DIABETES: NATURAL CONTROLS AND CURES

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Submitted in Partial Fulfillment for the Degree of Master of Science in Biology, Emphasizing Human Nutrition

October, 1987

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The diabetic state varies over a range of expressions in terms of age of onset, severity of biochemical defect, clinical manifestations, rate of progression, and response to therapy. The basic genotype may not be the same in individuals, but perhaps, as evidence may show, the phenotype may be more controllable than previously thought through proper modes of therapy. There is evidence of two forms of hereditary disease: Juvenile (growth onset) and adult (maturity onset). They are clearly separable by a difference in islet cell function. The juvenile has an absolute deficiency of insulin secretion; the adult has a less clear basis for the relative lack of insulin function. One widely used classification recognizes four stages: (1) Prediabetes, (2) Latent or Subclinical Diabetes, (3) Chemical Diabetes and (4) Overt or Manifest Diabetes.

1. Prediabetes

   This is a theoretical state through which all individuals pass who eventually develop overt diabetes. It implies a hereditary predisposition, applicable only to offspring of two diabetic parents or monovular twins, one of whom is diabetic. Some investigators add the mothers of unusually large babies.

2. Latent Sub-Clinical Diabetes

   This refers to the stage where the glucose tolerance test (GTT) is normal under normal conditions, but is abnormal under stresses such as pregnancy, emotional disturbances, infections and physical trauma. Cortisone administration provokes an abnormal GTT. (Fajans, 1959)

3. Chemical Diabetes

   This is the stage where the patient is asymptomatic, usually has a normal fasting GTT, but has a post-prandial hyperglycemia and a clearly abnormal GTT. This may be transformed to overt disease during periods of stress and excessive weight gain. Perhaps 50-60% of such individuals never develop the overt disease, but are all at high risk. (Fajans, 1969)

4. Overt or Manifest Diabetes

This represents the full-blown clinical symptom complex. Fasting and post-prandial hyperglycemia are regularly present and the patient has metabolic and vascular problems (to be discussed). Many with a mild form of overt diabetes shift back and forth between chemical and overt stages.

TYPES OF DIABETES

1. Pituitary Diabetes - generalized increase in all the anterior pituitary hormones (adrenocorticotropin, throid-stimulating hormones, and prolactin) causes elevated blood glucose concentration. The rate of glucose utilization by the cells is only moderately depressed. Adequate insulin is available, but the problem is failure to utilize it after it enters the cell. Many of the side effects associated with diabetes mellitus are absent. This type is said to be weakly insulin sensitive. (Guyton, 1976, pp. 997-1025)

2. Adrenal Diabetes - characterized by blood glucose concentration 50 percent or more above normal. This has many similarities to pituitary diabetes. (Guyton, 1976, p. 1025) Insulin lowers blood glucose concentration only a moderate amount in adrenal diabetes, not nearly as much as in pancreatic diabetes, but more than in pituitary diabetes. This type is said to be moderately insulin sensitive.

3. Pancreatic Diabetes (Diabetes Mellitus) - true or relative lack of insulin. (Guyton, 1976, p. 1025) This type is strongly insulin sensitive.

4. Diabetes Insipidus - large quantities of dilute urine are excreted due to increased anti-diuretic hormone secretion.

   a. Nephrogenic - failure of the renal tubules (kidney) to respond to anti-diuretic hormone (ADH). (Guyton, 1976, p. 510)

   b. Hypothalamic - tumor of portions controlling ADH secretion. (Guyton, 1976, p. 1002)

   c. Posterior Pituitary (Hypophysis) - the supraoptico-hypophyseal system secreting ADH fails. (Guyton, 1976, p. 1002)

5. Liver Diabetes - The liver is especially important for maintaining a normal blood sugar concentration - that is, the blood glucose concentration. For instance, storage of glycogen (the storage form of glucose) allows the liver to remove the excess glucose from the blood, store it, and return it to the blood when the blood glucose concentration begins to fall too low. This is called the glucose buffer function of the liver. As an example, immediately after a meal containing large amounts of carbohydrates, the blood glucose concentration rises about three times as much in a person with a normally functioning liver. (Guyton, 1976, p. 942)

   In the presence of excess insulin, excess glucose, or both, the liver takes up large quantities of glucose from the blood. In the absence of insulin, or when the blood glucose level falls too low, the liver gives glucose back to the blood. As one can see, the liver acts as an important blood glucose buffer mechanism, helping to keep the blood glucose concentration from going too high or falling too low. If the liver isn't functioning properly, even if all else is going well, one can have too high or too low of a blood concentration of glucose. (Guyton, 1976, p. 1039)

   When excess quantities of glucose enter the blood, about two-thirds of this is stored almost immediately in the liver. This prevents excessive increase in the blood glucose concentration. (Guyton, 1976, p. 1046)

   Therefore, there can be a form of liver diabetes, as the malfunctioning liver can cause an elevated blood glucose.

Symptoms of types 1-3 include coronary heart disease, neuro-muscular conditions, acidosis from fat utilized for energy, slow healing, muscle wasting from protein utilized for energy, elevated blood sugar from insufficient insulin production or utilization, dehydration from osmosis by elevated blood sugar, loss of amino acids and minerals (K^+, Mn^{++}, Mg^{++}, Ca^{++}, CL^-, CR^{++}) through neutralization of ketoacids for excretion by the kidneys and fluid loss, elevated blood pressure, vulnerability to infections because of acidity, atherosclerosis (plaques consisting of cholesterol and fats on blood vessel walls) from hypercholesterolemia, slowed intestinal motility, and osteoporosis. (Guyton, 1976, pp. 1040,42,48,68) (Berkow, 1977, p. 1298) (Krause, 1972, pp. 108,113,397) (Robbins, 1974, pp. 259-63,66,71,72,73) (Douglas, 1975) (Rolate, 1979)

Insulin

Insulin causes blood protein (amino acids), potassium, and sugar (glucose) to be taken from the blood into most body cells, decreases breakdown of fat and protein for energy and, therefore, decreases blood sugar, amino acid and lipid (fat) concentration. It also increases cellular hydration (water content) and promotes fat storage of the cell. (Helgason, 1982) (Orten, 1975, pp. 435-36)

Factors promoting stimulation of insulin release and, in excess, pancreatic exhaustion and diabetes, are the drugs (cortisol, progesterone, estrogen, etc.), amino acids, glucose, gastrointestinal hormones, caffeine, nicotine, and the oral contraceptives. (Guyton, 1976, p. 1043) (Orten, 1975, pp. 435-36)

Overstimulation from continued overconsumption of these can wear out the beta cells from overuse, causing them to under-function and secrete less insulin than needed. (Berkow, 1977, p. 1291)

Alloxan and Streptozotocin

Insufficient insulin production is induced in animals by the chemicals alloxan and streptozotocin. Alloxan is created by oxidation in the bleaching of wheat flour and sugar to make it white by combining chlorine dioxide, Benzoyl peroxide or agene with xanthine in these foods. (Wiley, 1956) (Heilbron, 1954) (Sharp, 1981) (Finar, 1968) Streptozotocin is an N-nitroso compound (Nitrosamine) formed by smoke-curing animal meats. (Helgason, 1982)

