DIABETES: THE BUTTER WITH THE SWEET
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March 1, 1954. The United States tested the largest nuclear device ever tested in the Marshall Islands of the Pacific. It was 1000 times larger than the atomic bomb dropped on Hiroshima. It sent a cloud of fire 100,000 feet into the air. The heat created gale force winds that blew vegetation from surrounding islands. The Marshallese were not amused. Enraged, they raced to court and sued the United States government. The United States conceded and money started rolling into these remote South Sea Islands.

But what does an islander do with money on an isolated island? Soon stuff had to be imported to spend cash on. People who once subsisted on tropical fruits, vegetables and fish, now became enamored with Spam and frozen turkey tails, [as well as our other convenience foods high in fat, salt and sugar, and low in nutrition.] The health results of such lifestyle changes quickly became apparent. Type II diabetes, almost unheard of in the Pacific islands before these dietary changes, now rendered 30% of the people over age 15 diabetic, and also high rates of hypertension, cardiovascular disease, kidney failure, eye disease, and amputations.[1]

"But I thought diabetes was inherited." someone may be thinking.
Inherited from the grocer, I might caution.

WORLD WIDE EPIDEMIC

Sadly diabetes proliferation is not limited to the Marshall Islands. World wide, diabetes is expected to increase by 46% in the next 10 years. The largest increases will be in the developing countries of Africa, China and South America[2]—countries that can ill afford the increased medical complications and costs associated with such a disease.
Nor is the United States immune to such increases in the number of diabetics. According to the CDC the U.S. had 5.8 million diabetics in 1980. By 2005 this number had jumped to an all time high of 20.8 million[3], and we know the population has not tripled during that same time. If diabetes were inherited we would have to conclude that diabetics are having far more babies than the rest of society! This is not actually possible because diabetics have difficult pregnancies.

"What are my chances of getting diabetes?" someone may be wondering.
Lifetime risk of getting diabetes in the United States for Caucasians is 39% for women and 33% for men. Hispanics suffer a little higher incidence at 53% for females and 45% males.[4]

The American Diabetes Association estimates the 2002 total cost for diabetes in the United States at $132 Billion. With the rise in diabetes they calculate that by 2020 we will be spending nearly $200 Billion on diabetes.[5]

COMPLICATIONS OF DIABETES

Diabetes causes multiple complications if blood sugar is not controlled. Complications can take many forms and can occur in various places throughout the body.
Heart disease[6] and stroke[7] kill 80% of diabetics.
Three out of four diabetics have high blood pressure.[8]
Diabetes is the number one cause of blindness accounting for 24,000 new cases each year.[9]
In 2005 there were 46,000 new cases of kidney failure resulting from diabetes, and an ongoing total of 179,000 cases.[10]

Thirty to 50% of diabetics suffer nerve damage that results in carpal tunnel syndrome,[11] pain or numbness in the feet or hands [peripheral neuropathy],[12] and slowed digestion of food.[13]

Diabetes is one of the greatest causes of amputations in the United States. A diabetic has ten times the risk of amputation. There are over 80,000 amputations per year in diabetics alone.[14] Diabetics who experience amputations on both legs never get back to walking like they once did.

Diabetics are 10 to 34% more likely to become depressed.[15] They experience more mood and memory changes and studies show that their brains actually shrink.[16]

Infections occur more frequently in diabetics making them more likely to die with a pneumonia or influenza. Life expectancy of diabetics is reduced by 12-14 years.[17] Not only does diabetes shorten ones life but it more than triples the risk of ending up in a nursing home in middle age.[18]
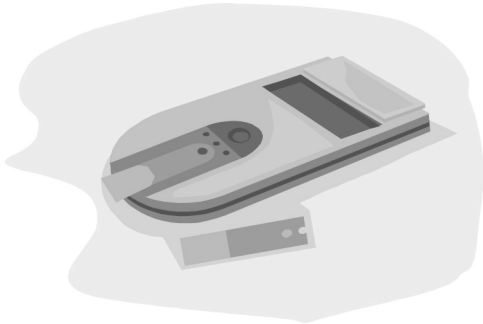
HISTORY OF DIABETES

The earliest recorded history of diabetes comes from Egypt in 1552 B.C.[19] It is interesting to note that this would have been around the time that the Israelites were enslaved in that country. Upon emancipation, scripture records that God told them, "If thou wilt diligently hearken to the voice of the LORD thy God, and wilt do that which is right in his sight, and wilt give ear to his commandments, and keep all his statutes, I will put none of these diseases upon thee, which I have brought upon the Egyptians: for I am the LORD that healeth thee."[20] Apparently, if the Israelites followed God's instructions they would be spared the metabolic syndrome embarrassment.

WHAT IS DIABETES?

Diabetes is too much sugar in the blood and urine. Blood sugar is tested on a fasting blood test. Normal blood sugar should be between 70-99 mg/dl. A blood sugar after fasting of between 100-125 mg/dl is defined as pre-diabetes. Any fasting blood sugar above 125 mg/dl diagnoses diabetes.[21]

WHAT CAUSES DIABETES?

In an effort to answer this question, Dr. James Anderson, renowned diabetologist, decided to feed healthy young men two pounds of sugar a day and check for signs of diabetes. Thirteen weeks into the study there were still no signs of diabetes.[22]

"I thought diabetes was too much sugar in the blood and urine," you may be thinking.

Recent research has confirmed the real culprit—fat. People on a low fat diet, (10-15% of calories), where the fat comes from a vegetable source, have a relatively low risk of getting diabetes. On the other hand, people eating 46% of their calories as fat have a 40% higher risk of diabetes. Certain fats are especially dangerous. Just 3% of calories coming from trans-fat will raise the risk of diabetes by 44% , and 270 mg of cholesterol, little more that that found in one egg, will increase the risk by 60%. If the majority of fat in the diet, (36% of calories), comes from saturated fat (usually animal sources) the risk of diabetes goes up to 64%.[23] Animal studies have shown that increasing the fat intake to 65% of calories increases the incidence of diabetes by 350%.[24] Fat makes a difference!

