Food Addiction: Evidence, Experience, and Clinical Implications

A Clinician’s Perspective Of The Science Behind Food Addiction

Marty Lerner, PhD
Faculty Disclosure

Marty Lerner, PhD

CEO

Milestones Eating Disorders Program
Defining Addiction: What are the common denominators?
- Tolerance
- Withdrawal symptoms
- More for longer periods than intended
- Unsuccessful effort to cut back or control
- Significant time to obtain or recover from effects
- Giving up social, occupational, recreational, activities because of substance use
- Continuation despite consequences

**DSM V- Substance Use Disorder-**

**Mild [2-3], Moderate [4-5], Severe [6+]**
Basic Assumptions

- Addiction in general is a complex combination of interactions between the biology of the “addict”, the nature of the substance abused, and the environment these take place.

- In other words, there is the nature of the person and the nature of the substance. The availability of the offending substances is the spark that starts the process.

- Food addiction is no exception.
Is Food Addiction a legitimate concept?

The “Naysayers” - Drug addiction, alcohol dependency, and process addictions [e.g. compulsive gambling] are substances and behaviors **not necessary for life**. Food is.

The “Believers” - But so is water and air – However, people do not consume water & air beyond their biological needs or in ways that threaten their survival.

- Drug Addiction – not all drugs are addictive.
- Food Addiction – not all foods are addictive.
Although there is no official definition of food addiction, many describe it much the same way as other drug dependence:

- Eating too much, despite consequences, even dire consequences to health
- Being preoccupied with food, food preparation and meals
- Trying and failing to cut back on food intake
- Feeling guilty about eating and overeating
- There is evidence some foods are more addictive than others. "It may be that doughnuts with high fat and high sugar cause more brain reward than soup."

*Mark Gold, M.D. Food and Addiction, 2012

Food Addiction- Description
Food Addiction is...

“Food addiction is a disease causing loss of control over the ability to stop eating certain foods. Scientifically, food addiction is a cluster of chemical dependencies on specific foods or food in general; after the ingestion of high palatable foods such as sugar, excess fat and/or salt the brains of some people develop a physical craving for these foods. Over time, the progressive eating of these foods distorts a person’s thinking and leads to negative consequences they do not want but cannot stop.”

- From: Food Addiction Institute Website
- http://foodaddictioninstitute.org/what-is-food-addiction/
<table>
<thead>
<tr>
<th>Binge Eating Disorder</th>
<th>Food Addiction</th>
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<tbody>
<tr>
<td>• Ate the whole box of chocolates in one sitting</td>
<td>• Ate the whole box over several sittings</td>
</tr>
<tr>
<td>• Emotional disturbance</td>
<td>• Physiological response</td>
</tr>
<tr>
<td>• DSM-5 clinical diagnosis, insurance reimbursement</td>
<td>• Not recognized or reimbursable</td>
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There are more similarities than there are differences...

*Obesity can exist without either one!*
Mood Disorders [Clinical Depression, Anxiety Disorder, Bi-polar] *
Substance Abuse/Dependency[alcohol, Rx Drugs, Etc.] *
PTSD – History of sexual abuse or emotional trauma*
Attention Deficit Disorders [ADD or ADHD]
Compulsive Disorders and “Process Addictions”
Gambling
Sex / Multiple Relationships
Spending / Shoplifting
Compulsive Approach To Work, School, Etc.[Perfectionism]
Self-Injury

*Most Frequent *No Stats on breakdown by ED Diagnosis

Frequently Accompanying an Over Consumptive Eating Disorder [BED, COE, FA, BN]
THE CASE FOR FOOD ADDICTION

Evidence
Food Addiction: What have we learned and who have we studied?
FOOD ADDICTION

- Stressing “moderation” to addicts is a moot point because the prefrontal cortex function is severely impaired.¹
- The message of “get it together”, “stop eating so much”, and “just become an intuitive eater” is not helpful²
- “Food can act on the brain as an addictive substance. Certain constituents of food, sugar in particular, may hijack the brain and override will, judgment, and personal responsibility, and in so doing create a public health menace.”³
- “Food addiction” versus “food and addiction”³

Genetics? Obesity Gene = FA Gene(s)?

Reward Circuits? – Dopamine/ Opioid Receptors

Role of Serotonin and Orexin

Classical Conditioning/Associative Learning?

External food [salient] cues “trumping” internal cues

Hormonal – ghrelin, leptin, insulin controlled?

Processed foods, “obesogenic environment”

Plasticity and Cross Addiction?

Stress > Cortisol > overeating

Dynamics of Pathological Eating?
Dopamine – The Reward /Feel Good Neurotransmitter

- The greater the expectation and experience of the substance, the “stronger” the dopamine signal.
- Drugs such as cocaine, amphetamines, alcohol, opiates stimulate increased levels of dopamine.
- Dopamine deficiencies will develop in response to repeated abuse of substances. The down regulation of D2 receptors creates tolerance.
- Dopamine deficiencies are thought to motivate drug seeking behavior / craving in an attempt to avoid withdrawal or experience the prior pleasant feelings. “Chasing the original high”
Functionally...