Alloxan and streptozotocin cause the beta cells of the Islets of Langerhans of the pancreas, which secrete insulin, to swell and finally degenerate. This decreases or destroys the ability of the cells to secrete insulin. (Guyton, 1976, p. 1047) (Helgason, 1982)

Incidence

Despite our massive medical effort against diabetes, the incidence of this fearsome degenerative disease is steadily increasing. (Levin, 1966) As of 1981, diabetes in the U.S.A. had increased 667% per 1000 population over 46 years despite the use of insulin. (See Table #1) Even if insulin is used to control the disease, it continues to progress. (Meinert, 1970) (Knatterud, 1971)

The lifespan and health of a "controlled" diabetic is way below average. A typical study completed in 1978 charted the lives of 800 diabetic men and 50 diabetic women. All had developed diabetes before age 31. All but 13 were "controlled" with insulin. The patients were matched by age and sex with others. As the disease progressed, nearly all the patients developed serious degenerative complications. By 1976 only 40% survived, compared with 90% of the others. (Deckert, 1979)

Insulin Sensitivity

Insulin sensitivity is decreased by excessive fatty acids (triglycerides) in the blood. (Yalow, 1965) (Solomon, 1968) This may be accomplished by excessive sugar ingestion. (Hallfresch, 1979) Insulin sensitivity is increased by complex carbohydrate ingestion and a high fiber ingestion. High fiber (indigestible carbohydrates) as opposed to low fiber diets significantly reduce blood fat and insulin, although caloric content is the same. (Albrink, 1979) However, it should be noted that the more one cooks a starch, the easier the digestion and absorption, and thus a higher insulin reaction. (Krause, 1972, p. 44)

Hyperinsulinemia

Reduced insulin sensitivity has been postulated to be the primary step that leads to hyperinsulinemia and then hypertriglyceridemia. (Olefsky, 1974) The high level of insulin, which is inhibited from dealing with the sugar, goes to the liver where it is used up by the body in making excess triglycerides. (Farquhar, 1966) Elevated insulin levels in response to glycemic stress have been associated with mild maturity onset diabetes and are considered to be one of the earliest detectible signs of diabetes. (Jackson, 1972)

Hyperinsulinism, Sodium, Potassium & Hypertension

Insulin stimulates the sodium-potassium pump. (Moore, 1973) It does it so much in the kidney, that at about twice the normal blood level, insulin can cause almost total resorption of sodium from the preformed urine back into the blood. (Nizet, 1971) This partly explains why obese people tend to retain sodium in their bodies and thus frequently develop high blood pressure.

Addition of potassium to the diet of people with primary hypertension significantly decreases blood pressure, and also shows a decrease in the level of noradrenalin in the blood. (Frujita, 1983) Therefore, the sympathetic nervous system became less active, since noradrenaline is released from sympathetic nerve endings. Potassium may have an effect upon the sympathetic nerves that go directly to the anterioles and cause dilation of these small resistance arteries. (Tannen, 1983)

Normal Blood Glucose Levels

According to Guyton, normal fasting blood glucose is 80-90 mg/100 ml. (Guyton, 1976, p. 1043)

Growth Hormone and Insulin

Insulin is nearly as essential for growth of an animal as is growth hormone. Furthermore, neither growth hormone nor insulin alone will cause very significant growth. Yet a combination of both will cause dramatic growth. The two hormones do not seem to affect growth by the same mechanism, but by different mechanisms, both of which are necessary. (Guyton, 1976, p. 1042)

Conversely, insulin lack causes extreme wasting of body proteins, with consequent dumping of amino acids and elevated plasma amino acid levels. These amino acids are mainly transported to the liver and used for liver gluconeogenesis (conversion to glucose). This leads to enhanced excretion of urea in the urine. The protein wasting is one of the most serious of all the effects of severe diabetes mellitus, leading to extreme weakness as well as many deranged functions of the organs. Protein wasting and lipolysis also leads to severe weight loss. (Guyton, 1976, p. 1042)

It must also be noted that prolonged administration of growth hormone causes osteoporosis (Guyton, 1976, p. 1043) and diabetes (Young, 1937). Growth hormone is demonstrated in milk and is not destroyed by pasteurization. (Robbin, 1956)

Sucrose and Milk

Wales suggests that milk (growth hormone) and sucrose (carbohydrate) combined are diabetogenic. (Wales, 1977)

Weight Loss and Fasting for Blood Pressure

Loss of only a third to one half of excess body weight has shown to reduce blood pressure significantly. (Eliahou, 1981)

Insulinase

Insulinase breaks down the insulin molecule. Niacin is one of a number of agents that inhibits the production of insulinase. Niacin's ability to reduce insulinase activity gives it a "striking hypoglycemic action" - action to bring down blood sugar level. In her book "Vitamins in Endocrine Metabolism" (Charles C. Thomas, Pub.), Dr. Isabel Jennings states that two other B vitamins - riboflavin and pantothenic acid - also are involved with insulinase breakdown. (Mirsky, 1956)

The amino acid L-tryptophan, copper and zinc also inhibit insulinase activity, causing a relative hypoglycemia. (Mirsky, 1956)

Obesity

Increasing body weight (obesity) and the development of diabetes is well documented. Approximately 80% of maturity onset diabetics are obese and 60% of grossly obese adults have carbohydrate intolerance (usually slightly abnormal glucose tolerance tests). The intolerance tends to return to normal following weight loss, and reduction of body weight in the diabetic is usually followed by reduced insulin requirements. It is postulated that fatty peripheral tissues cause an increased resistance to insulin. (Yalow, 1965) (Solomon, 1968)

Sugar metabolism improves in direct proportion to every pound of fat lost. (Jackson, 1969) Even in confirmed obese diabetics, weight reduction substantially improves their sugar metabolism and lowers their blood triglycerides. (Rudnick, 1965) (Hadden, 1975) (Olefsky, 1974)

Raw Food vs. Cooked Food and Transit Time

"Transit time" is the time it takes food to pass through the digestive tract. A diet of mostly raw food (which is rich in fiber) passes through the intestinal

tract in 18-24 hours. A diet of mostly cooked food takes 80-100 hours. It has been found that some diabetic patients drastically reduce their need for insulin when eating a diet of mostly (50-80%) raw food. Researchers felt the still active enzymes (deactivated when cooked) or the fast transit time of the raw diet caused this reaction. (Douglass, 1975)

Dietary Fat and Transit Time

It should also be noted that dietary fat greatly slows intestinal transit time (Orten, 1975, pp. 435-6) as well as elevating blood fats, and that eating a combined diet of carbohydrate, protein and fat slows the digestive time of each. (Krause, 1972, p. 85) The cellulose combined with protein may cause increased colon putrefaction (anaerobic decomposition) as a result of slowed digestion. (Krause, 1972, p. 92) Cooking softens and ruptures the cellulose cell structure, thus altering it. (Krause, 1972, p. 92) Since uncooked fiber increases speed of transit through the intestines and cooked decreases the time, the altering of the fiber by cooking is the root of the change. The cellulose itself, or the carbohydrate associated with it, or the cooking of it, is what increases intestinal putrefaction when eaten in association with proteins.

