**OBESITY**

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## Part 1

Thank you very much, Joan. (...) Ok. I'm going to tell you what the right answer is while we get the graph going. How many people answered "A"? Anybody answered "B"? Anybody answered "C"? Well done, very good, "C" is the right answer. And the reason that "C" is the right answer is that the way that we classify obesity is in terms of the body mass index (BMI). Now, the body max index is you weight in kilograms divided by your height squared, measured in metres. So, if you have a BMI of less than 18.5, then according to the World Health Organisation classification you are underweight, or a popular description is thin. A healthy, normal or acceptable weight, BMI, is 18.5 to 24.9. If you have a BMI of 25 to 29.9 that's classified as grade 1 overweight. If you have a BMI of 30 to 39.9, this is defined as obese. So, the definition of obesity is if you have a BMI of more than 30 kilograms per metre squared. And if you have a BMI of more than 40 this is described as morbidly obese, and what that means is obesity which is really likely to be associated with some sort of disease or illness.

Now, why are we concerned about the prevalence of obesity and obesity in our populations. Well, put very simply, being obese makes your risk of death, risk of mortality from all causes, very significantly higher. In fact, the relationship of body mass index, or BMI, to mortality is described as a "J"-shaped curve, so this curve is shaped like a "J". Along the X axis you have units of body mass index, with five unit increments from 20 to 40. And on the Y axis is mortality ratio; that just means risk of death and what you'll see, if you have a BMI between 20 and 25, within this range there's very little effect on your risk of death. So if you have a BMI within that healthy normal, acceptable range, there's no increased risk of death if you put on a little weight or you put off a little. Below 20, there is a slight kink up, and that gives you the end of the "J". Because being significantly underweight seems to also be associated with increased risk of death. If you look at what happens between a BMI of 25 and 30, you can see that the risk of death increases. But the risk of death increases very sharply from 30 to 35 and then even more sharply from 35 to 40. And this "J"-shaped curve signifies the fact that with each 5-unit increase in BMI, the increase in risk of death is ever so much steeper. So, the more weight, excess weight that you put on, the more your risk of death increases.

Now, it's interesting to see, that definitions of obesity, actually differ for different ethnic groups. And they've always been standardised according to European people, but actually their cut-off levels for BMI are different for different populations. So, as I said, BMI is calculated as your weight in kilograms over your height in meters squared, and for the European and Afro-Caribbean populations, the classifications of overweight is 25 to 29.9; and the definition of obesity is 30. But the cut-offs for South Asians seem to be different, in fact, they are lower. A South-Asian person is classified as being overweight when their BMI is only 23 to 24.9; and they are classified as obese when their BMI is more than 25, not more than 30. So the cut-offs are lower for South-Asians, and that is because, for South-Asians, a higher BMI is associated with an even greater risk of mortality, than for Europeans and Afro-Caribbeans. That means that is more dangerous for a person who is of south Asian origin to put on excess weight, than it is for a European. We don't exactly know the reasons for that, partly genetic, probably, but also perhaps the fact that South-

Asians tend to have a smaller, lighter build, naturally, means that they are less able - their bodies are less able to cope with excess weight if they acquire excess weight. So the cut-off seems to be different for South Asians and Europeans and Afro-Caribbeans. Putting on excess weight is a higher risk for a South Asian person.

**Part 2**

The other interesting thing to see is that there are different patterns of obesity. Obesity isn't just simple. You tend to find that people are either *apple-shape* or a *pear shape*. So an apple-shape is where you carry a lot of excess weight around your stomach, around the middle, and a pear shape is where the excess weight is on the hips and thighs. And on the whole you tend to find that women tend to be pear-shaped; and men, when they're overweight, tend to be apple-shaped. But actually is also possible for women to develop apple shape as well; less likely for men to develop a pear shape. Now, this is important, because it's been shown that the apple shape is more dangerous. There's higher risk of cardiovascular disease and death of all causes associated with the apple shape, than the pear shape. And we sometimes call this "Central Obesity" because the excess weight is central, and it seems to be a higher-risk associated with central obesity in particular. And because of that, people have suggested that in addition to BMI, we should also measure waist circumference, the circumference around the waist. And there are also cut-offs of waist circumference above which your risk of cardiovascular disease and other diseases is higher. And those cut-offs are 88 centimetres for women and 102 centimetres for men. So it is suggested that this should be measured along with BMI because a BMI might be high, but in some ways, you might not be at quite such a high-risk if you're a pear shaped than apple shaped. If you have a high BMI and you're apple-shaped, then that is a higher risk.

