Is there a link between High Fructose Corn Syrup and Obesity?

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Abstract

Even though there is some evidence in the literature supporting the idea that greater high fructose corn syrup (HFCS) consumption can be specifically linked to the onset of the obesity epidemic in the United States , the general scientific consensus is that HFCS and sucrose (table sugar) have identical impacts on overall health. Unfortunately, this consensus is likely mistaken as the current research makes critical errors and omissions that cause it to vastly understate the power of the link between HFCS and obesity. Evidence from timing data in the United States, international cross-country comparisons, and randomized controlled trials in both rats and humans are presented to make a compelling case that HFCS has a much stronger impact on obesity and health than sucrose.

Introduction

Obesity rates around the world have increased dramatically over the last several decades, and since obesity puts you at risk for a variety of debilitating and deadly conditions like diabetes and heart disease, this has created a worldwide public health crisis of an astonishing magnitude. One possible contributor to this epidemic, sugar sweetened beverages, has been identified as a possible cause, and a great deal of research has gone into trying to understand its effect on obesity. The literature is so great that citing it here in full would be too lengthy, though Malik et al (2013) provides a valuable meta-analysis that identifies and summarizes the research in this area. In this literature, however, the term sugar sweetened beverage is used to make the distinction with artificial sweeteners, but unfortunately lumps drinks using sucrose (table sugar) and drinks using high fructose corn syrup (HFCS) into one broad category.

Even if the literature showing links between sugar sweetened beverages and obesity is quite broad, the research identifying differences between sucrose and HFCS is limited to about a dozen studies. The current general scientific consensus is that even if sugar sweetened beverages, broadly defined, contributes to obesity, there is essentially no difference between soda sweetened with sucrose and soda sweetened with HFCS, and both types of soda should be avoided as much as possible. Sometimes researchers will be a bit more precise and say there is currently insufficient evidence to show that HFCS is worse for your health than sucrose, however most media reports usually emphasize there is no important difference. Unfortunately, there are important errors and omissions in the current literature that causes the links between HFCS and obesity and diabetes to be vastly understated. This report

identifies important lines of research that hopefully convinces you there is a critical difference between soda sweetened with sucrose and soda sweetened with HFCS in its effects on obesity and diabetes.

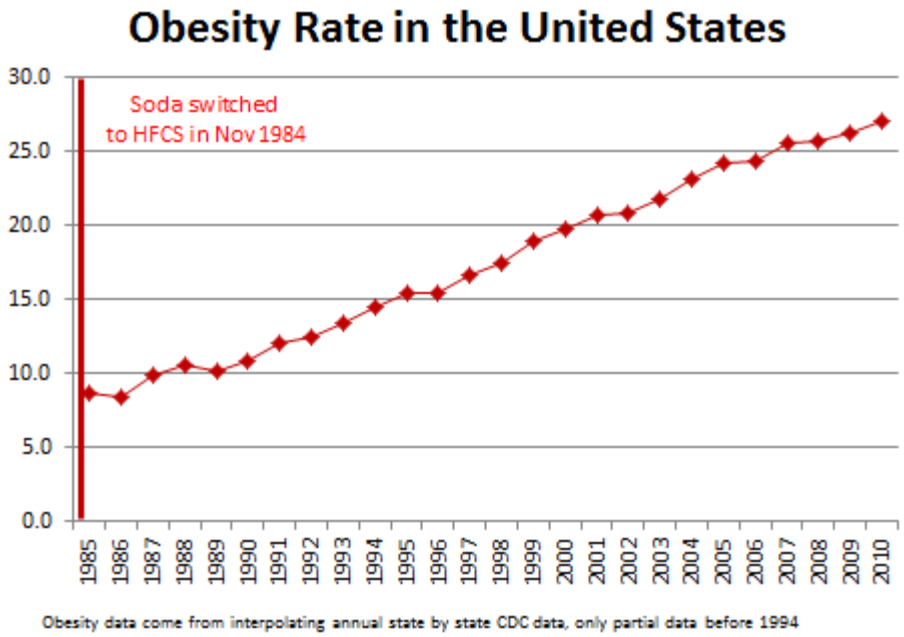
Part I – Evidence showing HFCS has a larger effect on obesity

Section 1 – Examining the timing link between HFCS and increase in obesity rates

The most prominent study highlighting the possible link between HFCS and the obesity epidemic comes from Bray et al (2004), which pointed out that the introduction of HFCS into our diet and the onset of the obesity epidemic occurred at approximately the same time. Using national surveys of obesity in the US, Bray et al found that obesity was relatively low in the late 1970s and significantly higher in the late 1980s and early 1990s. At the same time Bray et al used a sample of diet logs to show that HFCS consumption first started in the late 1970s, and ramped up significantly in the early 1980s. This coincidence in timing made HFCS a serious candidate for causing the onset of the obesity epidemic, but likely vastly understates the power of the link in timing. Once you make a couple of methodological changes when trying to identify this link, the evidence becomes much more compelling.

