Diabetes Mellitus

Diabetes is a chronic disorder of carbohydrate, protein, and fat metabolism characterised by fasting elevations of blood glucose levels and a greatly increased risk of heart disease, stroke, kidney disease, and loss of nerve function. It can represent an absolute insulin deficiency, impaired release of insulin by the pancreatic beta cells, inadequate or defective insulin receptors, or the production of inactive insulin or insulin that is destroyed before it can carry out its action. A person with uncontrolled diabetes is unable to transport glucose into fat and muscle cells. Glucose builds up in the bloodstream instead of being taken into and used by the cells, leading to hyperglycaemia. As a result, the body cells are starved, and the breakdown of fat and protein is increased. Hyperglycaemia also eventually leads to damaged blood vessels, which in turn, cause eye disease, heart disease, peripheral and autonomic neuropathy and diabetic nephropathy.[1,2]

Classification

As the common feature of diabetes is high blood glucose, it is often mistakenly thought to be a single disease. There are actually several types of diabetes, with different causal mechanisms. The three main types of diabetes are Type 1, Type 2 and gestational diabetes.

Type 1 diabetes is marked by a total or near-total lack of insulin. It results from the body destroying its own insulin-producing cells in the pancreas. People with this form of diabetes require daily insulin therapy to survive. It occurs more often in children and adolescents, and accounts for 10–15% of all people with diabetes. Surveys indicate that around 0.2–0.3% of the Australian population have Type 1 diabetes. [1,2]

Type 2 diabetes is marked by reduced levels of insulin, or the inability of the body to use insulin properly (insulin resistance). The disease is most common among people aged 40 years and over, and accounts for 85–90% of all people with diabetes. A 1999–2000 survey estimated that more than 7% of Australians aged 25 years or over have Type 2 diabetes. Many people with this form of diabetes eventually need insulin therapy to control their blood glucose levels. [1,2]

Gestational diabetes occurs during pregnancy in about 3–8% of females not previously diagnosed with diabetes. Screening tests for gestational diabetes are usually performed around the 24th–28th weeks of pregnancy. It is a temporary form of diabetes and usually disappears after the baby is born. However, it is a marker of increased risk of developing Type 2 diabetes later in life. [1,2]

Diagnostic Criteria

The diagnosis of diabetes mellitus in non-pregnant adults is based on fasting blood glucose levels, random blood glucose tests, or the results of a oral glucose tolerance test. All of the tests involve the measurement of glucose in the blood. This is expressed as the concentration of glucose in plasma. Testing for diagnosis should be considered in all individuals who are 45 years of age and older. Testing should be considered at a younger age in people who are obese, have a first-degree relative with diabetes, are members of a high-risk group, women who have delivered a baby more than 9 pounds, or those who have hypertension or hyperlipidaemia. A test may also be performed in people with classical symptoms of diabetes, such as excessive hunger, thirst and urination. [1,2]

Fasting Blood Glucose Tolerance

The fasting blood glucose test is the preferred diagnostic test due to ease of administration, convenience, patient acceptability and cost. Glucose is measured after food has been

withheld for 8 to 12 hours. If the fasting plasma glucose level is higher than 126 mg/dL (7.0 mmol/L) on two separate occasions, diabetes is diagnosed. A fasting plasma glucose level below 110 mg/dL (6.1 mmol/L) is normal. A level between 110 mg/dL and 126 mg/dL (6.1 to 7.0 mmol/L) is significant and is defined as impaired fasting glucose (IFG). [1,2]

Random Blood Glucose Test

A random blood glucose is one that is done without regard to meals or time of day. A random blood glucose concentration that is >200 mg/dL (11.1 mmol/L) in the presence of classic symptoms of diabetes such as polydipsia, polyphagia, polyuria and blurred vision is diagnostic of diabetes mellitus at any age.[1,2]

Oral Glucose Tolerance Test

The oral glucose tolerance test (OGTT) is an important screening test for diabetes. The test measures the body's ability to store glucose by removing it from the blood. In both men and women, the test measures the plasma glucose response to 75 g of concentrated glucose solution at selected intervals, usually one hour and two hours. In pregnant women, a glucose load of 100 g is given with an additional three-hour plasma glucose determination.[1,2]

In people with normal glucose tolerance, blood glucose levels should return to normal within two to three hours after ingestion of the glucose load. The patient is considered normal if the two hour plasma glucose is less than 140 mg/dL (7.75 mmol/L) and no value exceeds 200 mg/dL (11.1 mmol/L). If plasma levels are above 200 mg/dL (11.1 mmol/L) both at two hours and at least once between zero time and two hours, the patient is diagnosed as having diabetes mellitus. Patients with a two hour plasma glucose level between 140 mg/dL (7.75 mmol/L) and 200 mg/dL (11.1mmol/L) are said to have impaired glucose tolerance (IGT). [1,2]

Both impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) are associated with an increased risk of developing type 2 diabetes mellitus. Lifestyle changes, such as weight loss and exercise, are warranted in these patients.

Incidence and Epidemiology

Diabetes has become one of the leading threats to the health of Australians. There is now a growing epidemic due to the recent great rise of Type 2 diabetes, which contributes 85–90% of cases of diabetes. The Australian Diabetes, Obesity and Lifestyle Study (AusDiab), conducted in 1999–2000 to collect information on the prevalence of diabetes in Australia, revealed that approximately 940,000 Australians aged 25 or over have either Type 1 or Type 2 diabetes. The study also estimated that for every case of diabetes there exists an undiagnosed Type 2 case, thus about half a million Australians are unaware that they suffer from diabetes. Among some Aboriginal and Torres Strait Islander communities, as many as one-third of the community have diabetes.[3]

Prevalence of Type 1 diabetes

Based on self-reported information in the 1995 National Health Survey, the prevalence of Type 1 diabetes in Australia is about 39,400 people of all ages or 220 per 100,000 population.[3]

The National Heart Foundation Risk Factor Prevalence Study conducted in 1983 provided an estimate of prevalence of Type 1 diabetes in certain age groups based on plasma glucose levels. Among people aged between 25 and 64 years, the prevalence rate for Type 1 diabetes was 317 per 100,000.[4]

The Australian Diabetes, Obesity and Lifestyle Study collected diabetes prevalence information using an oral glucose tolerance test in persons aged 25 years or older only. The age-standardised prevalence of Type 1 diabetes was estimated at 298 per 100,000 for 1999–2000. This corresponds to 37,000 people aged 25 years or over.[3]

Due to the relatively low prevalence of Type 1 diabetes, there is no reliable information on differences in its occurrence among socioeconomic groups, or in urban, rural and remote areas.