Dopamine D2 Receptors are Decreased by Addiction

Cocaine
Meth
Alcohol
Heroin

Control
Addicted

Dopamine Availability

Dopamine Availability and Addiction
Dopamine and Food Addiction

- Highly palatable foods [sugar, highly refined foods, excessive fats, salt] will have a similar effect on the dopamine reward circuitry as drugs of abuse.

- There are a multitude of animal studies demonstrating a preference for sugar over several different drugs [cocaine, amphetamine, ETOH].

- Dopamine is thought to interact with a number of hormones and processes responsible for the regulation of appetite and satiety such as leptin, ghrelin, orexin, and insulin.

- Less palatable foods provoke less dopamine stimulation.
"The brain's pleasure center, called the nucleus accumbens, is essential for our survival as a species... Turn off pleasure, and you turn off the will to live... But long-term stimulation of the pleasure center drives the process of addiction... When you consume any substance of abuse, including sugar, the nucleus accumbens receives a dopamine signal, from which you experience pleasure, so you consume more.

The problem is that with prolonged exposure, the signal attenuates, gets weaker. So you have to consume more to get the same effect -- tolerance. And if you pull back on the substance, you go into withdrawal. Tolerance and withdrawal constitute addiction. Make no mistake, sugar is addictive."

* Dr. Robert Lustig, Professor of Pediatrics, Endocrinology at the University of California
BRAIN REWARD CENTER

What do the colors mean?

RED
high dopamine
normal pleasure and interest

YELLOW
medium dopamine
difficulty feeling joy or pleasure

GREEN
low dopamine
lack of pleasure
A Couple of “Volunteers”

STUDY SHOWS THAT OREOS MAY BE MORE ADDICTIVE THAN COCAINE
The cycle of declining dopamine receptors leading to addiction

Bingeing

Numb pleasure response
Cravings
Bingeing escalates
Even less pleasure response
Stronger cravings
Bingeing

Dopamine receptors (D2)
Day 2 … No Coffee, No Danish, No Problem…
μ-Opioid deficiency or Dopamine deficiency?

Overeating and binge eating both stimulate the release of endogenous opiates and Dopamine.

The neurobiological underpinnings in the reward circuit may be more similar to opioid addiction than to other addictive disorders.

The opioid system modulates motivation and reward processing, and low μ-opioid availability may promote overeating to compensate decreased hedonic responses in this system.

Deficiencies of opioid receptors have been observed with obese subjects. Whether cause or effect of obesity is not known [down regulation?]

*The Journal of Neuroscience, 4 March 2015, 35(9): 3959-3965; doi: 10.1523/JNEUROSCI.4744-14.2015*
Serotonin’s Role with Food Addiction

- Hi-Glycemic foods increase Serotonin levels
- High Serotonin Levels compete / lower D2 levels
- Tolerance develops to Hi-Glycemic Foods
- Over time, tolerance and need to consume more HG food in order to offset depressed mood and addiction ensues
- Serotonin is also a “feel good” neurotransmitter. It signals comfort, sense of well being, and satiety – When attenuating to serotonin levels, or having a genetic deficiency, there is a need to increase intake to control mood and sense of satiety.
More research is needed to examine the precise mechanisms by which deficient serotonin, dopamine, and/or opioid receptors regulate food preferences, appetite, addictive eating, and mood. Whether these are an effect of addiction or a contributing cause is an important question to answer.

However, it is evident from the current body of scientific literature investigating the interplay between an individual’s relationship to food and a frequently co-existing mood disorder is essential in order for successful recovery opportunities to exist.

Treating one problem in isolation is not enough. It is only by comprehensively assessing the neurochemical commonalities underlying such complex behavioral patterns that sustainable treatment solutions become possible.
Risk Factors - Role of Genetics

Factors involved in Obesity (Etiology)

Most of our obesity is explained by Non-Genetic factors

- Genetics: 25%
- Lifestyle, Environment, Culture: 75%

Source: International Journal of Obesity
As of 2006, more than 41 sites on the human genome have been linked to the development of obesity when a favorable environment is present.*

However, Food Addiction specifically, may involve the interaction between dysfunctional leptogenic genes and the “dietary habits” of the individual.

There is evidence of a genetic link accounting for a deficit of D2 receptors common to both drug addiction and obesity / overeating.

Likewise, there is speculation a similar genetic anomaly exists with respect to serotonin concentrations and overeating in the service of achieving homeostasis with respect to serotonin.
### Hormonal Influences

#### Appetite Hormones

- **Leptin** (produced by adipose tissue, signals brain to stop eating)
- **Ghrelin** (produced in stomach, hunger- hypothalamus)
- **Insulin** (regulates food intake)
- **Norepinephrine** (fight or flight)
- **Cortisol** (steroid hormone in response to stress, physiological antagonist to insulin)
- **Peptide YY** (induces satiety)
OREXIN

- Synthesized in the Hypothalamus
- Plays a role with appetite and sleep
- R1 appetite  R2 sleep
- Giving orexin will increase craving for food
- Leptin inhibits the release of orexin.

