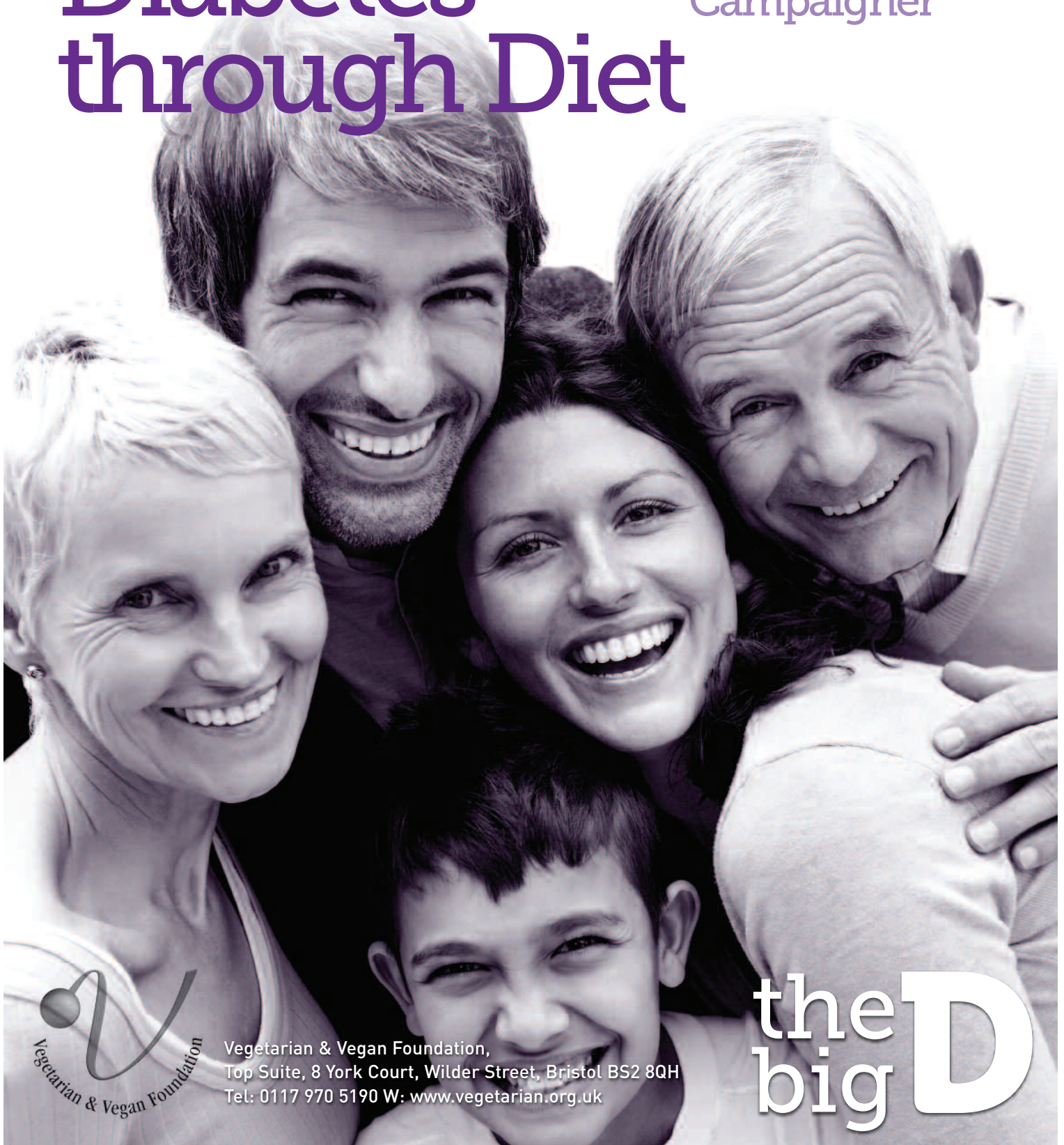


# The Big-D: Defeating Diabetes through Diet

A scientific report  
by Veronika  
Charvatova MSc,  
VVF Health  
Campaigner



Vegetarian & Vegan Foundation,  
Top Suite, 8 York Court, Wilder Street, Bristol BS2 8QH  
Tel: 0117 970 5190 W: [www.vegetarian.org.uk](http://www.vegetarian.org.uk)

the  
big **D**

# The Big-D: Defeating Diabetes through Diet

By: Veronika Charvatova MSc, Health Campaigner,  
Vegetarian & Vegan Foundation (VVF)

Edited by: Juliet Gellatley BSc DipDM, Founder &  
Director, VVF; Tony Wardle, Editorial Director, VVF

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T: 0117 970 5190

E: [info@vegetarian.org.uk](mailto:info@vegetarian.org.uk)

W: [www.vegetarian.org.uk](http://www.vegetarian.org.uk)

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The Vegetarian & Vegan Foundation (VVF) is a science-based charity that promotes human health through the promotion of a vegetarian or vegan diet. The VVF monitors and interprets research that links diet to health – explaining in simple terms how what we eat affects us, in both positive and negative ways. The VVF communicates this information to the media, the public, health professionals, schools and food manufacturers so providing accurate information on which to make informed choices.

# Executive summary

Diabetes has reached epidemic proportions all around the world and numbers are rising. The best available medication and current dietary recommendations are unable to limit the growth of this disease, never mind reverse it. For those who have diabetes, other health complications are likely to result, reducing their quality of life and potentially leaving them with damaged eyes and kidneys, prone to heart attacks and amputations and in constant pain due to neuropathies – which can eventually lead to depression and premature death.

Lifestyle and diet changes are recognised as necessary for diabetes management but none of the mainstream, currently recommended diet changes are truly effective. However, there is a diet that has proved to be effective in reversing diabetes, bringing blood glucose levels under control, inducing weight-loss and significantly improving the health of diabetics – yet it has no official recognition and plays no part in the official UK therapeutic approach. It is a vegan, low-fat, low GI diet which works on basic biological principles and has helped a large number of diabetics over the past 30 years because it strikes at the very core of the condition. The effectiveness of vegan diets is, however, endorsed by the American Diabetes Association when in its 2010 *Clinical Practice Guidelines* it stated that plant-based diets have been shown to improve metabolic control in people with diabetes. It is inexplicable that the UK has not endorsed these findings.

The effects of this vegan diet exceed the results of both medication and conventional diets combined. For this reason, the VVF has introduced its D-Diet, which has no adverse effects of any kind and does not restrict portion sizes. It requires only a change in mindset and the will to learn a new way of eating. If the recommendations of the D-Diet are adhered to, it not only brings blood glucose under control but also helps to prevent or reverse heart disease and other circulatory problems such as raised cholesterol, kidney disease, retinopathy and neuropathy. A number of studies have shown that a vegan diet is effective, well accepted by diabetics who benefit from

it in the short as well as the long term. However, the most important effect of this plant-based approach is its potential to save lives.

Although genes play an important role in the development of diabetes, lifestyle and environmental factors are even more important as they can determine whether the disease actually develops in the first place.

The occurrence of type 1 diabetes is strongly linked to early exposure to cows' milk together with a very short duration of breastfeeding. Individuals with genetic susceptibility are at high risk of developing diabetes if they ingest cows' milk proteins in an infant formula when their intestines are not yet fully capable of digesting such food. This risk is thought to be higher if the immune system has been compromised by a viral or another infection.

In very small children, milk proteins may be incompletely broken down by gut enzymes, pass through the gut wall into the bloodstream where the immune system then reacts to these foreign molecules. As the structure of some milk protein molecules closely resembles molecules found on the surface of insulin producing  $\beta$ -cells in the pancreas, the immune system attacks the body's own cells by mistake. The destruction of  $\beta$ -cells can be relatively fast but usually takes place over a number of years, eventually resulting in type 1 diabetes after all insulin-producing cells have been depleted. It is therefore highly advisable that children are not exposed to cow's (or any other non-human) milk, especially at an early age.

The story of type 2 diabetes is different. It still relates to the production of insulin by the pancreas, but in this case the body stops responding to it as its cells develop insulin resistance. The main culprit in this case is thought to be fat accumulated within muscle cells which influences cell metabolism. This fat accumulation interferes with the cells' insulin sensitivity and causes the decline of mitochondria (the cells' own energy-creating metabolic units).

Obese people, particularly those with abdominal obesity (where most fat is accumulated around their waist) are very likely to develop type 2 diabetes but fat accumulation inside cells can affect anyone regardless of their weight.

Because the process is long-term, insulin resistance develops gradually and people are sometimes first diagnosed with metabolic syndrome or pre-diabetes, both of which are reversible conditions. More often than not, they only discover that they have a disease when diabetes has fully developed, by which time they might already be experiencing other diabetic complications. The condition might still be reversible even at this stage but the longer a person lives with diabetes, the more damage is likely to be done to the body.

Diets limiting carbohydrate intake fail to work because when the body cannot obtain enough energy from carbohydrates, it uses fat instead and this substitution affects metabolism to a large extent, causing increases in blood lipids (fats), including cholesterol. It might seem desirable that the body burns fats but human metabolism is not adapted to fat being the main source of energy. This is highlighted by the fact that diabetics have an increased risk of heart attacks and other related health problems. Unless the diet is really low in fat and uses only vegetable oils, the lipids stored in the body's cells are not metabolised and the condition cannot improve significantly.

Our D-Diet (vegan, low-fat and low GI) helps those with both type 1 and type 2 diabetes. Although the causes of these two conditions are different, patients with either are likely to suffer very similar problems and complications. Type 1 diabetics will always be insulin dependent but they can avoid a number of related debilitating conditions simply by following the D-Diet that enables their body to cope with carbohydrates gradually, whilst avoiding potentially harmful animal fat and animal protein. Type 2



diabetics can follow a similar approach for reversing both diabetes and its associated complications.

One caution! Even though a vegan diet has helped many people to reduce or even eliminate medication, no one should ever make decisions about changes in their medication. This should always be done by their doctor.

The VWF is no longer alone in recommending a low-fat, wholesome, vegan diet as the most effective means of controlling diabetes – a number of experts are now echoing our words. It requires only an initial effort to learn how to shop, cook and eat to successfully follow the D-Diet's recommendations. With no side effects, no restrictions on portion size and no hunger pangs, there is every possibility of witnessing an improvement in the condition, an ability to reduce medication and an almost certain healthy weight-loss.

# What is diabetes?

Diabetes mellitus (the full medical name) is a condition characterised by high levels of glucose (sugar) in the blood which the body cannot properly use and which is eventually excreted in the urine, along with a good deal of water. As glucose passes through the kidneys, it attracts water which results in frequent trips to the toilet to urinate and a feeling of thirst caused by fluid loss. Diabetes is caused either by the pancreas entirely failing to produce the hormone insulin or producing insufficient quantities; or by the body cells' inability to react to insulin.

Insulin is produced by the pancreas and acts as a key, allowing glucose into the body's cells. Glucose is a vital source of energy for cells and is the main fuel for the body's processes. It comes from digesting carbohydrates but is also partially produced by the liver. Carbohydrates are the main nutrients in healthy foods such as wholegrain or rye bread, wholegrain pasta, oats, brown rice, pulses (beans, peas and lentils) and sweet potatoes. They are also in not-so-healthy foods such as white bread, white pasta, cakes, sweets and other sugary foods.

If the body cannot use glucose as a source of energy it uses fat instead, but this inevitably disturbs the biochemical balance of the body and can lead to further health complications. When glucose is prevented from entering the body's cells, they are denied their basic and most important fuel. This can result in tiredness, which is one of the symptoms of diabetes. Other symptoms are irritability, nausea, hunger, thirst, weight loss, blurred vision, tingling sensations in the hands and feet and dry, itchy skin.