Aboriginal and Torres Strait Islander people

Type 1 diabetes is generally thought to be rare among Aboriginal and Torres Strait Islander people, accounting for only 1–2% of all cases of diabetes among this group.[5]

Incidence

The National Diabetes Register collects information on insulin-treated diabetes in Australia. Coverage for 2000 is close to 100% among children with diabetes, who suffer predominantly from Type 1 diabetes. Incidence rates are around 19 per 100,000 population for both males and females aged 0–14 years. There were 743 new cases diagnosed in 2000.[3]

Prevalence of Type 2 diabetes

The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study estimated the prevalence of Type 2 diabetes in Australians aged 25 or over to be 7.2% (7.6% in males and 6.7% in females). This represents more than 850,000 Australians aged 25 years or over. The prevalence of Type 2 diabetes rises with age—a person aged 75 years or older is at least ten times more likely to have diabetes than someone aged 35–44 years. Available evidence suggests that there has been a rise in prevalence between 1995 and 1999–2000.[3]

Type 2 diabetes is more prevalent among people from lower socioeconomic backgrounds. In the lowest socioeconomic group (quintile 1) 3.6% of males and 4.3% of females reported having been diagnosed with Type 2 diabetes. A significantly lower proportion of people in the highest socioeconomic group (quintile 5) reported Type 2 diabetes: 2.0% of males and 1.7% of females.[3]

Aboriginal and Torres Strait Islander people

The prevalence of Type 2 diabetes is considerably higher among Aboriginal and Torres Strait Islander peoples than for the whole of the Australian population. Studies suggest that the prevalence may be as high as 30% in some Aboriginal communities, compared with 7% in the general population. Around 98–99% of diabetes in Aboriginal and Torres Strait Islander peoples is thought to be Type 2 diabetes.[5]

According to the 1994 National Aboriginal and Torres Strait Islander Survey, 3.5% of males and 4.7% of females reported having diabetes. Corresponding rates after the age of 45 years were 17% and 23%.[3]

Prevalence of Gestational Diabetes

The Australasian Diabetes in Pregnancy Society estimates the incidence of gestational diabetes to be around 5%.[6] The Australian Diabetes, Obesity and Lifestyle Study revealed that 3.6% of women who had been pregnant reported having been told that they had gestational diabetes.[3]

Aboriginal and Torres Strait Islander people

Available data suggest that Indigenous Australian women have a higher prevalence of gestational diabetes than non-Indigenous Australians. The incidence rate in Aboriginal and Torres Strait Islander women may be as high as 20% compared with a rate of 3–5% in Caucasian women.[7]

Indigenous Australians who have had gestational diabetes have a higher rate of conversion to Type 2 diabetes, up to 5% per year, compared with the Caucasian conversion rate of around 2% per year.[7]

Causes of Diabetes

Many factors are thought to contribute to the development of diabetes.

Genetic considerations

A genetic component is suspected in Type 1 diabetes. For instance, HLA-antigen analyses in Type 1 diabetes patients indicate substantial positive associations with HLA-DR_3, HLA-DW_3, HLA-DR_4, HLA-B_8 and HLA-B_{15}, and significant negative associations with HLA-B_7 and HLA-DR_{21}. Nonetheless, a large proportion of cases occur in people with no family history of the disease.[1]

While the above correlations are not observed in Type 2 diabetic patients, research on twins reveals that genetic factors also play a big role. In Type 1 diabetes, the concordance rate was 20-50% in identical twins and 5% in fraternal twins. These figures increased to almost 100% in identical twins and 10% in fraternal twins for Type 2 diabetes. In addition, family studies show that the presence of Type 2 diabetes in a family member is a risk factor; however, it is difficult to determine whether this represents the influence of genetics or shared environmental factors.[1,3]

Aetiology of Type 1 Diabetes Mellitus

Insulin dependent diabetes mellitus is generally recognized to be due to an insulin deficiency. Although the exact cause is unknown, current theory suggests a hereditary beta-cell disposition to injury coupled with some defect in tissue regeneration capacity. Causes of injury are probably free radicals, viral infections and autoimmune reactions.

Free Radicals

A diet high in bacon, ham, smoked salmon, and similar products has been linked to Type 1 diabetes. N-nitroso-compounds, found in smoked or cured meats, are similar in structure and function to Streptozotocin, which is a compound used to induce diabetes in animal studies. It works by destroying beta-cells. Various other chemicals in foods and the environment have also been implicated in beta-cell damage. These chemicals typically damage the pancreas by acting as free radicals.[8]

Viral Infection

Recent population studies have reinforced the theory that a viral infection is the cause of Type 1 diabetes in some patients. Viruses can infect pancreatic beta cells and induce an antibody attack. A viral cause was originally suspected due to the seasonal variation in the onset of the disease. During October to March, viral diseases, such as mumps, hepatitis, infectious mononucleiosis, congenital rubella, and coxsackie virus infections are much more common.[8]

Autoimmunity

Developing antibodies against the pancreatic beta-cells is probably the major causative factor, especially in individuals with a specific HLA-B_8 genotype. Antibodies to pancreatic cells are present in 75% of all cases of Type 1 diabetes, compared to only 1.5-2% in normal individuals. It is possible that as a result of other mechanisms, when normally concealed cellular antigens are exposed, the antibodies to the beta-cells develop in response to cell destruction. It appears that individuals who do not have diabetes either do not develop as severe an antibody reaction, or are more capable of repairing the damage once it occurs.[8]

Early Weaning and Cow's Milk Exposure

Recent studies have provided some strong evidence that exposure to a cow's milk in infancy may trigger the autoimmune process that subsequently leads to Type 1 diabetes. In fact, early cow's milk exposure may increase the risk by about 1.5 times. Studies show that patients with Type 1 diabetes were more likely to have been breast-fed for under three months and to have been exposed to cow's milk or solid foods before the age of four months.[8]

Aetiology of Type 2 Diabetes Mellitus

Type 2 diabetes results from a combination of genetic, environmental and behavioral risk factors. Insulin insensitivity is central to the development of Type 2 diabetes mellitus. The typical Type 2 diabetic patient normally suffers from hyperinsulinaemia. The pancreas is still secreting insulin, but the insulin is unable to perform its function as the cells have become unresponsive. Measures to restore insulin sensitivity usually improve Type 2 diabetes.

Age

The risk of developing Type 2 diabetes increases significantly with age; the incidence of Type 2 diabetes is low before 30 years of age.[3]

Ethnicity

Race and ethnic background are also associated with the development of Type 2 diabetes, with the prevalence of the condition being higher among Indigenous Australians and people of Pacific Islander, Asian and Southern European descent.[3]

Obesity

Excess weight, particularly abdominal obesity, is strongly linked to insulin resistance. Being overweight or obese increases the risk of developing Type 2 diabetes and, in those who already have diabetes, increases its severity. Overweight adults are up to three times more likely to develop Type 2 diabetes than those of ideal weight, while for those who are obese the risk is much greater, possibly up to ten times that of persons of ideal weight. Thus, weight loss, especially a significant decrease in body-fat percentage, is a prime objective in treating the majority of Type 2 diabetic patients since it improves all aspects of diabetes and may even bring about cure.[8]

Dietary Fat

A high percentage of fat in the diet, especially saturated fat, has been linked to Type 2 diabetes. The percentage of dietary fat intake is also used to predict the conversion of impaired glucose tolerance to Type 2 diabetes. This suggests that a high-fat diet increases the risk of subsequently developing Type 2 diabetes.[8]

Chromium Deficiency

Chromium deficiency is widespread, and may contribute to the prevalence of Type 2 diabetes. This is because chromium is vital for increasing sensitivity of cells to insulin. Chromium, as a critical component of the glucose tolerance factor (GTF), functions as a cofactor in all insulin-regulating activities. Supplementing the diet with chromium has been shown to significantly improve insulin action; decrease fasting glucose, cholesterol, and

triglyceride levels; and increase the HDL-cholesterol levels by increasing insulin sensitivity in normal, elderly and Type 2 diabetes patients.[8]

Prenatal Factors

Population research suggests that the nutritional status of the mother during pregnancy plays a role in determining whether the child will develop both types of diabetes later in life. Research indicates that overeating during pregnancy may increase the risk for diabetes for the unborn foetus later in life. Studies also show a significantly lower incidence of childhood diabetes when blood glucose levels are carefully controlled. A greater than 50% drop in the incidence of childhood diabetes was observed when mothers avoided an increase in blood sugar levels above normal. On the other hand, poor foetal nutrition leads to low birthweight, which may also predispose individuals to Type 2 diabetes. If such individuals are exposed to other risk factors (obesity, ageing and physical inactivity), the likelihood of developing Type 2 diabetes later in life also increases.[3,8]

Health Impact / Complications of Diabetes

Chronic elevations in blood glucose can lead to a variety of both short-term and long-term complications in diabetic patients. Monitoring and controlling the extent of hyperglycaemia is an essential component in the prevention of major diabetic complications.

