FATTY LIVER
What Is It?
How Did I Get It?
What Can I Do About It?

Fatty Liver…

Fatty liver is an unusual diagnosis because many people hear about it almost accidentally:

“Your ultrasound looks fine, except you have some fatty liver.”

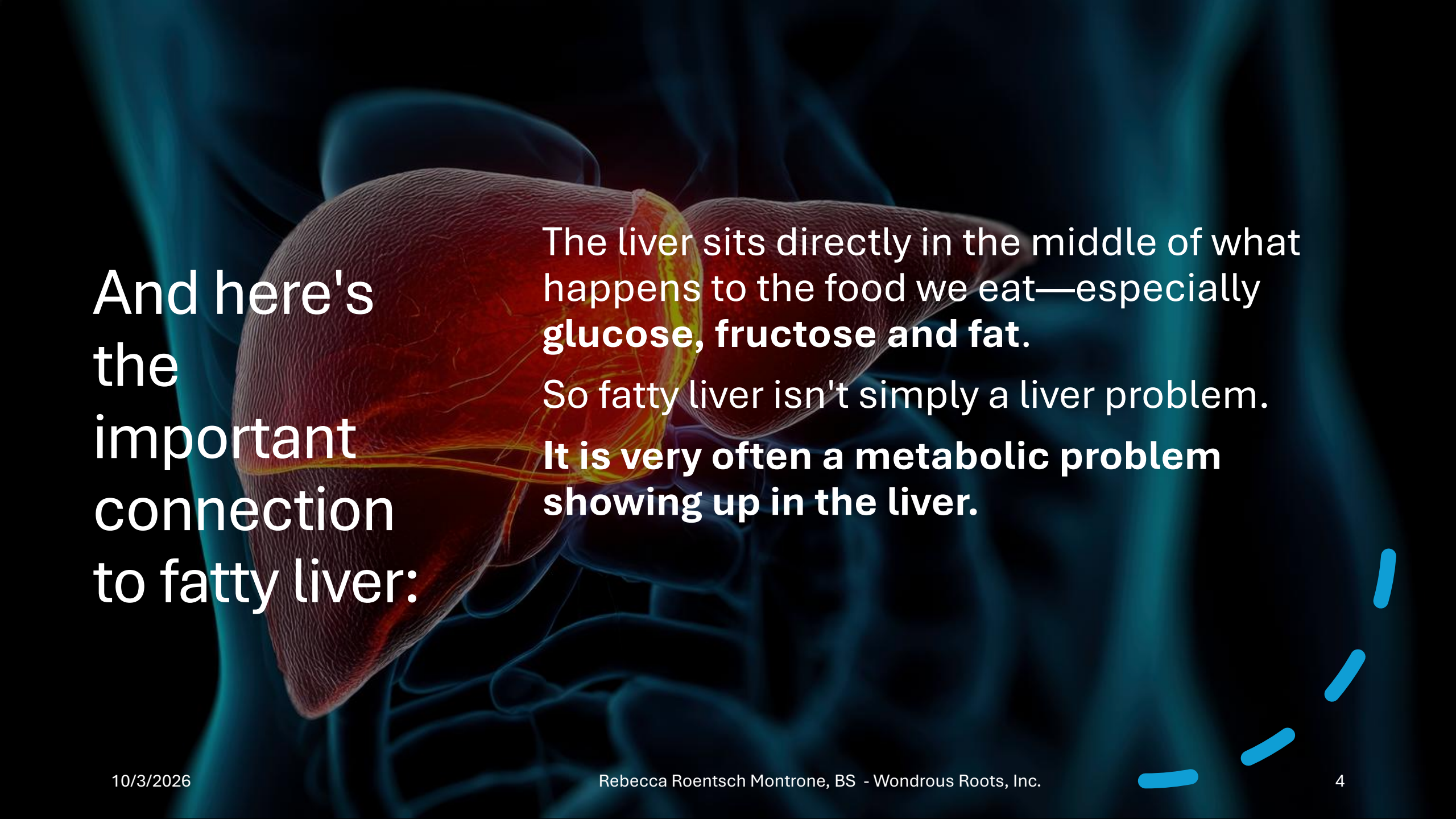
No pain. No obvious liver disease. Sometimes normal liver enzymes. And often very little explanation about what it means.

The encouraging part is that **the liver is remarkably responsive to metabolic change**, particularly before significant fibrosis has developed.

FIRST: MEET YOUR LIVER

The liver is essentially the body's **metabolic processing center**.

- Among its many jobs, it:
- Processes carbohydrates, fats and proteins
- Stores glucose as glycogen and releases it when needed
- Manufactures and packages fats and cholesterol
- Produces bile
- Processes hormones
- Metabolizes medications and alcohol
- Converts ammonia to urea
- Stores vitamins and minerals
- Produces important blood proteins and clotting factors
- Helps neutralize and eliminate potentially harmful compounds



And here's the important connection to fatty liver:

The liver sits directly in the middle of what happens to the food we eat—especially **glucose, fructose and fat**.

So fatty liver isn't simply a liver problem.

It is very often a metabolic problem showing up in the liver.

“FATTY LIVER” HAS A NEW NAME

For years we called the common metabolic form:

NAFLD — Nonalcoholic Fatty Liver Disease

The newer terminology is:

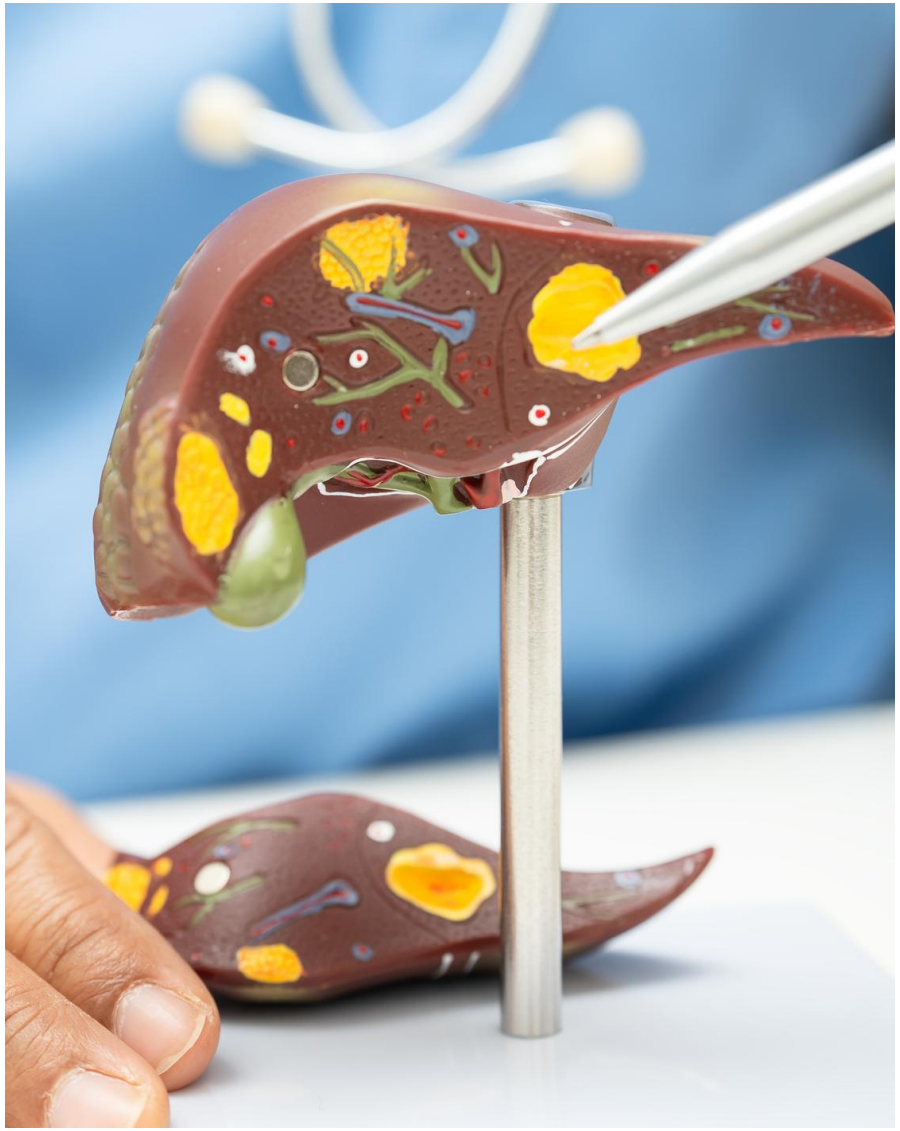
MASLD — Metabolic Dysfunction-Associated Steatotic Liver Disease

“Nonalcoholic fatty liver” told us what supposedly **didn't** cause it.

