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Beware of consumption of soft drinks containing fructose? : An Overview

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Abstract

The intake of sugar-sweetened soft drinks beverages has increased steadily over the past century and with this increase level has been reported to be associating with an increased risk of overweight, type 2 diabetes and cardiometabolic disease**.** Soft drink consumption and cardiometabolic risk, there was a 24% overall increased risk comparing the top and bottom quantiles of consumption. Several factors might account for this increased risk, including increased carbohydrate load and increased amounts of dietary fructose. Fructose acutely increases thermogenesis, triglycerides and lipogenesis as well as blood pressure, but has a smaller effect on leptin and insulin release than comparable amounts of glucose. In controlled feeding studies, changes in overweight, fat storage and triglycerides are observed as well as an increase in inflammatory markers.

Keywords: High fructose, obesity beverages, health risk, sucrose, weight gain.

Introduction

Sugar-sweetened drinks have been associated with several health problems. In the point narrative as presented below, we provide our opinion and review of the data to date that we need to reconsider consumption of dietary sugar based on the growing concern of obesity and type 2 diabetes (Bray., 2010). Obesity is on the rise and is reported by the World Health Organization (WHO) to affect 35 million children in developed countries. According to the WHO, overweight and obesity are more likely to be obese into adulthood and more likely to develop diabetes and cardiovascular diseases at a younger age. There is concern that high-fructose Sugar-sweetened drinks increases the risk of obesity in children compared with other caloric sweeteners. Global figures estimate that about 1 billion people are overweight and about 500 million are obese, with associated increases in the incidence of chronic disease and disability (WHO., 2013). Along with the increased consumption of soft drinks, there has been a rapid and large increase in the reported incidence of type 2 diabetes. Some of this increase is due to an ageing population, but the incidence of type 2 diabetes is also increasing in younger age groups. The intake of sugar-sweetened soft drinks containing either sucrose or high fructose may be associated with this increase in the incidence of type 2 diabetes either due to the influence of these soft drinks on the glycaemic or insulinaemic response or due to their contribution to an excessive energy intake, increasing the risk of weight gain, which is a key risk factor for the development of type 2 diabetes. (Malik et al.,

2010).This review paper aims to describe how fructose, compared with other sugars, is managed by the body, thus clarifying the impact of fructose on atherosclerosis, type 2 diabetes, and obesity.

Soft drink consumption of Fructose

Although acute fructose consumption could not stimulate leptin secretion, an increase in fasting leptin levels was detected after chronic high fructose intake there is some uncertainty about the mechanisms of fructose absorption, most of fructose seems to be absorbed by facilitated transport in the jejunum by the fructose transporter GLUT5 (Tappy et al., 2010) & (Jones et al., 2011). The body has limited ability to absorb pure fructose, and intake of fructose can therefore lead to malabsorption (Beyer et al., 2005). Malabsorption of fructose results in bacterial fermentation, which leads to formation of short-chain fatty acids (acetate, propionate, and butyrate) and gases (hydrogen, methane, and carbon dioxide) (Pimentel et al., 2006). These processes can affect the motility of the intestine and cause various symptoms such as abdominal pain, bloating, and altered stool (Gibson et al., 2007). A significant increase in fructose absorption has been shown when fructose is coingested with equal amounts of glucose.

Soft drink and Metabolic disorder

After Fructose absorption, fructose is transported by the portal vein to the liver, where it is effectively absorbed by liver cells (Litherland et al., 2004), resulting in only small amounts entering the systemic circulation. Metabolism of fructose thus occurs primarily in the liver, but fructose may also be metabolized by enterocytes. Although the artificially high blood fructose level, that study still shows that the kidneys have a relatively increased capacity to metabolize fructose (Paari et al., 2014). It has been shown that GLUT5 is expressed in the membrane of fat, kidney, muscle, and brain cells (Havel et al., 2005) but, due to very low levels of fructose in the blood, negligible amounts of fructose are probably metabolized in these tissues (Stanhope et al., 2012). As discussed above, the liver will metabolize a large majority of the ingested fructose Most of the reactions in liver fructolysis are the same as those occurring in glycolysis, but fructose enters at a later stage in the glycolytic reaction chain than glucose (Parks et al., 2008). In this way, the liver will metabolize fructose in an unlimited way, as opposed to the case of glucose.

This will influence the type and amount of metabolic products produced by the liver and is the main reason why fructose and glucose have different metabolic effects. In the liver, fructose can enter metabolic pathways: it can be oxidized, converted to glucose (and glycogen), or converted to lactic acid, or enter *de novo* lipogenesis (DNL).As a result of the metabolic difference between glucose and fructose, a higher percentage of fructose compared to glucose can be converted to fat in the liver via DNL (Crescenzo et al., 2013). This has been shown in a number of animal and human studies, in which these sugars have been consumed in equal quantities under similar experimental conditions.Intake of fructose together with glucose thus seems to affect the metabolic fate of fructose. To some degree, this effect may be due to higher insulin secretion after intake of glucose compared to fructose (Teff et al., 2004). Insulin will, amongst others, decrease glucose production from fructose (Girard et al., 2006), and insulin will also stimulate DNL (Boden et al., 2013). The extent to which fructose enters DNL is central to the health effects of fructose. Excessive intake of fructose, and hence increased DNL, may increase the risk of disease, because it may potentially cause both increased cholesterol levels in the blood and accumulation of fat in the liver (Le et al., 2009). The effect of fructose on lipid accumulation is thus unclear, but the effect of fructose on the blood lipid profile seems to be better documented.

