GLOBESITV

Why plant-based diets are the solution to the world's expanding epidemic

> **By Amanda Woodvine** BSc Nutrition, Health Campaigner

Vegetarian & Vegan Foundation (VVF)



# **CONTENTS**

4	EXE	CUTIVE SUMMARY	16
5	THE	GLOBAL INCIDENCE	
5	DEF	INITIONS	
	5	Assessing weight in adults	

- 6 Assessing weight in children
- 6 Assessing body composition
- **7** THE COST OF OBESITY

#### 9 HOW OBESITY AFFECTS ADULTS

- 9 Atherosclerosis, heart disease and stroke
- 10 Hypertension
- 10 Metabolic syndrome
  - (Insulin resistance syndrome)
- 11 Cancer

#### 11 HOW OBESITY AFFECTS CHILDREN

12 National Diet and Nutrition Survey: Young People Aged 4 to 18 Years

# 12 VEGETARIAN AND VEGAN DIETS FOR HEALTH AND WEIGHT LOSS

14 CAUSATION

**15 ACTIVE OVEREATING** 

**15 PASSIVE OVEREATING** 

38



Published by: Vegetarian & Vegan Foundation, Top Suite, 8 York Court, Wilder Street, Bristol BS2 8OH T: 0117 970 5190 E: info@vegetarian.org.uk W: www.vegetarian.org.uk © Vegetarian & Vegan Foundation 2006

# **CHANGES IN DIET COMPOSITION**

## 16 Fat

- 18 European Commission subsidies
- 18 The National Food Survey
- 20 Are all fats created equal?
- 21 Trans-unsaturated fatty acids
- 22 The role of added sugars, sugary drinks and alcohol

V

- 23 Glycaemic index and glycaemic load
- 24 Energy density and satiety
- 24 Fibre and satiety
- 24 High-protein diets
- 24 Dairy and weight loss

## 25 OTHER SOCIETAL CONDITIONS PROMOTING PASSIVE OVER CONSUMPTION

- 25 Food processing
- 25 Fast foods
- 25 Socio-economic trends in obesity
- 26 Changes in levels of physical activity
  - 26 Adults
  - 26 Children
  - 26 Recommendations
  - 27 10,000 steps per day

26 Globalisation and the rise of globesity

## **28 REFERENCES**

# **36 APPENDIX 1**

# **37 APPENDIX 2**

### **38 APPENDIX 3**

**GLOBESITY** | Why plant-based diets are the solution to the world's expanding epidemic

# **EXECUTIVE SUMMARY**

Obesity is now the most important nutritional disease in the Western world. And in even the poorest countries it is increasing at an alarming rate. For the first time in human history, the number of overweight people worldwide rivals the number of those who are underweight.

Obesity should not be dismissed as a mere cosmetic or moral concern. It is strongly linked to a number of chronic diseases, including heart disease, stroke, cancer, chronic respiratory diseases and diabetes.

With globalisation, people in low and middleincome countries are increasingly adopting Western dietary practices and a more sedentary lifestyle. Their diets are changing from one rich in grains, potatoes and other vegetables and legumes with a modest fat content, to a diet high in total energy, fats, salt and sugar. More and more meat, cheese, butter and other rich milk products and alcohol are being consumed at the cost of lower calorie carbohydrate-rich foods. This socalled 'nutrition transition' has led to both soaring obesity rates and chronic diseases, which blight and cut short the lives of people in low, middle and high-income countries alike. But in poorer countries these diseases tend to strike people at a younger age, leading them to suffer for longer and die sooner than those in the rich West.

The rising prevalence of obesity in children is of particular concern. Because of their excessive weight, an escalating number of children are experiencing health problems previously seen only in those of older years. High blood pressure, raised cholesterol levels and type 2 diabetes are accompanying the towering levels

of childhood overweight and obesity. Some predictions foresee that today's generation of children will die before their parents.

However, one group stands out among the general Western population. This group enjoys remarkably good health, exemplified by low rates of obesity, diabetes, heart disease and cancer, and an increased life expectancy. Obesity is much less common among vegetarians than it is amongst meat eaters. People who are vegetarian or vegan are slimmer than comparable meat eaters. Most overweight people shed pounds when they change to a vegetarian diet. Most importantly, losing weight this way is consistent with longterm health.

The solution to the reducing the prevalence of obesity on a global scale is clear. It shares the same foundations which minimise the risk of chronic disease. Reverting to our roots of a whole foods, plant-based diet rich in the grains, vegetables and legumes which, ironically, are increasingly fed to animals, combined with moderate physical activity can be the keys to both weight maintenance and improved health.

This is of course a long-term lifestyle change, rather than another quick-fix fad.

# THE GLOBAL INCIDENCE

The global incidence of obesity is soaring. More than one billion people in the world are now overweight and at least 300 million of them are clinically obese, according to the World Health Organisation (WHO).1 Overweight and obesity are now even prevalent in some of the poorest countries of the world where they affect 15 to 35 per cent of the adult population<sup>2</sup> – paradoxically, often co-existing with undernutrition. For the first time in human history, the number of overweight people worldwide rivals the number of those who are underweight.3 By WHO predictions, obesity is expected to emerge as a more serious world problem than malnutrition by 2025.

Obesity is particularly rife in the USA, where almost one-third (31 per cent) of adults are now affected.<sup>2</sup> Europe seems to be following this trend, but it is about 10 years behind. This means that in 10 years, rates of obesity in Europe are expected to reach the levels currently seen in North America.

Although it lags considerably behind the USA at present, the UK already has one of the worst rates of obesity in Europe. England ranked sixth and Scotland eighth in a study of obesity levels in 29 European countries.<sup>4</sup> Almost two-thirds of the English population is either overweight or obese, and it is showing one of the fastest accelerations in obesity. Obesity has almost quadrupled in the last 25 years, and if the present trend continues obesity will soon overtake smoking as the leading cause of premature deaths.4

The increasing global prevalence of obesity in children is of particular concern. About 22 million children aged under five are overweight.<sup>1</sup> In England, overweight or obesity affects over one-quarter of under 11s.5 Studies have shown that obesity in childhood and adolescence can persist into adulthood where its health risks are more severe<sup>6,10</sup> - obese children have double the chance of becoming obese adults.<sup>21</sup> Obesity in childhood is associated with a higher chance of premature death and disability in adulthood.<sup>193</sup> Some predictions foresee that today's generation of children will have a reduced life expectancy. This would be the first plummet in longevity seen in England for over a century.

The term 'obesity' is derived from the Latin *ob*, meaning 'on account of', and esum, meaning 'having eaten'. It is most commonly assessed by the body mass index (BMI). BMI is calculated by dividing a person's weight in kilograms (kg) twice by their height in metres (m). Whether a person is defined as underweight, normal weight, overweight or obese depends on which range their BMI falls into (Figure 1 and Figure 2). The normal weight range is taken as 18.5-24.9 kg/m<sup>2</sup>. The World Health Organisation recommends that adults maintain a BMI within this range and avoid weight gain of more than 5 kg (11 lb).114 People with a BMI of below 18.5 kg/m<sup>2</sup> tend to be classed as

•1/

# DEFINITIONS

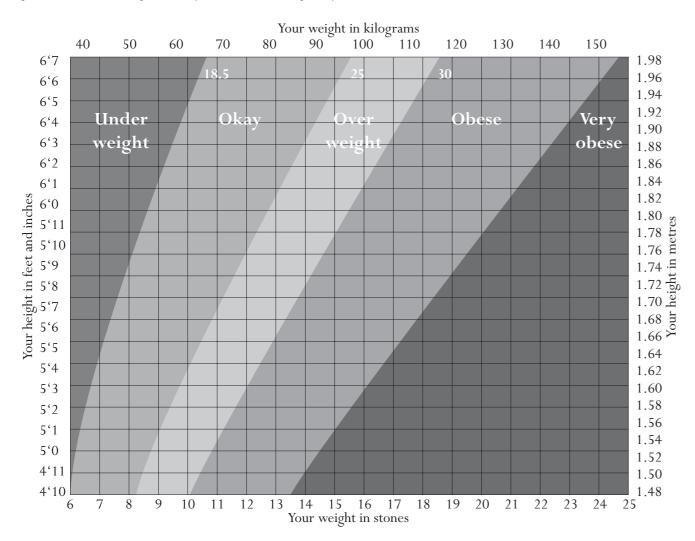
#### Assessing weight in adults

underweight, while a BMI of over 25 kg/m<sup>2</sup> is defined as overweight, and a BMI of over 30 kg/m<sup>2</sup> as obese. This 'normal' range of BMI was primarily calculated using North American mortality data, and so the cut-offs are different for Asian populations. At a given BMI, Asian Indians have seven to 10 per cent higher body fat; accordingly, a BMI of below 23 kg/m<sup>2</sup> is termed optimum; BMI 23 to 25 kg/m<sup>2</sup> overweight, and over 25 kg/m<sup>2</sup> obese in Asian Indians.<sup>279</sup>

Classification	BMI (kg/m²)	Risk of co-morbidity
Underweight	<18.5	Low for the non- communicable diseases associated with obesity, but increased mortality due to cancer and other infectious diseases
Normal range	18.5-24.9	Average
Overweight	25.0-29.9	Mildly increased
Obese		
Class I	30.0-34.9	Moderate
Class II	35.0-39.9	Severe
Class III (morbid)	>40.0	Very severe

Figure 1. WHO classification of overweight and obesity in White adults according to body mass index (BMI)

Figure 2. Assessment of overweight and obesity in White adults according to body mass index (BMI)



#### Assessing weight in children

A different method is used to assess the weight of children. Classifying overweight and obesity in children and adolescents is complicated by their continually changing height and body composition. The International Obesity Task Force (IOTF)<sup>9</sup> uses the BMI 'z score' to determine whether or not a child is on course for being an overweight or obese adult (see **APPENDIX I**). Calculated using international data, their system is based on whether or not a child is likely to reach the BMI cut-off points of 25 and 30 kg/m<sup>2</sup> in adulthood.

#### Assessing body composition

Strictly speaking, however, obesity is not defined as an excess of body weight, but an excess of body fat – to a point that seriously endangers health. Weight gain itself is not the only problem: both body shape and the way that fat is distributed in the body affect the risk of developing certain diseases. The highest risk is seen in people who tend to gain weight around the middle. This characteristic is called abdominal obesity (AO). AO is associated with metabolic syndrome and cancers of many sites, including breast, colon and the kidney (see **HOW OBESITY AFFECTS ADULTS**, page 9). Although the BMI measurement does correlate closely with excess body fat (adiposity), it does not assess a person's precise levels of fat, lean tissue (such as muscle) or water, nor does it identify whether the fat is accumulated in particular sites such as the abdomen where it has more serious consequences as outlined above. A person who was particularly heavy boned or muscular, for example, would have a lower percentage body fat for a given BMI. Thus someone like this might fall into an inaccurately high BMI range, yet be at low risk of the non-communicable diseases associated with obesity.

However, the waist-hip ratio, which is simply a person's waist circumference divided by their hip circumference, is one measure which *does* enable body fat distribution to be assessed. A waist-hip ratio of over one for men and of over 0.8 for women indicates an increased risk of diabetes, cardiovascular complications and related death.<sup>12, 118</sup>

An easy and valid measure of AO is simple waist circumference. A waist circumference of 102 centimetres (cm) or more for men, and 88 cm or above for women is defined as AO.<sup>13, 55</sup> Again, the optimum waist circumference is lower in Asian Indians than Whites with a cut-off of below 90 cm for men and 80 cm for women.<sup>279</sup> A recent study<sup>14</sup> aimed to derive cut-off levels of clothing size corresponding with increased heath risks. It concluded that men's trouser size equal to or larger than 38 in the UK, and women's dress size of 18 or above was associated with increased risk of heart disease, hypertension (high blood pressure) and type 2 diabetes (see **HOW OBESITY AFFECTS ADULTS**, page 9).

Although there are several relatively easy assessment methods available allowing obesity to be identified easily, some patients who are mildly or moderately overweight appear to be overlooked by health care professionals. Recent studies have shown that about one-quarter of overweight patients were thought to be of normal weight by doctors in primary care.<sup>15</sup> Opportunities for the treatment and diagnosis of this life-threatening condition are clearly being missed.

Figure 3. Adult health problems associated with overweight and obesity<sup>214</sup>

System	Effect	
Metabolic	Hyperinsulinism (excessive secretion of insu hyperglycaemia (raised blood glucose levels)	
	Insulin resistance, type 2 diabetes mellitus	
	Dyslipidaemia (a disruption in the amount o	
	Hyperuricaemia (high levels of uric acid in th	
	Syndrome X (a combination of medical disc blood pressure and abdominal obesity)	
Cardiovascular	Hypertension (high blood pressure – a risk t	
	Left ventricular hypertrophy (abnormal thick	
	Arrhythmias (irregular heart beats), sudden	
	Cerebrovascular disease (damage to the blo	
	Endothelial dysfunction (impaired function o	
	Low-grade chronic inflammation	
	Increased sympathetic activity	
Haematological	Impaired fibrinolysis (impaired breakdown c	
	Procoagulant state (blood stimulated to form	
	Hyperviscosity (increased blood 'thickness')	
	Atherothrombosis (the partial or complete related to a blood clot)	
Endocrine	Hirsuitism (increased hair growth in areas w	
	Elevated adrenocortical activity	
	Disturbances in circulating sex steroids and	
	Infertility	
	Polycystic ovary syndrome (multiple cysts in	
	Breast cancer	

# THE COST OF OBESITY

Overweight and obesity can open the gateway to many health problems. These can be non-fatal but debilitating, such as difficulties with physical activity, sexual problems (both psychological and physical), infertility, birth complications, incontinence, respiratory difficulties, heat intolerance, increased sweating and skin problems.<sup>8,213</sup> Low self-esteem, self-loathing and phobias are common in overweight people, who are frequent targets of discrimination, even in the health sector.<sup>215</sup>

¶/

The more life-threatening problems are of four main types: cardiovascular; conditions associated with insulin resistance such as type 2 diabetes; cancers, especially those which are hormonally related or affect the large-bowel; and gallbladder disease.<sup>213</sup> **Figure 3** and **Figure 4** contain a more comprehensive list of overweight and obesity-related health problems in both adults and children.

ulin, a hormone that regulates carbohydrate metabolism), s)

of lipids (fat) in the blood)

the blood), gout

orders including type 2 diabetes mellitus, insulin resistance, high

factor for coronary heart disease, stroke and kidney disease)

ckening of heart muscle), congestive heart failure

death

ood vessels in the brain), stroke

of cells lining blood vessels)

of blood clots)

m clots)

blocking of blood vessels), thrombophlebitis (vein inflammation

where it is normally minimal/absent)

binding globulins

n the ovaries)

Figure 3. continued			
Gastrointestinal	Hiatus hernia (the protrusion of the upper part of the stomach into the thorax through a tear or weakness in the diaphragm)		
	Gastroesophagic reflux		
	Gallstone formation, gallbladder hypomotility and stasis (risk factors for gallstone formation)		
	Gallbladder carcinoma		
	Steatosis (abnormally large quantities of fat within cells), cirrhosis		
	Colorectal cancer		
Respiratory	Restrictive ventilatory pattern		
	Shortness of breath in exercise and/or at rest		
	Obesity hypoventilation syndrome (inadequate breathing)		
	Obstructive sleep apnoea (a sleep disorder with irregular breathing at night and excessive sleepiness during the day)		
Renal	Proteinuria, albuminuria (an excess of blood proteins in the urine)		
	Enhanced sodium retention		
	Renin-angiotensin-aldosterone system stimulation (stimulation of the hormone system that helps regulate blood pressure)		
	Disturbed Na/K ATPase activity, NA/K co-transport (disrupted salt balance)		
Genitourinary	Incontinence		
	Prostate/endometrial/ovarian cancer		
Locomotor	Nerve entrapment		
	Low back pain, joint damage		
	Osteoarthritis (inflammation of the joints)		
Dermatological	Increased sweating		
	Oppositional intertrigo (skin disorder found in creases of neck, the skin folds of the groin, armpits or breasts or between the toes)		
	Wound dehiscence (wound re-opening)		
	Lymphoedaema (swelling that occurs when lymph fluid does not fully drain away from the tissues)		
	Acanthosis nigricans (brown/black velvety hyperpigmentation of the skin)		

Figure 4. Physical consequences of childhood and adolescent obesity<sup>276</sup>

Organ System	Obesity-related disorders
Pulmonary	Sleep apnoea (a sleep disorder in which the child has irregular breathing at night and is excessively sleepy during the day)
	Asthma
	Pickwickian syndrome (hypoventilation due to obesity)
Orthopaedic	Slipped capital epiphyses (a type of bone fracture)
	Blount's disease (causes bowlegs in children)
	Tibial torsion (twisting of the bone between the knee and the ankle)
	- Flat feet
	Increased risk of fractures

Neurological

Idiopathic intracranial hypertension (increased pressure in the brain and cerebrospinal fluid)

Figure 4. continued

Gastroenterological	Cholelithiasis (stones in the gallbladder or bile	
	Gastrooesophageal reflux	
Endocrine	Insulin resistance/impaired glucose tolerance	
	Type 2 diabetes	
	Menstrual abnormalities	
	Polycystic ovary syndrome (multiple cysts in t	
	Hypercorticism	
Cardiovascular	Hypertension (high blood pressure)	
	Dyslipidaemia (a disruption in the amount of	
	Fatty streaks	
	Left ventricular hypertrophy (abnormal thick	
Other	Systemic inflammation/raised C-reactive prot	

Weight tends to increase with age and the health risks increase with the length of time at an excessive weight.<sup>114</sup> A BMI of 40 kg/m<sup>2</sup> is associated with a decreased life expectancy of around 10 years. Being obese from the age of 40 has been observed to reduce life expectancy by about seven years – comparable with the impact of smoking 20 cigarettes a day.<sup>114</sup>

At least 2.6 million worldwide deaths each year are a result of overweight or obesity<sup>193</sup> and the estimated economic costs of obesity and overweight are a conservative £6.6-7.4 billion per year;<sup>4</sup> accounting for more than five per cent of all health costs.<sup>16</sup>

# HOW OBESITY AFFECTS ADULTS

Atherosclerosis, heart disease and stroke

Obesity, particularly AO, is associated with a significantly increased risk of atherosclerosis – the build up of lipids (fat, including cholesterol and triglycerides) and other cells, such as blood cells, on the artery wall.<sup>217</sup> Such 'plaques' can restrict the blood supply to organs and tissues. They may also rupture, causing the organs to which they supply blood to die. If atherosclerosis affects the arteries supplying the heart muscle then chest pains (angina) or heart disease can result. When a similar disease process affects the blood supply to the brain, the result is a stroke.<sup>245</sup>

The risk of developing heart disease is at least doubled, and risk of stroke is six times greater with a large waist measurement.<sup>19</sup> This is because obesity and abdominal fat are linked to high levels of harmful (LDL) cholesterol, low levels of protective (HDL) cholesterol and high triglycerides. Obese

8

the ovaries)

f lipids (fat) in the blood)

kening of heart muscle)

tein (a marker of inflammation in the blood)

people and those with abdominal fat also tend to have more blood clotting proteins (fibrinogen) in their blood. Less indication of damaged blood vessel repair (fibrinolytic activity) is also a common finding in obese individuals.<sup>19</sup>

•//

As well as losing weight, a range of dietary patterns appears to influence the development of heart disease. Certain harmful types of fat can elevate cholesterol levels. These are saturated fat components called myristic acid and palmitic acid which are found in animal fats such as butter and in coconut and palm oils (see **Are all fats created equal?**, page 20). A third saturated fat component called lauric acid has a similar, albeit lesser, effect.<sup>245</sup>

Trans fats, found in deep-fried fast foods, baked goods and, in low natural levels, in dairy products, lamb and beef fat<sup>157</sup> are, gram for gram, associated with an even higher risk of heart disease than saturated fat – the risk is anything from two-and-a-half to tenfold higher.<sup>156</sup> No safe limits of trans fat consumption have been shown.<sup>157</sup>

While some dietary components are harmful, certain foods can be heart protective. These include high intakes of raw or appropriately prepared fruit and vegetables which contain antioxidants: notably beta-carotene, vitamin E and vitamin C, and substances known as flavonoids (found especially in berries). LDL cholesterol is particularly damaging to arteries when it is oxidised. LDL oxidation can happen when naturally occurring, unstable molecules called free radicals are encountered. The antioxidants and flavonoids found in fruit and vegetables help to destroy such free radicals.<sup>255</sup> The consumption of wholegrain cereal is linked to lower risk of hypertension, heart disease, stroke, and deaths from cardiovascular disease.<sup>218-234</sup> Wholegrains have been the staple food worldwide for centuries, especially among vegetarians.<sup>237,</sup> <sup>238</sup>A 25 to 30 per cent reduction in stroke has been observed with the intake of wholegrains – which is similar in magnitude to the effect of statins.<sup>234-236</sup>

Several studies have shown an association between frequent nut consumption and reduced risk of heart disease.<sup>247-253</sup> Nuts are high in unsaturated fats and low in saturates, and may help lower cholesterol by contributing a better fat balance to the overall diet (see Are all fats created equal?, page 20).

