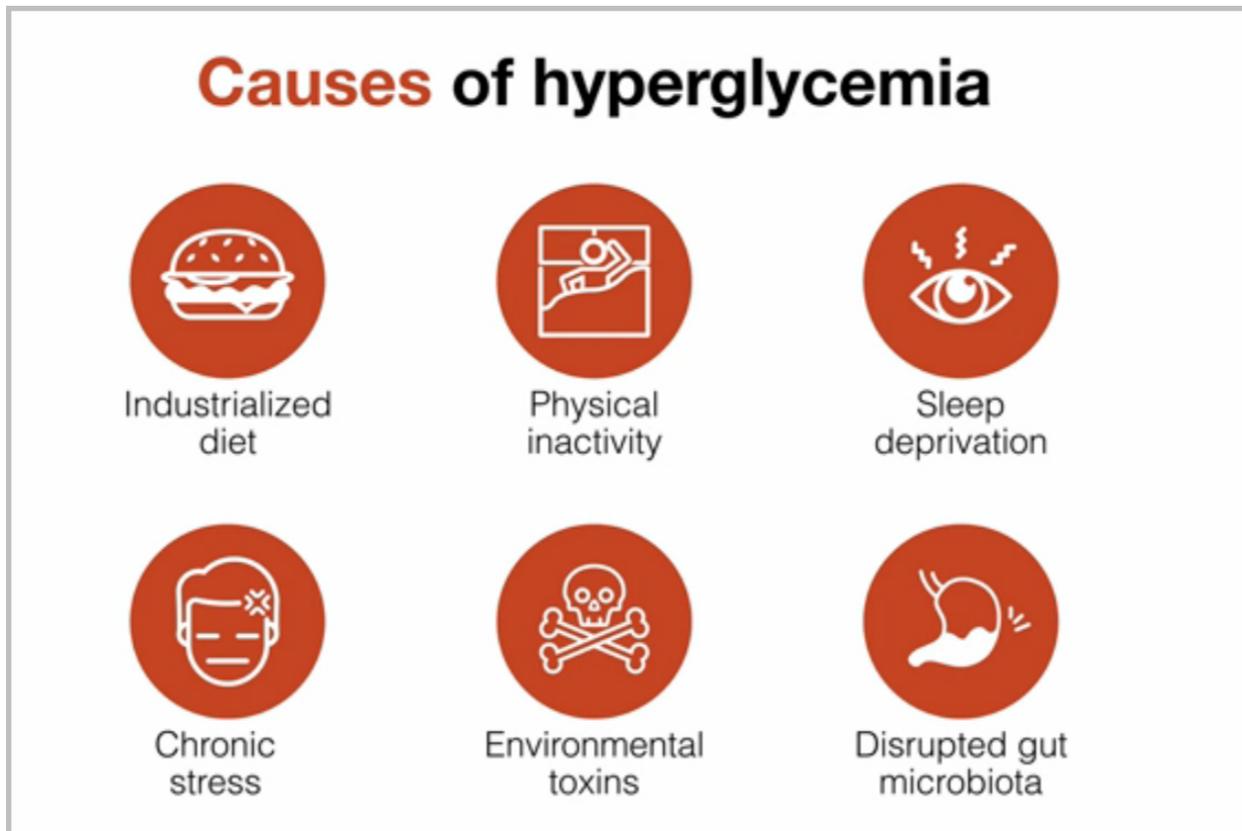


Hyperglycemia I - Part One

Hey, everybody. In this presentation, we're going to talk about hyperglycemia etiology and diagnosis, and then in the second presentation, we'll cover treatment of hyperglycemia.

Hyperglycemia, or high blood sugar, is a modern epidemic. In 2012, almost 10 percent of the U.S. population had high blood sugar, and 27 percent had prediabetes. Diabetes is the seventh leading cause of death, and if current trends continue, one in three people would have diabetes in two decades. Studies of contemporary hunter-gatherers suggest that hyperglycemia is a modern disease, since high blood sugar, obesity, and diabetes are extremely rare in hunter-gatherers following a traditional diet and lifestyle.



