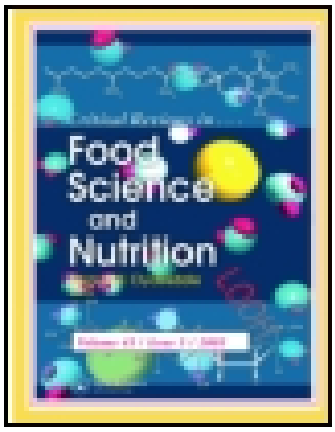

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Sugar and Nutritional Extremism

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Abstract: For many, sugar represents a threat to their health, a perception that is driven by the increase in the prevalence of obesity, diabetes and metabolic disorders which is directly or indirectly connected to consumption of sugar. But is sugar to blame for this health crisis, or are a sedentary lifestyle and an unhealthy diet just as important? Today, sugars and fats are being targeted for restriction or even prohibition. Should we get rid of sugar altogether and or does it

merit a reprieve? Is the effort to ócutlawô sugars a symptom of nutritional extremism that can be as harmful as any other type of extremism?

Most living creatures have evolved to take advantage of favourable conditions to store energy reserves and increase survival in times of shortage, but in the short time period since humankind has become sedentary and industrialized we have not adapted to the virtually unrestricted food availability. Famines and food shortages have been a part of human history and persist to this date. In addition, developed countries now deal with the opposite problem, obesity and excessive caloric intake. Obesity is so widespread that it has become a social and health problem. In his article ñThe toxic truth about sugarò, US endocrinologist Robert Lustig (2012) states that sugar is an addictive and dangerous substance responsible for increasing rates of obesity and diabetes worldwide. These authors even classify sugar as a toxin, along with cocaine and alcohol. This surely must lead some people to think that the world is turned on its head and that soon someone will present findings indicating that it is dangerous to breathe air, or even to live, because life after all leads ultimately to death. How is it possible that the food we have been consuming for centuries and mostly without apparent negative consequences has become all of a sudden a toxic substance that can kill us?

Every cell in our body depends on the intake of carbohydrates as a source of energy, making sugar the primary fuel for our bodies. In addition, humans are mammals, and our young are fed milk from birth. Milk contains lactose, or milk sugar, as one of its crucial components. Indeed, human milk has a high proportion of lactose in comparison to that of other species. It is little wonder, therefore, that we crave sweetness. Sweetness represents energy and satiation, and thus life itself.

It is true that in recent centuries sugar has become more abundant in our diets. Our distant forbearers only knew sugar in fruit and honey. Sugar cane has been known for thousands of years in tropical and subtropical areas. Its stems were favoured for chewing due to their pleasant, sweet taste. Only later ï in India and China ï was sugar exploited and ultimately also exported to Europe. It was, however, rare and expensive. Sugar consumption began to increase markedly in the early 19th century along with the development of sugar cane cultivation and cultivation of sugar beets in Europe. Eventually sugar became commonly available. Unfortunately, over the years consumption increased to levels that are truly unhealthy. Even worse, sugar began to be added to almost all processed foods and arguably is being misused by the food industry. According to some authors the food industry is going to great lengths to suppress information on the adverse health impact of sugar. In light of this controversy, many people have taken an interest in the impact of excessive sugar consumption on our health and have proposed to decrease consumption or regulating the use of sugar.

In their article ñThe toxic truth about sugarò, Dr. Lustig and colleagues (2012) compare sugar to alcohol on the basis of its chemical relatedness (alcohol is derived from the fermentation of sugar) and performance of the same criteria for regulating- unavoidability, toxicity, potential for abuse and negative impact on society. However, Lustig and colleagues primarily discuss fructose, and not sucrose which is commonly used for sweetening. The reason for this is that the food industry in the US uses in substantial measure high-fructose syrup made from corn, which is cheaper than classical sugar. HFCS is also easier to transport and, due to its liquid form, is easier to process by the food industry. Fructose syrup often contains a high ratio of fructose monosaccharide to glucose, while in the disaccharide sucrose the proportion of these simple

sugars is balanced. This causes fructose syrup to have greater sweetening power and to be metabolized differently in our bodies. HFCS is found in a number of foods and drinks worldwide, including in fruit juices, soft drinks, cereals, bread, yogurt, ketchup and mayonnaise. On average, Americans consume 27.2 kg of this sweetener per year per person.

