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DEVELOPING DIABETES

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Diabetes mellitus is the fourth leading cause of death in the United States. The number of diabetics has risen almost 50% since 1983 and tripled since 1958. There is now an average of 798,000 new cases each year. In 1997 an estimated 15.7 million Americans had diabetes (a third undiagnosed), nearly 6% of the population. From 90 to 95% of all cases are classed as non-insulin-dependent diabetes mellitus (NIDDM). Childhood-onset NIDDM has also sharply increased – 92% of the children are obese -- particularly in the 10- to 19-year age group.

Recent guidelines from the American Diabetes Association lowered the blood sugar threshold for diagnosis of diabetes from 140 milligrams glucose per deciliter of blood to 126. They recommend that all adults over age 45 be tested every three years, more frequently if there is any "red flag" such as family history or existence of high blood pressure. These guidelines will cause the number of registered diabetics to soar even more. [i]

DEFINITIONS

Diabetes mellitus is classed as either insulin-dependent diabetes mellitus – IDDM – (what used to be called type 1 diabetes mellitus) or NIDDM (what used to be called type 2 diabetes mellitus). IDDM can include juvenile-onset diabetes, but refers primarily to the treatment of the disease by insulin. NIDDM is no longer called adult-onset diabetes; the term refers to the management of the disorder through diet, exercise, and other lifestyle changes. Either way, diabetes is a multitude of metabolic imbalances, not a single disease, a collection of problems having an abnormal glucose/insulin relationship. There is disruption in the normally automatic regulation of blood sugar (glucose) metabolism – the conversion of glucose into fuel to power the body's cells. Insulin normally lowers blood sugar by inhibiting glucose production in the liver and by stimulating glucose use in skeletal muscle. Both actions are impaired in people with NIDDM.

IDDM relates to the group of problems associated with the **failure of the** *beta cells in the Isle of Langehans of the* **pancreas** to produce sufficient insulin on demand. Symptoms become severe and the person knows there is a problem. Secondary complications such as cardiovascular disease, kidney disease, gangrene, blindness, capillary disease, various neuropathies (nerve diseases) are more frequent than in NIDDM. And eventually (especially if insulin drugs are used) the pancreas stops producing insulin altogether.

IDDM is believed to be caused by a combination of genetic and environmental factors. An environmental insult is thought to initiate the process in susceptible individuals Among the environmental factors are aspects of the infant diet (pasteurized cow's milk, processed solid foods, etc., rather than exclusively breast milk). Later, "children's diet is greatly influenced by the diet of the parents." Consuming highly refined, processed, chemical-laden foods from an early age can create complications that result in pancreatic failure. Chemical poisons, "foul nutrition," and other toxic exposures may play a role. Viewed as an "autoimmune disease," IDDM can result from the inability of the immune system and endocrine system to properly protect and preserve the function of pancreatic, hepatic (liver), muscle, or adipose (fat) cells and tissues. Yet increasing evidence shows that IDDM may be prevented or halted if approached before total destruction of the pancreatic *beta cells* occurs.

NIDDM refers to a group of problems that develop in response to abnormalities in the **target tissues for glucose** – liver, muscle, or adipose tissue. The body's cells fail to properly respond to insulin. To compensate, the pancreas makes more insulin, but it remains ineffective so glucose accumulates in the blood. Or, due to a diet high in refined sugars, altered fats, and other denatured non-foods, the pancreas, liver, and other tissues are overwrought by attempting to deal with this persistent "foreign" onslaught. Eventually the overworked, exhausted pancreas may slow or stop functioning properly. Although the pancreas is key in governing proper glucose balance, other organs are involved. The liver, for example, stores sugar to be released as needed, and processes it again for use by other cells. The liver can transfer excess sugar into fat and store it in adipose tissue. These and other processes are complex and require many nutrients. The adrenal glands signal the release of glucose so body cells will be able to handle stress. Other tissues and organs can be cited, but the issue is that diabetes is a systemic (whole body) problem.

The incidence and prevalence of NIDDM in the world fluctuates widely. Its development is

tremendously influenced by "food abundance and economic status of the population." It occurs as a country advances technologically, bringing less physical activity and a more refined, processed, altered, denatured, nutrient-depleted food supply. Though there is likely a genetic influence, NIDDM is more responsive to environmental influences, lifestyle choices. Under-activity and overweight (or obesity) predominantly precede or accompany most cases of abnormal glucose tolerance. NIDDM is believed to develop in response to a variety of abnormalities in the structure of insulin, its transport to target tissue, target tissue receptor abnormalities, and abnormalities in one or more metabolic (processing) pathways needed to effectively use glucose. People with NIDDM are often unaware that there is a problem because there are no noticeable symptoms for many years as it develops and worsens. Studying NIDDM patients is only of limited help in understanding the cause because, once diabetes has developed, "the many coexisting metabolic disturbances make it impossible to distinguish primary cause from secondary effects." [ii]

SYNDROME X and INSULIN RESISTANCE

In 1988 Dr. Gerald Reaven, Stanford University, coined the term "**syndrome x**" to define a group of symptoms that "appeared to occur secondarily to cellular resistance to insulin-mediated glucose uptake." The theory is that excessive secretion of insulin may occur when normal insulin action is impaired. People with this "**insulin resistance**" – insulin insensitivity – compensate by secreting large amounts of insulin and are at increased risk for a number of health problems. The problems are clustered together as syndrome x and include glucose intolerance (or resistance), excess insulin secretion, obesity (particularly upper body), disturbance in blood fats (low HDL cholesterol, high triglycerides), high blood pressure. All these abnormalities may not be seen in an individual in order for the syndrome to be diagnosed. Insulin resistance and excess insulin are thought to directly contribute to the development of NIDDM, hypertension, and various vascular diseases such as coronary heart disease.