Soft drink and Atherosclerosis

It appears that high fructose intake can create an unfavorable lipid profile in blood via *de novo* lipogenesis. Create an unfavorable lipid profile in blood via DNL (Rizkalla et al., 2010). The main product of DNL is palmitic acid (Aarsland et al., 1998), a fatty acid specifically revealed to increase the risk of atherosclerosis (Teff et al., 2009). Fatty acids formed by DNL will mainly be packed in VLDLs delivered into the bloodstream. This may, in turn, increase the level of low density lipoproteins (LDLs) in the blood. In several studies, fructose has to a greater extent than glucose increased blood levels of triglycerides (Hallfrisch et al., 1998) and LDLs (Aeberli et al., 2011) showed that fructose increased the small dense LDLs, the type of LDLs that may in particular be linked to cardiovascular risk (Diffenderfer et al., 2014). Due to the insignificant levels of fructose in peripheral blood, as described above, only glucose has the potential to be a substrate for DNL in adipose tissue. Considering known

negative health effects of lipoprotein residues, DNL occurring in adipose tissue may be preferable compared with DNL in the liver. This may illustrate a metabolic difference between glucose and fructose when consuming large amounts of sugars.

Soft drink and Type 2 Diabetes

A high intake of sugar-sweetened beverages, with fructose as one of the major types of monosaccharides, has been associated with development of type 2 diabetes. Although this association does not prove causation, it is important to study the role of fructose in the development of type 2 diabetes. Fructose must be converted to glucose in the liver to cause an increase in blood glucose level. As the conversion takes time and only a portion of the fructose will form glucose, fructose increases blood glucose less than similar levels of glucose (Hashemi et al., 2007). The glycemic index for fructose consumption with lack of stimulation of the pancreatic β cells (Rodrigues et al., 2010), gives lower insulin secretion after intake of fructose compared with glucose. However, it is claimed that fructose may also contribute negatively to blood glucose homeostasis by causing insulin resistance in the liver. Inhuman studies, in which fructose has been reported to cause insulin resistance, the daily intake of fructose has been as high as 110 g, approximately 250 g and 138 g .This may indicate that the fructose intake must be high to potentially cause insulin resistance(Sievenpiper et al., 2014). Although it is conceivable that fructose, via lack of stimulation of satiety signals, could contribute to obesity, fructose has several properties that act against obesity. As previously mentioned, the small intestine has a limited capacity to absorb fructose that the body uses more energy after eating fructose rather than glucose, so less energy will be available to be stored as fat. On the basis of these properties, it does not appear that fructose is more fattening than other sugars. This also agrees with experimental studies of the relationship between fructose intake and obesity in animals (Stanhope et al., 2008) and humans (Sievenpiper et al., 2012).

Soft drink and Obesity

It is debatable whether fructose is less satiating than other sugars and thus can contribute to obesity through a high food intake. Although it is conceivable that fructose, via lack of stimulation of satiety signals, could contribute to obesity, fructose has several properties that act against obesity. As previously mentioned, the small intestine has a limited capacity to absorb fructose. This can lead to malabsorption at least if large amounts are consumed and consumption occurs without glucose-providing nutrients. the high relative sweetness allows smaller amounts of fructose than glucose and sucrose to be used to achieve a particular sweetness in most applications. On the basis of these properties, it does not appear that fructose is more fattening than other sugars. This also agrees with experimental studies of the relationship between fructose intake and obesity in animals and humans (Blakely et al., 1981) .The distribution of fructose into metabolic pathways is of key importance to the health effects of fructose. The distribution varies with the amount of fructose consumed, the duration of fructose exposure, the composition of diet/meal, and whether the measurement took place postprandially, after absorption, or under fasting conditions. Individual physiological, enzymatic, and endocrine factors are also important. Diet composition and the amount of fructose eaten and absorbed will be the focus of this discussion. The composition of diet and especially the amount of glucose/starch may have influence on the health effects of fructose. The effects of fructose on triglyceride and cholesterol levels in the blood, fat accumulation in liver, and insulin signaling intake of both pure fructose and fructose together with glucose. It is also important to note that, despite the metabolic difference between glucose and fructose, glucose consumption far exceeds fructose consumption in the human diet (White et al., 2013). This quantitative aspect must be considered when comparing the health effects of glucose and fructose.

Conclusion

In summary, metabolic disorders caused by excessive simple sugar, and specifically fructose, consumption. Although there is a lack of published literature regarding physiological effects of fructose in humans, current literature does not indicate that a normal consumption of fructose (approximately 50–60 g/day) increases the risk of atherosclerosis, type 2 diabetes, or obesity more than consumption of other sugars. However, a high intake of fructose, particularly if combined with a high energy intake in the form of glucose/starch, may have negative health effects via DNL. More studies are clearly needed, particularly studies under more realistic consumption levels of fructose.

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