Thiamin

Thiamin (Vitamin B1) is involved in intermediate metabolism of carbohydrates inside all cells of the body. However, acid retards the destruction of thiamin and thiamin contributes to the acid reaction, and in diabetes (acid condition) there is already a high blood thiamin level. (Goodhart, 1940) B1 deficiency results in severe damage to the heart and arteries. (Wintrobe, 1943) (Asburn 1944) Elevated serum erythrocyte transketolase indicates thiamin deficiency. (Wolfe, 1958)

Riboflavin and Niacin

Riboflavin (B2) and Niacin (B3) likewise contribute to the acidity of the body by virtue of their hydrogen liberating functions in the electron transport system. (Orten, 1975, pp. 598-602) (Mosler, 1970) (Everhart, 1984) Elevated serum erythrocyte glutathione reductase indicates riboflavin deficiency. (Bayoumi, 1976)

Elevated serum LDH could indicate niacin deficiency since niacin is biologically active in the enzyme NAD, which participates in reactions involving lactate dehydrogenase (lactate to pyruvate). (Brown, 1980)

Riboflavin deficiency in animals causes disorders of glucose tolerance similar to those seen in diabetics. Mice made even moderately riboflavin deficient show large disturbances of blood sugar and blood insulin following a feeding of glucose. Sixty minutes after the feeding, they show the typical pattern of high blood glucose and high blood insulin, which indicates a severe disorder of the mechanism in which insulin deals with sugar in the blood. Riboflavin supplements quickly restore these responses to normal. (Axelrod, 1951) (Reddi, 1979)

Nicotinamide and Nicotinic Acid have both been shown to be diabetogenic in elevated doses. (DiPalma, 1977) Individuals given high levels of Nicotinic Acid or Nicotinamide can get ulcers and GI tract bleeding, reduced blood pressure, increased heart rate, increased blood sugar levels (making them semi-diabetic), toxic liver and can become jaundiced. (DiPalma, 1977) (Mosler, 1970)

Pyridoxine

Pyridoxine (B6) may, as a supplement, aid one in diabetes since it decreases accumulation of xanthurenic acid by helping to completely metabolize the amino acid tryptophan, increases transport of amino acids and potassium into cells against a gradient, and is believed to be involved in the metabolism of unsaturated fatty acids. (Orten, 1975, pp. 602-5) This would seem to reverse several diabetic symptoms. Chemical diabetes brought on by oral contraceptive steroids has responded favorably to B6 supplementation. (Rose, 1978) Carpal tunnel syndrome, which includes painful or tingling hands and is known to be associated with pregnancy, oral contraceptives and diabetes responds well to B6 supplementation. (Folkers, 1978) (Ellis, 1979) (Taylor, 1971)

A 1970 international conference held in Madison, Wisconsin and sponsored by the University of Wisconsin Medical Center and the pharmaceutical house, Hoffmann-La Roche, Inc., was titled "The Biochemistry and Pathology of Tryptophan Metabolism and Its Regulation by Amino Acids, Vitamin B6, and Steroid Hormones." Raymond R. Brown, Ph.D., of the University of Wisconsin Medical Center, who was

chairman of the conference, summarized the findings of the three-day scientific sessions in an article in the American Journal of Clinical Nutrition (February, 1971).

In his summary of the conference, Dr. Brown gave an account of what harm can result when someone with a special need for high levels of B6 fails to receive adequate quantities. Abnormal tryptophan metabolites are found in the urine of the B6 deficient persons - xanthurenic acid being the best known of these substances and the one for which tests are usually made when checking an individual's B6 status. These metabolites, Dr. Brown said, "retard oxidative phosphorylation," which means they interfere with carbohydrate metabolism. They also "form inactive complexes with insulin suggesting a diabetogenic action."

At the joint meeting of the American Institute of Nutrition and the American Society for Clinical Nutrition, held at Cornell University in August 1973, Dr. David Rose reported on controlled tests showing impaired glucose tolerance in oral contraceptive users on a B6 deficient diet - as opposed to non-OC users on the same diet. One of the OC users, Dr. Rose said, had such a severe impairment that her condition was regarded as "chemical diabetes mellitus," an effect "reversed by pyridoxine."

Studies in Australia and Japan show Vitamin B6 concentrations significantly lower in diabetic than healthy people. (McCann, 1977) (Kotake, 1957)

Philpott hypothesizes that deficiency of the amino acid cystine causes a relative B6 deficiency because it is needed to transform B6 into its active form of pyridoxal-5-phosphate. (Philpott, 1983, p. 139)

B6 and Milk

Tomarelli has reported that sterilized milk, canned evaporated milk, or heat-processed milk powder have a substantial amount of the Vitamin B6 content destroyed, and that experimental rats cannot thrive on the milk treated in this way. (Tomarelli, 1955)

B6 and Neuropathy

Of 50 human adults in which B6 deficiency was induced, three developed peripheral neuropathy of a sensory type; motor function was later impaired. (Mueller, 1950) (Vilter, 1953) (Philpott, 1983)

High intakes of protein hasten the onset of B6 deficiency. (Baker, 1964)

Xanthurenic Acid and Homocysteine

Xanthurenic acid is an abnormal metabolite of the amino-acid tryptophan, and homocysteine of methionine. Vitamin B6 clears these metabolites from the system (and consequently the urine). Homocysteine in B6 deficiency has been found to cause arteriosclerosis initiation. (McCully, 1949) (Rinehart, 1949)

Folic Acid

Folic acid (B9) is probably undesirable as a supplement unless in the presence of ample B12, as folic acid uses up B12 to be converted to the citrovorum factor, which stimulates production of red blood cells. (Orten, 1975, pp. 609-12). However, B9 masks neurologic symptoms of B12 deficit (diabetic neuritis?) Philpott found that 47% of his diabetic patients registered low in testing for folic acid. (Philpott, 1983, p. 123). Low folic acid nets Forminoglutin aciduria (FIGLU test). (Wallach, 1984), p. 293)

Fifteen of seventeen patients with arteriosclerosis responded favorably to administration of folic acid; there was increased capillary blood flow and improved vision (believed to be a result of better supply to the retina, thus reversing to some degree the arteriosclerotic condition). (Kopjas, 1966) This could possibly aid diabetic eye symptoms.

P-Amino Benzoic Acid

P-Amino Benzoic Acid forms a part of the folic acid molecule, may have similar properties to biotin, stimulates lactation, and has anti-bacteriostatic effects on sulfonamide drugs. (Orten, 1975, pp. 620-1) As a free-standing supplement, this may not be helpful and may be harmful to a diabetic.