Insulin resistance means that receptors on cell walls have become resistant to insulin. The cells do not allow glucose to pass inside for energy production or storage. Insulin loses its effectiveness and has trouble clearing glucose out of the bloodstream. The pancreas reacts to what seems to be a lack of sufficient insulin, so churns out more. At first, the extra insulin forces blood sugar levels back down to normal. But eventually the pancreas may lose the battle to supply enough insulin. Then blood glucose levels get out of control and diabetes exists. Insulin resistance is diagnosed in many people with metabolic disorders: 65.9% in people with impaired glucose tolerance, 83.9% in those with NIDDM, 53.5% in people with hypertension, most all people with high blood triglyderides and low HDL cholesterol.

NIDDM is associated with obesity; from 50 to 80% of new cases each year are in people weighing too much. Yet many overweight people do not display insulin resistance and many normal weight people do. Not all people diagnosed with insulin resistance have high blood pressure and not all individuals with high blood pressure have insulin resistance. The incongruity goes on. It must be asked if the link between insulin resistance and the listed problems is "causal, consequential, or coincidental."

Insulin has many roles. Its primary function is to allow transport of glucose into cells and lower blood sugar levels. Insulin allows transport of amino acids into cells, essential for cell functions and survival. It stimulates glucose oxidation (energy from "burning" glucose), fatty acid synthesis, protein production, glycosaminoglycan (protein-carbohydrate complex) synthesis, RNA production. Insulin slows and balances the manufacture of glucose by cells, offsetting excessive breakdown of proteins. It prevents mobilization of fat from storage, increases glycogen (storage sugar) levels in muscles and the liver, activates formation of fats when needed. "Insulin is part of a constantly changing, dynamic system." Its actions require an abundance of nutrients including chromium, zinc, manganese, calcium, nucleic acids, and more. Are all its functions affected by "insulin resistance?" What of nutritional deficits or deviations?

"Professionals disagree about the causes of insulin resistance." NIDDM is thought to involve various degrees of pancreatic dysfunction and insulin resistance, "although the relative importance of these two factors is controversial." Obviously, much is not known and much is guesswork. Insulin resistance is "difficult to measure." The data is obtained by "surrogate measures" – fasting insulin concentration or insulin response to refined sugar. The rationale for the former is an assumption that the insulin response of the pancreas is undiminished and will rise to maintain normal blood sugar levels to a degree that is related to the degree of insulin resistance. Persons diagnosed with insulin resistance often have normal fasting blood sugar but severely elevated blood sugar after a "glucose load." In other words, the tests used to diagnose it include administering a huge dose of refined sugar. Could the test results be a reaction to this stressful, toxic insult? The "belief" that diabetes is preceded by insulin resistance "may be a self-

fulfilling prophesy because of the excessive emphasis put on the blood sugar level **after** refined sugar ingestion rather than the fasting state. Pervasive in the research is: "Sugars, refined carbohydrates and lots of hydrogenated fats may worsen or bring out insulin resistance." Thus, "uncertainty remains as to how a true measure of insulin resistance might perform in a prospective study." Really, it is insulin **sensitivity** – the opposite of insulin resistance – that is measured. And about 25% of apparently healthy people are considered insulin resistant. So, "shortcomings in our definitions of insulin resistance and the insulin-resistance syndrome limit effective use of these terms." Although sometimes treated as fact, "we need to be sure that the insulin resistance syndrome actually exists…" It is still theory.

The "fallacies" underlying the theory include: confusion between mechanism and cause, bias in studies with "dubiously selected controls" generally not randomly sampled, and publication bias. Negative reports are less likely to be referenced or published. Populations with the highest amounts of diabetes and high blood pressure often fail to be associated with insulin resistance markers and the syndrome is weak. Studies generally ignore the pancreatic/ portal vein/ liver relationship "because it is difficult to investigate." Enthusiasts ignore the question of whether infusion of various amounts of glucose into veins has any real physiological meaning. The question remains whether insulin resistance is confounded by nutritional and/or other environmental factors "as yet more difficult to measure as causes." It is easy to measure insulin levels but hard to honestly interpret results or meaning. If it is real, insulin resistance could be an effect rather than a cause. [iii]

AN ALTERNATE LOOK AT CAUSES

The problem of diabetes does not revolve entirely around the ability to produce or use insulin. Other organs and tissues – such as the pituitary, thyroid, adrenals, liver – all have effects on the prevention, onset, and progress of diabetes. Nutritional needs, stress levels, and toxic exposures must be considered. The body is an integrated, interrelated, complex system; any and all aspects may be involved.