Soya protein also has a favourable effect on several cardiovascular risk factors.<sup>254</sup> Its beneficial effect on harmful (LDL) cholesterol, triglycerides and possibly on protective (HDL) cholesterol has led to the US Food and Drug Administration's approval of a health claim that '25 g of soya protein a day, as part of a diet low in saturated fat and cholesterol, may reduce the risk of heart disease'.<sup>254</sup>

The British Medical Association acknowledged in its 1986 report that vegetarians tend to have lower rates of obesity, cholesterol levels, and heart disease - and that meat eaters could lower their cholesterol levels by switching to a vegetarian diet.<sup>211</sup> These findings were more recently echoed in research published in The Lancet<sup>110</sup> which showed that a vegetarian diet, together with other healthy lifestyle changes, could reopen blocked arteries in 82 per cent of research participants, without the use of either surgery or cholesterollowering drugs.

Dr Neal Barnard, president and founder of the Physicians Committee for Responsible Medicine in Washington DC states that: "...chicken-and-fish diets are not low enough in fat or cholesterol to do what vegetarian diets can... The leanest beef is about 28 per cent fat, as a percentage of calories. The leanest chicken is not much different, at about 23 per cent fat. Fish vary, but all have cholesterol and more fat than is found in typical beans, vegetables, grains, and fruits, virtually all of which are well under 10 per cent fat. So while white-meat diets lower cholesterol levels by only about five per cent,<sup>112</sup> meatless diets have three to four times more cholesterol-lowering power, allowing the arteries to the heart to reopen."

Along with weight loss and changing to a predominantly plantbased diet, cutting down on (or cutting out) alcohol, stopping smoking and cutting down on salt (sodium) - which essentially means cutting down on processed foods - are all heart protective actions. Exercise also has a beneficial effect.

10

#### **Hypertension**

The danger of developing raised blood pressure (hypertension) increases by up to six times with obesity.<sup>17</sup> Blood pressure tends to rise with both increasing waistlines and with the degree of obesity. The cause of this rise in blood pressure seems to be insulin resistance and excess insulin in the blood (hyperinsulinaemia). Insulin resistance is explained further below (see Metabolic syndrome, below). Insulin stimulates salt (sodium) re-absorption in the kidneys, rather than promoting its passage out of the body into the urine, which can cause the blood pressure to rise.<sup>17</sup>

Weight loss has been shown to lower blood pressure and lessen the need for blood-pressure lowering (antihypertensive) drugs in clinical trials.<sup>18</sup> Even a small weight loss can markedly reduce blood pressure, and weight loss is a much more effective treatment than salt restriction.

Aside from weight loss, increasing potassium intake and reducing salt intake is a common strategy for reducing blood pressure. Fruits and vegetables are rich in potassium and their liberal intake is recommended for the prevention and treatment of hypertension.<sup>239</sup>

Numerous studies have shown vegetarians to have lower blood pressure - with up to half of the risk of hypertension compared with meat eaters.<sup>240, 241, 242, 243</sup> The introduction of meat to the diet of vegetarians has been found to increase blood pressure by 10 per cent in as little as two weeks.<sup>244</sup>

## Metabolic syndrome (Insulin resistance syndrome)

The metabolic diseases of obesity, insulin resistance/diabetes (reduced sensitivity to the action of insulin), hypertension, raised LDL, triglycerides and reduced HDL cholesterol, and abdominal obesity often appear in a cluster, with two or more of them being present in the same person.<sup>256</sup> The term 'metabolic syndrome' is used to describe these diseases when they occur concurrently like this. They share the common factor of insulin resistance.

The more severe form of insulin resistance is type 2 diabetes. Although people with type 2 diabetes can make insulin - a hormone which lowers blood sugar levels - their cells are insensitive to its effects. Insulin helps the transport of glucose (sugar) across cell membranes. If cells lose their sensitivity to insulin, the result is that glucose (sugar) cannot enter the cells which need it for energy, and the individual may feel incredible hunger, despite having high blood sugar levels.

Insulin has other metabolic roles. For example, it stimulates fat cells to make fats from fatty acids, and it stimulates the liver and muscle to make protein from amino acids. This

leads to less fatty acids and amino acids in the blood. Complications of diabetes include heart disease and stroke, blindness, kidney disease, nervous system disorders, dental disease and limb amputation.258

Approximately 90 per cent of people with type 2 diabetes are overweight or obese<sup>257</sup> although exactly how overweight and obesity contribute to the metabolic syndrome is unclear.<sup>259</sup> Hormones and other substances secreted by the body's fat stores are thought to be the main cause.<sup>259</sup>

Treatment for metabolic syndrome is usually weight loss and physical activity<sup>259</sup> whereas diabetes itself can be treated with a high-fibre vegetarian diet. People who already follow this type of diet have just under half (45 per cent) the chance of developing the disease. However, people who eat meat six or more times per week have an almost fourfold chance of developing diabetes.<sup>260</sup>

The American Dietetic Association states that diabetes is much less likely to lead to death in vegetarians compared with meat eaters, and ascribes this to the higher intake of complex carbohydrates and lower weight amongst vegetarians.<sup>260, 261, 262</sup> A plant-based diet can also eliminate or even reduce a diabetic's need to medicate and it reduces the chance of nerve and eye (retina) damage.263-267

#### Cancer

Obesity and cancer are strongly linked.7 Raised BMI increases the risk of cancer of the oesophagus, colon, kidney, gallbladder, breast, cervix, endometrium and prostate.<sup>268, 269</sup> A body weight excess of over 40 per cent leads to a 50 per cent higher risk of cancer compared to maintaining a normal weight.269

Several mechanisms have been proposed to try and explain these findings. The increased risk for oesophageal cancer may be related to reflux of the stomach (gastric) contents back into the oesophagus.<sup>269</sup> The mechanisms by which obesity increases colon and kidney cancers are less well understood. Cancers of the breast and endometrium are almost certainly related to sex hormones.<sup>269</sup> Fat (adipose) tissue is a major source of production of the female hormone oestrogen among postmenopausal women. Obese women have higher blood levels of oestrogens than women of normal weight.<sup>269</sup> High blood levels of oestrogens stimulate the growth and division of the cells in these female tissues.62

Aside from losing weight, certain dietary factors can predispose or protect against cancer. The World Health Organisation lists these as saturated fat, which has a role in the development of breast, prostate, colon and rectum cancer; fruit and vegetables (which contain certain antioxidant

Many studies have found that eating more fruit and vegetables contributes to vegetarians' better chances but doesn't fully account for it, which indicates that there might be something in meat which acts as a cancer trigger.<sup>273, 274</sup> Indeed, potent cancer forming compounds (carcinogens) called heterocyclic amines and polycyclic aromatic hydrocarbons have been found in grilled or barbecued meat and fish.<sup>164</sup> Nitrates used in cured and smoked meats form carcinogenic substances in our bodies, too.<sup>164</sup> Researchers in the US looked at the carcinogens formed in cooking and found beef burgers produce 44 times more carcinogens than soya-based burgers. Bacon came top of the stakes, producing 346 times more.275

Because of their excessive weight, an escalating number of European children are experiencing the classic health problems previously seen only in those of older years.<sup>277</sup> High blood pressure, raised cholesterol levels, impaired insulin sensitivity and other risk factors (the metabolic syndrome) are accompanying rising levels of childhood overweight and obesity. Type 2 diabetes (which was once commonly termed 'maturity-onset diabetes' as it formerly affected only adults) now affects an estimated 2,000 to 10,000 children in Europe.277

vitamins, minerals and compounds such as flavonoids) which offer protection from oral cavity, oesophagus, stomach, bladder, colon, rectum, lung and cervix cancers; and high calorie and milk and beef fat intake are linked to deaths from breast cancer. Certain other milk components have been linked to cancers of the breast, bowel, ovaries and prostate (for a full discussion see the VVF's White Lies report).<sup>337</sup> Breast cancer risk seems to be reduced by certain components in plants, such as isoflavones derived from soya beans and lignans derived from wholegrain products.<sup>269</sup> Lycopene, the red pigment in tomatoes, may significantly reduce prostate cancer risk.164

Both the American Dietetic Association and the British Medical Association have found that vegetarians are less likely to develop certain cancers. The Oxford Vegetarian Study in 1994 concluded that vegetarians have a 40 per cent less chance of dying from cancer compared with meat eaters<sup>270</sup> Other studies have shown the risk to be reduced by between 25 and 50 per cent.<sup>271, 272</sup>

# HOW OBESITY **AFFECTS CHILDREN**

The 1991 Bogalusa Heart Study demonstrated that even mild obesity in children can lead to higher blood pressure, insulin and cholesterol levels, and that these track into adulthood to some degree. And evidence that atherosclerosis starts to

develop in childhood emphasises the importance of preventative dietary and lifestyle measures in early life.<sup>278</sup>

However, the most widespread consequences of childhood obesity are psychological and social. Obese children often become targets of discrimination. Studies have shown that 10 to 1 l-year-old boys and girls would prefer to befriend children with a wide variety of disabilities in preference to their overweight peers.<sup>65</sup> Additionally, children ranging from six to 10 years of age already associate obesity with a variety of negative characteristics such as laziness and sloppiness.<sup>66</sup> Although overweight young children do not have a negative self-image or low self-esteem67,68 obese adolescents develop a negative self-image that appears to last in adulthood.<sup>69</sup> A proposed explanation for this apparent discrepancy between children and adolescents is that self-image in young children comes from parental messages but as children become adolescents, self-image develops increasingly from society.

Swedish studies have shown obesity to be associated with parental neglect – with dirty and neglected children being at a much greater risk of obesity in adulthood than averagely groomed children.<sup>70</sup> A link has also been demonstrated between rapid weight gain in children and behavioural and learning difficulties.71

## National Diet and Nutrition Survey: Young People Aged 4 to 18 Years

A national diet and nutrition survey published in June 2000 looked into the eating habits of over 2,000 young people aged 4 to 18 years. It revealed that the diets of many children are low in many of the vital vitamins and minerals which are needed to help combat disease yet high in fat, salt and sugar convenience foods. The latter foods tend to be calorie-dense yet nutrient poor (see **Energy density and satiety**, page 24). Much of their daily nutrient intakes are gained from processed cereal, meat and dairy products with woefully low intakes of fresh fruit and vegetables. Meat and dairy products are central to most meals. Around one quarter of all children reported being unwell on at least one day during the seven-day dietary recording period.

A recent study found that vegetarian preschool children had in many ways a better nutritional profile than those who ate meat products. Compared to omnivore children, vegetarian children had lower intakes of total and saturated fat, cholesterol and sodium and higher intakes of beneficial nutrients such as potassium and the antioxidant vitamins beta-carotene (the precursor to vitamin A), C and E. The vegetarian children also ate more fruits and vegetables than their meat-eating counterparts.281

# **VEGETARIAN AND VEGAN DIETS** FOR HEALTH AND WEIGHT LOSS

There are two main ways that a person can lose weight. We can either consciously reduce calorie intake by restricting the amount of food that we eat, or make the transition away from eating junk foods – foods that are high in calories but low in nutrients - and towards eating foods which are nutrient-dense but relatively low in calories, such as fruits, vegetables, beans and whole grains. Nuts can also be added to the list, since, despite their calorie density, a scientific review in 2003 concluded that eating nuts every day certainly doesn't promote weight gain and might actually help people to lose weight.<sup>84</sup> Such foods are staples in a vegetarian diet.

Western vegetarians generally consume a healthier diet than omnivores; healthy foods such as soya, nuts, legumes and vegetables replace meat.<sup>318</sup> US vegetarians eat more wholegrain products, dark green and deep yellow vegetables, wholegrain bread, brown rice, soya milk, tofu, meat substitutes, legumes, lentils and nuts.<sup>319</sup> Although they eat the same quantity of food as omnivores (1,000 kg per year) they are usually slimmer.320

A healthy vegetarian diet, characterised by frequent consumption of fruits and vegetables, whole grains, legumes and nuts, results in higher intakes of dietary fibre, antioxidants and phytochemicals.<sup>321</sup> Thus a vegetarian diet contains a range of natural substances that can improve both the carbohydrate and lipid abnormalities in diabetes.321

Vegetarians eat about two-thirds of the saturated fat, and onehalf of the cholesterol of omnivores, and vegans consume onehalf of the saturated fat and no cholesterol.<sup>322, 323</sup> Vegans have very low levels of harmful LDL blood cholesterol.<sup>324, 325</sup> Staple foods of their diets, such as nuts, soluble fibre (from oats and barley) soya proteins, and plant sterols improve blood lipid levels.<sup>326</sup> In addition, substituting soya or other vegetable proteins for animal proteins reduces the risk of developing nephropathy (a disease affecting the kidneys) in type 2 diabetes. Vegetarians in Western countries enjoy remarkably good health, exemplified by low rates of obesity,<sup>327, 328</sup> diabetes, 329 heart disease 330-332 and cancer, 333 and a three-to-six year increase in life expectancy.334, 335

The biggest study on vegans to date<sup>86</sup> compared over 1,000 vegans in Europe to tens of thousands of meat eaters and vegetarians. The meat eaters, on average, were significantly heavier than the vegans. Even after controlling for exercise, smoking and other non-dietary factors, vegans came out slimmer in every age group. Less than two per cent of vegans were obese, compared to one in five English adults.85

Obesity is much less common among vegetarians than it is amongst meat eaters.<sup>100, 101</sup> Numerous research studies have shown people who are vegetarian or vegan to be up to 20 per cent (anywhere from six pounds to over two stones) slimmer than their meat-eating counterparts, ie compared to people of the same height, age and socioeconomic group, who smoke and exercise a similar amount.93-99, 347

Most overweight people shed pounds when they change to a vegetarian diet.<sup>102</sup> Adherents to Dr Dean Ornish's vegetarian (near vegan) programme for reversing heart disease have shown more weight loss compared to those on the Atkins, Weight Watchers and Zone diets – and the Ornish diet wasn't even designed for weight loss, but for health.<sup>341</sup> After five years, most of the adherents to the Ornish diet were able to maintain much of the 24-pound weight loss experienced in the first year despite them "eating more food, more frequently, than before without hunger or deprivation."342 Most importantly, losing weight this way is consistent with long-term health.<sup>104</sup> Vegetarians show an average body mass index about two units lower than non-vegetarians.<sup>117</sup> This means that for a given height, vegetarians weigh less and are likely to have less body fat than non-vegetarians.

Of course, some people do not lose weight despite following a plant-based diet. The answer is steering away from too many processed junk foods, which are often very high in fat. Says T. Colin Campbell, Professor Emeritus of Nutritional Biochemistry at Cornell University and Project Director of the China-Oxford-Cornell Diet and Health Project, the biggest study of nutrition ever undertaken: "These foods are not part of a plant-based diet that works to reduce body weight and promote health. Some people also become vegetarian only to replace meat with dairy foods, added oils and refined carbohydrates, including pasta made with refined grains, sweets and pastries. I refer to these people as 'junk-food vegetarians' because they are not consuming a nutritious diet."105

A study recently published in the Journal of Human Nutrition and Dietetics<sup>113</sup> observed 33 people who were in the earliest stages of becoming vegetarian. They were monitored for six months, and their new diets were self-selected vegetarian. The researchers concluded that the findings of their study "suggest that significant dietary changes, helping people to conform more closely to current dietary recommendations, occurred when people became vegetarian."<sup>113</sup> Although no significant changes in body weight were observed (the study participants were not consciously trying to lose weight) significant reductions were seen in body fat, waist and hip circumference. Those on the vegetarian diet took in significantly less calories, and importantly, less calories from saturated fats. They also derived more energy from

Body weight is the integrated product of a lifetime's diet and activity habits. Weight is often gained slowly, over a period of months and years, and therefore it is rather unreasonable to expect to take it off healthily in a matter of weeks. Each pound of body fat contains 3,500 kcal.335 Therefore, someone who takes in 500 kcal less than he expends each day can lose one pound of fat per week. Any higher weight loss is due to a more severe restriction of calories or a loss of water rather than fat. In medically unsupervised weight loss diets, men should eat at least 1,500 kcal per day and women 1,200 kcal.335

Treating weight loss as a race doesn't work; it only makes the dieter more eager to go back to the eating habits that put them in need of losing weight in the first place. One very large study of 21,105 vegetarians and vegans<sup>107</sup> found that BMI was lower among those who had adhered to their diet for five or more years compared to people who had been on the diet for less than five years.

Weight loss might also be elusive if a person does not engage in any physical activity. Given modern environmental factors, the International Association for the Study of Obesity considers that 30 minutes of moderate daily exercise may be insufficient for many people to prevent unhealthful weight gain. Additional exercise is recommended for those who find that this level of daily activity does not prevent weight gain.63

Certain people may have a family predisposition to be overweight, which can make the challenge more difficult. An especially rigorous diet and exercise regime is important in these cases. Says Professor Campbell, "In rural China, we noticed that obese people simply did not exist, even though Chinese immigrants in Western countries do succumb to obesity. Now, as the dietary and lifestyle practices of people in China are becoming more like ours, so too have their bodies become more like ours. For some of these people with genetic predispositions, it doesn't take much bad food before their change in diet starts to cause problems."106

carbohydrates and fibre. Their new vegetarian diets left them leaner with less harmful abdominal fat.113

In her book Eating Thin for Life<sup>87</sup> dietician Anne Fletcher looked into the habits of a few hundred successful 'dieters' - people who had not only lost over some four-and-a-half stones on average but also maintained their weight loss for an average of I I years. When she asked the dieters to describe their eating habits, the top responses were 'low fat', followed by 'eating less meat'. The dieters also commented that they ate 'more fruit and vegetables'. One scientific study<sup>88</sup> which tested the effects of an increased intake of fruit on weight loss found that a

significant weight loss could be sparked by adding three apples or pears to a person's daily diet. This effect was thought to decrease calorie intake by promoting feelings of fullness, without the fruit adding many extra calories to the diet. Similarly, a Harvard Study<sup>89</sup> of 75,000 women over a decade suggests that the more fruits and vegetables that women eat, the less likely they are to become obese. A scientific review conducted in 2004<sup>90</sup> suggests that in general increasing fruit and vegetable intake may be an important strategy for weight loss.