Increasing consumption of fructose over the past two to three decades in many developed countries correlates with increasing occurrence of obesity. However, research on the health impact of dietary fructose is inconclusive and has come to conflicting conclusions. H. Parker at Princeton University demonstrated that not all sweeteners contribute equally to body weight: rats fed HFCS reached markedly higher weights than did those fed sucrose, even though energy intake was identical. Long-term consumption of HFCS led to an abnormal gain of body fat, especially in the abdominal area, and an increase of circulating blood triglycerides in laboratory animals (Parker, 2010). These metabolic changes were accompanied by an important increase in body weight. Dr. Parker maintains that these findings may help explain the growing prevalence of obesity in the United States. These changes could be related to differences between HFCS and sucrose. Due to the production process, fructose molecules in HFCS are free, which means they are readily absorbed. By contrast, each sucrose molecule from cane or beet sugar contains fructose bound to a corresponding glucose molecule. Therefore, fructose must be metabolized before it can be used by the body. The surplus of fructose is then metabolized into fat, while glucose is largely processed as an energy source or saved in the form of glycogen in the liver and muscles. Johnson et al. (2013) state that fructose is distinct from other sugars in its ability to cause intracellular ATP depletion, nucleotide turnover, and the generation of uric acid. They maintain studies that show that fructose-induced uric acid generation causes mitochondrial

oxidative stress that stimulates fat accumulation independent of excessive caloric intake. Similarly, P. J. Havel (2005) has shown that fructose metabolism in the liver, in comparison to glucose metabolism, promotes lipogenesis, which can contribute to hyperlipidaemia and obesity. In addition, fructose does not increase insulin and leptin levels and therefore does not suppress hunger. In fact, fructose even seems to increase ï or at best not inhibit ï the hormone ghrelin which mediates the sensation of hunger. This lack of inhibition may constitute an endocrine mechanism that induces a positive energy balance. However, other authors (Sun and Empie, 2012) state that, in contrast to rats, which can metabolize more than 50% of fructose to fat, this figure is less than 1% for humans. Thus, it seems that the presumed negative impact of fructose on body weight increase and health is overrated. Sun and his colleagues (2011) also analysed data on sugar consumption between 1999 and 2006 from more than 25,000 Americans. They found neither a positive associations between the consumption of fructose and the levels of triglycerides, cholesterol or uric acid in the blood, nor a significant relationship with waist circumference or body mass index (BMI). Sievenpiper and colleagues came to similar conclusions, finding no harmful effects of fructose on body weight, blood pressure or uric acid level (Sievenpiper et al., 2012; Ha et al., 2012; Wang et al., 2012). The highest level of evidence from controlled feeding trials has shown a lack of cardiometabolic harm of fructose and sugar-sweetened beverages under energy-matched conditions at moderate levels of intake (Ha et al., 2013). These observations, therefore, contradict the claims of Lustig and colleagues (2012) that fructose consumption is associated with metabolic diseases such as hypertension (due to elevated uric acid level), increased level of triglycerides, insulin resistance and diabetes.

Squarely opposing Lustig's point of view is James M. Rippe. Rippe claims that there is no direct relationship between HFCS and obesity. He also states that the prevailing scientific view agrees that there is no significant difference between the effect of HFCS and sucrose on metabolism and health (Rippe and Angelopoulos, 2013). Rippe further maintains that metabolism of HFCS and sucrose and its influence on human health differ from the results emerging from studies, as these compare pure fructose or glucose, whereas these are present in negligible amounts in human food diet. Finally, recent clinical studies have shown that there is no detrimental effect of fructose consumption on total cholesterol, LDL and HDL cholesterol, even though certain scientists have demonstrated an increase in cholesterol and/or LDL cholesterol in subjects consuming either sucrose or HFCS. Rippeôs research trial involving 352 overweight or obese subjects who consumed up to the 90th percentile population consumption level of fructose did not show increases in either systolic and diastolic blood pressure, waist circumference, liver fat or ectopic deposition of muscle fat. Author admits that consuming diets rich in carbohydrates (especially simple sugars) increases the level of triglycerides. He concludes that the question of whether increased fructose consumption leads to a rise in uric acid level or blood pressure remains controversial. It will probably be necessary to perform further studies to clarify these issues. Manuel-y-Keenoy and Perez-Gallardo (2012) claim that experimental data at the cellular and tissue/organ level demonstrate that surplus calories in the form of various types of carbohydrates cause pathological changes leading to insulin resistance, body fat deposition, dyslipidaemia and vascular dysfunction, as well as altered appetite control. However, the evidence from dietary intervention studies is still insufficient to establish a causal link, partly due

to serious methodological limitations such as the definition and classification of carbohydrates and the lack of accurate biomarkers of the metabolic outcomes.