Refined, processed, denatured, nutrient-devoid non-foods are likely candidates in diabetes causation. For over 40 years Dr. J. Yudkin has examined the role of insulin in various disease states. He discovered links between dietary indiscretions, especially heavy refined sugar ingestion and excess intake of altered and saturated fats, with diseases that showed high insulin levels and signs of insulin sensitivity. "Suffice it to say, the modern diet plays a significant role in determining the development of insulin resistance with hyperinsulinemia [elevated insulin levels]." In fact, "nutrition may be a better explanation for this than genetics." Americans consume massive amounts of refined sugars. The average intake is now 149.2 pounds a year, about three pounds a week, 41 teaspoons a day! Many health professionals consider diabetes a result of years of punishing the body with heavy doses of sugar. Populations consuming the highest amounts of sugar have the lowest body levels of chromium -- an essential component of the glucose tolerance factor that is needed for proper blood sugar metabolism.

Refined sugars, refined flours, or altered fats can result in elevated blood levels of triglycerides, common with insulin sensitivity. NIDDM need not develop unless "additional extreme environmental factors" such as an unbalanced, denatured diet or excess food intake are "super-imposed on genetic background." High intake of simple sugars and other nutrient-stripped, altered non-foods is "known to impair glucose metabolism," and "micronutrient functions." People who eat the most refined sugars, highly processed foods – devoid of nutrients needed to process the sugars or starches – and the least fiber have two and a half times as great a risk of developing NIDDM as those who eat little refined or processed foods and lots of whole, fiber-rich foods.

Dietitians insist that refined sugars do not cause diabetes; that any starch or sugar in any carbohydrate can be disruptive. So they monitor amounts of daily carbohydrate intake. Fructose, a sugar found in fruits and vegetables, is now manufactured as a refined sweetener and added to soft drinks, bakery products, candy, etc. Claims are made that it does not harm diabetics, and may even improve their metabolic control. However, "too much" can elevate blood sugar, cholesterol, and triglycerides (as can any refined sugar) and long-term studies are lacking. Fructose – **if** consumed in fresh, whole fruits and vegetables – does not cause quick spikes of blood sugar levels as do refined sugars. But when extracted and processed – refined – it acts **exactly** like sucrose (white sugar), maybe worse.

Artificial sweeteners are also implicated in blood sugar disruptions. Over half the adult population currently consumes aspartame (NutraSweet, Equal). It can cause, aggravate, or accelerate a multitude of problems including diabetes and its complications, overweight, and a craving for refined carbohydrates. Caffeine can increase blood glucose levels. Two to four hours after ingestion, the blood sugar rises and then may

responsively fall (hypoglycemia). One possible reason is that caffeine can inhibit muscle glucose uptake even in the presence of insulin.

Food sensitivities or allergies, addiction to non-foods or chemicals, and nutritional deficits may contribute to diabetes. Some studies show that moderate intake of alcohol improves the ability to utilize insulin (post-glucose insulin concentrations). Other studies show alcohol may be related to the development of NIDDM and may produce low and high blood sugar fluctuations. Alcohol is a refined sugar. Years ago it was made naturally with associated nutrients left intact (including enzyme-rich yeast, trace minerals, B vitamins). Today's processing methods include pesticides on raw materials, additives for shelf life and clarity, and removal of natural live constituents and nutrients needed for sugar utilization.

Infants fed cow's-milk-based formula during the first three months of life have an increased risk of developing diabetes. Introduction of cow's milk products (pasteurized, homogenized) before the age of eight days more than doubles the risk of diabetes. Children exclusively breast-fed for at least the first two months and who receive cow's milk later (if at all), have much less chance of developing diabetes. Some studies examining family histories conclude that cow's milk is no problem. The conflicting results are attributed to different types of milk protein. Could the problem be pasteurization and homogenization which alter the protein, fats, sugars, and otherwise disrupt the natural complexities of milk? The Masaii people of Africa until recently consumed large amounts of cow's milk from early infancy – fresh, raw, unchanged – and had a very low incidence of diabetes. But mother's breast milk is the absolute best food for infants. Other altered foods including wheat and soy are also associated with later development of diseases such as diabetes. The delicate digestive and immune systems of babies are designed for breast milk, not altered, denatured, adulterated non-foods.

"Until the pasteurization of milk, the addition of chlorine to drinking water, and the bleaching of flour, diabetes was practically unknown." Chlorine and chloramines from chlorinated tap water have the power to induce diabetes. Chlorine chemicals make artificial sweeteners even more harmful. Flour bleaches (chlorine-based compounds used as preservatives) convert a food factor (xanthine) in wheat into *alloxan*, a very potent poison that destroys the insulin-producing cells in the pancreas. Nitrates, nitrites, and nitrosamines found in municipal water supplies significantly increase the risk of diabetes. Preserved meat and fish products, vegetables or fruits that have absorbed excess synthetic fertilizers, and other sources of nitrates add to the load, though up to 70% of total nitrate exposure may be from drinking water. The damaging effect of nitrates can be more pronounced in infants. Babies who are breast fed rather than given formula made with nitrate-contaminated tap water would have less risk of later developing diabetes.