In 1985, about 30 million people worldwide had diabetes of both types; a decade later this figure had increased to 135 million and by 2000 an estimated 171 million people had the disease. It is predicted that at least 366 million people will have diabetes by 2030 (Wild *et al.*, 2004). This increase is attributed to a range of factors, including population growth,

Country	Prevalence	Number of people
England	5.4 per cent	2,338,813
Northern Ireland	3.7 per cent	68,980
Scotland	4.1 per cent	223,943
Wales	4.9 per cent	153,175

ageing, unhealthy diets high in saturated fat and cholesterol, obesity and lack of physical exercise.

In the UK alone, 2.8 million people have been diagnosed with diabetes (Diabetes UK, 2010a), while an estimated half a million more have it but have not been diagnosed yet (Diabetes UK, 2010b).

In 2008 alone, 145,000 people were diagnosed with diabetes in the UK – around 400 people every day. According to the latest numbers, it is expected that by 2025 over four million people in the UK will have diabetes. Because of rapid increases in numbers of overweight and obese people, and also because the British population is ageing, most of these cases will be type 2 diabetes (Diabetes UK, 2010b).

In 2011, 85 percent of adult UK citizens with diabetes have type 2 and 15 per cent type 1 (Diabetes UK, 2010b).

## HbA1c & Glucose Blood Levels (Diabetes.co.uk):

HbA1c (%)	Average Blood Glucose (mmol/l)	Stage of diabetes
13	18	Levels of HbA1c above 6.5% are considered diabetic
12	17	
11	15	
10	13	
9	12	
8	10	
7	8	
6	7	HbA1c 6 – 6.5% is considered pre-diabetes or at risk of diabetes
5	5	HbA1c 3.5 – 5.5% is considered normal

Currently, there are two WHO (World Health Organisation) criteria for diagnosing diabetes. One is a test for blood glucose level after fasting – plasma glucose higher than 7.0mmol/l (126mg/dl) indicates diabetes. The other is a test for plasma glucose levels two hours after ingesting a special glucose drink – in this test plasma glucose level higher than 11.1mmol/l (200mg/dl) is considered diabetic (WHO, 2006).

Another criterion to be considered is glycosylated hemoglobin HbA1c, reflecting the average level of blood glucose over a period of weeks. Haemoglobin molecules are one of the main components of red blood cells and when glucose binds to haemoglobin in the bloodstream, HbA1c (glycosylated haemoglobin molecule) occurs. The more glucose in the blood, the more HbA1c will be present. Red blood cells survive for eight to 12 weeks before renewal, therefore by measuring HbA1c an average blood glucose reading can be returned. For non-diabetics, the usual reading is 3.5-5.5 per cent. For people with diabetes, an HbA1c level of 6.5 per cent is considered good control, although some prefer numbers closer to the non-diabetic level (Diabetes.co.uk).

## Type 1 diabetes

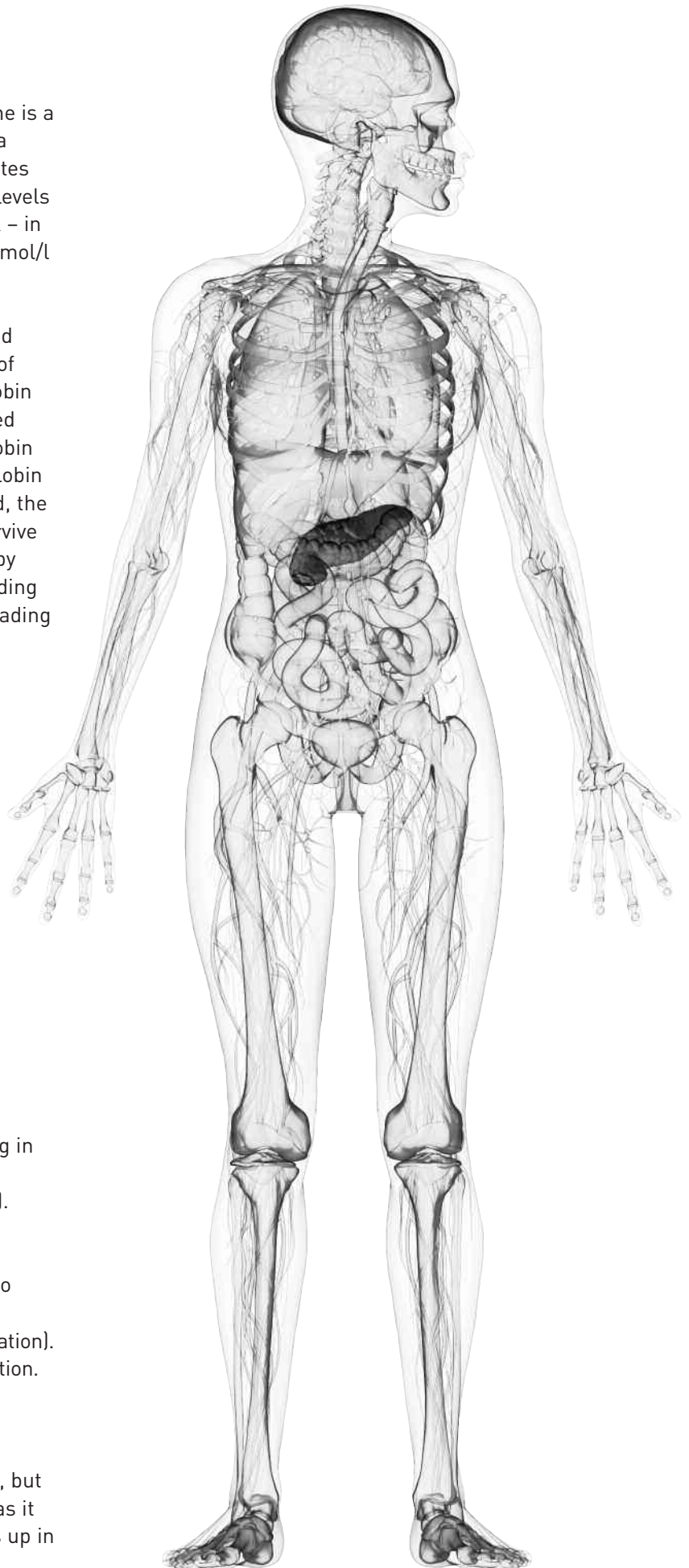
Type 1 diabetes typically develops early in life when the immune system attacks the insulin-producing cells in the pancreas and destroys them. It results in the body being unable to produce any insulin. Because insulin is a vital medium through which glucose is able to enter the body's cells, when it is absent glucose simply builds up in the blood.

Type 1 diabetes accounts for 10 per cent of all people (15 per cent of adults) with diabetes (Diabetes UK, 2010b). The incidence is increasing in all age groups but the rise is particularly steep among children under five years old (NHS, 2008).

Evidence is mounting up that a combination of susceptible genes combined with early exposure to cow's milk are responsible for this self-harming reaction of the body (see page 10 for more information). It might be also triggered by a virus or other infection.

## Type 2 diabetes

In type 2 diabetes, the body can still make some, but not enough, insulin or it fails to react to insulin as it should (insulin resistance). Again glucose builds up in the blood. This condition accounts for



approximately 90 per cent of all cases (85 per cent of adults). (Diabetes UK, 2010b).

Type 2 diabetes usually develops in people over the age of 40, although South Asian and Afro-Caribbean populations are at greater risk and tend to become diabetic relatively early in life – at around the age of 25 (Diabetes UK, 2010b). However, type 2 diabetes is rapidly becoming more common in children, adolescents and young people of all ethnicities. It is predominantly ascribed to the increase in childhood obesity (NHS, 2008).

## Gestational diabetes

This type of diabetes appears in women during pregnancy, usually after the first trimester, but mostly disappears when the pregnancy terminates. In most cases, it arises because the body cannot produce enough insulin for both mother and baby. However, if diabetes occurs during the first trimester, the condition is likely to have existed already before the pregnancy and may continue after it.

Being overweight or obese is a critical factor in contracting type 2 diabetes and puts people at higher risk, so it follows that the same principle applies to pregnant women.

Gestational diabetes affects up to five per cent of all pregnancies (Lancet, 2008). Those women who developed gestational diabetes which subsequently disappeared, are at an increased risk (about 30 per cent) of the disease reappearing later in life (Girling and Dornhorst, 2004).

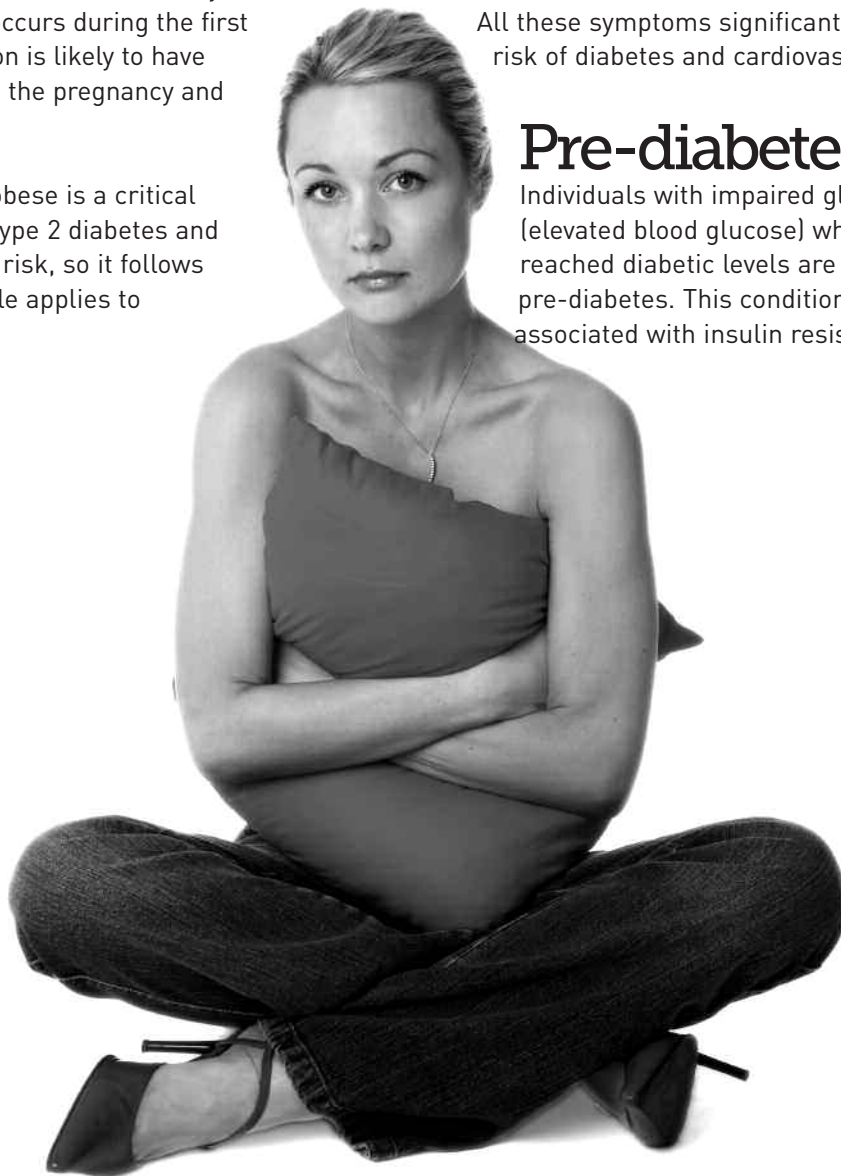
## Metabolic syndrome

Metabolic syndrome, or Syndrome X, is a condition characterised by central obesity (most weight accumulated around the waist), raised blood pressure, raised triglycerides (fats in the blood), low HDL cholesterol (the 'good' type that helps to clear cholesterol from the bloodstream) and impaired glucose metabolism. Impaired glucose metabolism means that the body is not using glucose properly and the level in the blood is elevated but has not yet reached diabetic levels.