So, I've indicated that obesity is associated with a higher risk of death, of many causes, and that is because it is associated with a number of health complications; the top is the non-insulin dependent form of diabetes, we sometimes called this "Type II" diabetes. It's the type of diabetes that you get in later life. It tends to appear after the age of about 40, and is very strongly associated with being overweight, whereas "Type I" diabetes is not usually associated with being overweight, and usually occurs in childhood.

So, this is an adult form of diabetes, very strongly associated with obesity. Obesity is also associated with heart-disease, with a number of cancers, with osteoarthritis, because of the excess weight that put pressure on your bones, with gallstones, with a condition called sleep apnoea; this is where excess weight around the neck and also the diaphragm makes it hard to breathe during the night when you're asleep and lying flat. And for some people this is a problem because they can actually stop breathing during the night for 10 seconds, sometimes more. And that causes them to wake up. And obviously it's very distressing and can be dangerous if they can't start breathing again.

It is very strongly associated with reproductive disorders, because a lot of hormones are activated by fat tissues, so if you have more fat then you can get an imbalance in the levels of hormones and that can lead to reproductive disorders. And it's also associated with pathological and psychological disorders and social penalties, and what I mean by that, is that there is evidence that people who are very significantly overweight, find it harder to get a job, and tend to have lower socio-economic status and tend to have more psychological problems. So a number of complications that are associated with obesity.

And this is just to highlight the proportion of some of the major diseases that we suffer in our populations and how much of those diseases are attributable to obesity. This is actually data from the USA. And this data suggests than more that 60% of Type II diabetes

is due to obesity as the primary cause. Similar story with some cancers, with osteoarthritis, about 17% of heart disease is due to excess weight and elevated BMI, due to obesity. So a lot of the disease that we see in our countries, in our populations is due to obesity. And of course you could argue that obesity can be treated, it's in some ways controllable. And so, we would expect that the prevalence of these diseases should decrease if we're able to tackle the problems of obesity that we have today.

**Part 3**

This shows you in a bit more detail the link between obesity and cardiovascular disease, which is a major cause of death in many countries. And these data are shown in terms of what we call the relative risk, so relative risk is where you assign a risk of 1 to what you want to use as your standard, and then everything else is relative to that. So if you have a relative risk of more than 1 it means higher risk; and if you have a relative risk of less than 1 it means a lower risk. So on the bottom here you have categories of BMI going from less than 21, of course that's healthy, up to more than 29, which is obese. And what you see is that as you increase BMI, the relative risk of cardiovascular disease increases. So what this means is that if you have a BMI between 25 and 28.9, you have doubled the risk of developing cardiovascular disease compared with if you had a BMI of less that 21. And similarly if you have a BMI of more than 29, you have three and a half times the risk of developing cardiovascular disease compared to if you have a BMI of less than 21. So, your risk of developing cardiovascular disease doubles, triples when you get - go from being healthy to being obese.

So this is the next question, and I hope this time the results are going to appear. I'd like you to guess what proportion of UK adults are obese at the moment. So your choices are one in three, one in four, one in ten, and one in 20. And your time - oh it's not working this time, let's see if this works (silence). I'm going to have to have a show of hands again, I'm really sorry. So, how many people think that the proportion of UK adults that are obese is 1 in 3. Anybody? Well, we have a few hands. One in 4 -quite a few more hands. 1 in 10 – a lot more, ok. It's about the same number. One in 20. OK, so you're being pessimistic. The right answer is one in 4. So, well done if you got that right!