Various fats have different physiological effects on the body. Saturated and trans-fats tend to increase cholesterol, raise blood pressure, and diminish the good HDL cholesterol levels. They also

increase the risk for heart disease, stroke, certain cancers and diabetes.[25],[26] Unsaturated fats, taken in their moderate amounts, (10%-15% of calories), tend to lower cholesterol, help maintain healthy HDL levels, provide essential fatty acids, and lower the risk for heart disease, stroke and diabetes.[27],[28]

Some of the healthiest fats come from natural plant sources. Five servings of nuts (raw or dry roasted) a week have been shown to decrease the incidence of diabetes by 30%.[29] The unhealthy fats tend to come from fast foods that are high in fat and low in nutrition. Two or more fast food meals per week will not only increase obesity but also can double the risk of diabetes.[30]

Fat is not the only culprit in fast foods; one sugar-sweetened soft drink per day can increase the risk of diabetes by 83%.[31]

There are other causes of diabetes. Remember the old "four food group" posters hung on grade school classroom walls, dairy, meat, grains, and plant foods? These posters were not an initiative of the National Institute of Health or National Academy of Sciences. Studies show that milk and red meat consumption increases insulin resistance leading to the development of obesity, cardiovascular disease, and diabetes.[32]

Stimulants can also increase diabetes. Smoking increases the risk of diabetes by 60%.[33] Caffeine increases diabetic blood sugars by 28%[34] and decreases the effectiveness of exercise in lowering blood sugar.[35] Alcohol increases obesity and destroys the pancreas' ability to produce insulin.[36] Narcotics increase insulin resistance within the cells.[37]

DRUGS AND DIABETES

There are certain prescription drugs that increase the risk of diabetes. Blood sugars tend to be harder to control with the use of some blood pressure medications (thiazide diuretics and beta-blockers, etc), atypical antipsychotic drugs (Clozapine, Zyprexa, Seroquel, etc), steroids such as prednisone,[38] and oral contraceptive pills.[39] Alternatives may need to be discussed with a physician.

"DIABESITY"

Obesity is one of the most powerful risk factor for type 2 diabetes.[40] While people that are considered underweight have a 7% lifetime risk of acquiring diabetes, those who meet the criteria for "very obese" have a 57% lifetime diabetes risk.[41] In fact, just 2 pounds weight gain can increase the risk of diabetes by 4%.[42] As more and more Americans become obese, the number of diabetics goes up proportionately. The most dangerous fat is that which accumulates inside the abdomen around the belly by the organs— what we call central fat or visceral fat. An increase in this fat increases resistance to the action of insulin and increases the risk of heart disease[43] and other complications.

STRESS: DIABETES AND OBESITY

People with type A personality tend to have more stress. Type A personalities have more than twice the risk of diabetes.[44],[45] Psychosocial stress unbalances the body's hormones promoting central obesity, diabetes, and cardiovascular disease.[46]

WHY IS DIABETES A PROBLEM?

When fat cells are too full, as in obese people, they lose the ability to respond to insulin. The fatigued pancreas eventually loses its capacity to produce enough insulin and blood sugar rises even higher.

Each fat cell has insulin receptors. When these receptors are stimulated by insulin they facilitate the passage of sugar into the cell. Think of insulin receptors as door knobs and insulin as the doorkeeper who opens the doors. The way the cells regulate how much sugar they take in is by increasing or decreasing the number of insulin receptors (door knobs) available for insulin to activate (open

the door to sugar). For example a normal cell puts some of its insulin receptors (door knobs) out into the blood stream where insulin can activate them (open sugar doors). Sugar then moves out of the blood stream into the cells lowering the blood sugar. Overfed fat cells pull all of their insulin receptors into the cell (leaving no doors to open). As a consequence the sugar accumulates in the blood stream increasing blood sugar to dangerous levels. When the diabetic starts to exercise, the cells get hungry and start putting more receptors into the blood stream thus making way for more sugar to enter the cells lowering the blood sugar.[47]

GLYCEMIC INDEX AND GLYCEMIC LOAD

Glycemic index indicates the effect specific carbohydrates have on blood sugar levels in comparison to the effect of pure sugar. High glycemic index foods raise blood sugar and insulin levels much higher and more rapidly than low glycemic index foods.[48] For example 50 gm of glucose has a glycemic index of 100, it enters the blood stream 100% as fast as pure sugar. A bowl of corn flakes and milk has a glycemic index of 92, meaning that the sugar in a bowl of corn flakes and milk enters the blood 92% as fast as pure sugar. Broccoli has a glycemic index of around 15, meaning that the carbohydrate in broccoli has 15% the effect of pure sugar on blood sugar.

The amount of food consumed is a major determinant of blood sugar. Glycemic load takes into account the amount of a certain glycemic indexed food eaten.[49] High glycemic load foods include calorie dense foods such as snack foods, fast foods, pastry, cookies, sweets, soda pop, white bread and white rice, refined carbohydrates and white potatoes. Low glycemic load foods include whole-grain breads and cereals, including oatmeal and brown rice, legumes, peas, beans, garbanzos, soy, tofu, fresh fruit and vegetables, nuts, protein rich foods and healthy fats.

We were not made to eat high glycemic load meals. Rats fed a high glycemic diet develop marked obesity in 32 weeks.[50] Fat rats are generally not seen out in nature. Humans fed high glycemic meals eat a larger volume of food, feel less satisfied, and get hungry sooner.[51],[52]

CONSEQUENCES OF HIGH BLOOD SUGAR

High blood sugar causes the triglycerides go up.
"Why would triglycerides go up?" Someone may be wondering, "I thought triglycerides were fats!"
The body has no little box in which to store sugar cubes. That's right. In order to store excess sugar the body needs to convert it to something it can store, like fat. So up go the triglycerides.
High blood sugar causes proteins to be glycated.
"Glycated! What's glycated?" you may be wondering.
Glycated is when sugar sticks to or coats the proteins in the body, like blood cells and blood vessels. Sugar clogs the system.
High blood sugar provokes insulin to rise.
Insulin is not just for chasing excess blood sugar into cells; it's also a growth factor. As a growth factor it needs building blocks for growth. Cholesterol is one of these building blocks. Elevated insulin results in elevated cholesterol. Elevated insulin also increases blood pressure.[53] Insulin not only makes the abdomen grow (in central obesity) but it also can make tumors grow increasing the chance of cancer.[54],[55]
Sugar weakens the body's white blood cell's ability to destroy bacteria.
Studies show that on a good day, one white blood cell can kill 14 dangerous disease causing bacteria. With the intake of just 12 teaspoons of sugar, the amount contained in most soft drinks, each white cell can only destroy 5-1/2 bacteria. Double the soft drink intake and the number of bacteria a white cell can destroy drops to just one![56] Most Americans consume more than 52 teaspoons of sugar a day![57]

