



FOOD & ADDICTION

CONFERENCE ON EATING AND DEPENDENCE
NEW HAVEN, CONNECTICUT

JULY 2007

RUDD REPORT

RUDD CENTER FOR FOOD POLICY AND OBESITY
YALE UNIVERSITY

309 Edwards Street, New Haven, CT 06511-8369
p: 203.432.6700, f: 203.432.9674, e: rudd.center@yale.edu
www.YaleRuddCenter.org



The Rudd Center for Food Policy and Obesity at Yale University organized a conference on Food and Addiction on July 9 and 10, 2007. World experts in the fields of addiction, nutrition, obesity and policy assessed the current science of food addiction.

The participants examined the connection of food to chemical, physiological, and psychological dependence. It represented the first time that leading researchers, clinicians and policymakers gathered to discuss food addiction.

The conference addressed two major questions:

1. Is food addiction a meaningful concept?
2. If food can create an addiction process, how should the issue be addressed and by whom?

The objectives of the conference were to distill the available science, connect clinical observations with scientific evidence, propose plans for future research, and consider implications for public policy.



Co-Chairs

Kelly D. Brownell, PhD
Director, Rudd Center for Food Policy and Obesity
Professor of Psychology, Epidemiology and Public Health
Yale University

Mark S. Gold, MD
Chief and Distinguished Professor, McKnight Brain Institute
University of Florida College of Medicine

Keynote Speaker

Nora D. Volkow, MD
Director, National Institute on Drug Abuse, National Institutes of Health

Participants

Robert Alpern, MD
Dean; Ensign Professor of Medicine, Yale School of Medicine

Louis J. Aronne, MD
Clinical Professor, Department of Medicine, Weill Cornell
Medical School

Claude Bouchard, PhD
Director, Pennington Biomedical Research Center
George A. Bray, Jr. Chair in Nutrition, Louisiana State University

Elissa S. Epel, PhD
Director of Research, Center for Obesity Assessment, Study &
Treatment, University of California, San Francisco

Wayne K. Goodman, MD
Director, Division of Adult Translational Research and Treatment
Development, National Institute of Mental Health



Participants (continued)

Shelly Greenfield, MD, MPH
Department of Psychiatry, Harvard Medical School
Associate Clinical Director, Alcohol and Drug Abuse Treatment
Program, McLean Hospital

Steven B. Heymsfield, MD
Executive Director, Clinical Research, Metabolism, Merck
Research Laboratories

Bartley G. Hoebel, PhD
Professor of Psychology, Princeton University

Tamas L. Horvath, DVM, PhD
Chair, Section of Comparative Medicine; Professor of
Comparative Medicine, Neurobiology, and Obstetrics,
Gynecology, and Reproductive Sciences, Yale School of Medicine

Ellen Hunt
Director of Marketing and Communications, The Rudd Group,
Executive Director, Rudd Foundation

Satya P. Kalra, PhD
Professor of Neuroscience, McKnight Brain Institute, University
of Florida College of Medicine

Samuel Klein, MD
Professor of Medicine and Nutritional Science, Washington
University School of Medicine

Allen S. Levine, PhD
Dean and Professor, College of Food, Agricultural and Natural
Resource Sciences, University of Minnesota

Yijun Liu, PhD
Associate Professor, McKnight Brain Institute, University of
Florida



Participants (continued)

Barbara J. Mason, PhD
Director and Professor, Division of Clinical
Psychopharmacology, Pearson Center for Alcoholism and
Addiction Research, The Scripps Research Institute

Marina Picciotto, PhD
Professor of Psychiatry, Yale University School of Medicine

Eric Rimm, ScD
Associate Professor of Epidemiology and Nutrition, Harvard
University School of Public Health

Dana M. Small, PhD
Assistant Fellow, John B. Pierce Laboratory, Assistant Professor
of Psychiatry and Psychology, Yale School of Medicine

