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Binge Eating: Neurochemical Insights from Animal Models

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Q. HOW CAN ANIMAL MODELS INFORM RESEARCH ON BINGE EATING?

A. Data generated from animal models of binge eating have yielded important insights pertaining to physiological and neurochemical alterations that may be the cause, or consequence, of abnormal eating behaviors. Several animal models have been developed for the study of binge-eating behavior (see Corwin & Buda-Levin, 2004 for review). Some models encourage voluntary binge eating in sated animals by offering limited access (e.g., 2-h/day) to a palatable food, such as those high in fat or sugar (Berner, Avena, & Hoebel, 2008; Dimitriou, Rice, & Corwin, 2000), in addition to ad libitum access to standard rodent chow. Other models incorporate periods of moderate food deprivation in order to stimulate animals to binge eat when a palatable food is presented (Avena, Rada, & Hoebel, 2008b). Sham feeding via a gastric fistula, in which the amount of food consumed is not constrained by stomach capacity, has also been used to model binge eating by allowing rats to consume large amounts of liquid food in a brief period of time (Avena, Rada, Moise, & Hoebel, 2006; Smith, 1989). Stressors can also precipitate binge eating in rats (Boggiano et al., 2005).

Q. IS THERE CLINICAL VALIDITY TO ANIMAL MODELS OF BINGE EATING?

A. Animal models of binge eating appear to have clinical validity. Binge eating in humans can occur in sated and hungry individuals (Marcus & Kalarchian, 2003). Periods of food restriction and bingeing have been shown in animal models to impact food consumption long after restriction has ceased (Hagan & Moss, 1997), a finding that models reports in humans (Polivy, 1996). There is also a connection between stress and binge-type eating in humans (Cattanaach, Malley, & Rodin, 1988) similar to that observed in animal models. Anxiety is comorbid with binge eating in humans, and signs of anxiety have been observed in animal models of binge eating (Avena, Bocarsly, Rada, Kim, & Hoebel, 2008a; Colantuoni et al., 2002).

Q. WHAT HAVE WE LEARNED ABOUT THE NEUROCHEMICAL BASIS OF BINGE EATING FROM ANIMAL MODELS?

A. Our knowledge of the effects of binge eating on several neurochemical pathways has been advanced through the use of animal models. Daily binge eating on a palatable sugar

solution or fat diet has been shown to repeatedly release dopamine (DA) in the nucleus accumbens (NAc), an area of the brain that processes reward and motivation, (see Avena et al., 2008b for review). Sugar-bingeing rats also show an increase in dopamine D1-receptor binding in the NAc and a decrease in dopamine D2-receptor binding in the dorsal striatum. These changes in DA are similar to the changes observed with drug dependency, and it has thus been suggested that binge eating of palatable foods may result in addictive-like behavior and concomitant neurochemical changes.

Animal models of binge eating have also revealed information regarding other chemicals in the brain. A role for opioids in binge eating has been supported by work showing alterations in opioid receptor-expression and genes in the NAc of binge eating rats (Colantuoni et al., 2001; Spangler, Goddard, Avena, Hoebel, & Leibowitz, 2003). Additionally, binge eating is stimulated by the injection of opiates into the NAc, an effect that is dependent on activation of the amygdala (Will, Franzblau, & Kelley, 2004). Animal models have also unveiled a role for NAc acetylcholine (Rada, Avena, & Hoebel, 2005) and GABA (Buda-Levin, Wojnicki, & Corwin, 2005) in binge-eating behavior.

The use of animal models is crucial for understanding the functions of neurochemicals that directly relate to binge eating. This information can complement and expand upon findings obtained from techniques used in human subjects, such as PET and fMRI.