Animal models, especially rodents, are not suitable models for metabolic changes due to their metabolic, physiological and behavioural differences from humans. There is perhaps one exception ï those are pigs. Clouard et al. (2012) imply that plenty of studies suggest that the pig can be a good and useful model in the field of nutrition and nutrition impact on health because pigs have many similarities with humans, including behavioural resemblance. Pigs are monogastric omnivores, such as humans, with proportionally similar organ sizes and very comparable gastrointestinal tract anatomy, morphology and physiology. With regard to hormonal regulation of feeding behaviour, pigs and humans share some taste receptors and hormones that are involved in appetite/satiety regulation. The extensive physiological similarities between the pig model and humans emphasize the research perspectives using a pig model to investigate the behavioural and neurophysiological mechanisms, in relation to human nutrition issues (Clouard, Meunier-Salaün and Val-Laillet, 2012). As observed in humans, in pigs diabetes and metabolic syndrome are related to obesity, insulin resistance and high triglycerides blood levels. We have used mini pigs at the Institute of Animal Science at Kostelec nad Orlici, Czech Republic, to assess their sensitivity to a diet promoting diabetes mellitus type 2 and metabolic syndrome (Klein et al., 2012). Mini pigs were fed a standard feed mixture supplemented with suet, sugar and salt either in regular (Group 1) or irregular (Group 2) intervals. Group 2 diet was intended to simulate the influence of an unhealthy food regimen. Each 3 months we performed blood analyses and measured glycaemia, glycated haemoglobin, triglycerides, total cholesterol, HDL and LDL. Backfat measurement and glucose tolerance was tested monthly. The diet rich in fat,

sugar and salt led to a significant increase in weight and even led to a state of morbid obesity in comparison to the control group, but it did not affect physiological parameters characterizing diabetes and metabolic syndrome. As the sensitivity of various pig genotypes to these diseases can differ (Chen et al., 2009), we can conclude from these results that Kostelec mini pigs are apparently resistant to these diseases, so they can enjoy life without worrying about their sugar intake.

According to Lustig, a further argument justifying the restricted consumption of sugar, is its potential for misuse. A number of scientists point to the addictive property of sugar, which has been demonstrated independently and was compared to addiction to hard drugs (Burger and Stice, 2012). Paul van der Velpen, head of Amsterdamôs health service, even claims that sugar is the most dangerous drug of our time ï also because it can easily be acquired (Waterfield, 2013). According to van der Velpen, needless sweetening of various foods can upset the nutritional balance and lead to a sugar addiction. It is allegedly as difficult to rid oneself of the craving for sugar as it is to stop smoking. As stated above, sugar affects dietary balance, by influencing the levels of insulin, leptin and ghrelin. Because these changes promote the sensation of hunger, sweet food makes us eat more than we need. This lack of negative feedback can potentially be misused by the food industry to boost consumption. Psychologists, however, argue that one can be addicted to almost anything. Whatôs your addiction?

Let us put hysteria and shocking claims aside and consider the matter objectively. It is true that high sugar intake is toxic and should be avoided. As members of the species *Homo sapiens sapiens*, we should be sapient and able to choose what we eat, and should teach healthy dietary habits to our children. Someone once said that the best thing we can do for our kids is to

teach them to drink pure water! And because they grow up in this world, we should make it easier for them by, for example, removing candy and sweet drink dispensers from schools. We need not worry that we will become gravely ill or die after sweetening our tea or coffee, so we can safely enjoy this pleasure. However, we should not surpass the tolerable limit, be it in the amount or frequency of sweetening. Unfortunately, sugar can also have chronic toxicity, so even small amounts served over a long time can endanger our health. However, genetic diversity among individuals is also important. What can kill one person might be mere icing on the cake for another. We expect in a foreseeable future, that each individual can adapt the general dietary recommendations according to his genetic profile and can thus benefit from personalized changes in the carbohydrates content of their diet (Manuel-y-Keenoy and Perez-Gallardo, 2012).

Dr. Lustig likes to compare sugar with alcohol; it is true that production and sale of alcohol is regulated. Perhaps some regulation of the food industry would be beneficial. Maybe even limits on the level of sweeteners in processed food should be considered. More helpful would be the promotion of quality food, healthy gastronomy and a balanced approach to life. Most people enjoy a glass of good wine and are not alcoholics. They would certainly not approve a complete alcohol prohibition. Letôs try the same with sugar. History teaches that extreme solutions are more often than a path to calamity, not to well-being.

