



**Diet and obesity:
Manna from the Gods or the food of destruction
Professor Keith Kendrick
11 November 2004**

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Whisper to an unknown shrine
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A hoard of apples in his desk,
Who calls the hooded masses
To crawl before a burnished throne
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When I wrote this poem some 25 years ago I had in mind the simple fact that our biological need for satisfying our own hunger, and that of our children, by obtaining and consuming food at almost any cost has made us highly vulnerable to self-delusion and exploitation. In the subsequent 25 years that vulnerability has brought us to a stage where the availability of cheap high-energy foods coupled with a more sedentary lifestyle is potentially destroying the quality of many people’s lives through obesity and their attempts to control it. In that period of time the numbers of overweight or obese adults worldwide has more than doubled to an estimated 1 billion and it is estimated that 17.6 million children under 5 years old are affected. For the first time in several centuries some are claiming that we are looking at the possibility of obesity-related health problems actually reversing the trend of increasing life-expectancy.

It is almost impossible these days to read a newspaper or magazine without an article dealing with the problem of obesity or some new form of diet to combat it. Are we really destined to become a world of bloated, fat-laden bodies moving ponderously in ever decreasing circles between the fridge and the television and with high risk of amputated limbs and premature death from diabetes, heart-disease and cancer? If there is a real problem, what has precipitated it and does the solution lie with us as individuals, better education, the food industry, the pharmaceutical industry or with strong government advice and legislation?

A brief introduction to food, how our body uses it and how we regulate our appetite for it?

This is a large topic and I will only confine myself to some of the basics. The cells which collectively make up our body need energy to function and food supplies that energy. The energy contained in food is expressed traditionally in calories with one calorie being defined as a unit of energy sufficient to raise the

temperature of 1 gram of water by 1 degree centigrade. A calorie is also defined as 4.184 joules, another measurement of energy (one joule is the heat energy given off when an ampere flows through a resistance of one ohm for one second). Most packaged foods that we buy will display the amount of calories contained although to make matters confusing the units used at kilocalories (1000s of calories) and can either be abbreviated as kcal or simply as “C” as opposed to “c”.

We take in our calories in food from three main sources – carbohydrates, protein and fat. While carbohydrates and protein both contain 4 kilocalories per gram, fat contains over double that number (9 kcal per gram). Alcohol should not be left out of the equation either and provides 7 kcal per gram. Dietary fibre can also produce around 3 kcal per gram. The total number of calories we need to take in a day to meet our energy needs can be calculated quite precisely, although it will vary as a function of gender, size, age and activity level. Most adult humans require between 2000 and 3000 kcals a day (in the UK average for women = 1940 and for men = 2550). If you want to perform an accurate personal calculation then you can do this on the internet (www.bmi-calculator.net/bmr-calculator/). This calculates your basal metabolic rate (BMR) first (the number of calories needed if you were lying in bed all day) and then you can apply a multiplication factor depending upon how active you actually are. If you can't be bothered to do this and have a calculator handy then the Harris Benedict equation for BMR is:

Women: $BMR = 655 + (9.6 \times \text{weight in kilos}) + (1.8 \times \text{height in cm}) - (4.7 \times \text{age in years})$
 or $= 655 + (4.35 \times \text{weight in lbs}) + (4.7 \times \text{height in inches}) - (4.7 \times \text{age in years})$

Men: $BMR = 66 + (13.7 \times \text{weight in kilos}) + (5 \times \text{height in cm}) - (6.8 \times \text{age in years})$
 or $= 66 + (6.23 \times \text{weight in lbs}) + (12.7 \times \text{height in inches}) - (6.8 \times \text{age in years})$

To calculate the number of calories you need to consume per day the BMR needs to be multiplied by an activity factor: Sedentary (little or no exercise) = BMR x 1.2; Lightly active (light exercise/sport 1-3 days a week) = BMR x 1.375; moderately active (moderate exercise/sports 3-5 days/week) = BMR x 1.55; very active (hard exercise/sports 6-7 days per week) = BMR x 1.725; extra active (very hard exercise/sports & physical job 6-7 days week) = BMR x 1.9. The table below provides some information on how many calories are burned by different activities:

Total energy used by a man aged 25 years (weighing 65kg) to do various activities.

Average energy expenditure

Everyday Activities

KJ/min

Kcal/min

Sitting

6

1.4

Standing

7

1.7

Washing, dressing

15

3.5

Walking slowly

13

3

Walking moderately quickly

21

5

Walking up and down stairs

38

9

Work and Recreation

Light (most domestic work, golf, lorry driving, carpentry, bricklaying)

10-20

2.5-4.9

Moderate (gardening, tennis, dancing, jogging, cycling up to 20km per hour, digging)

21-30

5.0-7.4

Strenuous (coal mining, cross-country running, football, swimming [crawl])

>30

>7.5

Source: Ministry of Agriculture, Fisheries and Food (1992) Manual of Nutrition. HMSO, London

Some of the high calorie snacks and foods we eat can take a long time to burn off even with vigorous exercise.

Does it matter how we get our calories?

The simple answer to this is that “yes” it does. The different sources of food energy all provide different things:

Carbohydrates

These are the body’s preferred fuel since they can be quickly broken down to simple sugars (glucose) by the small intestine and can most easily be utilised for energy by all cells in the body. They come from a wide variety of foods – bread, rice, cereals, pasta, potatoes etc. Carbohydrates either come as sugars, starches and fibers. The basic building blocks are sugar molecules with starches and fibers being

composed of chains of sugar molecules which need to get broken down before they are absorbed. Carbohydrates are grouped as simple (glucose, fructose, dextrose or sucrose) or complex (rice, grains etc). Modern convention is to classify carbohydrates in terms of a glycemic index (GI - a measure of how much and how quickly they change blood glucose levels). High GI foods are composed of simple sugars which can be absorbed quickly into the blood from the small intestine and result in a rapid and large increase in blood glucose and insulin release. Low GI foods tend to be complex carbohydrates which need to be broken down into simple sugars before they can enter the blood and this results in a slower and smaller increase in blood glucose and insulin. High GI foods are both more likely to provide too many calories too quickly for energetic purposes (so they can end up being stored as fat) and may end up promoting insulin insensitivity (metabolic syndrome and type 2 diabetes).