Since these hunter-gatherers have similar genetics as us, this suggests that hyperglycemia is mostly driven by environmental factors, and these are numerous, but I've listed what I think the major players are on this slide. These include the industrialized diet; physical activity, lack of physical activity in particular, and sitting too much; sleep deprivation; chronic stress; environmental toxins; and disrupted gut microbiota.

Now that said, this doesn't mean that genetics doesn't play a role in hyperglycemia, and, in fact, several genetic polymorphisms have been identified that predispose certain people to having high blood sugar. This explains why some people become obese and diabetic when exposed to a modern lifestyle while others do not. Instead they might develop an autoimmune disease or allergies and asthma or some other problem instead.

Long-term complications of hyperglycemia

Cardiovascular disease

Nerve damage (neuropathy)

Kidney damage (diabetic nephropathy) or kidney failure

Damage to the blood vessels of the retina (diabetic retinopathy),
potentially leading to blindness

Clouding of the normally clear lens of your eye (cataract)

Feet problems caused by damaged nerves or poor blood flow that can
lead to serious infections, and in some severe cases, amputation

Bone and joint problems

Skin problems, including bacterial infections, fungal infections and non-
healing wounds

Teeth and gum infections

Hyperglycemia is critical to address in any functional medicine treatment plan. It's a risk factor for many other serious diseases, some of which I've listed on the slide, including cardiovascular disease, nerve damage, kidney damage, damage to blood vessels of the retina such as diabetic retinopathy, bone and joint problems, skin problems, and periodontal infections.

The current reference ranges for markers related to blood sugar are far too high on the upper end. Many studies have shown that even high-normal blood sugar, according to the current lab reference ranges, is associated with a significantly higher risk of diabetes, heart disease, and other conditions, so we need to identify and address blood sugar issues early. This is our job as a functional medicine practitioner. It's not only to treat disease. It's to prevent it from occurring in the first place.

Once a patient with hyperglycemia progresses to a certain point along the continuum, the disease spectrum, it can be difficult, if not impossible, to reverse, but if you intervene early, for example, before any irreversible beta cell death has occurred, the prognosis is very good.

Primary Hyperglycemia Markers

Marker	Value
Glucose (fasting)	High
Hemoglobin A1c	High
Fructosamine	High
Post-meal glucose (GlycoMark, OGTT, glucometer)	High (Low for GlycoMark)
Triglycerides	High
HDL	Low
Triglycerides:HDL	High

These are the markers here on the slide that we'll be discussing that comprise the hyperglycemia pattern. They include fasting glucose; hemoglobin A1c; fructosamine; post-meal glucose, which can be measured in a few different ways, using a GlycoMark test, oral glucose tolerance test, or using a glucometer; triglycerides; HDL; and the triglycerides-to-HDL ratio.

There are certainly many, many more markers that could be used to look at blood sugar. Remember, we're covering functional blood chemistry as a screening tool, so we're using the basic markers that are readily available from many labs to identify patterns, and I'm teaching you how to dive deeper on those patterns if you identify one and the basics of how to treat them.

Also recall that you'll have the blood chemistry manual, which goes into detail about each individual marker and provides conventional and functional ranges for each along with indications for when the markers are high or low in both the conventional and functional ranges. So I'm not going to spend time doing that in the presentations. Instead, I can focus on the practical application.

Finally, recall the blood chemistry is all about patterns, not individual markers, so we want to learn to see the entire picture together, both the individual markers that comprise a pattern and then the

clinical signs and symptoms. As we go through not only hyperglycemia but all of the other patterns we're going to cover, keep that in mind.

Secondary Hyperglycemia Markers

Marker	Value
Uric acid	High
Fasting insulin	High
ALT	High
AST	High
LDH	Low
GGT	High

These are the secondary markers that comprise hyperglycemia. They include uric acid, fasting insulin, ALT, AST, lactate dehydrogenase, and GGT. So, again, there are many other markers we could use such as fasting insulin, fasting leptin, HOMA-IR, adiponectin, some of the fatty acids, and those can be helpful with follow-up testing, and you'll see some of these throughout the presentation, but these primary and secondary markers are the best ones to use on an initial case review blood panel to determine a blood sugar problem.

