Patients with hepatitis C or other liver diseases often ask me what to eat, what to avoid, and how to be healthier. I’m asked, “Is sugar bad for the liver? What about stevia, agave and coconut sugar? Is honey good for the liver?” In this article, I’ll discuss sugar and its many forms.

Since hepatitis C is a liver disease, I’ll start with some basic physiology and provide an explanation of the effects of sugar on the liver. The liver acts like the body’s processing plant; everything goes through it—whether you eat it, drink it, inhale it, or put it on your skin.

The Liver: Chief Metabolizer
One of the liver’s main jobs is to aid in digestion and metabolize what we eat. The liver processes fats, proteins, and carbohydrates, turning these into energy and other necessary components to keep us alive and healthy. Our energy levels rely on the liver’s ability to do its job.

Our primary source of energy comes from glucose. The liver acts as the body’s glucose fuel tank and regulator, and helps to keep our circulating blood sugar steady and constant. The liver both stores and manufactures glucose depending upon the body’s need.

— CONTINUED ON PAGE 2
The liver doesn’t do this completely on its own—it relies on insulin to guide it. After eating, blood glucose levels rise, which in people without diabetes triggers the pancreas to release insulin into the blood. Insulin signals the body to absorb glucose from the blood, which our cells use for energy.

When levels of glucose and insulin are high in the blood, the liver absorbs glucose and stores it as glycogen. The liver is like a silo for excess glucose. The liver dispenses this glucose when the body needs it. In people with diabetes, the liver doesn’t process and produce glucose normally.

The Liver and Sugar
Sugar is a simple carbohydrate; it is composed of two molecules—glucose and fructose. Glucose can be metabolized by every cell in the body and if we don’t get it from the diet, our bodies have a complicated way to make it. Glucose is essential to life.

Fructose is different. The liver is the only organ that can metabolize sugar. If your liver has stored all the glycogen it needs, fructose will be converted into fat. This fat can be redirected as blood triglycerides, but most will be stored in the liver, and may cause fatty liver disease.

If you have hepatitis C, your liver is already working hard, so it won’t like any additional work. Add in fatty liver disease and you have a formula for disaster.

What about Agave, Coconut Sugar, Honey & Stevia?
I love honey, so I’m always looking for any medical reason to keep it in my diet. Unfortunately, honey is still sugar. The body doesn’t like it any more than table sugar. However, since honey is sweeter than sugar, one may be able to use less, and in that way, it is slightly better. Some people believe that eating local honey may help allergy sufferers, but there isn’t scientific data to support this. Also, the potential harm too much sugar can do outweighs any potential benefit of allergy relief. The liver processes agave and coconut sugar just like any other sugar. From a manufacturing standpoint, these products are less processed. But from the liver’s standpoint, it is better to skip agave, coconut sugar, honey, molasses, and maple syrup.

As for stevia, I don’t know what to tell you. The Food and Drug Administration (FDA) declared stevia as “generally recognized as safe” (GRAS). Some experts have raised concerns. What disturbs me about stevia is that the products we get commercially have been processed. It isn’t like we are in Paraguay sucking on stevia plants. I use stevia and honey sparingly.

Sugar Recommendations
Don’t get me wrong, I am not a saint when it comes to sugar. I am careful about it, and I relish my small and infrequent treats. If I were facing execution, I’d dive in to vat of sugary baked goods. But, I don’t want to hasten my death by diving in there now.

The World Health Organization (WHO) set its added sugar intake recommendations to 5 percent of one’s daily calorie intake. For an adult of a normal body mass index (BMI), that works out to about 6 teaspoons (25 grams) of added sugar per day.