Aluminum can deactivate enzymes necessary in blood sugar metabolism. Elevated aluminum has been identified in many diabetic patients. This toxic metal adversely affects enzymes involved in the breakdown of blood sugar. Tap water and canned soft drinks are two sources of exposure. Seepage of aluminum into canned drinks increases with their acidity. Magnesium is among the nutrients that help protect against aluminum absorption and promotes it excretion. Diabetics are notorious for magnesium deficits. Smoking boosts the risk of diabetes and worsens it if it already exists. The HIB (hemophilus) vaccine has been linked with the development of IDDM and other immunological disorders. There is a dose-response relation between cumulative arsenic exposure and diabetes. Arsenic may be present in soil, water, and air as a common environmental toxin and in commercially-produced foods primarily due to pesticide use. [iv]

DIET

Some experts advocate a low-protein, low-fat, high carbohydrate diet for prevention and treatment of diabetes. Others promote a higher protein, higher-fat (primarily monounsaturated [MUFA]), lower carbohydrate diet. Protein foods are said to place stress on kidneys, increase saturated fat intake, increase cholesterol levels. High fat foods are considered bad for weight control and the cardiovascular system. MUFAs are thought to benefit blood fats, but studies with "realistic" dietary changes show their effects are only modest. Consuming large amounts of carbohydrates is thought to result in increased secretion of insulin to dispose of this "load." Protein is bad, fat is bad, carbohydrate is bad. Apparently "nobody knows how to deal with it." Sadly, "much of our dietary advice is still without adequate research substantiation."

It is agreed, even by the American Diabetes Association, that there is no single "diabetic diet" good for everyone. Advice should "always be individualized." Proportions of protein, fat and carbohydrates depend on many specific, unique, complex considerations.

Mexicans living in Mexico consume more carbohydrates and have higher serum triglycerides than Mexicans living in Texas. Yet the Mexicans in Mexico are less obese, have 26%

less diabetes, and have lower serum cholesterol concentrations. Native Americans who adopt the typical modern refined diet suffer greater percentages of obesity and diabetes. Their traditional diets often contained large amounts of fiber-rich, nutrient-dense carbohydrates. Returning to their native diet results in weight loss and blood sugar balance. Years ago a high-fat, high-protein diet was used to control diabetes. It was rich in eggs, bacon, cream, and butter. Diets containing up to 70% of calories in fat had no effect on cholesterol. Three out of four diabetics were able to consume a high-fat diet without insulin. Diets high in proteins such as meats, seafood, milk products, nuts and seeds have been consumed by traditional societies with no resultant cardiovascular problems, kidney problems, or diabetes. Rather than viewing diets from a quantitative perspective (how much protein, fats, carbohydrates) emphasis should be on **form, quality,** and **individualized needs**.

In recent decades animals raised for food by commercial ranchers are fed unnatural, pesticide-laden diets, given hormones and drugs to offset deplorable conditions and to quicken growth. The meat definitely contains more fat, unhealthy fats, poisons, and other factors that adversely affect health. Poisons accumulate in the fats. Fatty acids are unbalanced -- for example, containing little if any omega-3 fatty acids. The meats are not fresh and come from unhealthy animals so do not provide the nutrients found in naturally-raised or wild animals.

Carbohydrates such as grains are usually refined. Low fiber (refined) foods and altered-fat foods affect blood sugar, blood fats, and insulin sensitivity. People who eat a refined, starchy diet low in fiber – and who drink a lot of soft drinks – have two and a half times the rate of diabetes than those who eat less refined carbohydrates, more fiber (as in whole-grains), and less or no soft drinks. Not only the type of food, but the **structure** (is it unaltered as Nature made it?) is important to blood glucose and insulin responses after a meal. For example, chromium-rich **whole** barley is more beneficial to blood sugar and fluid consumption in diabetics than white (refined) wheat bread and whole-meal wheat bread. The negative effect of processing 'is now well established" and the potential of using "intact cereals" (in whole form) rather than flour has been clearly shown. The "negative impact of milling of intact cereal kernels" may be due to the fact that whole grains, once ground, quickly lose valuable nutrients and become rancid. High complex carbohydrate, high-fiber diets (whole, natural foods) have been shown to improve blood sugar control and insulin sensitivity in diabetics.

"Attempts should be made to substitute unrefined (complex) carbohydrates for refined (simple) sugars." Professionals have observed patients completely reverse the effects of NIDDM by consuming "unprocessed and whole foods." High intake of refined sugars "has been shown to have deleterious effects on glucose metabolism." Ingestion of beverages such as soft drinks has increased 68% and fruit juice drinks by 24% since 1972. These drinks contain mainly high-fructose corn syrup and other refined sugars. Fructose decreases activity of superoxide dismutase (a zinc- and copper-containing enzyme), reduces activity of other protective enzymes, reduces liver stores of copper, and increases stores of iron. Many minerals have beneficial "insulin-like" activities including chromium, vanadium, zinc, copper, nickel. Their presence stimulates insulin binding, blood sugar "burning," processing of fats, and proper production of storage sugar. Deficits of these and other nutrients "could be responsible for impaired insulin functions." Modern farming methods strip soils and food processing methods strip foods of these essential nutrients.