Marker	Value	Functional Range	Lab Range
Glucose	111	75 – 85	65 – 99
Hemoglobin Alc	6.3	4.4 – 5.4	4.8 – 5.6
Uric Acid	5.2	W: 3.2 – 5.5	2.5 – 7.1
BUN	21	13 – 18	6 – 24
Creatinine	0.63	0.85 – 1.1	0.57 – 1.00
Sodium	143	135 – 140	134 – 144
Potassium	4.3	4.0 – 4.5	3.5 – 5.2
Chloride	103	100 – 106	97 – 108
CO ₂	25	25 – 30	18 – 29
Calcium	9.2	9.2 – 10.1	8.7 – 10.2
Phosphorus	3.4	3.5 – 4.0	2.5 – 4.5
Magnesium	2.0	2.0 – 2.5	1.6 – 2.6
Protein, total	7.0	6.9 – 7.4	6.0 – 8.5
Albumin	4.6	4.0 – 5.0	3.5 – 5.5
Globulin	2.4	2.4 – 2.8	1.5 – 4.5
A/G ratio	1.9	1.5 – 2.0	1.1 – 2.5
Bilirubin, total	0.3	0.1 – 1.2	0.0 – 1.2
Alkaline Phosphatase	106	42 – 107	39 – 117
LDH	133	140 – 180	0 – 214
AST	32	W: 10-30	0 – 40
ALT	51	W: 10-22	0 – 32
GGT	65	10 – 26	0 – 60
TIBC	373	250 – 350	250 – 450
UIBC	292	150 – 375	150 – 375
Iron	81	85 – 135	35 – 155
Iron saturation	22	15 – 40	15 – 55
Ferritin	49	MW 33-263	15 – 150
Cholesterol, total	177	150 – 250	100 – 199
Triglycerides	161	50 – 100	0 – 149
HDL	43	55 – 85	> 39
LDL	102	0 – 175	0 – 99
Triglycerides / HDL Ratio	3.744	< 2	< 3.8
TSH	1.120	0.5 – 2.5	0.450 – 4.50
T ₄ , total	8.8	6.0 – 12	4.5 – 12.0
T ₃ Uptake	27	W: 28-35	24 – 39
T ₃ , Total	113	100 – 180	71 – 180
Vitamin D, 25-hydroxy	27.5	35 – 60	30.0 – 100.0
WBC	6.2	5.0 – 8.0	3.4 – 10.8
RBC	4.36	4.4 – 4.9	3.77 – 5.28
Hemoglobin	13.3	W: 13.5-14.5	11.1 – 15.9