References:

Burger, K.S. & Stice, E. (2012). Frequent ice cream consumption is associated with reduced striatal response to receipt of an ice creamï based milkshake, *Am. J. Clin. Nutr.*; **95:4,** 810ï 817. doi: 10.3945/ajcn.111.027003

Clouard, C., Meunier-Salaün, M.C. & Val-Laillet, D. (2012). Food preferences and aversions in human health and nutrition: how can pigs help the biomedical research? *Animal*. **6(1)**, 118-136. doi: 10.1017/S1751731111001315; available at: http://www.journals.cambridge.org/abstract_S1751731111001315

Ha, V. *et al.* (2012). Effect of fructose on blood pressure: a systematic review and meta-analysis of controlled feeding trials, *Hypertension*; **59(4):** 787ï 95. doi: 10.1161/HYPERTENSIONAHA.111.182311. Epub 2012.

Ha, V. *et al.* (2013). Fructose-Containing Sugars, Blood Pressure, and Cardiometabolic Risk: A Critical Review. *Curr. Hypertens. Rep.*; **15 (4)**: 281-297. doi: 10.1007/s11906-013-0364-1

Havel, P.J. (2005). Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. *Nutr. Rev.*; **63(5):** 133ï 57. PMID:15971409

Chen, H., Liu, Y.Q., Li, CH., Guo, X.M. & Huang, L.J. (2009). The susceptibility of three strains of Chinese minipigs to diet-induced type 2 diabetes mellitus. *Lab Anim (NY)*; **38(11)**:355-63

Johnson, R.J. et al. (2013) Sugar, Uric Acid, and the Etiology of Diabetes and Obesity. *Diabetes;* **62(10):** 3307-3315. doi: 10.2337/db12-1814

Klein, P. *et al.* (2012). Assessment of the susceptibility of the Kostelec minipigs to diet-induced type 2 diabetes mellitus and metabolic syndrome, in: *Research in Pig Breeding, International Workshop,* p. 20 (Kostelec nad Orlicí). ISBN 978-80-7403-101-4

Lustig, R.H., Schmidt, L.A. & Brindis, C.D. (2012). The toxic truth about sugar. *Nature*; **482:** 27ï 29.

Manuel-y-Keenoy, B. & Perez-Gallardo, L. (2012). Metabolic Impact of the Amount and Type of Dietary Carbohydrates on the Risk of Obesity and Diabetes, *Open Nutr. J.;* **6**: 21-34.

Parker, H. (2010). *A sweet problem: Princeton researchers find that high-fructose corn syrup prompts considerably more weight gain*, (Princeton University); available at http://www.princeton.edu/main/news/archive/S26/91/22K07/.

Rippe, J.M. & Angelopoulos, T.J. (2013). Sucrose, high-fructose corn syrup, and fructose, their metabolism and potential health effects: What do we really know? *Adv. Nutr.*; **4:** 236ï 245, doi: 10.3945/an.112.002824

Sievenpiper, J.L. *et al.* (2012). Effect of fructose on body weight in controlled feeding trials: a systematic review and meta-analysis**,** *Ann. Intern. Med.*; **156(4):** 291ï 304. doi:10.7326/0003-4819-156-4-201202210

Sun, S.Z. & Empie, M.W. (2012). Fructose metabolism in humans ï what isotopic tracer studies tell us. *Nutr. Metab.*; **9:** 89, doi:10.1186/1743-7075-9-89

Sun, S.Z., Anderson, G.H., Flickinger, B.D., Williamson-Hughes, P.S. & Empie, M.W. (2011). Fructose and non-fructose sugar intakes in the US population and their associations with indicators of metabolic syndrome. *Food Chem. Toxicol.*; **49(11):** 2875ï 82, doi: 10.1016/j.fct.2011.07.068

Wang, D.D. *et al.* (2012). The effects of fructose intake on serum uric acid vary among controlled dietary trials, *J. Nutr.*; **142(5):** 916ï 23, doi: 10.3945/jn.111.151951. Epub 2012

Waterfield, B. (2013). *Sugar is 'addictive and the most dangerous drug of the times'* (The Telegraph), available at http://www.telegraph.co.uk/news/worldnews/europe/netherlands/10314705/Sugar-is-addictive-and-the-most-dangerous-drug-of-the-times.html

