Weak link between food addiction and obesity in humans

Addiction to food cannot explain the current global obesity epidemic. In a recent review, researchers from the University of Cambridge, UK, closely examined the widely claimed link between obesity and addiction. They demonstrated that the evidence for food addiction in humans is limited, and that there are issues with the usefulness of the current food addiction model which need to be addressed.

According to the authors, the view that neurobiological concepts related to addiction could help explain obesity is gaining consensus among researchers. This 'food addiction model' encompasses two main ideas. The first is that certain foods may be addictive, acting on the brain to shape behaviours in a similar way to drugs of abuse. The focus here has so far been on processed foods, although questions remain as to exactly which nutrient (or nutrients) may be addictive, and how. The second idea is that food addiction consists of a set of behaviours which are found in a select group of obese people. Again, these behaviours are said to closely resemble those seen in drug addiction.

To find out how relevant the food addiction model is to obesity, the authors reviewed five key areas of evidence. First, they looked at the clinical overlap between obesity and drug addiction. While there are some similarities in patterns of behaviour, it is very difficult to apply the criteria used in defining drug addiction to the task of defining food addiction. Unlike drugs, food is necessary for survival and is relatively easy to obtain. The authors found that clinical overlaps are most apparent when looking at people with binge-eating disorder (BED). People with this disorder share many behaviours with people addicted to drugs, including loss of control of eating, escalating consumption, and compulsivity.

A second line of evidence comes from family studies, which hint at shared genetic susceptibilities between obesity and substance addiction. However, the authors argue that the underlying processes may not necessarily be the same in both conditions. In contrast, animal studies provide most evidence for food addiction. Research has shown how rats overexposed to high-sugar and/or high-fat diets can develop addictive behaviours over short periods of time. A combination of high-sugar and high-fat foods also produces overeating and obesity in these animals. Importantly, these results have not been replicated in humans.

The authors also reviewed the neurobiological evidence for food addiction. While it has been suggested that obese people's brains have reduced dopamine binding (dopamine is a chemical involved in reward and addiction), the results of studies in this area are very complex and do not

support an addiction model. Functional imaging studies have looked at altered brain responses to food-related stimuli among the obese. However, results have been variable and inconsistent here, too. The authors propose that overeating may be too complex to get a consistent result from these studies, and that the many genetic and environmental factors which contribute to obesity need to be considered in future.

In short, there is inconsistent evidence to support a direct link between food addiction and obesity. Moreover, the food addiction model currently has limitations for studying obesity. However, the authors are not wholly negative about the model. They argue that it could be potentially useful for understanding and treating obesity if it were made more sophisticated and precise in future to take into account the complexity of this condition.

References

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