The liver is essential to glucose homeostasis by controlling the balance between hepatic glucose production and glucose utilization by other tissues. It is the major organ that metabolizes and regulates fructose, other sugars, most other foods, alcohol, poisons, toxins, and a multitude of nutrients such as copper and iron. Refined foods deplete the body of nutrients required for liver function including B complex, manganese, calcium, and more. Liver disease is associated with abnormalities of glucose tolerance and insulin sensitivity. Liver damage induces storage of iron, leading to further liver breakdown. The ability of the pancreas to secrete insulin was impaired in rats fed a diet containing fructose and inadequate copper; there was also abnormal glucose tolerance. Many Americans (30%) take supplements of isolated, unnatural iron, while 60% take synthetic, isolated ascorbic acid (so-called "vitamin C"). Both supplements can disrupt copper levels in the body. Any synthetic, isolated supplement can lead to deficits of nutritional components protective to the liver, pancreas, blood vessel walls, and more.

Diabetic patients who followed a high-fiber, low-fat vegan diet (based on vegetables, fruit, grains, legumes) for three months experienced a fall in fasting blood sugars so significant that they were able to reduce their medications, lost more weight, and had a more substantial drop in cholesterol levels than the control group. The control group followed the American Diabetic Association diet. Was it the absence of animal products that brought the improvements? Or was it the elimination of refined foods and toxin-

containing foods? In the 1970s, people with diabetes who followed a diet consisting of 50 to 80% raw food were able to greatly reduce medication. When the diet was 90 to 100% raw, they were able to completely discontinue medication. There were no restrictions on fat, carbohydrates, or protein. Foods consumed basically the way Nature provides them are more bioavailable, more health promoting, and more protective and helpful against chronic diseases. Since 75% to 80% of people with NIDDM are obese, could the foods that cause diabetes also cause obesity? Worth considering. [v]

GLYCEMIC INDEX

The glycemic index (GI) was developed as a guide to measure how much a food affects a person's blood sugar. Although first intended for diabetics, it is applied to many other health conditions related to blood sugar regulation such as cardiovascular disease, hypertension, obesity, sports performance. Ranging in value from 1 to 150, the GI indicates how fast a high-carbohydrate food is digested into glucose and how much it causes the blood glucose to rise. The higher the GI, the greater the rise in blood sugar. Initially the reference (rated at 100) "food" was a refined sugar, glucose. Later, refined white bread was considered more "reliable." So GI charts may differ depending on the reference.

The concept is simple, but there are many complexities and flaws. The GI does not separate carbohydrates that are simple (sugars) or complex (starches). Many factors influence rating including the amount of fiber and fat – which lower the GI – how refined the food is, how fast the food is digested, whether it was cooked, what else is eaten with it. Refined sugar, refined honey, and most other refined carbohydrates (e.g. breakfast cereals, white bread, instant white rice, chips, crackers, sweets, etc.) have a high GI. But so do raisins, corn, potatoes, carrots, parsnips, turnips, etc. because their natural sugars are available for rapid digestion and absorption. Apples, apricots, peaches -- though sweet -- grapefruit, dairy products, nuts, and legumes have a low GI. Pasteurized dairy products contain poorly utilized altered milk sugar, lactose, so have little effect on blood sugar. When the enzyme lactase is added to milk, the rating goes up. Nuts contain fat which slows digestion and absorption of carbohydrates. Legumes contain soluble fiber that slows digestion and prevents a sharp rise in blood sugar. Grapenuts have a high GI because they are processed grains. Yet refined pita bread is densely packed so has a relatively low GI. Candy bars and ice cream often have a lower GI because of their fat content which slows the rate sugar enters the blood.

In other words, **only** the immediate effect on the blood glucose is considered, **not** whether the food is wholesome and healthy or refined and detrimental. Whole wheat bread ranks fairly high, higher than white bread, despite research showing grains "should be consumed in a minimally refined form to reduce the incidence of diabetes." Commercial whole wheat is finely milled, removing some bran and germ, and contains preservative bleaching chemicals that are toxic to the pancreas and used at four times the amount contained in white bread. Fiber prevents and helps diabetes. The overall quantity of fiber, higher in whole foods, affects glucose metabolism. Psyllium husks, fenugreek, marshmallow, and other mucilaginous herbs as well as guar gum and other fiber sources reduce blood sugar levels and insulin response. The **physical form** of foods affects the speed of sugar absorption. Intact, whole foods – regardless of how fast or slow the glucose shows up in the blood – do not cause powerful or stressful metabolic responses; they enhance blood sugar balance. The further a food gets from its natural state, the more difficult it is for the body to process. All foods should be consumed in the most unrefined, untouched form as possible for optimal biochemical response. People who frequently eat vegetables (both low and high on the GI) have an 80% lower risk of NIDDM. Fermented vegetables and breads (like real sourdough bread) have a beneficial affect on blood sugar levels. When vinegar is added to meals, blood sugar levels are lower. There are too many possibilities missed and ignored by the GI – it does not give a true picture of what the body eventually does with the food.