Cobalamin

Cobalamin (B12) is probably desirable as a supplement in diabetes because of diabetic neuritis and anemia, but may be of no effect as it is easily destroyed in acid conditions. (Orten, 1975, pp. 612-14) Low cobalamin nets methylmalonic aciduria. (Wallach, 1984, p. 293) B12 is needed for normal folic acid metabolism, so the FIGLU test is not a reliable test for B12. (Wallach, 1984, p. 293)

Inositol

Inositol has been found to be curative on fatty livers produced by administration of biotin. (Orten, 1975, p. 620) Salway and Finnegan gave inositol in doses far in excess of the RDA at 500 mg twice daily to seven diabetic patients who had peripheral nerve damage as a result of diabetes. This regime led to significant changes in the ability of the patients' nerves to conduct messages and it was concluded to be valuable for the purpose. (Salway, 1978) The normal concentration of inositol in nerves of the human body is fifty times what it is in the blood. (Gould, 1976)

Myo-Inositol

Diabetic patients and rats with experimentally induced diabetes show high myo-inositol levels in blood and urine. (Palmano, 1977) (Clements, 1977) (Anderson, 1976) (Winegrad, 1976) It is not clear whether these high levels are the results of hyperglycemia and competition with glucose for renal excretion, a possible dependence of myo-inositol on insulin for cell entry (Clements, 1977), or decreased catabolism of inositol by the kidney. (Melmed, 1974) (Palmano, 1977) (Clements, 1977) Recent evidence supports the theory of decreased inositol catabolism as a primary cause. (Melmed, 1977)

Pantothenic Acid and Biotin

Elevated doses may not be desirable as supplements, as they function in Coenzyme A, which acts in the biosynthesis of fatty acids. Biotin causes fatty liver in excess amounts. (Orten, 1975, pp. 605-9, 20)

Ascorbic Acid

Ascorbic acid (Vitamin C) is easily oxidized to its dehydro derivative and contributes to acidity. This would suggest Vitamin C supplementation in diabetes in ascorbic acid form be tempered with Buffered Vitamin C (the alkaline form of Vitamin C). It also converts folic acid to its physiologically active form of tetrahytrofolic acid and hyroxylates cholesterol to cholic acid. (Orten, 1975, p. 593)

Guinea pigs maintained on very low levels of Vitamin C developed degeneration of the Islets of Langerhans in the pancreas. (King, 1936) These guinea pigs demonstrated an extremely low sugar tolerance level, which was rapidly regained upon feeding high levels of Vitamin C. (Banerjee, 1943) (Banerjee, 1964) The pancreas' production was reduced to one-eighth that of normal guinea pigs. Other studies show deprivation of Vitamin C causes guinea pigs to be unable to convert glucose to glycogen for storage in their livers. (Altenburger, 1936)

Humans, including juvenile diabetics, have been shown, upon administering high amounts of Vitamin C, to have improved action of insulin, with lower levels of insulin being needed to control sugar metabolism. (Rogoff, 1944) (Pfleger, 1937)

The greater the deprivation of Vitamin C, the more cholesterol accumulates in our bodies, and administration of Vitamin C lowers cholesterol levels in humans. (Sokolaff, 1966) Localized ascorbic acid depletion often exists in segments of arteries afflicated with atherosclerosis. Adjacent arterial segments without lesions had a higher ascorbic acid content, and atherosclerosis was rare in these arteries. (Willis, 1955)

Chronic feeding of high acid-producing food (wheat) to humans and guinea pigs increased dehydro-ascorbic acid (the oxidized form of absorbic acid) with the emergence of a parallel hyperglycemia, and a corresponding lowering of ascorbic acid. (Chatterjee, 1975) Milk's effect on the acidity or alkalinity of urine is controversial. (Krause, 1972, p. 485) Pharmacological doses of dehydro-ascorbic acid are reported to be diabetogenic. (Patterson, 1950) This also degenerates the beta cells of the pancreas. (MacDonald, 1956) Serum dehydro-ascorbic acid is continuously elevated in patients with diabetes mellitus. (Chatterjee, 1975)

Vitamin C and Uric Acid

Ascorbic acid supplementation at levels of 4 grams or more have shown to significantly decrease (1.2-3.1 mg %) serum uric acid levels. (Stein, 1976)

Uric Acid and Heart Disease

Blood uric acid has been reported to be elevated in patients with heart disease. It has been valuable in prediction of cardiovascular mortality, but there is no known basis for a causitive role of uric acid in cardiovascular disease. (Jacobs, 1972) (Srivastava, 1974) (Takkunea, 1977)

Sucrose, Fructose, and Uric Acid

Serum uric acid was found to be significantly higher in twelve young women when they consumed a diet containing 43 percent of calories from sucrose as compared to starch. Also, ten men and nine women showed significantly higher fasting serum uric acid when they consumed a diet containing 30 percent of the calories from sucrose as opposed to starch. The fructose moiety of sucrose appears to be responsible for the increase in blood uric acid. (Kelsay, 1977) (Solyst, 1980) (Emmerson, 1974)

Vitamin A

Diabetics cannot convert beta-carotene into Vitamin A. (Davis, 1952)

Serum values of Vitamin A correlates fairly well with both dietary intake and stores in the liver. Lower limits for adults are 200 ug/litre and 300 for children. Serum concentrations of 100 ug/litre or less are an indication of definite deficiency as well as severe depletion of liver stores. (Sauberlich, 1974)

Vitamin E, Gangrene and Insulin Requirements

Tolggs showed that about 50 percent of patients with gangrene resulting from arteriosclerosis, diabetes and Buerger's Disease, were saved from amputation by Vitamin E. (Tolggs, 1957) Shute found that 25% of clinical diabetics on insulin

had a decrease of insulin requirement of 10 units or more when given Vitamin E. (Shute, 1973) Vogelsang also found insulin-sparing action. (Vogelsang, 1949) Vitamin E also helps maintain normal levels of blood fats. (Hermann, 1979) Vitamins A, E, C and selenium work synergistically in many instances. (Ames, 1969) (Ayres, 1979)

Vitamin E and PUFA

Polyunsaturated fatty acids, particularly linoleic acid, are easily oxidized and increased ingestion requires greater amounts of antioxidants in the diet, particularly Vitamin E. (Harris, 1963) (Horwitt, 1961)

Vitamin E

Low plasma concentration of -tocopherol and increased urinary creatine excretion is a usual laboratory finding in Vitamin E deficiency. (Sauberlich, 1974) The normal serum concentration of Vitamin E in serum is 0.8 to 1.1 mg/dl. (Bunnell, 1971)

Calcium and Insulin

Calcium may influence insulin secretion while calcium deficit causes decreased insulin secretion. (Gerich, 1976) (Gedik, 1977)

Calcium-Magnesium

Increasing the magnesium intake in humans, particularly when there are adequate levels of calcium, actually improves calcium utilization. (Henrix, 1963) High protein, low carbohydrate ingestion results in increased urinary calcium, phosphorus, iron, zinc and magnesium losses. (Pritikin, 1976)

High calcium, protein and Vitamin D intakes all function to increase the requirements for magnesium; therefore, it seems these as supplements in high amounts would not be desirable in diabetes. (Krause, 1972, pp. 108-9) Cow's milk fortified with Vitamin D has much more calcium, protein and Vitamin D than human milk. (Orten, 1975, pp. 695-704)

Magnesium

Magnesium is an essential mineral needed as an activator for enzymes of carbohydrate metabolism and of amino acid metabolism. Deficiency is seen in diabetes, neuromuscular conditions, and makes animals considerably more susceptible to atherosclerosis. (Krause, 1972, pp. 108-9)