All these symptoms significantly increase the risk of diabetes and cardiovascular disease.

## Pre-diabetes

Individuals with impaired glucose metabolism (elevated blood glucose) which has not yet reached diabetic levels are diagnosed with pre-diabetes. This condition is also associated with insulin resistance. The main







risk factor for pre-diabetes is obesity and related risk factors include high blood pressure, low HDL cholesterol and high triglycerides (fats in the blood).

Pre-diabetes almost always precedes type 2 diabetes but many people don't know they have the condition until diabetes has developed. However, pre-diabetes, as well as metabolic syndrome, is reversible with the D-Diet (see page 16).

Diabetes is not usually diagnosed immediately after its onset and by the time the diagnosis is made, many people will have already developed some health complications. However, even people who are aware of their condition and have adjusted their lifestyle accordingly, are still at considerable risk of heart disease, stroke, eye problems, kidney disease, nerve damage and amputations.

## Common health complications

### Cardiovascular disease

Cardiovascular disease (CVD) is a broad term and includes heart disease, stroke and other diseases of the heart and circulation – for example hardening and narrowing of the arteries supplying blood to the legs (peripheral vascular disease). CVD is more common in people with diabetes because high glucose levels in the blood enable fatty deposits to build up more easily on the inner walls of arteries. CVD is also the leading cause of death and disability in people with diabetes.

Because of prolonged periods of poorly controlled blood glucose levels, patients with diabetes also tend to have raised cholesterol and triglyceride levels and higher blood pressure. High blood glucose levels actually cause an increase in the blood lipids (fats) and make cholesterol more likely to stick to the walls of blood vessels. All these complications contribute to worsening CVD but can also cause further complications such as eye and kidney disease (see below).

### Kidney disease

High blood glucose levels and high blood pressure can cause damage to small blood vessels, particularly in the kidneys. Kidneys clean blood by filtering out waste products and excess compounds but are extremely sensitive to damage. Once the damage reaches a certain point, they start to fail. The sign of kidney damage is when protein albumin begins to appear in

the urine. The level of damage is measured by the amount of this protein present in the urine.

Almost one in three people with type 2 diabetes develops kidney disease (Department of Health, 2006) and one in five with type 1 diabetes dies of kidney failure (Morrish *et al.*, 2001).

### Retinopathy

Blood vessels in the eye are just as sensitive to high blood glucose and cholesterol levels and high blood pressure as those in the kidneys. Blood vessels supplying the retina – the seeing part of the eye – can become blocked, damaged, leaky or grow unpredictably. Any such damage or abnormal growth affects the vision and can ultimately lead to blindness. The condition where the retina is damaged is called retinopathy and it is estimated that 4,200 people in England are blind due to diabetic retinopathy (Scanlon, 2008). Almost all people with type 1 diabetes develop some degree of retinopathy within 20 years of diagnosis and almost two thirds of people with type 2 diabetes have retinopathy (Scanlon, 2008).

### Amputation

Any type of diabetes, especially when poorly controlled, can lead to nerve, muscle and sweat gland damage, which can seriously affect circulation and sensitivity in feet and legs and can result in injuries or infections. It can progress to a point where amputation is necessary.

### Depression

Diabetes and the complications that can spring from it, the side effects of medication or daily diabetes management itself can affect the emotional well being of anyone. As a result, depression, eating disorders, anxiety and various phobias can develop.

### Neuropathy

Changing levels of blood glucose can damage nerves which transmit impulses to and from the brain and spinal cord, to muscles, blood vessels, skin and other organs. The nerve damage can cause pain and may also lead to other problems, such as poor sensitivity in the feet making any minor injuries more likely to go unnoticed, which makes the damage more serious. Neuropathy can also lead to erectile dysfunction or impotence.

Neuropathy affects up to 50 per cent of people with diabetes (Boulton, 2005) whilst chronic painful neuropathy affects about one in six – compared to one in 20 non-diabetic people of the same age (Daousi *et al.*, 2004).

# Why do people become diabetic?

## Genetics

Our genetic make-up plays an important role in this disease and a certain set of genes can make us more or less susceptible to developing diabetes. However, even those with a greater susceptibility do not necessarily go on to develop the disease.

### Type 1 diabetes

This is a brief overview of the heredity factors which influence type 1 diabetes (Diabetes UK, 2010b):

- If a mother has type 1 diabetes, the risk for her child of developing it is about two per cent.
- If a father has it, the risk for his child is about eight per cent.
- If both parents have this condition, the risk is up to 30 per cent.
- If a child develops it, the risk is around 10 per cent for other siblings. If a twin develops it, the risk for a non-identical sibling is around 15 per cent. If it is an identical twin, the risk rises to 40 per cent.

Several gene variants have been identified as contributing to type 1 diabetes susceptibility but only a small proportion of genetically susceptible individuals – less than 10 per cent – go on to develop the disease (Knip *et al.*, 2005). This implies that environmental factors are necessary to trigger the autoimmune reaction which destroys insulin producing cells.

### Type 2 diabetes

This is a brief overview of the impact of heredity on type 2 diabetes (Diabetes UK, 2010b):

- If one of the parents has the condition, the risk for their child of developing it is 15 per cent.
- If both parents have the condition, the risk is around 75 per cent.
- If a non-identical twin develops this type of diabetes, the risk is ten per cent for the other twin. If an identical twin has the condition, the risk for the other twin is 90 per cent.

As shown in the next chapter, lifestyle and environmental factors play an enormous role in type 2 diabetes. Therefore, even individuals with susceptible genes, or people who have already developed type 2 diabetes, don't necessarily have to live with the condition for the rest of their lives.

## Lifestyle and environmental factors

Lifestyle plays an enormous role in either increasing or decreasing the risk of developing the disease. Even if both parents have diabetes, it doesn't necessarily follow that the child will develop it, too.

### Type 1 diabetes

It has been known for many years that type 1 diabetes is triggered by some external stimulus. The widely accepted theory is that a viral or another infection might be the culprit, making the body attack its insulin producing cells by mistake. However, the hypothesis that cow's milk is the main trigger was put forward in the 1990's (Karjalainen *et al.*, 1992; Gerstein, 1994; Åkerblom and Knip, 1998) and has been progressively more accepted ever since.

If an individual has a certain combination of genes making them more susceptible to type 1 diabetes, the environmental trigger is the key which opens the door to the disease but if the trigger is avoided, diabetes may never develop. The theory is as follows (Campbell and Campbell, 2004; Knip *et al.*, 2005):

A baby with a susceptible genetic make-up is exposed to cows' milk early in life, perhaps through an infant formula. The baby's immune system might be further compromised by a virus infection, increasing the risk for type 1 diabetes. When the milk proteins reach the small intestine they are not fully digested – i.e. broken down into individual amino acids – but are instead broken down into amino acid chains. These fragments may be absorbed through the gut wall into the blood where the immune system recognises them as foreign intruders and begins attacking them through an



immune response. Coincidentally, the structure of some of these fragments is identical to the surface structure of insulin producing cells ( $\beta$ -cells) in the pancreas (Karjalainen *et al.*, 1992; Martin *et al.*, 1991) and the body cannot distinguish between the two. Pancreas  $\beta$ -cells are therefore attacked and destroyed by the immune system as well as the milk protein fragments and the infant becomes diabetic. Type 1 diabetes is irreversible as the cells cannot regenerate.

The process of  $\beta$ -cell destruction can be fast and aggressive, leading to disease manifestation within a few months, or it can be slow and last for years, in some cases even more than 10 years with  $\beta$ -cells being gradually destroyed over this period (Knip *et al.*, 2005). However, the fast progression of the disease is rare (Knip, 2002).

Research has established which milk proteins are responsible for this dramatic autoimmune reaction. Karjalainen *et al.* (1992) suggest that the main one is bovine serum albumin (BSA), which is different in structure to human albumin (milk protein). They tested the blood of type 1 diabetic and non-diabetic children for the presence of antibodies against incompletely digested BSA. The results were astonishing – all diabetic children had antibody levels as much as seven times higher than the healthy children and there was no overlap in the antibody levels between the diabetic and healthy children – i.e. all diabetic children had high levels but none of the non-diabetic children did.

After that, a number of studies ensued and all but one found markedly elevated levels of BSA antibodies in the blood of diabetic children (Hammond-McKibben and Dosch, 1997).

Another protein abundant in cow's milk is  $\beta$ -casein, which also generates a specific immune response (Cavallo *et al.*, 1996). The structure of human  $\beta$ -casein is similar in many respects to bovine  $\beta$ -casein (from cow's milk) but 30 per cent of the molecule is different in structure. This difference is assumed to be the reason why the immune system reacts to it. Again there are structural similarities between bovine casein and the surface molecules of  $\beta$ -cells in the pancreas, just as there is with BSA, provoking an immunological cross-reactivity – the immune system attacks  $\beta$ -casein molecules as well as the  $\beta$ -cells (Cavallo *et al.*, 1996; Becker *et al.*, 1995).

A Chilean study conducted around the same time focused on the combination of susceptible genes and

cow's milk (Perez-Bravo *et al.*, 1996). The findings revealed that genetically susceptible children weaned too early onto cow's milk-based formula had 13.1 times greater risk of developing type 1 diabetes than children breast-fed for at least three months and who did not have susceptible genes.

In 2000, an extensive study of children from 40 different countries confirmed a link between diet and type 1 diabetes (Muntoni *et al.*, 2000). The study set out to examine the relationship between dietary energy from major food groups and type 1 diabetes. Energy intake per se was not associated with type 1 diabetes but energy from animal sources (meat and dairy foods) showed a significant association whereas energy from plant sources was inversely associated with diabetes. In other words, the more meat and milk in the diet, the higher the incidence of diabetes and the more plant-based foods in the diet, the lower the incidence.

In the meantime, it was discovered that there are five autoantibodies – antibodies which will attack their own host body – and the presence of these autoantibodies can predict the development of type 1 diabetes (Knip, 2002). In addition to the two which attack  $\beta$ -cells, together with two supporting antibodies, there is one which will attack insulin itself. It was suggested that cow's insulin present in formula milk increases the formation of these antibodies (Vaarala *et al.*, 1999). A Finnish study of children at increased risk of type 1 diabetes (having at least one close relative with the disease) showed that the immune system of infants given cow's milk formula as early as three-months old, reacted strongly to cow's insulin by forming these specific antibodies (Paronen *et al.*, 2000).

Results of another study following infants from birth (Åkerblom *et al.*, 2002) showed that exclusively breastfeeding for only a short period followed by the introduction of cow's milk, predisposed these children to  $\beta$ -cell-destroying autoimmune reactions by inducing formation of four culprit autoantibodies. Other population studies have shown that if three or four of these antibodies are present in blood, the risk of developing type 1 diabetes in the next five to 10 years is 60–100 per cent (Knip *et al.*, 2005).

## Type 2 diabetes

Type 2 diabetes often accompanies obesity. In fact, obesity is the main risk factor, particularly abdominal obesity, and over 80 per cent of people with diabetes are overweight or obese (U.S. Department of Health and Human Services, 2007). It has recently been

shown that the association between type 2 diabetes and abdominal obesity is equally significant for both sexes (Paek and Chun, 2010).

According to latest statistics (NHS, 2011), almost a quarter of adults in England (22 per cent of men and 24 per cent of women) were classified as obese. In addition, 44 per cent of men and 33 per cent of women in England were classified as overweight. The numbers of obese children – 16 per cent of boys and 15 per cent of girls (aged two to 15) – are also alarming. With these rising numbers of overweight people, the risk of diabetes is ever-increasing.

Comparing diets and diabetes rates in different countries reveals that as carbohydrate intake goes down and fat intake goes up, the number of diabetics rapidly increases (Campbell, 2004; Barnard, 2007). The difference cannot be ascribed to genetics as when people move to countries where the 'Western' style diet predominates and they adopt these eating

habits, their rates of type 2 diabetes increase above the national average (Tsunehara *et al.*, 1990).

