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THE NUTRI-SPEC LETTER

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PATIENTS FATIGUED & FAT? --- CYCLING FIXES THAT!

Not bi-cycling! Not motor cycling!

- Metabolic cycling –
- Glucogenic — Ketogenic cycling.
- Glucose — Fatty Acid cycling.
- Liver — Adipose — Muscle cycling.
- Randle Cycling.

Dear Doctor,

Fatigued patients cannot “ride” their cycle without running out of fuel.

Fat patients so weigh down their cycle it can barely move down the road.

All your patients have 2 engines — one energized with Glucose, the other fired up by fatty acids. But your Glucogenic patients? Your Ketogenic patients?

**FUEL LINES CLOGGED.
ENGINES WITH NO SPARK.**

Fatigued? Moving slow, feeling low? It's all about a mitochondrial energy deficit. It's all about the need for Energetics G or Energetics K, along with a very specific eating plan to restore cycling between engines.

Overweight? Again, it's all about mitochondrial energy deficit from clogged-up, sparkless engines --- the failure to cycle between Glucogenic and Ketogenic energetic pathways.

In your [May Letter](#), we gave you a deep dive into [OEA](#), highlighting how OEA cycles liver, adipose, and muscle energetics toward Glucogenic fat burning.

In your [September Letter](#), we echoed that message by placing the emphasis on [Carnitine](#), and how it teams up with OEA and the other nutrients in your Energetics G to break down the clogs in the Ketogenic pathways, providing the fatty acids as the fuel your Glucogenic patients so desperately need.

Lest you conclude that only Energetics G is about helping fatigued and fat patients --- in your [June Letter](#), we showed you how Energetics K has at least as powerful an effect on mobilizing body fat and increasing vitality as does Energetics G. The emphasis is on [Lipoic Acid](#) and [Niacinamide](#) and their mitochondria cycling effects --- helping the liver escape toxic metabolism of polyunsaturated fatty acids (HOHUM PUFAs = Heated, Oxidized, Hydrogenated, Un-Metabolizable Poly-Unsaturated Fatty Acids), and restoring the liver's capacity to cycle efficiently between the glucose and fatty acid metabolic engines.

When we say "cycling," we mean more than just the Randle Cycle (glucose/fatty acid cycling) that allows every cell to select, moment to moment, the ideal fuel --- glucose or fatty acids --- to load its mitochondrial fire. There is also cycling within both the glucose and fatty acid energetics pathways.

Pyruvate Cycling refers to the intracellular loop between the Mitochondria and the Cytosol using Pyruvate combined with Krebs Cycle intermediates. There is a Pyruvate-Malate Cycle, Pyruvate-Citrate Cycle, and Pyruvate-Isocitrate Cycle. This cycling is critical in both Glucogenic and Ketogenic Imbalances.

Pyruvate is the product of cytosolic Glycolysis that then penetrates the mitochondria for conversion to Acetyl-CoA and entry into the Krebs Cycle. In a Glucogenic Imbalance, with deficient fatty acid oxidation, this glucogenic pathway to Pyruvate production can be grossly over-efficient, with excess Pyruvate clogging the mitochondria.

Excess Pyruvate accumulates in a Ketogenic Imbalance as well, since the dominant fatty acid pathway overloads the mitochondria with Acetyl-CoA and NADH, both of which are needed for Pyruvate to be converted to Acetyl-CoA.

Pyruvate Cycling is the regulator, preventing the excess accumulation of Pyruvate in your healthy patients. But in a Glucogenic or Ketogenic Imbalance, the Pyruvate cannot be properly cycled, and follows alternative pathways. The end results are all anti-metabolic alternatives that lead to Insulin Resistance:

- Liver and kidney gluconeogenesis = elevated blood glucose
- Liver and adipose cells produce excess triglycerides
- Pancreas β -cell function is over-activated, releasing excess Insulin, thus accelerating the progression to Insulin Resistance.