Laurendeau observed a lower level of magnesium in the hearts of patients with myocardial necrosis, and was more marked in diabetic subjects. (Laurendeau, 1963)

Magnesium activates over 50 percent of the enzymes in the body including six of the nine glycolytic enzymes. With decreased levels of magnesium, the metabolic machinery of the body cannot function optimally. (Harper, 1973)

Lithium

Van der Velde observed that patients treated for depression with lithium had a definite improvement in their glucose tolerance levels, if before the treatment they were impaired. (Van der Velde, 1969)

Choline and Lecithin

Choline, a constituent of the lecithins, was discovered in the course of work on diabetic animals and cures fatty liver and several other factors associated with diabetes. (Orten, 1975, pp. 618-20)

Chromium

Sugar contains almost no chromium, and usually leads to a loss of body chromium because of the depleting effect of glucose. Upon feeding pure glucose or sucrose, blood sugar is elevated, insulin then elevates, then serum chromium increases. The chromium then travels to the kidneys where between 20 and 30 percent is excreted. (Glinsman, 1966) Also, diabetic individuals excrete chromium more rapidly in their urine than non-diabetics. (Doisy, 1971)

Chromium acts at the cell membrane facilitating the entry of glucose into the cells. Chromium is ineffective without insulin and the effectiveness of a given dose of insulin can be enhanced by 50-100 percent by the addition of chromium to the system. Chromium has been postulated to act as a disulfide bridge between the A-chain of insulin and membrane sulfhydryls (allowing insulin to be effectively bonded to the membrane of the target tissues). (Mertz, 1967)

Foods have been tested for both chromium content and biological value. For the latter test, tissue of lab animals was treated in the presence of insulin with measured quantities of a selected food, and an insulin-triggered reaction known to require chromium participation was measured. Though certain foods had a higher concentration of chromium (i.e., egg yolks and oysters), the amount of chromium with biological value was many times higher in brewer's yeast than any other food. On a chart of biological values, dried brewer's yeast rated 44.88, black pepper 10.21, calves liver 4.52, American cheese 4.39, and wheat germ 4.05. (Mertz, 1973)

It should be noted that chromium is easily lost in the milling of grains and the processing of foods. Dr. Armand Jeejeebhoy caused signs of diabetes to vanish by introducing chromium into a patients' system. (Jeejeebhoy, 1977)

Mertz reports that pure chromium as it appears in food, water or nature is not an accurate measure of dietary chromium, as this inorganic chromium is poorly absorbed in the human body - as little as 3 percent. (Mertz, 1974)

Twelve elderly patients with impaired glucose tolerance were given brewer's yeast extract. Half regained a normal ability to metabolize blood sugar within two months. (Doisy, 1974)

To determine the possible role of chromium in the treatment of hypoglycemia, researchers at USDA's Human Nutrition Research Center in Beltsville, Maryland studied eight hypoglycemic women. Insulin binding to red blood cells increased and remained elevated throughout the study. The number of insulin receptors also improved. (Anderson, 1985)

The Glucose Tolerance Factor

Dr. Walter Mertz of the U.S. Department of Agriculture found some 28 years ago that a substance is secreted from the liver in response to a meal which sensitizes tissues to insulin. He termed it "the glucose tolerance factor." (Mertz, 1959)

The only type of chromium presently thought to be biologically active in the human body is trivalent chromium. This form is commonly referred to as GTF (glucose tolerance factor). In this form, chromium is bound together with two niacin molecules (Vitamin B3) and three separate free amino acids: Glutamic acid, glycine, and cysteine. This natural chromium complex is much better absorbed and utilized than are the single chromium salts. (Mertz, 1971)

Manganese

The role of manganese in glucose metabolism is the least understood. Biochemically, it is important to the functioning of isocitric dehydrogenase, an important control enzyme in the regulation of the Krebs cycle. (Harper, 1973) Rats deficient in manganese exhibit severely diabetic glucose tolerance curves which revert to normal with the inclusion of manganese in the diet. (Everson, 1968)

Manganese deficient animals frequently produce offspring with pancreatic abnormalties or without a pancreas. In humans, 122 diabetics, ranging in age from 15 to 81, were examined and the manganese content of the blood was one-half that of normal individuals. The longer a patient had diabetes, the lower his manganese blood level. In many countries, plant extracts which are good sources of manganese have been used as home remedies in diabetes...such as blueberry, onion, brewer's yeast, and garlic. (Everson, 1968) (Everson, 1967) Manganese may play a small role in man. (Nutrition Reviews, 1968)

Potassium and Insulin

Potassium deficit causes glucose intolerance. (Conn, 1965) (Rapoport, 1964) Potassium is known to be involved in glucose entry into the cell. The insulin dependent glucose cellular pump requires potassium for normal functioning. (Schwartz, 1971)

Zinc

Zinc supplementation has been shown to improve arteriosclerosis, and diabetics actually excrete more zinc in their urine than do normal subjects. (Henzel, 1971) (Pidduck, 1970) Cadmium displaces zinc and interferes with certain enzyme systems requiring zinc. Higher ratios of zinc to cadmium are preferable because excess zinc prevents accumulation of cadmium - a slight deficiency allows it. Dairy products and butter have a very low zinc to cadmium ratio in relation to most other foods. (Schroeder, 1973)

Zinc is involved in the granulation and storage of insulin in the beta cells of the pancreas. It has also been established that the defect in dysinsulinemia, a pre-diabetic state, is one of the beta cells being unable to store and granulate insulin. (Bollin, 1964) (Zilva, 1972)

Zinc, Exercise and HDL Cholesterol

Physical activity levels were assessed for each individual in a study and were found to correlate with serum HDL cholesterol levels only in the third of subjects with the lowest Zn intake. HDL cholesterol levels tended to increase most in subjects with higher activity levels, and they remained constant or decreased in several people with low and moderate activity levels. Thus, moderate levels of Zn supplementation may offset exercise-induced increases in HDL cholesterol. (Goodwin, 1985)

Exercise

When prediabetics and diabetics exercise consistently, body fat and blood fats go down, and the insulin mechanism recovers. Even if no body fat is lost, 20 to 30 minutes of aerobic exercise a day causes blood fats and cholesterol to decline and glucose tolerance to improve. There is a beneficial reduction in blood glucose even with only three months of light exercise. (Calstrom, 1964) (Vranic, 1979) (Streja, 1979)

Amino Acids/Protein

Most, if not all, of the amino acids, stimulate insulin secretion. The most potent of these amino acids is alanine. Amino acids administered in the absence of a

rise in blood glucose cause only a small increase in insulin secretion. However, administered concurrent with elevated blood glucose concentration, glucose-induced secretion of insulin may be as much as doubled in the presence of excess amino acids. Therefore, the amino acids very strongly potentiate the glucose stimulus for insulin secretion. Normally, insulin rises rapidly in response to a rapid rise in blood glucose concentration, and falls rapidly with a rapid fall in blood glucose. (Guyton, 1976, p. 1043)

It is essential that glucose precursors (especially alanine) be delivered to the liver in adequate concentrations, especially when hepatic glycogen stores have been depleted and when gluconeogenesis is responsible for an increased proportion of the total hepatic output of glucose. (Felig, 1977) Regardless of the relative contributions of glucagon excess and/or insulin deficiency, the data indicates that protein feeding exaggerates the glucose over-production and hyperglycemia of the diabetic state. (Felig, 1977)

Histidine

Increased platelet aggregation has been observed in conditions with high risks of thromboembolic complications, including diabetes mellitus, progressive atherosclerosis and recent myocardial infarction, and neoplasms. L-histidine appears to be an effective inhibitor of spontaneous platelet aggregation. (Steinhauer, 1985)

Arginine

In rats fed Arg-deficient diets, insulin release in response to a glucose challenge was suppressed and glucose tolerance impaired. (Visck, 1985)

Other Amino Acids

Glutamic acid, glycine and cysteine - see also section on glucose tolerance factor of chromium.