Gene-Jack Wang, MD
Scientist, Nuclear Medicine PET Medical Imaging, Brookhaven
National Laboratory

Stephen C. Woods, PhD
Professor, Department of Psychiatry;, Director, Obesity
Research Center, University of Cincinnati College of Medicine

Susan Z. Yanovski, MD
Director, Obesity and Eating Disorders Program, National
Institute of Health

Douglas M. Ziedonis, MD, MPH
Chair, Department of Psychiatry, University of Massachusetts
Medical School

Eric Zorrilla, PhD
Assistant Professor, Committee on the Neurobiology of
Addictive Disorders, The Scripps Research Institute



The purpose of this paper is to report on the key issues and topics discussed by the participants of the conference. The topics included:

- Drivers of Diet and Obesity
- Animal Models of Eating and Addiction
- Psychological Processes
- Human Models
- Policy Barriers and Implications



The obesity epidemic has drawn much attention from the media, researchers, scientists and public health officials. Continued high rates of obesity underscore the intractable nature of the problem despite mounting efforts to prevent and treat it. Food contributes to the epidemic in many ways:

- Composition and palatability
- Availability and affordability
- Marketing
- Sedentary lifestyle
- The built environment
- The school environment
- Gene-environment interactions
- Biological mechanisms
- Anxiety and stress

Obesity is such a complex disease that scientists continue to search for additional explanations. Food addiction may be one factor. If it is, the implications could be substantial in many areas such as food marketing to children and corporate liability for diet-related health problems.

Existing research into food addictions, which included animal and human studies, offers a number of provocative findings. For instance, there are striking similarities in use and withdrawal patterns of sugar and of classic drugs of abuse.

Reciprocal relationships among food and other substances (e.g., people tend to gain weight when they stop smoking or drinking) raise the possibility that food and classic addictive substances compete for the same brain pathways. It is important to know whether food or things commonly added to food (e.g., additives, high fructose corn syrup) can hijack the brain in ways similar to drugs of abuse.



The two-day conference began with a reception at the Rudd Center, followed by a keynote address by Nora Volkow, director of the National Institute on Drug Abuse, National Institutes of Health.

Participants met for a full-day meeting the following day. Robert Alpern, Dean of the Yale School of Medicine, delivered a welcome speech. Kelly Brownell, director of the Rudd Center, discussed the epidemic of obesity, and Mark Gold, chief of the McKnight Brain Institute, recounted lessons from tobacco addiction.

The participants divided into working groups to discuss issues related to food and addiction. At the end of the conference, the participants gathered in a large assembly to discuss next steps and future directions.



Dr. Volkow focused on the reward circuitry in the brain and its relationship to addiction in her keynote address, entitled *Common Brain Mechanisms in Addiction and Obesity: Insights from Neuroimaging*.

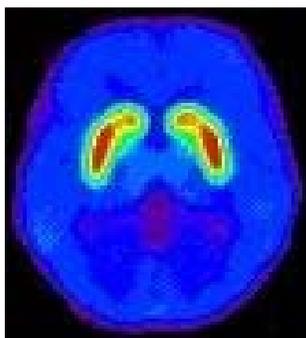
She said the reward circuitry is shared by addictive drugs and food, which might provide the theoretical foundation for studying food addiction.

Dr. Volkow paid particular attention to dopamine, a neurotransmitter well established as active in both food and drug intake mechanisms. Dopamine plays an important role not only in the reward system related to food and drug intake, but also in the motivation to consume both substances.

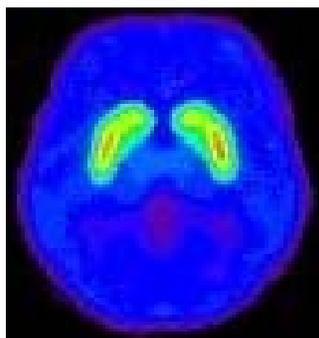
She also noted that compulsive overeating shares many of the same characteristics as drug addiction. Volkow said research into these two fields potentially can be mutually beneficial because of their similarities.

