2010 Annual Meeting Abstracts

Food Structure & Functionality Forum

MONDAY

MORNING
FS&FF 1: Food-Body Interactions
Chair(s): I. Appelqvist, CSIRO, Food and Nutritional Sciences, Australia; and M. Paques, Royal FrieslandCampina, The Netherlands

Keynote Lecture: Overview of Field. L. Huang, Monell Chemical Senses Center, USA

Humans and other vertebrates are believed to be capable of detecting at least five primary taste qualities: sweet, bitter, savory, sour and salty. The sensation of taste begins in the oral cavity. Taste buds, each of which consists of 50-100 cells, are the peripheral endorgans of taste. The activation of taste receptors on taste bud cells by sapid molecules triggers intracellular signal transduction cascades, which eventually lead to the release of transmitters such as ATP and serotonin onto the afferent gustatory nerves. While each taste quality seems to be sensed by a particular subset of taste bud cells, many of these signal transduction components are common among these sweet, bitter and savory taste signaling pathways. Interestingly, a number of these signaling molecules, including the G protein-coupled taste receptors, heterotrimeric G proteins, effector enzymes and downstream ion channels have also been found in the epithelial cells of the gastrointestinal tract. Stimulation of these transduction pathways regulates the release of glucagons-like peptide-1 (GLP-1) and other hormones in the GI tract. Therefore, taste receptors and their signaling pathways are not only essential in the initial food assessment in the oral cavity but also participate in monitoring ingesta along the alimentary tract, and taste bud cells and gut chmosensory cells function coordinately in regulating food intake and nutrient absorption.

Gut-expressed Sweet Taste Receptor and Regulation of Intestinal Glucose Transport. S. Shirazi-Beechey, University of Liverpool, Liverpool, UK

Many of the receptors and downstream signalling elements involved in taste transduction are also expressed in enteroendocrine cells where they underlie the chemosensory functions of the gut. We showed, for the first time, that taste receptor 1, T1R, family members, T1R1, T1R2 and T1R3 are expressed in the intestinal epithelium and proposed that the sweet taste receptor, T1R2+T1R3, functions as the luminal sugar sensor. Recent work in our laboratory has determined that T1R2+ T1R3, and the partner G-protein, gustducin, are co-expressed in glucagon like peptide 1 (GLP-1), glucose-dependent insulinotropic peptide (GIP) and serotonin containing endocrine cells. We have been interested in the role played by the intestinal sweet taste receptor in regulation of the intestinal glucose transporter, Na+/glucose cotransporter 1, SGLT1. SGLT1 is the major route for the transport of dietary sugars from the lumen of the intestine into enterocytes. Regulation of this protein is essential for the provision of glucose to the body and, thus, is important for maintenance of glucose homeostasis. We demonstrated that dietary sugars and artificial sweeteners increase SGLT1 mRNA and protein expression and glucose-absorptive capacity in wild type mice, but not in T1R3, or gustducin, knockout mice, indicating that T1R3 and gustducin are required for enhanced expression of SGLT1 in response to lumenal sugars and sweeteners. The findings that gustducin and T1Rs reside in enteroendocrine cells, whereas SGLT1 is expressed in neighbouring absorptive enterocytes implies a signalling event taking place between the chemosensory enteroendocrine cells and absorptive enterocytes. I shall discuss progress made in understanding the mechanisms underlying this cell to cell communication.

Chemosensory Functions of the Brain. Ivan E. de Araujo, The John B. Pierce Laboratory & Yale University School of Medicine, USA

Specialized populations of cells located in regions adjacent to the brain ventricles are thought to act as chemosensors capable of assesing the chemical composition of the bloodstream. Chemicals sensed by these specialized neuronal cells

include nutrients such as glucose, lipids and amino acids, as well as minerals and an array of molecular ions. Of particular interest are the mechanisms involved in the sensing of extracellular glucose levels by neurons located in the hypothalamic area. Specifically, it remains unclear if the detection of extracellular glucose levels by ?glucosensing? neurons depends on the intracellular metabolism of glucose or, alternatively, on currently unidentified neuronal glucose membrane receptors. In this talk we will review our current knowledge on the physiological and molecular mechanisms underlying neuronal chemosensation. We will in particular review evidence both in favor and against the notion that neuronal glucosensing relies on the existence of membrane glucose receptors, and will present recent data suggesting that ?sweet? T1R2/T1R3 receptors might operate as one such brain glucose receptor. We will consider the possible physiological roles that may be associated with brain ?sweet taste receptors? and a parallel between brain and gut chemosensory functions will be outlined.

