

NUTRI-SPEC



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THE NUTRI-SPEC LETTER

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From:
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Dear Doctor,

TRIUMPHANT TRUMPETS! FANFARE! VICTORY PARADE!

Which of your patients have you striking up the band in a celebration of glorious success? Over which of your NUTRI-SPEC successes do you feel the most satisfaction? Which of your NUTRI-SPEC patients show the most gratitude? For many NUTRI-SPEC practitioners some of their greatest joy, and some of their most enthusiastic praise comes from their ...

DIABETIC PATIENTS.

Why is helping a diabetic patient such a big deal? The gravity of their pathology is all too apparent to most diabetics. Most diabetics are well aware that:

- Diabetic retinopathy may cause them to go blind.
- Gangrene in leg ulcers may result in amputation.
- Diabetic neuropathy can cause bizarre and painful symptoms.
- Their only consolation is that the blindness, the amputation, and the neuropathy may be avoided because they are so likely to die of cardiovascular disease before the other three tragedies strike.

Yes, diabetics have a rosy future, indeed.

You, as a NUTRI-SPEC practitioner, have the power to save these patients from certain suffering and premature death. Some of you have been routinely exercising this power for years, revered by your diabetic patients for your heroism. You have reduced, or in some cases even eliminated, the insulin requirement of many insulin dependent diabetics. You have routinely eliminated the oral diabetic medications for your Type II diabetics. Some of you have witnessed the “miracle” of curing diabetic neuropathy. Many of your diabetic patients simply can no longer be labeled as diabetics. Many more of those patients, though still diabetic, thanks to you, have enjoyed added years of quality life they never dared hoped for --- no blindness, no leg ulcers, no amputations, and certainly a dramatic slowing of cardiovascular disease. For those of you who have not yet enjoyed the victory celebration with your diabetic patients, you will have, from the moment you finish reading this Letter, the power to enrich the lives of diabetics like no other doctor can.

Diabetes --- hyperglycemia --- where does the high blood sugar come from and why has your patient lost control of it? A person can become diabetic by two mechanisms that are unrelated, and even opposite in their physiology. Yet, once people have become diabetic, the treatment regimen required is very similar, regardless of which mechanism is the cause. What are these two mechanisms by which people become diabetic?

INSULIN INSUFFICIENCY and INSULIN RESISTENCE.

Here are the two conditions lined up side-by-side:

Insulin Insufficiency	Insulin Resistance
Type I Diabetes	Type II Diabetes
Juvenile Onset Diabetes	Adult Onset Diabetes
Insulin Dependent Diabetes	Non-Insulin Dependent Diabetes
(relatively) thin ankles and wrists	abdominal obesity
Triglycerides high, medium, or low	Triglycerides high
Metabolic Keto-Acidosis	Alkalosis, despite ketones
Sympathetic Imbalance	Ketogenic Imbalance

How are each of these two unrelated mechanisms initiated? In Type I diabetes, the functional capacity of the endocrine pancreas to produce insulin is at least partially lost. What destroys the endocrine pancreas? It appears that there is an auto-immune component to Juvenile Diabetes that combines with a genetic tendency. Several decades of observation indicate to me that the genetic component involves a tendency toward a

Sympathetic metabolic imbalance. Sympathetic types then, are those who are susceptible to auto-immune destruction of the Islets of Langerhans. And what triggers the auto-immune reaction? Though I have never seen any specific research on this, I can imagine several likely candidates, including (since the common denominator of all auto-immune diseases seems to be oxidative damage in the mitochondria) an excessive indulgence in dietary sugar, dietary polyunsaturated oils, and perhaps viral or bacterial infection.

Type I diabetes has one additional interesting aspect to its etiology --- that its onset often follows trauma. I have always thought that a better name than Juvenile Onset Diabetes would be Sudden Onset Diabetes. I remember reading many, many years ago what I think was Osteopathic literature that referred to the pancreas as the “shock organ” of the body. The clear relationship between trauma and the onset of diabetes was established, but without proposing any means by which the pancreas was vulnerable to sudden shock.

I have seen this trauma-induced component of Type I diabetes confirmed in my own practice in many instances. For example, a man whose diabetes began at age 13 when, as a frail and somewhat effeminate boy he was beaten up by bullies in Phys Ed class. New and bizarre symptoms began immediately, and he was diagnosed within two weeks as diabetic. In another case, a 5 year old girl was engaged in a tug of war with her brother who let go, causing her to fall backwards into the TV with enough force to smash it. She immediately became diabetic. In another case, a young man was responsible for the accidental death of two people, immediately after which he became diabetic. These cases of sudden onset diabetes occurred in people who had skinny ankles and wrists, and in whom I can imagined a preexisting sympathetic tendency. The key here is that the pancreas has been unexpectedly zapped --- such that the patient simply cannot produce enough insulin on demand.

The mechanism behind Type II diabetes involves no zap, as its onset is gradual --- over many years in which the patient just begs for trouble. The many causative factors include: ice-cream, sweet drinks (including juice), candy, cookies, pie, cake, honey or syrup, as well as ice-cream and sweet drinks (including juice). Your Type II diabetics are sugar babies, plain and simple. They have whipped their pancreas mercilessly, provoking it to produce all too much insulin all too often. Many of these patients (who typically show a Ketogenic imbalance on NUTRI-SPEC testing) had at one time a parasympathetic tendency. Parasympathetic types are predisposed to the production of super-physiological quantities of insulin --- which explains their ability to both build muscle and adipose tissue. Their naturally high insulin production is associated

with their genetically determined ideal diet --- largely protein and fat, with very little exposure to carbohydrate, and certainly to sugar.