BLOOD SUGAR RESPONSE TO WHOLE FOODS

The more carbohydrates are refined the higher their glycemic index. For example orange juice is the refined product of oranges. Not only is the fiber removed, but also in the process of preservation the juice is "pasteurized", meaning that it has been heat treated in an effort to reduce the number spoilage causing microbes. This process of heat-treating has a further refining influence on the carbohydrate in oranges breaking it down in to shorter chain starches and simpler sugars. Commercial orange juice is little different that soda pop in its effect on the body.[58] As a consequence drinking commercial orange juice elevates blood sugar very rapidly and to an excessive degree. What's more, once the body responds with insulin, the blood sugar drops precipitously leaving the individual faint and craving more refined carbohydrate. By comparison, eating a whole orange has a very different effect. The whole orange has not only sugar, but fiber, vitamins, phytochemicals, and minerals which help slow the passage of sugar into the blood stream and help the body more efficiently use the sugar. Because the sugar enters the blood more slowly and over a longer period of time, a precipitous fall in blood sugar that triggers hunger and faintness does not occur.[59]

Diabetics tend to eat foods of higher glycemic index.[60] The effect is that their blood sugar goes up quite quickly. The body responds with a surge of insulin to take care of the emergency. Insulin can rise rapidly but it cannot drop as rapidly as blood sugar. Consequently, before long the sugar runs out and the person becomes hypoglycemic—low on blood sugar—faint and hungry. They then look for food, probably long before the next *scheduled* meal. The foods they choose to fulfill their low blood sugar needs are usually junk foods that compound the process and the problem.

In order to ameliorate this problem, diabetics are told just to eat many little meals all day long.[61],[62] Does this work? Yes. For two reasons, first, if little meals are eaten all day long, eventually all the blood sugar spikes will coalesce into one long sugar swell with less variability and there will be no more dangerous peaks and troughs. If it weren't that frequent meals more than doubled the risk of cancer maybe it might have some merit.[63],[64]

The problems with eating more often are not limited to increased cancer risk. The stomach is a bit like the common household washing machine. A load of clothes is started washing. Halfway through the cycle some unruly person comes with more dirty clothes and adds them to the load. If the clothes are to be cleaned the whole cycle must be started over and perhaps more soap added. This is just the way the stomach works. If more food is added before it finishes its work, it has to start over and add more digestive juice.[65] Thus the machinery is jammed, sugar digestion impeded, and the diabetic controls this blood sugar at the cost of the delicate digestive organs.

What is the real solution? If the diabetic eats an unrefined whole grain breakfast—high in fiber (low glycemic index)—the blood sugar rises slowly, the sugar supply is steady, and there is no precipitous fall in blood sugar at the end of digestion. By lunch time the diabetic is just starting to feel hungry. Lunch is a meal of whole vegetables and legumes and the sugar is held at a manageable level all day long.

Sugar Clogs The System.

Glucose (blood sugar) is the preferred fuel of the cell. But, too much glucose in the blood clogs blood vessels and coats the blood cells with sugar. As blood sugar rises, excess sugar begins to stick to proteins—blood cell and blood vessel walls. These sugar coated blood cells are called hemoglobin A1c or HbA1c. HbA1c predicts increased risk of heart disease and overall mortality even for people without diabetes. HbA1c indicates the average blood sugar concentration over the past three months. A HbA1c of 7.0 or higher may indicate diabetes. An increase of just 1% in HbA1c is associated with roughly a 30% increase in mortality from all causes and a 40% increase in mortality from coronary heart disease. In one study 70%

of non-diabetics over 45 had a hemoglobin HbA1c of 5% or greater. Thus 82% of excess mortality due to blood sugar elevations then is in non-diabetes.[66] And here we were all patting ourselves on the back because we are not diabetic. Living the lifestyle of a diabetic may not make everyone diabetic, but it may still provide us with a full complement of diabetic complications.

When HbA1c goes up, so does glycation of the vessel walls. Thus not only do the blood cells have trouble functioning, due to the sugar coating, but the sugar-coated blood vessels pose an additional barrier to nutrients reaching body tissues. When this happens, body tissues starve for oxygen and nutrients making them more susceptible to fatigue, damage, and infection. This explains some of the complications listed earlier such as the elevated risk of amputation.

On the other hand, a 1% reduction in HbA1c lowers the risk of stroke by 17%, fatal heart attack by 18%, diabetic deaths by 25%,[67] amputation,[68] kidney failure, and diabetic retinopathy that leads to blindness each by 30%.[69]

SIGNS OF DIABETES

What are the signs of diabetes?

The signs of diabetes include:
- Low energy.
- Fatigue.
- Extreme thirst.
- Frequent urination.
- Blurred vision.
- Irritability and mood changes.
- Weight changes.
- Tingling and numbness in hands or feet.
- Frequent infections.
- Extreme hunger.
- Cuts and bruises that are slow to heal.
- Nausea and vomiting.
- Dehydration.
- Reduced conscious level.

Reduced consciousness! I was in the emergency room one day. In the stall next to where I was working an ER doctor was trying to awaken someone.

"Wake up! Wake up! Can you hear me?"

"What, who me? Where am I?"

"Did you know you blood sugar was 300? Are you a diabetic?"

"What? Who me? A diabetic?"

Sad to say this is the way all too many people discover they are diabetics. Their blood sugar goes too high, they pass out, and someone finds them and sends them to the hospital emergency room. This is not the way to discover diabetes. By this time the complications of diabetes are well on their way.

CAN PEOPLOE REVERSE THEIR DIABETES?

Kit Carson was taking 85 units of insulin daily. He was a big guy - 6'8" and 440 lbs. He relied on his pickup to go even short distances. Two days into the "Reversing Diabetes" lifestyle program his blood sugar, which had been as high as 500, returned to normal. In two years he lost 135 lbs. He never used insulin again. He says, "This program has changed my life."[70]

Can lifestyle changes really have that great of an effect on diabetes? The New England Journal of Medicine answered this question. They reported on an intervention trial to prevent diabetes in pre-diabetics that compared the effects of placebo (doing nothing), pharmacological medications, or lifestyle interventions. The results? Medications reduced the incidence of diabetes by 31% and lifestyle modification by 58%![71] This demonstrates that lifestyle change is a much stronger medical intervention than medications. Well, it makes sense; lifestyle caused the diabetes in the first place, not pills.

