

Mini-Symposium

“Homeostatic and hedonic control of food intake”

Centre Hospitalier Universitaire Vaudois (CHUV) Lausanne, October 16, 2014

Auditorium Yersin, CHUV main building, 8th floor

Organiser: Prof. Luc Tappy

PROGRAM

- 8:00 – 8:30 Welcome coffee
- 8:30-8:40** **Welcome: Prof. Luc Tappy, Department of Physiology, UNIL, Lausanne, Switzerland**
- 8:40 -9:25** **Prof. Jason Halford (University of Liverpool, UK)**
Is Obesity a Psychological Disorder: The Food Environment and Appetite Regulation
- 9:25-10:10** **Prof. Serge Ahmed (University of Bordeaux, France)**
Sugar addiction: Pushing the drug-sugar analogy to the limit
- 10:10 – 10:30 Coffee Break
- 10.30-11.15** **Dr. Loic Briand (University of Bourgogne, Dijon, France)**
Taste receptors in oral and extraoral tissues: role in nutrient and metabolic sensing
- 11.15-12.00** **Dr. Loredana Asarian (University of Zurich, Switzerland)**
Estrogens: roles in eating and bariatric surgery outcome
- 12.15 – 14.00 Lunch
- 14:00 – 16:00** **Afternoon workshops for PhD students with symposium speakers**

This mini-symposium will be accredited by the Association of Cantonal Veterinarians (SCAV), section Lausanne, as a half day of continuing education.

This meeting is free of charge but for organization purposes we would like participants (limited to a maximum number of 120) to register by filling the form [here](#) prior to October 1, 2014.

The UNIL doctoral school attributes 1.0 ECTS for PhD students who present a signed participation form for the mini-symposium (0.25 ECTS morning session, 0.75 ECTS afternoon session).

For additional information, please contact Dr. Ulrike Toepel (ulrike.toepel@unil.ch).

Talk abstracts

Is Obesity a Psychological Disorder: The Food Environment and Appetite Regulation

*Jason Halford
Institute of Psychology Health and Society
University of Liverpool, UK*

The word 'addiction' - in the physiological sense - when applied to sugar still remains controversial. Obesity in general does not share key etiological factors or diagnostic features with addictive disorders. Moreover, care needs to be taken that we don't psychopathologise the obese - as their response is arguably a normal one to a food environment that 'pushes' foods and beverages high in fat, sugar and salt. Nonetheless, behavioural addiction to key foods (combinations of nutrients) does appear to be a significant problem for some individuals, and the concept of 'food addiction' may fit better with phenomena such binge eating, which appears increasingly common. Appetite control (and failure to control appetite) can be understood on behavioural, nutritional and physiological levels. The concept of the 'satiety cascade' allows us to understand how food components influence the factors that trigger, sustain, terminate and further inhibit food intake. In pure energy terms, all calories are equal whatever their source - but in behavioural terms they may not be. The addition of sugar and fats to foods and beverages can significantly increase their energy density, undermining short term episodic appetite control (leading to passive over-consumption at the eating episode). Furthermore, their post-consumption effect on appetite may be comparatively short in duration - thus hastening the onset of the next eating occasion. Studies have demonstrated that compared to complex carbohydrates, sugars tend to have only transient effects in reducing appetite. This could lead to weaker post-meal satiety, leading to subsequent over-consumption through incomplete caloric compensation at the next meal. Perhaps one of the major concerns is not around physiological effects of sugars upon appetite control mechanisms, but around the role of sweetness in influencing consumption. Palatability delays satiation and can contribute to active over-consumption - both triggering and sustaining eating episodes, and over-riding appetite control. On an individual level this is consistent with a behavioural food addiction - and more generally, the difficulties experienced by the obese in controlling their appetite. However, active over-consumption is not just a function of the individual. Instead, the operation of appetite must be considered in the wider context of the environment - not only the immediate environment it is expressed in, but the broader environment that has shaped it over a life time. The extensive promotion of - and ease of access to - highly palatable, energy dense foods which are cheap, convenient and ubiquitous have distinct effects on both the homeostatic and hedonic mechanisms that are critical to appetite expression. This, in itself, is more than sufficient to make added sugar an issue of major concern in tackling obesity and related health problems.

Sugar addiction: Pushing the drug-sugar analogy to the limit

Serge H. Ahmed

Université de Bordeaux, Institut des Maladies Neurodégénératives, CNRS UMR 5293, 146 rue Léo-Saignat, F-33000 Bordeaux, France

I will review research that tests the validity of the analogy between addictive drugs, like cocaine and heroin, and palatable foods, notably those high in added sugar (i.e., sucrose). Food desires are by far the most frequent and intense desires in human daily life. Current evidence, though still scant, shows that sugar and sweetness can induce reward and craving that are comparable in magnitude to those induced by addictive drugs. This evidence is now supported by recent experimental research on sugar and sweet reward in laboratory rats. Overall, this research has revealed that sugar and sweet reward can not only substitute to addictive drugs, like cocaine, but can even be more rewarding and attractive. At the neurobiological level, the neural substrates of sugar and sweet reward appear to be more robust than those of cocaine (i.e., more resistant to functional failures), possibly reflecting past selective evolutionary pressures for seeking and taking foods high in sugar and calories. The biological robustness in the neural substrates of sugar and sweet reward may be sufficient to explain why many people can have difficulty to control the consumption of foods high in sugar when continuously exposed to them.