The first methodological change to make is to use a different dataset to identify when the increase in obesity started taking place. Bray et al used national surveys conducted relatively sparsely with large gaps in between the studies. However, the Centers for Disease Control in the late 1970s started conducting a representative phone survey every year to identify obesity rates in each individual state. The reasons this data was ignored by Bray et al is that early on relatively few states conducted the survey and all states were not included in the data until 1994. Since this was not a full national sample obesity rates for the entire US were not reported and only a state by state map of obesity rates for each year was released. Back in 2003, when I first started looking into this, I interpolated the incomplete state by state data from looking at the color coded map to generate an approximate estimate of a national obesity rate for each year from the late 1970s to the present. The evidence was striking. From the onset of the study to 1984, the obesity rate remained relatively flat bouncing around year to year but without exhibiting an increasing trend overall. Then in 1985 the obesity rate started rising by 1% a year, every year from 1985 to 2003. The state by state obesity data currently released by the CDC only goes back to 1985 but I reconstructed the graph I made in 2003 to present here. The critical insight into obesity that this new presentation of obesity trends provides is that something very important must have happened right around 1985 to dramatically change the trajectory of obesity rates in the US.

[Graph 1 – Obesity rates in the US over time as interpolated from CDC data]



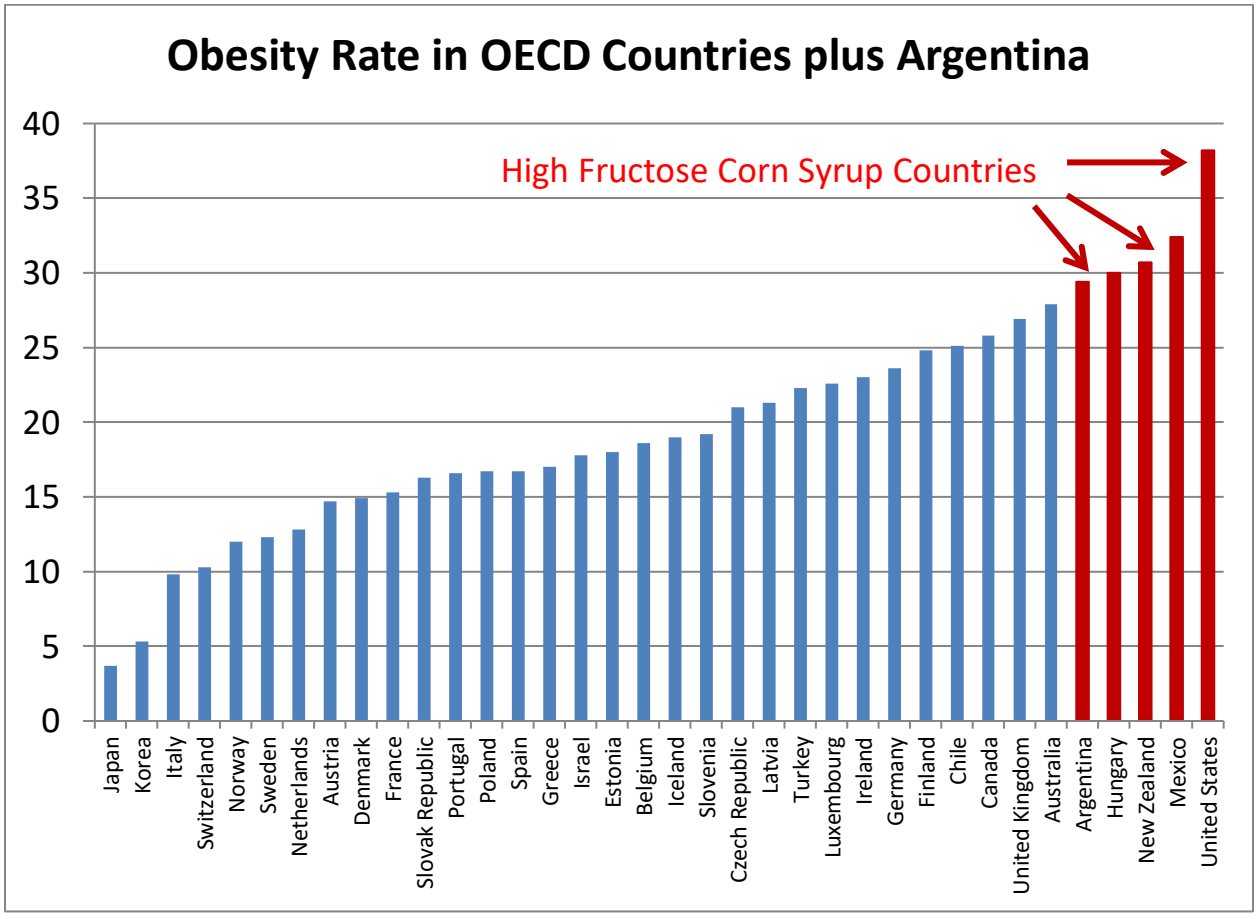
The second methodological change to make when trying to understand potential coincidence in timing between HFCS and obesity is to use the moment when beverage companies switched from using sucrose to HFCS in soda. Instead of using nationally representative diet logs to show a gradual increase in HFCS consumption in the early 1980s, both Coke and Pepsi switched over to using HFCS in their drinks in November of 1984. If the CDC obesity data shows that something dramatic must have happened around 1985 to impact obesity trends long term, then the fact that HFCS was broadly introduced into soda at exactly this time makes the case for a link between HFCS and obesity very strong indeed. The paper by Bray et al got a lot of media attention at the time and had a powerful impact on changing public perceptions of the health risks of consuming HFCS. The fact that it had such an impact and still vastly understated the case once you make a couple of methodological changes, makes this new evidence shown here likely to be even more persuasive. Still, this insight is only one piece of evidence, and once this hypothesis is suggested we need to find new sources of evidence to verify whether there is a good causal link between HFCS and obesity or whether this timing link was just a coincidence.