In your Ketogenic patients, Pyruvate Cycling is increased, resulting in increased Insulin secretion in response to chronically elevated blood glucose. Liver glucose production is still properly maintained in this condition. But, fat cell mass is expanded (particularly in abdominal adipose tissue) by the increased expression of fat synthesis enzymes.

In response to the high blood Glucose, fat cells expand concomitantly with the over-expression of Pyruvate Cycling. The excess Free Fatty Acids and Triglycerides are stored in adipose tissue as a result of Insulin action.

However, chronic exposure of beta-cells to elevated Glucose due to Insulin Resistance results in decreased Pyruvate Cycling activity. The over-production of hepatic Glucose caused by Insulin Resistance results in severe blood sugar increase, creating a “glucotoxicity cycle”. This condition, in turn, causes down-regulation of Pyruvate Cycling, concomitant with pancreatic failure.

In your Glucogenic patients, when they have progressed to Insulin Resistance, and particularly if they have reached the stage of Type 2 Diabetes, the regulation of peripheral Glucose levels is no longer under the regulation of Insulin. In Type 2 Diabetes, chronic exposure of β -cells to Glucose due to peripheral Insulin Resistance results in decreased Pyruvate Cycling.

The continued overproduction of Glucose (Gluconeogenesis) in the liver causes simultaneous inhibition of Insulin secretion. Thus, in both your Glucogenic and Ketogenic patients, the Type 2 Diabetes that derives from Insulin Resistance eventually results in Insulin-dependent Diabetes.

There is another nasty cycle involved here such that the excess triglycerides and free fatty acids in circulation (that can occur in both your Glucogenic and Ketogenic patients) further impairs pancreatic function, accelerating the decline to insulin-dependence. In essence, you have Type 1 Diabetes superimposed over Type 2 Diabetes.

Since both Glucogenic and Ketogenic Imbalances ultimately result in Insulin Resistance, they are both exacerbated to an extreme by a high-fructose diet (fruit, honey, sucrose). In response to the chronically elevated blood glucose, an early adaptive response of the pancreas is to increase Pyruvate Cycling, but over the years, the pancreatic β -cells burn out. Chronic exposure of the pancreas to elevated glucose due to Insulin Resistance results in decreased Pyruvate Cycling.

At the same time, in both Glucogenic and Ketogenic patients, there is an accumulation of triglycerides and free fatty acids in the circulation. These fats further impair pancreatic function and create a “lipotoxicity cycle”.

Simultaneously, over-eating (especially of HOHUM PUFAs & fructose) causes Insulin Resistance in adipose tissue with accumulation of Triglycerides and Free Fatty Acids in circulation. These changes result in the decline of β -cell function in diabetes.

FATIGUE & WEIGHT GAIN ARE 2 SIDES OF THE SAME COIN.

Every pound of excess body fat represents thousands of calories --- fatigue-relieving energy --- that should have been burned in the mitochondrial fire --- but wasn't.

Cycles within cycles within cycles? You need never concern over these complexities; Nutri-Spec handles that for you. Simply look at the fundamentals ...

Yes, both fatigue and weight gain are all about Glucogenic/Ketogenic Imbalance --- the failure of mitochondria to cycle efficiently between the Glucogenic and Ketogenic metabolic engines --- meeting the specific needs of brain tissue, muscle tissue, and adipose tissue --- while healthfully maintaining the liver as the driver of cycling from one power source to the other.

Glucogenic/Ketogenic Imbalance 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.

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 lose its way in the cytosol, even before the mitochondria are called upon.

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 be blocked 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 inefficient pathway.

Those compensations have further consequences --- a chain reaction of ever-decreasing Metabolic Cycling 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.

The key concept here is that INFLAM-AGING does not accrue due to getting older per se. Both Endogenous & Exogenous INFLAM-AGING have deficient energetics as fundamental causes. Restore strength in Metabolic Cycling with Energetics G & K.

To restore Randle Cycle dynamics to your fatigued & fat patients --- and --- to control the INFLAM-AGING caused by low energetics ----- Take advantage of ...

This month's SPECIAL --- 1 **FREE** with every 5 you buy of Energetics G, Energetics K & Rejuvenator.

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