An extensive, 21-year study involving over 25,000 adults in the USA found that diabetes is less frequent among vegetarians and vegans. Those on meat-free diets had a 45 per cent reduced risk of developing diabetes compared to the population as a whole. Meat consumption was positively associated with self-reported diabetes in both males and females (Snowdon and Phillips, 1985).

New research also suggests that eating just one serving of meat per week significantly increases the risk of diabetes (Vang *et al.*, 2008). It looked at the link between meat intake and the occurrence of diabetes in 8,000 adult Seventh Day Adventists Adventists (a Christian branch following a similar lifestyle but different diets), all of whom were non-diabetic at the start of the study. Those who followed a 'low-meat' diet over the 17 years of this long-term study had a staggering 74 per cent increase in their risk of developing

**Percentage of overweight and obese people in England in 2009 (NHS, 2011):**

Age group	sex	Overweight (%)	Obese (%)	Overweight or obese (%)
Over 16	Women	33	24	57
	Men	44	22	66
Two to 15	Girls	13	15	28
	Boys	15	16	31





type 2 diabetes compared to participants who followed a meat-free diet for the same period. Part of this difference was attributable to obesity and/or weight gain but even after allowances were made for this, meat intake remained an important risk factor.

So what is it that makes animal products so detrimental to health? The main enemy is fat.

A study published in 2004 produced an outstanding discovery (Petersen *et al.*, 2004) and confirmed the findings of previous studies (Phillips *et al.*, 1996; Krssak *et al.*, 1999). The researchers tested healthy young adults whose parents or grandparents had had type 2 diabetes for insulin resistance. Some were insulin resistant to one degree or another and further tests uncovered the reason why. Inside their muscle cells were microscopic drops of fat and this fat interfered with the cells' ability to correctly react to insulin. Even though their bodies produced sufficient insulin, fat inside their cells inhibited the appropriate reactions.

Muscle cells normally store small quantities of fat as energy reserve but in the insulin-resistant people, fat had built up to levels which were 80 per cent higher than in other young (healthy) people. Even though the affected people were slim, fat had nevertheless accumulated in their cells. The fat particles were intramyocellular lipids and the study showed (as did previous studies – Phillips *et al.*, 1996; Krssak *et al.*, 1999) that these lipids start accumulating many years before type 2 diabetes manifests.

It was later confirmed by other studies that insulin resistance in muscles and liver is strongly linked to fat storage in these tissues (Delarue and Magnan, 2007; Morino *et al.*, 2006).

Under normal conditions, fat is metabolised by the cells' own powerhouses – mitochondria – but it appears that people with type 2 diabetes have fewer mitochondria in their cells than they need to successfully burn all the supplied fat. As a consequence, the fat accumulates inside the cells (Barnard, 2007).

In order to understand the extent to which diet influences intracellular fat metabolism, another study was conducted (Sparks *et al.*, 2005). Healthy young men (average age 23 years) were put on a special, high-fat diet that drew some 50 per cent of its calories from fat – a diet not too different from that which many people in Western countries consume. After just three days, intracellular lipids had

increased considerably, showing that accumulation of fat inside cells is extremely rapid.

Further tests produced some surprising results – that fatty foods had a profound effect on those genes necessary for the existence and proper functioning of mitochondria. In fact, fatty foods turned off those genes that normally help mitochondria to burn fat. A high-fat diet therefore caused the body to accumulate more fat in muscle cells while at the same time slowing down the body's ability to burn this fat. This dual process then inhibited the ability of the cells to respond to insulin.

Humankind's evolutionary history may provide some answers as to why this happens. When food was predominantly scarce, our ancestors' bodies developed particular mechanisms to store fat on the occasions when they had access to energy-rich food – it was vital for their survival (Barnard, 2007). We live in a very different world now where for most of us food is rarely, if ever, scarce yet our bodies are still programmed to store fat when it is available.

To establish how insulin sensitivity changes when fat intake increases or decreases, and to determine the importance that genetic make-up has on diet, a study of healthy African-American and Caucasian women was conducted (Lovejoy *et al.*, 1998). The women were put on either a high-fat diet (50 per cent fat, 35 per cent carbohydrate, 15 per cent protein) or a low-fat diet (20 per cent fat, 55 per cent carbohydrate, 15 per cent protein) for three weeks. The results showed that insulin sensitivity of all women on the high-fat diet decreased by six per cent, whilst in African-American women on the low-fat diet it increased by six per cent and by 20 per cent in the Caucasian women. The study not only revealed the greater propensity of people on high fat diets to develop diabetes but revealed sharp ethnic differences.

A recent study looked at cell metabolism in relation to insulin resistance more closely (Hoeks *et al.*, 2010) and the conclusions were in line with the above – elevated fat levels in blood and/or intramuscular fat accumulation can cause reduction in mitochondrial function.

The predominant diet in many countries, including the UK, is high in fat, animal products and sugary foods and low in complex carbohydrates. Not only is this diet largely responsible for ever increasing numbers of overweight or obese people but it also increases the risk of diabetes and cardiovascular disease.



# Life with diabetes

Diabetes type 1 is a life-long condition but its progress can be slowed whilst with type 2 diabetes, it can be reversed. In both diseases, health complications can be avoided or reduced simply by adopting the correct approach.

Diabetes treatment is usually highly individual as everyone has different needs, according to their condition, the stage of development of the disease and other health complications. The classical approach to diabetes treatment is based on a combination of adjustments to diet, carbohydrate counting and medication, all of which can limit people's personal and professional lives. Our D-Diet approach is based on a radical change in diet, requires the patient to learn how to eat and how to think about food in a new way but does not require calorie restriction or combining foods from exchange lists.

## Classical approach to treatment

Healthcare professionals dealing with diabetes usually recommend diet adjustments to regulate carbohydrate intake in order to better control blood sugar levels. Because diabetes often goes hand in hand with obesity, they also recommend restricting calorie intake and appropriate physical activity.

### Medication Type 1 diabetes

In type 1 diabetes, the body doesn't produce any insulin so insulin has to be administered in the form of injections or an insulin pump. Depending upon the presence of further complications, type 1 diabetics might also need to take cholesterol and blood pressure-lowering drugs.

### Type 2 diabetes

If changes in diet and exercise don't bring about substantial change, those with type 2 diabetes are usually prescribed oral medication to help reduce high blood glucose levels and stimulate insulin production. These drugs might be insufficient on their own to bring blood glucose under control and are therefore sometimes combined with injectable drugs called incretin mimetics, which influence the body's glucose metabolism, stimulate the production of insulin and reduce the speed of digestion. If the oral and other non-insulin medications are not sufficient, insulin therapy is commenced. Because of the frequent presence of additional health problems, a range of cholesterol and blood pressure-lowering drugs is also taken by many type 2 diabetics.

It is worth noting the many adverse effects of drugs used to regulate insulin sensitivity and/or production (Diabetes UK, 2008):



Name	Trade name	Adverse effects
<b>Sulphonylureas (stimulate insulin production, increase insulin sensitivity)</b>		
Chlorpropamide		Hypoglycaemia, nausea, vomiting, diarrhoea, constipation, loss of appetite, abdominal pain, bloating, indigestion,
Glibenclamide	Daonil	weight gain, liver function problems, blood disorders,
Gliclazide	Diamicon, Diamicon MR	allergic skin reactions, low sodium, headaches, jaundice,
Glipizide	Glibenese, Minodiab	tinnitus, pins and needles, increased sensitivity to
Glimepiride	Amaryl	sunlight, intolerance to alcohol, visual disturbances,
Tolbutamide		confusion, dizziness, drowsiness, tremor, allergic reaction.
<b>Biguanide (helps to stop liver producing new glucose, increases insulin sensitivity)</b>		
Metformin (immediate release)	Glucophage	Nausea, vomiting, diarrhoea, abdominal pain, loss of appetite, metallic taste, reduced absorption of vitamin B12,
Metformin (prolonged release)	Glucophage SR	build up of lactic acid in the blood, allergic reactions, liver function problems.
<b>Prandial glucose regulator (stimulates insulin production)</b>		
Repaglinide	Prandin	Hypoglycaemia, allergic skin reactions, liver function
Nateglinide	Starlix	problems, abdominal pain, nausea, diarrhoea, vomiting, constipation, visual disturbances.
<b>Alpha glucosidase inhibitor (slows down the absorption of starchy foods from the intestine)</b>		
Acarbose	Glucobay	Flatulence, diarrhoea, abdominal pain, nausea, vomiting, indigestion, liver function problems, tissue swelling, blood disorders, allergic skin reaction, intestinal problems.
<b>Thiazolidinediones or Glitazones (reduce insulin resistance)</b>		
Pioglitazone	Actos	Visual disturbance, tissue swelling, weight gain, sinusitis, respiratory infection, numbness, insomnia, liver function problems, increased risk of bone fractures in women.
Pioglitazone/ Metformin	Competact	Blood disorders, visual disturbance, flatulence, weight gain, aching muscles, headaches, blood in urine, impotence, tissue swelling, respiratory infection.
<b>DPP-4 inhibitors or Gliptins (help the hormone incretin to stay in the blood for longer time, which stimulates insulin production and reduces the amount of glucose produced by the liver)</b>		
Sitagliptin	Januvia	Hypoglycaemia, drowsiness, diarrhoea, nausea, flatulence, constipation, upper abdominal pain, tissue swelling, headaches, dizziness, osteoarthritis, arm or leg pain, allergic reactions, weight loss, loss of appetite, respiratory infection.
Vildagliptin	Galvus	Hypoglycaemia, sore throat and runny nose (nasopharyngitis), tremor, headache, dizziness, weakness, constipation, weight gain, tissue swelling, allergic reactions, liver problems.
Vildagliptin/Metformin	Eucreas	Hypoglycaemia, tremor, headache, dizziness, fatigue, nausea.
<b>Incretin mimetics (influence the body's glucose metabolism, stimulate the production of insulin, reduce the speed of digestion and appetite)</b>		
Exenatide	Byetta	Hypoglycaemia, pancreatitis, decreased appetite, headache dizziness, nausea, vomiting, diarrhoea, indigestion, abdominal pain, heartburn, bloating, flatulence, burping, constipation, sweating, feeling jittery, weakness, reaction at injection site, allergic skin reactions, general allergic reaction, kidney function problems, dehydration, unusual taste in the mouth, drowsiness.
Liraglutide	Victoza	Hypoglycaemia, nausea, diarrhoea, vomiting, headache, heartburn, decreased appetite, dizziness, sore throat and runny nose (nasopharyngitis), bronchitis, anorexia, abdominal pain, constipation, gastritis, bloating, flatulence, indigestion, toothache, fatigue, high temperature, reaction at injection site, kidney function problems, general allergic reaction, pancreatitis, thyroid problems.

Singh *et al.* also found that Thiazolidinediones increase the risk of heart failure (Singh *et al.*, 2007).

## Diet

Although there are few diabetes-specific dietary recommendations from clinical practitioners, diabetics are usually recommended to consult a specialised dietician who will give them further advice based on their condition. However, most professionals are likely to prescribe a diet that limits carbohydrate and calorie intake and usually recommend cutting down on certain types of fat whilst encourage the consumption of high-fibre and low glycemic index food (Barnard, 2007; Diabetes Help, 2011; Anderson *et al.*, 2004). This diet might temporarily improve the condition by inducing weight-loss but it does not bring blood glucose levels under long-term control and, sooner or later, medication is usually needed.

People with type 1 diabetes invariably need to learn how to count carbohydrates as they base their insulin doses on the amount of carbohydrate eaten. However, recent recommendations encourage them to eat mostly foods with a low glycemic index to improve their blood glucose control (see page 20 for more information on glycemic index) and to avoid saturated fats (Barclay *et al.*, 2010).

