Is food addiction a useful concept to tackle obesity?

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Potatoes give you ‘drug fix’

You might not have to shoot it up to get a fix. But food is just as addictive as heroin and nicotine, research suggests. Substance abuse and high-glycemic foods – such as white bread and potatoes – trigger the same brain mechanism as that linked to addiction, according to Boston Children’s Hospital. They apparently cause excess hunger and stimulate reward and craving in parts of the brain.

London Evening Standard (July 2013)
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Outline

• Defining addiction
• Evidence for food addiction
  – Animal studies
  – Human studies (food craving, and binge eating)
• Implications: usefulness in explaining and reducing overeating

Junkie food?
What is addiction?

- **addict** *n.* a person addicted to a habit especially one dependent on a (specified) drug; (colloquially) enthusiastic devotee of sport or pastime

- **addiction** *n.* condition of taking a drug excessively and being unable to cease doing so without adverse effects

- **addictive** *a.* causing addiction and dependence

- **dependent** *a.* unable to do without something (esp. a drug)

The Concise Oxford Dictionary
(Drug) addiction

• ‘Addiction is restricted to the extreme or psychopathological state where control over drug use is lost.’

• ‘Dependence refers to the state of needing a drug to function within normal limits; it is often associated with tolerance and withdrawal (symptoms), and with addiction as defined above.’

• ‘Tolerance, sensitisation, withdrawal and craving are phenomena that may accompany dependence.’

Altman et al. (1996) *Psychopharmacology, 125, 285-345*
The food environment has changed dramatically with the influx of hyperpalatable foods that are engineered in ways that appear to surpass the rewarding properties of traditional foods (e.g. vegetables, fruits, nuts) by increasing fat, sugar, salt, flavors and food additives to high levels (Table 1). Foods share multiple features with addictive drugs. Food cues and consumption can activate neurocircuitry (e.g. meso-cortico-limbic pathways) implicated in drug addiction [1,2]. Animals given intermittent access to sugar exhibit behavioral and neurobiological indicators of withdrawal and tolerance, cross-sensitization to psychostimulants and increased motivation to consume alcohol [3]. Rats consuming diets high in sugar and fat demonstrate reward dysfunction associated with drug addiction, downregulation of striatal dopamine receptors and compulsive eating, including continued consumption despite receipt of shocks [4].
Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake

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Received 14 September 2006; received in revised form 19 April 2007; accepted 28 April 2007
‘Evidence for sugar addiction’

Rats lever press for 0.1mL 10% sucrose solution. Intermittent schedule is 12 hours access starting 4 hours into dark phase.

By day 21 intermittent-access rats consume an initial “binge” of sugar.

Note: total energy intake and body weight are unaffected.

Corwin et al. (2011) Physiology and Behavior 104, 87-97
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Gearhardt et al. (2011) Addiction 106, 1213-1220
Weight gain and ‘reward dysfunction’ in rats with extended access to a cafeteria diet

Cafeteria diet: Variety of energy-dense foods available 18-23 hours per day (‘extended’) or 1 hour per day (‘restricted’). All groups of rats had continuous access to chow (standard, nutritionally complete, low energy dense, lab rat food).

Measure of reward: current threshold at which rats respond (press a lever) to receive electrical stimulation of their brain through an electrode implanted in the lateral hypothalamus.

Some of Johnson and Kenny’s (2010) conclusions

• ‘The development of obesity in extended access rats was closely associated with a worsening deficit in brain reward dysfunction.’ (p 635)

• ‘Reward deficits in overweight rats may reflect counteradaptive decreases in baseline sensitivity of brain reward circuits to oppose their overstimulation by palatable food. Such diet-induced reward hyposensitivity may contribute to the development of obesity by increasing the motivation to consume high-reward ‘obesogenic’ diets to avoid or alleviate this state of negative reward.’ (p 639)
Dietary obesity in rats

Rogers, 1983. (Published in Mela & Rogers, 1998, Food Eating and Obesity, Chapman & Hall)
Underlying Johnson and Kenny’s arguments are parallels with results from similar studies on drugs of abuse.

