NUTRI-SPEC
METABOLIC BALANCE
SPECIFIC NUTRITION
Live Stronger Longer

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THE NUTRI-SPEC LETTER

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BURN FAT? ENERGETICS K DOES THAT!

(And so does Energetics G!)

Dear Doctor,

OEA burns fat. OEA prevents obesity. OEA controls regional fat distribution. OEA controls food consumption.

OEA supplementation yields significant improvement in all the pro-inflammatory and pro-aging sequelae of obesity --- as it brings down blood sugar, decreases Insulin, decreases Insulin Resistance, decreases HbA1c, and decreases C-reactive Protein.

OEA is a critical nutrient in your Energetics G.

Energetics G also gives you a broad array of other fat-burning nutrients --- most notably Carnitine, Ornithine, and Riboflavin.

Dysinsulinism → Insulin Resistance → Type 2 Diabetes + Elevated Triglycerides & Cholesterol → Weight Gain → Metabolic Syndrome + Fatty Liver → Increased incidence of Cardiovascular Disease & Cancer. Energetics G maintains healthy energetics in resistance to every step along the way.

You may have concluded from last month's Letter that Energetics G is your Nutri-Spec "fat-burning supplement." ----- And that Energetics K must do something else.

As it turns out, Energetics K gives you and your patients just as much fat-metabolizing benefits as Energetics G, but by entirely different mechanisms.

Do you remember The 60-60 rule for Fatty Liver? 60% of your patients over age 60 have some degree of Fatty Liver Disease. 35% of all your adult patients have some degree of MALFD (Metabolic-Associated Fatty Liver Disease).

Some are pushed through the pathway to MAFLD by a Glucogenic Imbalance, others by a Ketogenic Imbalance. With Nutri-Spec, you appreciate the concept of Biological Individuality. You do not offer remedies for high blood lipids, high blood sugar, or cardiovascular disease. Rather than treating diseases, you guide your patients toward regaining and maintaining their health span, and maximizing their life span …

LIVE STRONGER LONGER.

You will empower some patients by addressing their Glucogenic Imbalance, and others by reversing their Ketogenic Imbalance.

If you are Glucogenic (or, if you eat a high-fructose sugar diet), you are unable to access Fatty Acid Energetics. You become metabolically programmed to rely on sugar to produce energy.

The inaccessible fatty acids build up in your liver and fat cells. The excess sugar energetics drive you into blood sugar instability, with frequent spikes and crashes in blood sugar. That goes on for a period of years as you gradually become Insulin Resistant, eventually succumbing to a Metabolic Syndrome fatty acid pathway.

If you have a Ketogenic Imbalance (or if you eat a high-fat diet --- particularly a diet with even a little polyunsaturated or monounsaturated fatty acids), you are unable to access Glucose for a clean, hot, energetic fire. Instead, you are bogged down in an overload of fatty acids that you can only metabolize very inefficiently.

The sugar that cannot be pulled through the Glucogenic pathway via Glycolysis, then the Krebs' Cycle, and ultimately into the Electron Transport Chain, builds up

into elevated blood sugar, then backs up into fat cells, leading to weight gain and Insulin Resistance. As the liver and muscles do their best to create some energy while being bogged down with nothing but fatty acids to deal with, they go off-course into futile metabolic cycles.

A futile Ketogenic metabolic cycle simply means that the cycle goes in circles. It produces little to no energy, and ends up right back where it started, bogged down in gobs of fat. Some of that fat stays stored in the fatty liver, while the rest is shipped back to fat cells.

An in-depth look at Ketogenic Imbalance shows that there are many places where Glucose Metabolism is blocked.

The most fundamental is that excess fatty acids --- either from Ketogenic Imbalance or from unsaturated fatty acids in the diet --- blocks the action of GLUT4, the gatekeeper of Glucose entry through the cell membrane. The Glucose builds up in the blood, and backs up into fat cells.

Another major barrier to Glucose Energetics in a Ketogenic patient occurs in the mitochondria. As what little Glucose can trickle through the energetic path through Glycolysis to form Pyruvate ("mitochondria food"), there is a major barrier right there before the Pyruvate can move forward into the Krebs' Cycle.

The problem is that in a Ketogenic Imbalance, or in a person who is consuming excess vegetable/seed oil fatty acids, the fats block the enzyme PDH (Pyruvate Dehydrogenase) that converts the Pyruvate into Acetyl-CoA for entry into the Krebs' Cycle --- to be prepared for the Electron Transport Chain. If the Pyruvate is blocked, where does it go? There are many possible directions, and all of them create metabolic misery …

Some put major stress on the liver and kidney; some are pushed into what is called pancreatic "Pyruvate Cycling," which leads to Insulin Resistance; some push the Pyruvate into fat cells to be converted into more fat; excess Insulin production is provoked; and some of the Pyruvate is even directed toward the brain, particularly astrocytes, in a desperate attempt to provide the brain with the Glucose energetics it needs.

[All these mechanisms underlying Glucogenic & Ketogenic Imbalances are described and illustrated in some detail in your April Letter --- and are explained in

more detail than you ever need to know in Chapter 14 of your NUTRI-SPEC Manual, An Analytical System of Clinical Nutrition.]