Proteins

Proteins are chains of amino acids and these are broken down into their individual elements by the digestive system so that they can enter into the blood. These amino acids act as the building blocks for cells and help them to grow and maintain their structure. The liver can also convert amino acids into glucose, by a process called "gluconeogenesis, and so proteins are also an important source of energy as well. There are two different types of amino acids, essential and non-essential. Non-essential ones are those that can be created out of other chemicals found in your body (for example glycine synthesised from serine and threonine). Essential amino acids cannot be created and must be taken in from food (histidine, leucine, isoleucine, lysine, methionine, phenylalanine, threonine, tryptophan, valine). Most animal-based sources of protein (meat, milk, eggs etc) provide all essential and non-essential amino acids whereas vegetable-based sources are sometimes deficient in some of them (nuts, beans, soybeans etc).

Fats

When fats enter your digestive system they are broken down by an enzyme, lipase, into glycerol and fatty acids which are then reassembled into triglycerides for transport into the bloodstream. Muscle and fat cells can then absorb the triglycerides and either store or burn them for their energy requirements. Polyunsaturated, monounsaturated and saturated fatty acids are handled differently metabolically. Polyunsaturated fats (seeds, vegetable oils, fish oil etc – omega 3 fatty acids) are used for both energy and maintaining cell membranes and regulation. They are more readily mobilised from fat stores, especially during exercise and lower levels of both high (good)(HDL) and low (bad)(LDL) density lipoprotein forms of cholesterol. Monounsaturated fats (olive oil, nuts, avocados etc) are now largely regarded to be the most beneficial (good fat) since quite apart from the energy properties they have they not only lower levels of LDL cholesterol but increase those of HDL and may therefore help combat the development of cardiovascular problems. Saturated fats (meat, milk, butter, lard, cheese etc) are more difficult to break down, only provide energy, raise levels of both forms of cholesterol and are more likely to go into, and stay in, fat cells. The final category of fats to consider is called "transfats". These are created artificially by the food industry to prolong the shelf life of products and are generated by hydrogenating unsaturated fats. Unfortunately this ends up giving these transfats all the negative aspects of saturated fats – raising cholesterol and difficult to get rid of etc – even though they started off life as "good" unsaturated ones!

Suggested population averages for protein, carbohydrate and fat as a percentage of dietary energy.

Diet containing alcohol

Diet not containing alcohol

Protein

15

15

Total Carbohydrate

47

50

Non milk extrinsic sugars*

10

11

Total fat

33

35

Saturated fatty acids

10

11

Polyunsaturated fatty acids

6**

6.5

Trans fatty acids

2

2

Monounsaturated fatty acids

12

13

*NMES - free sugar not bound in foods, e.g. table sugar, honey and sugars in fruit juices, but excluding milk sugar.

** An individual maximum of 10% applies (with an individual minimum of 0.2% from linolenic acid, and 1% linoleic acid).

Alcohol should provide no more than 5% of energy in the diet.

Source: Department of Health (1991) Dietary Reference Values for Food Energy and Nutrients in the United Kingdom. HMSO, London.

How does the average British diet compare with the guidelines?

Contribution of fat, protein, carbohydrate and alcohol to the energy intake in the average British diet

Source: DEFRA & National Statistics (2001) National Food Survey 2000. The Stationery Office: London.

According to the British Nutrition Foundation, in order to achieve their recommendations many people will need to make significant changes to their pattern of eating. Achieving a balanced diet in practice will mean:

Eating more starchy foods such as bread, potatoes, rice and pasta. Assuming these replace fat-containing foods, this will help to reduce the amount of fat and increase the amount of fibre in the diet. Adding fat to these foods should be avoided or kept to a minimum.

Eating more fruit and vegetables. It has been suggested that individuals aim for at least 5 portions of different fruits and vegetables a day (excluding potatoes). As long as extra fat is not added to these foods, these changes will help to reduce fat intake, and increase intakes of fibre and important nutrients such as vitamin C.

Choosing leaner cuts of meat and lower fat versions of dairy products will help to reduce the amount of fat, particularly saturated fatty acids in the diet. Trimming fat, choosing cooking methods that do not require added fat and eating smaller portions of high fat foods can all be helpful.

Feast or famine

We, along with other animals have evolved highly efficient systems for acquiring, using and storing energy from food. Many of us now have food available on demand and eat regular main meals and snacks allowing us to get most of our energy requirements from the glucose that carbohydrates provide. It is also easy for us to eat more food than we need for immediate energy use and to store this as fat. The adaptability of this system to aid survival is however revealed when food is no longer available.

There is only around 40kcal of energy available as free glucose in the blood (enough for < 30 min). For the first 24 hours or so without food we can rely on stored glycogen in the liver which can be converted to glucose (about 2000 kcal). After that lipolysis starts breaking down the fat in fat cells to release fatty acids into the blood stream (around 100,000 kcals available). A number of cells in the body can utilise fatty acids for energy, notably muscles, although brain cells for example cannot. Once the liver has run out of glycogen it switches to converting the amino acids derived from protein into glucose (about 25,000kcal available). The liver then can also turn the fatty acids being produced by lipolysis of fats from fat cells into ketone bodies. Ketone bodies can also be utilised for energy by cells in the body and notably nerve cells can switch to using them as well.

Where individuals have normal levels of stored fat the above mechanisms can keep you alive for 30-40 days without food! A truly remarkable survival tool but one, fortunately, many of us no longer need.

Fat cells

Fat cells (adipocytes) are remarkable storage devices. We are born with around 5 million of them and this number increases during development (particularly during puberty) to reach around 30-50 billion in a normal weight adult. As mentioned above, an adult of normal weight has a total of around 100,000 kcal stored in fat cells.

So are fat cells just passive fat storage devices?

Up until around 10 years ago the only recognised additional, but very important, established role for fat was for making essential vitamins that are only fat soluble (vitamins A, D, E and K for example) available to the cells in our body. However, they are now recognised to play a much more active role in influencing a number of key physiological and behavioural functions including our vascular, immune and reproductive systems, sensitivity to insulin and even control of our feelings of hunger and satiety. These cells are now regarded as being highly complex hormone-producing factories which can secrete 25 or more different bioactive chemicals. As we will see in a moment, health problems associated with obesity may largely be due to the impact of having increased numbers and size of fat cells which change the magnitude of these chemical signals rather than fat-storage per se.