A recent study conducted by Dr Neal Barnard and colleagues from the Physicians Committee for Responsible Medicine<sup>108</sup> showed that low-fat vegan diets lead to significant weight loss, without requiring dieters to restrict calories, portion sizes or carbohydrates, or even to exercise. 64 overweight women were randomly assigned to either a low-fat vegan diet or to a more conventional lowfat comparison diet based on the guidelines of the US National Cholesterol Education Programme. As exercise can cause weight loss, the women were asked not to make any changes to their exercise patterns during the trial.

The control group lost just over half-a-pound per week, whereas the vegan group lost about one pound per week, which is similar to results seen with low-calorie diets. However, the weight loss on the vegan diet occurred with no limits on energy or portion sizes. The weight loss of the vegan group was attributed to it being lower in calories but more filling. Eliminating animal products meant that the vegan diet contained no animal fat, and making minimal use of oils meant that the diet was very low in fat overall.

The vegan group also showed a 16 per cent increase in its aftermeal calorie burning speed (referred to as the thermic effect of food). This appears to be due to the vegan diet having improved insulin sensitivity, causing people's cells to be able to pull glucose out of the bloodstream much more quickly.

The researchers comment that, although "At first glance, a vegan diet sounds like a challenge... research participants rate the acceptability of the vegan approach very similarly to that of other therapeutic diets. And while typical diets demand cutting calories and leave the dieter with nothing to assuage hunger pangs, a low-fat vegan approach provides plenty of choices to make up for whatever is missing. Hunger is not part of the equation."109

In its Global Strategy on Diet, Physical Activity and Health, the World Health Organisation also advocates that the following healthy behaviours are promoted to "encourage, motivate and enable individuals to lose weight": eating more fruit and vegetables, as well as nuts and whole grains; engaging in daily moderate physical activity for at least 30 minutes;

cutting the amount of fatty, sugary foods in the diet; and moving from saturated animal-based fats to unsaturated vegetable-oil based fats.80

Plant-based diets are not only advocated for adults, but children, too. The Paediatrician Dr Benjamin Spock advised in his book Dr Spock's Baby and Child Care<sup>81</sup> that weight-loss programmes for children should be based upon changing the type of food children eat, rather than the amount of food they eat. He encouraged shifting the entire family away from oily fried foods, meats and dairy products and toward low-fat, plantbased foods - grains, pasta, vegetables, legumes and fruit. When this is done, he stated, "weight loss typically occurs without anyone going hungry."81

This advice is echoed in the Physicians Committee for Responsible Medicine report, Weight Control and Obesity Prevention in Children: "Instead of centring meals around fatty meats and cheese, meals should be built from healthy grains, legumes, and vegetables."82

The solution to the reducing the prevalence of obesity on a global scale is clear. It shares the same foundations which minimise the risk of chronic disease. Reverting to our roots of a whole foods, plant-based diet rich in the grains, vegetables and legumes which, ironically, are increasingly fed to animals, combined with moderate physical activity can be the keys to both weight maintenance and improved health.

This is of course a long-term lifestyle change, rather than another quick-fix fad.

# **CAUSATION**

As summarised by the House of Commons Select Committee on Health in its Third Report of Session: "At its simplest level, obesity is caused when people overeat in relation to their energy needs."20

Overeating is of course a relative term. It describes taking in an inappropriately large amount of energy compared to how much energy a person expends. Energy needs do of course vary from person to person. While an energy intake of 3,000 kilocalories (kcal or 'calories') per day might be insufficient for an athlete undergoing training, it would be serious overeating for a petite office worker.

Overeating in the short-term, in the form of feasts and celebrations, is a common human ritual. In traditional societies where - because of extreme seasonality - feasting and fasting are a means of survival, overeating does no harm, and it may even do much good by topping up depleted body fat stores.<sup>140</sup> It is when overeating persists

over any length of time that it threatens health. Long-term overeating leads to excess body fat storage and to overweight and obesity.

There are two distinct types of overeating: active and passive. Active overeating can be brought on by many factors. These include a drive to carry on eating in spite of having satisfied your natural appetite; a defect in appetite regulation (as seen in many of the rare inherited forms of human obesity); an inappropriate psychological response to stress; or a disorder in the area in the brain which recognises feelings of fullness.203

Passive overeating is a separate phenomenon. It refers to passively taking in more food energy (calories) than the body can burn off. Two factors contribute to this: the energy-dense modern diet, and reduced levels of physical activity associated with modern sedentary living.203

# **ACTIVE OVEREATING**

Active overeating in humans can occur for cultural reasons among populations in which fatness is esteemed. In urban Gambia for example, there is a clear gender difference in the prevalence of obesity between middle-aged women (over 35 per cent) and men (less than two per cent).<sup>203</sup> Active eating here reflects a cultural desire for fatter women who are considered more affluent and more attractive.

In Western societies, however, active overeating is generally driven by marketing.<sup>203</sup> For example, 'meal-deals' in wellknown fast-food outlets provide well in excess of a teenage girl's entire daily energy and fat needs.<sup>203</sup> These meals are extremely cheap and are marketed at the poorer sections of society. They are most likely implicated in the social class gradient of obesity.

Overeating can often become addictive.<sup>204</sup> This sometimes starts as a response to life stresses and it can lead to bulimia nervosa, binge eating disorder and night eating disorder.204 Although these eating disorders have escalated in the past few decades, they are unlikely to contribute significantly to the global epidemic.

There are also rare inherited causes of human obesity. In these cases, the signals that make a person stop eating when full (satiety signals) work inadequately. This leads to an insatiable appetite and hence overeating. In rare cases, physical injury to the area in the brain which recognises feelings of fullness can also bring about gross obesity.

Food is constantly accessible to most people in developed countries and many suppliers endeavour to make their products as tempting and as easy to consume as possible. Cheap, energy-dense foods, which are highly calorific without being correspondingly filling, are widely available and greatly promoted by manufacturers. Simultaneously, there has been a transition towards sedentary lifestyles over recent decades. Before the technological revolution, people typically walked at least five to 10 miles a day, accounting for an additional 500 to 1,000 kcal of energy expenditure.<sup>115</sup> In our modern society, roughly two-thirds of men and three-quarters of women fail to meet the Department of Health's physical activity target of 30 minutes five times per week!<sup>20</sup>

Many prescription drugs also stimulate appetite and cause great weight gain.<sup>205</sup> Tobacco smoking suppresses the appetite and the nicotine it contains stimulates the nervous system. Fifteen to 20 cigarettes a day can increase daily energy requirements by 10 per cent.<sup>207</sup> Cigarette smoking habits seem to influence fat distribution patterns, although the exact mechanisms underlying this have not yet been elucidated.338 Smokers tend to be slimmer than nonsmokers, but have more abdominal fat distribution, which is a risk factor for cardiovascular disease and diabetes.<sup>207, 338</sup> The average weight gain after stopping smoking is three kilograms for men and four kilograms for women over a 10year period.<sup>207</sup> Although the health consequences of weight gain are secondary compared to the benefits of giving up smoking, weight increase is the most frequent reason for taking up smoking again.207

Fifty per cent of overweight women cite pregnancy as the main cause of their obesity.<sup>206</sup> A typical British woman gains around 12.5 kg (just under two stones) in weight during a typical pregnancy.<sup>210</sup> First-time pregnant, non-smoking, hypertensive and already overweight women are at special risk of excessive weight gain.<sup>206</sup> For severely overweight women (over 60 per cent overweight), restricting calories during pregnancy can be safe, as long as greater attention is placed on the quality of the diet.206

In summary, there are numerous causes of active overeating which can lead to severe ill health. However, evidence suggests that passive overeating has a greater effect on the weight of the nation than active overeating does.205

# **PASSIVE OVEREATING**

We are clearly living in a very different environment to that which we are adapted - one in which it is easy to consume more calories than we need.

# **CHANGES IN DIET COMPOSITION**

#### Fat

The incidence of obesity in the UK grew slowly from about 1920 and started to increase more significantly after the Second World War. While this trend is not driven by diet alone, changes in diet play a key part. The diet has changed during this time from one rich in complex carbohydrates (ie grains, wholemeal bread, potatoes and other root vegetables, legumes and other vegetables) with a modest fat content, to the modern diet. The national intake of meat, cheese, butter and other rich milk products and of alcohol has risen at the cost of lower calorie carbohydrate-rich foods.<sup>24</sup> The fat content of the diet has shown a significant increase.

Fat is very energy dense, containing more than twice as many calories, weight-for-weight (nine kcal per gram) as protein or carbohydrate (four kcal per gram). Not only is it the most calorie dense of the macronutrients, but, along with alcohol, it is also one of the least filling (satiating).<sup>39</sup> This means that in order to feel full, a larger amount of a fatty, carbohydrate-deficient diet must be eaten compared to a low-fat, high-carbohydrate diet.

There is a consensus across international agencies such as the World Health Organisation and national governments such as that in the UK that most people in developed countries eat too much fat for good health.<sup>38</sup> Fat, especially animal saturated fat, increases the risk of heart disease, diabetes and some cancers (see **HOW OBESITY AFFECTS ADULTS**, page 9). As the high calorie density of high fat foods increases the chances of eating too many calories (see **Energy density and satiety**, page 24), choosing a low fat diet is not only beneficial to health but is also a practical step to reduce the risk of weight gain.

**Figure 5** summarises the main sources of fat in the human diet. Any food that contains any of these products as an ingredient will also contain fat. Given the significant contribution of meat to the fat content of the UK diet, in 1976 the Royal College of Physicians/British Cardiac Society issued a report on diet and heart disease, recommending that more poultry be eaten in place of red meat because it contained less fat. This advice seems to have been taken on board: the *National Food Survey* shows a huge rise in the consumption of chicken, which was rarely consumed 50 years ago and has now become the most common form of dietary protein<sup>25</sup> (see **Figure 6**).

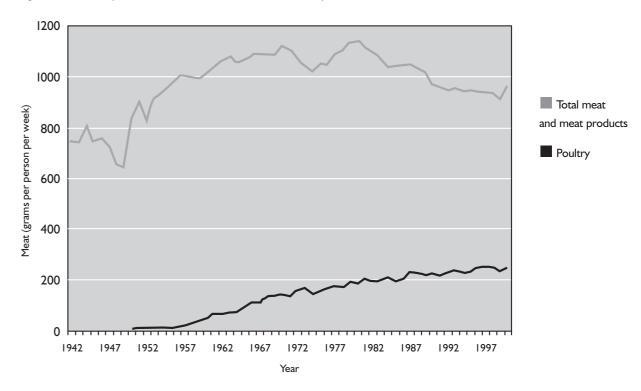
However, the competition to produce inexpensive meat, eggs and dairy products has led to changes in the way that farmed animals are reared. The worldwide trend is the replacement of small family farms with factory farms where animals are reared intensively. In the UK, 95 per cent of chickens, 99 per cent of ducks, over 90 per cent of meat pigs, and the majority of turkeys are reared in this way.<sup>27</sup> The greatest financial cost in rearing animals intensively is their feed. Therefore it is most cost-effective for farmers to maximise 'feed-conversion ratios' – ie to get the largest output of meat, eggs or dairy from the smallest input of feed. Animals are predominantly fed standardised industrial feed, which is carefully formulated to promote maximum weight gain. Chickens, for example, reach slaughter size almost twice as quickly as they did 40 years ago.

Professor Michael Crawford of London Metropolitan University recently analysed chicken thigh meat from several supermarkets and organic suppliers and found them to contain more than twice as much fat, and around 100 kcal more, weight-for-weight, as they did back in 1940.<sup>26</sup> Modern beef was also found to contain 30 per cent fat, compared with the five per cent found in wild beef. Meat has taken the place of lower calorie foods, which were once staples of the national diet. And portion-for-portion, the fat

Figure 5. The main sources of fat in the human diet

#### Source of fat Examples Type of fat Milk fat Milk, yoghurt, Almost inevitably highly saturated cream, butter and cheese. (typically 50 to 65 per cent of fat is saturated)132 Meat fat Meat (poultry and Tends to be highly red meat), meat saturated (typically products (such as 27 to 44 per cent of fat is saturated)132 sausages and burgers), cooking fats of animal origin such as lard. Fatty fish Mackerel, herring, Relatively low in trout and salmon. saturated fats (typically 17 to 25 per cent of fat is saturated)132 Seeds Seeds, nuts, vegetable Relatively low in and nuts oils and soft margarines. 'bad' saturated fats and high in 'good' polyunsaturated fats (except tropical oils such as coconut and palm oil). Typically eight to 25 per cent of fat is saturated<sup>132</sup>

Figure 6. Meat consumption, 1942 to 2000. Data from National Food Survey.



content of meat has doubled. The number of surplus calories that can lead to weight gain if taken in repeatedly is surprisingly low. An excess of just 30 kcal per day in energy consumed, compared with energy required, will cause a weight gain of about one kilogram (2 lb) over a year.<sup>114</sup> More calories and fat in meat (especially compared to staple foods of the past) is seemingly a key contributor to the 'passive over-consumption' of energy by society. It makes it easier to take in more calories than the body can burn off.

Researchers at the American Cancer Society followed more than 75,000 people for a decade to find out which behaviours were most associated with weight loss and which with weight gain.<sup>91</sup> The one dietary behaviour most associated with an increasing waistline was high meat consumption.<sup>91</sup> **Even after controlling for other factors, men and women who ate more than a single serving of meat per day seemed to be 50 per cent more likely to suffer an increase in abdominal obesity than those who ate meat just a few times per week.** 

In 1983, raised awareness of the changed composition of the national diet led to the introduction of quantified dietary targets for the UK population. The National Advisory Committee on Nutrition Education (NACNE) recommended that the nation's total fat intake be reduced to 30 per cent of calories and saturated fat reduced to 10 per cent of calories.<sup>30</sup> (The national averages are still currently above this, with fat providing 35.4 per cent of calories and saturated fat contributing 13.3 per cent of calories.)<sup>343</sup> Meat products were

6

identified as a leading source (28 per cent of dietary fat) of invisible fat, mainly saturated.<sup>32</sup> Butchers and food processors got into the practice of trimming off visible fat in response to consumer demand for leaner meat (see **Figure 7**). However, modern farming methods may have rendered the practices of trimming visible fat and removing skin from meat futile methods of eliminating dietary fat.

Figure 7. The process of visible fat being trimmed from pork; however 40 per cent of fat is 'invisible' and cannot be removed<sup>282</sup>



Despite these changing practices, the latest *National Diet and Nutrition Survey*<sup>31</sup> reports that meat and meat products are still the main source of total dietary fat, providing just under a quarter (23 per cent) of the average national dietary fat intake. This means that despite advice encouraging us to opt for leaner cuts of meat, a mere five per cent reduction in our total fat intake from meat has been observed.

Professor Crawford states: "This whole focus on rapid growth [in intensive farming], achieved through a highenergy, cereal-based diet has changed the lipid composition of the chicken meat itself, and you cannot escape that – even by removing the skin and scraping away the subcutaneous fat stuck to the meat."<sup>142</sup>

There is a particular problem with beef and mutton where 60 per cent is intramuscular and so is less easily removed. There is also a problem with pig meat, where 40 per cent of the fat is the hard to remove intramuscular kind.<sup>32</sup>

Figure 8. Intensive farming has caused the fat content of chicken meat to double since 1940



Catherine Geissler (Professor of Human Nutrition at King's College, London) and colleagues further report that the visible fat trimmed off by the food industry has remained in our food chain, by being used in other meat products.<sup>141</sup>

The increased awareness of the need to cut down on fat, especially saturated fat, has also increased the demand for low-fat and skimmed milk over full-fat milk. Butter has been partly replaced by margarines and low-fat spreads which became more available in the 1980s and, instead, vegetable oil consumption has increased (see **Figure 9**).<sup>141</sup>

#### **European Commission subsidies**

The Food Commission reports, however, that although consumers have reduced their purchases of butter to the lowest levels yet (1.2 million tonnes by the latest published figures) the European Commission purchased another 0.53 million tonnes.<sup>33</sup> The EC operates a *Butter for Manufacture* scheme which aims to 'dispose of surplus butterfat by encouraging manufacturers to use butter in manufactured products in preference to cheaper vegetable oils'. Under this scheme a subsidy is paid to food manufacturers on butter, butteroil and cream processed into certain eligible products, such as cakes, pastries, biscuits, ice cream and desserts.

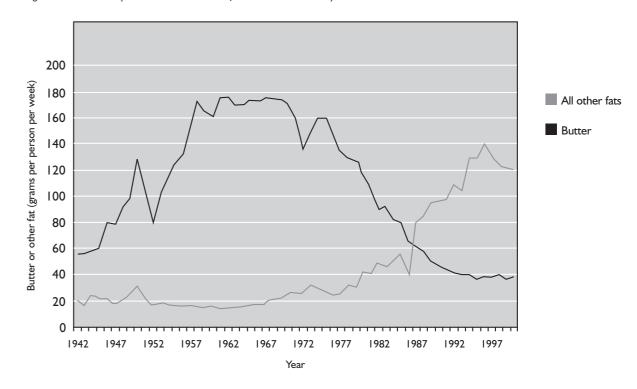
Another Common Agricultural Policy scheme which is in operation is the *Concentrated Butter for Direct Consumption* scheme, which aims to 'dispose of surplus butterfat by encouraging manufacturers to sell butteroil direct to the retail trade for direct consumption', ie use in the home, hospitals, restaurants etc.<sup>79</sup> The total now being bought by the EC amounts to nearly one-third of all butter produced, with 92 per cent of the surplus being sold off.<sup>33</sup> This means that an additional half-a-million or so tonnes of butter appears to be entering the food chain in the form of both manufactured products and foods eaten in hospitals and restaurants.

#### The National Food Survey

Meat and meat products are the leading source of fat in the national diet. Milk and milk products (such as cheese and semi-skimmed milk) are not far behind, providing 14 per cent of total fat.

But fat – and commonly animal fat, in the form of butter and milk – is a major ingredient of cakes, pastries, biscuits and chocolate (see **European Commission subsidies**, above). Vegetable oils (often hydrogenated) are sometimes also used in these products (see **Trans-unsaturated fatty acids**, page 21). Perhaps it is unsurprising, then, that cereals and cereal products (especially manufactured products such as pizza, biscuits, buns, cakes and pastries) fall just below meat in the *National Diet and Nutrition Survey* fat hierarchy, making up just under one-fifth (19 per cent) of our national fat intake (see **Figure 10**).

Figure 9. Butter consumption, 1942 to 2000. Data from National Food Survey.