High Protein Diet

A diet above 16% of caloric intake in protein causes the body to lose stores of minerals, and as protein increases, so does the mineral content of the urine. (Pritikin, 1976) Cow's milk has nearly three times the protein content of human milk. (Kraus, 1972, p. 255) The American Diabetic Association Diet is 43% carbohydrate, 23% protein, 34% fat, and because of the foregoing, has been heavily criticized as being of small importance. (Kiehm, 1976) (Pritikin, 1975) (West, 1973) A high protein diet also causes a diuretic effect, causing loss of water (dehydration), exacerbating diabetic symptoms - because it increases in the blood a byproduct of fat metabolism called ketones.

Dietary, Caloric, Fat and Carbohydrate Requirements

In 1923 Geyelin began to prescribe high-carbohydrate diets to diabetic patients treated with insulin. In 1935, after reviewing 10 years of his experience with the high-carbohydrate, low-fat diet, he concluded that the increased effectiveness of insulin that consistently followed the institution of a high-carbohydrate diet was "chiefly dependent on the degree to which fat is curtailed." (Geyelin, 1935)

Rabinowitch was another advocate of the high-carbohydrate, low-fat diet. His goal was to keep his patients 5 to 10 pounds under their average body weights. He suggested that even less fat be included in the diet than previous observers had advocated (i.e., less than 50 gm a day) and believed that the restrictions of dietary fat would help lower the incidence of cardiovascular-renal disease. He also observed that "potential diabetes can be activated and mild diabetes can be made severe by too rigid restriction of carbohydrate." (Rabinowitch, 1935)

Himsworth (Himsworth, 1934) demonstrated that diets high in carbohydrates improved glucose tolerance in normal persons when caloric intake was controlled. These observations of the effects of high-carbohydrate diet on oral glucose tolerance in normal subjects have been confirmed by others. (Wales, 1967) (Ford, 1968) (Anderson, 1968)

A number of dietary surveys have been made in conjunction with studies of the prevalence of diabetes. The Zulus consume about 85 percent of their calories as carbohydrate but they are thin and virtually free of diabetes. In the same area of the world, the Masai, who live on essentially raw meat, milk and blood, and eat only 20 percent of their calories as carbohydrate, also remain thin and are spared from diabetes. (Cleave, 1969)

Various American Indian tribes who have remained relatively homogeneous have a high incidence of abnormal glucose tolerance and clinical diabetes. (Henry, 1969) Like those of other low-income Americans, their diets are high in carbohydrate and fat with the result that over half the population exceed 125 percent of optimal weight. The diabetes is the maturity-onset type and is associated with a high incidence of large-vessel disease. Conversely, Mauratoff et al (Mouratoff, 1969) found two groups, the Eskimos and the Athabaskan Indians in Alaska, who have been spared from diabetes and atherosclerosis on a high fat, low-carbohydrate diet (less than 50 gm per day). Both of these groups have a low incidence of obesity. However, these diets were high in marine lipids EPA and DHA, and essentially raw.

The Indians in India have a low incidence of diabetes on a high-carbohydrate, low-calorie diet. However, those who migrated to South Africa and ate the same high-carbohydrate diet - but at a higher caloric intake - had a high incidence of maturity-onset diabetes and vascular disease. The Yemenite Jews who migrated to Israel and adopted the European diet high in calories and carbohydrates in place of their former diet (a high-fat, low-caloric diet) now have an increased incidence of diabetes and obesity. (Cleave, 1969)

Albrink and Davidson (Albrink, 1977) feel that the most important fact that emerges from clinical and epidemiologic studies is the deleterious effect of obesity on diabetes. They indicate that the fewest vascular complications are found when the habitual dietary intake results in leanness, regardless of the proportion of carbohydrate or fat in the diet.

Carbohydrate intake in the control of the plasma lipid levels was noted by Stone and Connor. (Stone, 1963) They increased the carbohydrate intake from

40 to 65 percent, together with other manipulations of the diet, and noted improvement in the hyperglycemia, hypertriglyceridemia and hypercholesterolemia in their patients.

The high complex carbohydrate, high-fiber (HCF) diet as researched by Dr. James Anderson and his co-workers has done wonders to reduce and eliminate insulin need. It consists of caloric intake low in simple sugars, 75 percent high-residue fibrous complex carbohydrates, 16 percent protein, and 9 percent fat. (Kiehm, 1976)

Glycemic Index

Dr. David Jenkins, an associate professor of nutrition and medicine at the University of Toronto, has recently formed a "Glycemic Index" which shows that complex and simple carbohydrates have variable blood sugar absorption rates, not necessarily correlated with their complexity. For example, potatoes (complex carbohydrate) deliver sugar into one's blood stream as fast as a Mar's bar, but peanuts are very slow (probably owing to the oil content). Phyllis Crapo of the University of Colorado, Health Science Center, found that pure uncooked starch gave virtually no rise in blood glucose, but cooked gave dramatic differences between different foods, and it was unpredictable how each food would be reacted to. (Kolata, 1983) Keeping with the context of this paper, I corresponded with Dr. Jenkins and found that all foods in his experiments were cooked, which would differ with raw foods because of factors related to state of the fiber content previously discussed in this paper (see correspondence letter).

Foods having hypoglycemic effects act as insulin substitutes. These include: Immature bean pods, olive leaves, celery, potato (raw), blackberry leaves, sugar beet, leaves and roots of banana, cashew, carrot, ginseng, burdock, sunflower, senna, Jerusalem artichoke, lettuce, dandelion, cabbage, turnip, papaya, spinach, sweet potato, wild cucumber, cranberry, pea, coconut, fenugreek, onion, garlic, lily-of-the-valley, all-spice, olive, oats, barley, corn, black cohosh, golden seal, red pepper and ginger. (Farnsworth, 1971)

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September 15, 1983

Alan R. Bonebrake D.C.
Prairie Village Shopping Center
6109 E 13th & Woodlawn
Wichita, Kansas

Dear Dr. Bonebrake:

Thank you for your letter. The foods were cooked. I hope your paper is successful.

With Good Wishes.

