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Does sugar addiction really cause obesity?

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A commentary on

Carbohydrate-biased control of energy metabolism: the darker side of the selfish brain

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Obesity has become the major health problem in many industrialized countries. But why do so many people – who are facing an abundant food offer – stay slim? All organs in the human organism like the heart, liver, kidney lose 40% of their weight during inanition, except the brain, which loses 1% or less (Krieger, 1921). According to the Selfish Brain theory, the brain uses its stress system, i.e., the sympathetic nervous system (SNS) and the hypothalamus–pituitary–adrenal (HPA) system, to actively demand energy from the body (Peters et al., 2011b). In this way, the brain can satisfy its high energy needs, while the rest of the body is only sparsely supplied. The function of the stress system to actively procure the brain with energy is called "brain-pull" function. It has been shown analytically that in the cerebral supply chain a competent brain-pull function protects against body mass gain, even if there is an abundant food offer available (Peters and Langemann, 2009). And why do other people become obese? If the brain-pull function is incompetent, then energy accumulates in the cerebral supply chain: accumulation of energy in the body stores leads to obesity, accumulation of energy (glucose) within the blood vessels culminates in type 2 diabetes (Peters and Langemann, 2009). Thus, the Selfish Brain theory states that people with incompetent brain-pull have to eat more in order to cover the energetic need of their brain, although their body stores are already overfull.

Tanya Zilberter refers to the Selfish Brain theory in her article entitled "carbohydrate-biased control of energy metabolism" (Zilberter, 2011). At the same time, she refers to an apparently related idea proposed by the psychiatrist DuPont (1997), who has used the term "selfish brain" in the context of addiction. Zilberter discusses in her opinion paper the role of carbohydrate addiction as a potential cause of obesity and calls this aspect "darker side of the selfish brain." She considers addiction as being "highly non-homeostatic" and concludes that "energy intake *beyond* rigid homeostatic regulation relies on behavior with hedonic rewarding and addictive nuances more characteristic for carbohydrates than for fat."

Here I would first like to pose the question whether carbohydrate addiction really affects the organism in a non-homeostatic way. Second, I would like to question whether carbohydrate addiction does result in obesity at all. Carbohydrate (sugar) addiction, including tolerance and withdrawal, has been demonstrated in rodents but not in humans (Garber and Lustig, 2011). Bartley G. Hoebel and his team have carried out ground-breaking animal experiments on this theme (Avena et al., 2008). The researchers have induced sugar addiction in rats by exposing them to a 20-days-experimental paradigm, the so-called "daily intermittent sugar and chow" regimen. In fact, the animals fed in this way enhanced their sugar intake. However, these rats regulated their caloric intake by decreasing their chow intake to compensate for the extra calories obtained from sugar, which results in a normal body weight (Colantuoni et al., 2002; Avena and Hoebel, 2003). These experiments clearly demonstrate that homeostatic control is maintained in the animals, which displayed signs of sugar addiction. Thus, there is no experimental evidence that sugar addiction affects metabolism in a non-homeostatic way, nor that sugar addiction is a cause of obesity.

How did "sugar addiction" develop in the experiments, which used the "daily intermittent sugar and chow" paradigm? Animals were food-deprived for 12 h, and food was offered only 4 h after onset of dark, which is the usual time of their first meal (Colantuoni et al., 2002; Avena and Hoebel, 2003). In principle, food deprivation constitutes a stressor, which threatens brain and body energy supply. The stressful effects of caloric restriction become evident to its full extent when the restriction lasts longer; then the brain has to strongly activate the SNS and the HPA-system in order to safeguard brain energy content and mass. In fact, long term caloric restriction in rats leads to a dose-dependent increase in serum corticosterone (Levay et al., 2010), and the animal's brain mass is conserved, while its body mass decreases (Greenberg and Boozer, 2000). Such brain-mass-preserving effects have also been observed in humans who were on weight reduction diet (Peters et al., 2011a). If now, in the "daily intermittent sugar and chow" paradigm, energy is offered to the rats with a 4-h delay, the prevailing cerebral energy crises can be most quickly resolved by the ingestion of sugar. The unexpected sudden resolution of the difficulties in cerebral energy procurement by intake of sugar prompts a striatal dopamine release as a rewarding signal. The characteristic of the rewarding system is that dopamine release is triggered by unpredicted successes (Schultz, 2007). Dopamine release in the nucleus accumbens helps to acquire and consolidate a behavioral strategy (Kelley, 2004), which safeguards brain energy homeostasis and allows to shut off the stress response; the strategy includes the choice and immediate intake of sugar. In this way, the "daily intermittent sugar and chow" paradigm favors acquisition and consolidation of feeding strategies, which are very effective in maintaining cerebral energy homeostasis in times of food insecurity.

In conclusion, I don't see any evidence supporting the view that carbohydrate addiction really causes obesity. As mentioned above, the Selfish Brain theory states that the underlying cause of obesity is a brain-pull incompetence. There are

many known causes for such an incompetence (i.e., reduced responsiveness) of the brain-pull system, e.g., the habituation to chronic psychosocial stress (Peters and Langemann, 2009; Peters et al., 2011b). The findings on "sugar addiction" in animals should not be linked to human obesity in an overhasty manner, since such ideas might be taken by others to offend those people who have gained weight. In this respect, scientists and clinicians should be particularly cautious, because humans with high body weight do already suffer from severe weight discrimination (Puhl and Heuer, 2009). These humans are known to exert even more rigid cognitive control over their eating behavior than slim subjects do, and these data contradict the notion that a lack of cognitive control causes weight gain (Timko and Perone, 2005; de Lauzon-Guillain et al., 2006; Snoek et al., 2008; Gallant et al., 2010). But even despite such scientific evidence people with high body weight are still accused of being week-willed and hedonistic – only striving at the satisfaction of their lust. The recent progresses in the field of brain energy metabolism, showing that the people who have gained weight just strive at covering their *cerebral energy needs* (Peters et al., 2011b), can be helpful to relieve them from the burden of weight discrimination.

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