Section 2 – Examining the international evidence

Luckily for us, there is a large scale long term natural experiment to help us understand whether HFCS consumption contributes to obesity. Some countries in the world have used HFCS in soda for the last several decades, and some countries have used sucrose in soda for the last several decades, so comparing the obesity trends in these two different sets of countries should give us some insight into the effects of HFCS on obesity. Using Google, I was able to identify a set of five very diverse countries that currently use HFCS in soda: the US, Mexico, New Zealand, Hungary, and Argentina. Most strikingly,

four of these countries are in the OECD and of the countries in the OECD these four countries all have the highest obesity rates. If the fifth country, Argentina, were in the OECD, then the five countries that use HFCS in soda would also have the five highest obesity rates in the OECD. If the timing evidence helped us come up with the HFCS hypothesis, then this independent evidence using international data strongly confirms what we originally suspected. The odds that it would happen solely by chance that the five countries that use HFCS also had the five highest obesity rates in the OECD are around 1 in 40,000, so really the evidence could not get much stronger than this.

[Graph 2 – Obesity rates in the OECD]



Part 2 – Arguments against hypothesis that HFCS has a greater effect on obesity

Section 3 – Understanding the chemical similarities and differences between HFCS and sucrose

Even though the article by Bray et al (2004) had a big impact on public opinions of the risks of HFCS, the scientific consensus has always maintained that there is essentially no difference between consuming HFCS or sucrose. There were some responses to the hypothesis suggested in the original study by Bray et al (White 2006, Forshee et al 2007, Tappy et al 2010, Klurfeld, et al 2013), and in these articles there

were contentions made that the link between high fructose corn syrup and obesity lacked sufficient evidence in support of it. Beyond citing a lack of evidence, the first main argument made on their behalf was one of chemical analogy. HFCS and sucrose are chemically quite similar so they argue their impact on people should be quite similar too. Table sugar, or sucrose, is just one molecule of glucose connected to one molecule of fructose, so this leads to an exact 50/50 mix in sucrose. HFCS can take various forms, but the one generally used in soda is HFCS-55, which is 55% fructose, 42% glucose, and 3% saccharides. Many people, both from the corn refiners industry and some independent scientists, point out that this combination is essentially the same, and that even if there is evidence that consuming high levels of fructose by itself has adverse health effects, these studies do not show what happens when glucose and fructose are consumed in approximately equal amounts.