Here is how to calculate your maximum (Note that 4 grams of sugar = 1 teaspoon) Take 5 percent of daily calories, and divide by 4. Example: 5 percent of 2000 calories = 100 calories/4 = 25 gm/6tsp added sugar per day. So now you know the goal, how do you determine how much added sugar is in a product? That is the hard part.
Although some food producers have voluntarily included the added sugar content, most haven’t. However, most food labels do not differentiate between added sugar and natural occurring sugars, and the only way to identify sugar is by reading the ingredient list. If sugar is listed in the first five ingredients and sugar content is high on the label, the food is likely high in added sugar. Sugar has multiple names, listed below:

- Agave nectar
- Brown sugar
- Cane crystals
- Cane sugar
- Coconut sugar
- Corn sweetener
- Corn syrup
- Crystalline fructose
- Dextrose
- Evaporated cane juice
- Fructose
- Fruit juice concentrates
- Glucose
- High-fructose corn syrup (HFCS)
- Honey
- Invert sugar
- Malt syrup
- Maltose
- Maple syrup
- Molasses
- Raw sugar
- Sucrose

We all have our own bottom lines and you get to decide what yours are. Avoiding added sugar or at least limiting added sugar is a good bottom line if you can do it. Fruit has natural sweetness and can satisfy a longing for sweets. Try adding cinnamon, ginger, or vanilla to create a sweet flavor.

For years, I blamed my fatigue on hepatitis C, but after I eliminated sugar, I noticed my energy levels were higher. On the rare occasions I eat sugar, I feel exhausted later. I missed it at first, but not anymore. Feeling good is better than sugar tastes. And when I partake in something sweet, I enjoy every morsel.

Lucinda Porter, RN, is a long-time contributor to the HCV Advocate and author of “Free from Hepatitis C” and “Hepatitis C One Step at a Time.” She blogs at www.LucindaPorterRN.com and HepMag.com
In case you missed this news in the HCV Advocate blog, here is the cold fact: roughly 97 percent of U.S. prison inmates with hepatitis C are not getting treated. This blatantly defies a 1976 Supreme Court ruling that states that inmates have a constitutional right to medical care. It also violates basic human rights, as well as sensible public health practices. Treatment is prevention, and every person who is successfully treated for hepatitis C reduces further virus transmission.

Click here to read this excellent piece of journalism, reported in Kaiser Health News.

What’s Being Done to Stop the Spread of Hepatitis A in the Midwest?

While more people in the U.S. die from hepatitis C every year than from all 60 reportable infectious diseases combined, another hepatitis virus is making a huge comeback. Hepatitis A outbreaks are occurring across the country, and the Midwest was hit hard this year. Kentucky has incurred one of the worst outbreaks of hepatitis A in the country, and the virus is spreading to Ohio, Tennessee and Indiana.

WOSU public media reported, “Since November, there have been more than 950 cases of hepatitis A in Kentucky, and six people have died. Indiana has had 148 cases in that same time.” Typically, Indiana sees 20 cases of hepatitis A annually. Although no hep A-related deaths have been reported in Indiana, 63 people have been hospitalized because of the disease. These are alarmingly high numbers for a disease that is completely preventable.

Click here to read WOSU’s story in its entirety, and find out what is being done to prevent further hepatitis A transmission.
Efficacy of sofosbuvir and velpatasvir, with and without ribavirin, in patients with HCV genotype 3 infection and cirrhosis—R. Esteban, et al.

Gastroenterology https://doi.org/10.1053/j.gastro.2018.06.042

Study Aims and Results
The aim of the study was to examine the effect of ribavirin on NS5A resistance-associated substitutions (RASs). This was a phase 3 clinical trial of 204 patients with chronic hepatitis C virus infection (HCV). All participants had genotype 3 with cirrhosis, and were treated with Epclusa (sofosbuvir plus velpatasvir) with and without ribavirin for 12 weeks. RASs are treatment-related substitutions that can prevent a NS5a drug—in this case, velpatasvir—from working. The primary endpoint of the study was virologic cure 12 weeks after treatment.

The overall cure rate was 91% in the people who did not receive ribavirin and 96% in the people who did receive ribavirin. But, when they analyzed the results of the people who had previously received treatment and who did and didn’t have RASs, the cure rates were different—84% (with RASs) vs. 96% (w/o RASs). In another analysis that looked at the same baseline characteristics but who received ribavirin, having RASs did not statistically affect the results—99% (with RASs) vs. 96% (w/o RASs).

Conclusions
Epclusa produced high cure rates in people with a difficult to treat population—HCV genotype 3, cirrhosis, prior treatment failures and some who had NS5A RASs.