Figure 1 Prolonged access to cafeteria food causes persistent elevations in threshold for BSR: comparison with drugs of abuse. (a) BSR threshold during daily intake of cafeteria food or drugs. (b) BSR threshold after loss of access to cafeteria food or drugs. Data were redrawn from Johnson and Kenny. In these studies, rats performed an operant response to obtain rewarding electrical brain stimulation into the median forebrain bundle at the level of the lateral hypothalamus. BSR threshold is defined as the minimum intensity of electrical stimulation that maintains operant responding. Increased BSR threshold is hypothesized to reflect decreased sensitivity of the brain reward system. Extended access to cafeteria food causes progressive disruption of the brain reward system that persists for long periods after loss of access to the food. In contrast, although extended access to abused drugs also causes progressive disruption of the brain reward system, this disruption dissipates in the first few days after withdrawal from the drugs.
Some of Johnson and Kenny’s (2010) conclusions reworked by Peter Rogers

• ‘The development of obesity in extended access rats was closely associated with a worsening deficit in reduced brain reward* dysfunction.’ (p 635)

• ‘Reward deficits Reduced reward in overweight rats may reflect counteradaptive decreases in baseline sensitivity of brain reward circuits to oppose their overstimulation by palatable food. Such diet-induced obesity-induced reward hyposensitivity may help contribute to oppose the development of obesity by increasing decreasing the motivation to eat consume high-reward ‘obesogenic’ diets to avoid or alleviate this state of negative reward.’ (p 639)

*measured by electrical self-stimulation of a brain area known to be involved in the control of eating (lateral hypothalamus)
In humans, diminished striatal dopamine receptor availability and striatal dysfunction have been associated with obesity [5] and prospective weight gain [6]. Foods and abused drugs may induce similar behavioral sequelae, including craving, continued use despite negative consequences and diminished control over consumption [7]. If foods are capable of triggering addictive processes, applying lessons learned from drug addiction to obesity, associated metabolic problems and diet-related diseases would suggest policy directions and prevention and treatment interventions [2,8].

Gearhardt et al. (2011) Addiction 106, 1213-1220
Results of studies on brain responses to food in obesity, etc

<table>
<thead>
<tr>
<th>Brain region</th>
<th>Response to presentation of food images</th>
<th>Response to cues signalling imminent presentation of food/ juice reward (anticipation)</th>
<th>Response to consumption of reward</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obese</td>
<td>BED</td>
<td>BMI</td>
</tr>
<tr>
<td>Regions associated with the reward circuity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Striatum</td>
<td>2 $\uparrow^{83,84}$, 1 $\uparrow^{85}$, 1 $\downarrow^{86}$</td>
<td>2 $\uparrow^{87,88}$, 1 $\uparrow^{89}$, 3 $\downarrow^{85,91,92}$, NA</td>
<td>1 $\uparrow^{91}$, 1 $\downarrow^{94}$, NA, NA, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>Midbrain</td>
<td>4 $\leftrightarrow^{83,84}$, 2 $\uparrow^{87,88}$, 5 $\uparrow^{85,89\rightarrow92}$, NA</td>
<td>2 $\leftrightarrow^{93,94}$, NA, NA, 1 $\uparrow^{95}$</td>
<td>1 $\uparrow^{86}$, 4 $\downarrow^{93,94,97,98}$, 2 $\leftrightarrow^{98,100}$, 1 $\uparrow^{94}$, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>PFC (orbital)</td>
<td>1 $\uparrow^{86}$, 3 $\uparrow^{85}$, 1 $\uparrow^{85}$, 1 $\leftrightarrow^{88}$</td>
<td>2 $\leftrightarrow^{87,88}$, 1 $\uparrow^{85}$, 1 $\downarrow^{89}$, 3 $\leftrightarrow^{85}$, NA</td>
<td>1 $\uparrow^{91}$, 1 $\downarrow^{94}$, 1 $\uparrow^{101}$, NA, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>PFC (lateral)</td>
<td>3 $\leftrightarrow^{84,86}$, 1 $\uparrow^{85}$, 1 $\uparrow^{85}$, 1 $\leftrightarrow^{83}$</td>
<td>2 $\leftrightarrow^{87,88}$, 1 $\leftrightarrow^{85}$, 1 $\downarrow^{89}$, 3 $\leftrightarrow^{85}$, NA</td>
<td>1 $\uparrow^{91}$, 1 $\downarrow^{94}$, 1 $\uparrow^{101}$, NA, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>PFC (medial)</td>
<td>2 $\leftrightarrow^{84,86}$, 1 $\downarrow^{85}$, 1 $\downarrow^{85}$, 1 $\leftrightarrow^{83}$</td>
<td>2 $\leftrightarrow^{87,88}$, 1 $\downarrow^{85}$, 1 $\downarrow^{92}$, 3 $\leftrightarrow^{85}$, NA</td>
<td>1 $\uparrow^{91}$, 1 $\downarrow^{94}$, 1 $\uparrow^{101}$, NA, 1 $\uparrow^{95}$</td>
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<tr>
<td>Amygdala</td>
<td>4 $\leftrightarrow^{83,86}$, 2 $\uparrow^{87,88}$, 5 $\uparrow^{85,89\rightarrow92}$, NA</td>
<td>2 $\leftrightarrow^{93,94}$, NA, NA, 1 $\uparrow^{95}$</td>
<td>1 $\uparrow^{93}$, 4 $\downarrow^{94,96\rightarrow88}$, 1 $\downarrow^{100}$, 1 $\downarrow^{94}$, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>Gustatory cortex (AI/FO)</td>
<td>1 $\uparrow^{83}$, 3 $\leftrightarrow^{84,86}$, 1 $\uparrow^{87}$, 1 $\leftrightarrow^{88}$</td>
<td>1 $\uparrow^{87}$, 1 $\leftrightarrow^{85}$, 3 $\leftrightarrow^{89,90,92}$, 2 $\uparrow^{85,91}$, NA</td>
<td>1 $\uparrow^{91}$, 1 $\downarrow^{94}$, NA, NA, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>Hippocampus/ PHG</td>
<td>2 $\leftrightarrow^{84,86}$, 1 $\uparrow^{85}$, 1 $\uparrow^{85}$, 1 $\leftrightarrow^{83}$</td>
<td>2 $\leftrightarrow^{87,88}$, 1 $\downarrow^{85}$, 1 $\downarrow^{89}$, 3 $\leftrightarrow^{85}$, NA</td>
<td>1 $\uparrow^{91}$, 1 $\downarrow^{94}$, NA, NA, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>Brain regions not associated with the reward circuity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thalamus</td>
<td>1 $\uparrow^{85}$, 3 $\uparrow^{81,84,86}$, 2 $\uparrow^{87,88}$, 5 $\uparrow^{85,89\rightarrow92}$, NA</td>
<td>2 $\leftrightarrow^{93,94}$, NA, NA, 1 $\uparrow^{95}$</td>
<td>2 $\leftrightarrow^{93,94}$, 5 $\uparrow^{93,94,96\rightarrow88}$, 2 $\leftrightarrow^{99,100}$, 1 $\uparrow^{94}$, 1 $\uparrow^{95}$</td>
</tr>
<tr>
<td>Rolandic operculum</td>
<td>4 $\leftrightarrow^{83,86}$, 2 $\uparrow^{87,88}$, 5 $\uparrow^{85,89\rightarrow92}$, NA</td>
<td>2 $\leftrightarrow^{93,94}$, NA, NA, 1 $\uparrow^{95}$</td>
<td>2 $\leftrightarrow^{93,94}$, 5 $\uparrow^{93,94,96\rightarrow88}$, 2 $\leftrightarrow^{99,100}$, 1 $\uparrow^{94}$, 1 $\uparrow^{95}$</td>
</tr>
</tbody>
</table>