The National Diet and Nutrition Survey and the National Food Survey have observed the nation's diet annually since the 1940s. In an apparent anomaly considering the rising obesity epidemic, data from the National Food Survey seems to indicate that people are eating fewer calories nowadays. Reported total fat consumption has also dropped – although when the accompanying reported decrease in calories consumed is considered, the proportion of fat in the diet remains unchanged. The contribution of fat to the UK diet has stayed virtually constant for more than three decades at around 40 per cent of calories.<sup>35</sup>

However, a degree of caution is required when interpreting the *National Food Survey* results. Underreporting the number of calories consumed is not an uncommon practice. Obese subjects under-report energy intake by an average of 30 per cent of calories consumed.<sup>37</sup> There is also justifiable feeling that the recent emphasis on the need to cut down on fat has encouraged people to *report* eating less fat, rather than to actually *eat* less fat. Dr Jebb and colleagues report that food disappearance records (which measure supplies moving through trade channels for domestic consumption) show as much, or even more, fat in our food supply than ever before.<sup>36</sup>

The falling trend in calorie intake suggested by the National Food Survey also excludes alcohol, confectionery, snacks and foods eaten outside of the home, such as in hospitals and restaurants. Eating outside the home is becoming increasingly popular – one-quarter of respondents to the Food Standards Agency Consumer Attitudes Survey in 2003 reported regularly

using some form of fast food or takeaway outlet.<sup>147</sup> Surveys indicate that food eaten out tends to be higher in fat than food eaten in the home (see **Fast foods**, page 25). Total recorded alcohol consumption in the UK has also doubled between 1960 and 2002 (see **The role of added sugars, sugary drinks and alcohol,** page 22).<sup>144</sup>

Source of total fat	% contribution to total fat intake
Meat and meat products of which	23
Bacon and ham	2
Beef, veal and dishes	3
Lamb and dishes	I
Pork and dishes	I
Coated turkey and chicken	I
Chicken, turkey and dishes	4
Burgers and kebabs	2
Sausages	3
Meat pies and pastries	4

Figure 10. Percentage contribution of food types to average daily total fat intakes in the diet of adults aged 19 to 64 years<sup>151</sup>

#### Figure 10. continued

Cereals and cereal products	19
of which	
Pizza	2
White bread	2
Biscuits	3
Buns, cakes and pastries	4
Milk and milk products of which	14
Whole milk	3
Semi-skimmed milk	3
Cheese (including cottage cheese)	6
Fat spreads of which	12
Butter	4
Margarines	I
Reduced fat spreads (60-80% fat)	5
Low-fat spreads (40% fat or less)	I
Potatoes and savoury snacks of which	10
Chips	5
Other fried or roast potatoes	I
Savoury snacks	3
Vegetables excluding potatoes	4
Fish and fish dishes	3

#### Are all fats created equal?

Fats and oils in the diet are largely made up of molecules called fatty acids, attached to the molecule glycerol. Three fatty acids combine with one molecule of glycerol to form so called 'triglycerides'. The fatty acids can be of three major types - saturated, monounsaturated and polyunsaturated, depending on how many double bonds they contain. A certain type of unsaturated fatty acid – trans-unsaturated – is often considered separately because of its effects on health, and because it is largely created by the manufacturing process.

Fat intake, per se, is implicated in the development of obesity and other associated conditions. All kinds of fat, whether unsaturated, monounsaturated or saturated provide the same amount of energy, and therefore curtailing total fat intake is important as a means to preventing obesity.

The body cannot function without some fat – but it is eating the right kind of fat that is vital in terms of our overall. We have no dietary requirement for saturated fats, which are strongly linked with raised levels of total cholesterol, hardening of the arteries and heart disease.<sup>151</sup> Evidence is

also emerging that reducing total and saturated fat intakes could also lower the risk of breast and prostate cancer.<sup>150, 340</sup> Figure 11 illustrates the major sources of saturated fats in the UK national diet.

Not all saturated fatty acids have the same effects. Those with the most cholesterol-raising properties are lauric acid (C12:0), myristic acid (C14:0), and palmitic acid (C16:0). These three fatty acids account for 60 to 70 per cent of the saturated fat in Western diets.<sup>283</sup>

Myristic acid is the most powerful cholesterol-raising saturated fatty acid.<sup>159</sup> It can increase total cholesterol levels by 50 per cent more than palmitic acid.<sup>284</sup> The major sources of myristic acid are butter, cream, whole milk and tropical oils.<sup>285-291</sup> Milk fat (from dairy cows) contains eight to 14 per cent myristic acid<sup>160</sup> and coconut and palm oils contain up to 18 per cent. Palmitic acid is the most common

Figure 11. Percentage contribution of food types to average daily total saturated fat intakes in the diet of adults aged 19 to 64 years<sup>151</sup>

Source of saturated fat	% contribution to
	saturated fat intake
Milk and milk products of which	24
Whole milk	4
Semi-skimmed milk	5
Cheese (including cottage cheese)	10
Meat and meat products of which	22
Bacon and ham	2
Beef, veal and dishes	4
Lamb and dishes	I
Pork and dishes	1
Coated turkey and chicken	I
Chicken, turkey and dishes	3
Burgers and kebabs	2
Sausages	3
Meat pies and pastries	4
Other	I
Cereals and cereal products	18
of which	
Pizza	2
White bread	Ι
Biscuits	4
Buns, cakes and pastries	4

5 U		
Figure 11. continued		
Fat spreads of which	П	
Butter	6	
Margarines	I	
Polyunsaturated reduced fat	I	
spreads (60-80%)		
Other reduced fat spreads	2	
(60-80% fat)		
Low-fat spreads (40% fat or less)	I	
Potatoes and savoury snacks	7	
of which		
Chips	3	
Other fried or roast potatoes	I	
Savoury snacks	3	
Chocolate confectionery	5	

fatty acid in the human diet, and the main saturated fatty acid in both animal fats (including red meats, poultry and eggs) and palm oil.

Lauric acid is the least harmful of these three saturated fats, with around one-third less cholesterol-raising power than palmitic acid. It is the main saturated fatty acid in coconut and palm kernel oils (they contain around 48 per cent).289-291

As tropical oils do not feature heavily in our typical national diet, they do not contribute significantly to average national saturated fat intake.<sup>343</sup> However, coconut is the chief source of energy for certain Polynesian populations.<sup>344</sup> The habitual diets of the toll dwellers from both Pukapuka and Tokelau are high in saturated fat (primarily from coconut) but low in dietary cholesterol (found only in animal products) and sucrose (sugar). Tokelauans take in many more calories from coconut than the Pukapukans (63 per cent compared with 34 per cent)<sup>344</sup> and so their intake of saturated fat is higher. As might be expected, Tokelauans have higher blood cholesterol levels. However, vascular disease is still uncommon in both populations.344

Coconut flakes (rather than coconut oil) have been found to lower harmful (LDL) cholesterol levels in people with moderately raised blood cholesterol.<sup>345</sup> Coconut is a good source of soluble and insoluble dietary fibre, which have cholesterol lowering powers and which may explain this apparent paradox. Of course, processing coconut in order to produce coconut oil does strip away the protective fibre. Conversely, dairy products and meat contain no protective fibre yet remain the leading sources of harmful saturated fats

in our national diet.<sup>346</sup> As perhaps would be expected, vascular disease (the build up of lipids and other cells on the artery wall) is a common finding in those who consume the modern Western diet.

### **Trans-unsaturated fatty acids**

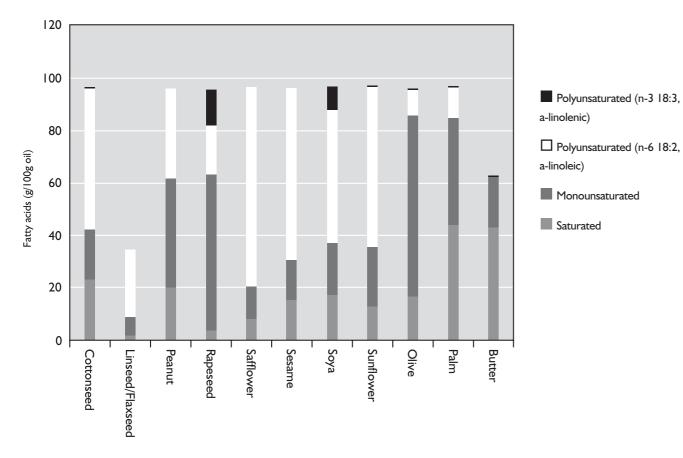
Another type of fat, namely trans-unsaturated fatty acids, has also been shown to increase the risk of heart disease by raising harmful (LDL) cholesterol levels and lowering protective (HDL) cholesterol levels. This combined effect on LDL and HDL cholesterol is double that of saturated fatty acids.<sup>155</sup> A recent review on the influence of trans fatty acids on health suggests that they are, gram for gram, associated with a two-and-a-half to tenfold higher risk of heart disease than saturated fat.<sup>156</sup> No safe limits of trans fat consumption have been identified.157

Trans fatty acids are often found in processed foods because fats containing these fatty acids can be prepared from liquid oils by an industrial process known as hydrogenation. The final product of this process is called hydrogenated vegetable oil, or hydrogenated fat. It is used in some biscuits, cakes, pastry, margarine and many processed foods. This means that foods that contain hydrogenated vegetable oil (which is always declared in the ingredients list) are likely to contain trans fats. Low levels of trans fatty acids are also found naturally in dairy products, lamb and beef fat, as small amounts of trans fat are produced in the gastrointestinal tract of ruminants.<sup>157</sup>

High intakes of dietary cholesterol also increase harmful LDL cholesterol.<sup>292</sup> Although saturated fat has 10 times the cholesterol-raising power of dietary cholesterol,<sup>295</sup> restricting dietary cholesterol is of special importance for those who are genetically prone to hypercholesteraemia.294 Dietary cholesterol is found only in the animal kingdom; even grilled skinless chicken breast contains 94 milligrams of cholesterol.<sup>293</sup> Humans have no dietary need for cholesterol as the body can manufacture all that we require.

The fats which are essential to the diet are linoleic acid (C18:2, n-6) and alpha linolenic acid (C18:3, n-3). These essential fatty acids (EFAs) have an important structural function in cell membranes; are involved in regulating cholesterol metabolism (ie its transport, breakdown and excretion) and are precursors of prostaglandins, thromboxane, leukotrienes, and of longer chain fatty acids. Seed oils such as linseed (flax), rapeseed (canola) and walnut oil are rich sources, as are seeds and nuts themselves.<sup>161</sup> Green leafy vegetables are also a source.<sup>162</sup> One teaspoon of flax seed oil or a handful of whole seeds and nuts (linseed, hempseed or walnuts) each day should provide most people with sufficient essential fatty acids.

#### Figure 12. Fatty acid composition of selected vegetable oils and butter for comparison<sup>143</sup>



As part of a healthy diet the Food Standards Agency recommends that we should try to reduce the amount of foods we eat that contain hydrogenated or saturated fats and replace them with unsaturated fats.<sup>149</sup> The total amount of fat that we eat should also be reduced.

**Figure 12** compares the fatty acid composition of butter and selected vegetable oils. Soya oil and sunflower oil are two vegetable oils that are typical of many vegetable oils in that they are low in saturates and high in polyunsaturates. The n-6 polyunsaturated fatty acids usually predominate in vegetable oils. Olive oil and rapeseed oil are both particularly high in monounsaturates and low in saturates. The composition of palm oil shows that there are exceptions to this general observation that vegetable oils are low in saturates and high in polyunsaturates. Clearly then, the switch should be made from animal to vegetable sources of fat (though using tropical oils such as coconut and palm oil only sparingly). The use of all types of oils in cooking should however be reduced.

The atherogenicity ('artery clogging potential') of the major fatty acids is summarised in **APPENDIX 2**.

# The role of added sugars, sugary drinks and alcohol

There is growing evidence that the rise in consumption of sugar-rich drinks may be fuelling the increase in obesity.<sup>134</sup> Experimental studies in which volunteers are given sugar-rich drinks compared to artificially-sweetened drinks or water show that people fail to decrease their intake of calories at the next meal sufficiently to allow for the calories previously taken in from the drink.<sup>41</sup> The overall effect is that sugar-rich drinks add to, rather than substitute, food intake and hence increase the risk of eating too much. This may be a particular problem for children, who are larger consumers than adults – soft drinks provide 26 per cent of the added sugars in the diet of four to 18 year olds,<sup>135</sup> compared with 16 per cent for adults.<sup>125</sup>

Raben and colleagues<sup>136</sup> compared the effects of sugar-rich versus artificially sweetened foods and drinks (approximately 80 per cent were beverages) on body weight in a group of overweight volunteers. Modest (but significant) weight increases were seen in the group consuming the sugar-rich diet during the 10-week study, whereas the group making use of artificially sweetened foods showed a modest weight loss. As previously commented, an excess of just 30 kcal per day in calories consumed, compared with calories required, can cause a weight gain of about one kilogram (two pounds) over a year.<sup>114</sup> 30 kcal could be obtained by drinking just one quarter of a can of cola. Although carbohydrate-rich foods with a low fibre content are typically less satisfying than similar products with a high fibre content (see **Fibre and satiety**), the significance of this for weight regulation is unclear. The CARMEN trial<sup>138</sup> was the first intervention trial to specifically investigate the role of simple sugars in the development of obesity. The trial, which was carried out in five European centres, compared the weightloss effects of two low-fat, high-carbohydrate diets – one rich in simple carbohydrates, and the other rich in complex carbohydrates. Subjects were allowed to eat freely during the six months of the study. A modest weight loss was seen in both groups of low-fat dieters, showing that it is favourable to replace dietary fat with carbohydrates, although no significant difference was seen between the two low-fat high-carbohydrate groups.

However, an intervention trial in Cambridge by Poppitt et al<sup>137</sup> saw a significantly greater weight loss in low-fat high complex carbohydrate dieters (who also tended to see a decrease in total cholesterol). Although those following a low-fat highsugar diet did not lose significant amounts of weight, importantly this group did not *gain* weight, despite the free eating (ad libitum) nature of the diet used in the Cambridge trial. The evidence then, suggests that replacing dietary fat with carbohydrates – preferably complex carbohydrates – may be a favourable strategy for weight loss.

The evidence regarding the effect of alcohol consumption on weight gain is somewhat conflicting. Alcohol is much more calorie dense than carbohydrate (seven kcal per gram compared with four kcal per gram) and alcoholic drinks frequently contain - or are served with drinks which contain - added sugar. As previously commented, total recorded alcohol consumption in the UK has doubled between 1960 and 2002.<sup>144</sup> Like soft drinks, alcoholic drinks do not appear to displace calories from food.<sup>139</sup> That said, the Health Survey for England indicated that non-drinkers are more likely to be obese than those who consume alcohol. Most of the current knowledge on alcohol's effect on weight is based on observational population studies, where confounding effects are possible. It cannot be ruled out, for example, that smoking tobacco at the same time as drinking alcohol could instead be responsible for the 'slimming' effect attributed to it.

## Glycaemic index and glycaemic load

The term glycaemic index (GI) describes the blood sugar (glucose) elevating potential of a food. Values over 90 are generally considered high,<sup>297</sup> meaning that foods with such values quickly release their natural sugars into the bloodstream. The foods that we evolved to eat would typically have had a lower GI of between 40 and 80.<sup>296</sup>

t ba supplex br e in mon h- ch hy ee tra dge fol at ca

> ter lov hu in lor los tw the foo int ch ch an co

22

The exact rise in blood sugar that is experienced depends on both the GI of the food and the amount of carbohydrate that it contains.<sup>296</sup> The glycaemic load (GL) calculation takes into account both of these considerations. High GL foods quickly release their natural sugars into the bloodstream, whereas low GL foods release their natural sugars slowly and evenly, unlike the quick hit provided by sugary snacks. Subtle changes in the food supply over the past few decades (see **Food processing**, page 25) have led to an abundance of more highly processed grains.<sup>299</sup> Less-processed foods are more likely to contain slowly digested carbohydrates, as the sugars they contain still have the protection of bran and other barriers which are removed in processing.<sup>299</sup>

Some scientists have warned against the fattening properties of foods with a high GI such as some types of potatoes, white bread, bagels and white rice and instead advise people to eat more wholegrain products, and types of rice and potatoes characterised by a low GI. The proponents of the GI hypothesis suggest that high GI foods produce rapid and transient surges in blood glucose and insulin which are in turn followed by rapidly returning hunger sensations and excessive calorie intake.

Low GI foods are beneficial for glycaemic control in diabetics and have a beneficial effect on cardiovascular risk factors<sup>299</sup> but their effect on body weight regulation is controversial. A scientific literature review looked at published human intervention studies which compared the effects of high and low GI foods or diets on appetite, food intake, energy expenditure and body weight.<sup>163</sup> Out of a total of 31 shortterm studies (less than one day's duration), 15 indicated that low GI foods were associated with greater satiety or reduced hunger, whereas reduced satiety or no differences were seen in the 16 other studies. Low GI foods reduced ad libitum food intake in seven studies, but not in eight other studies. In 20 longer-term studies (less than six months' duration), a weight loss on a low GI diet was seen in four and on a high GI diet in two, but 14 others recorded no difference. At present, then, there is no evidence that low GI foods are superior to high GI foods with regard to long-term body weight control.

However, as foods which quickly release their natural sugars into the bloodstream can decrease levels of protective HDL cholesterol and increase levels of dangerous VLDL and LDL cholesterol,<sup>298</sup> and are linked with increased risk of diabetes and heart disease,<sup>298</sup> glycaemic load does warrant consideration. Glucose itself has the ability to damage blood vessel (vascular) cells.<sup>299</sup>

Those who are overweight or carrying abdominal fat, diabetic, physically inactive and/or following a low-fat diet should pay particular attention to limiting GL. This can be achieved by choosing foods that have a relatively low GL per calorie. Pasta and dense chewy breads such as pumpernickel have a lower GL per calorie than most grain products, and the GL of lentils and beans is lower still.<sup>298</sup> Bread has a high GL, although other components in wholemeal bread are associated with reduced risk of heart disease and strokes.<sup>298</sup> Refined, processed foods should be avoided. APPENDIX 3 compares the GL per calorie of some common plant foods.