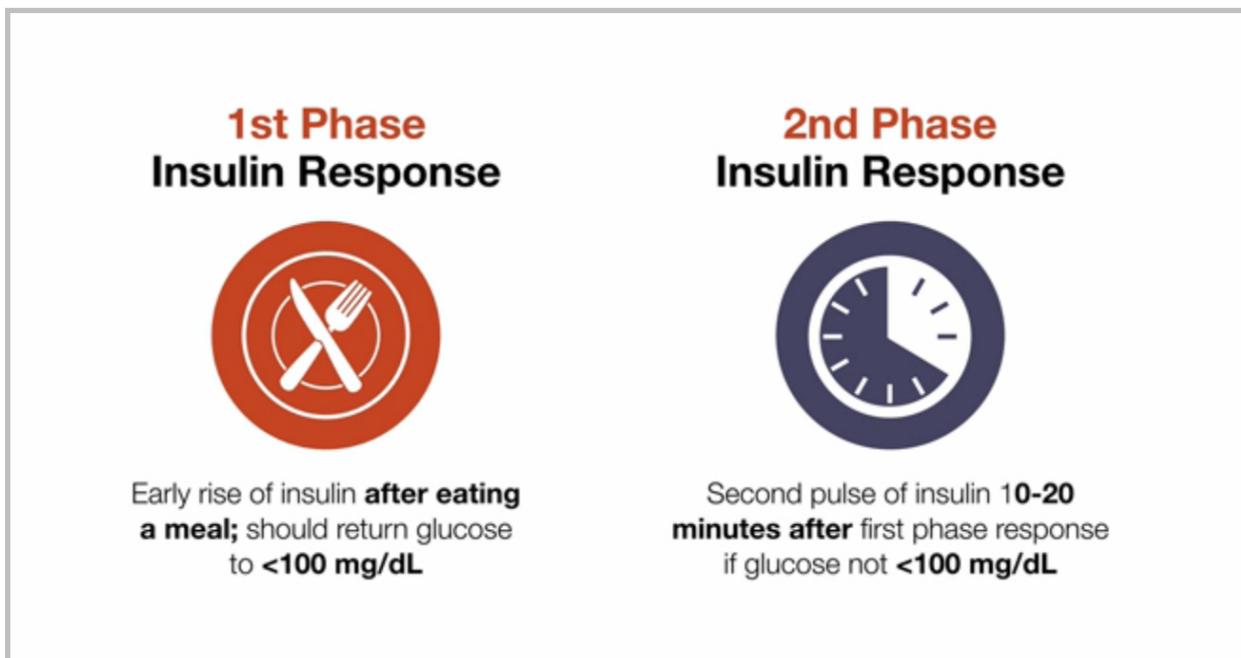
Okay, let's dive right into discussing lab results. I believe that's the best way to learn blood chemistry. So, the first patient is a 59-year-old female with a chief complaint of joint pain. Also has type 2 diabetes, which was not controlled, and she wanted to get off the medications she'd been

prescribed by her physician, if possible. Her fasting glucose is high at 111. As you can see, that's in the pre-diabetic range. A1c is 6.3, also in the pre-diabetic range. Triglycerides, ALT, and GGT were also out of the reference range. Her triglycerides-to-HDL ratio, HDL, lactate dehydrogenase, and AST were out of the functional, or optimal, range. Note that her vitamin D, 25-OH(D), is also low here, which is associated with diabetes and blood sugar issues.

Let's talk a little more about fasting glucose. The fasting glucose test measures the concentration of glucose in the blood after an eight- to 12-hour fast. It only tells us how blood sugar behaves in the fasted state. It tells us very little about how your blood sugar responds to the food that you eat. Fasting glucose is highly vulnerable. There are a number of variables, including recent food intake; sample storage; high intra-individual variability, so our fasting blood sugar varies a lot from day to day; acute stress and diurnal variations, so, of course, it varies throughout the day; common drugs that influence glucose metabolism such as corticosteroids, fibrates, cyclosporine, beta-blockers, methoxazole, thiazide diuretics, and thyroid hormones, among many others.

Fasting glucose is the least sensitive marker for predicting future diabetes and heart disease, so I just want to pause and let that sink in and reiterate it even, that fasting glucose is the least sensitive marker for predicting future blood sugar problems. Unfortunately, that seems little known in the conventional world, which relies heavily on fasting glucose. If you look at the scientific literature, post-meal blood sugar, hemoglobin A1c, and other markers can be better predictors of future issues, with some caveats that we'll cover.

Several studies have shown that a so-called normal fasting glucose level in the mid-90s predicts diabetes a decade later, or at least someone with a fasting glucose in the mid-90s is much more likely to develop diabetes a decade later than someone with a fasting glucose below that level.



So why is this? Well, we have to understand the first- and second-phase insulin response. When you eat a meal, the beta cells release stored insulin immediately. If blood sugar rises above 100 mg/dL, the beta cells release more insulin into the blood. This early release of insulin after a meal is called the first-phase insulin response. In a healthy person, it keeps blood sugar from rising too high because it's available to store most glucose coming from the digestion of a current meal. Now after completing the first phase, the beta cells pause. If blood sugar is still not below 100 mg/dL 10 to 20 minutes later, they push out another smaller second-phase insulin response. In a healthy person, this should bring blood sugar back down to the starting level that it was at before a meal within one to one-and-a-half hours after the meal has finished.