And this next slide will show you how the prevalence of obesity breaks down. So, this is data just from the UK. It is data that was collected by our National Audit Office and it shows you the prevalence of obesity, that is people who have a BMI of more than 30, percentage, in percentages in different age groups along the bottom. So you can see several things from this data. There are three important things that this graph shows. The first one is that the prevalence of obesity, the proportion of people that are obese increases with increasing age, up to the age of 55 to 64. So with increasing age there's an increase in the prevalence of obesity. The second thing to know is that after the age of 64 this decreases. And there are probably two main reasons for that. The first is that those people who are obese have a shorter life expectancy so they would die sooner. So, unfortunately that means that those people will not be there after the age of 64; or there will be less of those people there. So there's a natural decline. The other important point, is that after the age of 64, you see an increasing number of the types of diseases like cancer which cause weight loss, body wasting, and so there will be a decrease in body weight after that age; in addition to that older people tend to eat less, appetite is reduced, either because of psychological reasons or because of physical changes in their body - they loose their teeth, it makes things harder and so on. So, there is a decrease in BMI after the age of 64.

The other thing that's interesting is to compare the prevalence of obesity in men versus women, because you'll see that up to about the age of 54, the prevalence is about the

same in both sexes. But after the age of 55 the prevalence of obesity tends to be higher in women and that stays right up to 75+. And we think that is partly because women tend to live longer than men, that's one thing; but also that there might be some hormonal protection, some hormonal effects which means that women tend to have higher BMIs than men. Actually women naturally have a higher proportion of fat in their bodies than men anyway, and that seems to be prolonged throughout life.

**Part 4**

And this is also interesting data because, of course, in the UK we have a lot of populations who have emigrated here from other places. And this is data to show the prevalence of obesity in males and females in different ethnic groups. And this is also shown as a proportion. So this is a percentage of individuals classified as obese, men and women. This is in the general population of England, and this is in black Caribbean, Indian, Pakistani, Bangladeshi, Chinese populations who are living in the UK. And what we should do, really, is compare the prevalence of obesity of those ethnic minorities with the general population. It highlights some interesting data, because if you look at men, in the red, you see that on the whole, the prevalence of obesity in those ethnic minorities for men is either the same as the general population, or lower. In terms, especially for the Bangladeshi and the Chinese communities, the prevalence of obesity is particularly low in the men in those communities.

For women it is a slightly different story, because although some of these ethnic minorities have a similar or lower prevalence of obesity compared to the general population, for black Caribbean women and Pakistani women, the prevalence of obesity is quite significantly higher than the general population. So there's a special issue with prevalence of obesity in those two ethnic groups and perhaps that's something that people need to tackle especially. We don't know all of the reasons for that but it's something that seems to be highlighted in this data and the problem seems to be getting worse.

What about internationally? Well, we have a chart here which shows the, a number of different countries going from the lowest prevalence of obesity to the highest and you can see here, this isn't all countries in the world, it's just a selection, but China, is right at the top in terms of the lowest prevalence of obesity. So, do we have any people, any students from China here? Ok, you can be very pleased, so you're at the bottom of this table. Here is England, here is the US, here we have Saudi Arabia – I know we have a lot of students from Saudi Arabia here this year – and at the top here is Western Samoa. And one of the things that you'll notice in both the Saudi Arabian and the Western Samoan data is how much higher the prevalence of obesity is in women, compared with men. So we have that in the UK in older age groups, but in some countries that difference between men and women is even greater than in the UK.

And how has this changed over the time? Because obesity seems to be talked about an awful lot at the moment and people are always saying that "the rates are increasing", but is that really true? Well, I'm afraid it is. Because if you look at data going back to 1986, and compare that with current data, is quite clear that in both men and women, the rates of obesity, the prevalence of obesity has tripled. It has tripled in only 25 years, and that is quite a short period of time for such a rapid increase in obesity.

This has highlighted the fact that because obesity is associated with a number of different diseases, the way we think about the risk of disease has really changed, even

since 1984. In 1984, cardiovascular disease was still a great problem, but in 1984 the three things that we were most concerned about in terms of increasing our risk for cardiovascular disease was cholesterol, that means your blood cholesterol, smoking, and hypertension, which is high blood pressure. They were the three major risk factors for heart disease at that time. In 2006, the two major risk factors for heart disease are central obesity and diabetes. Now, that is not to say that cholesterol, smoking and high-blood pressure are not important anymore, of course they are. What it means is that central obesity and diabetes have overtaken those other risk factors as being the major cause of cardiovascular disease in our populations.