[11C]Raclopride

Binding in Food Deprived Controls During Neutral and Food Cues



Neutral



Food Cue

Source: Volkow, et al., Synapse 2002.



Working groups discussed various topics related to food and addiction.

During Phase 1, groups addressed the following questions:

- What is the definition of addiction?
- Why has the phenomenon of “food addiction” surfaced only in recent years?
- Are there alternate explanations for what may appear to be food addiction?
- What is known?
- What is suggested?
- What are the key unanswered questions?

During Phase 2, these questions were addressed:

- What would it take to accept that food addiction plays an important role in obesity?
- How can attention to the topic be raised?
- What can be done now?
- What can be done in the next five years?
- What can be done to build food addiction as a field of study?



Group A: Drivers of Diet and Obesity

Chair: Claude Bouchard, PhD, Director of Pennington Biomedical Research Center

The group made the following points in Phase 1 discussions:

- Addiction is a continuum of behavior from tolerance to withdrawal. It includes loss of control and continued use.
- Food addiction is plausible. It fits into the paradigm described above and can lead to weight gain.
- Some children today lack body signals that tell them they are full.
- The rise of a “food culture” and food environment supports overeating.
- Evidence points to food and food components as possibly addictive.
- Marketing has a key influence on food choice and the amount of food consumed.

During Phase 2 discussions the group discussed the need to gather more data to establish food as addictive, including:

- Replicate animal studies in humans to assess whether specific macronutrients promote addiction.
- Identify the circuitry in the brain that reacts to various foods.
- Ascertain which brain responses are most dramatic among those meeting the definition of addiction. Identify useful biomarkers or predictors.

**Group B:** Animal Models

Chair: Satya Kalra, PhD, Distinguished Professor Emeritus at the McKnight Brain Institute, University of Florida.

The group discussed the following regarding animal research in Phase 1 discussions:

- Deprivation of food components like sucrose and fat in a rodent diet gives rise to neurobiological functions that look as if rats are calorically deprived.
- Pre-treatment of animals with an opiate suppresses induced aversion to foods.
- Dopamine plays an important role in overall reward systems, and bingeing on sugar every day consistently produces dopamine. It is not yet known why sugar and perhaps fat might be different than other food components.
- Sugar-bingeing in some rat studies leads to symptoms of withdrawal when bingeing is prohibited. Rat studies, however, are controlled environments and do not necessarily translate to complex eating situations for humans.

In Phase 2, the group made these observations:

- The same behavioral criteria applied to drugs should be used for food when considering whether food is addictive.
- The overlap between reward systems for food and for drugs should be examined, especially in relation to cravings and continued consumption.
- Predictive models of obesity need to be developed to evaluate food addiction. The group was in agreement that food addiction research is a valid component of understanding obesity. The group believes food addiction does not require additional attention in the public. But it concluded that researchers should pay more attention to the relationship between drugs of abuse and obesity.



Group C: Psychological Processes

Chair: Marlene Schwartz, PhD, Deputy Director of the Rudd Center for Food Policy and Obesity.

The group members noted that the Diagnostic and Statistical Manual of Mental Disorders (DSM) has criteria for substance abuse and dependence, but does not include food addiction. For a condition to be characterized as a food addiction, the group suggested that three or more counterproductive behaviors must persist for a 12-month period. Some examples include: food consumption increases; attempts to control eating fail; other activities are neglected because of the food addiction.

The group explored the following questions:

- Are there subsets of people who are susceptible to abusing food as a substance? For example, one piece of cake might be satisfying to one child, whereas one piece might not satisfy another child.
- Are cravings to use drugs or substances different from cravings to eat food?
- What is the difference between people with binge-eating and overeating disorders versus those with “addictions”?