Editorial Comments
HCV genotype 3 is the most difficult to treat among the HCV population. Cirrhosis, prior treatment failure, and RASs further complicate treatment. The addition of ribavirin didn’t add much benefit to treatment in this study. The people who didn’t receive ribavirin and failed treatment may need to be re-treated with another treatment regimen. Additional studies are needed to find out what direct-acting antiviral (DAA) therapy is needed to re-treat people who don’t respond to Epclusa.

Ribavirin is an interesting drug. I have included some information about it at the end of this column.

“The people who didn’t receive ribavirin and failed treatment may need to be re-treated with another treatment regimen.”
Incidence and spontaneous clearance of hepatitis C (HCV) in PWID at the Stockholm needle exchange - importance for HCV elimination—M. Kaberg, et. al.

Study Aims and Results
The aim of the study was to understand the incidence, spontaneous viral clearance, predictors of new and re-infection of hepatitis C (HCV) among people who inject drugs (PWID) in Sweden.

The trial enrolled 2,320 patients between 2013 and 2016. The people were tested at baseline and answered questions regarding sociodemographic and injection risk behavior. Testing for HCV was repeated every 3 to 6 months.

At the beginning of the study, there were 100 study participants, 77% were HCV antibody positive, and 54% were HCV RNA (viral load) positive. Twenty-four percent (24%) were HCV antibody positive but HCV viral load negative indicating spontaneously HCV viral load clearance.

There was a high incidence of new HCV infections noted. The high rate of new infections was due to the sharing of needles and drug use equipment.

There were no significant differences between the two groups who became infected with HCV. However, there was a significant difference in the spontaneous clearance of HCV in the naïve (never been infected) group (20%) compared to the group who had previously spontaneously cleared the virus (44%).

Conclusions
In this study, there was a high rate of new infections. The authors noted that HCV infections among PWIDs would continue to rise. They also noted that increased harm reduction measures, more comprehensive needle exchange programs, effective drug treatment programs, and scaling up HCV treatment are needed to eliminate HCV.

Editorial Comments
The authors’ conclusions are not surprising. They pointed out that more treatment and services are needed to eliminate or even greatly reduce the prevalence of HCV in Sweden.

Note: the higher rate of spontaneous clearance of HCV among the people who had previously spontaneously cleared the virus has been noted in previous studies.

“The aim of the study was to understand the incidence, spontaneous viral clearance, predictors of new and re-infection of hepatitis C (HCV) among people who inject drugs (PWID) in Sweden.”
Microenvironment eradication of hepatitis C: A novel treatment paradigm—A. Cuadrado, et. al.

Source: *Am J Gastroenterol.* 2018 Jun 27. doi: 10.1038/s41395-018-0157-x. [Epub ahead of print]

**Study Aims and Results**

The study was conducted in a Spanish prison from 2016 to 2017 to create a permanent program to eliminate hepatitis C (HCV) in prisons. All eight hundred and forty-seven (847) inmates consented to participate in the study. The treatment consisted of Harvoni (ledipasvir plus sofosbuvir). The treatment period was 8 to 12 weeks. If participants did not respond to initial treatment, they were given phylogenic analysis and offered retreatment. New cases were evaluated every six months and upon release from prison. A teleconsultation program with a hospital was provided with patients for follow-up.

HCV antibodies were detected in 110 (13%); 86 (10%) had HCV RNA (HCV viral load). The majority were HCV genotype 1 or 3 (83%), and most (52%) had stage 2 or lower fibrosis.

Sixty-nine (69) inmates started treatment. These were prisoners incarcerated longer than 30 days. The cure rate was 97% (64 of 66 patients). Three of these patients were treated with a salvage regimen after failing initial therapy, and were subsequently cured. As of July 2017, no prisoners were reinfected.

**Conclusions**

The consent to participate in the study was 100%, and the cure rate was 97% in the Spanish Prison Program.

**Editorial Comments**

The Spanish model was a success and is a model program for Spain. Other countries should study this program and see if it can be implemented. Worldwide, we will need to cure the incarcerated populations and others if we are going to eliminate HCV in our lifetime.