Acute Complications

Diabetics are at risk of three main acute complications: hypoglycaemia, diabetic ketoacidosis and nonketogenic hyperosmolar syndrome.

Hypoglycaemia

As it is mainly Type 1 diabetic patients who need insulin injections, they are the ones who are typically at risk of developing hypoglycaemia from insulin or oral hypoglycaemic drugs. In addition to taking insulin, missing a meal or over-exercising can also cause hypoglycaemia. Symptoms include sweating, nervousness, tremor, and hunger in the day, and night sweats and unpleasant dreams at night, with the patient waking with an early morning headache.[1,2]

In response to the hypoglycaemia, hormones that raise blood glucose levels such as adrenaline, noradrenaline, growth hormone and cortisol increase. In turn, blood sugar levels will rebound, leading to hyperglycaemia.[1,2]

Diabetic Ketoacidosis

This acute complication is also more likely to occur in Type 1 diabetics. Ketoacidosis is caused by a lack of insulin, which results in a buildup of ketoacids. If allowed to progress, it can lead to metabolic problems and even coma. A day or more of polydipsia and polyuria as well as marked fatigue, nausea and vomiting usually precedes the coma. It is important to recognize these signs since ketoacidosis is a potential medical emergency.[1,2]

Non-Ketogenic Hyperosmolar Syndrome

This acute complication primarily affects Type 2 diabetes patients. It is a medical emergency with a mortality rate of more than 50%. It is usually caused by profound dehydration due to insufficient fluid intake or precipitating events like pneumonia, burns, stroke, a recent operation, or certain drugs. The onset of the syndrome may be subtle, occurring over days or even weeks. Symptoms include weakness, polydipsia, polyuria, and progressively serious signs of dehydration like weight loss, loss of skin elasticity, dry mucous membranes, rapid heart beat and low blood pressure.[1,2]

Chronic Complications

Diabetes can result in a range of long-term complications in addition to causing acute metabolic reactions. These complications are responsible for loss of working ability, invalidism, shortened life expectancy and reduced quality of life among people with diabetes.

People with diabetes are more prone to diseases of the large blood vessels (macrovascular disease) such as coronary heart disease, stroke and peripheral vascular disease as well as diseases of the small blood vessels (microvascular disease) such as retinopathy, kidney diseases and neuropathy (peripheral nerve disease).

Cardiovascular Disease

People with diabetes are two to four times more likely to develop cardiovascular disease compared with those without diabetes. Coronary heart disease, stroke and peripheral vascular disease, are the most common cardiovascular complications associated with diabetes. It is thought that diabetes increases the risk of these cardiovascular disease by increasing atherosclerosis. Other factors possibly contributing to the excess risk include high blood pressure and dyslipidaemia (low levels of HDL cholesterol and high levels of LDL cholesterol and triglycerides). The prevalence of these risk factors is higher among people with diabetes.[3,9]

Eye disease

People with diabetes are at an increased risk of developing eye disease, particularly diabetic retinopathy (retinal disease), cataract and glaucoma.

Diabetic retinopathy is a microvascular complication of diabetes caused by changes in the blood vessels of the retina. In some people with diabetic retinopathy, retinal blood vessels may swell and leak fluid, while in others, abnormal new blood vessels grow on the surface of the retina. It is the most common cause of blindness in people aged 30–69 years. [3,10-11]

Cataracts and glaucoma are also major causes of vision impairment and blindness among adults. A cataract is a clouding of the normally clear lens of the eye, leading to vision loss. Cataracts are more common and progress more rapidly in people with diabetes. Glaucoma is a condition where pressure builds up in the eye, pinching the capillaries that carry blood to the retina and optic nerve. Over time, the retina and optic nerve become damaged and vision is lost. People with diabetes are significantly more likely to develop glaucoma than people without diabetes.[12]

Kidney disease

Diabetes can affect the kidneys in a variety of ways, leading to serious and even life-threatening conditions. The most common kidney complications associated with diabetes are diabetic nephropathy and end-stage renal disease (ESRD).

Diabetic nephropathy results from high blood glucose levels damaging the blood-filtering capillaries (glomeruli) in the kidneys. The glomeruli's filtering efficiency declines and blood proteins such as albumin leak into the urine (albuminuria). As diabetic nephropathy progresses, the kidneys leak larger amounts of albumin. Microalbuminuria is a strong predictor of developing proteinuria, ESRD, high blood pressure and cardiovascular disease. Tight control of blood glucose and blood pressure may prevent microalbuminuria progressing to proteinuria or ESRD. [3]

ESRD is the final stage in the worsening of kidney function, when the kidneys lose the ability to remove waste products such as creatinine and urea from the blood. Diabetic nephropathy is the second most common cause of ESRD in Australia.[3]

Neuropathy

Neuropathy or nerve damage is a frequent complication of diabetes. Diabetic neuropathy usually manifests as either peripheral neuropathy, most commonly causing damage to the nerves in the feet, or autonomic neuropathy. The sequelae of diabetic neuropathy include pain, digestive problems, muscle weakness, non-healing ulcers and lower extremity amputation, and are associated with reduced quality of life and increased mortality. Diabetic neuropathy is generally a result of chronically high blood glucose levels which affect the metabolism of nerves. This in turn causes the accumulation of toxins which damage nerve structure and function.[1,3]

Peripheral nephropathy is classified into two broad types:

Sensory neuropathy – affects the nerves that carry information to the brain about sensations from various parts of the body. Symptoms may include pain, tingling in the limbs or absence of feeling in the feet, which predisposes people with diabetes to foot trauma.[1,3]
Motor neuropathy – affects the nerves that carry signals to muscles to allow the muscles to move. Motor neuropathy can lead to muscle weakness, particularly in the feet, which may become deformed as a result.[1,3]

Autonomic neuropathy affects the nerves that control involuntary body functions such as heart rate, blood pressure, sweating, and the action of the stomach, intestine and bladder. Symptoms may include dizziness and fainting, nausea, vomiting and diarrhoea, loss of bladder control and impotence in men.[1,3]

Foot complications

Diabetes is associated with nerve damage (peripheral neuropathy) and poor circulation (peripheral vascular disease) in the lower limbs. These factors increase the risk of developing foot ulcers and infections. Progression of these conditions in people with diabetes often leads to lower extremity amputations. Diabetes is estimated to account for about half of all non-traumatic amputations.[13]

Over time, diabetes can damage the nerves in the feet, resulting in a loss of sensation. Reduced sensation of pain and discomfort from foreign bodies, injury or even tightly fitting shoes can predispose people to foot trauma and ulceration. Damage to nerves also causes wasting of the foot muscles, reduced joint mobility and foot deformities such as claw or hammer toes that are vulnerable to ulceration. High blood glucose can also damage blood vessels in the lower limbs. Without a healthy supply of oxygen and nutrients, feet are predisposed to ulceration and infection.[1,3]

Nonhealing ulcers can result in gangrene and amputation of the affected area may be necessary as a limb-salvaging procedure if medical treatment is unsuccessful. Amputation is estimated to be 15 times more common in people with diabetes. Many patients with diabetes who undergo amputation will have a subsequent amputation on the other side within a few years. The remaining limb becomes more vulnerable to ulceration and infection because it has to bear extra pressure.[1,3]

Complications in Pregnancy

Two forms of maternal diabetes may occur during pregnancy: pre-existing diabetes and gestational diabetes.

