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THE NUTRI-SPEC LETTER

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From:
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Dear Doctor,

In the last several issues of this Letter we have assured that …

YOU HAVE THE POWER TO ADD DECADES OF GOOD LIVING TO YOUR DIABETIC PATIENTS.

You now understand the causes and the effects of both Type I and Type II diabetes. Applying NUTRI-SPEC principles in the care of your diabetic patients, you will offer them therapeutic specificity they can find nowhere else. You will give all your diabetic patients precisely the supplements and diet needed, not only to bring sugar under control, but to begin reversing all the sequelae to their diabetic condition, including:

- high cholesterol
- high triglycerides
- cardiovascular disease
- kidney disease
- neuropathy
- obesity
- elevated blood pressure
- retinopathy, and ultimately blindness
- leg ulcers, and ultimately amputation
- premature aging
- premature death

furthermore, you understand that …

FRUCTOSE …

is the evil villain destroying the health of your diabetic patients. Fructose causes Type II diabetes, while it initiates the pathological changes in your Type I diabetics.

Our several month discussion of diabetes fits into a larger clinical picture you must understand in order to provide the best nutrition care for your patients. That larger picture, that includes an understanding of each individual's status with respect to fructose intake, to insulin and glucagon production, and to insulin sensitivity, we can call …

GLYCEMIC CONTROL.

Considered in NUTRI-SPEC terms, responsibility for glycemic control falls mainly in the province of your Glucogenic/Ketogenic and Sympathetic/Parasympathetic metabolic balance systems. However, Anaerobic/Dysaerobic and Acid/Alkaline imbalances are frequently of major clinical importance as well. This month's Letter is devoted to further enhancing your power and your resolve to help your diabetic patients. It is most fundamentally essential that you understand how diabetes fits into your NUTRI-SPEC metabolic balance paradigm. Your NUTRI-SPEC key concept of …

BIOLOGICAL INDIVIDUALITY …

determines who will become diabetic, when, and how.

Suppose your patients John and Mary Doe sit down to a dinner of steak, salad with ranch dressing, and coffee. Later that evening they indulge in a little ice cream. The next morning they start their day off with coffee, orange juice, and corn flakes with milk. What exactly happens in John's and Mary's body chemistry over the 16 hours between the evening meal and mid morning the next day? No one can describe the biochemical changes that take place except you, a NUTRI-SPEC practitioner, and you must first know what metabolic types you are dealing with. Suppose John is ketogenic and anaerobic, while Mary has a glucogenic and acid tendency. Only you can appreciate that their biochemical reactions will be entirely individualized. So, let us look closely at how glycemic control is maintained, and how it breaks down in various states of metabolic imbalance.

In response to any meal, there are seven factors that determine an individual's ability to maintain glycemic control:

1. the <u>metabolic imbalances</u> the person has
2. the relative <u>proportions</u> of protein, carbohydrate, and fat in the meal
3. the amount of <u>fructose</u> in the meal

4. the glycemic index of the carbs in the meal
5. the amount of glucagon produced in response to the meal
6. the amount of insulin produced in response to the meal
7. the sensitivity of the patient to the insulin produced

Let us look closer. You know all about the importance of evaluating patients per their metabolic imbalances. You understand the NUTRI-SPEC Fundamental Diet, and how to modify that diet for each metabolic imbalance such that each patient has the proper proportions of protein, carbohydrate, and fat in the diet. After reading the last few issues of this Letter, you understand the evils of fructose, the most devastating sugar. Now, consider what is meant by a carbohydrate food's glycemic index. Carbs with a high glycemic index are those that do two things. They are fast sugars --- resulting in a sudden rush of sugar into the blood stream; also, the high glycemic index carbs cause an exaggerated insulin response. [Additional note: A high glycemic index meal, or a high carbohydrate diet in general, decreases the access to stored fat for energy.]

Glucagon is a hormone produced by the pancreas, mainly in response to protein in the diet. Glucagon causes the release of liver glycogen into the blood stream, thus increasing blood sugar. A secondary effect from the glucagon in response to adequate dietary protein is to keep under control delta 5 desaturase enzyme, thus decreasing the production of pro-inflammatory prostaglandins from arachidonic acid. Glucagon also decreases any tendency to over-synthesis of cholesterol in the liver.

Insulin is the pancreatic hormone released into the blood stream primarily in response to dietary carbohydrate, but also to a certain extent in response to a high protein meal. Insulin decreases the concentration of sugar and protein in the blood, causing sugar and protein to be stored as fat. It inhibits the release of stored fat. Insulin also inhibits the release of glycogen from the liver, and, increases liver synthesis of cholesterol. As explained in a recent NUTRI-SPEC Letter, high insulin also increases liver synthesis of triglycerides from carbohydrate. These effects of insulin are particularly pronounced in the presence of anaerobic, glucogenic, ketogenic, and parasympathetic imbalances, and, they are exaggerated in anyone after a high carbohydrate meal.

Insulin fits into the picture of glycemic control not just by its short-term effect in response to a meal, but also through the phenomenon known as insulin resistance. In patients whose diets habitually stimulate the production of excess insulin, there will come a time when that person becomes refractory to the effects of insulin. In those cases,

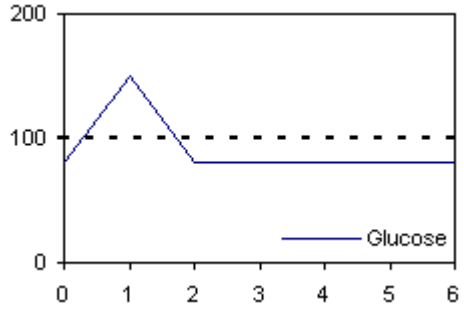
the insulin is still produced in excess, but, unable to complete its job of pushing sugar into the cells, circulates at high levels in the blood, sometimes for many hours. Now, here is perhaps the most important point you need to take from this month's Letter:

IN TYPE II DIABETICS THE CONTINUALLY HIGH INSULIN DOES DAMAGE AT LEAST EQUAL TO THAT DONE BY THE ELEVATED GLUCOSE.