To establish food as an addiction, researchers must:

- Identify macronutrients or combinations of macronutrients (or food additives) that create neurobiological symptoms similar to addiction.
- Isolate behavioral phenotypes through self-reporting and laboratory studies, including consideration of variable eating behaviors, macronutrient preferences, and eating in the absence of hunger.
- Create validated, self-reported and behavioral measures for eating behaviors.

**Group D: Human Models**

Chaired: Elissa Epel, PhD, Director of Research at the Center for Obesity Assessment, Study & Treatment, University of California, San Francisco

This group explored the idea that if food addiction exists, it should share traits with substance abuse and dependence. For instance, neurochemical changes in the brain occur with drug addiction. When people move from use to abuse to dependence, they no longer use the substance to get high, but to feel normal. Another example is nicotine addiction. It can be preceded by untreated depression and is often the first addiction a person develops. Can we find similar conditions that precede food addiction?

Foods and addictive substances have important differences, in particular the sensory properties. Are there neuroadaptations in relation to food that are similar to substance abuse or dependence? Do people who are at risk for obesity have a greater “wanting”? Do those who are “addicted” to food have greater anticipatory reactions to smell or other sensory stimuli?

In alcohol addiction, some work shows that about half of alcoholics respond to visual and olfactory cues with increased craving. The types of cues however, vary across individuals. Importantly, if an addictive process with food exists, obese people are not going to be the only people with a strong “want” for food.



To have a clear understanding of the possibility of food addiction, we need models to help explain signaling mechanisms and their roles. The D2 receptor, for instance, has received a lot of attention in relation to dopamine, but very little research exists on the D3 receptor. Research also suggests that normal satiation and other signaling might lag in obese individuals, including signals related to insulin and leptin.

Other areas to explore include:

- Evaluate how homeostatic systems may be perturbed in different ways.
- Establish whether or not food addiction meets criteria for classical substance dependence.
- Acquire access to food industry documents on sweeteners and fats.



Group E: Policy Barriers and Implications

Chair: Wayne K. Goodman, MD, Director, Division of Adult Translational Research and Treatment Development, National Institute of Mental Health

The policy group made the following recommendation:

- Advocacy groups must mediate between researchers and policymakers so that research findings are translated into meaningful public policy. Researchers are not actively charged with making public policy, including policy related to food and addiction.
- Obesity prevention, not treatment, is the policy direction that should be pursued.
- To reach policymakers and prompt reform, we must publish policy papers, hold conferences, organize allies, and launch local grassroots efforts.
- Policymakers and scientists in attendance at this meeting must form relationships and continue to be in close contact.
- The work that has been done in the food addiction field should be translated into lay terms and summarized in short position papers and fact sheets for policymakers.

Policy should be focused on children because they are the most vulnerable to addictive behavior. Policymakers might begin by considering the issue of sugar and addiction by examining links with children, advertising, and food consumption patterns.



The researchers concluded the conference by agreeing to meet again to assess the available science critically and to reach a consensus on pressing research questions.

Because funding is always an issue in research, participants want to consider ways to secure more grants, especially for young investigators.

Many questions and ideas emerged during the conference that could guide future research and action:

- What evidence can be gathered from the study of extreme cases of obesity and self-reported “food addiction”?
- Is there evidence that tolerance and withdrawal occur with highly palatable foods?
- What are the precise interactions between consumption of foods and the use of other substances?
- Can we distinguish between the hedonic value of high-sugar/high-fat foods and their potentially addictive nature?
- Can we identify biomarkers of the effects of macronutrients with neuroimaging and other diagnostic techniques?
- How would a label of addiction affect the stigma related to obesity?
- Would a model including obesity surgery patients be useful, given reports of differences in substance abuse before and after surgery?
- What is the construct of disinhibition? Is it biological? What are the pathways?
- What are the effects of dieting? What happens in the brain reward systems when people restrict their food intake?
- If an addictive process occurs, what are the active food constituents (e.g., macronutrients, additives, etc.)?
- What are the legal implications of the production, distribution, and marketing of specific foods to specific populations (e.g., children) if food is found to be addictive?
- What legislative possibilities might emerge as the science on food and addiction builds?