The table shows responses that were elevated (↑) or reduced (↓) in groups of obese individuals or those with binge-eating disorder (BED) relative to controls. No group difference is signified by ‘-’. Numbers before the arrows indicate the number of studies. The table also shows studies reporting positive (↑), negative (↓) or no (↔) reported group difference between neural activity and body mass index (BMI) or food addiction (FA) scores. AI, anterior insula; FO, frontal operculum; NA, no reports available (at the time of writing); PFC, prefrontal cortex; PHG, parahippocampal gyrus.
Effects on eating of acute tyrosine/phenylalanine depletion in humans

• Used ATPD to reduce brain dopamine function
  – Participants (17 men) consumed a TYR and PHE free amino acid drink versus balanced amino acid drink
  – Eating tested 5 hours later (peak depletion, confirmed by measurement of blood amino acids concentrations)

• ATPD did not increase eating
  – If anything it reduced food intake (by 18%, p=.06)
  – And hunger was lower after this meal (p=.03)

Hardman et al. (2012) Physiology and Behavior 105, 1202-1207
The case for food addiction – human studies
Craving

In humans, diminished striatal dopamine receptor availability and striatal dysfunction have been associated with obesity [5] and prospective weight gain [6]. Foods and abused drugs may induce similar behavioral sequelae, including craving, continued use despite negative consequences and diminished control over consumption [7]. If foods are capable of triggering addictive processes, applying lessons learned from drug addiction to obesity, associated metabolic problems and diet-related diseases would suggest policy directions and prevention and treatment interventions [2,8].
What is food craving?