## **Energy density and satiety**

Energy density refers to the amount of calories that different foods contain weight for weight (kcal per 100 g). Energy dense foods and energy dense diets have been blamed for the global obesity epidemic.177, 178, 179, 180, 181 Traditional African diets containing approximately 108 kcal per 100 g probably represent the levels at which human weight regulatory mechanisms have evolved.<sup>199</sup> The average British diet is almost fifty percent more energy dense than this, containing approximately 160 kcal per 100 g.<sup>199</sup> Most plant foods (boiled grains, lentils or beans, raw fruits and vegetables) provide under 120 kcal per 100 g and most fruits and vegetables provide much less than this.<sup>209</sup>

Laboratory studies suggest that energy-dense foods (foods high in calories but low in bulk) are less filling and may result in passive overeating and therefore weight gain.<sup>211</sup> Human observational studies suggest that diets like these also tend to be nutrient poor.211

Out of fat, protein and carbohydrate, fat is both the most energy dense (nine kcal/g) and the least filling (satiating) of all.<sup>300</sup> In order to achieve a feeling of fullness, a larger amount of a fatty, carbohydrate-deficient diet must be eaten compared to a low-fat, high-carbohydrate diet. Research has shown that when people are offered foods which have been secretly manipulated to alter the fat content, they eat far more energy when the meal is high in fat than when offered an apparently similar low-fat meal.<sup>40</sup> The body appears not to recognise that it is eating more calories and weight gradually increases. Conversely on a low-fat (less calorie dense) diet, despite eating as much as they wished and never feeling hungry, subjects have managed to lose weight.40

## Fibre and satiety

Whilst the importance of complex carbohydrates in appetite control warrants further study, fibre in foods has long been regarded as a factor which brings about feelings of fullness.<sup>300</sup> It brings about this in many ways - more chewing is required for a fibre-rich food; it distends, and spends more time in, the stomach; and, because it also tends to create a low glycaemic

index,<sup>303, 304</sup> it leads to the slow, steady release of nutrients.<sup>300</sup> Prolonged chewing reduces the rate of eating, which means that there is longer for the body to acknowledge that food is being ingested and to curb food intake appropriately. The distension of stretch receptors in the stomach also initiates feelings of fullness. A review carried out in 2001 showed that every 14 grams of extra fibre in the diet reduces calorie intake by 10 per cent.145

## **High-protein diets**

Data suggests that protein is the most filling nutrient of all<sup>301, 302</sup> and has been credited with helping to curb hunger in those following high-protein diets. This has not been tested objectively, and alternative explanations such as monotony and ketosis may also contribute.<sup>300</sup> While a few recent studies have observed that high-protein, carbohydrate-restricted diets can bring about modest short-term weight loss, 75-77 the long-term health consequences of following such diets in order to lose weight have not yet been investigated. Most of these diets contain less than 10 per cent carbohydrate, 25 to 35 per cent protein, and 55 to 65 per cent fat.<sup>305</sup> Because the protein is provided mainly by animal sources, these diets are high in saturated fatty acids and cholesterol, and could perhaps more aptly be renamed 'high-fat' diets.305

429 individuals following such a high-protein, high-fat, carbohydrate-restricted diet voluntarily logged their dietassociated health problems using an online registry.<sup>306</sup> Common findings included constipation, loss of energy, bad breath, difficulty concentrating, kidney problems, and heartrelated problems, including heart attack, bypass surgery, arrthymias and elevated blood cholesterol levels.305

## Dairy and weight loss

Research by Professor Zemel of the University of Tennessee Nutrition Institute has suggested that the consumption of dairy products may help people lose weight.<sup>307, 308</sup> His research compared weight loss in subjects consuming a diet high in dairy products with subjects taking in an identical number of calories but with a reduced amount of dairy. As weight loss was greater in the high dairy group, Professor Zemel suggests that calcium from dairy foods might affect fat cell metabolism in a way that promotes weight loss. However, no evidence that a diet high in dairy products promotes weight loss was found in a subsequent study which included Professor Zemel (but not as the first named author).309

A scientific literature review was carried out by researchers at the University of British Columbia in Vancouver, Canada, to look into the effects of dairy products or calcium supplements on body weight. Of nine studies on dairy products, seven showed no significant differences in weight, while two studies linked dairy consumption with weight gain.<sup>311</sup> Additionally, just one of 17 studies on calcium supplementation reported weight loss.

A recent study involving 12,000 children over a three-year period found that those who drank the most milk gained the most weight.348

# **OTHER SOCIETAL CONDITIONS PROMOTING PASSIVE OVER CONSUMPTION**

## Food processing

As previously commented, processing often raises the GI of a food, by removing bran and other barriers (and many valuable nutrients) which protect the sugars within. Processed foods can contain the visible fat trimmed from carcass meat products; and the use of butter, butteroil and cream in cakes, pastries, biscuits, ice cream and desserts is subsidised under the EC Butter for Manufacture scheme. Highly-calorific fat is a major ingredient of cakes, pastries, biscuits and chocolate, hence cereals and cereal products now account for 19 per cent of our national fat intake. Trans fatty acids can also be found in some processed foods such as biscuits, cakes, pastry and margarine. Human observational studies suggest that diets rich in processed foods tend to be nutrient poor. And laboratory studies suggest that they are less filling, resulting in passive overeating and therefore weight gain.

However, food processing is very profitable to the food industry. Potatoes, for example, are inexpensive and there is little profit to be made from selling them. But there is much more profit to be made after a modest (low-cost) labour input to transform the potatoes into crisps.<sup>198</sup>

Says Dr Linda Bacon, lecturer at City College of San Francisco: "Because of this profit incentive, the food industry plays a large role in promoting overeating and eating processed foods, which typically are much less nutrient dense than unprocessed foods... The food industry employs two practices: encouraging us to eat more food in general and to eat more processed food... They also act behind the scenes to influence media, non-profit organisations, health care and nutrition professionals, researchers, and government agencies."198

## **Fast foods**

A typical fast food meal has a very high energy density (ie is very high in calories but low in bulk). It is more than one-anda-half times more calorie dense than an average traditional British meal and two-and-a-half times denser in calories than a traditional African meal.<sup>199</sup> Typical 'meal-deals' in well-known fast-food outlets provide well in excess of a teenage girl's entire daily energy and fat needs.203

Obesity is most common amongst those with more limited financial means and and manual labourers.<sup>211</sup> Developments in farming and food technology have made added sugars and vegetable oils accessible globally at a remarkably low cost. Lower-income households tend to select diets high in lowcost meats, inexpensive grains, added sugars and added fats<sup>171, 172, 173, 174</sup> and their fruit and vegetable expenditures tend to be low.<sup>175</sup> One interpretation of this data is that fruit and vegetables are not considered a priority and lowincome households choose to spend their limited resources on items that are perceived to be more essential such as meat, clothing, or rent.<sup>197</sup> Human observational studies however suggest that such low-cost calorie-dense diets tend to be poor in nutrients.<sup>211</sup>

25

Professor Andrew Prentice, Head of the UK Medical Research Council International Nutrition Group, believes that diets high in fast foods could be particularly disadvantageous for children, whom, "have not yet developed any of the learned dietary restraint that needs to be exerted by anyone wishing to remain slim in the modern environment."200

A study recently published in the American Journal of Preventative Medicine<sup>201</sup> investigated the health impact if fast food users switched from beef-based hamburgers to plantbased burgers. It was concluded that the consumption of 100 billion McDonald's beef burgers instead of the same company's McVeggie burgers would provide an additional 550 million pounds of saturated fat and 1.2 billion total pounds of fat, as well as one billion fewer pounds of fibre, 660 million fewer pounds of protein, and no difference in calories.<sup>201</sup> The McVeggie burger was therefore seen as a less harmful fast-food choice.201

The practise by some fast food companies of introducing healthier options has been welcomed by Dr Susan Jebb, Head of Nutrition and Health Research at the Medical Research Council Human Nutrition Research Centre in Cambridge: "Fast food companies could play a major part in halting the rise in obesity if they adopted a more positive attitude to healthy eating such as providing meals of lower energy density, appropriately marketed and with point-of-sale nutrition labelling."201

Dr lebb adds: "Many supermarket ready-meals and convenience foods are also very energy dense. If we're going to stem the tide of obesity, it's important that we don't just swap one unhealthy meal for another. Research has shown time and again that to maintain a healthy weight, we need to eat foods with less fat and added sugars and to take more exercise."200

#### Socio-economic trends in obesity

The common perception of healthy food as expensive seems a barrier to healthier eating. In supermarkets, it is convenience foods which are heavily price-promoted and there are numerous special offers on these sorts of food compared with relatively fewer price promotions on raw foods or 'ingredients'.<sup>212</sup> In addition, healthy convenience foods are frequently costly and it is only the more wealthy who can afford them.<sup>212</sup>

However, studies have demonstrated the enormous potential to construct a healthy diet using inexpensive products.<sup>195, 196</sup> Dry foods with a stable shelf life such as beans actually provide more protein at lower prices than perishable meats, fish or dairy<sup>176</sup> – and unlike the latter foods, they are also a valuable source of fibre.

The food industry is putting out a clear message, so what about the message being put out by the UK Government? One scheme in operation is the 5 A DAY Programme, which aims to increase the average consumption of fruit and vegetables in the UK to the recommended level of around five portions a day. It has made use of television and radio advertising, leaflets, posters, booklets, a web site and magazine adverts and articles and has local and national partners including industry, government departments and other agencies.<sup>193</sup> Nearly two million children aged four to six years receive a free piece of fruit or vegetable each school day under its School Fruit and Vegetable Scheme.<sup>193</sup> Over a quarter of children and their families have reported eating more fruit at home after joining the scheme - including those in lower socio-economic groups.<sup>193</sup> Clearly then, the eating habits of *all* socio-economic groups are open to change; it is simply a matter of sending out the message of what constitutes a healthy diet more loudly and clearly.

# Changes in levels of physical activity

#### Adults

The rising trend in obesity seems to correspond with a decline in physical activity and a rise in sedentary behaviour. The level of physical activity has fallen considerably in the last 50 years – the technological revolution has meant that physical activity is no longer an essential part of daily life. Large shifts towards less physically demanding work are being seen worldwide, and moves towards less physical activity include the increasing use of the car and wider car ownership; mechanised tools; laboursaving devices; an increase in energy-saving devices in public places – such as escalators, lifts and automatic doors; warmer dwellings and more passive leisure pursuits.<sup>49</sup> Leisure time is dominated by television, videos and computers.<sup>121</sup> The average person in England watched over 26 hours of television a week in the mid-1990s, compared with 13 hours in the 1960s.<sup>42</sup> It has been estimated that the extra physical activity involved in daily living 50 years ago, compared with today, is the equivalent of running a marathon a week.<sup>339</sup>

There is increasing evidence that many people are not taking sufficient exercise to significantly benefit their health. As stated earlier, before the technological revolution, people typically walked at least five to 10 miles a day, expending an extra 500 to 1,000 kcal.<sup>115</sup> Nowadays, approximately twothirds of men and three-quarters of women fail to meet the Department of Health's physical activity target of 30 minutes five times per week.<sup>20</sup>

The estimated cost of physical inactivity in England is around  $\pounds 2$  billion per year and each 10 per cent increase in activity across the population has a potential gain of  $\pounds 500$  million.<sup>54</sup>

#### Children

For many children, energy expenditure both at school and at home are significantly lower than for previous generations. *The National Diet and Nutrition Survey (2000)* showed that most young people aged between seven and 18 were inactive, as indicated by time spent in moderate or vigorous intensity activities.<sup>46</sup>

In the 1970s, 90 per cent of primary school children in the UK walked to school, compared with 10 per cent today.<sup>51</sup> Despite academic studies stressing that children learn the critical lifelong motivation to take part in recreational sport primarily between the ages of seven and 10, less than half of English children received the Government's target of two hours per week of PE in school in 2002.<sup>53</sup> In the UK amongst children in particular, sedentary activities such as computer use and TV viewing have all risen dramatically.

#### **Recommendations**

Low daily physical activity is a risk factor for weight gain and one to two short weekly walks are insufficient compensation.<sup>165</sup> It is perhaps obvious to suggest that becoming more active will help weight control by using up calories. People who exercise the most are repeatedly shown to be the least likely to be obese.<sup>122</sup> More importantly, people who are the most physically active gain less weight as they get older than those who are sedentary.<sup>123</sup>

The International Association for the Study of Obesity considers that, given modern environmental factors, 30 minutes of moderate daily exercise may be insufficient for many people to prevent unhealthful weight gain. Therefore additional exercise is recommended for those who find that this amount of daily activity does not prevent weight gain.<sup>63</sup> Increasing activity levels would contribute to the prevention and management of over 20 conditions and diseases including heart disease, diabetes, cancer, positive mental health and weight management. Increasing activity levels would also beneficially affect musculo-skeletal health, reducing the risk of osteoporosis, back pain and osteoarthritis.<sup>312</sup>

The Chief Medical Officer recommends that: "Children and young people should achieve a total of at least 60 minutes of at least moderate-intensity physical activity each day. At least twice a week this should include activities to improve bone health (activities that produce high physical stresses on the bones), muscle strength and flexibility."<sup>313</sup>

The recommendation for adults to maintain good health is: "A total of at least 30 minutes a day of at least moderateintensity physical activity on five or more days of the week."<sup>313</sup> This can be achieved either by doing all of the daily activity in one session, or in several shorter bouts of activity lasting 10 minutes or longer. Older people are also advised to keep moving and to maintain their mobility through daily activity. Activities geared at improving strength, coordination and balance are highly advocated.<sup>313</sup>

#### 10,000 steps per day

A recommendation of 10,000 steps per day has gained popularity with the media. A fair degree of similarity has been found between this recommendation and current public health guidelines, if walking is the main activity mode.<sup>166</sup> Individuals who accumulate at 10,000 steps per day have less body fat<sup>314,315</sup> and lower blood pressure<sup>314</sup> than their less active counterparts.

Based on currently available evidence, Tudor-Locke and colleagues<sup>166</sup> propose the following cut-off bands to assess physical activity levels in healthy adults, based on the use of a pedometer to count steps (see Figure 13): (i) less than 5,000 steps per day may be used as a 'sedentary lifestyle index'; (ii) 5,000 to 7,499 steps per day might be considered 'low active' and is typical of daily activity excluding sports or exercise; (iii) 7,500 to 9,999 steps per day might be considered 'somewhat active'; and (iv) 10,000 steps per day or above can be used to classify people as 'active'. Those who take more than 12,500 steps per day are likely to be classified as 'highly active'.<sup>166</sup> It is recognised that 10,000 steps per day is likely to be too low for young people, who are recommended to accumulate 11,000 steps (for girls) to 13,000 steps (for boys) at least five days per week for a standard healthy base.316

Campaigns to encourage pedometer use, combined with clear guidelines such as these may have a useful role in raising awareness of how much physically activity people

Current number of steps per day	Activity level
Less than 5,000	Sedentary
5,000 to 7,499	Low active
7,500 to 9,999	Somewhat active
10,000	Active
Over 12,500	Highly active

Adapted from Tudor-Locke et all<sup>166</sup>

take in their day-to-day lives, and hence provide vital inspiration to increase physical activity.

## Globalisation and the rise of globesity

Obesity is seldom seen among people who traditionally live on a diet with moderate fat content and a high content of relatively low calorie vegetables and wholemeal products.<sup>22</sup> Human weight regulatory mechanisms probably evolved at an energy-density of about only 108 kcal per 100g.<sup>199</sup>

However, as is the case in the West, the diets in developing countries are also changing with rising incomes. The share of staples, such as cereals, roots and tubers, is declining, while that of meat, dairy products and oil crops is rising. Meat consumption in developing countries rose by 150 per cent per capita between 1964-1966 and 1997-1999.<sup>29</sup> During the same period, milk and dairy consumption increased by 60 per cent.<sup>29</sup>

Jeremy Rifkin, president of the Foundation on Economic Trends in Washington DC, USA and author of *Beyond Beef*: *The Rise and Fall of the Cattle Culture* (Plume, 1992), *and The Biotech Century* (Victor Gollancz, 1998), reports that as developing countries become richer, enlarging their meat supply seems to be a main priority: "They start with chicken and egg production and, as their economies grow, climb the protein ladder to pork, milk, and dairy products, then to grass-fed beef and finally to grain-fed beef. Encouraging this process advances the interests of agribusinesses and two-thirds of the grain exported from the USA goes to feed livestock."<sup>317</sup>

Dr David Brubaker, director of the Henry Spira/GRACE Project on Industrial Animal Production, Centre for a Liveable Future, Johns Hopkins University School of Public Health, USA, comments: "In the developing world, the share of grain fed to livestock has tripled since mid-century and now stands at 21 per cent. This percentage is likely to grow further as developing nations strive to emulate the model of industrial nations, where nearly 70 per cent of grain is fed to livestock."  $^{\scriptscriptstyle 127}$ 

Famine, however, is still widespread across the globe. An acre of cereal produces five times more protein than an acre used for meat production; legumes such as beans, peas and lentils can produce 10 times more protein and, in the case of soya, 30 times more.<sup>129</sup> The adoption of Western dietary habits has led to a paradox where extremes of under-nutrition now coexist with overweight and obesity which affects up to 35 per cent of the adult population in some of the poorest countries of the world.<sup>2</sup>

Ironically, reverting to our roots of a whole foods, plant-based diet rich in the grains, vegetables and legumes increasingly fed to animals, combined with moderate physical activity can be the keys to both weight maintenance and improved health.

# REFERENCES

I. World Health Organization, 2005. Preventing Chronic Diseases: A Vital Investment. WHO Global Report.

2. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 381. (Elsevier Churchill Livingstone.)

3. Powell C, 2003. Obesity — the time bomb waiting to explode. Veggiehealth. Issue 3, p 6. (Vegetarian & Vegan Foundation.)

4. House of Commons Health Committee, 2004. Obesity Third Report of Session 2003-2004. (London: The Stationery Office Limited.)

5. Jotangia D, Moody A, Stamatakis E, Wardle H, April 2005. Obesity among children under 11. Health and Social Care Information Centre/Department of Health.

6. Glenny A, O'Meara S, Melville A, Sheldon TA, Wilson C, 1997. The treatment and prevention of obesity: a systematic review of the literature. Int J Obes; 21: 715-737.

7. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 383. (Elsevier Churchill Livingstone.)

8. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 382. (Elsevier Churchill Livingstone.)

9. International Obesity Task Force. www.iotf.org

10. Blair SN, Brodney S, 1999. Effects of physical inactivity and obesity on morbidity and mortality: current evidence and research issues. Med Sci Sport Exer; 31: S646-S662.

11. Hans TS, Feskens EJ, Lean ME, Seidell JC, 1998. Associations of body composition with type 2 diabetes mellitus. Diabet Med.; 15:129-35.

12. Gibney MJ, Elia M, Ljungqvist O, Dowsett J (Eds.) 2005. Clinical Nutrition. p. 20. (Blackwell Publishing.)

13. National Institute of Health/National Heart Lungs and Blood, 1998. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. The Evidence Report. Obes Res.; 6:51S-2095.

14. Han TS, Gates E, Truscott E, Lean MEJ, 2005. Clothing size as an indicator of adiposity, ischaemic heart disease and cardiovascular risks. J Hum Nutr Dietet.; 18: 423-430.

15. Gibney MJ, Elia M, Ljungqvist O, Dowsett J (Eds.) 2005. Clinical Nutrition. p. 46. (Blackwell Publishing.)

16. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 381. (Elsevier Churchill Livingstone.)

17. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 382. (Elsevier Churchill Livingstone.)

18. Gibney MJ, Elia M, Ljungqvist O, Dowsett J (Eds.) 2005. Clinical Nutrition. p. 288. (Blackwell Publishing.)

19. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 383. (Elsevier Churchill Livingstone.)

20. House of Commons Health Committee, 2004. Obesity Third Report of Session 2003-2004. (London: The Stationery Office Limited.)

21. British Heart Foundation, November 2004. Obesity (1 of 4): The Causes of Obesity. www.bhf.org.uk/factfiles

22. Astrup A, 2001. Healthy lifestyles in Europe: prevention of obesity and type II diabetes by diet and physical activity. Public Health Nutrition 4(2B): 499-515.