Role of orexin-1 receptor mechanisms on compulsive food consumption in a model of binge eating in female rats. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology, 37 (9), 1999-2011*
Food and Drugs Activate Reward Circuitry Differently

**FOOD**
- Palatability (Sweets & fats)
- Changes in: Ghrelin, Insulin & Leptin
- Bliss Chemicals
  - Endogenous opioids
  - Cannabinoids

**DRUGS**
- Dopamine Cells
- Neurotransmitters that modulate Dopamine
  - Opiates, Nicotine, Cannabinoids, etc.

↑ Mesolimbic Dopamine
- Activate Reward Circuitry

Volkow, et al. 2008

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**Reward Circuitry**
Neuroplasticity, Chronicity, and Relapse

There appears to be plasticity associated with the addiction phenomenon in general as well as changes produced by addiction to specific addicting drugs. These findings also provide the basis for the current understanding of addiction as a chronic, relapsing disease of the brain with changes that persist long after the last use of the drug. Hence, the neuroplasticity in brain circuits and cell function induced by addictive substances [and behaviors] that are thought to underlie the compulsions to resume addictive behavior warrant further exploration. These investigations have significant implications for future therapies and treatments.
Sensitization, Cross-Addiction, and Relapse

- Opposite of tolerance – repeated abuse of a substance will create a significant “sensitivity” or dopamine release with related substances [cross addiction].

- Binge eaters, in particular with sugar, will be more “sensitive to the effects of alcohol and cocaine than non-binge eaters.

- Although tolerance is reversible it appears sensitization remains for extended periods and even a small amount of the offending substance [or behavior] will result in a heightened response.

- Supports “gateway” substances.

Summary of this research and theory appears in Nicole Avena’s text – Hedonic Eating, Oxford Press, Chapter 10, 2015.
Per Capita - Sugar Consumption

US Sugar Consumption, 1822-2005

\[ y = 0.5733x - 1041.4 \]

\[ R^2 = 0.9464 \]
Coke beneficiarys include the **Academy of Pediatrics**, as well as a number of respected medical and health groups, including $3.1 million to the **American College of Cardiology**, more than $3.5 million to the **American Academy of Family Physicians**, $2 million to the **American Cancer Society** and roughly $1.7 million to the country’s largest organization of dietitians, the **Academy of Nutrition and Dietetics**.

McDonalds has done the same as well as other fast and junk food manufacturers.

- N.Y. Times
- September 28, 2015
Processed Food Industry –
Anyone Remember the “Tobacco Debate?”
“Truth in Advertising”
The Perfect Storm

- Genetic predisposition [family history] +
- Dopamine / Serotonin deficiency +
- Learning history +
- Environmental Influences*
Focus of Recent and Future Research

- Is there a “disconnect” between the PFC [pre-frontal cortex] and reward center] – Can CBT / DBT psychological treatments / or abstinence from offending substances “trump” hedonic or compulsive eating? Short term or long term?

- Do people identified as FA harbor different sensitivities to specific food groups leading to addictive eating [e.g. sugar more than flour, flour products more than highly palatable fats, caffeine more than artificial sweeteners, etc?]
A Few Points to Consider

- Not all obese individuals are food addicts and not all food addicts are obese.
- Food Addiction or Eating Addiction [process addiction or substance addiction?]
- Food Addiction, Compulsive Overeating, Binge Eating Disorder, Bulimia: Semantics or conceptual differences?
- Can we draw conclusions from the obesity literature about Food Addiction?
Can we identify or predict children predisposed to addictive eating / food addiction?

Are people recovering from alcohol or drug dependency at greater risk for developing an addictive relationship with food? If so, is there a way to predict and/or prevent?

How effective are pharmacological interventions for FA [Naltrexone, Bupropion, Vyvanse, Topamax, SSRIs, SNRIs]?

What percentage of the obese population meet the criteria for FA or Substance Dependency?
Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry.

Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in the individual pursuing reward and/or relief by substance use and other behaviors.

The addiction is characterized by impairment in behavioral control, craving, inability to consistently abstain, and diminished recognition of significant problems with one’s behaviors and interpersonal relationships. Like other chronic diseases, addiction involves cycles of relapse and remission. Without treatment or engagement in recovery activities, addiction is progressive and can result in disability or premature death.
“All truth passes through three stages...

- First, it is ridiculed.
- Second, it is violently opposed.
- Third, it is accepted as being “self-evident.”

 Arthur Schopenhauer German philosopher (1788 – 1860)
Questions?

Marty Lerner PhD
CEO Milestones in Recovery
800 347-2364
mlerner@milestonesprogram.org