SIMILARLY, IN TYPE I DIABETICS ON INSULIN, THERE CAN BE INSULIN RESISTANCE SUCH THAT THE PATIENT INJECTS OUTRAGEOUS QUANTITIES OF INSULIN TO CONTROL THE SUGAR, AND AGAIN, THE DAMAGE FROM THE HIGH INSULIN IS AT LEAST EQUAL TO THAT CAUSED BY THE ELEVATED GLUCOSE.

I find it appalling that such a high percentage of medical physicians treat both Type I and Type II diabetic patients without ever even considering that insulin should be monitored just as closely as serum glucose.

Most physicians treat their diabetic patients without even bothering to do a 5-hour Glucose Tolerance Test (GTT). But even the rare doctor who considers a GTT on a diabetic patient is being negligent because insulin levels in the 5 hours after ingesting a glucose solution are more significant than the blood sugar levels. To responsibly treat both Type I and Type II diabetics, a doctor (who does not have the advantage of NUTRI-SPEC Testing) must perform a 5-hour Glucose-Insulin Tolerance Test (GITT). To understand the essentiality of a GITT, first consider a normal GTT, illustrated by the curve graphed below:



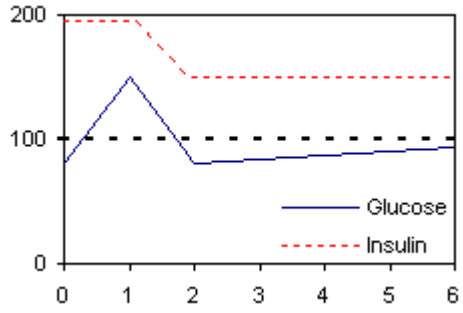
Having fasted for at least 8 hours, the patient has a fasting blood sugar of 80 at time 0, when he drinks the glucose solution. Within an hour his serum glucose peaks in the mid one hundreds, then falls to

100 or less by the end of the second hour. The blood sugar then remains stable at around 80 through the end of the test, after hour 5.

The problem with this normal GTT curve is that it is the curve shown by so many abnormal people. Many Type I diabetics on insulin will often show a GTT curve remarkably close to normal, even as they go blind and submit to amputation of a gangrenous leg. Many Type II diabetics have a perfectly normal GTT curve accompanying their blood pressure of 165/105 and their triglycerides over 300. (Many HYPOGLYCEMICS are diagnosed as merely neurotic after showing a normal GTT curve, even as performing the test causes them to experience jitteriness, heart palpitations, then a panic attack --- more on hypoglycemia later.)

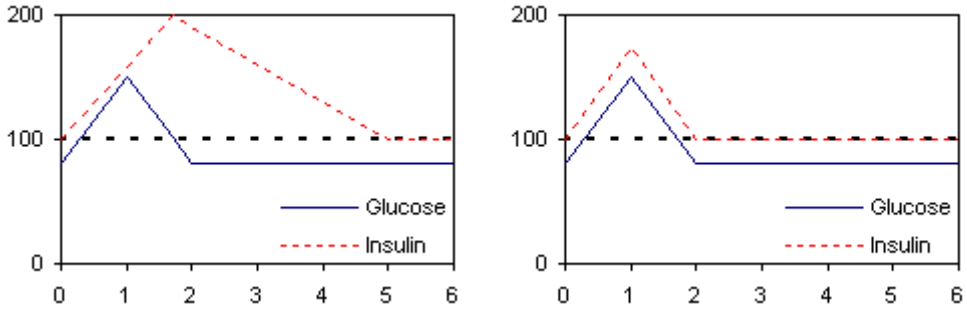
How can people so extremely abnormal in their symptoms associated with poor glycemic control have normal GTT curves? The answer is in their pathologically high insulin levels. Analysis of the GITT curves below tells all. (For illustration purposes, relative insulin levels are superimposed over the Glucose curves. The insulin is shown without units, with the line representing Glucose of 100 also representing normal fasting Insulin.)

Here is a graph of GITT for our blind, peg-legged Type I diabetic who has found that if she injects grotesque quantities of insulin twice daily, sugar can, on some days, be maintained at normal levels --- keeping her physician quite pleased:



There is enough insulin in this poor woman's blood to, on a "good" day, keep her sugar stable, which is enough insulin to assure that all her days are deadly bad. --- And, oh --- that smug physician has advised this slowly dying patient that she can freely eat fruit and juice, and that even cookies, cakes, pie, candy, and ice cream are OK in moderation as long as the sugar is reasonably well controlled --- but --- she is (of course) to carefully follow a low fat, low cholesterol diet.

Here is the GITT for our rolly-polly, pizza and Pepsi loving Type II diabetic (next to a normal GITT):



This Type II diabetic GITT curve might occur after a few days of avoiding sugar because the patient knew he was going to be tested. After his next pizza and Pepsi day, however, the Glucose curve could be about 25 higher before and after his typical meal.

In preparation for next month's Letter, here is a QUIZ. Consider your patients John and Mary Doe, and their meals as described on page 2. You will receive a FREE bottle of OXY B with your next order if you can match meals a,b,c,&d with GITTs 1,2,3,4. a = John's dinner b = Mary's dinner c = John's breakfast d = Mary's breakfast