Even though these arguments tend to carry the day among scientists, there are key differences between sucrose and HFCS that could have important effects on public health. The first difference is that in sucrose, the glucose and fructose molecules are bonded together, where in HFCS, the glucose and fructose molecules are freely floating. Since sucrose takes some digestion to free up the glucose and fructose molecules, this could lead to some differences in how the sweeteners are metabolized, but opponents suggest that this bond may be broken early on and have little effect overall. The second key difference between sucrose and HFCS is that HFCS is 55% fructose whereas sucrose is exactly 50% fructose. Since fructose by itself has studies showing adverse effects, the slightly higher composition of fructose in HFCS could have adverse effects as well, especially if consumed in large quantities.

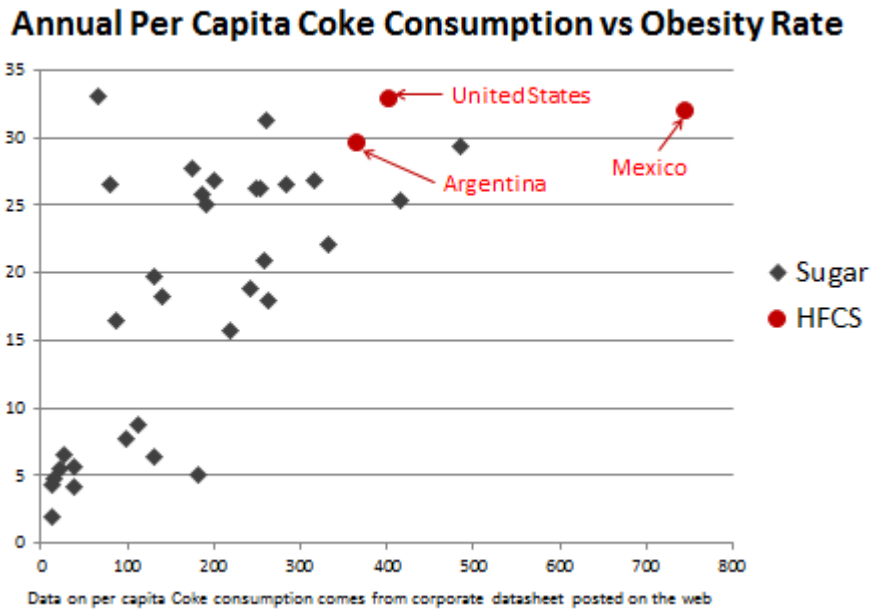
Section 4 – Examining the short term experiments

The second argument against the idea that HFCS might have a greater impact on obesity is that there are a few short term studies that show little difference in how sucrose and HFCS are metabolized. Five studies in particular analyze the very short term metabolic effects when fed HFCS compared to sucrose (Akgun et al 1985, Akhavan et al 2007, Melanson et al 2007, Soenen et al 2007, Stanhope et al 2008). Each study provided a small number of subjects with a fixed amount of sweetener to be consumed, then measured the effects on blood glucose, insulin, satiety, and food intake over the next day or two, depending on the study. These studies, however, are all very short term studies, and I discovered a critical idea that could explain why the short term studies show little difference, but the long term international data does.

The basic idea is that if you consume the ***same amount*** of sucrose and HFCS in soda then the effects are similar, but that when you consume HFCS it causes you to consume ***more soda***. The short term studies miss this effect because they strictly control the amount of sucrose or HFCS you consume, and then analyze the differences, whereas long term studies allow the individual to control how much sucrose or HFCS they consume, allowing greater differences since they can consume as much sucrose or HFCS as they want. One of the short term studies allowed you to vary how much food you ate on the second day of the study after consuming HFCS on the first day, but still gave you a fixed amount of sweetener over which you had no control.