Pre-Existing Diabetes

In women with pre-existing diabetes, glycaemic control worsens during pregnancy and insulin requirements increase in Type 1 diabetes. This is because the pregnancy hormones induce insulin resistance. Maternal diabetes affects the foetus and newborn as well as the mother.[3]

For these mothers, pregnancy can worsen kidney function in those with established nephropathy. Retinopathy may also deteriorate rapidly during gestation. Caesarean delivery is three to four times more frequent in pregnancies involving diabetes.[3]

In the foetus, pre-existing diabetes can cause major congenital malformations, as well as spontaneous abortions. Defects include absence of brain, malformations of the spine, skeleton and kidneys, and heart and great blood vessel abnormalities. The malformation rate is related to the degree of hyperglycaemia. International studies indicate that spontaneous abortions occur in 7–17% of diabetic pregnancies if diabetes is not well managed. The perinatal death rate is also increased by 1.5 to 2-fold in pregnancies with pre-existing diabetes compared with those without diabetes.[3]

International studies also show that large babies occur at a rate of 30% in diabetic pregnancies compared with 10% in non-diabetic pregnancies. Accelerated foetal growth is due to increased delivery of glucose and other nutrients from mother to foetus. This stimulates the pancreas in the foetus to produce extra insulin, which promotes abdominal fat deposition, growth of the skeleton and large size organs. Complications for these babies include birth trauma, and jaundice, hypoglycaemia and low levels of calcium in the newborn. Poor glycaemic control also leads to impaired lubrication of the lungs and respiratory distress in the newborn. These babies may also have a long-term greater risk of obesity.[3]

Gestational Diabetes

Women with gestational diabetes may have a greater risk of foetal perinatal death and disease, and are themselves at increased risk of developing Type 2 diabetes and perhaps cardiovascular disease later in life. Infants of women who develop gestational diabetes may have newborn hypoglycaemia, jaundice, respiratory distress and birth trauma resulting from being excessively large babies, much the same as those of women with pre-existing diabetes.[3]

Therapeutic Considerations

Nutritional Supplements

Chromium

Chromium is required for the maintenance of normal glucose metabolism. It is vital to proper blood sugar control as it functions as a key constituent of the "glucose tolerance factor". Glucose tolerance factor increases glucose tolerance by potentiating the effects of insulin. Without chromium, insulin's action is blocked, and glucose levels are elevated. Studies suggest that people who are deficient in chromium have difficulty regulating their blood sugar and that supplementation can improve impaired glucose tolerance.[14]

Chromium stimulates the activity of enzymes involved in the metabolism of glucose for energy and the synthesis of fatty acids and cholesterol. In the blood, it competes with iron in the transport of protein. Chromium may also be involved in protein synthesis by binding with RNA molecules.[15]

Clinical research has shown that supplementing the diet with chromium to decrease fasting glucose levels improves glucose tolerance, lowers insulin levels and decreases total cholesterol and triglyceride levels, while increasing HDL cholesterol levels.[16-17]

While some studies suggest that the effect of chromium in improving glucose tolerance in diabetes is insignificant, others have found it to be an undoubtedly important mineral in blood sugar metabolism. For instance, a randomized study of 180 patients with Type 2 diabetes clearly documented the benefit of chromium for Type 2 diabetic patients. In the study, one group was a placebo, one group was given 100 mcg of chromium picolinate twice per day, and the remaining group was given 500 mcg of chromium picolinate twice daily.

The results showed significant dose- and time-dependent decreases in glycosylated hemoglobin, fasting glucose, 2 hour postprandial glucose levels, fasting and 2 hour postprandial insulin values, and total serum cholesterol.[18]

Further benefits of chromium for Type 2 diabetic patients is its ability to lower body weight while increasing lean body mass. In a double blind study conducted in 1998, subjects who were supplemented with chromium picolinate show increased lean body mass while achieving weight loss. In comparison, treatment groups who did not receive supplementation still had reduced lean body mass. The study concluded that chromium picolinate is able to increase lean body mass in obese patients without counteracting the weight loss achieved.[19] This factor together with its ability to increase insulin sensitivity suggests that chromium supplementation may have an important role to play in patients suffering from diabetes, hypoglycaemia, and obesity.[1] Thus, in addition to the regular consumption of chromium-rich foods, the diabetic, obese and hypoglycaemic should supplement the diet with chromium picolinate.

Although no RDA has been established for chromium, at least 200 mcg/day appears necessary for optimal sugar regulation.[1] Chromium deficiency may be promoted by increasing chromium losses in response to physiological stressors. These include physical trauma, acute but not long term exercise, lactation and a high consumption of simple sugars. These factors, in addition to a marginal chromium intake, may promote chromium depletion in humans.[15]

Vitamin C

Vitamin C is involved in the manufacture of collagen, which is an important protein for connective tissue. Hence, vitamin C is vital for wound repair, healthy gums, and the prevention of excessive bruising, all of which are particularly crucial for the diabetic patient. In addition, vitamin C is also important for immune function, the manufacture of certain nerve transmitting substances and hormones, and the absorption and utilization of other nutritional factors.[1]

A point of consideration for diabetics is that the transport of vitamin C into cells is facilitated by insulin. Having no insulin or impaired functioning of insulin could result in insufficient intracellular vitamin C, and hence a deficiency in diabetics despite adequate dietary consumption. This is particularly detrimental for patients with diabetes as it could result in complications like an increased tendency to bleed, poor wound healing, microvascular disease, elevations in cholesterol levels, and a depressed immune system.[20]

Another reason that vitamin C is crucial for reducing complications in diabetic patients is that studies have found that at high doses of 2000 mg per day, vitamin C has the ability to reduce the accumulation of sorbitol in the erythrocytes and to inhibit the glycosylation of proteins.[21-22] In one study which ran for 58 days, young adults with Type 1 diabetes were supplemented with 100 or 600 mg vitamin C. RBC sorbitol was measured at baseline and again at 30 and 58 days. At baseline, RBC sorbitol levels were nearly doubled in these patients despite "adequate" dietary intakes of vitamin C. However, with the vitamin C supplementation at both dosages, RBC sorbitol levels were normalised within 30 days. This correction of sorbitol accumulation was independent of changes in diabetic control as monitored by fasting glucose, glycosylated hemoglobin, and glycosuria. The researchers hence concluded that vitamin C supplementation is effective in reducing sorbitol accumulation in the erythrocytes of diabetics.[23] Sorbitol accumulation and glycosyltion of proteins have been associated with complications of the nerves and eyes in diabetes, hence supplementation can be vital.

