



## What Can We Do About Rising Obesity? Professor Christopher Whitty

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This lecture considers the major public health challenge of obesity. It has increased and is increasing. Should we worry? And if so, what should we do about it? This lecture covers the epidemiology of obesity, health impacts, interventions at an individual level (where things are improving), and potential interventions at a societal level. These are notes of key themes raised rather than a transcript of the lecture. Central to the arguments in this lecture are that there is no reason for someone to have any feeling of shame about obesity. It is common and occurs for biological and societal reasons. What will be covered in the lecture is the medical consequences of this, and how we tackle them.

Here in the UK, over a two-decade period, obesity, defined as Body Mass Index (BMI) over 30 rose from 15% to 26%. The rate of increase has become more stable over the last few years but there is no sign of a decline and this is now feeding through to health impacts. In 2019, the last year for which there are currently full data, over 11,000 NHS hospital admissions were directly attributable to obesity and for over 800,000 obesity was a factor in the admission. Throughout this lecture we will use two definitions; obesity with BMI >30 and overweight BMI >25 kg/m<sup>2</sup>. These correlate with poorer health outcomes.

As well as adults there's been a steady increase in obesity in children, and in particular older children. 20% of year six children were classified as having obesity. There is a very substantial and widening gap between children who live in the least deprived and most deprived areas. Obesity has been steadily increasing in areas of relative deprivation but not in areas of affluence. In the UK the map of less affluent areas is very similar to the distribution of childhood obesity by year six. This is true when we consider a national picture, or an individual town like Blackpool. Areas with childhood obesity correlate very strongly with areas of lower life expectancy in adulthood. Obesity does not stop in childhood and tends to increase to late middle age.

Obesity has been rising rapidly across the globe. This is not just a problem for high-income countries although they are generally very affected. It is also very common in Latin America, North Africa and the Middle East and increasingly in many middle-income countries such as South Africa. Although this lecture considers calorific over nutrition leading to obesity, it is important to acknowledge that calorific undernutrition is still with us and has very severe health outcomes as well. Historically it was the consequence of poverty everywhere; many countries now have a double problem of over nutrition and undernutrition simultaneously. Even in high income countries many people who have obesity can also have malnutrition with unbalanced diets missing key elements.

Obesity causes health problems through multiple mechanisms. The most obvious are simple functions of having greater weight, for example the mechanical effects on joints. The health effects go far further than that however and that is partly because the adipose tissue where fat is stored, is highly biologically active and has many functions. In consequence having a lot of adipose tissue will lead to major physiological changes. This includes insulin resistance which affects glucose and energy storage and several other hormones including leptin and the important sex hormone

oestrogen. It is also associated with inflammation through several mechanisms, clotting and lipid metabolism. All have consequences for health.

*Osteoarthritis*, which leads to erosion of the joints, is strongly associated with obesity. The knee joints are frequently affected because of load bearing. Every five unit increase in BMI is associated with 35% increased risk of knee osteoarthritis and an 11% increase in hip osteoarthritis. The majority of knee replacements in many high-income countries are obesity related. There is also an increase in osteoarthritis on joints which are not load-bearing suggesting additional mechanisms.

*Diabetes*, specifically Type 2 diabetes is very strongly associated with obesity. Prevalence of Type 2 diabetes rose very rapidly from the 1960s as obesity increased. Type 2 diabetes has many consequences if not fully controlled, both direct due to raised blood glucose and indirect including heart disease, stroke, kidney disease and eye disease. 80 to 85% of Type 2 diabetes in the UK is accounted for by those with overweight or who have obesity. Where people with Type 2 diabetes lose weight diabetes may go away. The strong correlation of Type 2 diabetes and obesity is visible globally.

*Coronary heart disease* and *stroke* are both significantly increased by obesity. This is particularly true for people where fat is distributed centrally and raised waist size is as important as BMI, especially for some ethnic groups such as people of South Asian heritage. Obesity is associated with raised cholesterol, raised blood pressure, diabetes. The strongest association in stroke is between obesity and younger strokes in people under 65 years old.