And this obviously has led to a great burden on our health service, because when people become overweight, and when they start to develop these types of diseases, they need a health service, and health service costs money, it costs money to the tax-payers. Some people have argued that people who become obese, through whatever reason, should not have free treatment for operations which are a direct result of their obesity. For example, operations on their hips and knees, which might have been, whose damage might have been caused by excess weight. So in some Primary Care Trusts, in some hospitals in the UK, there have, in the past, been rulings that patients with a BMI of over 30 have to pay for their operations, they would not get them free. This is not something that is a general rule, and actually it is very controversial as you can imagine, but I think it's an important topic for debate and an important issue to address in the future, because some people feel very strongly that tax-payers shouldn't have to pay for those sorts of operations.

**Part 5**

So what I want to do now, is to try to look as some of the causes of obesity in our populations, to try to identify key things that we can suggest, could address these issues. So I'm going to touch on a number of factors to do with the cause of obesity, we sometimes call this the 'eatiology', this is the causes of obesity. I'm going to mention genetics, I'm going to talk about our metabolism, our macronutrients balance – that is the type of food that we eat, dietary factors, I'm going to hint at appetite control, food choice and eating patterns are important, psychological factors are very important but we don't understand them very much, and I'm going to talk about physical inactivity at the end.

Now, any explanation of obesity has to rely on something that we call the energy balance equation. Energy balance is energy intake minus energy expenditure, and that results in a change in body stores. Now, this is an important equation, so I want to explain this in a bit more detail. Energy intake is the food that you eat, because the food that you eat provides you with energy. So that's on the left hand side of this equation. Energy expenditure is the energy that you burn off. So, if you eat more than you burn off, you put on weight and if you do the opposite, if you eat less than you burn off, you will lose weight. That's the energy balance equation. It's a fairly simple concept, but is an important one for obesity, because any explanation of obesity has to rely on this energy balance equation. So when we're looking at causes of obesity in a population, we have to look at whether people are eating too much or not doing enough exercise, very simple. But the reasons why people don't eat – the reasons why people eat too much or don't do enough exercise can, sometimes, be quite complex. And that's what we need to find out.

So the first thing is to look at genes. And there's a lot of debate about whether obesity is due to genes or lifestyle. Now, I'm not going to use this system, I think I'm just going to rely on a show of hands. So I'd like to ask you before we start, what do you think, about whether obesity is a result of genes or lifestyles? Your choices are a) it's mostly due to

our genes, b) it's nothing to do with our genes, it's all about lifestyle, c) it is mainly about lifestyle but there may be some influence of genes and d) it is mainly about genes but lifestyle might affect it a bit. [Most students say c)]. You're very good. How many people would agree with c)? Well, you know the answer now. Ok, the answer is c). It's mainly about lifestyle but there may be some influence of genes. Now, I'm quite impressed about that, because some people would tend to think that genes don't have anything to do with it. So you've obviously heard something that genes, about genes being involved, so let me try to explain how genes are involved in risk of obesity.

So, when we're looking at obesity we have to consider both genetic and environmental factors and in terms of genetic factors, you can have one of two situations. Either you can have a situation where a condition is "monogenic", that means that you have a mutation in one gene, and a mutation in one gene is enough to cause a disease. In this case, we're saying obesity. Now, in reality there are, this is very, very rare for obesity to be caused by mutation in one gene. Very rare indeed. So, in a population, monogenic syndromes are not the cause of obesity, they are in a few very, very isolated cases. I'll give you an example a bit later. But that's not the general explanation of obesity.

The other situation is that obesity could be a polygenic disorder. In other words, it is determined by a large number of genes, not just one gene. And we call that large number of genes, susceptibility genes. And the idea is that a large number of genes can increase your risk for obesity, but they don't necessarily cause it. We sometimes say that obesity is the result of an adverse environment on a susceptible genotype. In other words, you might have some susceptibility genes, but they only become active, they only come into operation when you put yourself in an adverse environment. And we might think of an adverse environment as just having too much food on offer, being thrown at you; or an environment where you're not given the opportunity to do enough exercise.