In tests for the GI, potatoes are usually peeled and overcooked. New potatoes rank lower because the peel is left on and they contain less easily digested starch than russet potatoes. Mashed new potatoes have a higher GI than intact ones because crushing the flesh creates more surface area for faster digestion. Adding a pat of butter or a little olive oil lowers the GI as would consuming the potato with a protein or raw vegetable salad. The **way** a food is prepared or eaten determines the reaction is elicits. Potato crisps have a lower GI (because of the fat) than boiled potatoes. But potato crisps are not healthful foods! Even the ripeness of fruit contributes to the immediate blood sugar effect. The total amount and **type** of carbohydrate (starches convert easily to glucose but refined sugar, due to the presence of fructose, takes longer), the **physical form** of carbohydrate (the surface area of bread is digested faster, resulting in a quicker blood sugar response than pasta), the amount and type of fat, the protein, the fiber,

cooking (the extent of starch breakdown from cooking can affect the glycemic response), and many other factors enter the picture.

Extensive variability occurs in glucose response for the same foods with different people: "within-patient variability" or biochemical individuality. Because elevated blood sugar slows digestion and absorption, the level prior to eating affects glycemic response to foods. The GI concept may not apply to a mixed meal containing a variety of foods. The amount of time between meals has an effect. The GI was determined using the first meal of the day which can elicit a different response to an identical meal ingested four hours later. Sipping refined sugar slowly over several hours produces a much smaller increase in blood glucose than rapid intake of the same amount. Eating three carrots may take 15 minutes, whereas consuming their juice may take only a minute with differing blood sugar effects. Very few studies show that the GI has any clinical usefulness in treating diabetes. Less evidence exists to support the claim that lower GI foods prevent diabetes or assist in weight loss.

Over 600 foods have been given a GI value, but there are "no widely practiced standards" for food preparation, cooking, storage (e.g., if foods are cooked and allowed to cool, the result is formation of "retrograde" [degenerated] starches) or presentation. The GI seems to help blood sugar regulation in some, but **any** protocol that reduces or eliminates most refined carbohydrates from the diet will do so. Whole foods are complete, containing many complexes of nutrients both known and unknown that work together. Figs contain concentrated natural sugars, but they also contain B vitamins, calcium, and other nutrients the body uses to process the sugar properly. Potatoes are an easily digested starch, but are sources of potassium, zinc, chromium, vitamin C complex, B vitamins and other factors that aid biochemical equilibrium. Measuring **one** substance in the blood after ingestion of a food reveals very little about the use, value, and ultimate effect of that food on the body. Regarding the GI, "accuracy is not great, and its overall utility remains to be determined." [vi]

NUTRITION

An individual with a tendency for or the existence of diabetes needs to eat a diet of natural foods as minimally processed and whole as possible. With professional assistance, an individualized diet should be formed. Foods such as burdock root, Jerusalem artichokes, and dandelion root may be included as all contain inulin, a natural substance that helps balance blood sugar and insulin levels.

Chromium and the glucose tolerance factor in which it serves a primary role are essential to the use of carbohydrates and fats. Chromium supports blood sugar regulation and aides the interaction of glucose, insulin, and glucagon. It improves the uptake of glucose to cells where it can be used to produce energy, helps decrease cholesterol and triglycerides. Consumption of refined sugars and flours depletes chromium reserves. Most Americans and virtually all diabetics are deficient. Deficiency increases risk for IDDM, NIDDM, and gestational diabetes. Chromium given to diabetics significantly reduces requirements for insulin or other blood-sugar-lowering drugs. The glucose tolerance factor in foods has been studied extensively but attempts to synthesize the whole complex have been unsuccessful. Some food factors that improve glucose tolerance and promote insulin balance remain unidentified. Chromium content alone is not a valid indicator of bioavailability or effectiveness.

The most common mineral deficiency in diabetes is **magnesium** which helps to stabilize glucose and helps insulin to be released, produced, and function. It is involved in the maintenance of pancreatic beta cells; deficiency leads to atrophy of these cells. There is a direct relationship between intracellular magnesium concentrations and total body glucose metabolism. This mineral functions in over 300 essential enzymatic reactions involved in the metabolism of proteins, carbohydrates and fats. It is critical to offset metabolic acidosis, common in diabetes. Low magnesium is associated with carbohydrate intolerance, insulin resistance, hypertension, retinopathy, cardiovascular disease, vascular complications, neuropathy, and kidney disease.

Abnormal regulation of intracellular **calcium** appears in diabetics. Altered calcium metabolism may impair secretion of insulin and contribute to complications of diabetes including cataracts, cardiovascular disease, and premature aging. An increase in intracellular calcium may be the final triggering mechanism whereby glucose or other stimuli signal the pancreas to release insulin. Elevated blood calcium, which can indicate an inability to transport calcium from the blood to the tissues, may cause an imbalance and alteration of some metabolic functions.