And what were the lifestyle interventions? Lifestyle intervention included weight loss with a goal 7% of weight; daily exercise with a goal 150 minutes per week; improved eating including higher fiber intake, lower saturated fat, and lower glycemic load. After 3 years, the incidence of diabetes was 58% lower in the lifestyle intervention group.

LIFESTYLE INTERVENTIONS

What lifestyle changes was Kit Carson asked to make?

Change the diet to the "whole plant food whole" diet. Okay, so aren't there too many "wholes" in that sentence? The point to be made is, eat plant foods and eat them in their entirety—don't let any one "refine" them.[72] So, what are some examples of "whole plant foods whole"? –brown rice, whole wheat flour products, fresh carrots, broccoli, spinach, and granola, etc. What are some examples of foods that are not whole plant foods whole?- cows milk, eggs, pancake mixes, crackers that have refined flour, fast foods, most foods that come in crinkly packages, sugar and oil, fish, anything with oil as an added ingredient, etc.

One reason refined foods are dangerous is their lack of fiber. Only about 5% of Americans get as much fiber as is recommended. Fiber plays an important roll in diabetes prevention and management. Fiber protects against constipation, high cholesterol, heart disease, high blood sugar, diabetes, certain cancers, and obesity.[73]

In Harvard's Woman's Health Study, eating low fiber, high glycemic index foods more than doubled the risk of getting diabetes.[74] In another study oat bran bread reduced blood sugar response by 46% and insulin response by 19% compared to refined white bread in the diet.[75]

A patient came in with gestational diabetes (diabetes resulting from the changes that pregnancy does to the body) who did not want to take drugs or insulin for fear of what they might do to her unborn child. She was adamant—no pills, no shots. She was advised to eat ½ cup of oat bran three times a day. And she did eat those three half cups of oat bran - oat bran cookies, oat bran cereal, oat bran bread, oat bran in drinks. Her blood sugar was totally controlled; she delivered a normal healthy baby and her diabetes was gone.

Fiber slows the rate at which sugar enters the blood stream. Even more fiber than is recommended for the average American is recommended for diabetics, 50 gm or more of daily fiber.[76]

Another great benefit of the whole plant food diet is whole grains. Replacing refined grains in the diet with whole grains can reduce the risk of diabetes by 70%.[77] Increasing whole grains to 3 servings per day can decrease the risk of diabetes by 50%.[78]

Another benefit of eating whole plant foods is that they actually require chewing. Thorough chewing increases the early insulin response to eating, decreases blood sugar,[79] and helps reduce food intake.[80] Reduced food intake helps with weight control and increases insulin sensitivity.[81,82]

A healthy breakfast plays a major role in diabetes reduction. People who eat breakfast tend to eat less total calories for the entire day; have lower cholesterol levels; and end up with less diabetes.[83] One study shows a 37%-55% reduction in risk of diabetes in people who eat a regular breakfast compared to those who don't.[84]

Refined food products generally have had many nutrients removed from them that are necessary for life and for the processing of carbohydrates. If a person eats a food that is totally devoid of a nutrient that is necessary for life, the body has to take from its own stores just to survive, thus depleting its own reserves of that necessary nutrient.

Chromium is an example. Diabetics generally have no tissue chromium.[85] Consumption of refined carbohydrates, such as refined sugar, tends to deplete chromium stores. In diabetics, increasing chromium has been shown to decrease fasting glucose levels, improve glucose tolerance, lower insulin levels, decrease total cholesterol and triglycerides, increase HDL-cholesterol levels, and ameliorate the symptoms of hypoglycemia.[86] Whole wheat has eight times the chromium of white flour. Brown rice has four times the chromium of white rice.

Diabetes,[87,88] coronary heart disease,[89] hypertension and high triglycerides[90] are all associated with low dietary zinc. Whole wheat flour has four times the zinc as white flour. Pumpkin seeds and lentils are also good sources of zinc.

Magnesium levels are significantly lower in diabetics[91]—especially diabetics with complications such as poor glycemic control, retinopathy, obesity and hypertension.[92] The major dietary sources of magnesium include whole grains, legumes, nuts, and green leafy vegetables.[93]

A word of caution, the longer a person has been a diabetic, the longer it may take to respond to dietary changes.[94]

BENEFITS

Remember earlier we said that exercise makes the cells hungry again, reducing insulin resistance? Exercise lowers blood sugar and insulin, but it also helps to control weight. More than that, a good walk out in the fresh air and beautiful sunshine improves mental outlook, helping to deal with the diabetes promoting stress.[95] Compared to those who live a sedentary lifestyle, those who are highly active have a 46% lower incidence of diabetes.[96] It has been said, if you can't find time for exercise, you will have to find time to be sick. And, more people die for want of exercise than through over fatigue….

MAKE WEIGHT LOSS A HABIT

The most dangerous fat for the diabetic is the central fat or what is called visceral fat—that fat which settles in the abdomen around the organs. This fat is always at a higher temperature and releases toxins that increase diabetic complications. One thing the diabetic has in their favor is that all the fat reduces simultaneously—any reduction includes abdominal fat.[97] The goal then is not only to lose a little weight but also to make weight loss a habit. As long as weight is not gained back, the visceral fat will continue to melt away. Continuous weight loss, approaching ideal body weight, can reduce the risk of diabetes by 30-50%.[98]

Sunshine, Vitamin D and Diabetes

Part of the program for reducing diabetes involves getting adequate sun exposure. This helps maintain vitamin D levels. Studies show that vitamin D deficiency increases the risk of diabetes[99] and that vitamin D supplementation can actually reduce the risk.[100] Diabetics are at increased risk of osteoporosis. This risk is also reduced by sunshine and vitamin D.