What controls feelings of “hunger” and “satiety”

Maintaining our energy balance and metabolism is the job of the central nervous system. To carry out its job it is dependent on a wide range of hormonal and neural feedback signals from the digestive system and fat cells. It also receives inputs from the sense organs which assess the appearance, smell, taste and texture of food. All this information, together with that from brain memory systems which allow us to recall past experiences with food and reward systems that link this with the level of associated pleasure, are integrated in various centres of the brain's basement motivation control centre – the hypothalamus. This region also receives controlling inputs from other areas of the brain which act to regulate the timing of meals, their size and duration and even how they are consumed.

Early laboratory animal studies confirmed that within the hypothalamus there are separate satiety and hunger systems with lesions of the former producing obese individuals and of the latter anorexic ones. However, it is only in the last 10 years or so that we have begun to understand that to a large extent there

is considerable overlap between them and multiple neurochemical agents involved.

Hormones that act on the hypothalamus to control food intake either do so quickly, and influence particular meals, or more slowly and influence the stability of the fat stores in the body. Of these slow acting hormones, the one that has been known about for some time is insulin with the brain having receptors for this hormone just like cells in muscles and fat. However, some 30 years ago mutant mouse strains were discovered which were naturally obese through disruption of another unknown hormonal system. The two strains were called obese (ob for short) and diabetic (db for short) and different genes were implicated. The gene disrupted in the ob mice was shown by Jeffrey Friedman's group at Rockefeller University in the USA in 1994 to be for a hormone produced by fat cells and which they called "leptin". When leptin deficient mice were injected with leptin they lost weight quickly and the weight also stayed off. There was huge excitement with this discovery since it was felt that the way to an antiobesity drug had been found and Amgen reportedly paid \$20 million to Friedman's University (Rockefeller) for the rights. However, leptin did not turn out to be a miracle drug for humans although several years later Steven O'Rahilly in Cambridge discovered that some obese humans have this same leptin mutation and that they did respond to leptin by losing both their voracious appetite and excess weight. It now seems that a better target may be the receptors for leptin since obesity is associated with them becoming insensitive to leptin and the obese db/db mouse also has impairments in these receptors.

Since then, a number of other substances have been identified which are secreted by the gastrointestinal system which can either promote hunger (ghrelin) or satiety (cholecystokinin, pancreatic polypeptide (PP) and peptide YY (PYY), glucagon-like peptide-1 (7-36) amide (GLP-1) and oxytomodulin (Oxm). These influence the activity of a hunger promoting system in the hypothalamic arcuate nucleus (neuropeptide Y and agouti-related peptide AgRP) and a satiety promoting system (pro-opiomelanocortin – POMC and alpha-melanocyte stimulating hormone α MSH) and their various different receptors (see Murphy and Bloom 2004 for more details).

While the level of detail that has been uncovered in these appetite control systems is impressive, two notes of caution have already been demonstrated: firstly, what happens in mice does not necessarily predict what will happen in humans when these different chemical pathways are manipulated; secondly it seems far easier to make animals obese through manipulating single genes than it is to reduce hunger. Perhaps we have evolved more compensatory mechanisms for promoting hunger responses than for protecting against obesity as a result of impaired satiety regulation.

The hunger and satiety system works better with solids than liquids

It is becoming increasingly established that taking calories in liquid form has less impact on satiety than taking them in solid form. A recent study have shown that when individuals receive large numbers of additional calories in liquid form they end up in a state of positive energy balance and increase body fat and BMI whereas when the same number of additional calories are taken in solid form they do not. Thus routine consumption of high sugar soft drinks or alcoholic beverages is a very good way of consuming more calories than your body needs for its immediate energy requirements.

This is not really that surprising since from an evolutionary perspective the availability of high caloric fizzy drinks, fruit juices and alcoholic beverages is a relatively recent phenomenon and the pathways in the brain dealing with hunger and thirst are quite distinct.

What defines obesity?

As mentioned above, our body has a highly efficient storage mechanism for excess energy intake from food – fat. If we persistently take in more energy from food than our body needs to run itself, then we store more and more fat and the battle of the bulge begins as the fat that forms around our organs and under our skin pushes outwards. The main concern is about fat levels around our internal organs since they have most immediate access to dumping their secretions and content into the blood stream. Obesity level is quantified by assessing the extent to which excess fat is being stored by our body.

The general classification for levels of obesity is that produced by the World Health Organisation in 2000. It is based on a single measurement called the body mass index (BMI). This is your weight in kilograms divided by your height in metres squared ($BMI = wt(kg)/ht(m)^2$) (try www.bmi-calculator.net/). For the majority of individuals this is a reasonably accurate measure although it has some shortcomings in that it

takes no account of exactly where fat is being stored and is very misleading in athletes who build up a large muscle mass and have very little fat – the BMI will often classify the latter as obese simply because muscle is very heavy and if you build up a large muscle bulk you can have a high BMI score. However, for most of us the BMI is a good quantitative index. While it is simple to calculate, and there are numerous low-cost machines on the market to help in this respect, it is a measure that relatively few individuals bother to make routinely, if at all.

More accurate methods of assessing the proportion of fat compared with muscle and water now exist which use direct bioelectric impedance measures although the machines are more expensive and it can take a few minutes to obtain the necessary readings. However, another quick method that can be used is to simply measure hip and waist circumference since the main contribution to an expanded waistline is almost certain to be fat. Where waist circumference exceeds 35 inches in women and 40 inches in men this is generally considered to represent an obese condition that could represent a health risk.

Classification of Body Mass Index and Risk of Co-morbidities

Classification

BMI (kg/m²)

Risk of co-morbidities

Underweight

<18.5

Low (but risk of other clinical problems increased)

Normal range

18.5–24.9

Average

Overweight

25.0-29.9

Mildly increased

Obese

>30.0

Class I

30.0-34.9

Moderate

Class II

35.0-39.9

Severe

Class III severe (or 'morbid obesity' or 'super obesity')

>40.0

Very severe

Source: International Obesity task force

So do we really have a problem and does it really matter?

Unfortunately, the simple answer to this is “yes we most certainly do” and failure to act now will jeopardise the quality of life of both current and future generations.