“Metabolic dysfunction-associated” begins to tell us what **did**.

The inflammatory form previously called **NASH** is now:

MASH — Metabolic Dysfunction-Associated Steatohepatitis.



WHAT DOES “FATTY LIVER” ACTUALLY MEAN?

Quite literally: **Too much fat has accumulated inside liver cells.**

A healthy liver contains some fat.

The problem occurs when **triglycerides** begin accumulating excessively within hepatocytes—the working cells of the liver.

At first, this may cause relatively little injury.

That is **steatosis**.

But in some people, fat accumulation is accompanied by oxidative stress, cellular injury and inflammation.

Now we're moving toward **steatohepatitis—MASH.**

And chronic injury can stimulate the deposition of scar tissue.

THE CONTINUUM

Healthy liver

↓

Steatosis
Fat accumulation

↓

MASH
Fat + inflammation + liver-cell injury

↓

Fibrosis
Scar tissue begins accumulating

↓

Cirrhosis
Extensive architectural scarring

↓

In some individuals:
Liver failure / hepatocellular carcinoma

And an important distinction:

Fat is not the same thing as fibrosis.

Someone can have substantial liver fat without advanced scarring.

Fatty Liver: Are you at risk?
Yes
No

How common is fatty liver disease? Very.

About 1 in 4 to 1 in 3 U.S. adults
is estimated to have metabolic fatty liver disease (MASLD). NIDDK estimates approximately **25–30% of Americans** are affected. NIDDK

Worldwide: about 1 in 3 adults

A large 2024 meta-analysis involving **78 million people in 38 countries** estimated fatty liver disease in approximately **30% of adults worldwide.** PubMed

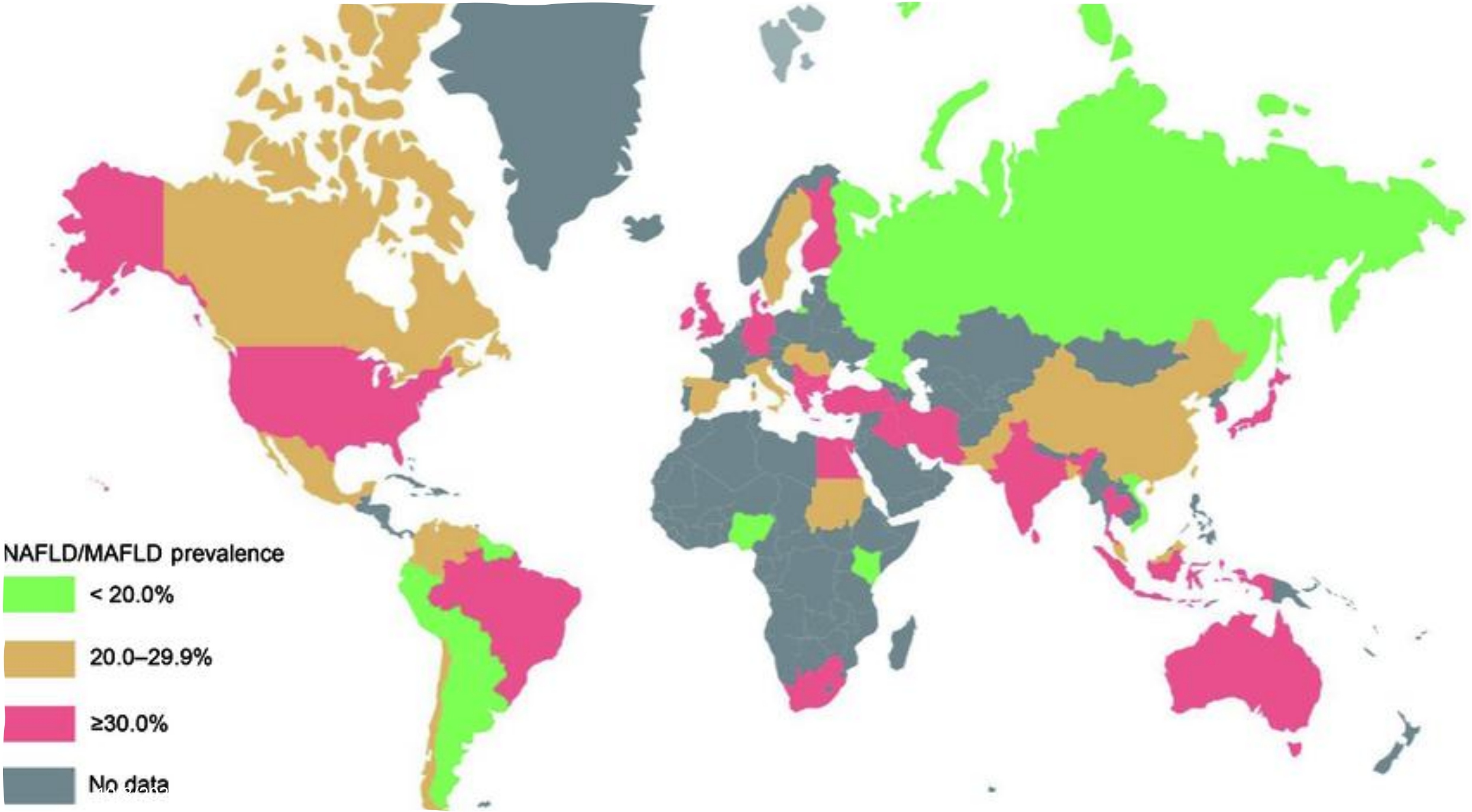
The numbers climb dramatically with metabolic disease:

Overweight/obesity:
Recent pooled data suggest fatty liver may be present in roughly **70%** of overweight/obese populations.

Type 2 diabetes:
Approximately **70%** in recent pooled data; NIDDK has historically estimated **one-third to two-thirds** of people with type 2 diabetes. PubMed

And many people don't know they have it.

Fatty liver frequently causes **few or no symptoms**, which means it may not be discovered until bloodwork or imaging is performed for another reason.


NAFLD/MAFLD prevalence
< 20.0%
20.0–29.9%
≥30.0%
No data

But why would my body put fat in my liver?

The liver isn't simply a passive storage bin that somebody filled with bacon and butter.

Liver fat can come from several sources.

Fatty acids arriving from adipose tissue

When insulin resistance develops, fat cells become less responsive to insulin's normal signal telling them to **stop releasing stored fat**.

More free fatty acids consequently circulate to the liver.

Fat arriving from the diet

Dietary fat can contribute to the liver's incoming supply of fatty acids.