Supplementation with a minimum of 2 g daily of vitamin C is essential for reaping the maximum benefits in diabetes. However, while vitamin C supplementation will be necessary to ensure this level of intake, patients should be encouraged to also obtain vitamin C from

vitamin C-rich foods that are rich in flavonoids and carotenes. Good dietary sources of vitamin C like broccoli, peppers, potatoes, Brussels sprouts and citrus fruits enhance the effects of vitamin C as well as exerting favorable effects of their own.[1]

Niacin and niacinamide

Also known as vitamin B_3, niacin has earned a reputation as a natural cholesterol-lowering agent that often rivals prescription drugs in mild to moderate cases. It may also help to prevent or treat a number of other disorders, from arthritis and depression to diabetes. Three forms of niacin supplements are commercially available: nicotinic acid (also called nicotinate), niacinamide and inositol hexaniacinate, a compound of niacin and inositol.

Normally, the body manages to absorb enough niacin from foods to carry out basic functions, working on the cellular level to keep the digestive system, skin and nerves healthy. This vitamin is also critical to releasing energy from carbohydrates and helping to control blood-sugar levels. Niacin, like chromium, is an essential component of the glucose tolerance factor, making it a key nutrient for hypoglycemia and diabetes.[24]

Supplementing the diet of diabetics with niacin in the form of niacinamide has been shown to exert many favorable effects. In fact, studies have shown that high doses of niacinamide, if given at the first signs of Type 1 diabetes, can help restore beta-cells or at least slow down their destruction. In turn, this helps to prevent complications and may even stop Type 1 diabetes from developing.[25]

Several studies on niacinamide treatment in recent onset Type 1 diabetes or Type 1 diabetes that is less than 5 years have indicated positive effects in terms of prolonged non-insulin-requiring remission, lower insulin requirements, improved metabolic control, and increased beta-cell function. With niacinamide supplementation, there has even been complete resolution of the condition in some newly diagnosed Type 1 diabetics.[26]

The mechanism of action behind niacinamide lies in its ability to inhibit macrophage- and interleukin-1-mediated beta-cell damage, and nitric oxide production. In addition, it is also an antioxidant.[27] The typical daily dose of niacinamide used in studies is 25 mg per kg of body weight. The studies in children used 100-200 mg per day.[1]

The inositol hexaniacinate form of niacin is also useful in both Type 1 and Type 2 diabetes to lower elevated blood lipids. Niacin or nicotinic acid has long been used to lower cholesterol levels. Unlike many cholesterol-lowering drugs that can potentially reduce life expectancy, niacin has been shown to lower cholesterol levels safely and even extend life. Moreover, unlike most prescription cholesterol-lowering medications, which simply lower levels of LDL cholesterol and triglycerides, niacin also raises levels of HDL cholesterol. (Lavie) As a result, this vitamin may prove more potent than conventional medicines in ultimately reducing the risk of cardiovascular diseases such as a heart attack.[28]

Although niacin's cholesterol-benefiting actions are well documented, because the dose required (1 g three times per day) to lower cholesterol levels often results in flushing of the skin, stomach irritation, ulcers, liver damage, fatigue, and other side effects, many people do not tolerate niacin very well. Hence, inositol hexaniacinate, composed of one molecule of inositol and six molecules of niacin, is the preferred form. It doesn't cause skin flushing and poses much less risk of liver damage with long-term use.[29]

Inositol hexaniacinate has long been used in Europe to lower cholesterol levels and to improve blood flow in patients with intermittent claudication. In addition to increased tolerance in patients, it has in fact yielded slightly better clinical results than standard niacin, including improved sugar level regulation. In one study of 153 patients treated with inositol hexaniacinate at dosages ranging from 600-1,800 mg/ day, no patients reported any side-

effects or adverse reaction.[29] In cases of hyperlipidemia, a dosage of 600-1,000 mg three times daily is usually sufficient to produce an 18% reduction in total cholesterol, a 26% reduction in triglycerides, and an increase of 30% in HDL cholesterol levels.[1]

As niacin can potentially disrupt blood sugar control in diabetics, patients taking it in any form must closely monitor blood sugar levels and discontinue treatment in the event of worsening of diabetic control.

Biotin

Although biotin is one of the lesser-known B vitamins, it plays an essential role in a number of important body processes. Taking its name from the Greek word *bios,* meaning "life," this nutrient assists the body in metabolizing protein, fats, and carbohydrates from food. It is particularly important for diabetic patients because it also plays a role in enabling the body to use blood sugar (glucose).

Biotin supplementation has been shown to improve glucose tolerance and enhance insulin sensitivity. In one animal study, 26 diabetic mice that were moderately hyperglycemic and insulin resistant, were treated for 10 weeks: 9 were given 2 mg of biotin per kg, 8 were given 4 mg of biotin per kg and 9 controls were given saline. Blood glucose levels, oral glucose tolerance, insulin response to oral glucose, and blood glucose decrease in response to insulin were recorded. Compared to controls, biotin treatment lowered post-prandial glucose levels, and improved tolerance to glucose and insulin resistance.[30]

Similarly in human studies, supplementation with biotin has also been shown to enhance insulin sensitivity. The mechanism of action appears to be biotin's ability to increase the activity of the enzyme glucokinase (GK). GK, expressed in hepatocyte and β cells, has a central regulatory role in glucose metabolism. Efficient GK activity is required for normal glucose-stimulated insulin secretion, postprandial hepatic glucose uptake, and the appropriate suppression of hepatic glucose output and gluconeogenesis by elevated plasma glucose. Hepatic GK activity is subnormal in diabetes and GK may also be decreased in the β cells of Type 2 diabetics. In supraphysiological concentrations, biotin promotes the transcription and translation of the GK gene in hepatocytes; this effect appears to be mediated by activation of soluble guanylate cyclase. More recent evidence indicates that biotin likewise increases GK activity in islet cells.[31]

In one study, 16 mg daily of biotin significantly reduced fasting blood sugar levels and improved blood glucose control in Type 1 diabetics.[30] In another study conducted in Type 2 diabetics, similar effects were noted with 9 mg / day of biotin.[32]

Vitamin B_6

Vitamin B_6, as the active coenzyme pyridoxal phosphate, plays an important role in the metabolism of carbohydrates. Pyridoxal phosphate serves as a coenzyme for enzymatic reactions in both gluconeogenesis and glycogenolysis. A deficiency of vitamin B_6 has been associated with impairments in gluconeogenesis and abnormal glucose tolerance. In addition, other major abnormalities in carbohydrate metabolism observed in a vitamin B_6 deficiency include hypoglycaemia, reduced liver glycogen levels, reduced serum and pancreatic insulin levels, degenerative changes in β cells, reduced growth hormone levels and reduced insulin response to glucose load. Hence vitamin B_6 supplementation is an essential component of treatment for patients with diabetes.[33]

Vitamin B_6 supplementation also appears to offer significant protection in terms of preventing diabetic complications. For one, it protects against the development of diabetic neuropathy. This is particularly important as diabetics with neuropathy have been shown to be deficient

in vitamin B_6 and to benefit from supplementation. Individuals with long-standing diabetes or who are developing signs of peripheral nerve abnormalities should definitely be supplemented with vitamin B_6. The standard dose prescribed is 150 mg.[34]

With its ability to inhibit glycosylation of proteins, which leads to tissue damage, vitamin B_6 may also prove to be important in preventing other diabetic complications of neuropathy, retinopathy, vascular damage and cataracts.[35]

Vitamin B_{12}

Not only does vitamin B_{12} help in the formation of healthy red blood cells, it is also involved in the maintenance of the myelin sheath, the fatty substance that covers nerves and enables them to function properly. In addition to fatigue, depression, confusion, memory loss and muscle weakness, a deficiency can cause numbness and tingling in the hands and feet due to nerve damage. These symptoms are typical of diabetic neuropathy, and a deficiency of vitamin B_{12} deficiency has been linked to the condition.[36] While supplementation seems to improve diabetic neuropathy, it remains unclear if this is due to the correcting of a deficiency state or the normalization of the deranged vitamin B_{12} metabolism seen in diabetics.[37] Oral supplementation with 1,000-3,000 mcg / day may be sufficient, but intramuscular vitamin B_{12} may be necessary in many cases.[1]

Vitamin E

Diabetics and experimental animal models exhibit high oxidative stress due to persistent and chronic hyperglycemia. This depletes the activity of the antioxidative defence system and thereby promote the generation of free radicals. As such, diabetics appear to have an increased requirement for vitamin E.