23. Raben A, Vasilaras TH, Moller AC, Astrup A, 2002. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. American Journal of Clinical Nutrition 76: 721-729.	4 R 4
24. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 385. (Elsevier Churchill Livingstone.)	v
25. Ministry of Agriculture, Fisheries and Food, 1991. Fifty years of the National Food Survey, 1940-1990 (ed. J M Slater). (HMSO, London.)	4 V
26. Wang YQ, Thomas B, Ghebremeskel K and Crawford MA, 2004. Changes in Protein and Fat Balance of Some Primary Foods: Implications for Obesity? Institute of Brain Chemistry and Human Nutrition (IBCHN). London Metropolitan University.	5 0 5
27. Currie A, 2004. Dishing the Dirt — The Secret History of Meat. (Viva!)	0
28. World Health Organization, 2000. WHO Consultation on Obesity. Obesity: Preventing and Managing the Global Epidemic.	5 H
29. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 18. (Elsevier Churchill Livingstone.)	5
30. NACNE, 1983. The National Advisory Committee on Nutrition Education. A discussion paper on proposals for nutritional guidelines for health education in Britain. (London: Health Education Council.)	V 5 a
31. Food Standards Agency/Department of Health, 2003. The National Diet & Nutrition Survey: adults aged 19 to 64 years. (London: TSO.)	5 M
32. Geissler C and Powers H (Eds.) 2005. Human Nutrition. Section CD 33.4(a) (Elsevier Churchill Livingstone.)	5 sj
33. The Food Magazine, October/December 2005. Issue 71. (The Food Commission).	5
34. Webb GP, 1995. Nutrition A Health Promotion Approach. (Edward Arnold.)	(I 9
35. Department for Environment, Food and Rural Affairs, 2001. National Food Survey 2000. (The Stationery Office: London.)	I
36. Jebb S and Steer T, 2003. Tackling the Weight of the Nation. (Flour Advisory Bureau/Grain Information Service.)	5 y T
37. Gibney MJ, Elia M, Ljungqvist O, Dowsett J (Eds.) 2005. Clinical Nutrition. p. 34. (Blackwell Publishing.)	0
38. WHO/FAO, 2003. WHO Technical Report Series 916. Diet, nutrition and the prevention of chronic diseases. (Geneva: WHO.)	C 6
39. Blundell JE, MacDiarmid JI, 1997. Fat as a risk factor for overconsumption: Satiation, satiety, and patterns of eating. J Am Diet Assoc.; 97: S63-S69.	n
40. Stubbs RJ, Harbron CG, Murgatroyd PR, Prentice AM, 1995. Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating ad libitum. American Journal of Clinical Nutrition; 62:316-329.	a G G
41. Tardoff MG, Alleva AM, 1990. Effect of drinking soda sweetened with aspartame or high fructose corn syrup on food intake and body weight. American Journal of Clinical Nutrition; 51: 963-969.	G 6
42. Office of Population Censuses and Surveys, 1994. General Household Survey. (HMSO.)	N
43. Sport England, 2000. Young People and Sport in England, 1999. (Sport England.)	6
44. Department of the Environment, Transport and the Regions, 1999. Transport Statistics Bulletin, National Travel Survey 1996-1998 Update. (DETR.)	6 u
45. Joint Health Surveys Unit on behalf of the Department of Health, 1999. Health Survey for England: Cardiovascular Disease '98. (The Stationery Office.)	R
46. Gregory J, et al, 2000. National Diet and Nutrition Survey: Young People Aged 4-18 years. Volume 1: Report of the Diet and Nutrition Survey. (The Stationery Office.)	P

47. World Health Organization,1998. Obesity: Preventing and Managing the Global Epidemic. Report of WHO Consultation on Obesity. (World Health Organization.)

9/

- 48. Hancox RJ, Milne BJ, Poulton R, 2004. Association between child and adolescent television viewing and adult health: A longitudinal birth cohort study. Lancet 364: 257-62.
- 49. House of Commons Health Committee, 2004. Obesity Third Report of Session 2003-2004 Volume I, p.41-43 para 133, 137, 144. (London: The Stationery Office.)
- 50. DEFRA, 2001. Cited in Henley Centre report prepared for Ofcom as reported in Childhood Obesity — Food Advertising in Context. 22 July 2004. Ofcom (the Office of Communications.)
- 51. DEFRA 2001. Cited in Henley Centre report prepared for Ofcom as reported in Childhood Obesity Food Advertising in Context. 22 July 2004. Ofcom (the Office of Communications.)
- 52. House of Commons Health Committee, Second Report of Session 2000-2001, Public Health, HC30, para 191.
- 53. House of Commons Health Committee (2004) Obesity Third Report of Session 2003-2004 Volume I, p.44 para 148. London: The Stationery Office Limited.
- 54. Game Plan (December 2002). A Strategy for Delivering Government's sport and physical activity objectives, p 47.
- 55. World Health Organization, 2000. WHO Consultation on Obesity. Obesity: Preventing and Managing the Global Epidemic.
- 56. Despres J-P, 1993. Abdominal obesity as an important component of insulin-resistance syndrome. Nutrition; 9:452-9.
- 57. W.H.O. (2004) Young people's health in context. Health Behaviour in School-aged Children (HBSC) study: international report from the 2001/2002 survey, Physical Activity pp 90-97 ISBN 92 890 I372 9. http://www.euro.who.int/Document/e82923.pdf. This report covers children aged 11, 13 and 15.
- 58. Marshall, S.J. et al (2002) Clustering of sedentary behaviour and physical activity among youth: a cross national study. Pediatric Exercise Science 14 (4) pp 401-417. Ho, S.M.Y. and Lee, T.M.C. (2001) Computer use and its relationship with adolescent lifestyle in Hong Kong. Journal of Adolescent Health, 29, pp 259-266.
- 59. See E. Stamatakis, 2002. Physical Activity (2002) In The Health of Children and Young People Chapter 4. Department of Health.
- 60. Measures of various forms of physical activity were taken and participation in activity for 30 minutes or more was converted into a summary physical activity levels variable to assess relationships between BMI/obesity prevalence and physical activity. The summary physical activity levels measure categories are as follows:
- Group 3: active for 30 minutes and over on at least 5 days a week (High activity levels).
- Group 2: active for 30 minutes and over on I-4 days a week (Medium activity levels).
- Group I: active at a lower level or not active at all (Low activity levels).
- 61. Spock B, Parker SJ. Dr. Spock's Baby and Child Care. 7th edition, Simon & Schuster, New York, NY, 1998.
- 62. Key TJ, Allen NE, Spencer EA, Travis RC, 2002. The effect of diet on risk of cancer. Lancet 360: 861-868.
- 63. Saris WH, Blair SN, van Baak MA, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obes Rev 2003;4:101–14.

- 64. Grundy SM, Blackburn G, Higgins M, Lauer R, Perri MG, Ryan D.
- Physical activity in the prevention and treatment of obesity and its comorbidities.
- Med Sci Sports Exerc 1999;31:S502-8.

65. Richardson SA, Goodman N, Hastorf AH, Dornbusch SM. Cultural uniformity in reaction to physical disabilities. Am Soc Rev. 1961;26: 241–247

66. Staffieri JR. A study of social stereotype of body image in children. J Perspect Soc Psychol. 1967;7:101–104

67. Kaplan KM, Wadden TA. Childhood obesity and self-esteem. J Pediatr. 1986;109:367-370

68. Sallade J. A comparison of the psychological adjustment of obese vs. non-obese children. J Psychosom Res. 1973;17:89-96

69. Stunkard A, Burt V. Obesity and the body image. II. Age at onset of disturbances in the body image. Am J Psychiatry. 1967;123:1443-1447

70. Lissau I, Sorenson TIA. Parental neglect during childhood and increased risk of obesity in young adulthood. Lancet. 1994;343:324-327

71. Mellbin T, Vuille J-C. Further evidence of an association between psychosocial problems and increase in relative weight between 7 and 10 years of age. Acta Pediatr Scand. 1989;78:576-580

72. Epstein LH, Myers MD, Anderson K. The association of maternal psychopathology and family socioeconomic status with psychological problems in obese children. Obesity Res. 1996;4:65-74

73. Dietz WH, Health Consequences of Obesity in Youth: Childhood Predictors of Adult Disease. 1998;101;518-525 Pediatrics

74. Hancox RJ and Poulton R. Watching television is associated with childhood obesity: but is it clinically important? International Journal of Obesity (2006) 30, 171–175

75. Westman EC, Yancy WS, Edman JS, Tomlin KF, Perkins CE. Effect of 6-month adherence to a very low carbohydrate diet program. Am J Med 2002;113:30-6.

76. Foster GD, et al. A randomized trial of a low-carb diet for obesity. N Engl J Med 2003; 348:2082–90.

77. Samaha FF, et al. A low-carbohydrate as compared with a low-fat diet in severe obesity. N Engl J Med 2003;348:2074-81.

78. 14th European Congress on Obesity International Obesity TaskForce Press Release June I 2005.

79. Rural Payments Agency, 2005. Available from: http://www.rpa.gov.uk/rpa/rpaweb.nsf?open

80. World Health Organization, 2003. Global Strategy On Diet, Physical Activity and Health.

81. Spock B, 1999. Dr Spock's Baby and Child Care. (Simon and Schuster.)

82. Physicians Committee For Responsible Medicine, 2005. Weight Control and Obesity Prevention in Children. Available from http://www.pcrm.org

83. Berkowitz VJ, 2000. A view on high-protein, low-carb diets. Journal of the American Dietetic Association 100: 1300, 1302-3.

Sabate J, 2003. Nut consumption and body weight. American Journal of Clinical Nutrition.
647s-50s.

85. Tackling Obesity in England, Report by the Comptroller and Auditor General

HC 220 Session 2000-2001: 15 February 2001. (The National Audit Office.)

86. Spencer EA, et al, 2003. Diet and body mass index in 38,000 EPIC-Oxford meat-eaters, vegetarians, and vegans. International Journal of Obesity. 27: 728-34.

87. Anne M. Fletcher, M.S., R.D., L.D. http://annemfletcher.com/

88. Oliveira M and Moura AS, 2003. Weight Loss Associated With a Daily Intake of Three Apples or Three Pears Among Overweight Women. Nutrition. 19: 253-256. 89. Dreon DM et al, 1999. A very-low-fat diet is not associated with improved lipoprotein profiles in men with a predominance of large, low-density lipoproteins. American Journal of Clinical Nutrition. 70: 412-8.

90. Rolls BJ, Ello-Martin JA and Tohill BC, 2004. What can intervention studies tell us about the relationship between fruit and vegetable consumption and weight management? Nutrition Reviews 62: 1-17.

91. Kahn HS et al, 1997. Stable behaviors associated with adults' 10-year change in body mass index and likelihood of gain at the waist. American Journal of Public Health 87: 747-54.

92. T. Colin Campbell, 2005. The China Study. p. 138. (Benbella.)

93. Ellis FR and Montegriffo VME, 1970. Veganism, clinical findings and investigations. Am J Clin Nutr 23: 249-255.

94. Berenson G et al, 1998. Association between multiple cardiovascular risk factors and atherosclerosis to children and young adults. The Bogalusa Heart Study. New Eng J Med. 338: 1650-1656.

95. Key T, Fraser GE, Thorogood M, et al, 1999. Mortality in vegetarians and nonvegetarians: detailed findings from a collaborative analysis of 5 prospective studies. Am J Clin Nutr. 70 (Suppl.): 516S-524S.

96. Bergan JG and Brown PT, 1980. Nutritional status of "new" vegetarians. J. Am. Diet. Assoc. 76: 151-155.

97. Appleby PN, Thorogood M, Mann J, et al, 1998. Low body mass index in non-meat eaters: the possible roles of animal fat, dietary fibre, and alcohol. Int J Obes 22: 454-460.

98. Dwyer JT, 1988. Health aspects of vegetarian diets. Am J Clin Nutr. 48: 712-738.

99. Key TJ and Davey G, 1996. Prevalence of obesity is low in people who do not eat meat. Brit Med Journ. 313: 816-817.

Pixley F, et al, 1985. Effect of vegetarianism on the development of gallstones in women.
British Medical Journal. 291: 11-12.

101. Frentzel-Beyme R, Claude J, Eilber U, 1988. Mortality among German vegetarians: first results after five years of follow up. Nutrition and Cancer. 11: 117-26.

102. Ornish D, Brown SE et al, 1990. Can lifestyle changes reverse coronary heart disease? Lancet. 336: 129-133.

103. Haffner SM, Valdez RA, Hazed HP, Mitchell BD, Morales PA, Stern MP, 1992. Prospective analysis of the insulin-resistance syndrome (syndrome X). Diabetes; 41:715-22.

104. T. Colin Campbell, 2005. The China Study. p. 139. (Benbella.)

105. T. Colin Campbell, 2005. The China Study. p. 140. (Benbella.)

106. T. Colin Campbell, 2005. The China Study. p. 140. (Benbella.)

107. Key TJ and Davey G, 1996. Prevalence of obesity is low in people who do not eat meat. Brit Med Journ. 313: 816-817.

108. Barnard ND, Scialli AR, Turner-McGrievy G, Lanou AJ and Glass J, 2005. The effects of a lowfat, plant-based dietary intervention on body weight, metabolism, and insulin sensitivity. Am J Med: 118: 991-997.

109. Physicians Committee for Responsible Medicine, Autumn 2005. PCRM's Study Shows the Weight-Loss Power of a Low-Fat Vegan Diet. Good Medicine Autumn 2005, p7. (Physicians Committee for Responsible Medicine.)

110. Ornish D et al, 1990. Can lifestyle changes reverse coronary heart disease? Lancet; 336: 129-33.

III. Barnard N, 2003. Breaking the Food Seduction, p66. (St. Martin's Griffin, New York.)

112. Hunninghake DB, Stein EA, Dujovne CA, 1993. The efficacy of intensive dietary therapy alone or combined with lovastatin in outpatients with hypercholesterolemia. N Eng J Med; 328: 1213-9.

113. Phillips F, Hackett AF, Stratton G and Billington D, 2004. Effect of changing to a self-selected	
vegetarian diet on anthropometric measurements in UK adults. Journal of Human Nutrition and Dietetics; 17(3): 249-55.	2
114. Walsh S, 2003. Plant Based Nutrition and Health p. 16. (The Vegan Society.)	
115. Walsh S, 2003. Plant Based Nutrition and Health p. 17. (The Vegan Society.)	-
116. Walsh S, 2003. Plant Based Nutrition and Health p. 19. (The Vegan Society.)	
117. Walsh S, 2003. Plant Based Nutrition and Health p. 21. (The Vegan Society.)	
118. Walsh S, 2003. Plant Based Nutrition and Health p. 23. (The Vegan Society.)	1
119. Diabetes Prevention Program Research Group, 2002. Reduction in incidence of type 2 diabetes with lifestyle intervention or metformin. New England Journal of Medicine; 346: 393-403.	
120. Tuomilehto J et al, 2001. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. New England Journal of Medicine; 344: 1343-1350.	1
121. Jebb S and Steer T, 2003. Tackling the Weight of the Nation. (Flour Advisory Bureau/Grain Information Service.)	
122. DiPietro L, 1995. Physical activity, body weight and adiposity: an epidemiologic perspective. Exercise and Sports Science Reviews; 23: 275-303.	1
123. Coakley EH et al, 1998. Predictors of weight change in men: Results from The Health Professionals Follow-Up Study. International Journal of Obesity; 22: 89-96.	
124. Fogelholm M, Kukkonen-Harjula K, 2000. Does physical activity prevent weight gain — a systematic review. Obesity Reviews; 1: 95-100.	ł
125. Henderson L, Gregoryn J, Irving K, Swan G, 2002. The National Diet and Nutrition Survey: adults aged 19 to 64 years. Energy, protein, carbohydrate, fat and alcohol intake. (London: HMSO.)	
126. The Worldwatch Institute, 2001. State of the World. Reported in Planet on a Plate. (Viva!)	
127. Brubaker D, 2004. Planet on a Plate. (Viva!)	I
128. Worldwatch Institute Press Release July 2, 1998. United States Leads World Meat Stampede. Reported in Planet on a Plate. (Viva!)	
129. Rifkin J, 2004. Feed the World. (Viva!)	I
I30. D'Silva J, January 2000. Factory Farming and Developing Countries, Compassion in World Farming Trust Briefing.	1
131. From National Food Survey http://statistics.defra.gov.uk/esg/publications/nfs/datasets/allfood.xls	I
132. Food Standards Agency, 2002. McCance and Widdowson's The Composition of Foods, Sixth Summary Edition. (Cambridge: Royal Society of Chemistry.)	ł
Milk fats used in analysis: skimmed milk, semi-skimmed milk, whole milk, yoghurt (plain), yoghurt (low fat), cream (single), butter, brie, camembert, cheddar, cottage cheese, cream	3
cheese, edam.	
Meat fats used in analysis: beef (trimmed lean, raw), lamb (trimmed lean, raw), pork (trimmed lean, raw), chicken breast (grilled without skin).	
Fatty fish used in analysis: mackerel (raw), herring (raw), trout (grilled), salmon (raw);	
Seeds and nuts used in analysis: almonds, brazils, peanuts, sesame seeds, sunflower seeds, walnuts.	
133. Food Standards Agency, 2002. McCance and Widdowson's The Composition of Foods, Sixth Summary Edition. (Cambridge: Royal Society of Chemistry.)	
134. Schwartz RP, 2003. Soft drinks taste good, but the calories count. Journal of Pediatrics; 142: 599-601.	
135. Gregory J, Low S, 2000. National Diet and Nutrition Survey of young people aged 4-18	

135. Gregory J, Low S, 2000. National Diet and Nutrition Survey of young people aged 4-18 years. Vol. I Report of the diet and nutrition survey. (London: HMSO.)

136. Raben A, Vasilaras TH, Moller AC, Astrup A, 2002. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 weeks of supplementation in overweight subjects. American Journal of Clinical Nutrition; 76: 721-729.

9/

137. Poppitt SD, Keogh GF, Prentice AM et al, 2002. Long-term effects of ad libitum low-fat, highcarbohydrate diets on body weight and serum lipids in overweight subjects with metabolic syndrome. American Journal of Clinical Nutrition; 75: 11-20.

138. Raben A, Astrup A, Vasilaras TH et al, 2002. The CARMEN trial: increased intake of carbohydrates – simple or complex – and unchanged blood lipids in overweight subjects. Ugeskr Laeger;164(5):627-31. (Abstract only)

139. Prentice AM, 1995. Alcohol and Obesity. International Journal of Obesity; 19: S44-S50.

140. Prentice AM, Diaz E, Goldberg GR et al, 1992. Famine and refeeding: adaptations in energy metabolism. In: Anderson GH, Kennedy SH, eds, 1992. The Biology of Feast and Famine: Relevance to Eating Disorders, pp. 245-67. (San Diego, CA: Academic Press.)

141. Geissler C and Powers H (Eds.) 2005. Human Nutrition. Section CD 33.3(a) (Elsevier Churchill Livingstone.)

142. Observer Food Monthly, Sunday May 15, 2005. It's supposed to be lean cuisine. So why is this chicken fatter than it looks?

http://observer.guardian.co.uk/foodmonthly/story/0,9950,1481443,00.html

143. Source: Ministry of Agriculture Fisheries and Food (1998), except \*where information is from US Department of Agriculture, Agricultural Research Service (2003). In: McKevith B, 2005. Nutritional aspects of oilseeds. British Nutrition Foundation. (London, UK.)

144. Leon DA and McCambridge J, 2006. Liver cirrhosis mortality rates in Britain from 1950 to 2002: an analysis of routine data. The Lancet; 367:52-56.

145. Howarth NC, Saltzman E, Roberts SB, 2001. Dietary fiber and weight regulation. Nutr Rev.;59:129 –139.

146. Jebb SA, 1997. Aetiology of Obesity. British Medical Bulletin; 53: 264-285.

147. Food Standards Agency, 2003. Consumer Attitudes Survey. Available at http://www.food.gov.uk/yourviews/surveys/foodsafety-nutrition-diet/cas2003survey

148. Prentice AM, Jebb SA, 2003. Fast foods, energy density and obesity: a possible mechanistic link. Obes. Rev.; 4(4): 187-94.

