Obesity and the eye

With increased emphasis on the primary care sector becoming involved in delivering messages about general well-being and lifestyle, **Ross Grant** explains the significance of obesity as a threat to general and ocular health.

The World Health Organization (WHO) convened a consultation on obesity in 1997 in the light of increasing concerns about its impact on health and society.[1] The condition is related to consumption of refined carbohydrates and sugar-sweetened drinks, consequently many agriculture ministries from countries whose economies were dependent on industries supplying these products opposed obesity's classification as a disease.[2] Obesity prevalence also correlates inversely with years of education,[3,4] the implication being that this in turn relates to access to health-related information and countermeasures comprising an understanding of the risks and need for self-control over preferences.

Once established in a population, obesity becomes a significant burden on health services because of its many associated illnesses. As with many conditions, prevention is preferable to cure. As primary care services such as optometry and dentistry regularly have contact with members of the public for routine checks, they offer the possibility of timely intervention or appropriate referral for nutritional advice. For this to work, an understanding of obesity and its implications becomes essential.

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Many conditions secondary to obesity have consequences for vision and the eyes

The ocular connection

While it is often difficult to establish a direct link between obesity and the eyes, many conditions secondary to obesity have consequences for vision and the eyes. Weight control would be aimed at prevention or as an adjunct to treatment to address the underlying cause associated with the excess weight.

Inflammatory conditions are associated with obesity[5] with the implication that any condition with an inflammatory element could be aggravated by excess weight. Probably the most common manifestation of this would be dry eye, which includes inflammation in its development cycle, the treatment of which could result in dramatic improvements in ocular comfort.[6]

Insulin sensitivity and type 2 diabetes are increasing rapidly in the population. They are a particular concern in children and young adults[7] where the long duration of the diabetes can lead to earlier onset of associated conditions such as cataract and diabetic retinopathy. There is a strong association with obesity, and prevention through weight control is seen as probably the best management approach. There is also a link between obesity and hypertension.[8]

Ocular manifestations range from earlier changes such as arteriosclerosis, tortuosity and nipping of veins at crossovers, through to full hypertensive retinopathy with vessel leakage leading to exudates and haemorrhaging.[9]

As well as these general conditions, obesity has been associated with the ocular conditions such as cataract, age-related macular degeneration (AMD) and increased ocular pressure.[10]

As obesity is an underlying cause for all of the above conditions, controlling it will arguably improve both general and ocular health, firstly as a preventative measure, and secondly as an addition to any other therapies that are applied.

TABLE 1
Interpreting body mass index

BMI	Assessment
<18.5	Underweight
18.5-24.9	Normal weight
25-29.9	Overweight
30+	Obese

The colour of fat

The distribution of lipid droplets in fat affects its composition and classification. Large lipid droplets result in white fat; smaller, more densely distributed droplets are found in brown fat, which also contains iron-rich mitochondria associated with muscle tissue, giving it its colour. These mitochondria can burn the fat in order to keep us warm.[11] Brown fat is found in hibernating animals, to prevent hypothermia, and in babies who are unable to shiver and so have proportionally larger quantities of brown fat in their necks and shoulders to keep them warm.

In obesity, white fat is more common and is found in two main locations; just under the skin (subcutaneous) and inside the abdominal cavity (visceral). Visceral fat can exert pressure and restrict proper function of the internal organs, arteries, veins and nerves. Higher fat levels are also associated with a bigger presence of macrophages and inflammatory markers,[12] with the result that fatter people are more prone to inflammatory conditions and associated chronic conditions. ▶

These include liver disease, metabolic syndrome, diabetes, hypertension and cardiovascular disease.[13]

A third type of fat, beige fat, occurs as a response to cold, when white fat cells acquire brown fat cell characteristics and can burn energy to generate warmth.

Measuring body fat

Body mass index (BMI)

This commonly used scale divides the weight in kilograms by the square of the person's height in metres. Table 1 shows generally used BMI assessments.

BMI is not infallible. Athletes can be assessed as overweight because their higher proportion of muscle weighs heavier than fat, giving a high reading, and loss of muscle in old age can result in artificially low BMI scores.

Anthropometric measurement

This measures body circumference, either simply around the waist, or including measurements for height and around the neck, hips, forearm and wrist. A table of these measurements against weight is used to estimate fat as a percentage of body weight. In another approach, a fold of subcutaneous fat at different locations on the body is pinched and measured with calipers and the results used to calculate percentage body fat.

Electrical resistance

A small current is passed through the body from one foot to the other, and as muscle is more conductive than fat, the resistance encountered indicates the body fat level. In more complex tests, the measurement is taken across several locations.

Displacement technique

This is a similar but more accurate measurement to BMI measurement using Archimedes' principle that an immersed body will displace a mass of fluid equal to the volume that it displaces. The difference in submerged and unsubmerged weight along with the displacement volume can be used to make a very accurate calculation of body fat.

DIABETES ON THE INCREASE

Diagnosed diabetes mellitus accounts for around 4 per cent of the UK population and this figure has worryingly doubled over the last decade with most projections suggesting a further increased incidence. There is a new diagnosis at least every five minutes and this results in over 5 per cent of the NHS budget going to the treatment and management of diabetes and its consequences. Each year in the UK, around 20,000 people die as a result of diabetes, and there are nearly 10,000 amputations and 2,500 lose vision due to the disease.

There is no evidence of any immunological involvement with type 2 diabetes and, frustratingly, no single gene has been found to be involved. The expression of the condition is triggered by a number of identified risk factors including:

- Age
- Obesity
- Family history
- Hypertension
- Previous gestational diabetes
- Low birthweight

Why do we put on weight?