The Food & Addiction Conference organized by the Rudd Center for Food Policy and Obesity enabled scientists from various fields to discuss and debate the issue of food and addiction. Participants believed the topic and its many layers of controversy offer great potential for future action and research.

They also agreed that another meeting is necessary with a wider cross-section of scientists and public health officials and should include presentations of relevant science for critical analysis.

Future efforts related to food and addiction should include strong recommendations that funding agencies increase support of work in this area, especially in support of young investigators.



- Adam TC, Epel ES. Stress, eating and the reward system. *Physiology & Behavior*. 2007;449-458.
- Avena NM, Long KA, Hoebel BG. Sugar-dependent rats show enhanced responding for sugar after abstinence: evidence of a sugar deprivation effect. *Physiology & Behavior*. 2005;359-362
- Avena NM, Rada P, Moise M, Hoebel BG. Sucrose sham feeding on a binge schedule releases accumbens dopamine repeatedly and eliminates the acetylcholine satiety response. *Neuroscience*. 2006;813-820.
- Christakis DA. The hidden and potent effects of television advertising. *From The Archives Journals*. 2006;1689-1699.
- Colantuoni C, Rada P, McCarthy J, et al. Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. *Obes Res*. 2002;10:478-488.
- DiFranza JR, Savageau JA, Fletcher K, et al. Symptoms of tobacco dependence after brief intermittent use. *Arch Pediatr Adolesc Med*. 2007;704-710
- Di Marzo V, Matias I. Endocannabinoid control of food intake and energy balance. *Nature Neuroscience*. 2005;585-589.
- Drewnowski A, Krahn DD, Demitrack MA, et al. Naloxone, an opiate blocker, reduces the consumption of sweet high-fat foods in obese and lean female binge eaters. *Am J Clin Nutr*. 1995;61:1206-1212.
- Epstein LH, Wright SM, Paluch RA, et al. The relationship between food reinforcement and dopamine genotypes on food intake in smokers. *Am J Clin Nutr*. 2004;80:82-88.
- Gold MS. Introduction. *Journal of Addictive Diseases*. 2004;1-3
- Hellmich N. Does 'food addiction' explain explosion of obesity? *USA Today*. 2007.
- Hoebel BG, Rada PV, Mark GP, Pothos EN. Neural systems for reinforcement and inhibition of behavior: relevance to eating, addiction, and depression. In: Kahneman D, Diener E, Schwarz N, eds. *Well-being: Foundations of Hedonic Psychology*. New York: Russell Sage Foundation; 1999:558-572.



James GA, Gold MS, Liu Y. Interaction of satiety and reward response to food stimulation. *J Addict Dis.* 2004;23:23-37.

Kalra SP, Kalra PS. Overlapping and interactive pathways regulating appetite and craving. *J Addict Dis.* 2004;23,5-21.

Kelley AE, Berridge KC. The neuroscience of natural rewards: relevance to addictive drugs. *The Journal of Neuroscience.* 2002;3300-3311.

Lam TKT, Schwartz GJ, Rossetti L. Hypothalamic sensing of fatty acids. *Nature Neuroscience.* 2005;579-584.

Spring B, Schneider K, Pagoto S, et al. Abuse potential of carbohydrate snacks for overweight carbohydrate eaters. *Ann Behav Med.* 2004;25:S035.

Volkow ND, Li TK. The neuroscience of addiction. *Nature Neuroscience.* 2005;1429-1430.

Volkow ND, Wise RA. How can drug addiction help us understand obesity? *Nature Neuroscience.* 2005; 555-560

Wang GJ, Volkow ND, Thanos PK, Fowler JS. Similarity between obesity and drug addiction as assessed by neurofunctional imaging: a concept review. *J Addict Dis.* 2004;23:39-53.