NUTRI-SPEC
METABOLIC BALANCE
SPECIFIC NUTRITION
Live Stronger Longer

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THE NUTRI-SPEC LETTER

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GLUCO/KETO? = Move Slow, Feel Low

It's All About Energy.

Dear Doctor,

Specifically, it's all about ENERGETICS.

WHAT is all about Energetics? Everything is all about energetics.

Our topic is Glucogenic/Ketogenic Imbalance. Our feature is your newly reformulated, mitochondria-feeding Energetics G & Energetics K.

Feeling a little old? --- Gluco/Keto is how that story is told.

Growing slowly fat? --- Gluco/Keto explains all that.

Suffering a little stress? --- Gluco/Keto makes your mind a mess.

Often sore & stiff? --- Either Glucogenic or Ketogenic, it makes no diff.

High or Low blood sugar? --- The essence of Gluco/Keto Imbalance.

Rising Triglycerides? --- Gluco/Keto in the liver-adipose & liver-muscle axes.

Rising Cholesterol? --- The ultimate expression of a Gluco/Keto liver.

Fatty Liver? --- 60% of your age 60+ patients suffer this Gluco/Keto tragedy.

Fatigue? --- Gluco/Keto starvation of mitochondria.

Oxidative Free Radical Damage? --- Gluco/Keto explosions in the mitochondria Electron Transport Chain.

INFLAM-AGING? --- This is HUGE … You already know Nutri-Spec gives you the power, with a combination of Activator, Rejuvenator, Immuno-Synbiotic, and an individualized blend of BALANCING PROCEDURE nutrients to …

DECREASE THE INFLAMMATORY CYTOKINE LEVEL OF 67-YEAR-OLD PATIENTS TO THE LEVEL OF HEALTHY 27-YEAR-OLD INDIVIDUALS.

We have offered many references from the Gerontology research literature confirming you have that power with Nutri-Spec --- power unmatched by any other professional --- to help your patients …

LIVE STRONGER LONGER.

You may want to review our write-up on both Endogenous and Exogenous INFLAM-AGING.

But as you consider both the Exogenous and Endogenous INFLAM-AGING pathways of aging, ask yourself this question --- what accelerates these pathways in patients with a decreased Health Span, compared to those who are robust on an age-adjusted level? ----- It is all about a lack of energy to maintain efficient metabolic processes.

It is all about energetics in the mitochondria.

It is all about Glucogenic/Ketogenic balance feeding mitochondria the ideal blend of Glucose and Fatty Acids to maximize energetics --- meeting the specific needs of brain tissue, muscle tissue, and adipose tissue, and healthfully maintaining the liver as conductor of the orchestra.

Glucogenic and Ketogenic Imbalances are the two major categories of deficient mitochondrial energy production. That means low ATP production and the accompanying deficient production of carbon dioxide. In a Glucogenic Imbalance, there is an impairment of Fatty Acid oxidation. A Ketogenic Imbalance shows an impairment of glucose metabolism. (See Pages 5 & 7 showing normal FA & Glucose pathways --- and blockage locations.)

In a Glucogenic Imbalance, the Fatty Acid pathway through β-oxidation to the Krebs Cycle can be thrown off course. In a Ketogenic Imbalance, the glycolysis pathway to the Krebs Cycle can be diverted. In both Glucogenic and Ketogenic Imbalances, the involved energetics pathway can go off course in the cytosol, before the mitochondria are even reached; energetics can also lose its way shortly after entering the mitochondria.

There is a complexity in Glucogenic/Ketogenic Imbalance that does not exist in your other Nutri-Spec Fundamental Metabolic Balance Systems.

The first level of complexity is that both Gluco/Keto energetic pathways can go off course in several different places. The second level of complexity is that once the pathway goes off course, there are health consequences, forcing an immediate attempt to compensate with a new pathway.

Those compensations have further consequences --- a chain reaction of ever decreasing metabolic strength.

The ultimate level of complexity is that each of these Imbalances will lead to Insulin Resistance, and once there is a state of Insulin Resistance, the prior attempts at compensation are magnified as manifestations of INFLAM-AGING.