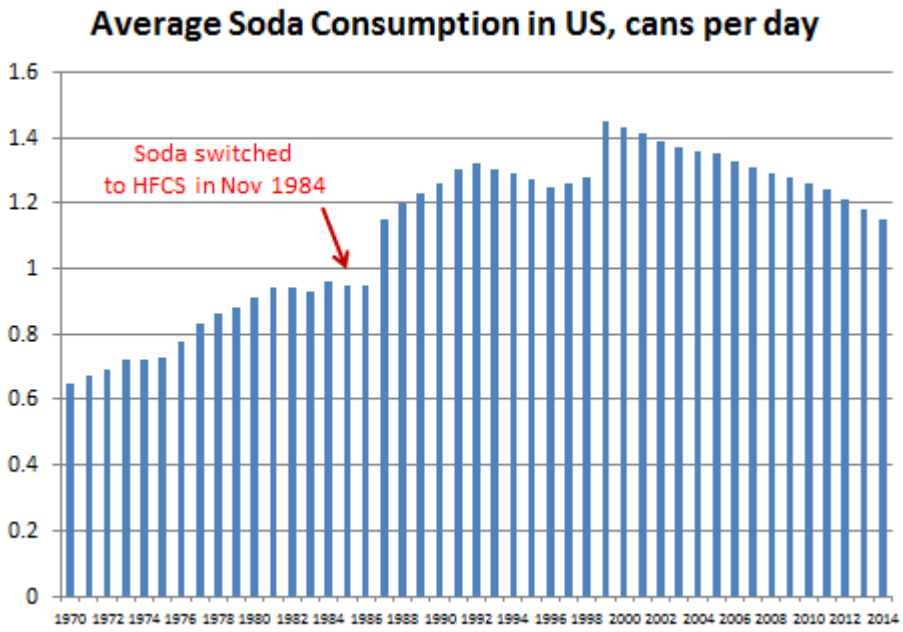
The first piece of evidence supporting this idea came when I came upon corporate data from Coke, showing per capita Coke consumption in a variety of countries around the world. If you simply graph the per capita Coke consumption for each country against their obesity rate, you find a very strong connection between Coke consumption and obesity. Every country that consumes a lot of Coke is also very obese, and consuming one can of Coke a day increases the obesity rate in a country by around 10-15 percentage points. Some countries have high obesity rates despite consuming very little Coke, but these are largely Middle Eastern countries addicted to a type of sweetened Arabic tea which mixes black tea, mint, and a fair amount of sugar. One important result is that HFCS consumption cannot explain the broader worldwide trends toward higher obesity rates since only five countries actually use HFCS in soda. What you also find is that given how much Coke a country consumes, HFCS countries are not more obese than you would expect, but that HFCS countries are generally among the highest per capita consumers of Coke around the world. This indicates that it might be that HFCS encourages you to consume more Coke that causes it to have a greater impact on obesity.

[Graph 3 – Per capita Coke consumption vs obesity worldwide]



The second piece of evidence supporting this idea that HFCS causes you to consume more soda came from a Washington Post article posted on their Wonkblog website that provided a graph showing per-capita soda consumption going back several decades in the US. The striking thing about this graph is that there was a massive one year 20% increase in soda consumption in the mid-1980s, right around the time that soda companies switched to HFCS. The graph is sparsely labeled, and the increase might have occurred in 1986 or 1987 rather than 1985, but this does provide additional evidence that HFCS might cause people to increase soda consumption.

[Graph 4 – Per capita soda consumption from 1970-2014]



If this were the case, then this could explain the differences between short term studies, which control how much sucrose or HFCS you consume, and long term studies, where individuals are free to consume however much sucrose or HFCS they want. Given that there is strong observational evidence in support of the HFCS hypothesis, and there are problems with all the arguments against it, what we need is some more powerful evidence from randomized controlled trials to determine whether the HFCS hypothesis is correct.

Part 3 – Evidence from Randomized Controlled Trials

Section 5 – Princeton rat study

The best evidence you can get to establish the validity of a hypothesis is a randomized controlled trial. Fortunately for our case, there have been three randomized controlled trials, one in rats and two in humans that have tested the hypothesis that HFCS causes obesity more than sucrose. In Bocarsly et al (2010), a team of Princeton researchers gave some rats water sweetened with sucrose and some rats water sweetened with HFCS, and then examined whether one set of rats became more obese than the other. In their study, rats that were given water sweetened with HFCS were significantly more obese than the rats given water sweetened with sucrose. The critical feature of this study, unlike the other short term experiments we examined in the last section, is that the rats were allowed to freely consume

as much sweetened water as they wanted. If the reason why HFCS has a greater effect on obesity is that HFCS causes you to consume more sweetener, then you want your study to allow individuals to choose how much sweetened beverage they want to consume. This is exactly what the Princeton rat study did, and it ended up producing evidence that HFCS has a greater impact on obesity than sucrose when consumed as a sweetened beverage, unlike the short term studies that failed to do this.