The House of Commons Health Committee report on Obesity published in May 2004 (www.parliament.the-stationery-office.co.uk/pa/cm/cmhealth.htm) has highlighted a number of worrying conclusions in its overall summary:

- Around two-thirds of the population of England are overweight or obese.
- Obesity has grown by almost 400% in the last 25 years.
- On present trends obesity will soon surpass smoking as the greatest cause of premature loss of life.
- It will entail levels of sickness that will put enormous strains on the health service.
- Today’s generation of children may be the first for over a century for whom life-expectancy falls.
- Obesity is associated with many health problems including coronary heart disease, diabetes, kidney failure, osteoarthritis, back pain and psychological damage.
- A strong association between obesity and cancer has only recently come to light.
- Estimated economic cost of obesity is £3.3–3.7 billion per year and of obesity plus overweight £6.6–7.4 billion.

In the UK the number of obese individuals has increased by 400% in the last 25 years and projected to reach over 50% by 2025. There are, of course, regional differences with a recent survey having revealed that Hull is the chubbiest town in Britain and Kingston-upon-Thames the leanest. The most likely individuals to be overweight are white, working class families who have poor education and do little exercise. By contrast lean towns are populated by a higher proportion of individuals who are well educated, have the money to eat well and exercise regularly. The rapid increase in obesity is also reflected in other countries (see below).

The Global Epidemic

Prevalence rates for obesity (body mass index [BMI] >30kg/m²) and overweight (BMI 25-30) continue to grow throughout the world.

Slide 12 is “The Global Epidemic” 3D projections graph which is too big to store with this file: apologies. It is on the disk as: project.wmf and can be inserted as a picture on a slide. It can also be accessed through our Web Site and copied from there.

<http://www.rri.sari.ac.uk/iotf>

A recent study has concluded that at least one in 13 annual deaths in countries in the European Union are likely to be related to excess weight. The UK has the highest individual percentage of all, with 8.7% of deaths being attributable to excess weight.

Obesity in children

Perhaps the most worrying trend is the increasing proportion of children who are obese or overweight. The figure below that summarises the current levels in Europe is taken from the International Obesity Task Force website. While the situation is certainly bad in the UK it can be seen from the figure that it is even worse in many Mediterranean countries.

Obesity, early sexual maturation and infertility

Many of us suspect that our children are maturing physically earlier and earlier but without necessarily a concomitant acceleration in mental maturity. If true such a scenario may be contributing not only to the alarming rise in teenage sex and pregnancies but possibly also to the equally alarming rise in the incidence of serious depression and suicide in the younger population (see my last lecture: “Stress, anxiety and depression – 7 th October 2004). Recent studies conducted in the USA have now confirmed that both overweight and obese girls are more likely to mature sexually earlier however the opposite is true for boys. The differences were most marked in obese children where girls were twice as likely to reach sexual maturity earlier than late and boys were half as likely. However, it should be pointed out that some studies have reported a significant increase in the prevalence of overweight and obesity in girls as a result of early sexual maturation and so cause and effect is far from clear.

How we get our calories may also be important with diets high in fat, protein and processed foods being associated with an earlier onset of menstrual cycles, and vegetarianism with a late onset. Similarly while the British derive over 40% of their calories from fat and generally start menstrual cycles at 13 years old, Chinese have only 15% of their calories as fat and do not start until 17.

Severe obesity is also linked with low fertility in both males and females with males having lower sperm counts and females irregular cycles.

The likely cause of these effects is that fat cells actually produce oestrogens and in obese individuals these enhance onset of puberty in females whereas in males they counteract the effects of androgens and so delay puberty. Oestrogens are also well known to suppress sperm production in adult males and excess oestrogen in adult females will disrupt the control of the menstrual cycle.

Obesity and cardiovascular disease

Obese individuals with a BMI >30 have around a 3-fold increase in risk of heart disease. The contributions of obesity to cardiovascular disease are likely to be multiple. Larger amounts of fat released into the blood will contribute to clogging up arteries and it is known that obesity is associated both with increased “bad” cholesterol (low-density lipoprotein – LDL) concentrations and decreased levels of “good” cholesterol (high-density lipoprotein – HDL).

Recent studies have found that fat also produces an enzyme “angiotensinogen” which acts to constrict blood vessels and both exacerbate this problem and increase blood pressure. Fat cells produce a number of inflammatory agents which may act to fragment the deposits clogging arteries and trigger heart attacks.

Fat cells produce a hormone “adiponectin” which recent studies suggest may protect against heart disease. Men with the highest levels of this hormone are 40% less likely to have heart attacks or die of heart disease than those with the lowest levels (Pischon et al 2004). While this hormone is produced by fat cells, obese individuals actually produce less of it than normal which might also help explain their greater risk of heart problems.

Obesity, metabolic syndrome and diabetes “diabesity”

Type 2 diabetes is the most common of the two forms (in the UK 1.5 million people have type 2 diabetes and 0.25 million type 1 in the USA around 14.4 million have type 2 and 1.6 million type 1) and rather than being the result of destruction of insulin-producing cells in the pancreas is associated with a condition whereby the body has a reduced ability to respond appropriately to insulin. This effectively means that cells have a reduced ability to take in the energy they require from sugar in the blood which both impairs their ability to function and leads to elevated blood sugar levels which may, for example, damage eyes, kidneys, nerves and heart.

In the UK diabetes costs around 9% of the total NHS budget and this is predicted to rise to 25% by 2025.

At least 80% of individuals with type 2 diabetes are overweight or obese. The risk of becoming diabetic becomes seriously increased even when individuals are still not classified as clinically obese, although it is even worse if you are. For example in women with a BMI of 28 there is an 18-fold increase whereas with a BMI of 35 there is a 92-fold increase (compared with a BMI of 22.5).

Diabetes is also associated with health inequalities: diabetes is three to five times more common in people of African and Caribbean origin living in the UK.

Two hormones produced by fat cells have so far been linked with the insulin resistance seen in metabolic syndrome and type 2 diabetes. The first, which I have already mentioned in relation to cardiovascular disease, is “adiponectin”. Studies in mice have shown that this helped diabetic animals make better use of insulin and we know that levels are reduced in obese individuals. More recently another hormone has been identified which has been called “resistin” (resistance to insulin). Normal mice given high levels of this hormone developed impaired insulin action and glucose intolerance (precursors of type 2 diabetes). The levels of this hormone rise after feeding and fall after fasting and are very high in mice with genetically or diet-induced obesity. However, at this stage the relevance of this hormone in humans has yet to be completely established.