Two Glucogenic patients can experience entirely different symptoms, and present somewhat differently to Nutri-Spec Metabolic Testing, as can two Ketogenic patients --- based on whether they have or have not yet reached a certain level of Insulin Resistance.

MECHANISMS OF GLUCOGENIC IMBALANCE

With the impairment of Fatty Acid energetics, there is an attempted (and failed) increase in reliance on glucose oxidation. In younger Glucogenic patients, the somewhat successful push toward glucose over-utilization can cause a reactive hypoglycemia. The blood sugar rarely drops extremely low, but there is extreme instability, with sudden drops in blood sugar.

With the stress of hypoglycemia, there is an attempted compensation with an increased release of Glucagon, and if that fails to elevate the blood sugar, then an increased release of Epinephrine and Norepinephrine to activate Lipolysis. The purpose of that compensation is to increase β-oxidation of Fatty Acids in all cells.

The compensatory Lipolysis transports Fatty Acids from adipose tissue to cells throughout the body (other than RBCs, since they have no mitochondria, and brain cells, since long-chain Fatty Acids cannot cross the blood-brain

barrier). The goal is to activate the Carnitine Shuttle and drive those Fatty Acids into the mitochondria for β-oxidation.

Now ask yourself --- is FA mobilization from fat cells good or bad for the Glucogenic patient? Think about it --- Fatty Acids are being pushed into a pathway that has already shown its inability to use them. (See Page 5.)

Some Glucogenic patients cannot stay on the cytosol pathway that leads to the Carnitine Shuttle. Instead, Fatty Acids are diverted to excess triglyceride formation.

Many Glucogenic patients show a breakdown of the Carnitine Shuttle.

Ideally, the hoped-for β-oxidation creates Acetyl-CoA, which ideally moves to Citrate and then into the Krebs Cycle. BUT, in some Glucogenic patients, before the Krebs Cycle can be reached, hypoglycemia causes diversion to Gluconeogenesis in the Liver. The liver releases the Glucose into the bloodstream (thus making even more Glucose available to these individuals who are already excessively Glucose-dependent), and the remainder of the Acetyl-CoA is used to produce Ketones.

In Glucogenic patients who are older, the system repeatedly flooded with even more excess Glucose, there will be no Glucagon and Norepinephrine attempted compensation. Instead, some Fatty Acids that make it through β-oxidation and into Acetyl-CoA are "stuck." The inability of Citrate to move forward in the Krebs Cycle, causes it to be pushed back from the mitochondria into the cytosol.