## How well does it work?

Dieticians frequently recommend using food exchange lists, which advocate combining certain types of food at every meal, counting carbohydrates and restricting portion sizes. This kind of diet, together with medication, focuses mainly on glucose management and weight-loss but doesn't limit animal products – the main sources of saturated fat in the diet. So, whilst it may keep blood sugar under control, it allows the body to feast on saturated fats and cholesterol and makes the kidneys work hard in coping with animal proteins.

High animal protein diets force both liver and kidneys to work harder in order to filter nitrogen products out of the bloodstream in the process of producing urine. Kidneys dilate their blood vessels to filter out protein waste – and animal

protein causes greater dilation than plant protein. In addition, plant-derived proteins are lower in sulphur and demand less from the kidney's filtration system. Moreover, fat droplets which accumulate in muscle cells remain exactly where they are and the condition gradually worsens.

## The D-Diet

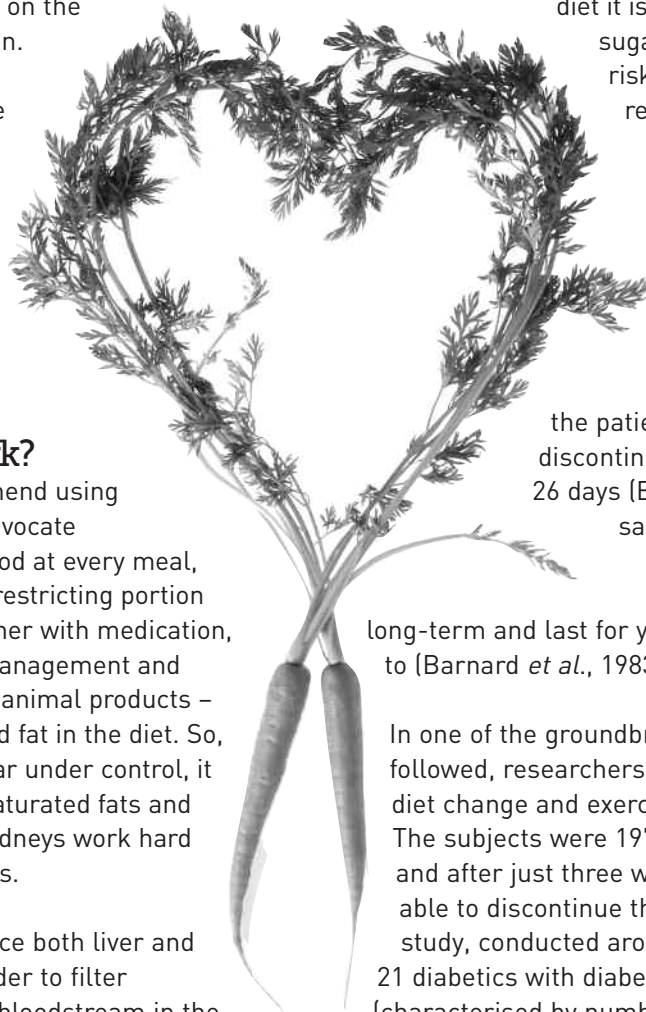
Those with either type of diabetes will benefit from this approach but unlike type 2 diabetics, those with type 1 will always need to take insulin. Nevertheless, type 1 diabetics can use the D-Diet to keep insulin doses to a minimum and markedly reduce their risk of further health complications.

This approach is based on what you eat, not on counting how much you eat because, perhaps surprisingly, what you eat actually regulates how much you eat (see page 20).

Research has shown that with the right diet it is possible to decrease blood sugar, limit medication, cut the risk of complications and even reverse type 2 diabetes.

One of the first studies to test the effects of a plant-based, low-fat diet and exercise on a group of 40 type 2 diabetic patients, had outstanding results – 36 of the patients were able to discontinue all medication after only 26 days (Barnard *et al.*, 1982). The same research group later demonstrated that the benefits of this diet are long-term and last for years, if the diet is adhered to (Barnard *et al.*, 1983).

In one of the groundbreaking studies that followed, researchers employed a combination of diet change and exercise (Barnard *et al.*, 1994). The subjects were 197 men with type 2 diabetes and after just three weeks, 140 of them were able to discontinue their medication. A further study, conducted around the same time, involved 21 diabetics with diabetic neuropathy (characterised by numbness and shooting or burning pains in the lower limbs), who volunteered to follow a vegan, whole food diet and exercise





programme for 25 days. Within 16 days, 17 of the patients reported that the neuropathic pain had been completely alleviated. Although the numbness persisted, it was noticeably improved within the 25 days of the programme (Crane and Sample, 1994).

A 2006 study, conducted by the Physicians Committee for Responsible Medicine with the George Washington University and the University of Toronto, tested health benefits of a low-fat vegan diet emphasising foods with a low glycemic index value and excluding all animal products on people with type 2 diabetes. It was compared to a diet based on the American Diabetes Association (ADA) guidelines which restricted calorie intake and limited carbohydrates (Barnard *et al.*, 2006). Portions of vegetables, grains, fruits and pulses were unlimited.

Over the 22-week study, 43 per cent of the vegan group and 26 per cent of the ADA group reduced their diabetes medications. Furthermore, the vegan group lost an average of almost one stone (13 pounds), compared with just over half a stone (9 pounds) in the ADA group.

Overall quality of this vegan diet was compared to the ADA diet on the basis of the Alternate Healthy Eating

Index (AHEI), which is used to estimate the risk of chronic diseases (Turner-McGrievy *et al.*, 2008). It employs a scoring system which assesses several dietary behaviours and rates food and nutrient intakes. The vegan group improved in every AHEI food category (vegetables, fruit, nuts and soya protein, ratio of white to red meat, cereal fibre, trans fat, polyunsaturated to saturated fat ratio) and significantly improved the overall AHEI score. The ADA group improved in only two categories (nuts and soya protein, polyunsaturated to saturated fat ratio) and did not improve the overall AHEI score of the group. An increase in AHEI score was also associated with decreases in HbA1c value (which measures blood sugar levels over time) and weight.

Following the success of the previous studies, a 74-week clinical trial using a low-fat vegan diet was conducted (Barnard *et al.*, 2009a). Participants were type 2 diabetics and they were randomly assigned a low-fat vegan diet or a diet following ADA guidelines. HbA1c changes from the beginning of the study to 74 weeks, or to the last available value before any medication adjustment, were -0.40 points for the vegan group and +0.01 points for the conventional diet group. In patients whose medication did not need to be adjusted, HbA1c fell 1.23 points over the initial 22



weeks, compared to 0.38 points in the ADA group. Glycemic control, therefore, improved more in the vegan group.

The reduction in triglycerides (fats in blood) in the vegan group was also remarkable as was the decrease in cholesterol levels (-20.4mg/dl in contrast to just -6.8mg/dl in the conventional group). Both groups managed to lose weight but unlike the vegan participants, volunteers on conventional (ADA) diet had restricted calorie intake whilst the vegan group did not.

Parallel to these intervention studies, another research group focused on analyzing dietary patterns of 2,875 volunteers without diabetes and determined their risk of diabetes by repeated measurements of basic indicators – blood glucose, insulin concentrations, cholesterol levels, and waist circumference (Liu *et al.*, 2009). Their findings were clear: consumption of a diet based mainly on plant foods protects against insulin resistance, while refined grains, high-fat dairy, sweet baked foods, candy and sugary soft drinks promote insulin resistance.

A similar study from 2011 (Rizzo *et al.*, 2011) focused on the risk or presence of metabolic syndrome (which often precedes diabetes) and dietary patterns of middle aged and elderly people. The study concluded

that vegetarians had significantly lower values for all risk factors apart from one (HDL cholesterol – which can be explained by the participants' consumption of dairy and eggs) and much lower occurrence of the metabolic syndrome than semi-vegetarians (eating meat occasionally) and meat-eaters.

Volunteers participating in some of the above studies preferred the vegan diet (Barnard, 2007) not only because it was effective but also because they found it better than the diet previously recommended. Participants in the 74-week study were repeatedly asked to rate the acceptability of their diets (Barnard *et al.*, 2009b) and the results showed that patients initially felt more restricted by the ADA diet and at the end of the study reported that the vegan diet was as acceptable as the conventional diet. These findings suggest that following a diet that reverses diabetes is no harder than following a conventionally recommended diet which produces only minor changes in metabolism.

The usefulness of vegan diets was eventually endorsed even by the American Diabetes Association when in 2010, their *Clinical Practice Guidelines* stated that plant-based diets had been shown to improve metabolic control in persons with diabetes (American Diabetes Association, 2010).



# Basic principles of the D-Diet

In this section, the basic principles of a diet used for preventing, treating and reversing diabetes are described together with explanations of each of them. The next section ('Why and how it works' on page 20) then provides a more detailed insight into how this diet influences the body's processes. Anyone switching to the D-Diet should remain in close touch with their doctor as glucose control and insulin sensitivity can improve relatively quickly and there might be need for medication adjustment – a decision that needs to be made by a doctor, never by the patient.

## 1st principle: no to animal products

By eliminating all animal products (meat, fish, dairy, eggs) diabetics avoid eating substantial amounts of fat and their cholesterol intake will be zero. Even lean, white meat and fish contain surprisingly high amounts of fat. For example, 38 per cent of calories from roast chicken and 40 per cent of calories from salmon come from fat (Food Standards Agency, 2002).

Although promoted as a source of omega-3 fats, fish oils also contain cholesterol and a significant proportion of their fat is saturated fat – between 15 and 30 per cent, depending upon the species (Barnard, 2007). Dairy products, even low-fat varieties, inevitably contain fat and most of it is saturated fat. There is no requirement for saturated fats in our diet.

Reducing fat intake is vital for many reasons – in order to reduce the amount of intramyocellular lipid (which interferes with muscle cells' insulin sensitivity), for cardiovascular health and for general weight loss.

This diet excludes all animal products also because animal protein from meat, fish, dairy or eggs places an additional strain on the kidneys and can damage them (Knight *et al.*, 2003; Barclay *et al.*, 2010). Plant proteins do not appear to cause the same problem (see pages 16 and 21). Protecting the kidneys is another key issue on the way to better health.

All foods should be of plant origin and unrefined wherever possible, which means they will be naturally high in fibre and complex carbohydrates, and low in fat (except oils, nuts and seeds). Animal products contain no fibre or healthy carbohydrates while plant foods contain all the essential nutrients we need.

It has been found that avoiding certain foods entirely is easier than attempting to moderate their intake (Trapp *et al.*, 2010). Reducing the intake of problem foods may not have the desired effect and cravings for them may never be lost. Avoiding them entirely, on the other hand, enables the taste to adjust to the new way of eating and problem foods are likely to lose their appeal.

## 2nd principle: low-fat

Even though vegetable oils are better for the body than animal fats because they contain essential fatty acids, less saturated fat and no cholesterol, it is important to keep them to a minimum. In order to reverse or improve the diabetic condition, it is essential to eliminate intracellular fats (Barnard, 2007) but this can only happen if excessive fat consumption is avoided. By eating low-fat foods, the body is still likely to obtain the essential amount of fat it needs, but not more.

One of the pilot studies on the effects of a vegan diet showed that there are important differences between types of fat in a low-fat regime (Nicholson *et al.*, 1999). Researchers compared the potential of a low-fat vegan diet with a conventional low-fat diet. Participants were all type 2 diabetics and they followed the assigned diet for 12 weeks. At the end of the study period, the vegan group had 28 per cent reduction in fasting plasma glucose levels (tests carried out after approximately 12 hours without eating). The reduction in the conventional group was significantly lower – only 12 per cent. The average weight loss was 7.2kg in the vegan group compared to 3.8kg in the conventional group. Medication was reduced in all vegan participants, in one of them completely, whilst there were no reductions in medication in the conventional group.