• Private experience – reported through verbal behaviour (or self-report rating)

• Studies of food craving:
  ‘a strong desire to eat a particular food’

• Dictionary:
  to long for, to desire intensely, to need urgently
ROGERS, P. J. AND H. J. SMIT. Food craving and food “addiction”: A critical review of the evidence from a biopsychosocial perspective. PHARMACOL BIOCHEM BEHAV 66(1) 3–14, 2000.—Although certain commonalities exist between eating and drug use (mood effects, external cue-control of appetites, reinforcement, etc.), it is argued that the vast majority of cases of (self-reported) food craving and food “addiction” should not be viewed as addictive behavior. An explanation is proposed that instead gives a prominent role to the psychological processes of ambivalence and attribution, operating together with normal mechanisms of appetite control, the hedonic effects of certain foods, and socially and culturally determined perceptions of appropriate intakes and uses of those foods. Ambivalence (e.g., “nice but naughty”) about foods such as chocolate arises from the attitude that it is highly palatable but should be eaten with restraint. Attempts to restrict intake, however, cause the desire for chocolate to become more salient, an experience that is then labelled as a craving. This, together with a need to provide a reason for why resisting eating chocolate is difficult and sometimes fails, can, in turn, lead the individual to an explanation in terms of addiction (e.g., “chocoholism”). Moreishness (“causing a desire for more”) occurs during, rather than preceding, an eating episode, and is experienced when the eater attempts to limit consumption before appetite for the food has been sated. © 2000 Elsevier Science Inc.
Craving as a consequence of restraint

Desire to eat (elicited by exposure to eating-related cues, anticipation of the pleasure of eating, ‘hunger’, etc.)

Eating resisted or impeded

Craving

Attribution of addiction (e.g. ‘chocoholic’)
Addiction as an attribution

..prevailing notions tend to see addiction as something that happens to people; that is, as something imposed from outside by the inescapable pharmacological properties of an alien substance. *The Myth of Addiction* argues that explaining one’s behaviour as either within, or outwith, one’s control has consequences according to the situation, and in a climate of moral and legal censure it makes sense to choose the latter.

J. B. Davies, 1997, page vii
Attribution of food addiction – helpful or unhelpful?

• Effects of attributing overeating and obesity to addiction (physiological inevitability)
  – Attitudes of others (e.g., support for public funding of obesity treatments*, stigma**, etc)
  – ‘Obesity is a disease’ (in obese participants reduced body-image dissatisfaction, but reduced concerns about weight which led to higher-calorie food choices***)
  – Diminished personal responsibility and motivation to change (e.g., dieting success****)

*Lund et al. (2011) *Obesity* 19, 1580-1585  
**Latner et al. (2014) *Appetite* 77, 79-84  
***Hoyt et al. (2014) *Psychological Science* 25, 997-1002  
****Ogden & Wardle (1990) *British Journal of Clinical Psychology* 29, 445-446
Attribution of food addiction – helpful or unhelpful?

• A new study (Liverpool/Bristol)
  – Participants read a passage confirming or dis-confirming existence of food addiction
  – Food intake (cookies, crisps, breadsticks, grapes)
  – Scores on Yale Food Addiction Scale

• Context (e.g., effect on smokers versus non-smokers)
The case for food addiction – binge eating

• Binge eating is defined as (DSM-V)
  – “episodes of eating significantly more food in a short period of time than most people would eat under similar circumstances, with episodes accompanied by feelings of lack of control.”
  – Occurs in Binge Eating Disorder, Bulimia Nervosa and Anorexia Nervosa

• Is this food addiction (addictive behaviour)?
  – That depends on how addiction is defined
  – Out of a sample of women (n=79) with a diagnosis of Binge Eating Disorder, 92% met adapted criteria for substance dependence (DSM-IV), and 41% met adapted criteria for addictive disorder (Goodman, 1990)*

*Cassin & von Ranson (2007) Appetite 49, 687-690
The case for food addiction – binge eating

• Association between Binge Eating Disorder and obesity (US study), proportion obese*:
  – Women with diagnosis of BED: Black 83%, White 56%
  – Matched controls (no BED): Black 35%, White 13%

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- Relationship between ‘food addiction’ and weight status

<table>
<thead>
<tr>
<th>Weight category</th>
<th>BMI (kg/m²)</th>
<th>Sample size</th>
<th>Diagnoses (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt;18.50</td>
<td>n = 60</td>
<td>10.0</td>
</tr>
<tr>
<td>Normal-weight</td>
<td>18.50–24.99</td>
<td>n = 572</td>
<td>6.3</td>
</tr>
<tr>
<td>Overweight</td>
<td>25.0–29.99</td>
<td>n = 86</td>
<td>14.0</td>
</tr>
<tr>
<td>Obese</td>
<td>≥30.00</td>
<td>n = 32</td>
<td>37.5</td>
</tr>
</tbody>
</table>

***Meule (2011) Frontiers in Psychiatry 2, 1-4  
****Pedram et al. (2013) PLoS ONE 8, e74832
Is the food addiction model appropriate?

• On a reasonably stringent model of addiction
  – food poses a low risk of addiction
  – food addiction does not explain most overeating and obesity

• Attributing overeating to food addiction may be counterproductive to dietary change

To restate the two caveats, whatever entity we call food addiction should not be seen as an excuse for unhealthy eating and the unhealthy eating associated with food addiction should not be equated with obesity.