149. Food Standards Agency, 2005. How to be a healthy weight. Available at http://www.eatwell.gov.uk/healthydiet/healthyweight/howtobe/

150. Bingham SA, Luben R, Welch A et al., 2003. Are imprecise methods obscuring a relation between fat and breast cancer? Lancet; 362 (9379); 212-14.

151. Henderson L and Gregory J, 2002. The National Diet and Nutrition Survey Adults aged 19-64 years. Volume1: Types and quantities of foods consumed.

152. Department of Health, 1991. Dietary Reference Values for Food Energy and Nutrients in the United Kingdom. Report of the Panel on Dietary Reference Values of the Committee on Medical Aspects of Food Policy. (London: HMSO.)

153. Keys A, 1980. Seven Countries: multivariate analysis of death and coronary heart disease. (Cambridge Mass: Harvard University Press.)

154. Hegsted DM, McGandy RB, Myers ML et al, 1965. Quantitative effects of dietary fat on serum cholesterol in man. Am J Clin Nutr; 17: 281-295.

155. Mensink RPM, Katan MB, 1990. Effect of dietary trans fatty acids on high-density and lowdensity lipoprotein cholesterol levels in healthy subjects. N Engl J Med; 323: 439-45.

156. Stender S, Dyerberg J, 2004. Influence of trans fatty acids on health. Ann Nutr Metab; 48(2): 61-6.

157. Murray S, 2005. Chewing the fat on trans fats. CMAJ; 173(10): 1158-1159.

158. Institute of Medicine, September 2002. Dietary reference values for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Available at: http://books.nap.edu/html/dri\_macronutrients/reportbrief.pdf

159. Kris-Etherton PM, Yu S, 1997. Individual fatty acid effects on plasma lipids and lipoproteins: human studies. Am J Clin Nutr; 65 (suppl): 1628S-44S.

160. German JB and Dillard CJ, 2004. Saturated fats: what dietary intake. Am J Clin Nutr; 80: 550-9.

161. Buttriss J, 1999. n-3 Fatty Acids and Health. P.I. (BNE.)

162. Pereira C et al, 2001. The Alpha-Linolenic Acid Content of Green Vegetables Commonly Available in Australia. Int. J. Vitamin. Ntr. Res.; 71(4): 223-228.

163. Raben A, 2002. Should obese patients be counseled to follow a low-glycaemic index diet? No.Obes rev; 3(4): 245-56.

164. Physicians Committee for Responsible Medicine, 2002. Healthy Eating for Life to Prevent and Treat Cancer. (John Wiley & Sons, Inc., New York.)

165. Saris WHM, Blair SN, van Baak MA et al, 2003. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obesity Reviews; 4(2): 91-100.

166. Tudor-Locke C and Bassett DR, 2004. How Many Steps/Day Are Enough?

Preliminary Pedometer Indices for Public Health. Sports Med; 34 (1): 1-8.

167. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 86. (Elsevier Churchill Livingstone.)

168. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 87. (Elsevier Churchill Livingstone.)

169. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 98. (Elsevier Churchill Livingstone.)

170. Geissler C and Powers H (Eds.) 2005. Human Nutrition, p. 95. (Elsevier Churchill Livingstone.)

171. Smith AM, Baghurst KI, 1992. Public health implications of dietary differences between social status and occupational category groups. | Epidemiol Commun Health; 46: 409-16.

172. Hulshof KF, Brussaard JH, Kruizinga AG et al, 2003. Socio-economic status, dietary intake and 10 y trends: the Dutch National Food Consumption Survey. Eur J Clin Nutr; 57: 128-37.

173. Roos E, Prattala R, Lahelma E et al, 1996. Modern and healthy?: socioeconomic differences in the quality of diet. Eur J Clin Nutr; 50: 753-60.

174. Worsley A, Blasche R, Ball K et al, 2003. Income differences in food consumption in the 1995 Australian National Nutrition Survey. Eur J Clin Nutr; 57: 1198-211.

175. Leibtag ES, Kaufman PR, 2003. Exploring food purchase behaviors of low-income households. Current issues in economics of food markets. AIB 747-07. Washington, DC: ERS/USDA.

176. Drewnowski A and Darmon N, 2005. The economics of obesity: dietary energy density and energy cost. Am J Clin Nutr; 82 (suppl): 2655-735.

177. WHO technical support series 916, 2003. Diet, nutrition and the prevention of excess weight gain and obesity. Report of a joint WHO/FAO expert consultation. Geneva: WHO.

178. Swinburn BA, Caterson I, Seidell JC et al, 2004. Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutrition 7: 123-46.

179. French SA, Story M, Jeffery RW, 2001. Environmental influences on eating and physical activity. Annu Rev Public Health; 22: 309-35.

180. Stubbs RJ, Whybrow S, 2004. Energy density, diet composition and palatability: influences on overall food energy intake in humans. Physiol Behav; 81: 755-64.

181. Popitt SD, Prentice AM, 1996. Energy density and its role in the control of food intake: evidence from metabolic and community studies. Appetite; 26: 153-74.

182. McCrory MA, Fuss PJ, Hays NP et al, 1999. Overeating in America: association between restaurant food consumption and body fatness in healthy adult men and women ages 19 to 80. Obes Res; 7: 564-71.

183. French SA, Harnack L, Jeffrey RW, 2000. Fast food restaurant use among women in the Pound of Prevention study: dietary, behavioral and demographic correlates. Int J Obes Relat Metab Disord; 24: 1353-9.

184. Prentice AM, Jebb SA, 2003. Fast foods, energy density and obesity: a possible mechanistic link. Obes Rev: 7: 564-71.

185. Bowman SA, Gortmaker SL, Ebbeling CB et al, 2004. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. Pediatrics; 113: 112-8.

186. Zizza C, Siega-Riz AM, Popkin BM, 2001. Significant increase in young adults' snacking between 1977-1978 and 1994-1996 represents a cause for concern! Prev Med 32: 303-10.

187. Kant AK, 2000. Consumption of energy-dense, nutrient-poor foods by adult Americans: nutritional and health implications. The third National Health and Nutrition Examination Survey, 1988-1994. Am | Clin Nutr; 72: 929-36.

188. Bray GA, Nielsen SJ, Popkin BM, 2004. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. Am J Clin Nutr; 79: 537-43.

189. Berkey CS, Rockett HR, Field AE et al, 2004. Sugar-added beverages and adolescent weight change. Obes Res; 12: 778-88.

190. Wiehe S, Lynch H, Park K. Sugar high: the marketing of soft drinks to America's schoolchildren. Arch Pediatr Adolesc Med; 158: 209-11.

191. Rolls BJ, Morris EL, Roe LS, 2002. Portion size of food affects energy intake in normal-weight and overweight men and women. Am J Clin Nutr; 76: 1207-13.

192. Young LR, Nestle M, 2002. The contribution of expanding portion sizes to the US obesity epidemic. Am J Public Health; 92: 246-9.

193. World Health Organization, 2005. Preventing Chronic Diseases: A Vital Investment. WHO Global Report.

194. Spencer C. Fruits of the Past. (Viva!)

195. Raynor HA, Kilanowski CK, Esterlis I et al, 2002. A cost-analysis of adopting a healthful diet in a family-based obesity treatment program. J Am Diet Assoc; 102: 645-56.

196. Mitchell DC, Shannon BM, McKenzie J, 2000. Lower fat diets for children did not increase food costs. J Nutr Educ; 32: 100-3.

197. Blisard N, Stewart H, Jollife D, 2004. Low income households' expenditures on fruit and vegetables. Economic Research Service of the US Department of Agriculture. (Agricultural Economic Report publication 833.)

198. Bacon L, 2005. Eat Well: An Activist's Guide to Improving Your Health and Transforming the Planet, pl2.

199. Prentice AM and Jebb SA, 2003. Fast foods, energy density and obesity: a possible mechanistic link. Obes Rev.; 4(4): 187-94.

200. 'MRC study explains probable link between fast foods and obesity', MRC press release, 22 October 2003. http://www.mrc.ac.uk

201. Spencer EH, Frank E, McIntosh NF, 2005. Potential effects of the next 100 billion hamburgers sold by McDonalds. Am J Prev Med.; 28(4): 379-81.

202. Powell C, 2003. Obesity – the time bomb waiting to explode. Veggiehealth. Issue 3, p 6. (Vegetarian & Vegan Foundation.)

203. Prentice AM, 2001. Overeating: The Health Risks. Obesity Research; 9 Suppl 4:234S-238S

204. Stunkard A, Berkowitz R, Wadden T et al, 1996. Binge eating disorder and the night-eating syndrome. Int | Obes Relat Metab Disord.; 20: 1-6.

205. Prentice AM, Jebb SA, 1999. Energy expenditure and regulation of human energy balance. In: Kopelman P, ed. Appetite and Obesity: Disorders of Over- and Under-eating. London, UK: Royal College of Physicians; pp. 25-38

206. Linne Y, Barkeling B, Rossner S, 2002. Long-term weight development after pregnancy. Obesity Reviews; 3(2): 75-83

207. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 388. (Elsevier Churchill Livingstone.)

208. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 391. (Elsevier Churchill Livingstone.) 209. Food Standards Agency, 2002. McCance and Widdowson's The Composition of Foods, Sixth

Summary Edition. (Cambridge: Royal Society of Chemistry.)

210. Webb GP, 1995. Nutrition A Health Promotion Approach. (Edward Arnold.)

211. British Medical Association, 1986. Diet, Nutrition & Health, BMA Report, 4.11, p49.

212. Childhood Obesity – Food Advertising in Context. 22 July 2004. Ofcom (the Office of Communications.)

213. World Health Organization, 2003. Global Strategy On Diet, Physical Activity and Health.

214. Gibney MJ, Elia M, Ljungqvist O, Dowsett J (Eds.) 2005. Clinical Nutrition. p. 49. (Blackwell Publishing.)

215. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 382. (Elsevier Churchill Livingstone.) 216.British Medical Association, 1986. Diet, Nutrition and Health, BMA Report, 4.11, p49.

217. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 383. (Elsevier Churchill Livingstone.)

218. Steffen LM, Jacobs DR Jr, Stevens J, Shahar E, Carithers T, Folsom AR. Associations of wholegrain, refined-grain, and fruit and vegetable consumption with risks of all-cause mortality and incident coronary artery disease and ischemic stroke: the Atherosclerosis Risk in Communities (ARIC) Study. Am J Clin Nutr 2003; 78: 383–390

219. Liu S, Manson JE, Stampfer MJ, Hu FB, Giovannucci E, Colditz GA, et al. A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. Am J Public Health 2000; 90: 1409–1415

220. Bazzano LA, He J, Ogden LG, Loria CM, Vupputuri S, Myers L, et al. Fruit and vegetable intake and risk of cardiovascular disease in US adults: the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. Am J Clin Nutr 2002; 76: 93–99

221. Liu S, Lee IM, Ajani U, Cole SR, Buring JE, Manson JE. ; Physicians' Health Study Intake of vegetables rich in carotenoids and risk of coronary heart disease in men: The Physicians' Health Study. Int J Epidemiol 2001; 30: 130–135

222. Joshipura KJ, Ascherio A, Manson JE, Stampfer MJ, Rimm EB, Speizer FE, et al. Fruit and vegetable intake in relation to risk of ischemic stroke. JAMA 1999; 282: 1233-1239

223. Joshipura KJ, Hu FB, Manson JE, Stamfer MJ, Rimm EB, Speizer FE, et al. The effect of fruit and vegetable intake on risk for coronary heart disease. Ann Intern Med 2001; 134: 1106–1114

224. Gaziano JM, Manson JE, Branch LG, Colditz GA, Willett WC, Buring JE. A prospective study of consumption of carotenoids in fruits and vegetables and decreased cardiovascular mortality in the elderly. Ann Epidemiol 1995; 5: 255–260

225. Knekt P, Reunanen A, Jarvinen R, Seppanen R, Heliovaara M, Aromaa A. Antioxidant vitamin intake and coronary mortality in a longitudinal population study. Am J Epidemiol 1994; 139: 1180–1189

226. Albert CM, Gaziano JM, Willett WC, Manson JE. Nut consumption and decreased risk of sudden cardiac death in the Physicians' Health Study. Arch Intern Med 2002; 162: 1382–1387

233.	l
cons	u
Nutr	
234.	I
cons	u
1534	
235.	J
ische	2
Clin	N
236.	
men	2
237.	J
Nutr	
238.	I
Am J	
239.	١
pota	S
277:	I
240.	I
preli	
241.	F
pros	ta
Jour	
242.	I
treat	r
243.	I
cont	r
244.	I
Medi	c
245.	
Livin	8

- 227. Ellsworth JL, Kushi LH, Folsom AR. Frequent nut intake and risk of death from coronary heart disease and all causes in postmenopausal women: the Iowa Women's Health Study. Nutr Metab Cardiovasc Dis 2001; 11: 372–377
- 228. Hu FB, Stampfer M, Manson J. Frequent nut consumption and risk of coronary heart disease in women: prospective cohort study. BMJ 1998; 317: 1341-1345
- 229. Fraser GE, Sabate J, Beeson WL, Strahan TM. A possible protective effect of nut consumption on risk of coronary heart disease. The Adventist Health Study. Arch Intern Med 1992; 152: 1416–1424
- 230. Fraser GE, Shavlik D. Risk factors for all-cause and coronary heart disease mortality in the oldest-old. The Adventist Health Study. Arch Intern Med 1997; 157: 2249-2258
- 231. Jiang R, Manson JE, Stampfer MJ, Liu S, Willett WC, Hu FB. Nut and peanut butter consumption and risk of type 2 diabetes in women. JAMA 2002; 288: 2554–2560
- 232. Jacobs DR Jr, Meyer KA, Kushi LH, Folsom AR. Whole-grain intake may reduce the risk of ischemic heart disease death in postmenopausal women: the Iowa Women's Health Study. Am J Clin Nutr 1998; 68: 248-257
  - Liu S, Stampfer MJ, Hu FB, Giovannucci E, Rimm E, Manson JE, et al. Whole-grain umption and risk of coronary heart disease: results from the Nurses' Health Study. Am J Clin 1999; 70: 412–419
  - . Liu S, Manson JE, Stampfer MJ, Rexrode KM, Hu FB, Rimm EB, et al. Whole grain sumption and risk of ischemic stroke in women: a prospective study. JAMA 2000; 284: 1–1540
  - Jacobs DR Jr, Meyer KA, Kushi LH, Folsom AR. Whole-grain intake may reduce the risk of emic heart disease death in postmenopausal women: the Iowa Women's Health Study. Am J Nutr 1998; 68: 248–257
  - Jacobs DR Jr, Meyer HE, Solvoll K. Reduced mortality among whole grain bread eaters in and women in the Norwegian County Study. Eur J Clin Nutr 2001; 55: 137–143
  - Jenkins DJ, Kendall CW, Marchie A et al. Type 2 diabetes and the vegetarian diet. Am J Clin 2003; 78 (Suppl): 610S-616S
  - Liu S. Whole-grain foods, dietary fiber, and type 2 diabetes: searching for a kernel of truth. Clin Nutr 2003; 77: 527–529
  - Whelton PK, He J, Cutler JA, Brancati FL, Appel LJ, Follmann D, et al. Effects of oral issum on blood pressure. Meta-analysis of randomized controlled clinical trials. JAMA 1997; 1624–1632
  - . Riboli E, 22.06.2001. Meat, processed meat and colorectal cancer. EPIC Study iminary results.
  - Rouse IL, Beilin LJ, Mahoney DP et al, 1986. Nutrient intake, blood pressure, serum, urinary taglandins and serum thromboxane B2 in a controlled trial with lacto-ovo-vegetarian diet. nal of Hypertension; 4: 241-50
  - Margetts BM, Beilin LJ et al, 1985. Randomised controlled trial of a vegetarian diet in the tment of mild hypertension. Clinical Exp. Of Pharmacology & Physiology; 12: 263-66
  - Margetts BM, Beilin LJ et al, 1986. Vegetarian diet in mild hypertension: a randomized rolled trial. Br Med Journal; 293: 1468-71
  - Donaldson AN, 1926. The relation of protein foods to hypertension. Californian and Western icine; 24: 328
  - . Geissler C and Powers H (Eds.) 2005. Human Nutrition. pp. 364-65. (Elsevier Churchill 1gstone.)
- 246. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 367. (Elsevier Churchill Livingstone.)

247. Albert CM, Gaziano JM, Willett WC, Manson JE. Nut consumption and decreased risk of sudden cardiac death in the Physicians' Health Study. Arch Intern Med 2002; 162: 1382–1387

248. Ellsworth JL, Kushi LH, Folsom AR. Frequent nut intake and risk of death from coronary heart disease and all causes in postmenopausal women: the Iowa Women's Health Study. Nutr Metab Cardiovasc Dis 2001; 11: 372–377

249. Hu FB, Stampfer M, Manson J. Frequent nut consumption and risk of coronary heart disease in women: prospective cohort study. BMJ 1998; 317: 1341–1345

250. Fraser GE, Sabate J, Beeson WL, Strahan TM. A possible protective effect of nut consumption on risk of coronary heart disease. The Adventist Health Study. Arch Intern Med 1992; 152: 1416–1424

251. Fraser GE, Shavlik D. Risk factors for all-cause and coronary heart disease mortality in the oldest-old. The Adventist Health Study. Arch Intern Med 1997; 157: 2249–2258

252. Barnard ND, Scialli AR, Bertron P, Hurlock D, Edmonds K, Talev L. Effectiveness of a low-fat vegetarian diet in altering serum lipids in healthy premenopausal women. Am J Cardiol 2000; 85: 969-972

253. Fraser GE. Nut consumption, lipids, and risk of a coronary event. Clin Cardiol 1999; 22 (Suppl): IIIII- IIII5

254. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 371. (Elsevier Churchill Livingstone.)

255. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 370. (Elsevier Churchill Livingstone.)

256. Gibney MJ, Elia M, Ljungqvist O, Dowsett J (Eds.) 2005. Clinical Nutrition. p. 78. (Blackwell Publishing.)

257. World Health Organization, 2003. Global Strategy On Diet, Physical Activity and Health.

258. Centers for Disease Control and Prevention, 1998. National Diabetes Fact Sheet: National Estimates and General Information on Diabetes in the United States, Revised Edition. (Atlanta, GA: Centers for Disease Control and Prevention.)

259. Geissler C and Powers H (Eds.) 2005. Human Nutrition. p. 383. (Elsevier Churchill Livingstone.)

260. Snowdon DA, Philips RL, 1985. Does a vegetarian diet reduce the occurrence of diabetes? American Journal of Public Health; 75: 507-12.

261. West KM, Kalbfleisch JM, 1966. Glucose tolerance, nutrition and diabetes in Uruguay, Venezuela, Malaya and East Pakistan. Diabetes; 15: 9-18.

262. West KM, Kalbfleisch JM, 1971. Influence of nutritional factors on prevalence of diabetes. Diabetes; 20: 99-108.

263. Anderson JW et al, 1991. Metabolic effects of high-carbohydrate, high-fibre diets for insulindependent individuals. American Journal of Clinical Nutrition; 54: 936-43.

264. Barnard RG et al, 1983. Long-term use of a high-complex carbohydrate, high-fibre, low-fat diet and exercise in the treatment of NIDDM patients. Diabetes Care; 6: 268-73.