There is a clear association between some *cancers* and BMI. Raise BMIs clearly associated with endometrial (uterine) and postmenopausal breast cancer in women is probably due to the effects of adipose tissue on oestrogen. Oesophageal adenocarcinoma and renal (kidney) cancers are also associated with high BMI.

Non-alcoholic fatty *liver disease* which is in large part associated with obesity is potentially a very dangerous cause of cirrhosis and liver cancer. It is the second most common reason for liver transplants after alcohol in the UK.

Obesity is associated with *poor pregnancy outcomes*. The overall chance of miscarriage under 12 weeks is one in five (20%) but in those with a BMI over 30 the chance is one in four (25%). There are also increase risks of gestational diabetes, blood clots, high blood pressure and pre-eclampsia, and difficulties during delivery.

Some *infections* are more common or more severe in people with obesity. This has been very clear in COVID-19. The higher the BMI the greater the chance of hospitalisations, admission to ICU and dying from COVID-19.

In addition to these specific risk factors for multiple diseases that come with obesity and overweight *all-cause mortality*, communicable disease mortality and noncommunicable disease mortality are strongly associated with BMI. This is irrespective of age, gender or smoking status.

Since obesity has risen relatively recently this means that we now have a major future problem for individuals, families, society and the NHS as the effects of the rise in obesity feed through to serious health problems in middle and later life.

Recognising obesity is a problem is easier than tackling it. But we must. And there is a way through this. It is difficult for people to lose weight *and sustain that loss*; they need support. People with obesity often feel highly stigmatised, meaning they may be late to ask for help. Many people, especially those living in areas of deprivation, live in obesogenic environments where high-calorie

foods are widely available, heavily promoted and are more affordable than alternatives. The tendency to blame individuals for being relatively overweight is both unhelpful and scientifically wrong. Individuals, society, health services and industry all have a role to play in addressing this major public health problem now and for the future.

To understand what is possible it is important to understand why obesity arises. The reasonable question is actually why is obesity not universal? And why has it increased over time? Weight gain is, crudely, calories in, and absorbed, compared to calories expended. Most people enjoy eating. The control of how much we want to eat, known as satiety, is however highly regulated biologically. The body controls very closely how much food is taken in. There is a strong genetic component. The key to how much we want to eat is the brain, especially the hypothalamus, and multiple systems which act on it. These include gut hormones and multiple signalling. These pathways in principle provide a route to treatment in the long run.

The genetic component of obesity is strong but complex. At the extreme end is the very rare Prader-Willi syndrome where a single chromosome change leads to children being constantly hungry. For most people multiple genes affect hunger and satiety. Individual variation within particular environments can largely be explained genetically. Several medical conditions, including hypothyroidism, can also cause weight gain. If either the mother or father have obesity, then children of either gender are also more likely to as well but with many exceptions both ways. This is a combination of genetic and social factors.

The body tries to hold on to weight gained. By making lifestyle changes it is possible to lose significant amounts of weight. The body however responds to weight loss by 'trying' to get back to the previous, maximum weight. Someone who has lost weight can have a prolonged physiological response like a leaner person who is starving. This can lead to yo-yoing between obesity and healthier weight.

The reason that obesity has changed over time can largely be explained by availability of calories, fat supply and a strong industry pushing food that is very high in calorific value. As calories available increases, overall obesity in society also increases. It is however not just the total number of calories that are consumed. Ultra-processed foods lead to more weight gain per calorie ingested<sup>1</sup>. They are very high calorie density and far more of the calories are absorbed than unprocessed food where many calories will simply pass through. Unsurprisingly there is a strong correlation between childhood obesity prevalence and how common fast food and other ultra-processed food types are available.

Responding to obesity has to combine elements from the individual, medical and other healthcare practitioners, and society at large. Professional medical and healthcare staff have a central role in secondary and tertiary prevention based on individual consent. They give advice on not gaining weight and losing it sustainably; where necessary prescribing drugs and in some cases surgery where lifestyle changes have failed to achieve sufficient return towards a normal weight.