So, that's an important point, that obesity is the result of an adverse environment working on a susceptible genotype. And I think the environment part of it is very important because food intake, lack of exercise, cultural factors, have a huge impact on your original genetic make up. And it's very important to remember that the prevalence of obesity in the UK has tripled in just 20 to 30 years, but our genes haven't changed in that time. So genes can only account for so much of the risk, the rest is about environment.

**Part 6**

This is another way of looking at the interaction between genes and environment. So, everybody here has a complement of genes, some of which make you more susceptible to obesity, and some of which actually protect you. So they're not all bad, some of your genes will be protective. Different people will have a different balance of protective versus susceptible. So some people will have a greater proportion of protective genes, a smaller number of susceptible genes, others will be at higher risk because their genes are not so great. But then they can choose to live a higher-risk lifestyle, or a healthy lifestyle. So, if you were to have a lot of protective genes and you live a very healthy lifestyle you're doing exactly the right thing, because you will swing this pendulum towards health.

If, on the other hand, you're born with a lot of susceptible genes and you have a high-risk lifestyle, you will push this pendulum towards disease. But of course, most people have a combination of both, so you might have quite a large complement of protective genes, but live a high-risk lifestyle and they where you end up depends on the balance between the two.

So, the problem with this model is that we still don't understand very much about these genes at all. We've identified a few genes, which are linked with obesity, but we don't understand them very well. And that's why you see newspaper articles where people claim that an obesity gene has been discovered. But they always follow almost the same line, and this is what they usually say. They say: that "researchers have discovered a commonly occurring gene that may explain why some people become overweight while others do not. However, they point out that it is unlikely to be the cause of the global obesity epidemic". You always see that sentence at the end of the article. And that is because that one gene can't account for obesity by itself.

The way that these researchers will have done their studies, is they would take a very large population. They would take blood samples from all of those people and find out whether they've got the good or the bad form of that gene. They will then measure the BMI of those people and will see whether the BMI of the people with the bad gene is higher on average than the BMI of the people with the good gene. So what they're doing is they are associating BMI, with the presence or absence, of a good or bad gene. But that doesn't show you cause, does it. It's just an association; it's just showing that the people carrying that gene tend to have a higher BMI. So it cannot possibly show you a cause. And this is the point of the susceptibility genes, that every time somebody discovers one, it's interesting, but it doesn't tell you that that gene, how important that gene is, in terms of obesity in general.

Now, the most excitement occurred when the gene for leptin was discovered. And I'm going to focus on this as a special example of the gene that is associated with obesity. It was discovered in 1994-1995 and it was discovered in a mutant strain of mice, they are called the obese mice or the "ob-ob" mice. These mice had a mutation in the leptin gene, and they were very obese, they had type II diabetes, they had *hypofaygia* which means they couldn't stop eating. And they had very reduced physical activity, which is probably not surprising when you see how fat they were. Those mice could not produce any leptin because they had a mutation in their leptin gene, so they had no leptin at all and they became huge and could not stop eating. And you can see why that caused so much excitement because leptin is made by your body fat; it's made in human beings as well as mice and is made in response to eating. It's a hormone and it controls food intake because it makes you feel full. It circulates in your blood and it signals to the brain, so it's a hormone that is produced when you eat food, it enters the blood and it tells your brain that you are full and so you can stop eating.

Now, when those mutant mice who couldn't make any leptin themselves were injected with leptin, they stopped eating within thirty minutes and they didn't eat again for six and a half hours. So, leptin, in those mice was really important. And it is interesting to see that the name leptin comes from the Greek word leptose, which means thin.

**Part 7**

So, this is a simple diagram to show how leptin works. You eat food; this is your gut, so you absorb nutrients, your body fat takes up those nutrients, it absorbs them, and then it makes leptin, because you've eaten food. The leptin enters the blood stream and then it signals to your brain that you've eaten plenty of food, and your brain, normally when it's hungry, produces another hormone called "neuropeptide y". But leptin stops your brain from making neuropeptide y. So neuropeptide 'y' is the hunger signal that tells you to eat. When leptin appears it stops the production of neuropeptide y so you stop eating. So it's quite a simple scheme.

In the mutant mice, so the ob-ob mice - what happens is that they eat food, they absorb the nutrients from the food but they can't make leptin. Their body fat can make no leptin at all, their brain doesn't receive any signal from leptin, it carries on making this hunger hormone, neuropeptide y, so the mice carry on eating. There's nothing that can make them stop eating, because they do not have any leptin.

