicts. This is similar to what Childress and colleagues (1999) found after showing recovering cocaine addicts a simulated video of the purchase and smoking of crack cocaine. They also reported that there was activation in the anterior cingulate gyrus and the right amygdala. Clearly, just seeing an image of the drug of choice or food of choice, given that there is a memorial foundation of the use and experience of euphoria or tranquility (from psychological pain), is apparently enough to activate one or more regions implicated in drug or food craving. In addition, seeing a visual cue may kindle thought contemplation and even planning to obtain and consume the food or drug.

Other types of sensory or behavioral cues may also trigger drug and food craving. For example, Schneider and colleagues (2001) used the odor of hard liquor (whiskey) among a number of newly recovering alcoholics to assess whether a two second olfactory exposure could potentiate craving in specific neural pathways. A total of ten recovering alcoholics as well as ten healthy control subjects were evaluated, using functional MRI, with the olfactory stimulus. On average, the testing of the patients occurred seven days after they had gone through detoxification. Schneider and colleagues found that the brief olfactory exposure activated the right amygdala and the cerebellum in the recovering alcoholics. There was no activation in the control subjects. It appears that the right amygdala may be a common marker for drug or food craving, regardless of the sensory cue.

Following the initial assessment, the men were treated with a pharmacologic agent (doxepin) designed to blunt or reduce drug craving, as well as with behavioral therapy (cognitive behavioral therapy) for a total of three weeks. What is quite notable and even encouraging is that upon re-exposure to the olfactory cue, none of the ten recovering alcoholic subjects showed any activation (using fMRI) in the amygdala. These results suggest that pharmacologic agents and/or behavioral therapies can be very effective in reducing relapse. In other words, there are some potentially efficacious therapeutic interventions.

There are numerous clinical implications here. For example, is the odor or aroma of palatable food a powerful trigger of food addiction or other eating disorders? Logic would say yes; nonetheless, this needs to be formally evaluated using fMRI as well as validated psychological scales. Further, are there effective pharmacologic and behavioral interventions for individuals diagnosed as food addicts, as there are for alcohol and drug dependent individuals? Such craving-reducing medications include naltrexone and acamprosate for alcohol dependence and bupropion for tobacco dependence. Again, logic would suggest that there are numerous potential interventions, both behavioral and pharmacologic, that need to be evaluated.

Clinical Similarity 2: Disinhibition and Tolerance (Cellular Adaptation)

A second clinical similarity is the issue of disinhibited food or drug consumption, that is, decreased inhibitory control of food or drug intake. Several researchers have suggested that, over time, a small quantity of food may no longer produce euphoric or palliative effects as it did early on in the history of food or drug consumption (Gearhardt et al. 2011; Volkow et al. 2008). The seminal research of Blum and colleagues (1996) has noted that in many individuals, there may be an insufficient number of dopamine 2 receptors, which mediate pleasure, in the striatum. They further described this as a reward deficiency syndrome. This appears to be a common neurobiological phenomenon in both drug dependence and possibly in food addiction or dependence. In the latter case, the individual might binge eat as a compensatory behavior for a hypoactive dopaminergic system in the striatum (Volkow et al. 2004).

Gearhardt and colleagues (2011) have also suggested that the consumption of specific tasty foods might actually override the desires to limit caloric food consumption. Moreover, using fMRI, their research noted that those individuals who met behavioral criteria as a food addict exhibited hypoactivation in the lateral orbitofrontal cortex (OFC), suggesting less inhibitory control in response to reward cues. Whereas there was notable activation in the amygdala and anterior cingulate cortex after seeing a photo of a chocolate milkshake (anticipatory reward), there was hypoactivation in the lateral OFC. This finding suggests several things. First, the anticipatory high might be greater than the actual food-induced high. As such, this may be a tolerance issue: the neurons have become genuinely less responsive to a small amount of a food, as in cases of substance dependence where increasingly higher doses of the drug are required to achieve euphoria or tranquilization (e.g., disengagement). Moreover,
the reduced activation in the lateral OFC suggests that individuals with a high food addiction diagnosis possess less inhibitory control upon consumption of palatable foods. Similarly, substance-dependent individuals also exhibit hypoactivation in the lateral OFC (Goldstein et al. 2007). Previous research has noted that when substance-dependent individuals are presented with a small dose of a drug, this in turn triggers excessive consumption of that drug.