WATER: ELIXIR OF LIFE

Which is sweeter, a raisin or a fresh grape? Usually a raisin is. So it is with blood, drinking more water thins the blood and lowers blood sugar by sheer dilution,[101] protects against ketoacidosis (the condition that sends diabetics to the emergency room with alter consciousness),[102,103] and decreases diabetic neuropathy.[104] Humans need between 8 and 12 eight ounce glasses of water daily. Diabetics are no exception and benefit greatly from drinking water.[105]

REVERSING DIABETES SUMMARY

- Regular exercise in the open air and sunshine (a little is better than none).
- Eat an unrefined plant based diet, low in fat, high in fiber.
- Make breakfast a healthy habit.
- Make weight control a habit.
- Drink plenty of water.
- Get adequate sleep.

- Avoid stimulants such as coffee, alcohol and tobacco.
- Turn stress over to God who alone can handle it.

HAS THIS PROGRAM WORKED?

It has. In the "Reversing Diabetes" program, diabetic patients were studied for 25 days on a special diet, exercise program with adequate sunshine, rest and pure filtered water. Patients were fed an unrefined plant based diet, low in fat (10%-15%), high in fiber with no cholesterol.

The average weight loss was 11 pounds. One fourth of diabetics no longer needed insulin or drugs to control blood sugar. Those who still needed insulin saw their requirements cut nearly in half. Blood pressures dropped from an average of 155/81 to 132/77, and 81% had complete relief of peripheral neuropathy.[106]

One example from the "Reversing Diabetes" program is John Rowe, R.N., E.R. nurse and a diabetic for eleven years, who was injecting up to 144 units of insulin a day. Within two days of adopting the "Reversing Diabetes" lifestyle, his blood sugar returned to normal without medication. He lost thirty-five pounds in four months. His blood pressure fell to normal and his vision greatly improved.[107]

And exactly what dietary program are we really talking about? - the original Bible diet!

Then God said, "I give you every seed-bearing plant on the face of the whole earth and every tree that has fruit with seed in it. They will be yours for food." "And you will eat the plants of the field."[108]

Recall that the earliest record of diabetes comes from the pyramids of Egypt during the time that the Israelites were liberated, and that God said, "If thou wilt diligently hearken to the voice of the LORD thy God, and wilt do that which is right in his sight, and wilt give ear to his commandments, and keep all his statutes, I will put none of these diseases upon thee, which I have brought upon the Egyptians: for I am the LORD that healeth thee."[109]

Why die of diabetes? Why not make it a point to exercise regularly and eat only a whole plant based diet?

Diabetes The Butter With The Sweet: The End.

References

[1] The Baltimore Sun, Oct 26, 1997.

[2] Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes epidemic. Nature. 2001 Dec 13;414(6865):782-7.

[3] CDC, Diabetes Data and Trends, and CDC, Diabetes Fact Sheet, Oct. 26, 2005

[4] Narayan KM, Boyle JP, Thompson TJ, Sorensen SW, Williamson DF. Lifetime risk for diabetes mellitus in the United States. JAMA. 2003 Oct 8;290(14):1884-90.

[5] Hogan P, Dall T, Nikolov P; American Diabetes Association. Economic costs of diabetes in the US in 2002. Diabetes Care. 2003 Mar;26(3):917-32.

[6] Sowers JR, Epstein M, Frohlich ED. Diabetes, hypertension, and cardiovascular disease: an update. Hypertension. 2001 Apr;37(4):1053-9.

[7] Hu G, Sarti C, Jousilahti P, Peltonen M, Qiao Q, Antikainen R, Tuomilehto J. The impact of history of hypertension and type 2 diabetes at baseline on the incidence of stroke and stroke mortality. Stroke. 2005 Dec;36(12):2538-43.

[8] Kabakov E, Norymberg C, Osher E, Koffler M, Tordjman K, Greenman Y, Stern N. Prevalence of hypertension in type 2 diabetes mellitus: impact of the tightening definition of high blood pressure and association with confounding risk factors. J Cardiometab Syndr. 2006 Spring;1(2):95-101.

[9] Klein R, Klein BEK. Vision disorders in diabetes. In: National Diabetes Data Group, editors, Diabetes in America, 2nd ed. Washington, DC: U.S. Department of Health and Human Services, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, 1995. NIH Publication No. 95-1468. 293 336.

[10] U.S. Renal Data System, USRDS 2007 Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda, MD, 2007.

[11] Mota M, Panuş C, Mota E, Sfredel V, Patraşcu A, Vanghelie L, Toma E. Hand abnormalities of the patients with diabetes mellitus.Rom J Intern Med. 2000-2001;38-39:89-95.

[12] Gregg EW, Sorlie P, Paulose-Ram R, Gu Q, Eberhardt MS, Wolz M, Burt V, Curtin L, Engelgau M, Geiss L; Prevalence of lower-extremity disease in the US adult population >=40 years of age with and without diabetes: 1999-2000 national health and nutrition examination survey. Diabetes Care. 2004 Jul;27(7):1591-7.

[13] Horowitz M, Wishart JM, Jones KL, Hebbard GS. Gastric emptying in diabetes: an overview. Diabet Med. 1996 Sep;13(9 Suppl 5):S16-22.

[14] Centers for Disease Control and Prevention (CDC), National Center for Health Statistics, Division of Health Care Statistics, data from the National Hospital Discharge Survey and Division of Health Interview Statistics, data from the National Health Interview Survey. U.S. Bureau of the Census, census of the population and population estimates and National Center for Health Statistics, CDC, bridged-race population estimates. Data computed by personnel in the Division of Diabetes Translation, National Center for Chronic Disease Prevention and Health Promotion, CDC.

[15] Wexler DJ. Low risk of depression in diabetes? Would that it were so. CMAJ. 2006 Jul 4;175(1):47.

[16] Reagan LP. Insulin signaling effects on memory and mood. Curr Opin Pharmacol. 2007 Dec;7(6):633-7.

[17] Narayan KM, Boyle JP, Thompson TJ, Sorensen SW, Williamson DF. Lifetime risk for diabetes mellitus in the United States. JAMA. 2003 Oct 8;290(14):1884-90.

[18] Valiyeva E, Russell LB, Miller JE, Safford MM. Lifestyle-related Risk Factors and Risk of Future Nursing Home Admission. Arch Intern Med 2006; 166 (May8):985-990.

[19] Loriaux, DL. Diabetes and The Ebers Papyrus: 1552 B.C. Endocrinologist. 16(2):55-56, March/April 2006.

[20] Exodus 15:26 King James Version of the Holy Bible.

[21] American Diabetes Association. Standards of medical care in diabetes. Diabetes Care. 2005 Jan;28 Suppl 1:S4-S36.