A deficit of **essential fatty acids** is a key to proper calcium distribution and utilization. Essential fatty acid metabolism is impaired in diabetics. For example, the enzyme needed to convert linoleic acid to gamma-linolenic acid (GLA) is inhibited. Sources of GLA (evening primrose oil, black currant seed oil, borage oil) help normalize fat metabolism and decrease the

amount of insulin required. Omega-3 fatty acids are often deficient; flaxseed oil, a rich source, lowers the amount of insulin required, lowers high serum triglycerides, and lowers high blood pressure. Eating a cup of ground flaxseed daily for 90 days dramatically improved blood sugar control in persons with NIDDM. Some prosta-glandins, products of unsaturated fatty acids, help insulin work more effectively, relax blood vessels, regulate calcium metabolism, improve nerve function. Fatty acids produced from refined sugars interfere with essential fatty acid function and increase the chance of developing diabetes. Hydrogenated fats, other *trans* fatty acids, and any refined oils – altered, void of nutrients, chemical-laden – rob the body of nutrients, lead to fatty degeneration, and contribute to the cause and complications of diabetes.

Persons with IDDM and NIDDM exhibit deficiencies in **zinc** levels. Zinc may exert an insulin-like effect, helping to control blood sugar levels, and evidently protects the pancreatic islet cells from destruction. Zinc increases cellular glucose assimilation and glucose effectiveness.

Vanadium, a trace mineral that lowers blood sugar by "mimicking" insulin and improving cells' sensitivity to insulin, affects many aspects of carbohydrate metabolism such as glucose transport, breakdown of glucose, glucose output, sugar-processing enzymes, decrease of storage sugar production and increase of deposition. Vanadium compounds, particularly vanadyl sulfate, have been used to improve fasting glucose and other diabetic measures. But fractionated, high-potency supplements have adverse effects. Organic complexes are better absorbed, require lower doses, and have no signs of toxicity. Long-term results include decreases in plasma glucose, triglyceride and cholesterol levels; an amelioration of some complications (vascular hyperactivity, cataract formation, kidney disease, hypertension, cardiomyopathy). The protective effects are improved with the addition of vitamin E; in foods it appears with E complex. Natural sources include seafood, liver, whole grains, mushrooms, parsley, corn, soy, dill.

Effects of **manganese** deficiency include reduced insulin activity, impaired glucose transport, and lowered insulin-stimulated glucose oxidation ("burning") and conversion to triglycerides in adipose cells. This trace mineral is needed for glucose balance. The average manganese content of whole blood in diabetics is about one-half that of non-diabetics. **Selenium**, the trace element activator of the vitamin E complex, prevents degeneration of the pancreas. **Lithium** has insulin-like effects and assists insulin sensitivity. Lithium disbalance has been found in diabetics. "Avoiding deficiencies of trace elements will enable the reduction of the incidence of diabetes (67 references)." Modern farming methods deplete trace minerals from soil; food processing methods remove any remaining vestige of these nutrients.

Vitamin E complex may assist insulin use by making cell membranes more permeable or receptive to insulin, allowing more glucose into cells. Glucose tolerance is improved and pancreatic beta cells are protected. There is a strong association between low vitamin E status and increased risk of diabetes. This is thought to be due to free radical stress. However, "we don't know if these extra free radicals cause diabetes or result from the disease…" Tissue breakdown due to deficiency-induced and poison-induced stress could certainly result in more free radicals. Glycated hemoglobin and triglycerides (elevated in diabetics) are significantly lowered when vitamin E status is improved. Only "mixed" results occur with fractionated alpha-tocopherol.

Plasma and white blood cell levels of **vitamin C** are low in diabetics, predisposing to capillary fragility, atherosclerosis, elevated blood fats, slow wound healing, eye problems, nerve pathology, and kidney stress. There may be impaired vitamin C tissue storage capabilities. Intracellular scurvy can contribute to many degenerative complications. The C complex participates in insulin regulation by inhibiting glucose-induced insulin release in pancreatic islets. Increased intake results in improved fasting blood sugar, lower cholesterol and triglycerides. Excessive glucose leads to accumulation of sorbitol (sugar alcohol) in cellular cytoplasm, altering osmotic properties. Vitamin C complex limits sorbitol production, reduces glycosylation (how much sugar attaches abnormally to proteins), and improves glucose tolerance. Various **flavonoid** compounds (natural parts of vitamin C complex) reduce sorbitol accumulation, aid blood sugar balance, reduce elevated cholesterol levels, and help prevent diabetic complications such as capillary fragility, cataracts and retinopathy. **Copper**, another constituent in vitamin C complex, improves glucose tolerance.