This is the same mutant mice, so again, they're eating food, they're absorbing nutrients, they can't make any leptin themselves, but then if you inject leptin into their blood, that leptin that you injected signals to their brain to stop eating. So their brain then does stop making neuropeptide y and they stop eating. So that's why injecting leptin into these mice stops them eating.

So, what about humans? Well, as you can imagine there was huge excitement, because if leptin is so important to stop food intake and prevent obesity in the mice, then maybe it was just as important in humans. But, there are very few cases of leptin deficiency that have been found in humans. There are in the UK, there are one or two families, where it's been shown that children born in those families have a leptin mutation just like in the mice. And very similar to the mice, those children from when they were babies, just could not stop eating, were hungry all the time and got very, very big, very, very fast. And those children actually being treated by injections of leptin, just like the mice. But that condition is very, very, very rare. There are only one or two families in the UK and I don't know about the rest of the world.

So, it's a very rare condition and in fact what people had found is that obese humans actually have higher levels of leptin than non-obese. So, it's the opposite of what you might think. In addition to that, the circulating concentration of leptin is positively associated with body weight, BMI and body fat. So what that means is the, the greater your body weight, the greater your BMI, the more body fat you have, actually the more leptin you have, that doesn't make sense because if you have more leptin you should stop eating more easily. So the situation in obese humans is obviously different and there's a new theory which tries to explain this. The new theory suggests that in an obese person, they eat food, they absorb nutrients, their fat tissue makes plenty of leptin but for some reason their brain is resistant to leptin. So the idea is that obese people are leptin resistant. It's not like the situation in the mutant mice, it's just that for some reason the brain cannot respond to leptin in the same way. So as a result, it carries on making neuropeptide y, the hunger signal, and the person carries on eating. It hasn't been absolutely proven but is the most popular theory at the moment.

Ok, so this is just to test you on your understanding of leptin. One of these statements is incorrect, and I want you to decide which one is incorrect. a) leptin makes you feel full, b) obese people might be leptin resistant, c) obese people are leptin deficient, d) obese people have higher leptin levels than lean people. So which one is wrong? (Students shout answers) Ok, how many people think a) is wrong? How many people think b) is wrong? How many people think c) is wrong? And how many people think d) is wrong? Ok, not everybody is answering but the correct answer is c). 'C' is wrong because obese people are not leptin deficient, they are leptin resistant, so they can make leptin, in fact, they have higher levels of leptin than non-obese people, but their brains don't respond to the leptin.

**Part 8**

Ok, I'm going to move on now to the last part of my lecture which is to talk about food, and our food intake. Now, you're all quite young I can see, so you probably don't appreciate quite how much our food intake might have changed over the last 20 or 30

years. But if you're more close to my sort of age, you might appreciate the fact that portion sizes seem to have increased over time. And this is just an example of a typical portion size in 1954, so a Burger King, 2.8 ounces, 202 calories, compared with 2004, 4.3 ounces, 310 calories. And you'll see similarly portion sizes of other commonly consumed food products. These are all bad examples that seemed to have increased. So, it's important to ask whether our energy intake has changed over the last 30 years, because if it has then that is one part of the energy balance equation. So, I'd like to tell me what you think. a) Do you think that energy intake is more than before? b) Do you think that energy intake is less than before? c) Or do you think that energy intake is the same? How many people think a)? Ok. How many people think b)? How many people think c)? Ok. I can tell you that the answer is actually B. Surprised!

Here is some evidence that the answer is B. Our energy intake appears to be lower than before. This is information that we have going from 1950 up to 1990. We actually have some more up-to-date data as well, but it is showing the same trend. And you can see on this graph. This graph shows total energy intake and it's broken down into different types of food. Between 1950 and 1970 energy intake was increasing. From 1970 to 1990 it's actually decreased. You can see that the type of food that we're eating now is quite different from 1970. In 1970 quite a large proportion of our energy intake was household food; that is food that you cook at home. It's much smaller now, and we have a greater proportion now of alcohol, confectionary, soft drinks, than we used to. But it's undeniable from this data that energy intake seems to have decreased. There are some arguments about the accuracy of data because as people have eaten less food in the home, over time, they now eat more food outside the home. And some of these data doesn't always take into account the food that you eat outside the home, so there are some people who think that this is a slight underestimate. But even still, our energy intake is not significantly higher than it was in 1970. So, if we're eating less, why are we getting fatter? a) Is it because our energy expenditure has decreased? b)Is it because our metabolism has changed? c) Is it because our genes have mutated? d) Or do you have absolutely no idea? So how many people think a)? How many people think b)? How many people think c)? Good. How many people think d)? Ok, some people have no idea, that's fine. The answer is that our energy expenditure has decreased and I'll show you some data about energy expenditure.