One study that examined the effects of vitamin E on oxidative stress and membrane fluidity in the brain of diabetes-induced rats found that dietary vitamin E strengthened the antioxidative defense system with an increased activity of the antioxidant enzymes, such as superoxide dismutase (SOD) and glutathione peroxidase and increased vitamin E content in the brain. Accordingly, vitamin E was found to reduce the accumulation of reactive oxygen species such as superoxide radicals, decrease the generation of oxidative damage substances, increase the membrane fluidity lowered by oxidative damage, and significantly improve lipid compositions.[38] All of these findings may aid may aid in preventing the long-term complications of diabetes.

In addition, vitamin E appears to play a significant role in the prevention of diabetes. In one study which involved 944 men, 42-60 years of age, who did not have diabetes at baseline, 45 men developed diabetes during the 4 year follow-up. A low lipid standardized plasma vitamin E concentration was associated with 3.9-fold risk of incidence of diabetes. A decrement of 1 micro-mol/ L of serum vitamin E was associated with an increment of 22% in the risk of diabetes.[39]

Magnesium

Magnesium plays a variety of roles in the body. Not only is it critical for energy production and proper nerve function, it also promotes muscle relaxation and helps the body produce and use insulin. Magnesium is involved in several areas of glucose metabolism and there is considerable evidence that diabetics need supplemental magnesium. This is because diabetes can cause low magnesium levels.[40] A poor intracellular magnesium concentration, as found in Type 2 diabetics and in hypertensive patients, may result in a defective tyrosine-kinase activity at the insulin receptor level and exaggerated intracellular calcium

concentration. Both events are responsible for the impairment in insulin action and a worsening of insulin resistance in these diabetic and hypertensive patients.[41]

By contrast, daily magnesium administration to Type 2 diabetics, restoring a more appropriate intracellular Mg concentration, contributes to improve insulin-mediated glucose uptake. The benefits deriving from daily supplementation are further supported by epidemiological studies showing that high daily magnesium intake are predictive of a lower incidence of Type 2 diabetes.[41] Studies also suggest that magnesium may prevent some of the complications of diabetes, such as retinopathy and heart disease. Magnesium levels are lowest in those with severe retinopathy.[40]

In addition to eating a diet high in magnesium-rich foods like tofu, legumes, nuts, whole grains and green leafy vegetables, supplementation with 300-500 mg of magnesium as aspartate or citrate is recommended. Also, diabetics should take at least 50 mg per day of vitamin B6, as the level of intracellular vitamin B6 appears to be intricately linked to the magnesium content of the cell. Without vitamin B6, magnesium is unable to get inside the cell.[1]

Potassium

A high-potassium diet is particularly beneficial for diabetics because potassium helps to improve insulin sensitivity, responsiveness and secretion, insulin administration induces a loss of potassium, and a high potassium intake reduces the risk of cardiovascular complications like heart disease and atherosclerosis.[42-43] The RDA for potassium is 1.9-5.6 g. If body potassium requirements are not being met through diet, supplementation is essential to good health, especially so for diabetics, the elderly and athletes.[1]

Physicians commonly prescribe potassium salts in the dosage range of 1.5-3.0 g/ day. However, potassium salts can cause nausea, vomiting, diarrhoea, and ulcers. Thus a better option is using potassium-rich foods like vegetable juices, or food-based potassium supplements to meet the human body's potassium requirements. [1]

Another point to consider for diabetic patients and patients with kidney disease is that excess potassium intake may lead to heart disturbances and other consequences of potassium toxicity. Individuals with kidney disorders usually need to restrict their potassium intake. Most diabetics can consume a high-potassium diet, but their kidney function should be properly evaluated before prescribing a potassium supplement. [1]

Manganese

Manganese is a cofactor in many enzyme systems involved in blood sugar control, energy metabolism, and thyroid hormone function.[44-45] In animal studies, a manganese deficiency was linked with increased incidence of diabetes and the frequent birth of offspring who develop pancreatic abnormalities or who have no pancreas at all. Moreover, diabetics have also been found to have only one-half the manganese of normal individuals. Thus supplementation of a daily dose of 30 mg is beneficial for diabetics.[1]

Zinc

Zinc is involved in virtually all aspects of insulin metabolism: synthesis, secretion, and utilization. By improving levels of insulin, zinc supplements may help people with Type 1 or 2 diabetes manage their disease more effectively.[46] Zinc also protects against beta-cell destruction, and its antiviral effects are well known. In addition, some people with diabetes have wounds that fail to heal well; this problem relates in part to the presence of high blood sugar levels and zinc may help by regulating insulin metabolism. Moreover, zinc also helps to

repair the skin's top layer in part by helping to process the essential fatty acids that encourage healing.[47] Zinc is found in good amounts in whole grains, legumes, nuts, and seeds. Diabetics are found to excrete excessive amounts of zinc in the urine and therefore require supplementation. The recommended level of zinc supplementation for diabetics is at least 30 mg per day.[1]

Flavonoids

Flavonoids is the umbrella term given to some 4,000 compounds that impart the colorful pigment to fruits, vegetables and herbs. They appear to act as effective antivirals, anti-inflammatories, antihistamines and antioxidants, and are useful for reducing cancer risk and preventing or treating a wide variety of conditions. Recent research suggests that among these conditions, flavonoids may be useful in diabetes.[48-50] For instance, flavonoids, such as quercetin, promote insulin secretion and are potent inhibitors of sorbitol accumulation. Other therapeutic benefits of flavonoids include the increase of intracellular vitamin C levels, a decrease in the leakiness and breakage of small blood vessels, the prevention of easy bruising, and immune system support.[50] All of these are factors that could help reduce complications in diabetes. In addition to consuming a diet rich in flavonoids, the diabetic should take an extra 1-2 g per day of mixed flavonoids.[1]

Essential fatty acids

Supplementation of essential fatty acids (EFA) have been found to benefit various aspects of diabetes. More specifically, the omega-6 gamma-linolenic acid has been shown to protect against the development of diabetic neuropathy while the omega-3 oils protect against atherosclerotic processes and enhance insulin secretion in Type 2 diabetes.

