We are being killed by fructose *by Robert Harrison Black* bob@life401.com

Executive summary:

We are having an epidemic of metabolic syndrome which includes, obesity, fatty liver disease, type two diabodies and heart disease. This increase correlates with the increased intake of total added sugars which include sucrose, high fructose corn syrup, maple syrup and honey. These sugars are easily broken down into the components which are 45% or more fructose and the balance primarily glucose.

Fructose and glucose have different metabolic pathways. Glucose is quickly absorbed by the intestine and passed through the liver into the blood stream. This signals the pancreas to produce insulin. The insulin escorts the glucose to where it is used for energy, stored as glycogen or converted to fat. The human metabolic system is able to handle glucose if the glucose is delivered over an extended time. Glucose spikes from added sugars or easily metabolized foods like rice, and potatoes can overload the system. Glucose causes a satiety signal that would limit eating. High levels of free glucose in a diet can cause an increase in fasting triglycerides.

Fructose is passed from the intestine and held in the liver where it is primarily metabolized into fat. The fat is either deposited in the liver, in the visceral cavity or escapes into the blood stream. The result is visceral fat and the risk of fatty liver disease. Post meal triglycerides area under the curve are higher with fructose than glucose. Fructose consumption causes lower levels of HDL, higher level of fasting LDL, small dense LDL, oxidized LDL and concentrations of remnant-like particle–triglycerides. Fructose consumption also increased fasting plasma glucose and insulin levels and decreased insulin sensitivity. These effects are in subjects consuming fructose but not in those consuming glucose. Unlike glucose that can provide a satiety signal fructose provokes a feeding signal.

The metabolic syndrome does not depend on being obese. The evidence would indicate the risk for heart failure and having metabolic syndrome and not being obese is at least as high as being obese and having metabolic syndrome. Because metabolic syndrome blocks the satiety signal it would seem metabolic syndrome causes obesity not the other way around.

Below are the references that support the summary:

From the Journal of the American College of Cardiology:

Lamers and others found that heart failure (HF) was more correlated with metabolic syndrome (MetS) than obesity. The work is summarized in the chart below.

BMI Group	MetS		Pre-Incidence of HF, n (%)
Normal	No	109	17 (15.6)
Normal	Yes	68	43 (63.2)
Overweight	No	127	18 (14.2)
Overweight	Yes	107	51 (47.7)
Obese	No	43	4 (9.3)
Obese	Yes	96	52 (54.2)

The main finding of the present study is that after adjustment for well-known cardiovascular risk factors, MetS was independently and significantly associated with an increased 6-year incidence of HF in a population without diabetes and baseline macrovascular complications. Obesity status or increased BMI were not independent predictors of 6-year HF-risk in this studied population. Moreover, obese participants without MetS displayed the lowest risk of incident HF compared with normal-weight participants with MetS. Finally, hypertension, central obesity, and inflammation were demonstrated as the strongest possible mediators of the independent association between MetS and 6-year incidence of HF. (Lamers et al., 2013)

From the Journal of the American Medical Association:

Page and others found increases in fructose consumption have paralleled the increasing prevalence of obesity, and high-fructose diets are thought to promote weight gain and insulin resistance. Fructose ingestion produces smaller increases in circulating satiety hormones compared with glucose ingestion, and central administration of fructose provokes feeding in rodents, whereas centrally administered glucose promotes satiety.

There was a significantly greater reduction in hypothalamic CBF Central Blood Flow after glucose vs fructose ingestion (−5.45 vs 2.84 mL/g per minute, respectively; mean difference, 8.3 mL/g per minute [95% CI of mean difference, 1.87-14.70]; P = .01). Glucose ingestion (compared with baseline) increased functional connectivity between the hypothalamus and the thalamus and striatum. Fructose increased connectivity between the hypothalamus and thalamus but not the striatum. Regional CBF within the hypothalamus, thalamus, insula, anterior cingulate, and striatum (appetite and reward regions) was reduced after glucose ingestion compared with baseline (P < .05 significance threshold, family-wise error [FWE] whole-brain corrected). In contrast,

fructose reduced regional CBF in the thalamus, hippocampus, posterior cingulate cortex, fusiform, and visual cortex ($P < .05$ significance threshold, FWE whole-brain corrected). In whole-brain voxel-level analyses, there were no significant differences between direct comparisons of fructose vs glucose sessions following correction for multiple comparisons. Fructose vs glucose ingestion resulted in lower peak levels of serum glucose (mean difference, 41.0 mg/dL [95% CI, 27.7-54.5]; $P < .001$), insulin (mean difference, 49.6 μU/mL [95% CI, 38.2-61.1]; $P < .001$), and glucagon-like polypeptide 1 (mean difference, 2.1 pmol/L [95% CI, 0.9-3.2]; $P = .01$). (Page et al., 2013)

From the Journal of Clinical Investigation:

Stanhope and others found consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. The study confirmed the previous work on animals matched their results with their human subjects.

