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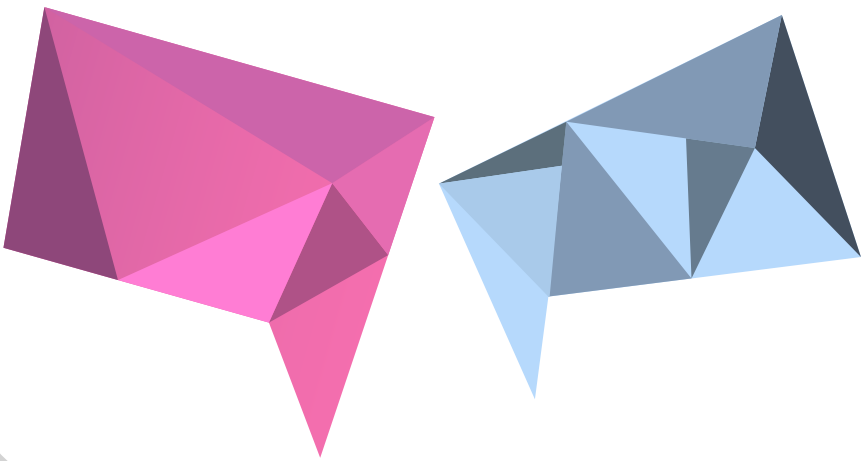
CONSENSUS

NeuroFAST consensus opinion on food addiction

Researchers of NeuroFAST met in Utrecht in February 2013 to discuss addiction in the context of food intake and to develop a common consensus on this important issue. The novel category "Substance-Related and Addictive Disorders" which has just recently been included in the Fifth Diagnostic and Statistical Manual of Mental Disorders (DSM-5) published by the American Psychiatric Association in May 2013 allowed us to structure our discussion accordingly. The prior version of the Manual (DSM IV TR) had only referred to Substance Use Disorders. The inclusion of non-substance based addictive disorders opens the door for the future definition of such behaviors. Indeed, the first and currently only disorder termed Gambling Disorder has been subsumed under this extended DSM 5 category.

The psychiatric classification is important because the listed disorders represent official disorders whose treatment should be reimbursed e.g. by health insurers. Disorders newly listed in the Manual can thus be perceived as becoming part of official medicine. It is however important to realize that the DSM classification system is mainly used for research purposes; in Europe and many other parts of the world, psychiatric disorders are classified according to the International Classification System published by the World Health Organization. The current version ICD-10 was published in 1992; efforts are ongoing to develop the future ICD-11 whose publication is scheduled for 2015. It is currently unknown if the ICD 11 category Substance Use Disorders will also be changed to allow inclusion of non-substance based addictive disorders. Undoubtedly, DSM 5 is setting the trend, but the recent controversies related to novel disorders listed in DSM 5 will influence which additions will also make it into ICD 11.



Consensus statements

After a thorough discussion of issues related to the potential existence of food addiction as a disorder, the NeuroFAST research group agreed on the following statements:

- Current evidence does not allow us to conclude that a single food substance via a single specific neurobiological mechanism (e.g. specific brain receptors or specific neuronal pathways) can account for the fact that people overeat and develop obesity.
- In humans, there is no evidence that a specific food, food ingredient or food additive causes a substance based type of addiction (the only currently known exception is caffeine which via specific mechanisms can potentially be addictive). Within this context we specifically point out that we do not consider alcoholic beverages as food, despite the fact that one gram of ethanol has an energy density of 7 kcal.
- Addictive (over)eating is clearly distinct from substance use disorders that cause addiction via specific mechanisms (e.g. nicotine, cocaine, cannabinoids, opioids, etc).
- An addiction-like eating behavior may, in rare instances, be caused by mutations in single genes which entail an elevated feeling of hunger and reduced satiety.