Obesity and cancer

The National Obesity Forum has presented evidence to suggest that around 20 different cancers have been linked to obesity. They also noted that in the morbidly obese, death rates from cancer were 52% higher for men and 60% higher for women. The cancers most strongly linked to obesity are those of the breast (post-menopausal), uterus, colon, kidney, oesophagus, pancreas and gallbladder. The American Cancer Society estimates that reducing obesity levels could reduce annual cancer deaths in the USA by 90,000.

Once again there may be a number of factors whereby obesity could cause cancer and at this stage most of the links are epidemiological. Probably the strongest link is in relation to breast cancer in post-menopausal women with obese individuals having up to 3-fold higher oestrogen levels than non-obese ones. Before menopause, obese women appear less likely to develop breast cancer, probably because fewer ovulations occur as a result of high oestrogen levels. However, a disturbing general finding is that the heavier women are when they are diagnosed with breast cancer the more likely they are to die from the disease (for example, a study from the USA published in 2003 reported a 2.5-fold increased risk of dying).

Hormones produced by fat, such as leptin, have also been linked with cell proliferation and obese people with large numbers of fat cells usually have high levels. Obese, leptin-deficient mice, for example, seem to be protected against developing tumours as a result of treatment with cancer-inducing agents. Many of the substances produced by fat cells are also pro-inflammatory which could also help promote cancer development.

Obesity and sleep apnea

As I discussed in my lecture “To sleep perchance to dream” (December 2003), overweight males are prone to both snoring and the development of sleep apnea - where breathing is prevented during sleep for periods of up to 10 seconds or more (often as many as 100 times a night) by excess fat in the neck helping to block the airway. This can result in the individual experiencing persistent headaches and feeling tired and depressed.

Obesity and osteoarthritis

A combination of the extra load put on our joints from having to carry around extra weight and excess pro-inflammatory chemicals produced by large numbers of fat cells may increase the risk of developing arthritis in load-bearing joints.

Obesity and life expectancy

In my lecture on ageing (Turning back the hands of time: growing old gracefully – March 2004), I discussed how animal work had shown that the most reliable way of prolonging life expectancy was to significantly reduce calorie intake it should therefore not come as a surprise to learn that obesity is associated with the opposite outcome. The Governments recent Health Committee report concluded that there is a nine-year reduction in life expectancy amongst obese patients. This prognosis is made even worse if obese individuals also smoke. Generalised obesity (fat distributed around the whole body) results in alterations in the blood circulation and heart function, while central/abdominal obesity (fatness mainly around the chest and abdomen) further restricts chest movements and alters breathing function. Fat around the abdomen is

also a major contributor to the development of diabetes, hypertension, and alterations in blood lipid (fat and cholesterol) concentrations.

Obesity and psychological problems

In both children and adults obesity is linked to higher rates of low self-esteem and depression. Obese individuals are often on the receiving end of unwanted attention and negative comments which can be a particular problem with children where there is often no middle ground i.e fat individuals tend to end up either as bullies or being bullied. This can easily lead to a sense of being cut-off from society and its values, leading to stress, anxiety, depression and comfort binge eating. As we have also been seeing in recent years this scenario can often lead to suicide.

There are also psychological problems associated with long-term exposure to high fat and high sugar diets in relation to both mood swings and cognitive skills.

Obesity vs dieting vs lack of exercise?

The official view of the US and UK Government Health advisors is that these health problems are directly associated with obesity. However, there are those who urge caution in blaming the above health problems simply on excess storage of fat. It should be emphasised that it is very difficult to establish cause and effect directly. There is some evidence being produced that repeated weight loss and weight gain in individuals trying to fight their weight problems, coupled with the fact that overweight and obese individuals are more likely to have lifestyles where exercise and general fitness levels are poor, may be more potent risk factors than being obese per se (see Paul Campos 2004 "The Obesity Myth"). Some research by Steven Blair in the USA has actually claimed that obese individuals who exercise have half the death rate of those who are normal weight but do not exercise.

What has caused the problem?

The answer to this in a nutshell could not be simpler. Food supplies the body with energy and where energy input exceeds energy requirement it gets stored as fat. This energy stored as fat acts as a useful reserve for periods when energy supplies from food become scarce. You only need to eat 50 kcal a day more than you need for energy and in one year this will convert to an extra kilogram of fat. The major problem of obesity therefore resides in the simple conclusion that we are taking in more energy from food than we need and the days of food scarcity are largely forgotten.

To make matters worse we now know that as we pile on the pounds we don't just simply fill up our complement of 30-50 million fat cells to maximum size, we actually generate more of them so that an overweight individual might have 100 million and severely obese individuals can get up to 270 million. These extra fat cells are very hard to get rid of and so when we diet and start to empty them of their fat contents, the volume of the chemical messages they send to our brain to tell us to eat more reaches fever pitch! This is why so many of us lose weight on diets only to put it all back again in a short period – it is really difficult to ignore the chemical pleadings of all those extra fat cells telling us they are empty.

As Joseph Conrad expressed in "Heart of Darkness":

"No fear can stand up to hunger, no patience can wear it out, disgust simply does not exist where hunger is; and as to superstition, beliefs, and what you may call principles, they are less than chaff in the breeze. It's really easier to face bereavement, dishonour, and the perdition of one's soul than this kind of prolonged hunger."

So, have we simply become gluttons?

Our motivation to eat food and the pleasure we experience from doing so is strong and high caloric foods can easily spawn bingeing and withdrawal behaviours reminiscent of "addiction" although whether we can become addicted to food in the same way as to drugs is still a matter of dispute. However, it may come as a surprise to many that there is no overall evidence supporting a conclusion for the rapid increase in

obesity over the last 50 years being due to an increase in average calorie intake. On average we actually take in fewer calories than we did 50 years ago although the form in which we consume calories shows an overall increase in consumption of fat (particularly saturated fats) in relation to carbohydrates (see Prentice and Jebb, 1995).

What has declined in close association with our increase in obesity is our level of physical activity. Fewer jobs now involve a significant amount of physical labour, there are more labour-saving devices in the home, we make greater use of cars for travelling even short distances and spend increased amounts of our leisure time in front of televisions and computers rather than playing sport or taking some form of exercise. The patterns of the curves on a graph plotting the rise in obesity against the rise in hours spent in front of a television are almost identical!