Last month's Letter featured OEA in your Energetics G for its ability to mobilize and burn fat --- (but we could just as easily have featured Carnitine, Ornithine, or any of the other nutrients). By what mechanism does Energetics K increase efficiency of lipid metabolism and facilitate weight loss? ----- Consider Lipoic Acid and Niacinamide.

Lipoic Acid can be considered the ultimate supplement for metabolic efficiency. It is both an Adaptogen and a Rejuvenin. That is why you find it prominently in Adapto Max, Oxy Max, and Rejuvenator (and of course, in Activator).

Some of the most highly toxic products of lipid peroxidation exhibit their toxicity by inhibiting enzymes --- and that includes enzymes such as PDH mentioned above, required to maintain the balance between glucose and fatty acid energetics. The Lipoic Acid sulfhydryl group blocks the toxic effects of Free Radical Oxidation, maintaining mitochondrial enzyme action.

Lipoic Acid increases intracellular Coenzyme Q10, which is critical to the Electron Transport Chain.

Lipoic Acid is shown in clinical studies to decrease elevated triglycerides by as much as 45%.

Lipoic Acid is a cofactor of other energetics enzymes in addition to mitochondrial dehydrogenase complexes (PDH). It also activates lipid kinases, tyrosine kinases, and serine/threonine kinases, thereby increasing the efficiency of glucose uptake, and supporting balanced glucose–fatty acid energetics.

Lipoic Acid has been called "Pyruvate Oxidation Factor," and as an α-keto-acid dehydrogenation coenzyme, it is the link between lipid and carbohydrate metabolism.

Type 2 Diabetics have elevated fasting blood levels of Lactate and Pyruvate. Interestingly, the increased Lactate and Pyruvate double after glucose loading in obese Type 2 Diabetics, but not in lean Diabetics. Lipoic Acid decreases the excess of Lactate and Pyruvate and the elevation in blood sugar after Glucose loading. This shows that the need for Lipoic Acid is directly proportional to the degree to which a person is overweight.

Now consider Niacinamide/Nicotinamide/NAM, absolutely essential to restoring the liver-adipose axis to prevent excess fat buildup in both the liver and fat cells.

Chengting Luo, et al. Nicotinamide Reprograms Adipose Cellular Metabolism and Increases Mitochondrial Biogenesis to Ameliorate Obesity. J Nutr Biochem. 2022 Sep:107:109056. https://pubmed.ncbi.nlm.nih.gov/35609856/

Using a mouse model of human obesity, this study demonstrates remarkable effects of Niacinamide supplementation on decreasing obesity, increasing lean body mass (in the absence of any exercise regimen), improving Insulin Resistance and glycemic control, and restoring liver function in test animals with compromised liver function from a high-fat diet.

Mice were divided into 3 groups: a control group on normal chow and no NAM supplementation; a group on normal chow with NAM supplementation; a group made obese with a high-fat diet and then supplemented with NAM. After only 3 weeks of NAM supplementation at a human equivalent dose of less than 200 mg/day:

- Fat mass in the obese group was decreased by 47%, while lean mass increased by 40%. [You need to place 3 exclamation marks (!!!) in your brain after reading that statement again.]

- The test group on normal chow but with NAM supplementation also showed a significant decrease in fat mass. Mass of subcutaneous adipose tissue, abdominal adipose tissue, brown adipose tissue, and liver fat mass were all decreased, even in these normal, healthy mice. (That is to say --- not all your Glucogenic & Ketogenic patients are obese --- just as not all the 35% of your patients developing a fatty liver are obese. Energetics G & K have broad application among your patients.)

- NAM lowered the glucose level of obese mice at each time point in a glucose tolerance test. The hair color of the obese mice returned to the black, glossy quality of the control group, with less oil on the surface that resulted from a high-fat diet.

- The decreased fat mass and improved lean mass were achieved even though the food intake between the obese group and the control group was the same.

- In the obese group, NAM supplementation decreased LDH and Uric Acid.

- The NAM treatment did not alter the quantity of Glycolysis Energetics. Rather, it increased the NAD+ (--- the OXPHOS "energizer") directly, and increased NAMPT, the enzyme that enhances NAD+ synthesis.

- NAM supplementation increased Carnitine synthesis in the obese mice, thus facilitating β-oxidation of fatty acids.

- NAM dramatically increased proteins associated with mitochondrial OXPHOS, with the Electron Transport Chain fed by both fatty acid β-oxidation and Krebs Cycle glucose oxidation. NAM increased O2 consumption and CO2 production.

- Amazingly, the NAD+ in high-fat diet obese mice was increased 32-fold.

In summary, NAM reprograms adipose cellular metabolism, enhancing adipose mitochondrial functions to ameliorate symptoms associated with obesity.

This study also confirms that the NAD+ levels in adipose tissue, liver, brain, and muscle is much lower in obese subjects, and that the mechanism by which exercise and calorie restriction benefit metabolic disorders is by increasing NAD+.

Tubby Tummy fade away? ----- The patient needs Energetics K.

Hips expanding far too wide? ------ Get Energetics G on her side.

Special for June ----- 1 FREE for every 5 you buy
Energetics G or Energetics K.