Sincerely,

David J.A. Jenkins
Associate Professor

DJAJ/mmh

Mono and Disaccharides (Sugars)

Human studies show greater insulin responses following the feeding of glucose or corn syrup with a dextrose equivalent of forty-two than after the feeding of sucrose. (MacDonald, 1978) (Thompson, 1978) The simple sugars (glucose, fructose, etc.) which are monosaccharides or disaccharides are absorbed directly from the small intestines and either used as energy after absorption into the body cells, converted to glycogen in the muscle or liver cells, or converted into triglycerides in fat tissue or liver cells, which send the triglycerides through the blood to peripheral tissue, thus elevating serum triglycerides.

Glucose moderately promotes fat production and elevated blood fat, but fructose does much more so by stimulating much more fat conversion in the liver. Sucrose (table sugar), which is glucose combined with fructose (a disaccharide), can produce what is called "the disaccharide effect" - their combination will elevate blood fats more than either separately. (Thompson, 1979) Moreover, feeding simple sugars (especially sucrose) increases both blood sugar and insulin levels because insulin sensitivity is decreased (that is, the insulin is there, but cannot function properly). (Hallfresch, 1979)

Sucrose

Sucrose may be an etiological factor in genetically predisposed diabetics, causing hyperinsulinemia, high fasting blood sugar, peripheral insulin resistance and retinal and renal vascular complications in genetically selected rats. (Cohen, 1974) Weight gain induced in humans by the intake of excess calories increased serum triglycerides when sucrose, as opposed to starch, consumption was also increased. (Naismith, 1974) Rats show greater insulin response to sucrose as compared to glucose. (Norden, 1971)

Sucrose and Triglycerides

In human studies in which the intake of sucrose has been either eliminated or reduced, significant decreases in fasting serum triglycerides occurred. (Roberts, 1973) (Rifkind, 1966) (Mann, 1970)

Dietary Fats

Low fat diets improve the insulin mechanism and reduce the need for insulin medication. (Conner, 1963)

Fasting

"Starvation in obese patients causes considerable improvement in carbohydrate tolerance unrelated to actual weight loss. Starvation may activate mechanisms which promote nitrogen retention." (Jackson, 1969)

Fasting decreases blood pressure in only 3-4 days in almost all obese people. (Mineur, 1983) This is long before any sizeable loss of weight, indicating high blood pressure is not due to the mechanical effects of body fat. While caloric intake was restricted, sodium excretion in the urine was significantly increased.

Insulin, Weight Loss and Blood Pressure

Insulin also affects blood pressure by acting on the hypothalamic area of the brain, causing it to accelerate the activity of the sympathetic nervous system. (Rowe, 1981). It then elevates blood pressure by causing constriction of arterioles. Weight loss also results in a fall in plasma levels of insulin, noradrenalin, renin, angiotensin II, and aldosterone - all hormones that tend to increase blood pressure. (Marks, 1985)

Meals: Number and Quantity

When one eats carbohydrate is important. Prediabetic patients were given their carbohydrate in four equal small meals per day. Blood sugar levels were the same as normal persons used as controls. Given the same amount of carbohydrate at one sitting, their blood sugar rose into the danger zone. (Deckert, 1979)

Salt and Size of Meal

Australian researchers at the University of Sydney report in the British Medical Journal (292:1697, 1986) that ingestion of salt with a starchy meal causes an

increase in the height and duration of the blood sugar curve following the meal. Normally, they say, the larger the meal (starchy), the higher the blood sugar rises and the longer it takes to fall back down. With salt, one need not increase the amount of starch in the meal to accomplish this. They conclude that salt either boosts digestion of food in the intestine so that more sugar is released from it, or it stimulates the intestine to absorb sugar more efficiently. It is also possible that the salt may have inactivated insulin or suppressed its production.

Arterial Disease

Dr. Rachmiel Levine of the City of Hope Medical Center in Duarte, California, writing in the Journal of the American Geriatrics Society (November, 1971), estimated that "seventy percent of deaths in diabetic patients are the direct result of vascular (blood vessel) disease. This is about two-and-a-half times the proportion of deaths from vascular disease among the non-diabetic population of all ages."

Dr. Levine pointed out that the vascular problems exist on every level from the capillary system all the way up to plaque formation in the aorta. "The introduction of insulin led to an enormous rise in the life expectancy of diabetic patients and has, therefore, revealed the width and extent and severity of the vascular lesions," he wrote. "For diabetes, insulin has shifted the center of gravity from glycosuria (presence of glucose - or blood sugar - in the urine) and coma to the heart, the limbs, the kidney, and the eye," in Dr. Levine's opinion.

Atherosclerosis

Atherosclerosis is not only caused by elevated blood fats, but also by eating overcooked foods or foods cooked in the presence of oxygen (which converts cholesterol from a non-toxic to toxic forms) (Pollak, 1958) (Taylor, 1979) (Peng, 1979), chlorine used in water supplies and to clean milk containers (Price, 1976), xanthine oxidase from homogenized milk (which also damages blood vessel walls) (Zikakis, 1974), and milk containing butterfat (Hepner, 1979).

Since diabetics are particularly susceptible to atherosclerosis and its complications, it seems prudent to consume a diet that will favor the reduction of serum cholesterol and triglycerides. Limiting the saturated fat and cholesterol in the diet has been shown repeatedly to cause a fall in serum cholesterol even in the absence of weight loss. (Stone, 1963) (Van Eck, 1960) (Kempner, 1958)

The arterial wall is an insulin-sensitive tissue and responds to high levels of insulin with the development of lipid-filled lesions. (Stout, 1977) Diabetics who live in underprivileged areas have less macroangiopathy than others in the United States or Canada. (West, 1972) They usually live on a high-carbohydrate, low-saturated-fat diet which suggests that the major problem is the saturated fat. (Tsuji 1971)

The atherosclerotic plaque may be reversed by a low fat diet (Brandt, 1977), a diet enriched with alfalfa (Malinow, 1978), or impaired by a diet enriched with garlic or onions (which inhibits blood platelet aggregation and adhesion), or cold-water fish or oil from the fish (especially cod liver oil). (Dyerberg, 1975) (Makhyd, 1979) (Dyerberg, 1978) (Siess, 1979)

Blood Fats/Hypertriglyceridemia

High levels of blood fats interfere with the ability of the body to use insulin. If one lowers them, the body's use of insulin returns to normal. (Baqdade, 1967)

Controlled hypertriglyceridemic patients consumed greater amounts of sucrose, alcohol, and total calories than controlled normo-triglyceridemic patients. Tendency to overweight also contributed to the hyper condition. (Maruhama, 1977)

Fiber

Anderson found that fiber in the form of pectin, guar, and certain polysaccharides extracted from legumes lower blood cholesterol levels as much as 80 percent in rats. (Anderson, 1979) He also showed a 63 percent triglyceride level reduction in experimental subjects maintained on a high complex carbohydrate, high fiber diet. (Anderson, 1978)

A variety of dietary fibers from potato and other tubers are capable of normalizing blood glucose. (Doi, 1979) High fiber diets, as opposed to low fiber diets, show lowered carbohydrate sensitivity. Increased insulin sensitivity is also induced. (Albrink, 1979) (Jenkins, 1929) (Wigand, 1979)

Oat Bran Fiber

Geriatrics (41 #8:28, 1986) reports that oat bran reduces blood levels of cholesterol (even in diabetics) up to 15 percent. Firstly, it stimulates the liver to include more acid (produced by tearing down cholesterol) in the bile juice secreted into the intestine. Secondly, oat fiber (water soluble) is broken down in the intestine into chemical fragments which, after being absorbed, inhibit cholesterol production by the tissues.

