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Editorial overview: Diet, behavior and brain function: You are what you eat: Effects of the modern food environment on brain and behavior

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Dana M Small is the deputy director of The John B Pierce Laboratory and professor of Psychiatry at Yale University School of Medicine. She received her MSc degree in Neuroscience in 1998 and her PhD in Clinical Neuropsychology in 2001 from McGill University. Professor Small is a pioneer in the neuroimaging of taste, flavor and feeding in humans. Her early work characterized the neural circuits underlying food reward, highlighting a role for dorsal striatal dopamine circuits. More recently her laboratory has described the deleterious effects of obesity and diet on these circuits. Professor Small has received several awards for her work including the Allan Epstein award for contributions to understanding ingestive behavior and The Ruth Pike Award for nutrition research. In 2014 she was appointed to the National Academy of Sciences Board on Behavioral, Cognitive and Sensory Sciences (BBCSS) where she is leading an effort to further understand the influence of diet, adiposity and metabolism on cognitive functions. Her laboratory is funded by NCI, NIDDK, NIDA and NIDCD.

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''*Tell me what you eat and I will tell you who you are*,'' — *Jean Brillat-Savarin 1826*

Changes in the food environment can have a profound impact on health and physiology. In his book 'Catching Fire: How Cooking Made us Human' Harvard anthropologist Richard Wrangham argues that cooking made us human by reducing the need for metabolically costly intestinal tissue which resulted in the selection of bigger brains and smaller guts. The crux of the hypothesis is that cooking helps to break down difficult to metabolize components of foods so that the energetic cost of metabolizing is reduced and the net energy gained from consumption is increased.

Although the changes in today's food environment may not equate with the advent of fire, the effects on energy balance may nevertheless be similarly profound. In many ways the crux of Wrangham's hypothesis plays forward. Today food costs less, is easier to obtain and not only is it easier to metabolize 'modern' foods but foods and beverages proffer up nutrients in doses, mixtures and forms not previously encountered in our evolutionary past. We also eat more frequently and we have greater choice. The effect of this mismatch is the obesity epidemic.

This special issue brings together findings from diverse perspectives suggesting that the mismatch between our food environment and our physiology is producing deleterious effects on the brain and brain function that reaches beyond homeostatic regulation, to affect perception, cognition, and affective regulation. Four emerging themes are highlighted; first, direct effects of nutrients on brain and behavior, second, evidence and mechanisms linking diet/obesity to cognitive dysfunction, third, pathways to compulsive feeding, and fourth, moderating factors such as stress, food advertisements and peer pressure.

Direct effects of nutrients on brain and behavior

Growing evidence suggests that dietary lipids and carbohydrates can directly impact behavior and brain function. In their paper Berland *et al*. consider evidence that dietary triglycerides (TGs) act directly on mesolimbic dopamine neurons to reduce desire for food following a meal. However, when chronically elevated this TG-sensing mechanism becomes desensitized so that motivation for food reward becomes resistant to TG-mediated homeostatic control. They also propose a novel model by which chronic intake of dietary TGs might produce adaptations in dopamine D_2 receptors (D_2R)

in Neuroscience in 2001 from the University of Amsterdam. She then worked as a postdoctoral fellow with Prof MF Dallman at the University of California, San Francisco. Dr la Fleur pioneered the idea to provide animals with choice to consume fat and sugar separate from pelleted chow as a new obesogenic animal model and showed with this the importance of frequent snacking in obesity development and stress reducing properties of palatable intake. Her current interest is to understand how nutrients affect the brain and how these changes mediate the overeating and metabolic problems as observed in obesity. Dr la Fleur received several awards for her work including the Novo Nordisk award for Endocrinology and the Alan N Epstein award from the Society of the Study of Ingestive Behavior. Her work is currently funded by the Dutch Technology Foundation (STW), the Dutch Science Foundation (NWO), and the European Union.

leading to the development of compulsive intake as described in the paper by O'Connor and Kenny.

The paper by de Araujo distinguishes two pathways by which dietary fats gain access to reward circuits. The first is a sensory pathway whereby the orosensory properties of fat-containing foods are relayed to the brain and elaborated to produce flavor perceptions and pleasure. The second pathway, by contrast, involves lipid-triggered gut signals that are conveyed to the dorsal striatum via the vagus nerve. This pathway is proposed to operate subliminally so that information about the nutritional content of food gains direct access to reinforcement circuits. As a result gut-derived signals bypass cortical circuits to directly engage motor programs outside of awareness or insights in to the nature of the choices. Repeated activation of this axis is then suggested to permit the emergence of habit-like, automatic action schemata that prompt fat ingestion while precluding overt insights in the nature of dietary choices. A similar opinion is reached by Burke and Small who review evidence that it is the energetic, rather than the pleasurable properties of foods that regulate nucleus accumbens responses to food cues, a demonstrated predictor of weight gain susceptibility.

Décarie-Spain *et al*. bring attention to the variability of findings from studies examining effects of high-fat feeding on brain and behavior. They highlight the need for considering differences in diet content, duration, weight gained and metabolic alterations in understanding effects of fat intake. For example, saturated dietary lipids can promote metabolic dysfunction and adversely affect dopamine signaling and function whereas mono- and poly-unsaturated fatty acids can be protective. They also suggest a timeline in which fat intake and adiposity interact so that alterations in dopamine transporter functions occur first followed by an inhibition of dopamine availability and release after full-scale obesity is reached.

Akin to fats, all sugars are not created equally. Page and Melrose offer a review of literature showing that glucose and fructose have differential effects on physiology and feeding, mediated in part by their distinct effects on neuroendocrine circuits. Whereas glucose metabolism leads to the release of satiety hormones and induction of other physiological events associated with satiety, fructose fails to stimulate these signaling cascades and may even produce opposing effects on hypothalamic circuits leading to increased intake. In her paper Swithers provides a critical review of the literature examining the effects of non-nutritive sweetener consumption on body weight and metabolic health. She makes a convincing case for the existence of 'probable doubt' in the safety of these substances and discusses potential mechanisms by which non-nutritive sweeteners may produce rather than prevent metabolic disorders.

De Jong and colleagues further shine the spotlight on sugar consumption asking whether there is sufficient evidence to conclude that sugar has addictive qualities that are comparable to those of addictive substances. They provide an incisive review of the literature and argue that while sugars clearly produce short and long term effects on the brain they do not induce plasticity changes that readily occur with drugs of abuse. Rather, they suggest that ingestive behaviors such as binging play a causal role and propose reframing food addiction as 'eating' addiction.

Finally, considering both fat and sugar, Keast examines the role of diet on perception. Although this literature produces inconsistent findings, bi-directional associations appear to exist where individual differences in