

# Niacin Effect on Triglycerides and Cholesterol

Elevated triglycerides and cholesterol result largely from poor liver health. That decreased liver function is associated with a Glucogenic/Ketogenic Imbalance and/or an Anaerobic/Dysaerobic Imbalance.

What do you think of prescribing a drug that interferes with liver function to treat a condition caused by poor liver function? Surely you see that is illogical, at best.

What if that drug places a huge burden on liver function to the point of being hepato-toxic, including elevating liver enzymes? Worse than illogical, this type of nonsense is all too typical of allopathic medicine.

Now go a step further --- and consider that the drug that interferes with liver function in an attempt to treat a condition caused by liver dysfunction not only further compromises the unhealthy liver, but whose side effects include exacerbating the very hepatic dysfunction that causes the elevated triglycerides and cholesterol?

Beyond illogical; beyond irrationally damaging; this is insanity.

Yet, the drug in question is prescribed for thousands upon thousands of patients, including many of your own. That drug is Niacin, the Nicotinic acid form of vitamin B3.

Those who are taking Niacin thinking they are doing something “natural” to lower their elevated lipids, are just kidding themselves. The Niacin dose prescribed is entirely pharmacological ---exceeding the nutrition dose by a factor of 100. Yes, 100 times the ideal dose of Niacin is prescribed, doses that are toxic to the liver, and that specifically further impede hepatic control of triglycerides and cholesterol production.

The elevated triglycerides/cholesterol is invariably associated with a fatty liver --- Metabolic Associated Fatty Liver Disease (MAFLD). Every clinical indicator of MAFLD is exacerbated by high doses of Niacin...

One study (Jing Zhou) specifically quantifies the effect of Niacin on MAFLD. It is found that Niacin in nutritional quantities up to 24 mg daily decreases the risk of MAFLD. In other words, those who routinely do not get 24 mg of Niacin daily have an increased incidence of fatty liver. But there is a U-shaped intake curve. Anything beyond 24 mg daily (19 mg in females and 27 mg in males) immediately begins to gradually increase the risk of MAFLD. In other words, it damages liver function. The hepato-toxicity is minimal at first, and accumulates over time, at a very slow pace at first, but then gradually increases with age. After age 60, excess intake of Niacin increases MAFLD incidence more rapidly than that same dose does in the young.

We repeatedly alert you that fatty liver exists in between 30 and 60% or more of adult Americans (--- your patients!), with the incidence of 60% in your patients age 60+. Think of that as “The 60-60 Rule” of fatty liver disease. We have also explained repeatedly that MAFLD is

not something that your patient suddenly “comes down with”. Rather it is a steadily progressing pathology, slow at first, then accelerating in rate as the years go by --- and that the process is evident even in teenagers and children.

This study by Zhou looked at a large group of subjects, male and female, average age 37. Forty percent of these 37-year-olds showed MAFLD by ultrasound examination.

Here are the MAFLD metabolic comorbidities found in these relatively young adults:

- Hemoglobin A1c	+	11%
- Fasting Blood Glucose	+	21%
- Fasting Blood Insulin	+	116%
- Fasting Triglycerides	+	64%
- Total Cholesterol	+	3%
- HDL Cholesterol	-	19%
- High-Sensitivity C-Reactive Protein	+	70%

The implications from these data is clinically valuable to you and your patients. First, all patients whose blood labs show findings in these unhealthy directions are telling you they have created some degree of MAFLD. From a Nutri-Spec perspective that is the same as saying these patients have some degree of Glucogenic/Ketogenic Imbalance and/or Anaerobic/Dysaerobic Imbalance.

The second huge implication of these data is that there are 3 Metabolic abnormalities that are the MAJOR indication of MAFLD. Scan the list above and you will see very clearly --- Fasting Blood Insulin, Fasting Triglyceride, and C-Reactive Protein are the major red flags. The elevated insulin and triglycerides (showing Insulin Resistance, the forerunner of Type 2 Diabetes) along with CRP are the most significant indicators of vascular disease, yet they also define MAFLD.

Insulin Resistance, diabetes, CVD, Metabolic Syndrome, and MAFLD are not separate disease entities, but all part of one unified patho-physiology.

You clearly see the inflammatory component of all those “separate” conditions --- fatty liver (cirrhosis), vascular disease and diabetes --- with all their additional comorbidities such as obesity (particularly abdominal), hypertension, myocardial strain, kidney overload, and cognitive decline (brain inflammation) --- are inter-related aspects of INFLAM-AGING.