Omega-6 fatty acids. It is thought that intracellular deficiencies of key nutrients like vitamin C, magnesium, vitamin B6, and zinc is responsible for the considerable disturbance in essential fatty acid (EFA) metabolism found in both human and experimental diabetes. This disturbance leads to a variety of microvascular, hemorrheological, and other abnormalities, which eventually result in reduced blood flow and neuronal hypoxia.[51]

One key disturbance is impairment in the process of converting linoleic acid to gamma-linolenic (GLA), dihomo-gamma-linolenic (DHGLA), and arachidonic acids.[51] In the Gamma-Linolenic Acid Multicenter Trial, 111 patients with mild diabetic neuropathy were given 480 mg of GLA in the form of evening primrose oil daily for 1 year. Sixteen different parameters were assessed, including conduction velocities and amplitudes, hot and cold thresholds, sensation, tendon reflexes, and muscle strength. After 1 year, all 16 parameters improved, 13 of which were statistically significant.[52] The researchers concluded that supplementing GLA in the form of evening primrose oil may thus help to bypass disturbance in EFA metabolism in diabetic patients. Other sources of GLA include borage and black currant oils.[51]

Omega-3 fatty acids. With regards to the effect of omega-3 fatty acids on diabetes, findings have been contradictory. On the one hand, the cholesterol and triglyceride lowering abilities of omega-3 fatty acid-rich oils have been widely publicised in hundreds of studies, including those performed on diabetics.[53] For these patients, omega-3 fatty acids have been shown to lower total cholesterol, LDL cholesterol, and triglyceride levels while simultaneously raising HDL cholesterol levels. In addition, diabetics also experienced other benefits like decreased platelet reactivity, decreased blood viscosity, increased plasma levels of tissue plasminogen activator, reduced endothelial permeability and improved insulin.[53]

On the other hand, another group of studies have reported potentially deleterious effects, including increased levels of plasma glucose, total cholesterol, LDL cholesterol, and serum

apolipoprotein B.[54-55] These have raised doubts regarding the safety of fish oil supplementation in diabetes.

Further studies have thrown some light on the contradictory findings. Specifically, dosage appears to be a critical element. Adverse effects tended to occur at larger doses of 4 – 10 gl per day of eicosapentaenoic acid (EPA) and docosahexanoic acid (DHA).[54-55] At the lower dosage of 2.5 g of omega-3 fatty acids, fish oil supplementation inhibited platelet aggregation and thromboxane A2 production, reduced elevated systolic blood pressure, reduced triglyceride levels, and caused small, but statistically significant, increases in HBAlc and total cholesterol.[54]

In one study, a 96% pure EPA product at a dose of 900 mg per day for 8 weeks reduced platelet aggregation due to platelet-activating factor. However, it had little effect on diabetes regulation, lipid levels, and blood rheology. In contrast, 1,800 mg / day for 8 weeks reduced platelet aggregation, but LDL cholesterol increased significantly. It is generally concluded that dosages above 900 mg daily, may potentially produce negative effects on blood sugar control, and perhaps on blood lipids too.[55]

With this in mind, it is hence advisable to be cautious when prescribing fish oils to diabetics. One of the reasons fish oil products may be negatively affecting diabetes is the high level of lipid peroxides in these preparations coupled with their effects on depleting antioxidant nutrients when ingested. Thus fish consumption or the use of flaxseed oil, neither of which is associated with negative effects in diabetes, may be a safer option to help reap the benefits of omega-3 oils. In fact, increased consumption of cold-water fish has been shown to produce effects that are equal or even superior to fish oil supplementation.[1]

In one study, 25 men suffering from hypercholesterolaemia were studied over for 5 weeks to compare the effects of eating an equivalent amount of fish oil from whole fish with those of a fish oil supplement. Although both fish and fish oil supplements lowered triglycerides and raised HDL cholesterol, dietary fish produced additional benefits over the fish oil supplements.[56] Another study demonstrated an inverse correlation between fish intake and impaired glucose tolerance and diabetes. A mean daily intake of 24.2 g was associated with a significantly lower incidence of glucose intolerance. In addition, mortality was lower in fish consumers than in those who did not eat fish.[57] These studies, along with additional epidemiological studies showing a low prevalence of diabetes in cultures consuming cold-water fish, indicate that omega-3 fatty acids may offer some protection against the development of diabetes. An average daily intake of 30 g or two servings per week of fish is recommended.[1]

In addition to fish oils, flaxseed oil may offer similar benefits because it contains alpha-linolenic acid (ALA), an omega-3 oil which the body can convert to EPA. Moreover, flaxseed oil supplementation may avoid some of the problems associated with EPA supplementation in diabetics.[1]

In summary, diabetics can benefit from both omega-6 and omega-3 fatty acids. At this time, it appears the best approach in diabetics is to increase the consumption of cold-water fish, supplement the diet with 480 mg of gamma-linolenic acid as provided from evening primrose, borage, or blackcurrant oil, and consume 1 tablespoon or 10 g of flaxseed oil daily.[1]

Carnitine

Carnitine is an amino acid-like compound that helps the body produce energy. While readily abundant in meats and dairy foods, some people take carnitine in supplement form to increase vitality. Carnitine helps to mobilize fat stores, which are converted into energy that the heart, muscles, and other body tissues can use. In diabetics, carnitine supplementation

has significantly reduced total serum lipid and increased HDL cholesterol levels. In addition, it increases α-oxidation of fatty acids, which may aid in preventing diabetic ketoacidosis.[58]

Inositol

In animal studies, inositol supplementation has successfully treated diabetic neuropathy, since it helps to re-establish normal myoinositol levels in the deficient neuron.[59] A combination of glucose competition with myoinositol for active transport and accumulation of sorbitol within the cell, resulting in the loss of intracellular myoinositol, is thought to be responsible for the neuronal myoinositol deficiency. Oral supplementation in human diabetics has not, however, resulted in significant clinical improvement.[60]

Dietary

Nutrition therapy is an integral component of diabetes management. In general, the main goal of nutrition therapy that applies to all diabetic patients is to attain and maintain optimal metabolic outcomes. This includes blood glucose levels in the normal range or as close to normal as is safely possible to prevent or reduce the risk for complications of diabetes, a lipid and lipoprotein profile that reduces the risk for macrovascular disease, and blood pressure levels that reduce the risk for vascular disease. In addition, we should address risk factors like obesity and dyslipidemia.[61-63]

An appropriate healthy eating plan will assist with:

- control of blood glucose levels
- control of serum lipid levels (cholesterol & triglycerides)
- control of body weight
- preventing short term complications such as hyperglycaemia or hypoglycaemia
- preventing long term complications of the heart, blood vessels, eyes, kidneys and nerves
- improvement of overall health by optimum nutrition

Overall Dietary Principles for Diabetes

1. Increase intake of complex carbohydrates

Including high fibre carbohydrate foods that lead to a slow and gradual rise in blood glucose levels. Carbohydrates are important for providing energy for the body; however diabetics should avoid refined simple carbohydrates. Instead they should choose complex carbohydrates that have a low glycaemic index (GI). Slow acting carbohydrates include:

- Wholegrain bread – such as pumpernickel and wholemeal
- Wholegrain cereals - such as rolled oats, untoasted muesli, barley bran, rice bran
- Fruit - especially apples, pears, oranges, grapes, cherries, grapefruit, peaches, plums, kiwi fruit and firm bananas
- Grains - such as barley, buckwheat and bulgur
- Rice – like brown rice and basmati rice
- All vegetables - especially sweet potato, corn and yam

Try to include at least one slow acting carbohydrate food at each meal.[61-63]

2. Limit saturated fat and have a moderate total fat intake

Excess saturated fat can increase cholesterol and triglyceride levels, which can increase the risk of developing heart and blood vessel disease.This is especially detrimental to people with diabetes as they are at greater risk of cardiovascular disease. Thus it is important for diabetics to eat less saturated fat and have a moderate total fat intake. In addition, many people with Type 2 diabetes are overweight. Keeping fat, especially saturated fat, to a minimum can assist with weight control, also vital for control of blood glucose levels.[61-63]

Saturated fat can be reduced in the diet by:

- Selecting small serves of lean meat, chicken and fish
- Trimming fat from meat and skin from chicken
- Avoiding processed meat and sausages
- Minimising intake of dairy products which are high in fat
- Avoiding snack foods such as cakes, biscuits, chocolates, chips, and pies
- Avoiding takeaway foods which are crumbed, fried, in batter or creamy sauces
- Using low fat cooking methods, such as grilling, microwaving and barbecuing
- Avoiding the use of fats like butter or cream sauces to cook foods
- Maximising intake of healthy fats, such as olive oil, nuts and avocado[61-63]

3. Eat moderate amounts of protein

Protein is needed by the body for growth and repair. However, protein foods often contain fat, thus choose those that are low in fat, such as:

- Dried peas, beans and lentils
- Fish and seafood
- Lean meat, organic where possible
- Poultry – without the skin, organic where possible[61-63]

4. Keep salt intake to a minimum

High salt diets can raise some people's blood pressure, and increase their risk of developing heart disease. Many people with diabetes also suffer from hypertension. Thus where possible, diabetics should choose low-salt, salt-reduced or no-added-salt foods. They should also try not to add any salt to food while cooking or at the table. Instead of salt, use herbs and spices to add flavour instead.[61-63]

5. Consume only moderate amounts of sugar

Sugars do not have as detrimental effect on blood glucose levels as was once thought, and moderate amounts can be included as part of a diabetic's diet. However, confectionary, soft drinks, cordials and other foods that contain concentrated amounts of sugars should be avoided.[61-63]

6. Minimise intake of alcohol

A moderate intake of alcohol may have some health benefits, but larger amounts are detrimental to health. It is recommended that people with diabetes who drink alcohol only drink in moderation. Alcohol recommendations for Australians are no more than 2 standard drinks per day for women and no more than 4 for men. Two alcohol free days are also recommended.[61-63]

Also, it is important for diabetics to remember that medications can react with alcohol. Thus people who are on tablets or insulin for their diabetes should see their doctor to discuss whether they can safely include alcohol in their diet.[61-63]

A Typical Meal Plan for One Day

Breakfast: wholegrain cereal like porridge with rice milk and an apple

Morning tea: one serve of fruit like cherries

Lunch: wholemeal sandwich with a small serve of meat, tuna or chicken and salad

Afternoon tea: a handful of mixed nuts or a serve of fruit

Dinner: A small serve of meat, fish or chicken with one cup of brown rice and vegetables

Supper: one carton of yoghurt

A Note on High Protein, Low Carbohydrate Diet

Some advocates of high protein, low carbohydrate diets suggest that this type of diet can provide benefits ranging from weight loss and disease prevention, to improvements in blood glucose levels. The recommended dietary protein intake for Australians is 0.75g/kg for adults. The maximum protein requirements is around 2.0g/kg per day. A high protein diet advocates up to 4.0g/kg, which is far in excess of what most people need. The dietitians at Diabetes Australia – Victoria do not recommend this diet for both healthy individuals and especially diabetics.[64] In their opinion, potential problems include:

- Too much protein can lead to dehydration through the removal of nitrogen, which requires fluid.
- Protein intake increases calcium excretion, thus an excess of protein results in the excretion of too much calcium, which in turn decreases bone strength.
- With a high protein intake, saturated fat intake may increase. Intake of saturated fat should be minimised by people with diabetes and/or heart disease.
- For people with diabetes, an excess of protein can cause kidney damage, a primary complication of diabetes. People with diabetes who also have renal impairment show a worsening of kidney function with excess protein intake.
- High protein diets may have insufficient carbohydrate to provide enough soluble and insoluble fibre, and B vitamins.
- Low carbohydrate diets have inadequate variety. Eating a variety of foods is important to obtain all of the nutrients needed for good health.
- Obsession with diets that are prescriptive are known to fail in the long term.[64]

Lifestyle

Prevent Weight Gain

Maintenance of a healthy body weight is important for the management of diabetes, as well as reducing the risk of health problems such as heart disease and high blood pressure. In addition to total body weight, where the weight is carried is important. In general, 'apple' shaped people, who carry weight around the middle, are more likely to develop health problems than 'pear' shaped people, who carry weight around the hips and thighs.

Weight loss can be achieved by reducing total calorie intake and increasing physical activity.[63]

Exercise

With emerging research, it is becoming increasingly clear that exercise may be a therapeutic tool in a variety of patients with, or at risk for diabetes.

Regular exercise training has a potential benefit on carbohydrate metabolism, hyperlipidaemia and insulin sensitivity, which can be maintained for at least 5 years. An improvement in insulin sensitivity in turn helps to prevent cardiovascular disease like atherosclerosis, and hypertension. In addition, exercise may enhance weight loss and, in particular, weight maintenance when used along with an appropriate calorie-controlled meal plan. The possible benefits of exercise for the Type 2 diabetic patient are hence substantial, and studies strengthen the importance of long-term exercise programs for the treatment and prevention of diabetic complications.[65]

While the abovementioned benefits of exercise may not affect the Type 1 diabetic patient to the same extent, it can still be part of a general healthy lifestyle program in Type 1 diabetics who do not have complications and are in good blood glucose control. For people with Type 1 diabetes, the emphasis must be on adjusting the therapeutic regimen to allow safe participation in all forms of physical activity consistent with an individual's desires and goals. Ultimately, all patients with diabetes should have the opportunity to benefit from the many valuable effects of exercise.[65]

However like any therapy, the effects of exercise must be thoroughly understood to analyse its risks and benefits in a given patient. Before beginning an exercise program, the individual with diabetes mellitus should undergo a detailed medical examination to screen for the presence of macro- and microvascular complications that may be worsened by the exercise program. By doing so, we can design an individualized exercise plan that will minimise risk to the patient. A careful medical history and physical examination should focus on the symptoms and signs of disease affecting the heart and blood vessels, eyes, kidneys, and nervous system.[65]

There are several considerations that are specific for diabetics. While aerobic exercise is recommended, precautionary measures involving the feet is essential. The use of silica gel, air midsoles and proper socks to prevent blisters and keep the feet dry is vital for minimizing trauma to the feet. Proper footwear is essential, especially for diabetics with peripheral neuropathy. Patients must be taught to monitor closely for blisters and other potential damage to their feet, both before and after exercise.[65]

Another point to consider is proper hydration. Dehydration can affect blood glucose levels and heart function adversely. Thus diabetic patients who are exercising in hot environments must pay special attention to maintaining hydration. Adequate hydration prior to and during exercise is important to compensate for losses in sweat.[65]

High-resistance exercise using weights may be acceptable for young individuals with diabetes, but older individuals or those with long-standing diabetes should avoid this form of exercise. Moderate weight training programs that utilize light weights and high repetitions can be used for maintaining or enhancing upper body strength in nearly all patients with diabetes.[65]

Other Lifestyle Considerations

Everyone who has diabetes should be seen at least once every six months by an endocrinologist. He or she should also ideally see an exercise physiologist for help in developing a physical activity plan, and, perhaps, a social worker, psychologist or other

mental health professional for help with the stresses and challenges of living with a chronic disease.[66]

Everyone who has diabetes should also have regular eye exams once a year by an ophthalmologist to make sure that any eye problems associated with diabetes are caught early and treated before they become serious.[66]

Also, people with diabetes need to learn how to monitor their blood glucose. Daily testing will help determine how well their meal plan, activity plan, and medications are working to keep blood glucose levels in a normal range.[66]

~ End ~