Garlic and Onion

Garlic and onion, though a little slower in action, is as effective as Tolbutamide (an oral drug for diabetics) in clearing the bloodstream of excess glucose. (Jain, 1973) Garlic and onion have preventive effects on fat-induced hyperlipemia, each of which increases fibrinolytic activity. (Bordia, 1973)

Dessicated Liver

Blotner and Murphy, in studies made at Peter Brigham Hospital in Boston, showed that dessicated liver could be used as a substitute for insulin without the side-effects. (Blotner, 1929)

Alcohol

Alcohol use tends to magnify risk factors for coronary heart disease by increasing blood pressure and body weight and impairing glucose tolerance. (Eichner, 1985)

Reactive Hypoglycemia

This form of low blood sugar shows itself some 2 to 6 hours after a meal. It may be associated with excessively rapid emptying of the stomach after a meal and may indicate the early warning stages of the maturity onset form of diabetes. If not managed properly, it may develop into the maturity onset form of diabetes. The suggested therapy is a diet low in simple sugar and high in complex carbohydrate and fiber. (Johnson, 1980) (Leichter, 1979)

Provocative Food Testing

Potts found that two-thirds of the insulin-dependent adult-onset diabetics did not need insulin after they had withdrawn from their maladaptive reactive substances. Those still using insulin required only one-third the amount used before their work-up. (Potts, 1977) (Potts, 1980) (Potts, 1981)

Seven poorly controlled obese adult onset diabetics fasted four days while being monitored. During the fast, the blood sugar levels of these patients decreased to a normal or near-normal level. Provocative testing which followed revealed sensitivities to a number of both carbohydrate and protein foods which elevated the patients' blood sugar levels. When the patients avoided the incriminated foods, their insulin requirements decreased considerably, four of the patients being able to get along without any insulin. Results suggest that avoidance of incriminated foods may favorably influence the dietary management of diabetes. (Potts, 1977)

Bicarbonate

Randolph showed the therapeutic value of using sodium and potassium bicarbonate to reduce provocatively evoked symptoms. (Randolph, 1976)

Generalized Pancreatic Deficiency

Frier has evidence that diabetes mellitus is a "generalized pancreatic deficiency," meaning that in the diabetic state, the pancreas' production of bicarbonate is the most deficient, followed by its secretion of proteolytic enzymes, as well as insulin production. (Frier, 1976)

Exocrine Pancreatic Function

Deficiencies in the exocrine function of the pancreas have long been associated with endocrine insulin deficiencies. Abnormal exocrine pancreatic function has been reported in patients with juvenile-onset and maturity-onset diabetes by many investigators. (Baron, 1973) (Bock, 1967) (Domschke, 1975)

Graham found, while supplementing pancreatic exocrine insufficiency, that other factors such as a positive nitrogen balance, a return of a sense of well-being, and a stabilization and/or reduction in insulin requirements were often accomplished. The therapy was pancreatic enzymes, bicarbonate, and amino acids. (Graham, 1977)

Chlorophyll and Pancreatitis

Trypsin (Beck, 1964) and enterokinase (Mann, 1979) can produce pancreatitis. Chlorophyllin (Oda, 1971) has antitrypsin activity. Enterokinase (Yamashima, 1956) (Kunitz, 1939) converts trypsinogen to trypsin. Pancreatic edema and hemorrhage are the result of vascular dilation and increased capillary permeability brought on by activated kinins. Free trypsin in the pancreas releases kinins. (Mann, 1979) Hyperamylasemia and pancreatitis were prevented by chlorophyll-a (the natural form of active chlorophyll). (Mann, 1979) Chlorophyll-a is converted to chlorophyllin in vivo. (Oda, 1971) Diabetes stemming from this source can be prevented.

Chlorophyll and Suppurative Disease

Chlorophyll has a bacteriostatic effect on anaerobic infections, such as streptococcus hemolyticus. (Gruskin, 1940) Further, it helps stimulate the production of connective tissue so as to aid the repair of the tissue affected. (Gruskin, 1940) Chlorophyll is the green coloring matter of plants, and is present in all growing vegetable cells: The photosynthesis of starch from CO_2 does not proceed in the absence of chlorophyll. (Gruskin, 1940) This could deal with diabetics being prone to infections.

Myrtillin

In the November 5, 1927 issue of the Journal of The American Medical Association, Dr. Frederick M. Allen describes the beneficial role of myrtillin: "Myrtillin was found to reduce alimentary glycosuria and hyperglycemia in normal dogs, to reduce glycosuria and prolong life in depancreatized dogs, and to reduce or abolish glycosuria in diabetic patients. Our experience to date indicates that myrtillin tends to stabilize the blood sugar, which otherwise fluctuates wildly, and that it spares insulin. It never causes hypoglycemia."

Myrtillin is found in all green plants, but most abundantly in blueberry or huckleberry leaves and various myrtles. It is also present in yeast and oatmeal.

Hypochlorhydria and Achlorhydria

These are reportedly quite common in diabetics. (Richardson, 1978) (Rabinowich, 1949) Therapeutic use of HCL has relieved some cases of diabetic neuropathy. (Rabinowich, 1949) The reduced exocrine pancreatic output in some diabetics might result from a lack of acid stimulation from the stomach. If that were the case, then acid, not alkali, would be the treatment of choice.

Neuropathy

See Inositol, B6 and Neuropathy, Folic Acid, and Cobalamin.

CONCLUSIONS

Several factors are involved with the health of the diabetic and potential diabetic. Several methods can be used to prevent the onset of diabetes, improve the health of the diabetic, and reduce insulin needs of the diabetic.

By way of prevention and cure, one should eat a high amount of raw foods with a high fiber content. Diet should be high in complex carbohydrates, low or void in simple carbohydrates. Diet should include sources of trivalent chromium,

choline and healthy liver. Overt diabetics should take additional Vitamin A. Diet should not contain alcohol, excessive protein or saturated fat. Diet should not contain excessive unsaturated fat. Meals should be small and many rather than large and few, and contain several green leafy vegetables. Meals should be void of processed foods deprived of fiber, and vitamins and minerals. One should avoid drugs promoting pancreatic exhaustion and diabetes.

One should eat cooked foods having a low glycemic index (which are slowly absorbed). One should eat foods liberally which have a hypoglycemic effect (except in individuals prone to hypoglycemia). One should exercise daily and avoid milk and milk products. One should avoid smoked meats and bleached foods.

Overt diabetics, latent sub-clinical diabetics, chemical diabetics, or individuals with diabetic symptoms should, in addition, have glucose tolerance testing, provocative food testing, tests for exocrine pancreatic function, hypochlorhydria and achlorhydria, hyperinsulinemia, and blood and urine tests indicating deficiency of vitamins and minerals. Corrective measures should then be administered and results monitored.

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