Gut Nutrient Sensing, Gut Hormone Release and Appetite Regulation. G. Frost, Imperial College London, UK

The aim of the lecture will be to give an overview of the relationship between foods, nutrient sensing in the gastro intestinal tract and appetite regulation. The lecture will focus on the role played by gut hormones and/or triggering CNS response. The intention is to debate the role of nutrient receptors in maintaining energy homeostasis. We will bring in the work carried from infusion of gut hormones as evidence for gut hormones as short term satiety signals, and our own team investigating central signalling. We will highlight our recent work investigating the sweet taste receptor T1R2-T1R3 and the fatty acids receptors GPR40, 41 and 43. One of the aims will be to highlight the importance of integrated physiology to understand these systems. I would also like to highlight the impact of physiological change in food structure as a potential method of regulating appetite.

Nutrient and Taste Receptors in the GI-Tract: Consequences for Food Preferences and Intake. R. Mattes, Purdue University, USA

It is widely acknowledged that the sensory properties of foods are critical determinants of food choice. The affective responses they elicit have inherent and learned components. The former provides the hedonic framework for judging food acceptability, but is modified through experience. This starts in utero and continues throughout life. The primary mechanism entails associative learning where the sensory properties of a food are paired with consequences of its ingestion and can be used subsequently to guide ingestive decisions. Recent findings have expanded understanding of the mechanisms and functions of sensory systems in this context. The concept of a limited number of taste primaries is being challenged by evidence of gustatory responses to compounds like fat that do not fall within traditional categories. Further, there is accumulating evidence that the transduction mechanisms for taste compounds in the oral cavity are functional throughout the gastrointestinal tract. Thus, the chemical signals in foods influence not only decisions to internalize a substance, but also its digestion, absorption and peripheral metabolism. Feedback from the combination of these processes then become the substrate upon which future decisions are made regarding what, when, how much and in what form that food should be consumed in the future.

Food Preferences Conditioned by Nutrient Actions in the Gut. A. Sclafani, Department of Psychology, Brooklyn College-CUNY, Brooklyn, NY, USA

Food preferences are determined not only by the orosensory (taste, odor, texture) properties of food but also by the post-oral actions of nutrients. Flavor preferences are conditioned in rodents by intragastric (IG) or intraduodenal nutrient infusions. These conditioned preferences are very persistent, can reverse the animal?s natural taste aversions (e.g., to bitter) and are produced by a variety of nutrients (carbohydrate, fat, protein). The presence of taste signaling proteins in the GI tract suggests that conditioned preferences might be mediated by gut ?taste? cells, but this has not been confirmed. Knockout mice missing the T1R3 sweet taste receptor do not prefer sweet solutions, but learn to prefer flavors paired with IG sucrose solutions. Also, the effectiveness of IG glucose but not fructose infusions to condition flavor preference suggests that carbohydrate conditioning is mediated by a glucose sensor rather than a sweet receptor. Similarly, although CD36 may act as a gustatory lipid receptor, CD36 knockout mice learn to prefer flavors paired with IG fat infusions. A greater understanding of the gut nutrient sensing pathways that mediate food preference learning may lead to new approaches to diet-related disorders.Supported by DK31135

A Gut Feeling for Satiety?Are Gut Receptors a Potential Target for Energy Intake Control?. Martin Foltz,

Unilever R&D, Vlaardingen, The Netherlands

Developing functional foods calls for fundamental understanding of the physiology of the benefit area and includes identification of key regulators targetable with foods. The GI tract is a potential target region for influencing body weight as recent findings show that energy intake is *inter alia* determined by gastrointestinal stimuli.A variety of nutrients triggers gut hormone secretion and activate the vagus. The underlying initiation events seems to include activation of receptors and transporters sensitive towards fatty acids, peptides, amino acids, sweet and bitter compounds. The true physiological role of specific gastrointestinal nutrient receptors and their overall contribution for regulation of energy intake is poorly understood. We have recently characterized selected bitter receptors from the TAS2R family, the peptide receptor GPR93 and the umami receptor T1R1/R3. In the presentation *in vitro*, *ex vivo*, and animal *in vivo* data will be presented. The results point to a potential route of action by which selected natural compounds might influence appetite control. Our data provide additional evidence that bitter and peptide sensing mechanisms are coupled to hormone release from enteroendocrine cells. Their relevance as molecular target for influencing energy intake in man needs testing using specific food grade receptor antagonists.

Moderated Discussion.