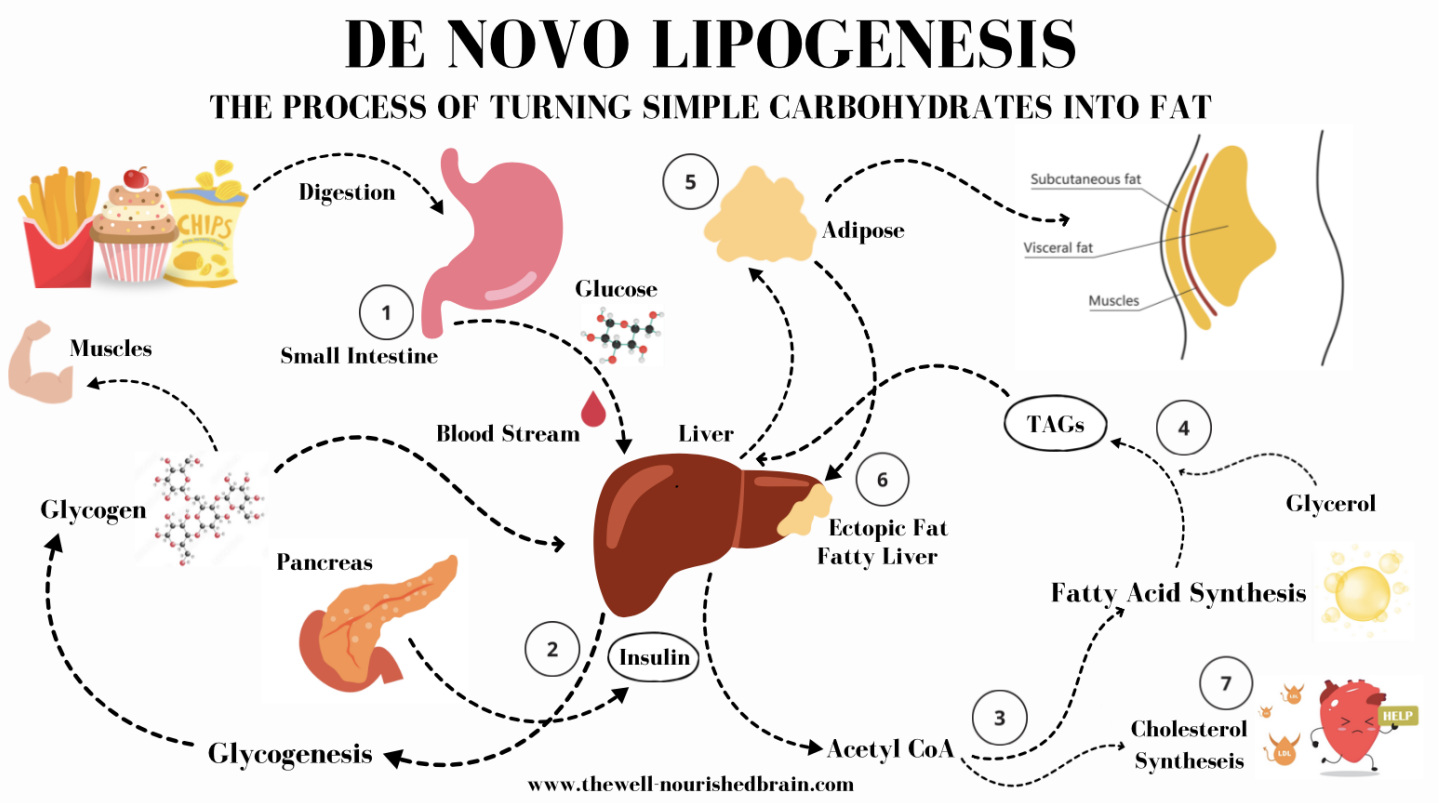
And—the fascinating one—the liver can MAKE fat.

De novo lipogenesis

The liver can convert excess carbohydrate into fatty acids and ultimately triglycerides.

So:

Eating fat is not the only way to develop a fatty liver.



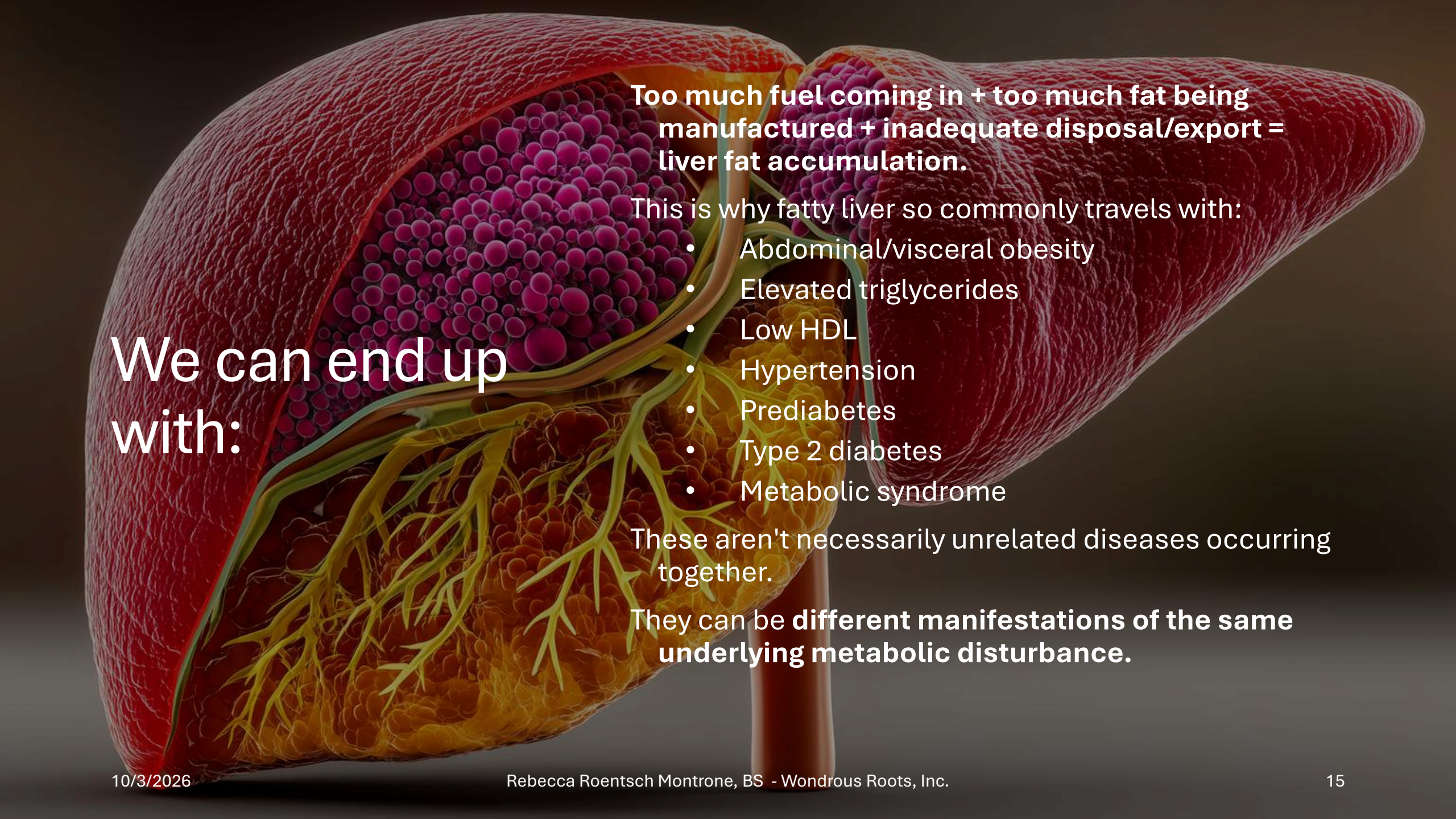
ENTER INSULIN RESISTANCE

Normally insulin helps coordinate what happens to incoming energy after a meal.

With insulin resistance:

- Muscle doesn't handle glucose as effectively.
- Blood glucose begins trending upward.
- The pancreas compensates by producing more insulin.
- Fat tissue releases more fatty acids.
- The liver receives increasing amounts of metabolic fuel.
- The liver can increase production and storage of triglycerides.

Eventually the liver itself can become insulin resistant.



We can end up with:

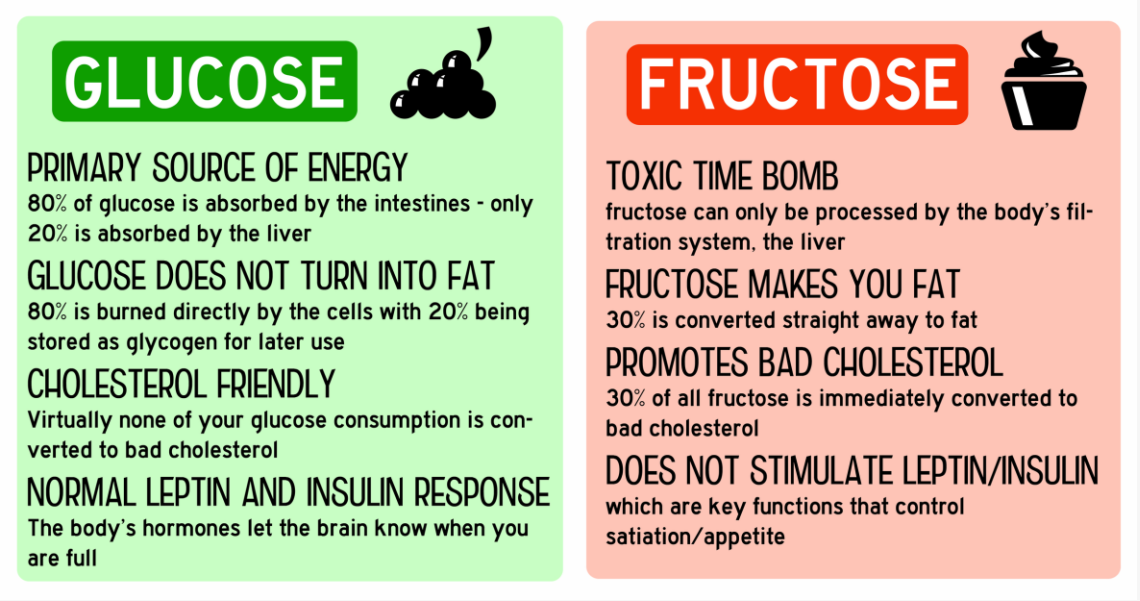
Too much fuel coming in + too much fat being manufactured + inadequate disposal/export = liver fat accumulation.