In addition, during the early stage of drug use a small amount of the drug triggered a “priming effect,” that is an exaggerated euphoric response. It is postulated that over time, individuals become physiologically dependent, and a small dose is ineffective, and a larger dose is necessary to achieve euphoria or tranquilization. This appears to be due to cellular differences and/or adaptation in central nervous system (CNS) reward circuitry as well as hypoactivation in the lateral OFC. Likewise, in the naïve food addict, the consumption of a small amount of a highly palatable food produced euphoria or tranquilization. Over time, this effect diminished and notably higher quantities of food were required (e.g., bingeing) to achieve the desired effect. This appears to be related to both differences in reward circuitry, as well as hypoactivation of the lateral OFC. This is an important clinical similarity between substance dependence and food dependence.

So, the loss of control manifests through hypoactivation in the lateral OFC when consuming a hyper palatable food or a drug. This leads to periods of binging, as well as increased impulsivity when specific foods or drugs are available. Numerous recent studies (Gearhardt et al. 2011; Stice et al. 2010) have also found that obese subjects that they evaluated using functional MRI showed less activation in the striatum following the consumption of palatable food, when compared with lean subjects. Again, these findings suggest tolerance and/or a loss of control, based upon hypoactivity and/or a lack of responsiveness in the striatum and the lateral OFC. Moreover, this seems to predict that bingeing on a palatable food is required to achieve the euphoric and/or calming effect. This episodic or regular binging on palatable foods clearly contributes to the obesity epidemic.

In addition, numerous studies have noted that the anterior cingulate is stimulated through exposure to specific cues. Because the anterior cingulate is involved in contemplation and planning, this implies that there is some degree of volitional control in food consumption prior to the loss of control. Nevertheless, hypoactivity in the lateral OFC and a lack of appropriate responsiveness to routine, natural rewards (a good book, a funny movie, music, exercise) in the striatum seems to override volitional control in many individuals.

In the case of food addiction, Gearhardt and colleagues (2011) also noted that body mass index (BMI) was not an important factor in whether individuals demonstrated craving for hyper palatable foods, as well as loss of control in the lateral OFC. Both lean and heavy individuals appear to crave palatable foods. It is probable that the tolerance phenomenon occurs in either lean or heavy individuals.

In both food addiction and substance dependence, it may be that an individual may binge eat or binge with a drug as a compensatory behavior for a hyperactive dopaminergic system within the striatum (Wang et al. 2004). Food dependence and drug dependence share many of the same neural pathways, regarding both craving and loss of control. Future research will be required to document and validate these clinical similarities and differences.

Clinical Difference

Acute tryptophan depletion (ATD) consistently leads to binging on carbohydrate/fat rich highly palatable foods (Corsica & Spring 2008), particularly in carbohydrate cravers (they could be lean or heavy). For example, Pagato and colleagues (2009) noted that when overweight women underwent an acute tryptophan depletion (ATD) process, they increased their intake of sweet food. This study confirmed the results of previous research that links sweet, palatable food consumption with a serotonin deficiency (Asin, Davis & Bednarz 1992). Sugar dense (sweet) foods catalyze the absorption of tryptophan through the large neutral amino acid carrier complex in the brain. Fortuna (2009) had previously detailed that tryptophan is scarce in food, but that consumption of carbohydrates, particularly carbohydrate foods that trigger a substantial insulin increase, kindle tryptophan absorption and its subsequent conversion into serotonin. Moreover, serotonin is the principal mood elevating neurotransmitter in the CNS.

ATD will also trigger depression in many individuals who have had an episode of depression in the past or others with subclinical depression or dysthymia (Delgado et al. 1990). In his seminal research, Delgado reported that 67% of subjects with a prior history of major depressive disorder relapsed following acute tryptophan depletion. Subjects consumed a tryptophan-free beverage for eight hours that contained all of the essential and nonessential amino acids except tryptophan.

The fact that ATD can trigger binging on palatable foods in individuals who are carbohydrate cravers, as well as individuals with a history of depression, strongly suggests that binging on palatable foods is a form of self-medication for a mildly dysphoric mood state. The argument can be made that people binge eat to attenuate psychological stress, while others enjoy the inherent tastes and textures of palatable foods. Another reason why some individuals binge eat is to compensate for an underactive dopamine reward system. Wang and colleagues (2011)
have recently reported that binge eating predicts significant extracellular dopamine release in the striatum. Moreover, they suggest that binge eating appears to be a greater predictor of extracellular dopamine release than obesity or BMI. However, they elaborate that this phenomenon is only seen in a subset of individuals with a history of depression, bulimia, obesity, and binge eating disorder. It is postulated that the subset has a polymorphism of the serotonin system.