So you'll remember that energy expenditure is the other part of the energy balance equation. This is the energy balance equation, but it's drawn in a slightly different way, so here you have energy intake, and these are all the components of energy expenditure and a change or an imbalance in that equation leads to energy storage. Now, your energy expenditure is composed of three different types. About 65% of your energy expenditure is called maintenance or base or metabolic rate. That's the energy that you need just to stay alive, to breathe, to make sure that blood pumps around your body for your heart to beat, that sort of thing, so very, very basic level just to keep you alive. About 10% is to make sure that your body temperature stays at 37°C degrees centigrade. And then the other 25% or so is what we called physical activity and that's the activity you do. Of course this varies very much for different people, so for some people this green part is really, really small. And for others it's much bigger. For a long time, people thought that those people who became obese might have a much lower basal metabolic rate, a lower metabolism than people who didn't become obese, but that's now been proven to be wrong. And what that means, is that the green section, your physical activity, the extra activity that you do is the most important component of energy expenditure.

Let me show you proof that people who are obese don't have a lower metabolism than people who are not obese. This shows you energy expenditure over the course of a whole

day, you've got a whole 24-hour period, sorry this is 12 hours. And this is an obese person compared with a lean person. And you can see that the energy expenditure in the obese person in the dark line is higher the whole day compared with the lean person, even when they're asleep. So even the basal metabolic rate which is when they're asleep, is higher in obese person than a lean person. And that's because in an obese person they are carrying more weight so it requires more energy to do everything, even breathing, than a lean person. And more evidence for that is here if you compare a lean, with an obese and a super obese person, (so this is ideal body weight plus 50%, ideal body weight plus 100%). So this is somebody who is twice the ideal body weight. You can see that their 24-hour energy expenditure over a 24-hour period is increased when you go from lean to obese to super obese. So somebody who is obese actually has a higher metabolism, than somebody who is lean because they need more to expend more energy to do the same thing.

**Part 9**

So, what that means is that we really have to look at physical inactivity and this is a chart showing the degree of physical inactivity in men and women in the UK where the proportion of individuals that are classified as sedentary is almost 30%. Sedentary means that these people are doing less than 30 minutes of moderate activity on five days a week. So it's quite a small amount of activity. And we know that levels of physical activity have decreased. This is actually indirect data. So on the left hand side - this is data that I have already shown you- showing that energy intake increased between 1950 and 1970 but actually decreased between 1970 and 1990, and that goes against the sharp increase in the prevalence of obesity that we've seen over the same period. On the other hand, in this graph you see the number of cars per household and the amount of television viewing in hours per week over that same period. And you can see that is increased. These are indirect measurements of inactivity. If we're watching more TV, we've got more cars, we're obviously doing less in terms of walking and activity. And that coincides with the very sharp increase in rates of obesity. So physical activity is a very important component of the energy-balance equation, and it must play a big part in prevalence of obesity in the UK.

So, I want to finish by reminding you again of the energy balance equation. And I've shown you that energy intake, minus energy expenditure leads to a change in body energy stores. Our data seems to suggest that we're actually eating less energy than we used to, but our energy expenditure has decreased, but on an individual level, it's a question of balance between these two and obesity is a result of an imbalance. On an individual basis if you put on weight, you're eating too much compared with your expenditure, so you either have to exercise more or you have to eat less.

So I hope this has encouraged you to take up that basketball session at lunchtime, or whenever it was, and I'm going to leave you with one last slide which I think is quite ironic. This is in the US, it's a 24-hour fitness studio but everybody going in is using the escalator. Thank you very much for your attention.