This is why fatty liver so commonly travels with:

- Abdominal/visceral obesity
- Elevated triglycerides
- Low HDL
- Hypertension
- Prediabetes
- Type 2 diabetes
- Metabolic syndrome

These aren't necessarily unrelated diseases occurring together.

They can be **different manifestations of the same underlying metabolic disturbance.**



THE FRUCTOSE QUESTION

Glucose and fructose are both sugars—but the body handles them differently.

Much of ingested glucose can be utilized throughout the body.

The liver plays a particularly prominent role in fructose metabolism.

Large, rapidly delivered quantities—particularly from:

- Sugar-sweetened beverages
- Soda
- Sweet tea
- Energy drinks
- Fruit drinks
- Added sugars
- Highly processed foods

can provide substrate favoring hepatic **de novo lipogenesis**.

“BUT I'M NOT Overweight!”

Important:

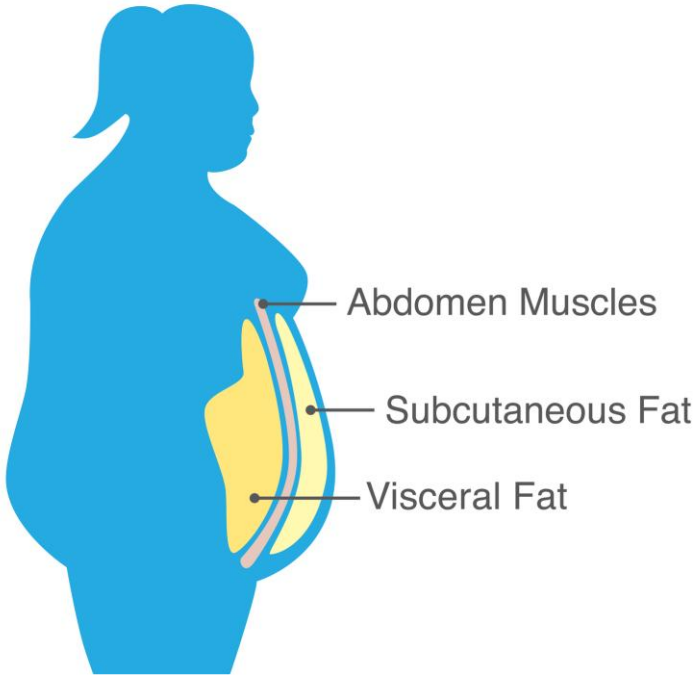
A person does **not** have to have obvious obesity to develop fatty liver.

Body weight doesn't tell us:

- Where fat is stored
- How much visceral fat is present
- How insulin sensitive someone is
- Genetic susceptibility
- Muscle mass
- Dietary pattern
- Metabolic health

Someone can therefore look relatively lean and still develop MASLD.

Subcutaneous vs. Visceral Fat



Subcutaneous fat — the fat we can see

and

Visceral/ectopic fat — fat accumulating around and within organs where we would rather not have it.

It isn't always metabolic

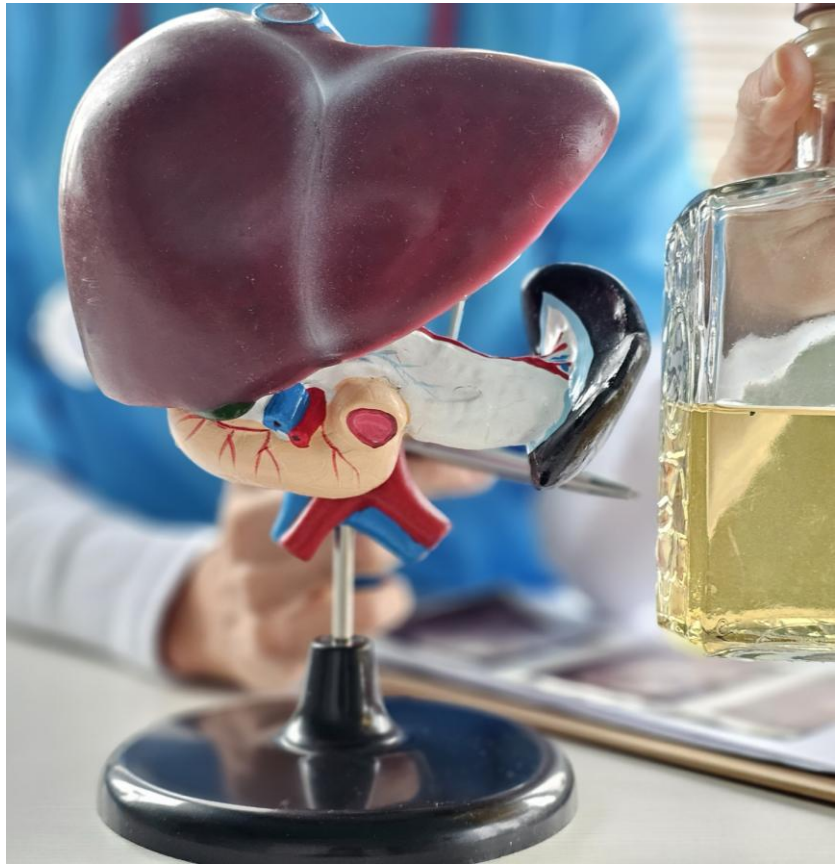
Before blaming everything on sugar and insulin, fatty liver has a differential diagnosis.

Fat can accumulate because of:

- Alcohol
- Certain medications
- Rapid weight loss
- Malnutrition
- Lipodystrophy
- Some endocrine disorders
- Certain genetic/metabolic disorders
- Other liver diseases

*And **metabolic dysfunction** and **alcohol exposure** can coexist.*

So **“fatty liver” describes what we see—not necessarily why it happened.**



“How would I know I have it?”

“Wouldn't my liver enzymes be elevated?”

Not necessarily.

Fatty liver can be remarkably quiet.

Many people have few or no symptoms. When symptoms occur, fatigue and vague right-upper-abdominal discomfort can occur. It may first appear because someone had:

- Routine bloodwork
- An abdominal ultrasound
- CT
- MRI
- Imaging performed for something completely unrelated

What do AST and ALT tell us?