[22] Anderson JW, Herman RH, Zakim D. Effect of high glucose and high sucrose diets on glucose tolerance of normal men. Am J Clin Nutr. 1973 Jun;26(6):600-7.

[23] Adapted from: Salmerón J, Hu FB, Manson JE, Stampfer MJ, Colditz GA, Rimm EB, Willett WC. Dietary fat intake and risk of type 2 diabetes in women. Am J Clin Nutr. 2001 Jun;73(6):1019-26.

[24] Wang Y, Wang PY, Qin LQ, Davaasambuu G, Kaneko T, Xu J, Murata S, Katoh R, Sato A. The development of diabetes mellitus in Wistar rats kept on a high-fat/low-carbohydrate diet for long periods. Endocrine. 2003 Nov;22(2):85-92.

[25] Storlien LH, Jenkins AB, Chisholm DJ, Pascoe WS, Khouri S, Kraegen EW. Influence of dietary fat composition on development of insulin resistance in rats. Relationship to muscle triglyceride and omega-3 fatty acids in muscle phospholipid. Diabetes. 1991 Feb;40(2):280-9.Links

[26] Hu FB, van Dam RM, Liu S. Diet and risk of Type II diabetes: the role of types of fat and carbohydrate. Diabetologia. 2001 Jul;44(7):805-17.

[27] Trichopoulou A, Lagiou P. Worldwide patterns of dietary lipids intake and health implications. Am J Clin Nutr. 1997 Oct;66(4 Suppl):961S-964S.

[28] Picinato MC, Curi R, Machado UF, Carpinelli AR. Soybean- and olive-oils-enriched diets increase insulin secretion to glucose stimulus in isolated pancreatic rat islets. Physiol Behav. 1998 Nov 15;65(2):289-94.

[29] Jiang R, Manson JE, Stampfer MJ, Liu S, Willett WC, Hu FB. Nut and peanut butter consumption and risk of type 2 diabetes in women. JAMA. 2002 Nov 27;288(20):2554-60.

[30] Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, Ludwig DS. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. Lancet. 2005 Jan 1-7;365(9453):36-42.

[31] Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, Hu FB. Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. JAMA. 2004 Aug 25;292(8):927-34.

[32] Papakonstantinou E, Panagiotakos DB, Pitsavos C, Chrysohoou C, Zampelas A, Skoumas Y, Stefanadis C. Food group consumption and glycemic control in people with and without type 2 diabetes: the ATTICA study. Diabetes Care. 2005 Oct;28(10):2539-40. Related Articles, Links

[33] Hur NW, Kim HC, Nam CM, Jee SH, Lee HC, Suh I. Smoking cessation and risk of type 2 diabetes mellitus: Korea Medical Insurance Corporation Study. Eur J Cardiovasc Prev Rehabil. 2007 Apr;14(2):244-9.

[34] Lane JD, Hwang AL, Feinglos MN, Surwit RS. Exaggeration of postprandial hyperglycemia in patients with type 2 diabetes by administration of caffeine in coffee. Endocr Pract. 2007 May-Jun;13(3):239-43.

[35] Lee S, Hudson R, Kilpatrick K, Graham TE, Ross R. Caffeine ingestion is associated with reductions in glucose uptake independent of obesity and type 2 diabetes before and after exercise training. Diabetes Care. 2005 Mar;28(3):566-72.

[36] Greenhouse L, Lardinois CK. Alcohol-associated diabetes mellitus. A review of the impact of alcohol consumption on carbohydrate metabolism. Arch Fam Med. 1996 Apr;5(4):229-33.

[37] Li Y, Eitan S, Wu J, Evans CJ, Kieffer B, Sun X, Polakiewicz RD. Morphine induces desensitization of insulin receptor signaling. Mol Cell Biol. 2003 Sep;23(17):6255-66.

[38] Izzedine H, Launay-Vacher V, Deybach C, Bourry E, Barrou B, Deray G. Drug-induced diabetes mellitus. Expert Opin Drug Saf. 2005 Nov;4(6):1097-109.

[39] Spellacy WN. Carbohydrate metabolism during treatment with estrogen, progestogen, and low-dose oral contraceptives. Am J Obstet Gynecol. 1982 Mar 15;142(6 Pt 2):732-4.

[40] Anderson JW, Kendall CW, Jenkins DJ. Importance of weight management in type 2 diabetes: review with meta-analysis of clinical studies. J Am Coll Nutr. 2003 Oct;22(5):331-9.

[41] Narayan KM, Boyle JP, Thompson TJ, Gregg EW, Williamson DF. Effect of BMI on lifetime risk for diabetes in the U.S. Diabetes Care. 2007 Jun;30(6):1562-6.

[42] Mobley CC. Lifestyle interventions for "diabesity": the state of the science. Compend Contin Educ Dent. 2004 Mar;25(3):207-18.

[43] Mori Y, Hoshino K, Yokota K, Itoh Y, Tajima N. Differences in the pathology of the metabolic syndrome with or without visceral fat accumulation: a study in pre-diabetic Japanese middle-aged men. Endocrine. 2006 Feb;29(1):149-53.

[44] Hu C, Li L, Lu M. Case control study of the relationship between type A character and type II diabetes mellitus. Zhonghua Yi Xue Za Zhi. 2001 Feb 25;81(4):205-7.

[45] Gogiberidze OG, Gogiberidze KO, Kavtaradze GV. Behavioral risk factors in patients with diabetes mellitus type II. Georgian Med News. 2005 Jan;(118):29-31.

[46] Björntorp P. Body fat distribution, insulin resistance, and metabolic diseases. Nutrition. 1997 Sep;13(9):795-803.

[47] Borissova AM, Tankova TI, Koev DJ. Insulin secretion, peripheral insulin sensitivity and insulin-receptor binding in subjects with different degrees of obesity. Diabetes Metab. 2004 Nov;30(5):425-31.

[48] Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, Bowling AC, Newman HC, Jenkins AL, Goff DV. Glycemic index of foods: a physiological basis for carbohydrate exchange. Am J Clin Nutr. 1981 Mar;34(3):362-6.

[49] Venn BJ, Green TJ. Glycemic index and glycemic load: measurement issues and their effect on diet-disease relationships. Eur J Clin Nutr. 2007 Dec;61 Suppl 1:S122-31.

