How junk food primes the brain's food-seeking behavior

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Credit: Maliz Ong/public domain

(Medical Xpress)—The current epidemic of obesity in developed countries should be a warning for health officials in the developing world with newly opened markets. Food manufacturers, restaurant

franchising companies, food supply chains and advertisers collaborate to create environments in which extremely palatable, energy-dense foods and their related cues are readily available; however, people still have adaptive neural architecture best suited for an environment of food scarcity. In other words, the brain's programming may make it difficult to handle the modern food ecosystem in a metabolically healthy way.

Humans, like all animals, have ancient genetic programming adapted specifically to ensure food intake and food-seeking survival behaviors. Environmental cues strongly influence these behaviors by altering neural architecture, and corporations have refined the science of leveraging human pleasure response and perhaps inadvertently reprogramming people's brains to seek surplus calories. In an environment that is rich in highly palatable, energy-dense foods, the pervasiveness of food-related cues can lead to food seeking and overeating regardless of satiety, a likely driver of obesity.

A group of Canadian researchers at the University of Calgary and the University of British Columbia recently published the results of a mouse study in the *Proceedings of the National Academy of Sciences* in which they explored the neural mechanisms behind these changes in food-seeking behavior.

Programming future food approach behaviors

They report that the short-term consumption of extremely palatable food—specifically, sweetened high-fat food—actually primes future food approach behaviors. They found that the effect is mediated by the strengthening of of excitatory synaptic transmission onto dopamine neurons, and lasts for days after initial 24-hour exposure to sweetened high-fat foods.

These changes occur in the brain's ventral tegmental area (VTA) and its

mesolimbic projections, an area involved in adapting to environmental cues used for predicting motivationally relevant outcomes—in other words, the VTA is responsible for creating cravings for stimuli found to be rewarding in some way.

The researchers write, "Because enhanced excitatory synaptic transmission onto dopamine neurons is thought to transform neutral stimuli to salient information, these changes in excitatory synaptic transmission may underlie the increased food-approach behavior observed days after exposure to sweetened high-fat foods and potentially prime increased food consumption."

Possible therapeutic approaches to obesity

The enhanced synaptic strength lasts for days after exposure to high-energy-density food, and is mediated by increased excitatory synaptic density. The researchers found that introducing insulin directly to the VTA suppresses excitatory synaptic transmission onto dopamine neurons and completely suppresses food-seeking behaviors observed after 24-hour access to sweetened high-fat food.

During that period of food access, the number of glutamate release sites onto dopamine neurons increases. Insulin acts to block those sites, competing with glutamate. Noting that this suggests a possible therapeutic approach to obesity, the authors write, "Thus, future work should determine whether intranasal insulin can decrease overeating due to food priming induced by palatable food consumption or food-related cues."

More information: Consumption of palatable food primes food approach behavior by rapidly increasing synaptic density in the VTA. *PNAS* 2016 ; published ahead of print February 16, 2016, DOI: 10.1073/pnas.1515724113

Abstract

In an environment with easy access to highly palatable and energydense food, food-related cues drive food-seeking regardless of satiety, an effect that can lead to obesity. The ventral tegmental area (VTA) and its mesolimbic projections are critical structures involved in the learning of environmental cues used to predict motivationally relevant outcomes. Priming effects of food-related advertising and consumption of palatable food can drive food intake. However, the mechanism by which this effect occurs, and whether these priming effects last days after consumption, is unknown. Here, we demonstrate that short-term consumption of palatable food can prime future food approach behaviors and food intake. This effect is mediated by the strengthening of excitatory synaptic transmission onto dopamine neurons that is initially offset by a transient increase in endocannabinoid tone, but lasts days after an initial 24-h exposure to sweetened high-fat food (SHF). This enhanced synaptic strength is mediated by a long-lasting increase in excitatory synaptic density onto VTA dopamine neurons. Administration of insulin into the VTA, which suppresses excitatory synaptic transmission onto dopamine neurons, can abolish food approach behaviors and food intake observed days after 24-h access to SHF. These results suggest that even a short-term exposure to palatable foods can drive future feeding behavior by "rewiring" mesolimbic dopamine neurons.