Section 6 – Randomized controlled trials in humans

One of the two randomized controlled trials done on humans was conducted by Yu et al (2013) which examined the impact of HFCS on obesity compared to sucrose. In this study, one set of participants was given milk sweetened with sucrose, and another set of participants were given milk sweetened with HFCS for 10 weeks and then weighed at the beginning and end of the study to identify the impact on obesity. The participants were divided into three groups, where the low consumption group was given enough sweetener to represent the 25th percentile of sugar or HFCS consumption found in everyday society, the medium consumption group was given enough sweetener to represent the median amount of sugar or HFCS consumption, and the high consumption group was given enough sweetener to represent the 90th percentile of sugar or HFCS consumption. So for each group, the low, medium, and high consumption groups, half were given a certain amount of milk sweetened with sucrose, and half were given a certain amount of milk sweetened with HFCS, each one corresponding to the amount they were supposed to consume for each group. Each participant was instructed to drink the sweetened milk provided, but then was free to consume whatever else they wanted for the rest of the day. This is a key feature of the study, since it allows the HFCS participants to consume more sweeteners throughout the day, which could be the mechanism through which HFCS has a greater impact on obesity.

What is interesting in this case is that publically the researchers reported there was no difference between the sucrose and HFCS groups, but horribly misinterpreted the results of their study. If you look at the tables, you can see there is no difference in weight between the sucrose and HFCS participants in the low consumption group, but in the medium consumption group the HFCS participants weighed 6-7 pounds more, and in the high consumption group the HFCS participants weighed about 11-12 pounds more. The problem is that there were only 40 participants in each low, medium, or high consumption group, 20 of which consumed sucrose and 20 of which consumed HFCS. So even though the effects on obesity were measurable and important given that the study only lasted 10 weeks, the results themselves were not statistically significant, so the researchers made a critical error implying that the evidence showed no difference between the sucrose and HFCS groups. In fact there was a difference, an important and measureable difference, and this randomized controlled trial confirms the evidence from timing data in the US and the international obesity data from the OECD and directly refutes the arguments suggesting there is no difference between sucrose and HFCS when it comes to obesity.

There was another randomized controlled study (Lowndes et al 2012) examining the effects of sucrose and HFCS on weight loss that confirms my hypothesis that HFCS has a greater effect on obesity by changing how much sweetener you consume. In this study, researchers put around 250 participants on

a reduced calorie diet, some who were given a certain amount of sucrose to be consumed in their diet and some who were given a certain amount of HFCS to be consumed each day. In addition, the HFCS and sucrose groups were divided into low consumption and average consumption groups (representing consumption levels equal to the 25th percentile and 50th percentile) and then compared to an exercise only group that did not get a low calorie diet at all. Over the 12 week study period, the HFCS group lost slightly more weight than the sucrose group (4 pounds compared to 3 pounds in the low consumption group) but the weight loss was lower in the average consumption groups where they consumed more sweetener as you would expect (only 2.5 pounds for the HFCS average consumption group).

The critical difference between the first and second randomized controlled trial is that in the first study they allowed participants to eat whatever they wanted in addition to the daily dose of sweetener (which may have allowed them to consume more sweetener), where in the second study they were on a fixed low calorie meal plan that gave them no ability to change what they ate at all (which restricted their ability to consume more sweetener). As I suggested, these studies that strictly control the amount of sweetener you consume might be misleading, because the way HFCS might cause people to become obese is by encouraging them to consume more sweetener. As you would expect then, the first study revealed a significant difference between HFCS and sucrose once you were allowed to change your eating habits in response, but showed little difference if your eating habits were not allowed to adapt. At this point, the supporting evidence in favor of the idea that HFCS has a greater effect on obesity is overwhelming, even though the published research on the subject presents a mixed view on the subject.

Part 4 – Effect of HFCS on diabetes

Section 7 – Study linking HFCS consumption to diabetes

So far we have only examined the link between HFCS and obesity, but given the strong connection between obesity and diabetes, we should also try and see if we can determine whether HFCS is to blame for the dramatic rise in diabetes as well. One study by Goran et al (2013) looks at this problem directly, using data on HFCS production in a variety of countries around the world and comparing it to reported diabetes rates in each of these countries. They use a big cross-country multivariate regression and find that high levels of HFCS production in a country is associated with a diabetes rate 20% higher than countries with little or no HFCS production in the country. Now I tend to be skeptical of large cross-country multivariate regressions because it is difficult to tell what is really going on in the data, and in this case the researchers make an important mistake that probably causes them to understate the connection between HFCS and diabetes.

The key mistake is that in their regression they included the obesity rate as a control. The argument for this inclusion is that obesity is generally considered a cause of diabetes, so if you want to see whether HFCS is the cause, you need to separate the effect that obesity has on diabetes. If the cause of high obesity rates was something completely different, then this would be the correct way to go, however if HFCS is the cause of obesity ***and*** diabetes then this is a big mistake because a big part of the effect is

going to be attributed to obesity rather than HFCS if you include obesity as a control. If obesity is also caused by HFCS, then the part attributed to obesity is also caused by HFCS, so the best thing to do is to not include obesity as a control since it would lead to a dramatic understatement of the impact of HFCS on diabetes. At the very least, you should run the regression with and without the obesity control to see how big of a difference this makes.