When individuals with impaired glucose tolerance were tested repeatedly for diabetes and their eating habits were examined (Marshall, *et al.*, 1994), it was found that excessive fat consumption significantly increased the risk of developing diabetes. A recent review of studies on bariatric surgery (a surgical procedure reducing the size of the stomach and gut available for nutrient absorption, performed on severely obese people) (Andreelli *et al.*, 2009) revealed a startling result. Type 2 diabetes can be reversed within days of the surgery, even before any significant weight loss is achieved. The main mechanism for this dramatic change is the sudden decrease of triglycerides and free fatty acids in the blood and rapid reduction of fats in liver and muscle

cells. Such drastic changes are obviously not achievable immediately through diet but the effects of this surgery clearly illustrate the efficacy and importance of a low-fat diet.

The amount of fat per serving should not exceed three grams (or ten per cent of calories from fat). Apart from added oils, diabetics should also limit their consumption of nuts and seeds.

### 3rd principle: low glycemic index (GI)

Glycemic index, or GI, is a measure of the effects of carbohydrates on blood sugar levels. Carbohydrates that break down quickly during digestion and rapidly release glucose into the blood have a high GI. Carbohydrates that break down more slowly, releasing glucose gradually into the bloodstream, have a low GI.

To help the body deal effectively with the carbohydrate content of different foods, those that release their energy slowly should be preferred. Low GI means that after ingestion, blood glucose will not reach high levels, which is exactly what people with diabetes need. It allows them to better control their blood sugar and reduces the likelihood of complications caused by hyperglycaemia, such as retinopathy, neuropathy and nephropathy (kidney disease).

Results from 14 studies on glycemic index show that choosing low GI foods alone reduces HbA1c (for both types of diabetes) by 0.3 to 0.4 percentage points. In some of the studies, the difference was even higher – 0.6 points (Brand-Miller, 2003). The authors concluded that the benefit of low GI food consumption is similar to that offered by medication targeting postprandial (after eating) hyperglycaemia. Another review (Willett *et al.*, 2002) came to a similar conclusion and the authors suggested that low GI foods improve glycemic control and reduce hypoglycaemic episodes in people treated with insulin.

In summary, the D-Diet should contain only foods from plant sources, a minimum of oils and should be rich in foods with a low glycemic index. For these reasons, the D-Diet is based on whole grains, pulses, vegetables, fruit and nuts and seeds (see the D-Diet nutrition basics, page 22, for more information on plant-based nutrition). Whilst limiting the types of food eaten, this

## Glycemic index of selected foods (from Glycemic Index Database and The GI Diet Guide)

Classification GI range Examples

Classification	GI range	Examples
Low GI	55 or less	Most fruits and vegetables, pulses (beans, soya, peas, lentils, chickpeas), barley, buckwheat, hummus, pasta, nuts and seeds, sweet potatoes, dried apricots and prunes, rolled oats, all-bran cereals, wholegrain pumpnickel bread, soya yoghurt and products low in carbohydrates
Medium GI	56–69	wholewheat bread, rye bread, crisp bread, brown rice, basmati rice, corn, porridge oats, shredded wheat, pineapple, cantaloupe melon, figs, raisins, beans in tomato sauce
High GI	70 and above	potatoes, watermelon, pumpkin, white bread, French baguette, white rice, rice cakes, corn flakes, processed breakfast cereals, dates, sugary foods

diet does not limit the amount of food consumed. Being high in fibre and digested gradually, the recommended foods make the consumer feel full sooner and for longer, while calorie intake is reduced by the minimal amount of fat it contains (per volume of food eaten).

## Why and how it works

What we eat has an enormous effect on our metabolism. As we have shown, the accumulation of fat in body cells can seriously damage our health and trigger a whole range of other problems. The D-Diet – a plant-based, wholesome and low-fat diet – works on many levels and for both types of diabetes. This is why:

- 1) It eliminates intramyocellular lipids and thus improves cell metabolism, enabling mitochondria to work properly by removing the fatty obstacles which stand in the way of insulin sensitivity.

A study comparing intramyocellular lipids in muscle cells of vegans and non-vegans, matched for age and body weight, revealed that vegans had 31 per cent less fat in their muscles (Goff *et al.*, 2005). Another study of morbidly obese people who had to undergo gastric bypass surgery (Greco *et al.*, 2002) – which reduced the size of their stomach and shortened the length of intestine available for the absorption of nutrients – showed that the drastic weight-loss that followed had a striking effect on their cells. After the first six months, their intramyocellular lipid levels dropped by 87 per cent and their insulin resistance had largely disappeared.

Obviously, we do not recommend taking such extreme measures but these findings illustrate an important point: insulin resistance is reversible.

As a part of the same study, researchers tested whether a low-calorie diet can also deplete intramyocellular lipids. They found that although weight loss was slower, the effects were equally significant. Nevertheless, the diet focused only on restriction of calories, not on types of food eaten. As other studies have shown, plant-based, low-fat and low GI diets can produce better results in terms of intracellular fat elimination (Barnard, 2007) and improved insulin sensitivity (Lovejoy *et al.*, 1998).

- 2) When refined carbohydrates and fats are avoided and animal products are eliminated from the diet, the risk of heart and circulation-related problems (high blood pressure, raised cholesterol and triglycerides and atherosclerosis) plummets. There is no cholesterol in plant foods. A recent review of studies (Ferdowsian and Barnard, 2009) on vegetarian and vegan diets concluded that a plant-based diet, including nuts, soya and soluble fibre, can reduce LDL (bad) cholesterol by 25-30 per cent – a figure comparable to the effects of statin drugs.
- 3) The kidneys can cope with plant protein much more easily than animal protein. By switching to a vegan diet, the kidneys are less stressed. Research has shown that among people with any degree of kidney damage, consumption of animal protein increases the risk of further kidney deterioration (Knight *et al.*, 2003), while a vegan diet has a protective effect

(Kontessis *et al.*, 1990; Soroka *et al.*, 1998). Anderson *et al.* (2004) also suggested that substituting soya protein for animal protein significantly reduces renal hyperfiltration, a condition that may develop into diabetic nephropathy (kidney disease).

- 4) Many diabetes-associated complications are caused by damage to blood vessels of all sizes by poor blood sugar control (resulting in high glucose levels) and by raised cholesterol levels, which can harm artery walls. A diet based on starchy, high-fibre foods can protect blood vessels by reducing excess cholesterol, and enables the body to digest carbohydrates gradually, therefore preventing peaks in blood glucose levels (Chandalia *et al.*, 2000). This is extremely important especially for the eyes, kidneys and the heart itself because these organs tend to suffer most as the result of diabetes. An investigation into the links between nutrition and retinopathy in diabetics (Roy *et al.*, 1989) found that patients without retinopathy had a significantly higher daily intake of carbohydrates and fibre, and a lower intake of protein, than diabetics with retinopathy.
- 5) The D-Diet also induces the desired effect of weight loss without portion restriction, ensuring that those who follow it need never feel hungry. This is of particular importance as too many dietary restrictions, limitations and a lack of positive results have the potential to induce depression in many diabetics (Diabetes UK, 2010b). An increased intake of fibre slightly decreases the intake of calories. It was suggested that every 14 grams of fibre reduce the calorie intake by approximately 10 per cent (Howarth *et al.*, 2001).



## The D-Diet nutrition basics

A healthy diet to reverse diabetes, or to produce a significant improvement in the condition, should be based on the following food groups.

No. of Servings	Foods	Healthy Portion Size	To Provide
At least 5	<p><b>Fruits</b> Apples, pears, peaches, oranges, kiwi fruit, bananas, berries, grapes, etc. Eaten preferably whole or in smoothies (juices have higher GI because they don't contain fibre).</p> <p><b>And Vegetables</b> Broccoli, cauliflower, spinach, kale, leeks, carrots, peppers, tomatoes, squash, green beans, sweet potatoes, celery, lettuce, cabbage, Brussels sprouts, etc.</p>	<p><b>Fresh fruit:</b> 1 medium piece (the size of a tennis ball)</p> <p><b>Dried fruit:</b> 1-1 ½ tablespoons or 1 golf ball</p> <p><b>Green or root vegetables:</b> 2-3 tablespoons or ½ tennis ball</p> <p><b>Salad vegetables:</b> 1 large cereal bowl or 80g</p>	Folate (folic acid), Calcium, Vitamin A, Vitamin C, Fibre, Iron, Antioxidants
3-4	<p><b>Whole grains</b> Wholegrain pasta, brown rice, bran cereal, oats, rye bread, couscous, grains such as wheat, spelt, barley, millet, quinoa, etc.</p>	<p><b>Cooked brown rice:</b> 2-3 heaped tablespoons or ½ teacup</p> <p><b>Breakfast cereal:</b> 25g or 1 regular sized cereal bowl</p> <p><b>Cooked wholemeal pasta:</b> 1 cup as side dish or 2 cups as main dish</p> <p><b>Wholemeal or rye bread:</b> 2 slices</p>	Energy, Fibre, B Vitamins, Calcium, Iron, Protein
2 or 3	<p><b>Pulses</b> Beans (kidney, pinto, black-eyed, butter, soya), lentils, peas, chickpeas, tofu and low-fat soya and bean products burgers, sausages, mock meat, etc.)</p>	½ cup (cooked)	Protein, Energy, Fibre, Iron, Calcium, Other Minerals
1	<p><b>Nuts or seeds</b> Walnuts, cashew nuts, almonds, pecan nuts, Brazil nuts, pumpkin seeds, sunflower seeds, etc.</p>	1 tablespoon	Protein, Energy, Fibre, Calcium, Other Minerals
Small amounts	<p><b>Vegetable oil</b> Flaxseed, hemp seed or rapeseed oil, used cold; olive oil or rapeseed oil for cooking</p> <p><b>Margarine</b></p>	1 teaspoon per portion	Energy, Vitamin E (oils), Vitamin A & D (fortified margarine), Essential Omega-3 and Omega-6 Fats (flaxseed, soya, walnut, hemp)
At least 1	<p><b>B12 Fortified Foods</b> e.g. fortified soya milk, vitamin B12 fortified breakfast cereal, yeast extract (e.g. Marmite)</p> <p><b>Or B12 supplement</b></p>		Vitamin B12

1-2 litres of water per day (at least eight glasses) should also be consumed as part of a healthy, balanced diet. Tea, especially herbal teas, can be counted as water.

Research shows that results in diabetics who follow a low-fat, wholefood, vegan diet are better than any single drug can produce (Barnard, 2007).

A plant-based diet not only helps with the management of diabetes but is also extremely effective in reducing the common complications associated with it – cardiovascular disease, raised cholesterol, kidney and eye complications. Plant-based means no cholesterol, no animal protein and only a little saturated fat, which puts less strain on the kidneys, can help the heart work better and cuts down the risk of cancer.

A review of 11 scientific papers on diabetes and diets based around high-fibre foods and carbohydrates showed, in every study, that diet can improve blood sugar control and cholesterol levels (Anderson *et al.*, 1987). These findings support a more recent large population study which examined the relationship between dietary factors, insulin resistance and metabolic syndrome (McKeown *et al.*, 2004). It revealed that the greater the intake of high-fibre, whole grain foods with a low GI, the lower the incidence of insulin resistance and metabolic syndrome.

Whole grains are rich in complex carbohydrates, pulses are high in protein and all have a low GI. Virtually all vegetables have a low GI and contain many essential vitamins and minerals, as well as cancer fighting antioxidants.

It is a common misconception that because fruits are sweet, they should be avoided by diabetics. In fact, nearly all fruits have a low GI, the only exceptions being watermelon and pineapple. Moreover, they are rich in antioxidants and contain a wealth of vitamins and minerals.