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References

- Avena NM, Bocarsly ME, Rada P, Kim A, Hoebel BG. After daily bingeing on a sucrose solution, food deprivation induces anxiety and accumbens dopamine/acetylcholine imbalance. *Physiology and Behavior*. 2008a; 94(3):309–315. [PubMed: 18325546]
- Avena NM, Rada P, Hoebel BG. Evidence of sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience and Biobehavioral Reviews*. 2008b; 32(1):20–39. [PubMed: 17617461]
- Avena NM, Rada P, Moise N, Hoebel BG. Sucrose sham feeding on a binge schedule releases accumbens dopamine repeatedly and eliminates the acetylcholine satiety response. *Neuroscience*. 2006; 139(3):813–820. [PubMed: 16460879]
- Berner LA, Avena NM, Hoebel BG. Bingeing, self-restriction, and increased body weight in rats with access to a sweet-fat diet. *Obesity*. 2008 Jun 26. Epub ahead of print.
- Boggiano MM, Chandler PC, Viana JB, Oswald KD, Maldonado CR, Wauford PK. Combined dieting and stress evoke exaggerated responses to opioids in binge-eating rats. *Behavioral Neuroscience*. 2005; 119(5):1207–1214. [PubMed: 16300427]
- Buda-Levin A, Wojnicki FH, Corwin RL. Baclofen reduces fat intake under binge-type conditions. *Physiology and Behavior*. 2005; 86(1–2):176–184. [PubMed: 16140347]
- Cattanaach L, Malley R, Rodin J. Psychologic and physiologic reactivity to stressors in eating disordered individuals. *Psychosomatic Medicine*. 1988; 50(6):591–599. [PubMed: 3237948]
- Colantuoni C, Rada P, McCarthy J, Patten C, Avena NM, Chadeayne A, Hoebel BG. Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. *Obesity Research*. 2002; 10(6):478–488. [PubMed: 12055324]
- Colantuoni C, Schwenker J, McCarthy J, Rada P, Ladenheim B, Cadet JL, Schwartz GJ, Moran TH, Hoebel BG. Excessive sugar intake alters binding to dopamine and mu-opioid receptors in the brain. *Neuroreport*. 2001; 12(16):3549–3552. [PubMed: 11733709]

- Corwin RL, Buda-Levin A. Behavioral models of binge-type eating. *Physiology and Behavior*. 2004; 82(1):123–130. [PubMed: 15234600]
- Dimitriou SG, Rice HB, Corwin RL. Effects of limited access to a fat option on food intake and body composition in female rats. *International Journal of Eating Disorders*. 2000; 28(4):436–445. [PubMed: 11054791]
- Hagan MM, Moss DE. Persistence of binge-eating patterns after a history of restriction with intermittent bouts of refeeding on palatable food in rats: implications for bulimia nervosa. *International Journal of Eating Disorders*. 1997; 22(4):411–420. [PubMed: 9356889]
- Marcus MD, Kalarchian MA. Binge eating in children and adolescents. *International Journal of Eating Disorders*. 2003; 34(Suppl):S47–57. [PubMed: 12900986]
- Polivy J. Psychological consequences of food restriction. *Journal of the American Dietetic Association*. 1996; 96(6):589–592. quiz 593–594. [PubMed: 8655907]
- Rada P, Avena NM, Hoebel BG. Daily bingeing on sugar repeatedly releases dopamine in the accumbens shell. *Neuroscience*. 2005; 134(3):737–744. [PubMed: 15987666]
- Smith GP. Animal models of human eating disorders. *Annals of the New York Academy of Science*. 1989; 575:63–72. discussion 72–74.
- Spangler R, Goddard NL, Avena NM, Hoebel BG, Leibowitz SF. Elevated D3 dopamine receptor mRNA in dopaminergic and dopaminoceptive regions of the rat brain in response to morphine. *Brain Research Molecular Brain Research*. 2003; 111(1–2):74–83. [PubMed: 12654507]
- Will MJ, Franzblau EB, Kelley AE. The amygdala is critical for opioid-mediated binge eating of fat. *Neuroreport*. 2004; 15(12):1857–1860. [PubMed: 15305124]