AFTERNOON

FS&FF 2: Design of Successful Performing Interfaces

Chair(s): K. Dewettinck, Ghent University, Belgium; and Y. Wang, Kraft Foods Inc., USA

Factors Governing Partial Coalescence in Oil-in-Water Emulsions: a Review. K. Dewettinck[1], E. Fredrick[1], P. Walstra[2], [1]Ghent University, Belgium, [2]FrieslandCampina, The Netherlands

The consequences of the instability mechanism partial coalescence in oil-in-water food emulsions show a discrepancy. On the one hand, it needs to be avoided in order to achieve an extended shelf life in food products like sauces, creams and several milk products. On the other hand, during the manufacturing of products like ice cream, butter and whipped toppings partial coalescence is required to achieve the desired product properties. It contributes to the structure formation, the physicochemical properties (stability, firmness, overrun) and the sensory perception, like fattiness and creaminess of the final food products. Both the process parameters, like the flow condition, the temperature history and the actual temperature, and the formulation of the oil-in-water emulsions, defining the distribution of the components between the continuous aqueous phase, the dispersed oil phase and the interfacial layer, determine to a large extent the rate of partial coalescence. This review critically summarises the findings of partial coalescence in oil-in-water emulsions in order to provide insight in how to enhance and retard it. Next to the pioneering work, a large set of experimental results of more recent work is used to illustrate the key factors.

Mechanism of Lipolysis and Micellization of β-carotene from Oil-in-Water Emulsions Stabilized with Soy Protein Isolate. A. Malaki Nik, M. Corredig, A.J. Wright, University of Guelph, Guelph, Ontario, Canada

The design of novel interfaces to control the behavior of food colloids during digestion, including lipolysis is an exciting area of research. In the current study, the effect of the oil-water interfacial structure on the lipolysis of an oil-in-water emulsion stabilized with SPI and the subsequent release of β-carotene (BC) was investigated. Particle size distribution of emulsion was significantly altered during simulated gastric digestion. However, the introduction of bio-surfactants, mainly bile salts (BS) and phospholipids (PL) during the duodenal stage resulted in re-emulsification of the oil droplets. Combined with particle size distribution, a significant increase in negative charge was obtained during the 2 h duodenal digestion, i.e. from -40 to -70 mV, indicated that proteins and peptides were displaced in the presence of BS. The pancreatic lipase activity was monitored by the release of free fatty acids. In the presence of BS alone, 60% lipid hydrolysis occurred, while addition of PL was found to inhibit lipolysis and the introduction of colipase significantly increased the extent of lipolysis up to 85%. Investigations of BC micellization showed 70 % transfer in presence of BS alone and addition of PL and colipase affect the rate of BC micellization. The results of this study

would enable food industry to design high-quality healthy foods.

Stability and Controlled Release Properties of Double Emulsions. L. Sapei, D. Rousseau, Ryerson University, Toronto, ON, Canada

Food-grade water-in-oil-in-water (W/O/W) double emulsions for the controlled release of food ingredients were developed. Emulsions were prepared with deionized water and canola oil, as well as polyglycerol polyricinoleate (PgPr) and polysorbate 80 as emulsifiers for the primary water-in-oil (W/O) emulsions and secondary (W/O/W) emulsions, respectively. Sodium chloride (NaCl) was entrapped within the internal aqueous phase as a simple model compound to evaluate the stability and release properties of the emulsions. The internal aqueous phase was gelled with gelatin to improve emulsion stability and slow the release of NaCl from the internal aqueous phase towards the external aqueous phase. Stability of the W/O/W emulsions was characterized via sedimentation, optical microscopy, particle size analysis, and conductivity to examine NaCl release profile. There was a significant decrease in the total release and release rate of NaCl with gelatin incorporation compared to the control double emulsions. Release of NaCl was also dependent on initial loading. The initial average oil globules size increased from 45 to 65 μm with an increase of NaCl concentration from 2% to 8% in the internal aqueous phase. All W/O/W emulsions had a high initial salt retention in the inner aqueous phase (>90%), which was very promising for the encapsulation of food ingredients and their controlled release.

The Development of a One-step Double Emulsion Formation Protocol. M. Pradhan, D. Rousseau, Ryerson University, Toronto, ON, Canada

The development of double emulsions typically involves a two-step process whereby an internal emulsion is dispersed into another continuous phase via secondary emulsification. We have now developed a single-stage double emulsification protocol for the development of oil-in-water-oil (O/W/O) emulsions that only requires one surfactant. Two different n-alkane oils and three different emulsifiers were studied, with focus placed on a formulation containing mineral oil, glycerol monooleate (GMO) and deionized water. Depending on the ratio of GMO and water, a critical zone emulsification existed where the O/W/O emulsion yield and stability were highest. Phenomenologically, double emulsion formation originates from the charging of the oil/water interface during emulsification which is balanced by the emulsification capacity of the GMO. At a critical ratio, GMO?s capacity to promote water-in-oil (W/O) emulsion formation is counterbalanced by interfacial charging leading to extensive double emulsification. The role of pH was also investigated, and showed that higher double emulsification occurred at basic pH (