Diet is of course very important. Sustainable weight loss is possible, with substantial benefits. The key is to have calorie/fat intake lower than needed to maintain the current weight. 600 kcal/day is ideal. It needs to be nutritionally balanced and enjoyable.

Exercise also has an important role but it is very difficult to lose weight when someone has obesity without reducing calories. In order to prevent obesity, most people may need 45 to 60 minutes of moderate intensity activity a day. Exercise is essential to wider health benefits, whatever people's weight, and can help keep weight down. It is a misconception however to think it can be the single

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<sup>1</sup> <https://www.nhs.uk/live-well/eat-well/what-are-processed-foods/>

mechanism by which people who have obesity can lose weight. Physical activity drops off in areas of the greatest deprivation for a variety of reasons.

One area that is advancing, and is likely to advance rapidly over the next decade in the view of those working in the field, is *drug treatment for people with obesity*. Current drug treatments have limited efficacy or unacceptable side-effects. The most widely used drug to date has been orlistat which prevents around 1/3 of the dietary fat that is eaten from being absorbed by the body but has side-effects in many cases. Using the biochemical and hormonal mechanisms by which appetite is controlled by the body is potentially a much better way of treating it. This year the drug trial of semaglutide, a glucagon like peptide previously used in diabetes showed significant benefit in clinical trials. Over time those with the greatest problems with obesity where lifestyle changes alone have not been sufficient will be able to be supported with drug treatment.

The most invasive treatment is *bariatric surgery*. For people who have significant obesity and who have not been able to control weight by other mechanisms, three different approaches are used; a gastric band placed around the stomach; gastric bypass; a sleeve gastrectomy where some of the stomach is removed. The second two of these have most of their effect through changing the hormonal control of appetite. They can lead to sustained weight loss and if they have Type 2 diabetes then this often resolves. Bariatric surgery is most commonly needed in late middle age and in areas of deprivation.

Waiting until people have obesity to intervene is far less good than creating an environment where it is much less likely in the first place. This is the role of *primary prevention*, and therefore this falls to society and potentially the State. This is the most contested area in how we tackle obesity. In the first lecture in this series, I talked about the ladder of possible State intervention ranging from minimal impact such as public information and supporting science to test possibilities, through more active intervention such as nudge taxes or regulation, and all the way up to outright banning or making individual citizens subject to the law. Any use of State levers and powers in a democracy has to be the decision of democratic leaders.

What is not controversial is that we, the public, need to engage with the food industry on this. Pleasure from food, profits for the industry, and health are not mutually incompatible, but the current approach is leading to a highly obesogenic environment for many. Advertising, supermarkets, distribution of fast-food outlets, sponsorship of sports events all lead to strong incentives and signals for people to consume more calories. The aim should not be to reduce enjoyment but reduce unnecessary intake of energy especially in ultra-processed form.

We will need multiple interventions, each with modest incremental impact. No single intervention will be sufficient. We do not yet know the optimal mix. Governments around the world are struggling with this and try different approaches. I will highlight just one which has been tried in the UK and elsewhere (e.g. Mexico) which is a sugar levy on higher sugar soft drinks. Over the period this has been in place in the UK, the intake of soft drinks has not gone down in any social group, but the amount of sugar consumed by this mechanism has decreased by 35%. This is therefore a good example of a win for public health without any obvious loss for enjoyment by any segment of society or industry. The changes were achieved by soft drinks manufacturers reformulating their soft drinks, so they were below the threshold.

Obesity has increased and is increasing in the UK and globally. There are multiple health impacts of obesity on individuals, and implications of this for society in the long run are profound. At an individual level this is heavily determined by genetic factors mainly through their impact on appetite. But at a societal level the increase in obesity is associated with deprivation and ultra-processed foods amongst other factors. No single intervention is going to solve the problems associated with



obesity, but we are advancing both in treatment such as drugs, and have the potential to intervene socially.

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