When these meat eaters become sugar eaters, the pancreas gets revved up out of control. After years and years of such overstimulation, the individual becomes refractory to the action of insulin. Also, insulin production becomes disconnected from insulin demand. These two factors combined add up to the slow but sure development of insulin resistance. The insulin is there in the blood, along with the high sugar, but the insulin does not do its job of carrying the glucose into the cells. By the way, many of the pathologies associated with Type II diabetes are caused as much by the high insulin as they are by the high sugar. By the time these patients present in your office with Type II diabetes, they generally show abdominal obesity and elevated triglycerides along with the Ketogenic NUTRI-SPEC test pattern.

[Side note: There is a third mechanism by which people can become diabetic. It involves the excess production of glucagon, the other pancreatic hormone. Glucagon, contrary to insulin, mobilizes sugar, thus raising blood glucose. There are parts of Europe where a common treatment for diabetes is to surgically remove a portion of the pancreas that produces glucagon --- very effective in many cases. Where this third mechanism fits into your NUTRI-SPEC system, I am still not certain, despite looking at the matter for many years. If and when I ever reach even tentative conclusions on how we can deal with this glucagon mechanism clinically, I will let you know.]

Suppose tomorrow you have a new patient who is diabetic. You know you have the power to turn this person's life around. What is your first step? After taking a thorough history, you will, of course, do your NUTRI-SPEC testing. You will likely find an Electrolyte Stress imbalance; if so, treat it according to your QRG analysis. You may find an Anaerobic or Dysaerobic imbalance; if so, treat that according to your QRG as well. Your main focus, however, is to determine whether this patient is sympathetic or ketogenic. Whether you are dealing with a sympathetic or a ketogenic diabetic is nearly always readily apparent from your NUTRI-SPEC testing, but, since most of these patients are on medication, the picture is not always that clear. Here are the rules to follow in making the distinction between a ketogenic and sympathetic diabetic:

If the patient tests ketogenic, then treat ketogenic.

If the patient tests sympathetic, then treat sympathetic.

For patients who show neither a ketogenic nor a sympathetic test pattern: If the patient is on insulin and tests parasympathetic, then treat as sympathetic --- but --- consider the likelihood that this patient is getting too much insulin. Often these patients are “running low” too often, and should be cutting back a bit on their dosage of insulin. Doing so should be quite easy once the NUTRI-SPEC regimen is begun.

If the patient is taking insulin but tests glucogenic, then treat with Complex S, but accompanied by the glucogenic (not the sympathetic) dietary recommendations. These patients tend to respond very quickly to Complex S accompanied by a very low carbohydrate diet.

If the patient is on insulin but tests neither sympathetic nor parasympathetic nor ketogenic nor glucogenic, then treat the patient as sympathetic unless the diabetes came on after age 35, and the patient has chubby ankles (--- be careful to distinguish between chubb and edema.). In the case of later onset and chubby ankles, treat as Ketogenic.

If the patient is on oral diabetic medication and tests glucogenic, then treat with Complex S, plus the glucogenic (not sympathetic) dietary recommendations.

If the patient is on oral diabetic medications and tests parasympathetic, then treat with Oxy K plus the parasympathetic dietary recommendations.

If the patient is on oral diabetic medications and tests neither ketogenic nor glucogenic nor sympathetic nor parasympathetic, then treat as Ketogenic.

There will be an occasional diabetic patient with an Electrolyte Stress imbalance whom you will supplement with Complex S (from the ES page of your QRG analysis), and Oxy K (from the Ketogenic page of your QRG analysis).

You must also supplement all diabetic patients with Oxy Power, 2 twice daily, in addition to whatever other Oxy Power may be indicated by your QRG analysis. This is because of the oxidative damage (glycation) that is rampant in the diabetic condition.

Those are the rules for adding measurably to the length of life and immeasurably to the quality of life in your diabetic patients. The only obstacles to phenomenal success are: failure to get patient compliance on diet, and, interference from medications that may no longer be appropriate. Regarding dietary compliance, you will find that most

patients have been already thoroughly indoctrinated in the understanding that food choices are important in their condition. The problem you may have is convincing these patients to ignore the diets recommended by the various diabetic associations and medical associations. These inane diets are incredibly high in sugars, particularly fruit sugar (fructose), the most devastating poison to diabetics. (If you know any diabetics who have ever stayed in the hospital, ask them about the "special diabetic diet" they received. Absurd --- dangerously absurd.) The NUTRI-SPEC Fundamental Diet accompanied by either the sympathetic, ketogenic, or glucogenic dietary recommendations will, in itself, often bring patients' blood sugars tumbling down --- even before adding in the beneficial effects of your NUTRI-SPEC supplementation.

Managing a diabetic patient on medications can keep you on your toes. For patients who are on insulin, you must be ever vigilant that they will go low. You must alert them to that possibility on day one. Often within days of following your dietary recommendations and taking Complex S the sugar, on their current dose of insulin, can plunge --- sometimes dangerously low. This potential plunge in sugar is particularly evident in Type II diabetics who were inappropriately put on insulin.

I once asked an acquaintance internist why he prescribed insulin for Type II diabetics even though their insulin was already high and doing damage at least equal that done by the elevated glucose. He paused for a moment and then replied, "Well, we have to do SOMETHING to get the sugar down." The idea seems to be that even though the body cannot respond efficiently to insulin, if you load the patient with gross amounts of it, you will at least get some response. That the patient's length and quality of life may actually decrease is not considered. Please understand that your patients must be prepared to alter their insulin intake virtually immediately upon your NUTRI-SPEC regimen.

More on your diabetic patients next month --- but in the meantime begin helping these people. They will be some of your most satisfying success stories.

Sincerely,

Guy Schenker, D.C.

P.S.: Special this month --- 2 OXY K and 2 Complex S **FREE** with every 10 you buy.