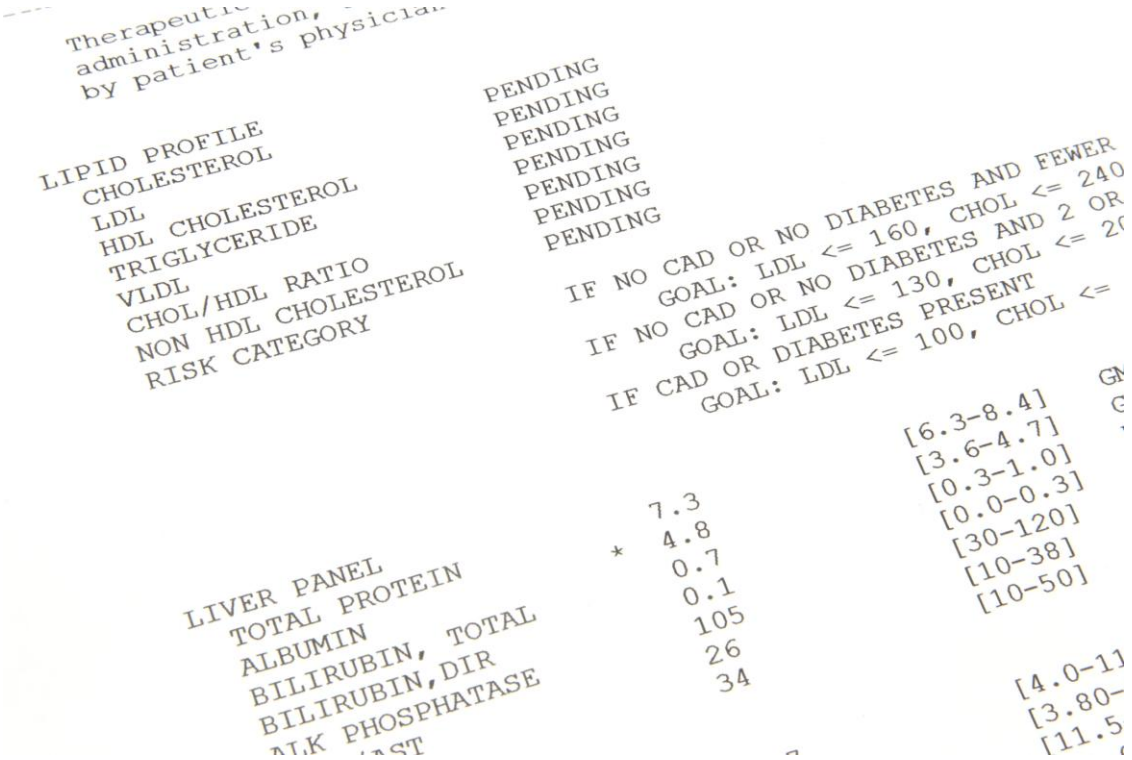
Elevated AST and ALT (liver enzymes) can suggest liver-cell injury.

But they do **not directly measure how much fat is in the liver.**

And they don't reliably tell us how much fibrosis is present.

So normal liver enzymes should not automatically be interpreted as:

“My liver is fine.”



If I have “fatty liver,” how much damage has occurred?

Now the question changes from: **“Do I have liver fat?”** to: **“Has it caused fibrosis?”**

Diagnostic tools can include:

Routine laboratory testing

- AST, ALT, platelets and other markers provide useful information.

FIB-4

A calculation using:

- Age
- AST
- ALT
- Platelet count

It is inexpensive and can help identify people unlikely to have advanced fibrosis versus those who need further assessment.

FIB-4 Score Interpretation Table

Score Range	Interpretation	Next Steps
< 1.45	Low risk of advanced fibrosis	Usually no further immediate testing needed; continue routine monitoring.
1.45 - 3.25	Intermediate risk	Consider additional non-invasive tests (e.g., elastography) or specialist referral.
> 3.25	High risk of advanced fibrosis	Strongly suggestive of advanced liver fibrosis; refer to a hepatologist for further evaluation and management.

This table is for informational purposes only. Consult your healthcare provider for medical guidance.

Other Testing

Ultrasound

Can identify steatosis but is much less useful for determining inflammation or early fibrosis.

Elastography/FibroScan

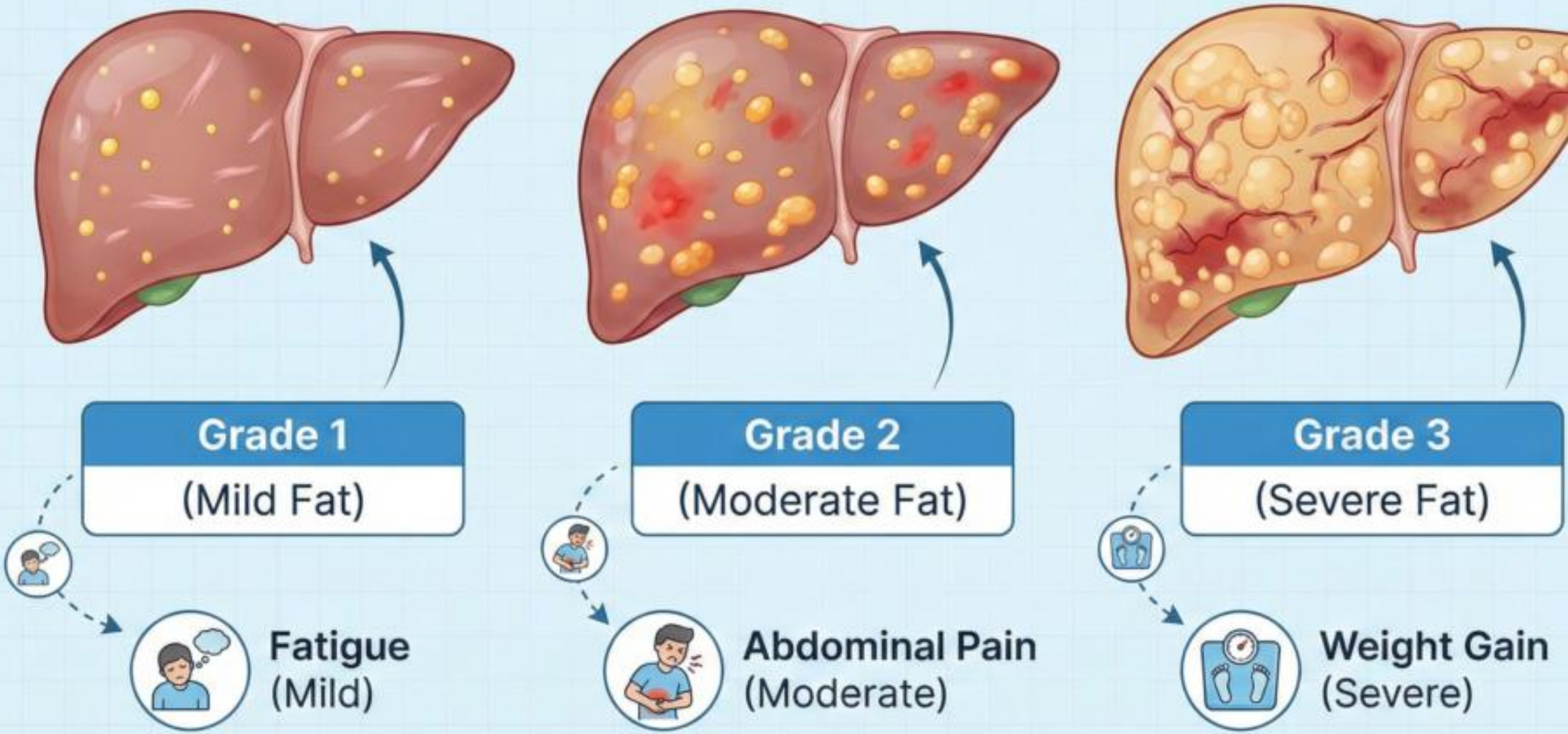
Measures liver stiffness as a surrogate for fibrosis and can also assess steatosis.

MRI-based techniques

Can quantify liver fat and, with MR elastography, assess stiffness.

Liver biopsy

Still provides information that noninvasive testing cannot completely duplicate, but most people with suspected MASLD don't automatically need one.


Grade 1
(Mild Fat)
Fatigue
(Mild)
Grade 2
(Moderate Fat)
Abdominal Pain
(Moderate)
Grade 3
(Severe Fat)
Weight Gain
(Severe)