Zhou makes the clear point that this same metabolic indicator list of fading Health Span also applies to overdosing with something as seemingly innocuous as Niacin. That is to say --- and to illustrate --- all your patients with elevated triglycerides are exacerbating their condition by consuming high quantities of Niacin --- and certainly by taking the 2,000 mg or more daily commonly prescribed to lower triglycerides.

Again --- do you get it? The very drug prescribed to lower cholesterol and triglycerides will actually further elevate triglycerides.

An ideal fasting triglyceride level is around 100. If 37-year-old individuals unhealthy enough to show MAFLD typically show 64% elevated triglycerides, then these 37-year-olds, otherwise in “good health” (not diabetic, etc.), are statistically speaking showing a triglyceride level of 164. Yet, the so-called “normal” range of blood triglycerides is often claimed to be well over 200. Absurdity? Yes! ----- But, give these individuals Niacin to lower their triglycerides and cholesterol while further compromising hepatic function? No! Restore liver health by correcting the underlying Glucogenic/Ketogenic or Anaerobic/Dysaerobic Imbalances!

Something seen in Zhou’s data is how comparatively insignificant is the cholesterol level in these 37-year-old individuals with MAFLD. Elevated cholesterol is not specifically a part of MAFLD. Fasting cholesterol is elevated in many with MAFLD, but it is elevated in individuals who show no MAFLD --- simply because the deranged liver function resulting in elevated cholesterol relates to liver functions not specifically either cause or effect in fatty liver disease. That is why you have some patients with high triglycerides and normal cholesterol, and other patients with high cholesterol and normal triglycerides.

How can the medical/pharmaceutical establishment “legitimately” claim efficacy for prescribing 2,000 or more mg daily of Niacin for high cholesterol and triglycerides? Does the drug “work”? Pharmacological doses of Niacin very definitely lower both triglycerides and cholesterol. But the mechanism involves overriding the Glucogenic/Ketogenic or Anaerobic/Dysaerobic pathway that led to elevated triglycerides or cholesterol --- by superimposing an additional blockage in hepatic function.

Specifically, damaging doses of Niacin lower triglycerides by several mechanisms. First it blocks the liver enzyme DGAT2, responsible for the final step of triglyceride synthesis. Picture this --- your Anaerobic/Dysaerobic or Glucogenic/Ketogenic patient suffers a liver that has been pushed into excess triglycerides synthesis because of blockages in either the glucose or fatty acid energetics pathway. Now, just as that compensatory triglyceride-producing pathway is ready for completion, the Niacin drug steps in and blocks it. The consequence? Liver damage. Look up the side effects of using Niacin as a drug. Right down the line you will recognize them as signs of liver pathology --- including elevated liver enzymes, jaundice, and the list goes on and on.

Besides blocking DGAT2, 2,000+ mg of Niacin daily also slows triglyceride production by increasing Apolipoprotein B, which is essential for both very low-density lipoprotein and low density lipoprotein synthesis. This is also the means by which Niacin as a drug decreases serum cholesterol. In other words, slowing triglyceride production automatically decreases cholesterol production.

There is yet a third mechanism by which drugging with Niacin will reduce triglyceride production, and consequently cholesterol production. This mechanism reaches an absurd level of insanity. Niacin does not just act on the liver, it acts on fat cells. **It inhibits the breakdown and release of fatty acids stored in adipose tissue.** So now, even less of the triglyceride fatty acids can be delivered to the liver for resynthesis into either triglycerides or cholesterol. ----- BUT --- all you have really done is severely decreased the ability to mobilize fat from that tubby tummy and burn it as energy. Since abdominal weight gain is a primary feature of Insulin Resistance

and MAFLD, and Type 2 Diabetes, the patients swallowing Niacin as a drug are just shooting themselves in the foot in the weight loss department.

INTERESTING SIDE NOTE: Another researcher (Zhao) did essentially the same work as Zhou, but relating Niacin intake to the incidence of depression. The same U-shaped incidence curve exists. At intakes below something near 24 mg daily, there is a higher incidence of depression, and depression incidence improves as individuals' intake approaches 24 mg daily. But beyond that ideal intake, the incidence of depression begins to increase, gradually, but in direct proportion to the excess Niacin. The rate of which the incidence of depression increases at any particular Niacin intake accelerates with age.