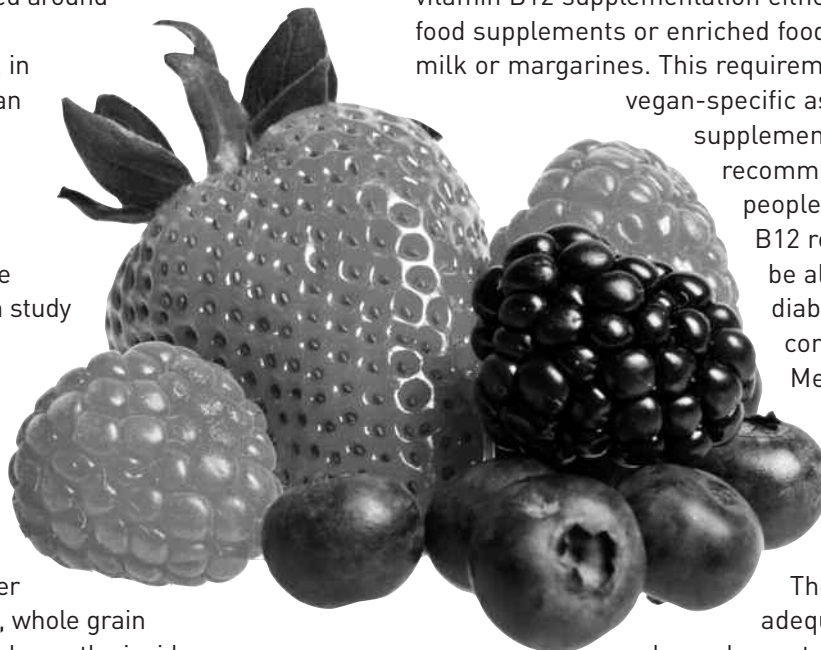
**Important note on grapefruit: grapefruit or grapefruit juice can influence the effects of some medications (Saito *et al.*, 2005). For example, they can increase the**

**blood concentration of some blood pressure and cholesterol lowering drugs, antihistamines and some psychiatric medications. If you are on any kind of medication, it is necessary to consult your doctor on how grapefruit might affect you.**

To ensure sufficient intake of essential omega-3 fats, natural sources should be made part of a daily diet – flaxseed (linseed), hempseed, walnuts and their oils and rapeseed oil. Nevertheless, the body requires only small amounts of these oils, so the low-fat rule still applies, even to these ‘good’ fats.

A vegan diet based on these principles is the healthiest possible, however, there is a need for vitamin B12 supplementation either in the form of food supplements or enriched foods, such as soya milk or margarines. This requirement is not solely

vegan-specific as B12 supplementation is recommended for all people over the age of 50. B12 requirements may be also higher in diabetics as the commonly taken drug, Metformin, can reduce absorption of this vitamin (Diabetes UK, 2008).



The nutritional adequacy of a well-planned, vegetarian or vegan diet has been consistently confirmed. As the latest review concluded (Craig, 2010) – a vegetarian diet, including fortified foods, is nutritionally suitable for adults and children and promotes better health. The same review states that vegetarians have lower body weight, total and LDL (bad) cholesterol, blood pressure, reduced rates of deaths from heart disease; and decreased incidence of high blood pressure, stroke, type 2 diabetes and certain cancers.

A review which focused specifically on diabetes diets (Anderson *et al.*, 2004) came to the conclusion that nutritional therapy is essential for the successful treatment of diabetes and that the most effective diabetes diet is a high carbohydrate, high fibre and low-fat diet, emphasising whole grains, vegetables, fruits, pulses and low GI foods, and soya protein.

# Conclusion

As diabetes continues to spread worldwide, it is essential that an effective approach is adopted for its prevention and treatment. Neither current mainstream recommendations nor the available medication treat the condition successfully.

The D-Diet, based predominantly on low GI foods, has proved highly efficient in inducing the key metabolic changes needed for diabetes reversal. It reduces the ability of intracellular lipids to interfere with glucose sensitivity and prevents high blood cholesterol and other excessive lipids from damaging the heart and blood vessels. It increases the intake of complex carbohydrates vital for slow glucose release and replaces animal protein with plant protein to protect the kidneys. It improves the health of diabetics to such an extent that their medication might need to be lowered or even discontinued and it has the power to reverse conditions such as pre-diabetes and metabolic syndrome.

It has been established that type 2 diabetes is preventable yet there appears to be little knowledge that type 1 diabetes can also be prevented. According to decades of research, cows' milk proteins, ingested by genetically-susceptible young children, can cause a specific immune reaction leading to accidental destruction of insulin producing cells. Type 1 diabetes may therefore be prevented by prolonging the duration of breastfeeding or by using soya infant formula instead of formulas based on cows' milk.

What we eat can have a profound effect on our bodies and choosing the right diet can literally save our lives. Diabetes does not have to be a fatal diagnosis. Scientific research and clinical studies show that lifestyle is the single most important factor in the development of diabetes and it is therefore never too late to try a new – and effective – approach.





# References

- Åkerblom, H.K., Knip, M., 1998. Putative environmental factors and Type 1 diabetes. *Diabetes/Metabolism reviews*. 14(1) 31-67
- Åkerblom, H.K., Vaarala, O., Hyöty, H., *et al.*, 2002. Environmental factors in the etiology of type 1 diabetes. *American Journal of Medical genetics*. 115 (1) 18 – 29
- American Diabetes Association: Standards of medical care in diabetes – 2010. *Diabetes Care*. 33 (Suppl. 1) S11-S61
- Anderson, J.W., Gustafson, N.J., Bryant, C.A., *et al.*, 1987. Dietary fiber and diabetes: a comprehensive review and practical application. *Journal of American Dietetic Association*. 87: 1189-97
- Anderson, J.W., Randles, K.M., Kendall, C.W.C., Jenkins, D.J.A., 2004. Carbohydrate and fiber recommendations for individuals with diabetes: A quantitative assessment and meta-analysis of the evidence. *Journal of the American College of Nutrition*. 23 (1) 5-17
- Andreelli, F., Amouyal, C., Magnan, C., Mithieux, G., 2009. What can bariatric surgery teach us about the pathophysiology of type 2 diabetes? *Diabetes & Metabolism*. 35 (6) 499-507
- Barclay, A., Gilbertson, H., Marsh, K., Smart, C., 2010. Dietary management in diabetes. *Australian Family Physician*. 39 (8) 579-83
- Barnard, N.D., 2007. *Dr. Neal Barnard's program for reversing diabetes: the scientifically proven system for reversing diabetes without drugs*. USA, New York: Rodale Inc.
- Barnard, R.J., Lattimore, L., Holly, R.G., *et al.*, 1982. Response of non-insulin-dependent diabetic patients to an intensive program of diet and exercise. *Diabetes care*. 5: 370-4
- Barnard, R.J., Massey, M.R., Cherny, S., *et al.*, 1983. Long-term use of high-complex-carbohydrate, high-fiber, low-fat diet and exercise in the treatment of NIDDM patients. *Diabetes Care*. 6: 268-73
- Barnard, R.J., Jung, T., Inkeles, S.B., 1994. Diet and exercise in the treatment of NIDDM: The need for early emphasis. *Diabetes Care*. 17 (12) 1469-72
- Barnard N.D., Cohen J., Jenkins D.J., *et al.*, 2006. A low-fat, vegan diet improves glycemic control and cardiovascular risk factors in a randomized clinical trial in individuals with type 2 diabetes. *Diabetes Care*. 29 (8) 1777-83
- Barnard, N.D., 2007. *Dr. Neal Barnard's program for reversing diabetes: the scientifically proven system for reversing diabetes without drugs*. New York, NY, USA: Rodale Inc.
- Barnard, N.D., Cohen, J., Jenkins, D.J.A., *et al.*, 2009a. A low-fat vegan diet and conventional diabetes diet in the treatment of type 2 diabetes: a randomized, controlled, 74-wk clinical trial. *American Journal of Clinical Nutrition*. 89 (5) 1588S-96S
- Barnard, N.D., Gloede, L., Cohen, J., *et al.*, 2009b. A low-fat vegan diet elicits greater macronutrient changes, but is comparable in adherence and acceptability, compared with a more conventional diabetes diet among individuals with type 2 diabetes. *Journal of the American Dietetic Association*. 109 (2) 263-72
- Becker, F., Wasmuth, H.E., Hahnen, J., *et al.*, 1995. Prediction of common epitopes between cow's milk proteins and  $\beta$ -cell antigens. *Autoimmunity*. 21 A342 (abstract)
- Boulton, A.J.M., 2005. Management of diabetic peripheral neuropathy. *Clinical Diabetes*. 23; 9-15. The figure is based on four different studies in which estimates of neuropathy range from 66 per cent in people with Type 1 diabetes over 60 years of age to 41.6 per cent in people who been diagnosed for over seven years.
- Brand-Miller, J., Petocz, P., Hayne, S., Colagiuri, S., 2003. Low-Glycemic Index Diets in the Management of Diabetes: a meta-analysis of randomized controlled trials. *Diabetes Care*. 26 (8) 2261-7
- Campbell, T.C. and Campbell, T.M.II., 2004. *The China Study*. Dallas, Texas, USA: BenBella Books
- Cavallo, M.G., Fava, D., Monetini, *et al.*, 1996. Cell-mediated immune response to casein in recent-onset insulin-dependent diabetes: implications for disease pathogenesis. *Lancet*. 348 (9032) 926-8
- Chandalia, M., Garg, A., Lutjohann, D., *et al.*, 2000. Beneficial effects of high dietary fiber intake in patients with type 2 diabetes mellitus. *The New England Journal of Medicine*. 342 (19) 1392-8
- Craig, W.J., 2010. Nutrition concerns and health effects of vegetarian diets. *Nutrition in Clinical Practice*. 25 (6) 613-20
- Crane M.G. and Sample C., 1994. Regression of diabetic neuropathy with total vegetarian (vegan) diet. *Journal of Nutritional Medicine*. 4: 431-9
- Daousi, C. *et al.*, 2004. Chronic painful peripheral neuropathy in an urban community: a controlled comparison of people with and without diabetes. *Diabetic Medicine*. 21 (9) 976-82
- Delarue, J., Magnan, C., 2007. Free fatty acids and insulin resistance. *Current Opinion in Clinical Nutrition and Metabolic Care*. 10 (2) 142-8
- Department of Health, 2006. *Turning the corner: improving diabetes care*. [online]
- [www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH\\_4136141](http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4136141)
- Diabetes Help: *Diet and Exercise, 2011*. [online] <http://www.diabetes-help.co.uk/diabetes-diet-exercise.php?s=206>
- Diabetes UK, 2008. *Treatments – Tablets*. <http://www.diabetes.org.uk/Guide-to-diabetes/Treatments/Tablets/>
- Diabetes UK, 2010a. [online] <http://www.diabetes.org.uk/Professionals/Publications-reports-and-resources/Reports-statistics-and-case-studies/Reports/Diabetes-prevalence-2010/>
- Diabetes UK, 2010b. *Diabetes in the UK 2010: Key statistics on diabetes*. [online] [http://www.diabetes.org.uk/Documents/Reports/Diabetes\\_in\\_the\\_UK\\_2010.pdf](http://www.diabetes.org.uk/Documents/Reports/Diabetes_in_the_UK_2010.pdf)
- Diabetes.co.uk: *Guide to HbA1c* [online] <http://www.diabetes.co.uk/what-is-hba1c.html>
- Ferdowsian, H.R., Barnard, N.D., 2009. Effects of plant-based diets on plasma lipids. *American Journal of Cardiology*. 104 (7) 947-56
- Food Standards Agency [2002]: *McCance and Widdowson's The Composition of Foods*. Sixth summary edition.
- Gerstein, H.C., 1994. Cow's milk exposure and type 1 diabetes mellitus. *Diabetes care*. 17 (1) 13 – 19
- Girling, J. & Dornhorst, A., 2004. Pregnancy and diabetes mellitus, in Pickup J.C. and Williams G. (ed.) *Textbook of Diabetes*, (3rd edition). Oxford: Blackwell Science
- Glycemic Index Database. [online] <http://www.glycemicindex.com/>