[50] Pawlak DB, Kushner JA, Ludwig DS. Effects of dietary glycaemic index on adiposity, glucose homoeostasis, and plasma lipids in animals. Lancet. 2004 Aug 28-Sep 3;364(9436):778-85.

[51] Roberts SB. High-glycemic index foods, hunger, and obesity: is there a connection? Nutr Rev. 2000 Jun;58(6):163-9.

[52] Augustin LS, Franceschi S, Jenkins DJ, Kendall CW, La Vecchia C. Glycemic index in chronic disease: a review. Eur J Clin Nutr. 2002 Nov;56(11):1049-71.

[53] Reaven GM. Pathophysiology of insulin resistance in human disease. Physiol Rev. 1995 Jul;75(3):473-86.

[54] Hammarsten J, Högstedt B. Hyperinsulinaemia: a prospective risk factor for lethal clinical prostate cancer. Eur J Cancer. 2005 Dec;41(18):2887-95.

[55] Lawlor DA, Smith GD, Ebrahim S. Hyperinsulinaemia and increased risk of breast cancer: findings from the British Women's Heart and Health Study. Cancer Causes Control. 2004 Apr;15(3):267-75.

[56] Kijak. E.; Foust G; Steinman R.R.; Relationship of Blood Sugar Level and Leukocytic Phagacytosis; Southern California Dental Association 1964; 32(9):349-351.

[57] United States Department of Agriculture, Office of Communications. Agriculture Fact Book 2001-2002. March 2003. http://www.usda.gov/factbook/2002factbook.pdf

[58] Sullivan MJ, Scott RL. Postprandial glycemic response to orange juice and nondiet cola: is there a difference? Diabetes Educ. 1991 Jul-Aug;17(4):274-8.

[59] Bolton RP, Heaton KW, Burroughs LF. The role of dietary fiber in satiety, glucose, and insulin: studies with fruit and fruit juice. Am J Clin Nutr. 1981 Feb;34(2):211-7.

[60] Laitinen JH, Tuorila HM, Uusitupa MI. Changes in hedonic responses to sweet and fat in recently diagnosed non-insulin-dependent diabetic patients during diet therapy. Eur J Clin Nutr. 1991 Aug;45(8):393-400.

[61] Jenkins DJ. Carbohydrate tolerance and food frequency. Br J Nutr. 1997 Apr;77 Suppl 1:S71-81.

[62] Bertelsen J, Christiansen C, Thomsen C, Poulsen PL, Vestergaard S, Steinov A, Rasmussen LH, Rasmussen O, Hermansen K. Effect of meal frequency on blood glucose, insulin, and free fatty acids in NIDDM subjects. Diabetes Care. 1993 Jan;16(1):4-7.

[63] de Verdier MG, Longnecker MP. Eating frequency--a neglected risk factor for colon cancer? Cancer Causes Control. 1992 Jan;3(1):77-81.

[64] Franceschi S, La Vecchia C, Bidoli E, Negri E, Talamini R. Meal frequency and risk of colorectal cancer. Cancer Res. 1992 Jul 1;52(13):3589-92.

[65] Ewe K, Press AG, Bollen S, Schuhn I. Gastric emptying of indigestible tablets in relation to composition and time of ingestion of meals studied by metal detector. Dig Dis Sci. 1991 Feb;36(2):146-52.

[66] Khaw KT, Wareham N, Luben R, Bingham S, Oakes S, Welch A, Day N. Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of european prospective investigation of cancer and nutrition (EPIC-Norfolk). BMJ. 2001 Jan 6;322(7277):15-8.

[67] Moss SE, Klein R, Klein BE, Meuer SM. The association of glycemia and cause-specific mortality in a diabetic population. Arch Intern Med. 1994 Nov 14;154(21):2473-9.

[68] Moss SE, Klein R, Klein BE. Long-term incidence of lower-extremity amputations in a diabetic population. Arch Fam Med. 1996 Jul-Aug;5(7):391-8.

[69] The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group. N Engl J Med. 1993 Sep 30;329(14):977-86.

[70] http://www.reversingdiabetes.org/?cat=hiw&page=testimonies

[71] Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med. 2002 Feb 7;346(6):393-403.

[72] Barnard ND, Cohen J, Jenkins DJ, Turner-McGrievy G, Gloede L, Jaster B, Seidl K, Green AA, Talpers S. A low-fat vegan diet improves glycemic control and cardiovascular risk factors in a randomized clinical trial in individuals with type 2 diabetes. Diabetes Care. 2006 Aug;29(8):1777-83.

[73] Anderson JW, Randles KM, Kendall CW, Jenkins DJ. Carbohydrate and fiber recommendations for individuals with diabetes: a quantitative assessment and meta-analysis of the evidence. J Am Coll Nutr. 2004 Feb;23(1):5-17.

[74] Salmerón J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. JAMA. 1997 Feb 12;277(6):472-7.

[75] J Am Diet Assoc. 1996 Dec;96(12):1254-61. Oat bran concentrate bread products improve long-term control of diabetes: a pilot study. Pick ME, Hawrysh ZJ, Gee MI, Toth E, Garg ML, Hardin RT.

[76] Anderson JW, Gustafson NJ, Bryant CA, Tietyen-Clark J. Dietary fiber and diabetes: a comprehensive review and practical application. J Am Diet Assoc. 1987 Sep;87(9):1189-97.

[77] Liu S, Manson JE, Stampfer MJ, Hu FB, Giovannucci E, Colditz GA, Hennekens CH, Willett WC. A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. Am J Public Health. 2000 Sep;90(9):1409-15.

[78] Jukka Montonen, Paul Knekt, Ritva Järvinen, Arpo Aromaa, and Antti Reunanen. Whole-grain and fiber intake and the incidence of type 2 diabetes. Am J Clin Nutr 2003 77: 622-629.

[79] Suzuki H, Fukushima M, Okamoto S, Takahashi O, Shimbo T, Kurose T, Yamada Y, Inagaki N, Seino Y, Fukui T. Effects of thorough mastication on postprandial plasma glucose concentrations in nonobese Japanese subjects. Metabolism. 2005 Dec;54(12):1593-9.

[80] Sakata T, Yoshimatsu H, Masaki T, Tsuda K. Anti-obesity actions of mastication driven by histamine neurons in rats. Exp Biol Med (Maywood). 2003 Nov;228(10):1106-10.