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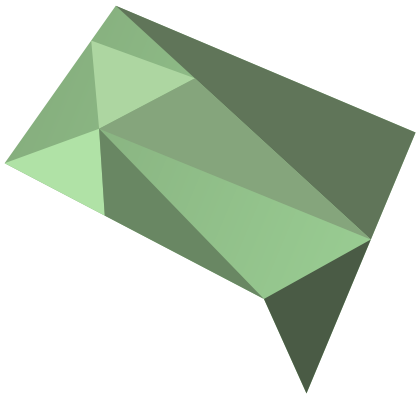
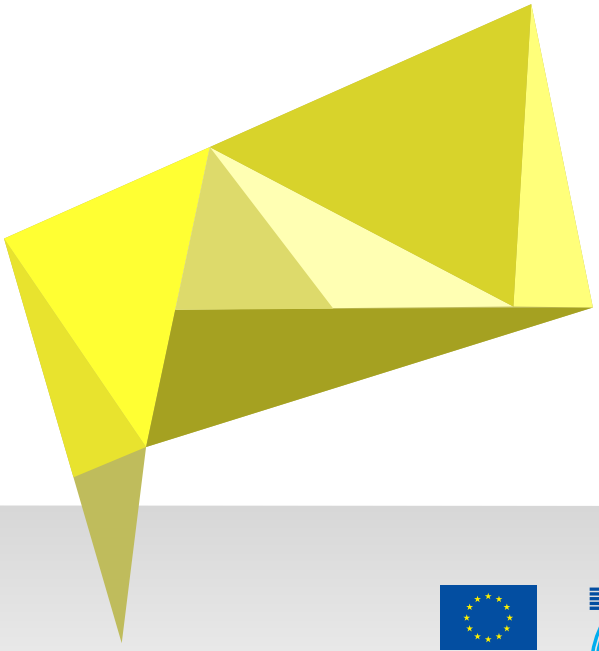


An example of a genetic disorder in which disordered eating behavior occurs is leptin deficiency caused by mutations in the leptin gene. If leptin, which is mainly synthesized in fat cells and secreted into the blood stream, is not produced by a rodent or a human, then this hormone cannot bind to specific brain receptors. This can give rise to overeating and early onset development of obesity i.e. in infancy. This overeating has a specific neurobiological basis. Despite extensive efforts, this particular disorder has been identified in members of very few families worldwide. It is also important to realize that the hunger drive of these affected individuals is not targeted to specific foods. Instead their almost insatiable hunger results in overeating of any food that is available.

The ingestion of food can have a rewarding effect, particularly if we are hungry or develop an appetite for a given food. In neurobiological terms, this rewarding feeling of pleasure results from complex neuronal signaling processes, which are generated upon seeing, smelling, and tasting food. The food texture can also generate pleasure via specific sensors located in the mouth. Finally, ingestion of food entails signaling processes that initiate in the mouth, larynx, esophagus, stomach and small and large intestines. With respect to the rewarding property of foods, there is overlap with neuronal pathways involved in substance use disorders. However, this overlap in itself does not validate the concept of food addiction.

Undoubtedly many people eat more than is healthy. This can (but does not have to) result in obesity and potentially other more or less serious medical conditions such as cardiovascular disorders and type 2 diabetes mellitus. In our opinion, a subgroup of such individuals conceivably may overeat as a consequence of an addiction-like eating behavior. This implies that, as in drug addiction, these subjects crave food, will spend a substantial part of the day thinking of, purchasing, preparing and eating food despite knowledge that this is unhealthy and potentially also despite manifestation of medical consequences of overeating. We hypothesise that a wide range of physiological and psychological issues or problems may underlie the development of an addiction-like eating behavior. Thus, a genetic or acquired propensity to overeat may be activated, if an individual experiences prolonged stress, anxiety, depressed mood and boredom.

Finally, it should be pointed out that food addiction cannot be diagnosed according to any set of criteria which have gained general medical or psychological recognition. Based on current knowledge, the term food addiction appears inappropriate. We suggest that the term addiction-like eating behavior or addictive eating is better suited to describe a phenomenon encompassing symptoms which overlap with the criteria used to define substance use disorders. It may be worthwhile to consider addiction-like eating behavior as an explanation for overeating in a subgroup of individuals with obesity. However, this subgroup by no means accounts for a large proportion of individuals with obesity. Future research is required in humans and animal models to improve our knowledge of mechanisms involved in the development of an addiction-like eating behavior and to assess its relevance for obesity.



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