265. Munoz JM, 1984. Diabetes Care; 7: 297-300.

266. Crane MG, Sample CJ, 1995. Regression of diabetic neuropathy on total vegetarian (vegan) diet. Journal of Nutritional Medicine.

267. Roy MS et al, 1989. Nutritional factors in diabetics with and without retinopathy. American Journal of Clinical Nutrition; 50: 728-30.

268. World Health Organization, 2003. Global Strategy On Diet, Physical Activity and Health.

269. Geissler C and Powers H (Eds.) 2005. Human Nutrition. pp. 383 and 422. (Elsevier Churchill Livingstone.)

270. Thorogood M, et al, 1994. Risk of death from cancer and ischaemic heart disease in meat and non-meat eaters. British Medical Journal; 308: 1667-70.

271. Chang-Claude J, et al, 1992. Mortality pattern of German vegetarians after 11 years followup. Epidemiology: 3: 395-401.

272. Thorogood M, et al, 1994. Risk of death from cancer and ischaemic heart disease in meat and non-meat eaters. British Medical Journal; 308: 1667-70.

273. Block G, 1991. Epidemiological evidence regarding vitamin C and cancer. American Journal of Clinical Nutrition; 54: 13105-145.

274. Negri E, et al, 1991. Vegetable and fruit consumption and cancer risk. Int. Journal of Cancer; 48: 350-54.

275. Marcus E. Vegan - The New Ethics of Eating. (McBooks Press.)

276. International Obesity TaskForce, 2004. Obesity in children and young people, a crisis in public health. http://www.iotf.org

277. International Obesity TaskForce Press Release, Junel 2005. 14th European Congress on Obesity. International Obesity TaskForce. . http://www.iotf.org

278. National Forum for Coronary Heart Disease Prevention, May 1993. Food For Children: Influencing choice and investing in health. p.20.

279. Enas A, Senthilkumar A, Chennikkara H, Bjurlin MA, 2003. Prudent Diet and Preventive Nutrition from Pediatrics to Geriatrics: Current Knowledge and Practical Recommendations. Coronary Artery Disease in Asian Indians (CADI) Research Foundation, and University of Illinois, Chicago, USA

280. Cole TJ et al, 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. BMJ; 320:1240-3.

281. Thane CW and Bates CJ, 2000. Dietary intakes and nutrient status of vegetarian preschool children from a British national survey. J. Hum. Nutr. Dietet.;13:149-162.

282. British Meat Food Service:

http://www.britishmeatfoodservice.com/foodservice/pdfs/Choose\_Pork\_Guide.pdf

283. Renaud S, Delorgel M. Dietary lipids and their relation to ischemic heart disease from epidemiology to prevention. J Int Med 1989; 225 (Suppl): 39-46

284. Enas A, Senthilkumar A, Chennikkara H, Bjurlin MA, 2003. Prudent Diet and Preventive Nutrition From Pediatrics to Geriatrics: Current Knowledge and Practical Recommendations. Coronary Artery Disease in Asian Indians (CADI) Research Foundation, and University of Illinois, Chicago, USA

285. Grundy SM, Denke MA. Dietary influences on serum lipids and lipoproteins. J Lipid Res 1990; 31: 1149-1172

286. Knuiman JT, West CE, Katan MB, Hautvast JG. Total cholesterol and high density lipoprotein cholesterol levels in populations differing in fat and carbohydrate intake. Arteriosclerosis 1987; 7:612–619

287. Denke MA, Grundy SM. Comparison of effects of lauric acid and palmitic acid on plasma lipids and lipoproteins. Am J Clin Nutr 1992; 56: 895–898

288. Bonanome A, Grundy SM. Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. N Engl J Med 1988; 318:1244–1248

289. Grundy SM. Comparison of monounsaturated fatty acids and carbohydrates for lowering plasma cholesterol. N Engl J Med 1986; 314: 745-748

290. Renaud S, Delorgel M. Dietary lipids and their relation to ischemic heart disease from epidemiology to prevention. J Int Med 1989; 225 (Suppl): 39-46

291. Katan MB, Zock PL, Mensink RP. Dietary oils, serum lipoproteins, and coronary heart disease. Am J Clin Nutr 1995; 61 (Suppl): 1368S-1373S

292. Grundy SM, Denke MA. Dietary influences on serum lipids and lipoproteins. J Lipid Res 1990; 31: 1149–1172	3 d
293. Food Standards Agency, 2002. McCance and Widdowson's The Composition of Foods, Sixth Summary Edition. (Cambridge: Royal Society of Chemistry.)	3 A
294. Brown MS and Goldstein JL, 1984. How LDL receptors influence cholesterol and atherosclerosis. Scientific American; 251(5): 52-60.	a 3
295. Enas A, Senthilkumar A, Chennikkara H, Bjurlin MA, 2003. Prudent Diet and Preventive Nutrition from Pediatrics to Geriatrics: Current Knowledge and Practical Recommendations. Coronary Artery Disease in Asian Indians (CADI) Research Foundation, and University of Illinois, Chicago, USA	3 h 3 N
296. Walsh S, 2003. Plant Based Nutrition and Health p. 97. (The Vegan Society.)	3
297. Barnard N, 2003. Breaking the Food Seduction, p93. (St. Martin's Griffin, New York.)	t
298. Walsh S, 2003. Plant Based Nutrition and Health p. 97-99. (The Vegan Society.)	4
299. Brand-Miller J, 2005. Optimizing the cardiovascular outcomes of weight loss. Am J Clin Nutr; 81: 949-50.	3 tl
300. Mattes RD, Hollis J, Hayes D, Stunkard AJ, 2005. Appetite: Measurement and Manipulation Misgivings. J Am Diet Assoc; 105: S87-S97.	3 J
301. Eisenstein J, Roberts SB, Dallal G et al, 2002. High-protein weight-loss diets: Are they safe and do they work? A review of the experimental and epidemiological data. Nutr Rev; 60: 189-200.	3 ci 3
302. Halton TL, Hu FB, 2004. The effects of high protein diets on thermogenesis, satiety and weight loss: A critical review. J Am Coll Nutr; 23: 373-385.	n 3
303. U.S. Drug Administration. The Food Guide Pyramid. Washington, DC: U.S. Government Printing Office; 1992.	h 5
304. Ludwig DH. Dietary glycemic index and obesity. J Nutr. 2000;130:2805-3.	3
305. Physicians Committee for Responsible Medicine, Updated May 25, 2004. Analysis of Health Problems Associated with High-Protein, High-Fat, Carbohydrate-Restricted Diets Reported via an Online Registry. http://www.pcrm.org	3 V
306. http://www.AtkinsDietAlert.org/registry.html	a
307. Zemel, M.B., Thompson, W., Milstead, A., Morris, K. and Campbell P. 2004. Calcium and dairy acceleration of weight and fat loss during energy restriction in obese adults. Obesity Research. 12 (4) 582-590.	3 tl 4
308. Zemel, M.B., Richards, J., Russel, J., Milstead, A., Gehardt, L. and Silva, E. 2005. Dairy augmentation of total and central fat loss in obese subjects. International Journal of Obesity. 29 (4) 341-7.	3 a 3
309. Thompson, W.G., Rostad Holdman, N., Janzow, DJ, Slezak, J.M., Morris, K.L. and Zemel, M.B. 2005. Effect of energy-reduced diets high in dairy products and fiber on weight loss in obese adults. Obesity Research. 13 (8) 1344-53.	P 3 fi
310. Lanou A.J. 2005. Data do not support recommending dairy products for weight loss. Obesity Research. 13 (1) 191.	3 h
311. Barr S.I. 2003. Increased dairy product or calcium intake: is body weight or composition affected in humans? Journal of Nutrition. 133 (1) 245S-248S.	3 v
312. Department of Health, 2005. Choosing Activity: a Physical Activity Action Plan. (London: Department of Health.)	a 3
313. Department of Health, 2004. At least five a week: evidence on the impact of physical activity and its relationship to health. (London: Department of Health.)	h

314. Hatano Y. Use of the pedometer for promoting daily walking exercise. ICHPER 1993; 29: 4-8

9/

316. President's Council on Physical Fitness and Sports, 2001. The President's Challenge Physical Activity and Fitness Awards Program. Bloomington (IN): President's Council on Physical Fitness and Sports, US Department of Health and Human Services.

317. Rifkin J, 2004. Feed the World. (Viva!)

318. Singh PN, Sabate J, Fraser GE, 2003. Does low meat consumption increase life expectancy in humans? Am J Clin Nutr; 78 (Suppl): 5265–532S

319. Haddad EH, Tanzman JS, 2003. What do vegetarians in the United States eat? Am J Clin Nutr; 78 (Suppl): 626S-632S

320. Appleby PN, Thorogood M, Mann JI, Key TJ, 1998. Low body mass index in non-meat eaters: the possible roles of animal fat, dietary fibre and alcohol. Int J Obes Relat Metab Disord; 22: 454-460

321. Rajaram S. The effect of vegetarian diet, plant foods, and phytochemicals on hemostasis and thrombosis. Am | Clin Nutr 2003; 78 (Suppl): 5525–558S

322. Jenkins DJ, Kendall CW, Marchie A, et al, 2003. Type 2 diabetes and the vegetarian diet. Am J Clin Nutr 2003; 78 (Suppl): 6105–616S

323. Davis BC, Kris-Etherton PM. Achieving optimal essential fatty acid status in vegetarians: current knowledge and practical implications. Am | Clin Nutr 2003; 78 (Suppl): 6405-6465

324. Janelle KC, Barr SI. Nutrient intakes and eating behavior scores of vegetarian and nonvegetarian women. J Am Diet Assoc 1995; 95: 180–186, 189, 187–188

325. Jenkins DJ, Kendall CW, Popovich DG, Vidgen E, Mehling CC, Vuksan V, et al. Effect of a veryhigh-fiber vegetable, fruit, and nut diet on serum lipids and colonic function. Metabolism 2001; 50:494-503

326. Sacks FM, Castelli WP, Donner A, Kass EH. Plasma lipids and lipoproteins in vegetarians and controls. N Engl | Med 1975; 292: 1148–1151

327. Key TJ, Fraser GE, Thorogood M, Appleby PN, Bernal V, Reeves G, et al. Mortality in vegetarians and non-vegetarians: a collaborative analysis of 8300 deaths among 76,000 men and women in five prospective studies. Public Health Nutr 1998; 1: 33–41

328. Appleby PN, Thorogood M, Mann JI, Key TJ, 1998. Low body mass index in non-meat eaters: the possible roles of animal fat, dietary fibre and alcohol. Int J Obes Relat Metab Disord; 22: 454-460

329. Singh PN, Lindsted KD. Body mass and 26-year risk of mortality from specific diseases among women who never smoked. Epidemiology 1998; 9: 246-254

330. Snowdon DA, Phillips RL, Fraser GE. Meat consumption and fatal ischemic heart disease. Prev Med 1984; 13: 490–500

331. Fraser GE, Lindsted KD, Beeson WL. Effect of risk factor values on lifetime risk of and age at first coronary event. The Adventist Health Study. Am | Epidemiol 1995; 142: 746–758

332. Thorogood M, Mann J, Appleby P, McPherson K. Risk of death from cancer and ischaemic heart disease in meat and non-meat eaters. BMJ 1994; 308: 1667–1670

333. Key TJ, Fraser GE, Thorogood M, Appleby PN, Bernal V, Reeves G, et al. Mortality in vegetarians and non-vegetarians: a collaborative analysis of 8300 deaths among 76,000 men and women in five prospective studies. Public Health Nutr 1998; 1: 33–41

334. Singh PN, Sabate J, Fraser GE, 2003. Does low meat consumption increase life expectancy in humans? Am J Clin Nutr; 78 (Suppl): 5265–532S

GLOBESITY ~ |~ Why plant-based diets are the solution to the world's expanding epidemic

335. Enas A, Senthilkumar A, Chennikkara H, Bjurlin MA, 2003. Prudent Diet and Preventive Nutrition from Pediatrics to Geriatrics: Current Knowledge and Practical Recommendations. Coronary Artery Disease in Asian Indians (CADI) Research Foundation, and University of Illinois, Chicago, USA

336. Food Standards Agency, 2002. McCance and Widdowson's The Composition of Foods, Sixth Summary Edition. (Cambridge: Royal Society of Chemistry.) Analysis based on a 330 ml can of cola.

337. Butler J, 2006. White Lies: The health consequences of consuming cow's milk. (Vegetarian & Vegan Foundation.)

338. Canoy D, Wareham N, Luben R et al, 2005. Cigarette smoking and fat distribution in 21,828 British men and women: a population-based study. Obes Res. 13(8):1466-75

339. Tackling Obesity in England, Report by the Comptroller and Auditor General

HC 220 Session 2000-2001: 15 February 2001. (The National Audit Office.)

340. Armstrong B and Doll R, 1975. Environmental factors and cancer incidence in different countries. International Journal of Cancer; 15: 617-631.

341. Dansinger ML et al, 2003. One year effectiveness of the Atkins, Ornish, Weight Watchers and Zone Diets in decreasing body weight and heart disease risk Presented at the American Heart Association Scientific Sessions, November 12, 2003, Orlando, Florida.

342. Freedman MR, King J and Kennedy E, 2001. Popular diets: A scientific review. Obesity Research 9:IS-40S.

343. Food Standards Agency/Department of Health, 2003. The National Diet & Nutrition Survey: adults aged 19 to 64 years. (London: TSO.)

344. Prior IA, Davidson F, Salmond CE at al, 1981. Cholesterol, coconuts, and diet on Polynesian atolls: a natural experiment: the Pukapuka and Tokelau Island studies. Am J Clin Nutr; 34 (8): 1552-61.

345. Trinidad TP, Loyola AS, Mallillin AC et al, 2004. The cholesterol-lowering effect of coconut flakes in humans with moderately raised serum cholesterol. J Med Food; 7(2): 136-40.

346. Food Standards Agency, 2002. McCance and Widdowson's The Composition of Foods, Sixth Summary Edition. (Cambridge: Royal Society of Chemistry.)

347. Berkow SE, Barnard ND, 2006.Vegetarian Diets and Weight Status. Nutrition Reviews; 64(4): 175-88.

348. Berkey, C.S., Rockett, H.R., Willett, W.C. and Colditz, G.A. 2005. Milk, dairy fat, dietary calcium, and weight gain: a longitudinal study of adolescents. Archives of Pediatric and Adolescent Medicine; 159 (6): 543-50.

## APPENDIX I

Assessing weight in children

International cut-off points for body mass index (BMI) for overweight and obesity in children^{280}

International obesity cut-off points for BMI for overweight and obesity by gender between 2 and 12 years, defined to pass through BMI index of 25 and 30 kg/m<sup>2</sup> at age 18, obtained by averaging data from Brazil, Great Britain, Hong Kong, Netherlands, Singapore and United States

Age	BMI 25 kg/m2		BMI 30 kg/m2	
(years)	Males	Females	Males	Females
2	18.4	18.0	20.1	20.1
2.5	18.1	17.8	19.8	19.5
3	17.9	17.6	19.6	19.4
3.5	17.7	17.4	19.4	19.2
4	17.6	17.3	19.3	19.1
4.5	17.5	17.2	19.3	19.1
5	17.4	17.1	19.3	19.2
5.5	17.5	17.2	19.5	19.3
6	17.6	17.3	19.8	19.7
6.5	17.7	17.5	20.2	20.1
7	17.9	17.8	20.6	20.5
7.5	18.2	18.0	21.1	21.0
8	18.4	18.3	21.6	21.6
8.5	18.8	18.7	22.2	22.2
9	19.1	19.1	22.8	22.8
9.5	19.5	19.5	23.4	23.5
10	19.8	19.9	24.0	24.1
10.5	20.2	20.3	24.6	24.8
	20.6	20.7	25.1	25.4
11.5	20.9	21.2	25.6	26.1
12	21.2	21.7	26.0	26.7

## **APPENDIX 2**

Chemical characteristics and atherogenicity of major fatty acids295

Fatty acids	Chemical structure	Atherogenicity	Comments
	Saturated fatty acids (SAFA)		Highly atherogenic and thrombogenic. Markedly increases LDL level. Slightly increases HDL level.
Lauric acid	C12:0	t	Coconut oil 48%, palm oil 48% and butter fat 3%.
Myristic acid	C14:0	t t t	Most potent cholesterol-raising SAFA. Coconut 18%, palm kernel oil 18%, butter fat 18%, animal fats 1%-5%
Palmitic acid	C16:0	t t	Most common and reference standard of SAFA. Palm oil 45%, butter fat 26%, beef fat 26%, mutton fat 24%, chicken fat 23%, pork fat 25%, cocoa butter 26%, coconut oil 9% and palm kernel oil 8%.
Stearic acid	C18:0		Raises HDL level without raising LDL level. Butte fat 13%, beef fat 22%, mutton fat 25%, chicken fat 6%, pork fat 12%, cocoa butter 35%, coconut oil 3% and palm oil 4%.
	Trans fatty acids (TRAFA)		Increases Lp(a), TG, small, dense LDL levels. Decreases HDL level: 3-fold increase in cardiac arrest.
Elaidic acid	C18: 1 n-9 trans	t t t	Fried food, crispy food, cakes, biscuits, donuts, pizza, reused frying oils.
	Monounsaturated fatty acids (MUFA)		Significantly lowers LDL level. Raises HDL level. Lowers insulin resistance. Antiatherogenic and antithrombogenic.
Oleic acid	C 18:1 n-9	++	Butter fat 28%, beef fat 39%, mutton fat 33%, chicken fat 42%, pork fat 45%, cocoa butter 35%, coconut oil 7%, palm kernel oil 14%, and palm oil 39%.
	n-6 Polyunsaturated fatty acids		Lowers LDL levels. Lowers HDL level to a small small extent <sup>5</sup>
			Antiatherogenic.
Linoleic acid	C18:2 n-6		Predominant PUFA in western diets.
	n-3 Polyunsaturated fatty acids		Decreases LDL and TG levels, blood pressure and risk of sudden death. Increase HDL level. Antiarrhythmic and antithrombogenic effects.
Alpha-linolenic acid (ALNA)	C18:3 n-3		Precursor to EPA and DHA. Flaxseed oil 50%, rapeseed (canola) oil 10%, mustard oil 10%.
Eicosapentaenoic acid (EPA)	C20:5 n-3	++	Fatty fish (sardines, mackerel, salmon), plant algae
Docosahexaenoic acid	C22:6 n-3		Fatty fish (sardines, mackerel, salmon), plant algae

# GLOBESITY ~~ |~ Why plant-based diets are the solution to the world's expanding epidemic

## **APPENDIX 3**

Glycaemic load of common plant foods<sup>298</sup>

Food load	Energy (per 1000 calories)	Glycaemic (per 100 grams)
Potatoes	85	276
Sugar, brown	376	232
Sugar, white	387	232
Rice, white	120	220
Banana, ripe	90	217
Rice, brown	110	210
Bread, wholemeal	250	180
Bread, white	270	180
Oranges	50	150
Pasta, wholemeal	120	126
Pasta, white	130	126
Bread, pumpernickel	250	114
Banana, underripe	90	110
Beans	120	95
Cherries	60	75
Lentils	120	60
Tomatoes	20	29
Cashews	560	25
Avocados	160	23
Almonds	600	15
Hazel nuts	630	14
Oil	890	0



Vegetarian & Vegan Foundation (VVF) Top Suite, 8 York Court, Wilder Street, Bristol BS2 80H

T: 0117 970 5190

E: info@vegetarian.org.uk