The World Health Organisation have also identified increased consumption of energy-dense foods high in saturated fats and simple sugars coupled with reduced physical exercise as the key causes of the rise in obesity.

While current dogma considers the root cause of the obesity problem to be high energy diets and lack of exercise causing an expansion and proliferation of fat cells that is difficult to reverse, changes in the rewarding aspects of food with obesity may also contribute.

Eating food is not only necessary for survival but is also pleasurable (as already discussed). Evidence from both human brain imaging and laboratory animal studies has shown that obesity is associated with decreased levels of dopaminergic activity in reward centres in the brain. A simplistic interpretation of these findings is therefore that obese individuals need to eat more in order to gain the same level of reward as obtained by normal weight individuals. The net result of this would be that obese individuals will be motivated to eat more.

Is the problem down to genes or environment?

The simple answer to this is that both genetic and environmental factors contribute to obesity. However, we cannot lay the blame for the recent rise in obesity on genetic changes since they could not have occurred in the last 50 years. While it is certainly true that some individuals are more genetically predisposed to lay down greater numbers of fat cells and a small proportion do have genetic mutations that result in obesity, the greatest influence for many still resides with the environment. The original ideas that fat people had lower metabolic rates or had altered glucose levels or metabolism have all been discarded. The proposal that the human genome was somehow unique in allowing obesity to occur has also gone out the window with diet-induced obesity having been demonstrated in a large range of domestic and laboratory animal species.

While Steven O’Rahilly in Cambridge has found humans exhibiting voracious appetites and obesity involving leptin (Farooqi et al 2001) these mutations are extremely rare with only around a dozen individuals recognised in the world so far.

While we consider obesity problems in the developed world as being a serious concern there are small countries where prevalence of obesity and type 2 diabetes is truly appalling. In a number of the island nations in Micronesia the import of western diets and loss of traditional ways of life – including more active methods of obtaining food such as fishing – have produced devastating effects. In Hawaii and Samoa the problems of obesity are worse than in the USA but the island that has really focussed scientific attention is Kosrae where a staggering 50% of the population are obese and most suffer from type 2 diabetes and/or hypertension. Life expectancy, not surprisingly is unacceptably low. If anyone wants evidence of what a cheap imported western food culture can do to people that have not evolved sufficiently to deal with it then this clearly provides it.

What we have learned from these unfortunate islanders is that there is a strong genetic element of failure to cope with our cheap high energy western food. Not everyone on Kosrae becomes obese and it seems that those who don’t are more likely to have some Western genes provided by visitors to the island (Whalers, Pirates etc). Not surprisingly work is under way to try to identify which are the key genes which provide protection against obesity.

It seems that many other cultures find western diets equally difficult to defend against with Asians also appearing to be susceptible to this obesity problem. This has led to the evolutionary concept of “thrifty” genes that are more prevalent in cultures that have a long history of constant struggle against famine.

These genes would predispose individuals to eat as much as possible whenever food became available. The argument goes that most countries in the current developed world have not had to select for these thrifty genes for a considerable period and that their influence has been weakened (although they are clearly still causing us serious problems!).

If this theory is correct, then the fact that the large producers of the least healthy cheap western foods are expanding rapidly in developing countries around the world in the search for new lucrative markets, suggests that these countries are importing an obesity time bomb.

The power of advertising and the celebrity cult

Advertising expenditure on high-calorie drinks and snacks and fast food restaurants in the UK during 2002 amounted to a staggering £450 million

A report prepared by the University of Strathclyde for the Food Standards Agency in September 2003 entitled "Review of research on the effects of food promotion to children"

(www.food.gov.uk/healthiereating/promotion/readreview/) produced the following summary:

"This first UK systematic review of the research literature shows that:

1. There is a lot of food advertising to children.
2. The advertised diet is less healthy than the recommended one.
3. Children enjoy and engage with food promotion.
4. Food promotion is having an effect, particularly on children's preferences, purchase
 1. behaviour and consumption.
5. This effect is independent of other factors and operates at both a brand and category level.

This does not amount to proof of an effect, but in our view does provide sufficient evidence to conclude that an effect exists. The debate should now shift to what action is needed, and specifically to how the power of commercial marketing can be used to bring about improvements in young people's eating".

In summarising the general methods used by advertising companies to promote food for children the report states that:

"Overall, the creative appeals in children's food advertising were found to concentrate on 'fun' and 'taste', rather than on health or nutrition (true both in comparison to other food promotions (aimed at adults) and other promotions aimed at children). The dominance of animation as a creative device was thought to illustrate this tendency. Fast-food advertising, which has become more prominent in recent years, tends not to describe the product advertised and focuses on the experience of the meal and the brand."

In general, studies carried out to date do provide strong evidence that food promotion influences which foods and brands children buy. The main effects reported were for buying foods high in fat, sugar or salt although at least one study has also reported promotional effects on increasing low fat snack sales in relation to vending machines. Bolton (1983) produced a much cited study which concluded that food advertising exposure, as calculated from children's television viewing diaries, was significantly related with children's snacking frequency, calorific intake and nutrient efficiency. Coon et al (2001) found a significant association between the television being on during meals and children's diet. Taras et al (1989) and Gracey et al (1996) found weak evidence of a relationship between television watching and food purchase requests (in the first study) and fat intake (in both studies). The other two studies found significant relationships between television viewing and obesity (Dietz & Gortmaker 1985), and between television viewing/video game playing and high cholesterol (Wong et al 1992).

Parental influence and advertising

Donkin et al's (1992 & 1993) survey of English parents of 7-11 year olds found that the largest category of children's requests for foods seen advertised on television was for cereals (18%), followed by biscuits and cakes (11%), fruit and vegetables (11%), sweets and chocolates (10%), drinks (10%), and meat and meat products (9%). Eleven percent of requests were specifically for Kellogg's cereals. Forty five percent of the requested products had added sugar. In Hitchings et al (1998) research with 9-10 year old children and parents in Newcastle upon Tyne, parents reported granting 96% of children's food requests. Four of the ten

foods which children most frequently asked their parents to buy also appeared in the top ten most frequently recalled food adverts by children. Radkar & Mundlay (2001) found that 'child's demand' for the product was reported by Indian parents as a substantial influence on buying decisions for several categories of food product. One study (Williams 1974) found that North American 9-13 year olds spent 'almost half' their weekly allowance on snacks and that 44% reported buying snacks they saw advertised on television.