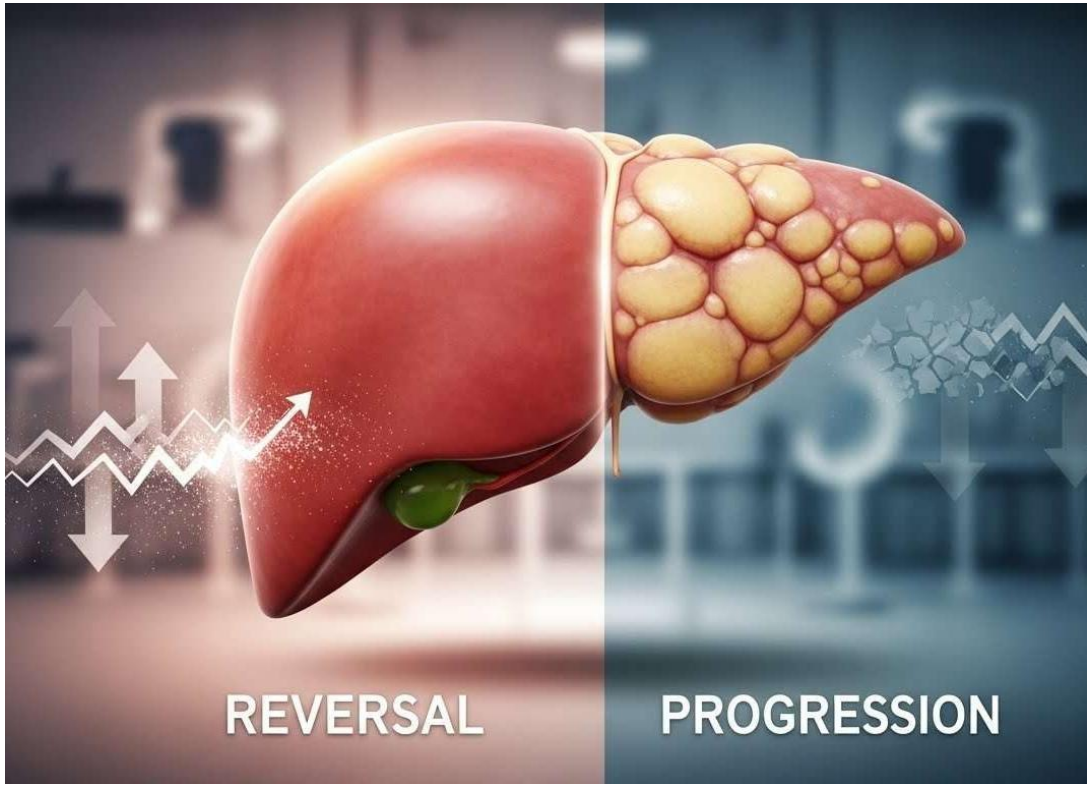
Now for the good news…

Fatty liver is not automatically a one-way trip toward cirrhosis.

Liver fat is metabolically dynamic.

Change the metabolic environment and the liver can respond.

In people with overweight or obesity, even roughly **3–5% weight loss can reduce liver fat**, while greater loss—often around **7–10%**—can produce greater improvement in inflammation and fibrosis. Physical activity itself can be beneficial even without weight loss.



“What can I do about it?”

STOP OVERLOADING THE LIVER

Reduce the major metabolic drivers.

EMPTY THE WAREHOUSE

Mobilize excess stored energy gradually.

BUILD BETTER GLUCOSE DISPOSAL

And here comes one of our stars:

MUSCLE.

Resistance training isn't merely about stronger arms and legs.

Skeletal muscle is an enormous glucose-disposal organ.

More metabolically active muscle + regular contraction = another place for glucose to go.

IMPROVE INSULIN SENSITIVITY

Food composition, meal pattern, activity, sleep, visceral-fat reduction and metabolic health all enter here.

CHOOSE FOOD THAT HELPS

Dietary Do’s for the Liver:

- This is where we can unpack:
- Mediterranean-style dietary patterns
- Protein
- Fiber
- Legumes
- Vegetables
- Whole fruit versus juice
- Omega-3s

Dietary Don’ts for the Liver:

- Refined carbohydrates
- Added sugar/fructose
- Alcohol

COFFEE — GOOD FOR THE LIVER?

Perhaps surprisingly, **regular coffee consumption has consistently been associated with better liver health**, particularly a lower risk of significant fibrosis and advanced liver disease. Benefits are often seen with roughly **2–3 or more cups per day** and appear to involve more than caffeine alone. Coffee contains chlorogenic acids and other polyphenols, diterpenes and additional compounds with potential **antioxidant, anti-inflammatory and antifibrotic effects**. Evidence that coffee actually prevents fatty liver itself is less consistent, but its association with **reduced progression and fibrosis** is quite compelling.

Nutritional & Botanical Support

Lifestyle and metabolic changes remain the foundation for addressing fatty liver, but a number of **nutrients and botanical compounds can provide additional support**. Some help the liver **process and export fat**, others encourage **fatty-acid oxidation and insulin sensitivity**, while still others support **mitochondrial function, antioxidant defenses, healthy inflammatory balance, bile flow and the gut-liver axis**. Rather than looking for a single "liver supplement," the goal is to support the multiple metabolic processes that help the liver **reduce excess fat, protect its cells and restore healthier function.**

CHOLINE

Choline is essential for normal transport of fat out of the liver. It is needed to produce **phosphatidylcholine**, which helps package triglycerides into VLDL particles so they can be exported rather than accumulating within liver cells. Choline deficiency itself can cause fatty liver. Food sources include **eggs, liver, meat, fish, soybeans and other legumes**.

OMEGA-3 FATTY ACIDS — EPA & DHA

EPA and DHA can help **lower triglycerides, reduce hepatic fat synthesis and improve fatty-acid metabolism**. They also produce inflammation-resolving mediators that may help reduce the inflammatory environment accompanying metabolic fatty liver. Omega-3s are especially attractive when fatty liver occurs alongside **elevated triglycerides**.

VITAMIN E

Vitamin E is a major **fat-soluble antioxidant** that helps protect liver-cell membranes from lipid peroxidation and oxidative damage. This becomes particularly relevant when simple fat accumulation progresses toward inflammation and cellular injury. Vitamin E has received particular attention in **MASH**, the inflammatory form of metabolic fatty liver disease.

BERBERINE

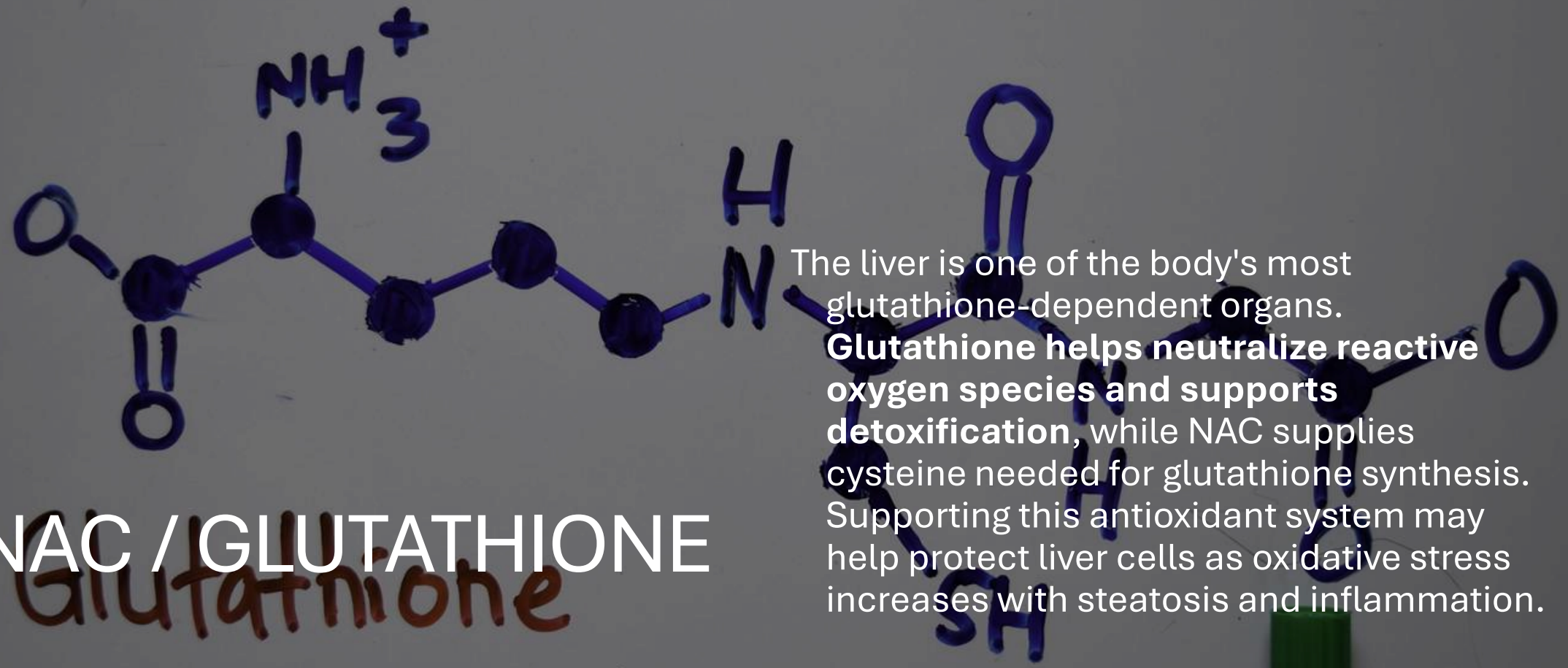
Berberine influences several metabolic pathways involved in fatty liver, including **AMPK**, an important cellular energy sensor. It may improve insulin sensitivity, decrease hepatic glucose production, reduce triglyceride synthesis and encourage fatty-acid oxidation—addressing several of the metabolic disturbances that drive liver-fat accumulation.