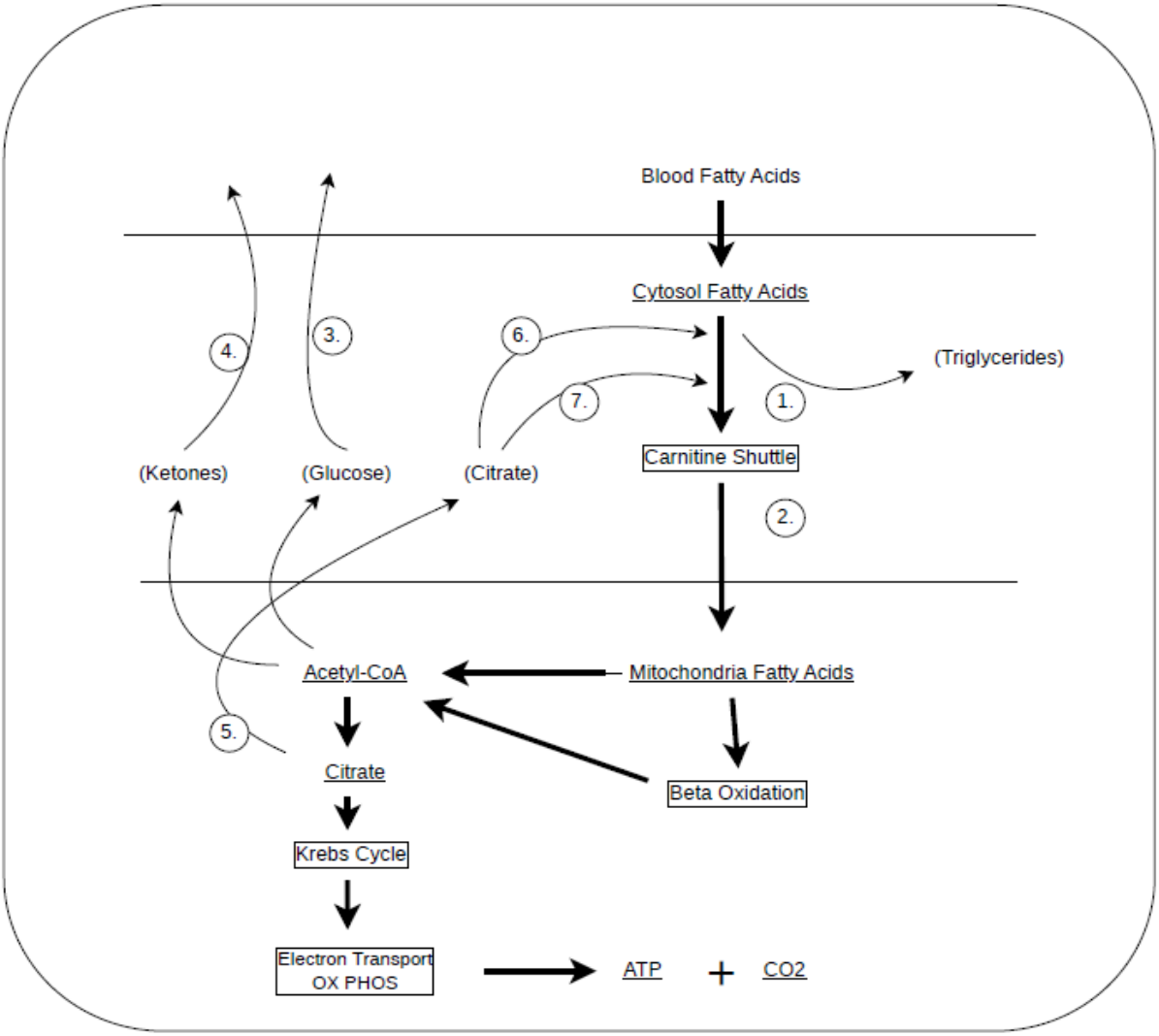
Once Citrate floods the cytosol, it can do absolutely nothing but stir up trouble.

It activates enzymes that further divert cytosol Fatty Acids off track before they get to the Carnitine Shuttle. Other Citrate-activated enzymes further shut down the Carnitine Shuttle itself. There can be excess triglyceride production, excess cholesterol synthesis in the liver, build-up of triglycerides in adipose cells, and above all, the development of Insulin Resistance --- ultimately leading to Metabolic Associated Fatty Liver Disease.

The liver attempts yet another compensation. It activates alternative peroxisomal and cytochrome oxidation of Fatty Acids, producing peroxide products and large amounts of Reactive Oxygen Species (ROS).

GLUCOGENIC IMBALANCE

Fatty Acid Energetics Blocked



1. Fatty Acids diverted into triglyceride excess
2. Carnitine Shuttle "CLOSED due to metabolic difficulties"
3. Liver Glucogenesis, causing Glucose release into the bloodstream
4. Acetyl-CoA converted to Ketones and released into bloodstream
5. Citrate kicked up into the cytosol with disastrous consequences
6. Another blockage diverting FA from the Carnitine Shuttle
7. Another blockage of the Carnitine Shuttle
8. + Triglycerides; + Cholesterol; + ROS; Insulin Resistance; MAFLD

This Nutri-Spec model of Glucogenic Imbalance recognizes how critical the insufficient β-oxidation is. The most important enzyme in β-oxidation is decreased by 40 – 50% in patients with MAFLD.

The excess production of ROS further impairs hepatic β-oxidation, while also damaging mitochondrial function by excessively oxidizing proteins, lipids, and DNA. The overwhelming flood of ROS exacerbates the Insulin Resistance.

* At the point of MAFLD, the Glucogenic Insulin Resistant patient clinically resembles the Ketogenic Insulin Resistant patient. Neither is producing energy efficiently with Glucose.