Stress and depression

A more controversial suggestion has been that increased problems with stress and depression in the developed world have contributed significantly to our current rise in obesity. Binge eating is a recognised compensatory behaviour used by many individuals to help combat bouts of stress or depression. Stress hormones also directly influence brain systems controlling appetite – the idea is that the initial stress response actually suppresses appetite but that prolonged changes in adrenal hormones (cortisol and adrenaline) which can outlast a period of acute stress by an hour or more actually stimulate appetite and so the net result of stressful episodes is to promote hunger and increase food intake.

Do we want a solution and where can we find an acceptable one?

There can be no doubt that we desperately want a solution. The diet industry around the world is enormous, with Americans estimated to spend around \$40 billion a year trying to lose weight. This is not however necessarily a recognition of the health risks of obesity with so many cultures equating beauty with thinness. For most individuals losing weight is about trying to become both more attractive and not having to deal with the psychological problems of feeling an object of curiosity, humour or disgust.

Dieting

The size of the dieting products and service industry worldwide is testament to the simple conclusion that for the majority of individuals "what comes off soon goes back on again" – often worse than before – and it is easy to become locked in a vicious cycle of "feast" and "famine" which progressively saps our resolve to try again and can damage both the quality of our lives and our bank balance. Despite the large amounts of hype associated with many of these dieting schemes they all do the same thing in the end – significantly reduce your caloric intake. This is even true of the infamous Atkins diet that for a long time was thought to confound this rule. It has subsequently been found to work because with diets high in protein and low in carbohydrates we are more easily satiated and tend to eat less calories.

If you think about it, our dietary and lifestyle habits that led to us becoming overweight took a number of years to cause this change and are difficult to alter overnight. Simply adopting a radical change in dietary habits for a matter of months, without having already adapted to a more suitable routine diet before hand is often a recipe for disaster. Advice given is often to use success from dieting as a platform to adopt a better routine diet afterwards. However, for many it would probably be better to adapt to a more appropriate routine diet first which will start you on the road to gradual weight loss and making it possible to then use a more radical dieting approach to accelerate the process.

As I have already pointed out, the problem for dieters is that if they already have an increased number of fat cells the strength of the signal they send to the brain to eat more after a diet can be very strong and so even the most committed and resolute individuals can face an up-hill battle in keeping the weight off.

Anti-obesity drugs

Despite the huge size of the obesity-treatment market worldwide and the many scientific breakthroughs that have been achieved in the last 10 years to identify new targets for therapeutic intervention there are only a few major drugs available currently. These either try to reduce hunger signals at the level of the brain (mostly making you feel satiated more quickly after a meal) or inhibit the absorption of nutrients so you take in less energy from food. These are not equivalent to the kind of fictional agent shown in the film "The Nutty Professor" where one dose turns a fat person into a thin one with a vastly accelerated metabolic rate and "attitude". They are only likely to achieve modest weight loss (generally 10% or less) in around

half of the individuals who take them.

Orlistat (Xenical) – this inhibits pancreatic lipases and blocks absorption of around one-third of fat consumed. It is approved for long-term use and can cause mild to moderate gastrointestinal upset as its main side effect.

Sibutramine (Meridia, Reductil) – this inhibits noradrenaline, serotonin and dopamine re-uptake in the brain and seems to act by reducing hunger cravings. Average weight loss is 8% and main reported side effects are increased blood pressure, insomnia, constipation and a dry mouth. However, there have been some deaths linked to this drug and it is banned in some European countries.

Metformin – this is often prescribed for type 2 diabetes and also for treatment of polycystic ovarian syndrome. It helps with improving glucose utilisation through enhanced sensitivity to insulin and does seem to promote some weight loss as a result.

There are likely to be a number of new drugs on the market within the next 5 years. Cannabinoid receptor blockers (Rimonabant) are claimed to offer combined therapy for obesity and smoking by reducing the feelings of pleasure associated with eating and smoking. At present there are a number of further attempts to reduce hunger at the level of the brain by targeting melanocortin, ghrelin and neuropeptide Y based systems. We now know that strategies whereby levels of leptin are increased have little beneficial effects in the majority of cases in humans and so Amgen's investment in this seems unlikely to pay off. It seems that most obese people produce large amounts of leptin and the problem is that its receptors seem less able to respond to it. We would therefore probably need drugs which could increase the sensitivity of this receptor.

Some promising drugs which act by dissolving fat cells have also been reported to have beneficial effects in mice but the general experience in the whole field to date has been that where dramatic treatment effects can be found in mice they very often do not seem to work in humans.

Surgery

Weight-loss surgery has had a pretty chequered history and it became clear quite early on that just removing large amounts of abdominal fat from an individual carried high health risks (liposuction for cosmetic purposes does not remove that much fat and concentrates on subcutaneous stores). Bariatric surgery (on the stomach) on the other hand is increasing at an astonishing rate as a successful treatment for the morbidly obese. It carries a significant risk of death (1% during surgery), and some very uncomfortable side effects, but can produce huge and permanent weight loss – a recent study showed patients lost around 70% of their excess weight in 6-12 months and over the next 14 years this only dropped to 50%. Surgery typically promotes weight loss by drastically reducing the size of the stomach so that individuals feel satiated very quickly and will vomit if they overeat. Other procedures alter the ability of the digestive tract to absorb nutrients and this also promotes weight loss. The most common technique used is the Roux-en-Y gastric bypass which creates a small gastric pouch (15-30ml) by placing staples across the proximal stomach and dividing it. In the USA 103,000 of these kinds of operations were carried out last year and this is predicted to increase to 126,000 next year.

Better nutrition: A global strategy for reducing obesity that relies on surgery, drugs or crash diets is clearly untenable. What we need is a strategy whereby we better adapt our routine diet to our chosen lifestyle. The simple fact is that our body is no longer adapted optimally to balance energy intake with energy expenditure in a more sedentary culture where high energy foods are freely available. We need to provide it with some help from our brain!

Better education: At the heart of any national or international strategy for dealing with the problem of obesity must be educating the population about what and how much they should be eating in order to maintain a healthy diet. This undoubtedly requires continued Governmental backing and funding, but well above current levels. While educating the young may be an obvious priority in this respect it is clear that the most effective strategies will need to target both parents and children.