- Goff, L.M., Bell, J.D., So, P.-W., Dornhorst, A., Frost, G.S., 2005. Veganism and its relationship with insulin resistance and intramyocellular lipid. *European Journal of Clinical Nutrition*. 59 (2) 291-8
- Greco, A.V., Mingrone, G., Giancaterini, A., *et al.*, 2002. Insulin Resistance in Morbid Obesity Reversal With Intramyocellular Fat Depletion. *Diabetes*. 51 (1) 144-51
- Hammond-McKibben, D., Dosch, H.-M., 1997. Cow's milk, bovine serum albumin, and IDDM: can we settle the controversies? *Diabetes care*. 20: 897 – 901
- Hoeks, J., van Herpen, N.A., Mensink, M., *et al.*, 2010. Prolonged fasting identifies skeletal muscle mitochondrial dysfunction as consequence rather than cause of human insulin resistance. *Diabetes*. 59 (9) 2117-25
- Howarth, N.C., Saltzman, E., Roberts, S.B., 2001. Dietary fibre and weight regulation. *Nutrition Reviews*. 59 (5) 129-39
- Karjalainen, J., Martin, J.M., Knip, M. *et al.*, 1992. A bovine albumin peptide as a possible trigger of insulin dependent Diabetes mellitus. *New England Journal of Medicine*. 327 (5) 302-7
- Knight, E.L., Stampfer, M.J., Hankinson, S.E., *et al.*, 2003. The impact of protein intake on renal function decline in women with normal renal function or mild renal insufficiency. *Annals of Internal Medicine*. 138 (6) 460-7
- Knip, M., 2002. Can we predict type 1 diabetes in the general population? *Diabetes Care*. 25 (3) 623-5
- Knip, M., Veijola, R., Virtanen, *et al.*, 2005. Environmental Triggers and Determinants of Type 1 Diabetes. *Diabetes*. 54 (Suppl.2) S125-36
- Kontessis, P., Jones, S., Dodds, R., *et al.*, 1990. Renal, metabolic and hormonal responses to ingestion of animal and vegetable proteins. *Kidney International*. 38: 136-44
- Krssak M., Falk Petersen K., Dresner A., *et al.*, 1999. Intramyocellular lipid concentrations are correlated with insulin sensitivity in humans: a <sup>1</sup>H NMR spectroscopy study. *Diabetologia*. 42 (1) 113-6
- Lancet, 2008. The global challenge of diabetes. *The Lancet*. 371(9626) 1723
- Liu, E., McKeown, N.M., Newby, P.K., *et al.*, 2009. Cross-sectional association of dietary patterns with insulin-resistant phenotypes among adults without diabetes in the Framingham Offspring Study. *The British Journal of Nutrition*. 102 (4) 576-83
- Lovejoy, J.C., Windhauser, M.M., Rood, J.C., *et al.*, 1998. Effect of a controlled high-fat versus low-fat diet on insulin sensitivity and leptin levels in African-American and Caucasian women. *Metabolism*. 47 (12) 1520-4
- Marshall, J.A., Shetterfly, S., Hoag, S., Hamman, R.F., 1994. Dietary fat predicts conversion from impaired glucose tolerance to NIDDM. *Diabetes Care*. 17 (1) 50-56
- Martin, J.M., Trink, B., Daneman, D., Dosch, H.-M., Robinson, B., 1991. Milk proteins in the etiology of Insulin-Dependent Diabetes mellitus (IDDM). *Annals of medicine*. 23 (4) 447 – 52
- McKeown, N.M., Meigs, J.B., Liu, S., *et al.*, 2004. Carbohydrate nutrition, insulin resistance, and the prevalence of the metabolic syndrome in the Framingham offspring cohort. *Diabetes care*. 27 (2) 538-46
- Morino, K., Petersen, K.F., Shulman, G.I., 2006. Molecular mechanisms of insulin resistance in humans and their potential links with mitochondrial dysfunction. *Diabetes*. 55 (Suppl. 2) S9-S15
- Morrish, N.J., Wang, S.L., Stevens, L.K. *et al.*, 2001. Mortality and causes of death in the WHO multinational study of vascular disease in diabetes. *Diabetologia*. 44 (suppl 2) s14-s21
- Muntoni, S., Cocco, P., Aru, G. and Cucca, F., 2000. Nutritional factors and worldwide incidence of childhood type 1 diabetes. *American Journal of Clinical Nutrition*. 71 (6) 1525-9
- NHS, 2008. Diabetes in England. [online] <http://www.diabetes.nhs.uk/document.php?o=559>
- NHS, 2010. *Statistics on obesity, physical activity and diet: England, 2010* [online] [http://www.ic.nhs.uk/webfiles/publications/opad10/Statistics\\_on\\_Obesity\\_Physical\\_Activity\\_and\\_Diet\\_England\\_2010.pdf](http://www.ic.nhs.uk/webfiles/publications/opad10/Statistics_on_Obesity_Physical_Activity_and_Diet_England_2010.pdf)
- NHS, 2011. *Statistics on obesity, physical activity and diet: England, 2011* [online] [http://www.ic.nhs.uk/webfiles/publications/003\\_Health\\_Lifestyles/opad11/Statistics\\_on\\_Obesity\\_Physical\\_Activity\\_and\\_Diet\\_England\\_2011.pdf](http://www.ic.nhs.uk/webfiles/publications/003_Health_Lifestyles/opad11/Statistics_on_Obesity_Physical_Activity_and_Diet_England_2011.pdf)
- Nicholson, A.S., Sklar, M., Barnard, N.D., *et al.*, 1999. Toward improved management of NIDDM: A randomized, controlled, pilot intervention using a low-fat, vegetarian diet. *Preventive Medicine*. 29: 87-91
- Paek, K.-W., Chun, K.-H., 2010. Sex difference of type 2 diabetes affected by abdominal obesity versus overall obesity. *Yonsei Medical Journal*. 51 (6) 850-6
- Paronen J., Knip M., Savilahti E., Virtanen S.M., *et al.*, 2000. Effect of cow's milk exposure and maternal type 1 diabetes on cellular and humoral immunization to dietary insulin in infants at genetic risk for type 1 diabetes. Finnish Trial to Reduce IDDM in the Genetically at Risk Study Group. *Diabetes*. 49 (10) 1657-65
- Perez-Bravo, F., Carrasco, E., Gutierrez-Lopez, M.D., *et al.*, 1996. Genetic predisposition and environmental factors leading to the development of insulin-dependent diabetes mellitus in Chilean children. *Journal of Molecular Medicine*. 74 (2) 105-9
- Petersen, K.F., Dufour, S., Befroy, D., *et al.*, 2004. Impaired mitochondrial activity in the insulin-resistant offspring of patients with type 2 diabetes. *New England Journal of Medicine*. 350 (7) 664-71
- Phillips D.I., Caddy S., Illic V., *et al.*, 1996. Intramuscular triglyceride and muscle insulin sensitivity: evidence for a relationship in nondiabetic subjects. *Metabolism*. 45 (8) 947-50
- Rizzo, N.S., Sabate, J., Jaceldo-Siegl, K., Fraser, G.E., 2011. Vegetarian dietary patterns are associated with a lower risk of metabolic syndrome. *Diabetes Care*. March 16 [Epub ahead of print]
- Roglic, G., Unwin, N., Bennett, P.H. *et al.*, 2005. The burden of mortality attributable to diabetes: realistic estimates for the year 2000. *Diabetes Care*. 28; 2130-2135
- Roy, M.S., Stables, G., Collier, B., 1989. Nutritional factors in diabetics with and without retinopathy. *American Journal of Clinical Nutrition*. 50 (4) 728-30
- Saito, M., Hirata-Koizumi, M., Matsumoto, M., *et al.*, 2005. Undesirable effects of citrus juice on the pharmacokinetics of drugs: focus on recent studies. *Drug Safety*. 28 (8) 677-94
- Scanlon, P.H., 2008. The English national screening programme for sight threatening diabetic retinopathy. *Journal of Medical Screening* 15 (1) 1-4
- Singh, S., Loke, Y.K., Furberg, C.D., 2007. Thiazolidinediones and heart failure. *Diabetes Care*. 30 (8) 2148-53
- Snowdon D.A. and Phillips R.L., 1985. Does a vegetarian diet reduce the occurrence of diabetes? *American Journal of Public Health*. 75 (5) 507-512

- Soroka, N., Silverberg, D.S., Gremland, M. *et al.*, 1998. Comparison of a vegetable-based (soya) and an animal-based low-protein diet in predialysis chronic renal failure patients. *Nephron*. 79: 173-80
- Sparks, L.M., Xie, H., Koza, R.A. *et al.*, 2005. A high-fat diet coordinately downregulates genes required for mitochondrial oxidative phosphorylation in skeletal muscle. *Diabetes*. 54 (7) 1926-33
- The GI Diet Guide. [online] <http://www.the-gi-diet.org/lowgifoods/>
- Trapp, C., Barnard, D., Katcher, H., 2010. A plant-based diet for type 2 diabetes: Scientific support and practical strategies. *The Diabetes Educator*. 36 (1) 33-48
- Tsunehara, C.H., Leonetti, D.L., Fujimoto, W.Y., 1990. Diet of second generation Japanese American men with and without non-insulin-dependent diabetes. *American Journal of Clinical Nutrition*. 52: 731-8
- Turner-McGrievy, G.M., Barnard, N.D., Cohen, J., *et al.*, 2008. Changes in nutrient intake and dietary quality among participants with type 2 diabetes following a low-fat vegan diet or a conventional diabetes diet for 22 weeks. *Journal of the American Dietetic Association*. 108 (10) 1636-45
- U.S. Department of Health and Human Services, 2007. *Overweight and Obesity: Health Consequences*. [online] [http://www.surgeongeneral.gov/topics/obesity/calltoaction/fact\\_consequences.htm](http://www.surgeongeneral.gov/topics/obesity/calltoaction/fact_consequences.htm)
- Vaarala, O., Knip, M., Paronen, J., *et al.*, 1999. Cow's milk formula feeding induces primary immunization to insulin in infants at genetic risk for type 1 diabetes. *Diabetes*. 48 (7) 1389-94
- Vang A., Singh P.N., Lee J.W., Haddad E.H. and Brinegar C.H., 2008. Meats, Processed Meats, Obesity, Weight Gain and Occurrence of Diabetes among Adults: Findings from Adventist Health Studies. *Annals of Nutrition and Metabolism*. 52 (2) 96-104
- WHO, 2005. *Diabetes*. [online] Available at: <http://www.who.int/dietphysicalactivity/publications/facts/diabetes/en/index.html>
- WHO, 2006. *Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia*. [online] [http://www.who.int/diabetes/publications/Definition%20and%20diagnosis%20of%20diabetes\\_new.pdf](http://www.who.int/diabetes/publications/Definition%20and%20diagnosis%20of%20diabetes_new.pdf)
- Wild, S., Roglic, G., Green, A., *et al.*, 2004. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care*. 27 (5) 1047-53
- Willett, W., Manson, J.A., Liu, S., 2002. Glycemic index, glycemic load, and risk of type 2 diabetes. *American Journal of Clinical Nutrition*. 76: 274S-80S



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Top Suite, 8 York Court, Wilder Street, Bristol BS2 8QH  
T: 0117 970 5190  
E: [info@vegetarian.org.uk](mailto:info@vegetarian.org.uk)  
W: [www.vegetarian.org.uk](http://www.vegetarian.org.uk)  
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