"Studies in animals have documented that, compared with glucose, dietary fructose induces dyslipidemia and insulin resistance. To assess the relative effects of these dietary sugars during sustained consumption in humans, overweight and obese subjects consumed glucose- or fructose-sweetened beverages providing 25% of energy requirements for 10 weeks. Although both groups exhibited similar weight gain during the intervention, visceral adipose volume was significantly increased only in subjects consuming fructose. Fasting plasma triglyceride concentrations increased by approximately 10% during 10 weeks of glucose consumption but not after fructose consumption. In contrast, hepatic de novo lipogenesis (DNL) and the 23-hour after meal triglyceride AUC were increased specifically during fructose consumption. Similarly, markers of altered lipid metabolism and lipoprotein remodeling, including fasting apoB, LDL, small dense LDL, oxidized LDL, and after meal concentrations of remnant-like particle–triglyceride and –cholesterol significantly increased during fructose but not glucose consumption. In addition, fasting plasma glucose and insulin levels increased and insulin sensitivity decreased in subjects consuming fructose but not in those consuming glucose. These data suggest that dietary fructose specifically increases DNL, promotes dyslipidemia, decreases insulin sensitivity, and increases visceral adiposity in overweight/obese adults."
(Stanhope et al., 2009)

From Hepatology:

Nonalcoholic fatty liver disease (NAFLD) is the most common chronic liver disease in adults and children. A number of genetic and environmental factors are known to predispose individuals to NAFLD. Certain dietary sugars, particularly fructose, are suspected to contribute to the development of NAFLD and its progression. The increasing quantity of fructose in the diet comes from sugar additives (most commonly sucrose and high fructose corn syrup) in beverages and processed foods. Substantial links have been demonstrated between increased fructose consumption and obesity,

dyslipidemia, and insulin resistance. Growing evidence suggests that fructose contributes to the development and severity of NAFLD. In human studies, fructose is associated with increasing hepatic fat, inflammation, and possibly fibrosis. Whether fructose alone can cause NAFLD or if it serves only as a contributor when consumed excessively in the setting of insulin resistance, positive energy balance, and sedentary lifestyle is unknown. Sufficient evidence exists to support clinical recommendations that fructose intake be limited through decreasing foods and drinks high in added (fructose-containing) sugars. (HEPATOLOGY 2013;57:2525–2531) (Vos & Lavine, 2013)

From Acta Physiologica:

Giorgino and others found That Visceral adipose tissue (Belly Fat) is how insulin effectiveness is lost. The reference provides the complete review of the negative effects of visceral fat.

Adipose tissue is now recognized to have a multitude of functions that are of importance in the regulation of energy balance and substrate metabolism. Different hormones, in particular insulin and catecholamines, govern the storage and utilization of energy in the triglyceride depots. In addition, adipocytes produce several different substances with endocrine or paracrine functions, which regulate the overall energetic homeostasis. With excess energy storage, obesity develops, leading to increased risk for type 2 diabetes and cardiovascular disease. The distribution of body fat appears to be even more important than the total amount of fat. Abdominal and, in particular, visceral adiposity is strongly linked to insulin resistance, type 2 diabetes, hypertension and dyslipidaemia, leading to increased risk of cardiovascular disease. The adverse metabolic impact of visceral fat has been attributed to distinct biological properties of adipocytes in this depot compared with other adipose tissue depots. Indeed, regional variations in the metabolic activity of fat cells have been observed. Furthermore, expression studies aiming at defining the unique biological properties of adipose tissues from distinct anatomical sites have identified depot-related differences in the protein content of fat-produced molecules. (Giorgino, Laviola, & Eriksson, 2005)

From the Journal of Heptology:

Ouyang and others found similar detrimental effects of fructose. Fructose increased the severity of nonalcoholic fatty liver disease. Increased fructose consumption was univariately associated with decreased age ($p < 0.0001$), male gender ($p < 0.0001$), hypertriglyceridemia ($p < 0.04$), low HDL cholesterol (< 0.0001), decreased serum glucose ($p < 0.001$), increased calorie intake ($p < 0.0001$) and hyperuricemia ($p < 0.0001$). After controlling for age, gender, BMI, and total calorie intake, daily fructose consumption was associated with lower steatosis grade and higher fibrosis stage ($p < 0.05$ for each). In older adults (age > 48 years), daily fructose consumption was associated with increased hepatic inflammation ($p < 0.05$) and hepatocyte ballooning ($p = 0.05$). (Ouyang et al., 2008)

From touch Endocrinology:

VAT (Visceral Adipose Tissue) plays a potential role in the pathogenesis of insulin resistance. Abdominal obesity is characterized by elevated Triglycerides, reduced high-density lipoprotein (HDL) cholesterol and an excess of small dense low-density lipoprotein (LDL) particles. It seems as though visceral adiposity promotes the onset of cardiovascular risk factors, such as high glucose levels, high blood pressure. This increases cardiovascular disease risk. (Verrijken, Francque, & Gaal, 2010)

From the International Journal of Epidemiology:

Luke and Cooper found that while exercise is important for physical and mental health, exercise alone is not a practical way to lose weight. Only calorie restriction can prevent and reverse obesity. (Luke & Cooper, 2013)

From a Study by the National Institute of Health

In a study funded by the National Institute of health with 439 women at risk of diabetes where ≥45 minutes of moderate-to-vigorous intensity exercise, 5 days/week alone was compared to reduced caloric intake alone and a program of both exercise and restricted calorie intake was compared to a control where no intervention was attempted. The results were a 2.4 % weight reduction for exercise alone, 8.5 % weight reduction for diet alone and 10.8% reduction to the combination. The exercise alone reduced their fat intake by 3.3%. It appears that exercise made it easier to eat less. The exercise alone and the diet plus exercise participants were healthier. (Foster-Schubert et al., 2012)

As a guide to fructose avoidance;

Sucrose (Table sugar) is approximately 45% Fructose and 55% Glucose.
High fructose corn syrup is approximately 45% Fructose and 55% Glucose.
The sugar in fruit Juice is approximately 45% Fructose and 55% Glucose
The sugar in soft drinks is approximately 45% Fructose and 55% Glucose
Honey is approximately 56% fructose to 44% glucose,
Agave syrup, sorghum and maple syrup each have over 50% fructose.
Malt extract is approximately 95% Glucose and Poly Glucose and 5% Fructose
Food companies are tricky, they will call sucrose: Cane Sugar, Unrefined sugar, Florida crystals etc. Do not be fooled.

The sugar, as starch, in rice, wheat, oats, corn and white potatoes is mostly glucose. Since glucose in these foods is in the form of starch, it is broken down and absorbed. If we eat slowly it is better managed by our insulin system. Even better are foods with more complex starches that are absorbed more slowly. These include nuts, beans, and whole grain cereals like oats and wheat.

I checked our cupboard and shelves of the local grocery store and found it is hard to avoid added sugar. All of the prepared meals in the frozen section I looked at had sugar, even the ones for those on a diet. Many did not have the ingredients listed so the amount of added sugar could not be determined. We are easily fooled into thinking that with a title like granola it would be healthy. In trying to avoid fructose note that peanut butter without added sugar and pasta without added sugar each have about 3% sugar. This sugar is not fructose. As a general rule fruits that spread their seeds by being eaten with the seed surviving the digestive system and being deposited are high in fructose. In the table below I took the grams of sugar and divided by the serving size. For example: Post Great Grains lists their serving size as 55 grams and the sugar is listed as 13 grams. 13÷55= .236, .236X100=23.6% Here is what I found:

Product	Percent added sugar
Great Value Old fashioned oats	2.5
Quick Oats	0.0
Grape Nuts	8.6
Post Shredded Wheat	0.0
Quaker Granola	25.4
Post Great Grains	23.6
Kellogg Raisin Bran	23.6*
Wheat Chex	10.6
Cheerios	3.6
Jif extra crunchy peanut butter	9.3**
Kraft Mac and Cheese	10.0

* Some of the sugar is from the raisins and is not added sugar.
** Some comes from the peanuts but most is added.

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