There is a vicious cycle here: deficient Fatty Acid oxidation in the liver creates a compensatory Fatty Acid oxidation pathway, which generates ROS that damage the mitochondria, and thus further decreases β-oxidation. The ROS damage also increases mitochondrial membrane permeability, leading to the release of mitochondrial damage-associated mitochondrial DNA, cytochrome, cardiolipin, and heat shock protein into the cytoplasm --- which then stimulates an inflammatory response and fibrosis = INFLAM-AGING.

MECHANISMS OF KETOGENIC IMBALANCE

The deficient Glucose energy production, with reliance on Fatty Acid energetics of a Ketogenic Imbalance involves blockages in the Glucose Metabolic pathway in several places. These barriers to Glucose Energetics create several vicious cycles that potentiate the mitochondrial starvation for Glucose and the reliance on less efficient Fatty Acid energy production.

In several places, there are futile Fatty Acid Metabolic pathways by which fats that have been transported from adipose tissue literally go in circles, and then are shipped back to fat cells. Is weight loss your goal? Frustration!

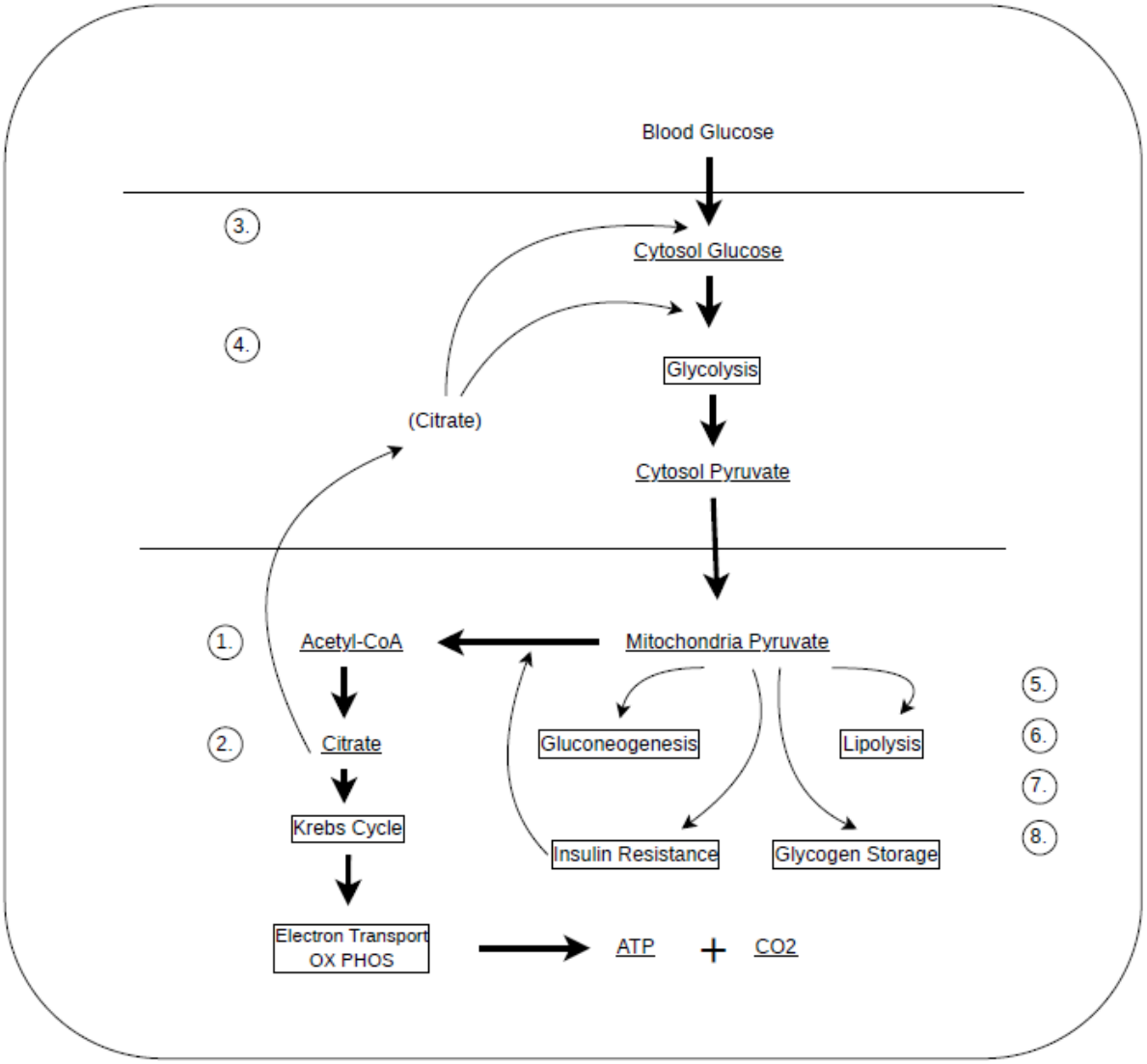
The place where Glucose Metabolism is most severely thrown off course is at the mitochondrial conversion of Pyruvate to Acetyl-CoA. The excess Acetyl-CoA derived from Fatty Acids pushes Pyruvate into alternative pathways.

