A Shared Molecular and Genetic Basis for Food and Drug Addiction

Overcoming Hypodopaminergic Trait/State by Incorporating Dopamine Agonistic Therapy in Psychiatry

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KEYWORDS

• Molecular basis • Genetic basis • Food addiction • Drug addiction
• Hypodopaminergic state • Dopamine agonistic therapy • Psychiatry

KEY POINTS

• A brief history is presented of the importance of the molecular neurobiology and neurogenetics of reward brain circuitry in addiction.

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- Shared common mechanisms exist between food and drug addiction with emphasis on similar neurochemical brain changes in acute and chronic conditions.
- Treatment approaches are listed arguing for dopaminergic agonistic therapy rather than dopaminergic antagonistic therapy.
- A genetic addiction risk score needs to be developed for early age identification of food and or drug addiction risk.
- Policymakers should be convinced that they must develop public health messages like they did with tobacco and use other tactics including taxation to find new treatments that target our youth as preventive measures.

INTRODUCTION

How we feel when we are starving or miss a meal, or two, may be linked to the brain's complex role in controlling appetite. This understanding may be crucial in efforts to develop better ways of helping the millions of Americans afflicted with obesity.[1]

There may be useful ways to identify the role of food addiction in the obesity pandemic. Obesity is rapidly surpassing smoking as the number one killer in the industrialized world. The cost is an estimated $117 billion annually in related illnesses and loss of productivity.[2,3] Food addiction does not explain all cases of obesity, and as the number of persons diagnosed with obesity continues to increase, many people are seeking answers. The increased number of people who eat more food than is required for the basic energetic needs suggests that food intake is no longer simply for purposes of survival.[4] Behavioral and brain changes resembling the effects of drugs of abuse have been observed in rats trained to overeat sugar solution.[5–7] Another similarity was observed in that the dopaminergic reward circuitry of the brain system was involved in animals overfed highly palatable foods.[8–13]

The Molecular Aspects of Dopamine in Reward Circuitry

Feelings of well-being are controlled by dopamine (DA), a neurotransmitter in the brain. The healthy interaction of neurotransmitters, such as serotonin, the opioids, and other brain chemicals, with DA results in feeling well and happy. Depression in contrast has been associated with low serotonin levels.[14] Drug development for the treatment of neurologic, psychiatric, and ocular disorders has targeted DA receptors, a class of G-protein-coupled receptors.[15]

Recently, Salamone and Correa,[16] Sinha,[17] and Nutt and colleagues[18] have debated the claim that DA is the "antistress" or "pleasure" molecule. Nutt and colleagues[18] argue that although addiction is viewed as a disorder of the DA neurotransmitter system, this view has not led to new treatments. Although true, it is not because DA is not a key neurotransmitter in the addiction process requiring not only release in the human striatum but also appropriate responsivity in terms of receptor function. Nutt suggests that only stimulants like cocaine and alcohol require dopaminergic function not cannabis, heroin or nicotine.

The authors do not agree with this oversimplification and intend to show the relationship between glucose cravings and other drugs of abuse. Accordingly, they have argued[19–21] that of depression and other disorders.[22] According to Salamone and Correa, labeling DA neurons as reward neurons may be an overgeneralization;