In the same study, there was another underappreciated source of evidence indicating a link between HFCS and diabetes. The researchers produced a graph of the overall diabetes rate over time in the US, and as in the obesity data, this graph showed diabetes rates abruptly increasing staring right around 1985. Just as the obesity data indicated something important and dramatic happened in 1985, the diabetes data shows exactly the same thing. If HFCS was likely the cause of the increase in obesity in 1985, and we know the strong connection between obesity and diabetes, then HFCS is likely the cause of the increase in diabetes rates that happened at approximately the same time. As a result, getting rid of HFCS in our food supply could potentially lead to dramatic public health benefits not only through reducing obesity rates, but also by reducing the incidence of diabetes as well.

Part 5 – Policy Implications

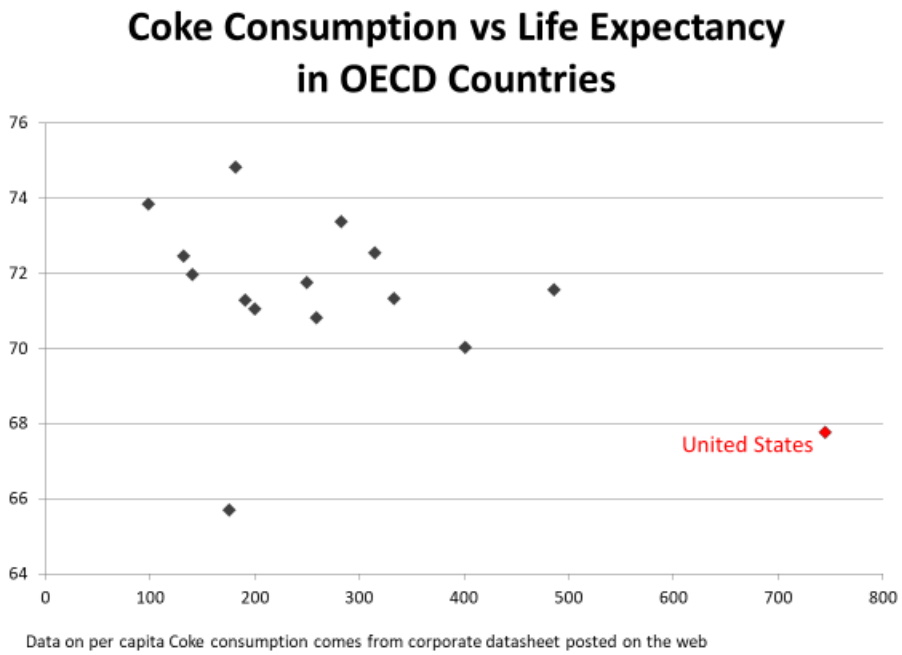
Section 8 – Should we ban HFCS or impose a soda tax?

The most obvious solution to the negative impact of HFCS in soda is to simply ban the use of HFCS in all food and beverage products. The model for this would be the ban on hydrogenated oils enacted by the Obama administration in 2015 (which has not yet taken effect), which was done without the need for legislation simply by having the FDA no longer classify hydrogenated oils as "Generally Regarded as Safe". This required the food companies to come up with scientific evidence showing hydrogenated oils were safe to use, which the FDA knew would be impossible because the bulk of the scientific studies showed hydrogenated oils to be harmful. Banning HFCS could follow a similar regulatory course, though this would not take place until the academic literature changed expert opinion on the difference between sucrose and HFCS, which is probably necessary to convince the regulators to make the change.

The most prominent and debated public policy intervention designed to reduce obesity is the soda tax, even though the evidence presented here, when analyzed carefully, raises serious questions about the efficacy of such a solution. When looking at the problem of obesity, the epidemic began in the United States specifically after HFCS was introduced into soda, at which point obesity started rising about 1 percentage point a year for several decades (see graph 1). The rest of the world, however, also experienced elevated obesity rates without any HFCS in soda (see graphs 2 and 3), which implies that the problem was also the quantity of soda consumed even if it was sweetened with sucrose. If HFCS were the likely cause of the increase in obesity, and this trend would best be brought to an end by banning HFCS. If the rise in soda consumption is the driving cause of obesity, then imposing a soda tax makes more sense since obesity rates might possibly be driven down long term by reducing the consumption of soda.