CURCUMIN

Curcumin, the principal polyphenol associated with turmeric, has **anti-inflammatory and antioxidant** activity and influences pathways involved in insulin sensitivity and lipid metabolism. It may help reduce hepatic fat accumulation while moderating inflammatory signaling and oxidative stress within the liver.

MILK THISTLE / SILYMARIN

Silymarin is a group of flavonolignans extracted from milk thistle (*Silybum marianum*). It has **antioxidant, anti-inflammatory and hepatoprotective** properties, helps preserve glutathione activity and may help protect hepatocyte membranes from injury. It has a long history of use as a liver-supportive botanical.



NAC / GLUTATHIONE

The liver is one of the body's most glutathione-dependent organs. **Glutathione helps neutralize reactive oxygen species and supports detoxification**, while NAC supplies cysteine needed for glutathione synthesis. Supporting this antioxidant system may help protect liver cells as oxidative stress increases with steatosis and inflammation.

TAURINE

Taurine is highly concentrated in many tissues and participates in **bile-acid conjugation, mitochondrial function, antioxidant defense and lipid and glucose metabolism**. It may help improve insulin sensitivity, decrease oxidative stress and favor healthier handling of fats within the liver.

Magnesium & Vitamin D

MAGNESIUM

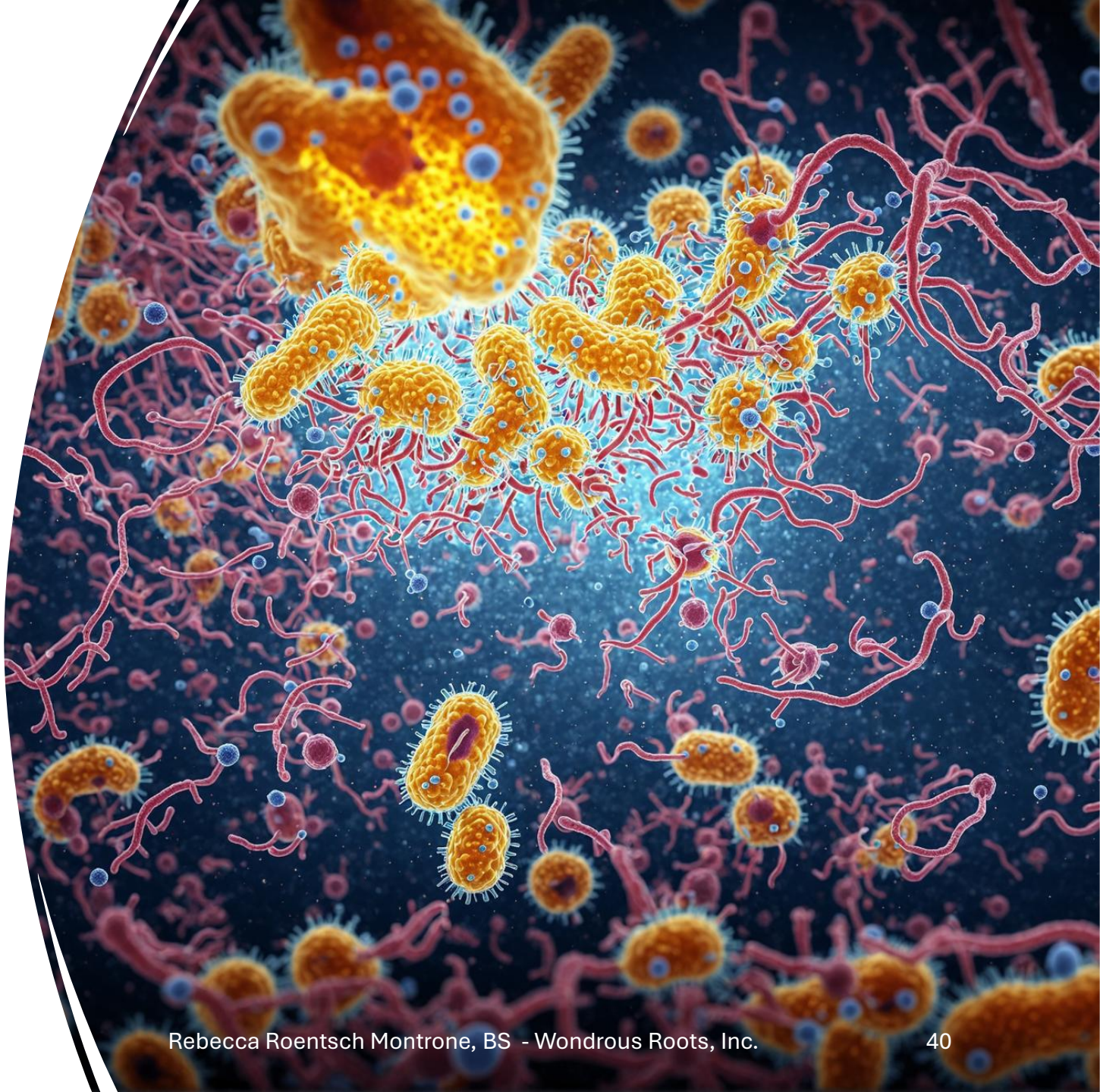
Magnesium is required for hundreds of enzymatic reactions, including those involved in **glucose metabolism, insulin signaling, mitochondrial energy production and lipid metabolism**. Low magnesium status commonly accompanies metabolic dysfunction, making adequate magnesium particularly important when fatty liver occurs with insulin resistance or type 2 diabetes.

VITAMIN D

Low vitamin D status frequently accompanies obesity, insulin resistance and metabolic fatty liver. Vitamin D participates in **insulin signaling, immune regulation and inflammatory control**. Correcting deficiency supports overall metabolic health and removes one potentially unfavorable factor in the metabolic environment surrounding MASLD.