Key-words

Sugar, cocaine, addiction, craving, dopamine, animal models

Taste receptors in oral and extraoral tissues: role in nutrient and metabolic sensing

Loïc Briand

Center for Taste and Feeding Behaviour - UMR6265 CNRS - UMR1324 INRA

Université de Bourgogne, Dijon, France

The sense of taste along with the sense of smell is one of the most important senses involved in the perception of food by humans. This sense is stimulated when fundamental nutrients or harmful compounds, such as toxic molecules activate specialized receptors located in taste buds. Humans are able to perceive and discriminate five different taste qualities, sweet, salty, sour, bitter, and umami (the taste of some amino acids such as glutamate). The perception of sweet taste is mediated by the T1R2/T1R3 receptor, which is expressed in the oral cavity, where it provides input on the caloric and macronutrient contents of ingested food. Cellular assays have shown that T1R2/T1R3 receptor recognizes all the chemically diverse compounds perceived as sweet by human beings, including natural sugars and sweeteners. Interestingly, the expression of a functional sweet taste receptor has been described in numerous extra-gustatory tissues including the gastrointestinal tract, pancreas, bladder, adipose tissues, and brain, where it has been proposed to regulate metabolic processes. This newly recognized role of the sweet taste receptor makes this receptor a potential novel therapeutic target for the design of inhibitors to treat certain related metabolic dysfunctions, including obesity and diabetes.

Estrogens: roles in eating and bariatric surgery outcome

Lori Asarian

*Institute of Veterinary Physiology and Center for Integrative Human Physiology
University of Zurich, 8057 Zurich, Switzerland*

Interest in sex differences in physiology and medicine is increasing; for example, the NIH of the USA is phasing in a requirement to balance sex in cell and animal studies, similar to the humans-subjects policy announced in 1993. Although markedly more women than men suffer from morbid obesity in the USA and other countries, and sex differences in physiology apparently contribute to this, work on sex-differences in eating behavior is relatively neglected. This lecture will focus on the role of female gonadal steroid hormones on eating and body weight regulation. This includes my recent interest in how Roux-en-Y gastric bypass surgery (RYGB) affects eating in females, who make up 85 % of patients who opt for bariatric surgery.

It is well known that rats eat about 20% less during the periovulatory (estrous) phase of the ovarian cycle than during diestrus and that ovariectomy both abolishes the cyclicity of eating and increases daily food intake above the diestrus maximum for several weeks, leading to increased body weight and adiposity. A near-physiological cyclic regimen of estradiol treatment that we developed was sufficient to maintain normal eating and body weight in ovariectomized rats. We have also discovered that estradiol treatment increases the potency of the gastrointestinal hormones CCK and GLP-1 to decrease eating. At least some estrogenic inhibitions of eating observed in rats also operate in women, supporting the clinical relevance of the basic animal research.

We recently reported described the first analyses of the effects of estradiol in a rat RYGB model. We found that estradiol treatment increased weight loss in ovariectomized RYGB rats, suggesting that the effects of RYGB may be lessened after menopause in women. An analysis of weight-loss outcome in a sample of women receiving bariatric surgery was consistent with that possibility.

The surgical re-arrangement of the gastrointestinal tract in bariatric surgery procedures and the resulting increase in release of GLP-1 and other hormones is thought to contribute importantly to changes in eating after RYGB. We discovered that the satiating efficacies of endogenous GLP-1 and endogenous CCK were significantly increased by estradiol treatment in our RYGB model. Unexpectedly, however, we did not detect a difference in these effects between RYGB and sham-operated rats.

Because previous studies did not assess the contributions of delivery of food to the jejunum *per se* after RYGB, we tested the effects of intrajejunal (IJ) lipid infusions on eating in RYGB or sham-operated female rats. We showed that the satiating action of IJ lipid is increased by RYGB and that antagonists to the gut peptides CCK and GLP-1 reversed the satiating effect of IJ lipid in RYGB rats, indicating that increases in endogenous CCK and endogenous GLP-1 satiation are increased by RYGB. The GLP-1 data extend reports that RYGB leads to (i) jejunal hypertrophy with an increased number of L-cells and (ii) increased prandial GLP-1 secretion by showing for the first time that contribution of endogenous GLP-1 to the satiating action of food in the jejunum is increased by RYGB. The CCK data similarly extend reports that RYGB increases prandial CCK secretion in human patients and should increase attention to this peptide in the therapeutic effect of RYGB. In these tests, however, estradiol did not affect the response to IJ lipid infusions. We are now conducting tests of IJ glucose infusions, and initial data suggest that estradiol does increase the response to IJ glucose in RYGB rats.

Supported by NIH grants DK 092608 and DK54523.