The Distasteful Detriments of Fructose (Conversational Science)

Senior year of high school I found myself faced with the culminating assignment of my secondary schooling career, an extensive research paper. While other students were choosing standardized, boring, topics they had no interest in, I decided to base my topic in an actual quandary I had been wrestling with personally. Months later I proudly finished my paper on "The Benefits and Detriments of Protein Powder and Artificial Sweeteners." Although this was a high school project, I dug into it with gusto and emerged with satisfied, research-backed opinions that I have ascribed to ever since. However, although I drew the general research conclusion that sugar was deleterious to our health, I lacked clarity in the nuances. Are all sugars bad? How about the sugar in fruits and vegetables? If I skip the fruit tray and choose some Snickers with a multivitamin chaser, is that a comparable substitute? Do different sources of sugar come with different serving sizes that are acceptable? These are just a few of the many questions I have wrestled with in the last few years. Encouraged by a fascinating video on the topic of sugar I was motivated to once again dive in and update my knowledge. Hopefully in reading this, you will be a beneficiary as well! I hope you are ready to jump into the rush of the sugar quandary with me. Just be careful who you discuss this with, sugar conversations with friends and family can get rather sticky.

My inspiration for this Battle Plan was stoked after watching a video from the YouTube channel: What I've Learned (What I've Learned, 2016). As an aside: Although the reliability of YouTube as a platform is questionable at best, there remains high quality, research backed

content present within certain spheres. Upon looking into journal articles on this topic, I feel confident in saying relevant research was correctly integrated into this video, in a manner that is sufficient for me to consider the video as a reliable, digestible source for biochemically centric information. The video exceptionally laid out how three substances are metabolized in the body (alcohol, glucose, fructose), and the subsequent impact of each on our health. For most of this paper I will generally follow the order of the video lesson, primarily providing value by putting the content into new words and taking out some of the complex pathways that the average person (not a nutrition nerd like me) would get bored with. To be clear, this is not original research of mine, and I give production credit to the content creator.

Alcohol consumption is linked to a host of both short- and long-term health concerns, impacting everything from motor control to metabolism. For the purposes of this paper, I will specifically focus on the metabolic and morbidity elements. To understand the challenges and dangers of metabolizing alcohol, we must first understand how something is properly metabolized. Our classic forms of complex carbohydrates (potatoes, breads, pasta, etc.) are eaten and eventually broken down in the digestive system to glucose. Glucose is one of three simple sugars, or monosaccharides (along with fructose and galactose), that represent the basic form of digestible carbohydrates. Glucose is the currency of the metabolic system. Our entire body is wired to accept glucose, in fact our cells use it as a basic form of fuel. That means that when we digest glucose, much of it is used by the cells along the way. It doesn't require remanufacturing in the liver to turn it into a suitable form of energy for the masses. As a result, approximately 80% of the glucose consumed is utilized by the collective cells in the body before it even reaches

the liver (What I've Learned, 2016). This last 20% goes into the liver via a gatelike entrance provided by Insulin Receptor Substrate (IRS-1) and then triggers a pathway through which glucose is transformed into Glucose 6-P. At this point approximately half of this product (obviously dependent on many factors such as glycogen saturation, activity level, etc.) is converted into glycogen to be stored in the liver for future quick energy use. The last 10% of our glucose is turned into pyruvate, goes through the TCA cycle, and produces ATP (raw energy) and citrate an (intermediary compound). Citrate in the liver is generally bad news. It contributes to VLDL fat formation which leaves the liver and results in unfavorable consequences anywhere it goes (augments fat cells, creates atherosclerosis, increases muscle insulin insensitivity). Now keep in mind glucose is not a high impact food for these health detriments. The video creator reminds us that only about 2% of the glucose from the aforementioned foods will make it to this negative pathway. However, such is not the case with our next two metabolic players.

If you are not already aware or maybe need to hear this truth one more time: Alcohol is a toxin. That assertion is not merely the product of an overbearing parental opinion, or hearsay, the body processes it metabolically as a toxin. Alcohol is not instantly usable by our cells when it is ingested, instead the majority of it (80%) goes to our liver. Once in the liver, glycogen is not an option due to the molecular makeup of alcohol. Instead, it is converted into acetaldehyde which produces Reactive Oxygen Species (ROS). Last year I completed an extensive literature review comparing eNOS and ROS. In summary, eNOS is the good guy that fights ROS the bad guy. ROS is responsible for a plethora of negative health conditions (including atherosclerosis,

cancer, and fat accumulation). When health professionals or the general health community recommend ingesting antioxidants, the goal is to counteract ROS and its oxidative nature.