Better food labelling: While packaged foods do generally contain all the information required for an

informed individual to assess their caloric and nutritional values we all know that only a tiny fraction of consumers either have the information or time to interpret them. With food labelling, just like financial information associated with loans and credit cards, the small print hides a number of potential shocks. It would be a tough task to come up with a simple set of standards that would allow the use of some kind of simple visual scaling system (such as a traffic light system – red = high; amber = medium and green = low for example) so that consumers could tell at a glance whether a product had a high caloric value and had high levels of fat, sugar, salt etc. However, this type of approach has to be an essential component to supporting the efforts spent in improving better nutritional education. In general we will never spend lots of time reading the small print on labels. There has to be a simple visual guide that tells us at a glance all we need to know. The use of such a system would also be likely to force the food and advertising industries to focus on producing and promoting healthier foods.

Better information on content of prepared meals: How many fast-food outlets, restaurants, schools, hospitals etc give their consumers details of the caloric and nutritional content of the meals they are providing? The simple answer to this is of course – very few. Providing simple nutritional information on menus should also help consumers to make informed choices and promote wider availability of healthy choice meals. Eating out is hopefully both a pleasant gustatory and social experience but one we should not be paying for by putting on weight needlessly!

Exercise

Which ever way you look at it, maintaining regular levels of exercise is essential for a human body to function normally. We have not evolved to cope with a life of total inactivity and even if we wanted to it would take thousands of years to make the necessary changes to our genome. Taking insufficient exercise is a danger to our health irrespective of whether we are fat or thin.

Various attempts have been launched to try to convince us to take a minimum of 10,000 steps a day or to visit the gym for 30-60min a day for 5 days a week. Most of us find it difficult to restrict our food calorie intake too much and so the only way is to find a reliable route to achieve higher levels of physical exercise. This is easiest if you simply put a little more exercise into your daily work or leisure routine so that you don't end up almost totally sedentary all day. At the very least this means getting off your bums and walking around more. The idea of wearing a pedometer and setting a daily target of 10,000 steps a day is a good one since at least it shows you how little exercise you may be doing and gives you a target to aim for. There are lots of ways you can introduce greater levels of physical activity into your every day life. Apparently just abandoning use of your television remote and getting up and changing the channels yourself can end up with you burning thousands of extra calories a year. The table below gives some examples of how many extra calories can be burned by more active forms of the same tasks:

Energy expenditure and activity - sedentary behaviour

Kcalories

Using TV remote control

<1

Getting up to change TV channel

3

Sitting, talking on the phone, 30 min

4

Letting dog out of the back door

2

Walking the dog, 30 minutes

125

Using pre-cut vegetables

0

Washing, cutting vegetables, 15 min

12

Using auto car wash

18

Washing and waxing car, 1 hour

300

Using a lift, 3 floors

<1

Walking up 3 floors

15

Sending email to colleague, 4 min

2

Walking and talking to colleague, 4 min

6

Shopping on-line, 1 hour

30

Shopping, pushing trolley, 1 hour

200

Source: Mayo Clinic Proceedings (77) 2002

Burning calories during different activities

The following chart shows approximate calorie expenditure for a range of activities:

Activity

Kcalories used in 20 minutes of activity

Aerobic dancing - low intensity, equivalent to walking

80

Aerobic dancing - medium intensity, equivalent to jogging

130

Aerobic dancing - high intensity, equivalent to running

170

Bed making

100

Cleaning windows

60

Cleaning stairs

65

Climbing stairs (72 steps per minute)

95

Climbing stairs (92 steps per minute)

130

Cycling on flat ground ('own speed')

125

Dancing (waltz)

130

Dusting

70

Gardening

110

Golf

100

Knitting

25

Office work (general)

25

Operating electric sewing machine

25

Playing cricket

160

Playing pool

65

Playing squash

200

Playing tennis

140

Playing football

140

Playing table tennis

90

Playing cards

40

Running (speed unspecified)

190

Sitting typing

30

Walking on the level (1-2 km per hour)

45

Walking on the level (4-5 km per hour)

85

Watching football

40

Source: Human Energy Requirements: A manual for planners and nutritionists, by WPT James and EC Schofield, published by the Oxford University Press (1990).

Going to the gym and playing sport can also be a positive social experience rather than simply a necessary grind!

Global solution: reduce childhood obesity?

A sad fact of life is that if both parents are obese there is a 70% likelihood that their children will be too and this has more to do with food and exercise practices in the family than genes. Another sad fact is that if you are obese as a child you are likely to have weight control problems for the whole of your life. A final important fact is that successful treatment of obesity in children is much easier to achieve when the whole family is involved.

If we are really going to solve the obesity problem we therefore need to target children in particular so that the next generation of parents can use their positive influences on dietary and exercise habits of their children to help reduce the incidence of obesity to more acceptable levels.

Actions:

- Better nutrition education and information for children and parents
- Better education for children and parents on how to prepare meals from basic ingredients
- Routine measurements of children's fat levels (BMI etc)
- Incentives for food industry to produce more healthy options
- Incentives for consumers to purchase healthy options
- Tighter regulation on food advertising
- Better quality children's meals in restaurants
- Better quality school food
- School vending machines only to provide healthy snacks/drinks
- More relevant outlets for children's exercise
- More encouragement from TV programmes and celebrities to exercise
- Increase numbers of physical activity periods in school curriculum

Some final "take-away" messages

- We eat too much fat, salt and processed sugar
- Try to eat a greater proportion of monounsaturated fats
- Extra fat cells can be for life not just for Christmas!
- Fat cells are hormone factories that, in excess, can damage health
- Obesity linked with heart disease, type 2 diabetes, cancer, osteoarthritis, sleep apnea,

psychological problems

- Obesity is caused by taking in more calories than we use
- An extra 50 kcal a day will add a kilo that stays!
- Reduced exercise/fitness more responsible than increased calorie intake
- No miracle diets or treatments are available
- Yo-yo dieting can impair your immune system
- Current drugs only achieve 5-10% weight loss and have side effects
- Surgery can be very effective but is a drastic step
- Only effective National strategy requires Government intervention
- Food industry needs encouragement to produce more healthy options
- We need better simple information on nutritional content of food
- Food advertising needs better regulation
- We need to get up and exercise more
- The key battleground is dealing with childhood obesity

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