The work of Corsica and Spring (2008), Fortuna (2010), and others has suggested that carbohydrate craving and subclinical depression (dysthymia) are mildly dysphoric states where individuals self medicate to alleviate negative feeling states. This may be a result of low brain serotonin levels, as well as low serum tryptophan levels. Low brain serotonin levels may be a result of low amounts of tryptophan in food as well as individual genetic factors (polymorphisms of the serotonin system: tryptophan hydroxylase gene, serotonin transporter gene; Wedekind et al. 2010). As such, individuals may be incorrectly diagnosed as food addicts when they are in fact carbohydrate cravers, based upon relatively low serotonin activity and turnover in the brain. The biologic foundation in those individuals could be genetic differences, together with the paucity of tryptophan in food. Again, tryptophan is the scarcest essential amino acid in food.

Dysregulated or low serotonin pathways within the hypothalamus may underlie a wide variety of eating disorders, with or without concurrent depression or dysthymia. Moreover, low central serotonin levels appear to reduce the normal inhibitory control involved in food and drug craving and compulsive food or drug seeking. Roiser and colleagues (2006) have postulated that decreased serotonin levels, particularly those further decreased via ATD, heighten the incentive salience of sweet foods. This may undermine impulse control when exposed to rewarding foods (Olausson, Engel & Soderpalm 2002).

What is not known is if ATD will trigger a relapse to the drug of choice, such as a major stimulant or sedative drug, in a recovering drug user. This may be a significant clinical difference between drug dependence and food addiction. Nonetheless, recent research in recovering alcoholics has suggested that ATD may trigger alcohol craving (Ait-Daoud et al. 2009). Ait-Daoud and colleagues (2009) have noted that alcoholics who have the L-carrier variant of the 5-HTT gene tend to have much greater alcohol craving than those with the short form. The long form increases synaptic uptake of serotonin and thereby decreases serotonin levels in the synapse. Moreover, the craving state can be worsened by acute tryptophan depletion.

Even though there is a growing body of evidence that implicates ATD in alcohol craving, it is unclear in other forms of drug dependence. Further research is required to clarify whether ATD is a factor in other forms of drug dependence. Nonetheless, many alcohol and other drug dependent individuals who are still using or who are in recovery may suffer from comorbid depression.

**DISCUSSION**

Regardless of how it is labeled, it is clear that some foods may possess addictive properties, at least in some individuals. Multiple investigations have noted that obese and bulimic individuals are at heightened risk to binge on hyper palatable foods. Clearly, the consumption of large quantities of palatable foods is a factor in the obesity epidemic.

Given the obesity epidemic in the United States, together with the concurrent epidemic of type 2 diabetes, primary prevention measures should be of utmost importance. The fact that the three major manufacturers of soda in the U.S. were willing to voluntarily comply with restricting the sales of soda in schools is a definitive indication that prevention efforts are gaining traction (Kaledin 2009). Other promising public health efforts include the Healthy Kids, Healthy Communities Program, which has over 50 funded sites throughout the United States. This commendable program, operated by the Robert Wood Johnson Foundation, may serve as a template for other primary prevention programs designed to increase access to healthy food and increase physical activities, particularly in poor, underserved communities.

The recent recommendation by the Institute of Medicine that all adults and children have 60 minutes of physical activity every day is another indication of increased awareness of the importance of physical activity and healthy diet to quality of life (Brooks et al. 2004). First Lady Michelle Obama should also be commended for her investment in the Move First program.

Nonetheless, the implications of ubiquitous advertising of sugar dense and fat dense foods may affect the dietary choices of many individuals in high-risk groups, including but not limited to those who are obese. This is also true regarding the ubiquitous advertising of beer and other alcoholic beverages on television and on billboards. Moreover, Gearhardt and colleagues (2011) have suggested that such advertisements of highly palatable foods (i.e., pizza, sweets) may induce craving and impulsive purchase of these foods. This is supported by research indicating that just seeing an image of a milkshake or other palatable foods can induce craving. It is especially true in obese and bulimic individuals, as well as those with binge eating disorder. Moreover, a number of recent studies have suggested that certain sugar dense and fat dense foods are addictive.

Further research clarifying the effects of such advertisements and other cues in specific brain regions implicated in craving is timely and warranted.
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