[81] Holloszy JO, Fontana L. Caloric restriction in humans. Exp Gerontol. 2007 Aug;42(8):709-12. Epub 2007 Mar 31.

[82] Wing RR, Blair EH, Bononi P, Marcus MD, Watanabe R, Bergman RN. Caloric restriction per se is a significant factor in improvements in glycemic control and insulin sensitivity during weight loss in obese NIDDM patients. Diabetes Care. 1994 Jan;17(1):30-6.

[83] Farshchi HR, Taylor MA, Macdonald IA. Deleterious effects of omitting breakfast on insulin sensitivity and fasting lipid profiles in healthy lean women. Am J Clin Nutr. 2005 Feb;81(2):388-96.

[84] Mark A Pereira, Alex I Kartashov, Children's Hospital, Boston, Boston, MA; Linda Van Horn. Reported Breakfast Habits and Incidence of Obesity and the Insulin Resistance Syndrome in Young Black and White Adults: The CARDIA Study Program and Abstracts of the 43rd Annual Conference on Cardiovascular Disease Epidemiology and Prevention: in association with the Council on Nutrition, Physical Activity and Metabolism Circulation 2003;107;e7001-e7039. p. 35.

[85] Hummel M, Standl E, Schnell O. Chromium in metabolic and cardiovascular disease. Horm Metab Res. 2007 Oct;39(10):743-51.

[86] Mertz W. Chromium in human nutrition: a review. J Nutr. 1993 Apr;123(4):626-33

[87] Quraishi I, Collins S, Pestaner JP, Harris T, Bagasra O. Role of zinc and zinc transporters in the molecular pathogenesis of diabetes mellitus. Med Hypotheses. 2005;65(5):887-92.

[88] Simon SF, Taylor CG. Dietary zinc supplementation attenuates hyperglycemia in db/db mice. Exp Biol Med (Maywood). 2001 Jan;226(1):43-51.

[89] Diabetes Care. 2007 Mar;30(3):523-8. Serum zinc level and coronary heart disease events in patients with type 2 diabetes. Soinio M, Marniemi J, Laakso M, Pyörälä K, Lehto S, Rönnemaa T.

[90] Singh RB, Niaz MA, Rastogi SS, Bajaj S, Gaoli Z, Shoumin Z. Current zinc intake and risk of diabetes and coronary artery disease and factors associated with insulin resistance in rural and urban populations of North India. J Am Coll Nutr. 1998 Dec;17(6):564-70.

[91] He K, Song Y, Belin RJ, Chen Y. Magnesium intake and the metabolic syndrome: epidemiologic evidence to date. J Cardiometab Syndr. 2006 Fall;1(5):351-5.

[92] Sharma A, Dabla S, Agrawal RP, Barjatya H, Kochar DK, Kothari RP. Serum magnesium: an early predictor of course and complications of diabetes mellitus. J Indian Med Assoc. 2007 Jan;105(1):16, 18, 20.

[93] Larsson SC, Wolk A. Magnesium intake and risk of type 2 diabetes: a meta-analysis. J Intern Med. 2007 Aug;262(2):208-14.

[94] Nagulesparan M, Savage PJ, Bennion LJ, Unger RH, Bennett PH. Diminished effect of caloric restriction on control of hyperglycemia with increasing known duration of type II diabetes mellitus. J Clin Endocrinol Metab. 1981 Sep;53(3):560-8.

[95] Chipkin SR, Klugh SA, Chasan-Taber L. Exercise and diabetes. Cardiol Clin. 2001 Aug;19(3):489-505.

[96] Hu FB, Sigal RJ, Rich-Edwards JW, Colditz GA, Solomon CG, Willett WC, Speizer FE, Manson JE. Walking compared with vigorous physical activity and risk of type 2 diabetes in women: a prospective study. JAMA. 1999 Oct 20;282(15):1433-9.

[97] Hallgreen CE, Hall KD. Allometric relationship between changes of visceral fat and total fat mass. Int J Obes (Lond). 2007 Dec 18 [Epub ahead of print].

[98] Moore LL, Visioni AJ, Wilson PW, D'Agostino RB, Finkle WD, Ellison RC. Can sustained weight loss in overweight individuals reduce the risk of diabetes mellitus? Epidemiology. 2000 May;11(3):269-73.

[99] Boucher BJ. Inadequate vitamin D status: does it contribute to the disorders comprising syndrome "X"? Br J Nutr. 1998 Apr;79(4):315-27.

[100] Pittas AG, Lau J, Hu FB, Dawson-Hughes B. The role of vitamin D and calcium in type 2 diabetes. A systematic review and meta-analysis. J Clin Endocrinol Metab. 2007 Jun;92(6):2017-29.

[101] Lorenz I. Retrospective study of serum glucose concentration in cattle with mucosal disease. J Vet Med A Physiol Pathol Clin Med. 2000 Oct;47(8):489-93.

[102] Burge MR, Garcia N, Qualls CR, Schade DS. Differential effects of fasting and dehydration in the pathogenesis of diabetic ketoacidosis. Metabolism. 2001 Feb;50(2):171-7.

[103] Jayashree M, Singhi S. Diabetic ketoacidosis: predictors of outcome in a pediatric intensive care unit of a developing country. Pediatr Crit Care Med. 2004 Sep;5(5):427-33.

[104] Andersen H, Jakobsen J. Diabetes mellitus. Curr Opin Neurol. 1997 Oct;10(5):376-80.

[105] Sawka MN, Cheuvront SN, Carter R 3rd. Human water needs. Nutr Rev. 2005 Jun;63(6 Pt 2):S30-9.

[106] Crane, MG and Sample, C: Regression of diabetic neuropathy with total vegetarian (vegan) diet. J Nutr Med 1994; 4:431-439.

[107] http://www.reversingdiabetes.org/?cat=hiw&page=testimonies

[108] Genesis 1:29; 3:18 (NIV). Scripture taken from the HOLY BIBLE, NEW INTERNATIONAL VERSION®. Copyright © 1973, 1978, 1984 International Bible Society. Used by permission of Zondervan. All rights reserved. The "NIV" and "New International Version" trademarks are registered in the United States Patent and Trademark Office by International Bible Society. Use of either trademark requires the permission of International Bible Society.

[109] Exodus 15:26 King James Version of the Holy Bible.