“Worldwide, we will need to cure the incarcerated populations and others if we are going to eliminate HCV in our lifetime.”
All oral direct acting antiviral therapy against hepatitis C virus (HCV) in human immunodeficiency virus/HCV–coinfected subjects in real world practice: Madrid coinfection registry findings—J. Berenguer, et. al.

Source: Hepatology https://doi.org/10.1002/hep.29814

Study Aims and Results
The authors assessed predictors of treatment response and curing patients in this clinical study of people coinfected with HIV and hepatitis C (HCV) treated with direct-acting antiviral (DAA) medications in Madrid, Spain. The study was conducted between 11/2014 and 8/2016.

There were 2,369 people treated. The predominate genotypes were: genotype 1a (42%); genotype 1b (15%); genotype 3 (15%); genotype 4 (22%). Thirty-four percent (34%) had cirrhosis and 7% had decompensated cirrhosis. The DAA medications included sofosbuvir plus ledipasvir (62%); sofosbuvir plus daclatasvir (15%); dasabuvir plus ombitasvir/paritaprevir/ritonavir (13%) and other regimes (10%). Thirty-one percent (31%) of the combination of DAA therapies included ribavirin. Less than 1% of the patients in the study discontinued therapy.

The overall cure rate was 92%. The cure rate was 94% in people with no cirrhosis, 91% for people with compensated cirrhosis, and 81% of people with decompensated cirrhosis.

The factors that predicted non-response to treatment were male sex, low CD4 cell count (<200/mm³), high viral load ≥800,000 IU/mL, compensated cirrhosis, decompensated cirrhosis, and the use of sofosbuvir plus simeprevir, sofosbuvir plus ribavirin, and simeprevir plus daclatasvir.

Conclusions
This real-world trial resulted in high HCV cure rates in the majority of patients coinfected with HIV and HCV except in known predictors of non-response including older HCV DAA medications.

Editorial Comments
The cure rates in this trial of HIV/HCV coinfection were high except as stated above. Now we are better equipped to treat people coinfected with HIV and HCV with better strategies:
• Using the more potent DAA HCV drugs
• Restoring CD4 cell counts with HIV therapy before starting HCV drug therapy

However, just as in the case as with hepatitis C mono-infection, there are going to be people that may be difficult to cure. But eventually, with the right combination of DAA drugs, we should eventually cure everyone with HCV.
**Abstract:** Use of ribavirin in viruses other than hepatitis C. A review of the evidence—G. Ramirez-Olivencia, et. al.


**Summary:** In the history of hepatitis C treatment, ribavirin was an important component of treatment; when combined with other HCV drugs, it cured hundreds of thousands of people. Now, it is seldom used to treat HCV except on an individual basis.

The authors conducted a review of studies to find out about other diseases that may respond to ribavirin treatment. The two disease families included:
- Hemorrhagic fevers – the most widely known are Ebola, and
- Coronavirus - SARS

These two families of diseases are some of the most lethal diseases known. More research is needed, but if ribavirin can be used to treat these two families of diseases, it would be a major milestone! But it is probably a long-shot.

**A note about ribavirin and HCV treatment:** the exact action of ribavirin is poorly understood. When used as a mono-therapy, it does not cure hepatitis C but used in combination with another HCV drug, it enhances the viral suppression and eradication of HCV. The action of ribavirin is believed to be that of lethal mutagenesis—that is it triggers the hepatitis C virus into a mutation process that when combined with a more powerful HCV drug can help eradicate the hepatitis C virus.

---

**Article:** Genotype from Punjab, India: Expanding classification of hepatitis C virus into 8 genotypes—S. M. Borgia, et. al.


**Summary:** Genotype 8, a newly discovered HCV genotype, was recently identified in Canada. The diversity of the HCV genotypes are classified by their genetic difference of at least 30% from the other HCV genotypes. The four patients with genotype 8 were originally from Punjab, India and were treated with HCV direct-acting antiviral medications. Good news! All four of the treated genotype 8 patients were cured.
The HCV Advocate offers information about various forms of intervention in order to serve our community. By providing information about any form of medication, treatment, therapy or diet we are neither promoting nor recommending use, but simply offering information in the belief that the best decision is an educated one.

Reprint permission is granted and encouraged with credit to the Hepatitis C Support Project.

© 2018 Hepatitis C Support Project