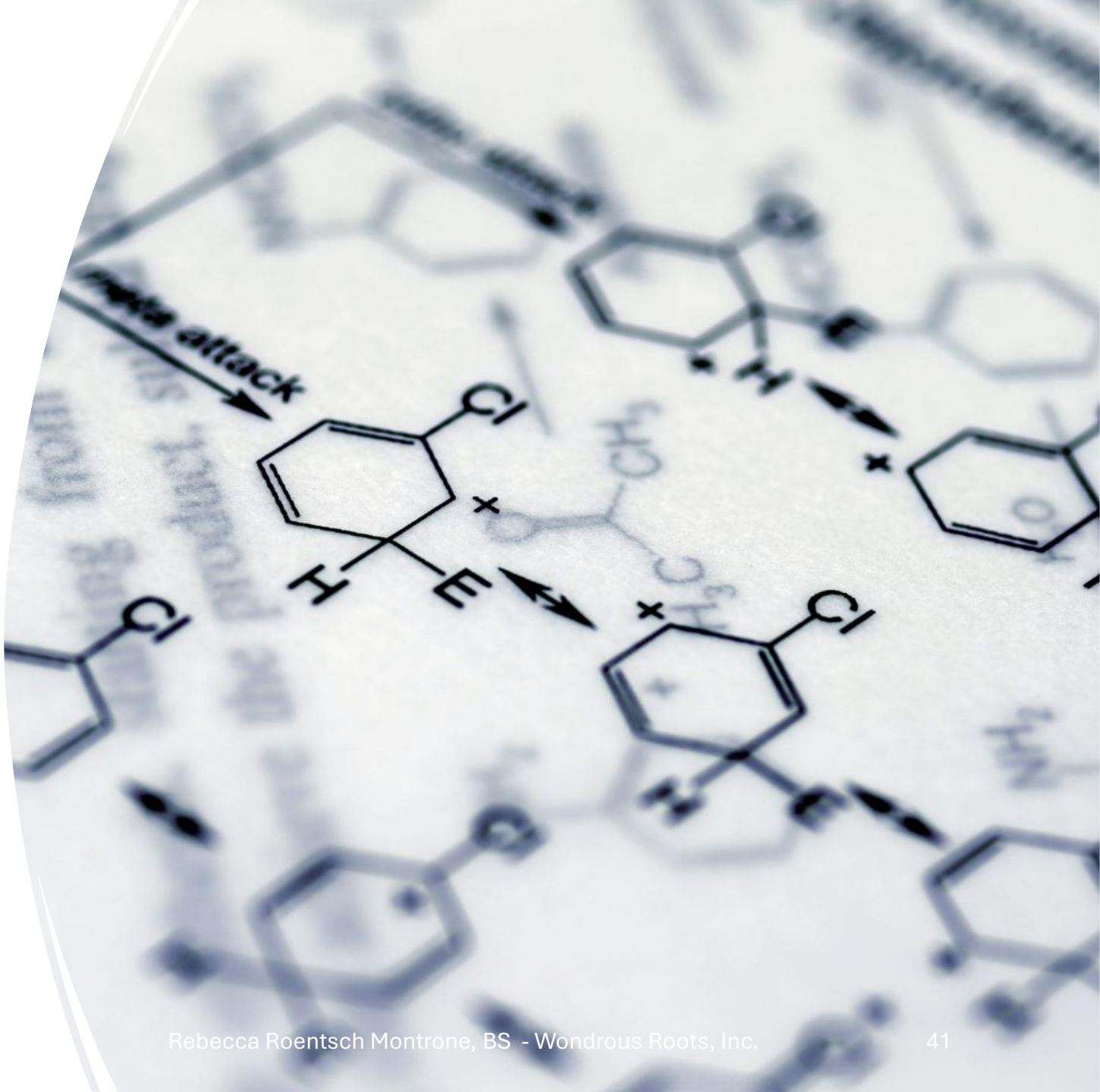
PROBIOTICS / PREBIOTICS

The **gut-liver axis** gives intestinal health a surprisingly direct connection to fatty liver. The liver receives blood from the intestine through the portal circulation, exposing it to microbial metabolites and other intestinal products. Supporting a healthier microbiome and intestinal barrier may help reduce **endotoxin exposure, inflammation and metabolic dysfunction** affecting the liver.



BETAINE

Betaine (trimethylglycine) acts as a **methyl donor** and helps convert homocysteine back to methionine through the BHMT pathway. Methylation is important for phosphatidylcholine production and normal hepatic lipid transport. Betaine also functions as an osmolyte and may support healthy **fat metabolism and liver-cell function**.



ARTICHOKE LEAF & GREEN TEA

ARTICHOKE LEAF

Artichoke leaf (*Cynara scolymus*) has traditional and modern use for **liver, bile and digestive support**. Its constituents—including cynarin and other polyphenols—have antioxidant activity and may favorably influence cholesterol, triglyceride and hepatic lipid metabolism. It also has **choleretic activity**, encouraging bile production.

GREEN TEA / EGCG

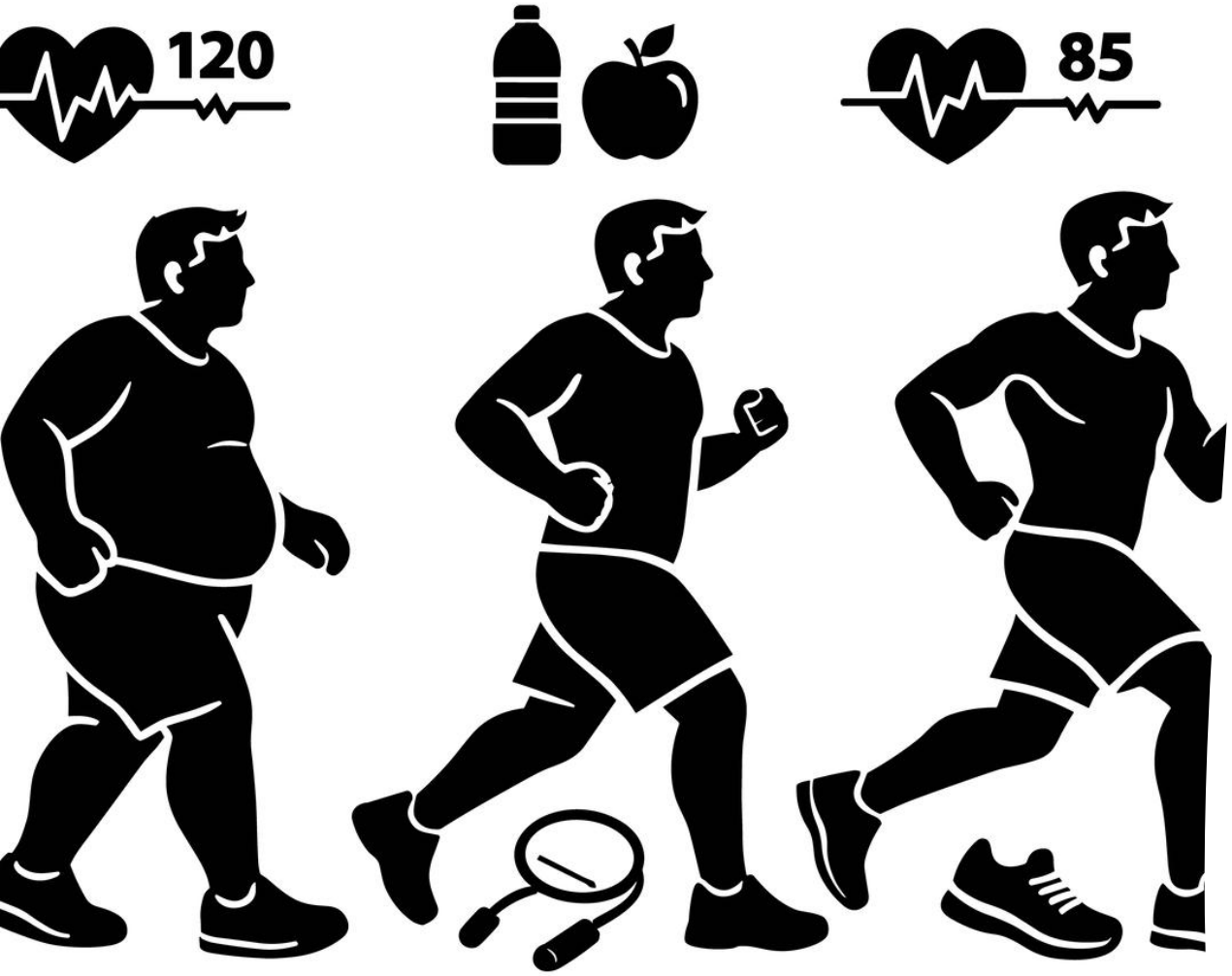
Green tea catechins—particularly **EGCG**—influence antioxidant defenses, inflammation, insulin sensitivity and lipid metabolism. They may help decrease hepatic fat synthesis while encouraging fatty-acid oxidation. For routine use, **green tea as a beverage is distinct from highly concentrated green-tea extracts**, which in excessive doses can themselves injure the liver.

SULFORAPHANE

Sulforaphane, concentrated in **broccoli sprouts**, activates **Nrf2**, a master regulator of the body's antioxidant and cellular-defense systems. It also influences **AMPK, mitochondrial function, fatty-acid oxidation, inflammation and hepatic fat synthesis**. Particularly interesting research suggests sulforaphane may stimulate **lipophagy—the cellular breakdown and removal of accumulated fat droplets from liver cells.** A human trial using sulforaphane-rich broccoli-sprout extract in men with fatty liver found improvements in **ALT, GGT and oxidative-stress markers** after two months.

L-CARNITINE

L-carnitine plays a central role in **fat burning within the mitochondria**. It transports long-chain fatty acids across the mitochondrial membrane so they can undergo **beta-oxidation and be used for energy** rather than remaining available for triglyceride storage. Carnitine may also support mitochondrial function, insulin sensitivity and healthier lipid metabolism, making it especially relevant when fatty liver occurs as part of broader metabolic dysfunction.



Wrapping Up!

Fatty liver is **common, often silent, and closely connected to metabolic health**—but it is also remarkably responsive to change. Improving insulin sensitivity, reducing excess sugar and refined carbohydrate intake, achieving gradual weight loss when needed, exercising regularly and building metabolically active muscle can all help the liver begin unloading stored fat. Alongside these foundational changes, targeted nutrients and botanicals can support **fat metabolism, mitochondrial function, antioxidant protection, inflammation control and healthy liver function**. The important message is that fatty liver is not simply something to watch—it is an opportunity to recognize metabolic imbalance early and **take meaningful steps toward reversing it.**