Similar to a Glucogenic Imbalance, the excess Acetyl-CoA produces excess Citrate, which is pushed back up into the cytosol. In the cytosol, the Citrate completes two vicious cycles. It blocks the uptake of Glucose from the bloodstream, and it blocks Glycolysis enzymes.

Now, the Glucose Energetics, which was already overwhelmed by Fatty Acid β-oxidation, is inhibited even before Glucose can be converted to Pyruvate to enter the mitochondria.

KETOGENIC IMBALANCE

Glucose Energetics Blocked



1. Excess Acetyl-CoA from β-oxidation blocks Pyruvate conversions
2. Citrate kicked out of mitochondria
3. Citrate blocks Glucose entry in cytosol from blood
4. Citrate blocks Glycolysis enzymes
5. Liver Gluconeogenesis
6. Liver & muscle Glycogen storage
7. Conversion to and storage of fat
8. INSULIN RESISTANCE → MAFLD

Meanwhile, Pyruvate, which was blocked from its conversion into Acetyl-CoA, needs some place to go. Any pathway it follows, it further inhibits Glucose utilization. There is increased Gluconeogenesis in the liver; there is increased Glycogen storage; there is increased fat storage, and over time, the individual becomes Insulin Resistant.

This deficiency of Glucose Energetics is exacerbated to an extreme in Ketogenic Individuals by …

- fasting
- long-duration (low-intensity) exercise
- low-carb meals
- Poly- and Mono-unsaturated Fatty Acid intake, even in small quantities

The dominance of Fatty Acid oxidation over glucose oxidation leads to a bigger proportion of electrons being transported to Complex 2 rather than Complex 1 of the Electron Transport Chain. This leads to a less efficient Oxidative Phosphorylation. Mitochondrial production of ROS is increased.

Another mechanism is involved here. The free Fatty Acids released from adipose tissue by Lipolysis, or derived from a high-fat diet, can act directly on the pancreatic Beta cells, decreasing their Insulin secretion. This Ketogenic Imbalance and its reliance on Fatty Acid oxidation and its impairment of glucose oxidation is typical of Diabetes, and in those who fast regularly.

The sad truth is that Glucogenic or Ketogenic Imbalance exists to some degree in almost all your patients. Remember, 60% of your patients are headed for Fatty Liver Disease by age 60. But the process starts early --- really in childhood, with the typical American diet high in sugar and HOHUM PUFAs (Heated, Oxidized, Hydrogenated, Un-Metabolizable polyunsaturated fatty acids).

Energetics G & Energetics K provide all the nutrients necessary to boost energetics --- reestablishing balance between the Glucose and FA pathways to ATP. Supra-physiological quantities of certain Adaptogens and vitamins are required to break down the barriers in the liver-adipose axis, the liver-muscle axis, and the liver-brain axis --- achieving healthy glycemic control, fat deposition and mobilization, and supply of enough energy to drive cellular functions in all organs, and prevent INFLAM-AGING.