Vitamin A (retinol) complex and **carotene** complexes are associated with enhanced insulin-mediated glucose disposal -- improved insulin sensitivity. Vitamin A and E complexes contribute to proper relaxation of blood vessel walls, increased insulin delivery to and glucose uptake by muscle tissues, improved wound healing, proper mineral and fat metabolism, and the health of eye, kidney, nerve, skin, and other tissues and organs. Uncontrolled diabetes may affect metabolic availability of vitamin A. Plasma

concentrations are decreased in subjects with IDDM. Livers of people with NIDDM may fail to properly convert carotenoids to vitamin A. Volunteers with impaired glucose tolerance had lower concentrations of carotenes than healthy people; those with diabetes had even lower levels. **Vitamin D** complex is required for normal insulin secretion and glucose tolerance.

Vitamin B complex is very important to anyone with blood sugar or insulin imbalances. **Thiamin** (B_1) aids in glucose metabolism. **Riboflavin** (B_2) metabolism is abnormal in diabetics. **Niacinamide** (B_3) promotes pancreatic beta-cell integrity, stimulates pancreatic insulin secretion, and increases insulin sensitivity within cells. Some diabetics are able to reduce or stop insulin treatment with supplementation, but dependence on insulin resumes within a year if discontinued. Niacinamide can extend the honeymoon or remission phase (indicative of some pancreatic function) after initial diagnosis of diabetes, may halt disease progression and sometimes help restore pancreatic function so insulin is no longer needed. Synthetic niacin causes insulin resistance in normal subjects; only the natural food form is protective. Many diabetics have low blood levels of **pyridoxine** (B_6), even lower if there is nerve damage. Reduced levels of B_6 can cause a significant increase in insulin and glucose levels, problems with amino acid transport and possibly protein synthesis. Diabetics have a high rate of protein breakdown. Pyridoxine improves glucose tolerance and insulin sensitivity. **Vitamin B_{12}** is required for normal function of nerve cells and can reduce nerve damage. Some diabetics show deficiency or disordered metabolism of **biotin** particularly in the presence of neuropathy. Biotin is needed to process glucose. Supplementation may result in a significant drop of fasting glucose levels and reduction of pain when nerve damage exists. **Inositol**, associated with B complex, is needed for normal nerve function; supplementation has resulted in reversed nerve damage. Both **choline** and inositol aid the liver, glucose metabolism, and nerve health. All these vitamins are complexes, work synergistically, and contain many other nutrients. B complex deficits are commonly associated with depression, poor blood sugar control, high blood pressure, and elevated cholesterol and triglycerides.

Coenzyme Q10 and the other coenzyme Qs are required for normal carbohydrate metabolism, are low in diabetics, and are related to nerve damage. In one trial, blood sugar levels fell substantially in 31% of diabetics after supplementation with coenzyme Q10. Liver and heart glandulars are excellent food sources of all coenzyme Qs and associated coworkers.

Lipoic acid is used to treat diabetic neuropathies (nerve degeneration), aids control of diabetes, helps prevent many complications, and "may even help to prevent the onset of diabetes in the first place." Reported to improve blood flow and distal nerve conduction, reverse damage to nerves, heart, and eyes, it has been used in Europe for 30 years. Lipoic acid is a critical sulfur-bearing coenzyme (also called thioctic acid) involved in pyruvate and *a*-ketoglutarate oxidation in the citric acid cycle – the breakdown of carbohydrates, proteins, and fats for energy. In other words, it is needed to convert glucose to chemical energy. It is derived from and works with vitamin B complex in foods. Lipoic acid has been isolated from yeast and liver, foods rich in B complex. The isolate is used pharmacologically in high doses with possible side effects. Whole food complexes are preferable such as organ meats, red meats, nutritional yeast, potatoes, carrots, beets, sweet potatoes, spinach, etc.

Herbs used to assist blood sugar metabolism include: Bitter melon aids in normalizing glucose levels. Bilberry, blueberry, and huckleberry are rich in the blood-sugar lowering compound, myrtilliin, and aid retinopathy. Dandelion root, burdock, and Jerusalem artichoke, inulin-containing herbs, help balance blood sugar. Ginseng may reduce fasting blood sugar and body weight, elevate mood, and improve glycated hemoglobin and physical activity. Nettles, aloe vera, fenugreek, garlic, onions, and Pau D'Arco have been used to effectively lower blood glucose levels. Capsicin-containing peppers and Gingko biloba support peripheral vascular disease and vascular complications. Maitake mushroom may be a tool to manage glucose and insulin sensitivity. Fig leaf tea can allow diabetics to reduce insulin doses. ***Gymnema sylvestre***, appears to help the pancreas produce insulin, improve the ability of insulin to lower blood sugar, and repair beta cells in the pancreas, generally improving blood sugar control. [vii]

EXERCISE

Moderate to vigorous physical activity can reduce the risk of and actually prevent NIDDM as well as help control diabetes by greatly improving blood sugar metabolism and reducing blood lipid levels. Glucose tolerance and insulin sensitivity are improved. Better circulation, reduced blood pressure, improved cardiovascular health, and balanced weight are other benefits. It is well known that being sedentary for a long time impairs the body's ability to handle blood sugar. Diabetics who take only oral medication or no medication receive more dramatic effects than those using insulin injections. [viii]

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