Since glycogen cannot be made, this acetaldehyde goes into the mitochondrial TCA cycle similar to the glucose (just in much high quantities). Once again ATP is produced, but this time because of the overload on the system, much more citrate is created. Significant VLDL is formed and can once again go in several directions. The first, fat storage is uniquely visceral in depository location, due to the proximity of the visceral region to the liver (where the VLDL is made). In essence this is what creates the "beer belly," a relatively high percentage of fat in the stomach area compared to the rest of the body. The second option is for the liver to distribute excess free fatty acids to the muscle to use as fuel. Using this method for fuel is less then ideal and can predispose the muscles to insulin insensitivity. Finally, with the other two options exhausted the liver may not be able to go anywhere with the fat that it has manufactured through alcohol metabolism. The last option is for the fat to sit in the liver, and this is how Fatty Liver Disease occurs. This is a bad sign for the liver, as the liver accumulates fat it becomes less efficient and eventually could be rendered useless (potentially bad news if your largest organ becomes useless). All of these bad players together promote an interesting signaler known as JNK1 which upregulates inflammation. This can cause liver insulin resistance by closing the IRS gate responsible for letting in glucose, amongst other health issues (general rule; inflammation is almost always bad).

That was a meaty section with lots of detail, yet now the explanation for fructose should be incredibly simple. Many people have heard of fructose but would have a hard time describing where it comes from, and how it is unique. To answer the first question; the most common sources of fructose are: various fruits, honey, and most prevalently, table sugar. Table sugar (sucrose) is made of one molecule of glucose and one molecule of fructose. We already discussed glucose and its path through metabolism, but what about fructose? Fructose behaves very similarly to alcohol, because it is identified as a foreign substance in our body (think foreign currency vs. the native currency of glucose). Because of this, fructose is almost entirely processed in the liver compared with ethanol (80%) and glucose (20%). Like alcohol the fructose makes a beeline for the mitochondrial TCA cycle and overloads it in similar fashion. A glut of citrate results and the subsequent VLDL is then disposed in the same 3 ways discussed with alcohol (DiStefano, 2020). That means that yes, fructose can predispose you to depositing fat viscerally, leading to unnaturally distributed bodyfat (and a significantly higher risk for heart disease). It can also increase your likelihood of developing both muscle and liver insulin resistance. Finally, it can cause Non-Alcoholic Fatty Liver Disease, a legitimate and underrealized condition that is currently experienced by ~20% of the U.S. population (What I've Learned, 2016).

With all of this said it seems reasonable to conclude that fructose in any and all forms is to be demonized for its metabolic aftermath. However, one very important caveat is demonstrated when fructose is eaten from whole foods. Whole foods contain the essential fiber that slow digestion when the food is consumed. This fiber diminishes the absorption rate of the

fructose so the liver can process at a more reasonable pace, thus making the cycle more efficient with less undesirable products. A perfect example of the role of fiber is found in comparing fruit juice with whole fruits. Juices and many fruit byproducts are sold after the fiber has been removed to increase their shelf life. For example, one particular brand of orange juice offers a 110-calorie serving of nutrition packed goodness. However, those calories primarily come from 22 grams of sugar (most of which is fructose), with 0 grams of fiber. It is fair to note that this product boasts significant amounts of calcium, Vitamin D and Vitamin C, however upon further examination each of these three are supplemented by ingredient additives. Conversely, a similar calorie count from raw orange contains slightly less sugar, 6 grams of fiber, and a higher amount of Vitamin C (naturally). One easy way to see how this fiber plays a role in digestion is through the glycemic index. The glycemic index rating for orange juice is 76, categorizing it as a high glycemic food. Conversely an orange comes in at 45, thus making it a low glycemic option (Jedha, 2021). Therefore, I would conclude that naturally occurring foods containing fructose were created for our enjoyment and consumption and we should partake in them with satisfying moderation. Conversely, the vast majority of our packaged grocery items have been stripped of most that is “natural” (primarily fiber for this conversation), and over 80% of them have added sugar (mainly in the form of sucrose which contains fructose. As such, they should be avoided when possible, to combat the negative hepatic (liver), and metabolic effects.

I want to provide a final point to ponder in view of this new information. The term diet is not always synonymous with “beneficial for health.” In the same vein, “healthy” as a term has become warped by merciless marketing, shaped by non-professionals and overall has become

highly subjective. Diet foods can cut fat at the expense of added sugar and smooth it over with biased data and progressive marketing techniques. However, because of these food recommendations, our American diet has become saturated with fibreless quick digesting foods that consistently put our liver into overdrive. And it shows. When looking at the data objectively, it is impossible to mistake the clear correlation between the obesity epidemic and the introduction of diet (sugar laden) grocery alternatives on shelves and in our homes.

Just because something has been marketed as healthy does not mean it is the best option for people to choose.

As always, I hope this was informative for you to take in! If you have any questions arise after reading my summary of the metabolic effects of fructose, feel free to reach out and ask me! Most of all, I hope you took away knowledge that you can later use as wisdom for everyone's well-being.

Always Striving,
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