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Sugar and reward system: Is high sugar intake generating akin effects like drugs of abuse?

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Sugar is an imperative part of our diet. Only a tiny part of us pass a usual day deprived of adding sugar to typical meal. It is travail to exactly estimate sugar content in diet and food stuffs owing to its accessibility and affordability. Glucose, a genre of sugar, is the prime cradle of energy for every cell in the body [1]. Among the human body organs, the brain is the cardinal sugar energy-utilizing organ. Augmented sugar intake is considered one of the foremost factors for copious snags, including obesity and type II diabetes, but little is identified concerning the effects of enduring sugar intake on the brain.

Evidence proposes that high-sugar intake is accountable for intense neural vicissitudes in the brain areas intricate in reinforcement. The amygdala, particularly the basolateral amygdala (BLA) neurons is recognized to facilitate the reinforcing assets of drugs of abuse [2]. Ambroggi *et al.* reported that BLA input is requisite for dopamine to augment the firing of nucleus accumbens (NAc) [3]. The NAc is linked in the cognitive processing of reward, comprising particular penchant retorts to certain pleasant stimuli, motivational salience in addition to reinforcement [4,5]. The BLA gets dopaminergic input from the VTA and conveys glutamatergic projections to the medium spiny neurons (MSNs) in the NAc. MSNs are crucial assembly of the mesolimbic reward circuit [6].

Currently Shariff *et al.* examined the consequence of short- and long-term binge-like sucrose intake on the morphology of the BLA neurons by the help of an intermittent-access two-bottle choice paradigm [7]. The researchers used Golgi-Cox staining to impregnate principal neurons from the BLA of short- (i.e. 4 weeks) and long-term (i.e. 12 weeks) sucrose consuming adolescent rats and compared these to age-matched water controls. The denouements indicate possible maladaptive changes to the dendritic architecture of BLA principal neurons, particularly on apical dendrites following long-term sucrose consumption. Specifically, the consequences show reduced total dendritic arbor length of BLA principal neurons following short- and long-term sucrose intake. Furthermore, researchers found that long-term binge-like sucrose intake caused a significant reduction in the length and complexity of apical dendrites. Taken together, the results highlight the differences between short- and long-term binge-like sucrose intakes on BLA principal neuron morphology and are suggestive of a perturbation in the diverse synaptic inputs to these neurons.

Klenowski *et al.* stated that persistent intake of sucrose in a binge-like way, changes the morphology of medium spiny neurons of the NAc [8]. The conundrums presented ominously declined in the total dendritic length of NAc shell MSNs with respect to age-matched control rats owing to continual binge-like sucrose intake. They also reported that the reform of these neurons caused predominantly from abridged distal dendritic complexity. Contrariwise, in case of rats ingesting sucrose an increased in spine densities at the distal branch orders of NAc shell MSNs was observed. The concerns of this study augment excellence to the assumption that sugars (i.e. sucrose) have addictive possessions succeeding durable, binge-like intake (Figure 1).

Numerous studies have suggested that chronic acquaintance to drugs of abuse (i.e. alcohol, morphine, opium, cocaine, amphetamines) is accountable for cellular and morphological hallmark [9,10] that are also similar to sugar. Not only sugar, but also high fat diet is also causative to generate addiction like state. In a study by Dingess *et al.* inspected exposure to high fat diet diminishes dendritic spine density in the medial prefrontal cortex [11]. Another study by Thompson *et al.* examined obesity-persuaded structural and neuronal plasticity in the lateral orbitofrontal cortex [12]. The outcomes of these studies suggested a substantial decrease in the density of spines and declined in inhibitory synaptic transmission in the prefrontal cortex which is profoundly linked with reward processing.

The comparison between sugar and drugs of abuse is a gullible alternative way. Undoubtedly, it is observed that natural rewards (i.e. sugar) and drugs of abuse (i.e. morphine, opium, cocaine, amphetamines) may share a few usual neuronal characteristics, but the real outcome of drugs of abuse is very precarious. This incongruity between sugar and drugs of abuse prompted structural alterations may

Figure 1. Sugar exerts a similar sort of addictive effect on society that drugs of abuse also reach.

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specify that both rewards (i.e. sugar and drugs) may affect distinct landscapes of the neuronal homeostasis. However, further researches are indispensable to illustrate the exact mode of actions triggering sugar addiction facts.

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Competing interests

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