The problem with this argument is that the levels of soda consumption has been falling ever since 1999 in the United States (see graph 4), and yet obesity rates still continue to rise by about 1 percentage point a year. This indicates that lower soda consumption will not end the continuous rise in obesity, even if a new soda tax does reduce consumption by as much as 20 percent, since that is the decline the US has already experienced so far. There still might be long term health benefits to imposing a soda tax (see graph 5 below), where the lower soda consumption might not be effective in getting people who are already obese to lose weight, but might be useful in keeping a new generation of people from gaining weight and becoming obese. If the soda tax is sold to the public based on the promise of dramatic reductions in obesity over the short run, and then the benefits fail to materialize, this might prevent the public from enjoying the long term intergenerational health benefits of a soda tax when the public demands its repeal.

[Graph 5 – Annual Per Capita Coke Consumption vs Life Expectancy for Rich OECD Countries]



There could be benefits from the extra tax revenue raised by the soda tax, however this requires the governments imposing the tax to do a particularly effective job spending the money. The most obvious solution is to spend the new tax revenue on public health programs designed to reduce obesity, however, some of the most attractive options often have little or no evidence to prove they actually work. Since reducing obesity often proves difficult to do, many programs attempting to reduce to obesity (say by encouraging exercise and healthy eating) may also end up being completely ineffective, and much of the money raised from the tax would ultimately not go to good use. A better option would be to spend most of the money on programs already proven to have tremendous long term benefits (like Philadelphia did when they dedicated the money to universal pre-K), and then devote the rest of

the tax revenue to funding a lot of small pilot programs to reduce obesity that can then be carefully studied.

Ideally, a ban on HFCS could be enacted quickly with the hope that this would end the steady increase in obesity rates. Then, after the ban took effect, the goal could be to reduce obesity long term by reducing soda consumption through a soda tax. When enacting the soda tax, however, advocates should carefully manage public expectations by emphasizing the long term intergenerational health benefits rather than contending that a tax will lead to a large decrease in obesity over the short term. In addition, a lot of effort should be put into ensuring the revenue from the soda tax is used effectively. In this case, a soda tax could become attractive to the public and unlikely to repealed in part due to the benefits of the programs funded by the tax as well as the long term health benefits which might take decades to show up.

Conclusion

Hopefully in this paper I showed you dramatic compelling evidence that the link between HFCS and obesity (and diabetes too) is vastly understated in the current research literature. In this report, I show four separate, independent sources of evidence (timing link, international data, rat study, randomized controlled trial in humans) that together make a convincing case that HFCS has a greater adverse effect on obesity than sucrose. Unfortunately, the scientific community and media largely stick to the idea that there is no difference between sucrose and HFCS, and until we manage to change the minds of these critical players, we will not be able to see the public health benefits from banning HFCS from our food supply.

Even though I believe the evidence for the link between HFCS and higher obesity rates is quite convincing, there are some further steps we could take in coming up with more definitive research. The first thing you could do is look at timing data in Hungary, where Hungary had relatively low obesity rates a couple decades ago, but now has the highest obesity rate in Europe. If we could link the date that Hungary started using HFCS in soda to the date obesity started to rise, this could be another powerful source of evidence to confirm our hypothesis.

The second research project we could undertake is to replicate the randomized controlled trial in humans that showed a significant difference between HFCS and sucrose with a larger sample size. In the new study, we could focus on the medium consumption group and instead of testing just between sucrose and HFCS we could have groups test for sucrose, HFCS, artificial sweeteners, and a control group. Most importantly we could increase the sample size dramatically from say 20 per subgroup to 250 per subgroup (for a total of 1000 participants) while also increasing the length of the study from 10 to 15 weeks.

Engaging in this new research could speed up the time it takes to move from public health insights to new preventative legislation. It took around 25 years to ban hydrogenated oils from the time researchers started to provide evidence of negative impacts on public health. Hopefully with HFCS we

can shorten this time, and it should only take some relatively inexpensive randomized controlled trials to provide the definitive evidence. Given the cost of the obesity and diabetes epidemics in the US, spending $10-25 million on a few randomized controlled studies would be minimal. The purpose of this paper is first to correct the mistakes and omissions in the current literature, and this alone could have a tremendous impact on the public health debate of HFCS. If secondarily it encourages new research on HFCS, obesity, and diabetes, then this could represent a significant gain as well, though until we get an actual ban on using HFCS in food, the benefits to society will go unrealized.

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