Nutrient availability

The Paleolithic diet was sporadic and not assured as it depended on finding dispersed food in order to eat it, and success in this was not guaranteed. Because of this, if food was available, we developed the instinct to eat and if possible store the energy in our bodies pending the next time that availability was restricted. We still have this instinct but, in modern society, the ready availability of food means that if we indulge it we can overeat.

Cooking food releases very much more nutrients for digestion than there are if it is eaten raw. The cell walls of both meat and vegetables are broken down, releasing the nutrients to our digestive systems. Processing foods, for instance by grinding grains into flour, also releases the nutrients, and greatly increases the surface area of the food accessible to digestive enzymes. It is suggested that the time saved from foraging by these more concentrated forms of food has freed up time for us to develop our intellects and societies.

The human microbiome is a population of some 1-2kg of symbiotic bacteria in the gut, and we are coming to realise that this has a much more important part to play in both digestion and health than was previously known. The bacteria break down many foods indigestible to us into forms that we can more readily absorb, and the profile and nature of this population therefore play a considerable part in our use of the food we eat. As mice share our predilection for eating cooked food,[14] they are reasonably good proxies for people in research on diet and obesity. If gut organisms are transferred from obese mice to normal-weight ones, and both groups are then fed on the same diet, the normal mice begin to put on weight. In the reverse case, obese mice who are recipients of normal mice gut bacteria start to lose weight. The bacterial populations and diets are interdependent, and changing one will result in a change in the other.

Weight and activity

An inactive lifestyle is one of the greatest risk factors for obesity.[15] Even relatively small amounts of exercise, so long as they are regular, are beneficial. Here again, a gradual approach is safer – someone who is at risk of a heart condition because of their weight should not launch straight into an aggressive exercise programme. The slow development of fitness in line with weight loss is safer.

Weight loss

The simplified approach to reducing weight is to eat fewer calories than are burnt in a normal day of activity. As with anything biological, this is an over-simplification and subject to the influence of several other factors. Our calorific need goes up with body weight, as we have to support the additional weight that we put on. Consumption of fewer calories leads to loss of weight until our body mass adjusts to the new intake and then weight loss will stop. To continue to reduce weight, we need to reduce our intake to a level supporting an even lower mass.[16]

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A diabetic patient checks their blood sugar levels

With a reduced intake, the first step is to burn up sugars stored in the liver, and as these are dissolved in water, their use means that the water is no longer needed. It is therefore excreted, leading to fairly rapid initial weight loss that slows when all the water is excreted – the body then switches to burning fat and subsequent weight loss will tend to be slower. A slow rate of loss is not to be underestimated – a kilogram does not sound like much when looking at the scales, but if you consider the size of a kilogram block of butter or lard, it is considerable, and its loss should be appreciated.

Exercise builds muscle which, whether it is working or not, burns more calories than fat. However, muscle is also denser than fat, and so weighs more, possibly making fat-loss look less than it actually is when using scales.

Dieting

According to some authorities, people fall into three main groups defined by the underlying causes for their putting on weight. Tackling the dominant trait that applies to any individual will yield the best results in achieving weight loss.

The first group has a genetic disposition to feeling hungry all the time, and therefore have difficulty sustaining a diet. The approach for these people is to encourage them to eat a reduced intake of about 800 calories on each of two days of the week, with a good intake of liquids. This enables them to eat normally for the rest of the week and is easier to sustain than continually holding back. It is combined with healthier eating habits, such as a Mediterranean diet with higher protein and lower fat content on the other days.

The second group produces a lower-than-normal amount of the satiety hormone GLP-1, which tells us when we are full and need to stop eating. These people receive no strong signal to tell them when they are full, and so continue to eat as long as food is in front of them. As the release of satiety hormones takes place quite late as food passess through the digestive tract, the situation for these people is aggravated if they eat foods that are absorbed quickly. There is simply not enough present to trigger the hormone release.

Speed of absorption is assessed using the glycaemic index, which measures the time taken for sugar to peak in the bloodstream when a food-type is eaten. There are two glycaemic index scales, one of which uses white sugar to define the highest level, and the other white bread. People whose satiety hormone release is low need to eat lower glycaemic index foods and more proteins in their diet. As hormone release tends to happen some 20 minutes after eating, developing slower eating habits will also help, a habit that is less prevalent in a fast-food society. Fast eating results in overshoot of satiety and can bring on indigestion.

Surges in blood sugar from high glycaemic index foods trigger a corresponding surge in insulin production by the pancreas, and this high fluctuation is thought to be instrumental in the development of insulin insensitivity and type 2 diabetes. Low glycaemic index foods are generally less processed, making access to their nutrients slower.[17] For instance, wholegrain flour contains more fibre and is less finely ground than white flour, exposing a smaller surface area to digestive enzymes.

The final group eat for psychological reasons associated with anxiety or stress. They will benefit most from joining a weight loss group to provide emotional support and encouragement during the process. Online weight management groups have also been found effective.[18] Their approach, as well as following a diet, is to identify the triggering moments and feelings that lead to eating, and use these to change their behaviour.

The BBC has posted an initial questionnaire which will help to identify which group patients might fall into at www.bbc.co.uk/guides/z2csfg8.

Keeping weight off is a long-term game, and requires a sustained effort to cultivate the habits that keep it off. Two hormones have a significant role to play in energy balance. Ghrelin is a hunger hormone secreted when the stomach is empty, and leptin is a satiety hormone which acts in the hypothalamus to suppress hunger and to create a feeling of fullness. Fat generates leptin, and when body fat is reduced, the amount of leptin produced also goes down,[19] so we feel hungry. The lost weight needs to be kept off until the leptin production normalises, and while this has begun by about 10 weeks after weight loss, the process is gradual and can last longer than a year.[20] A diet producing a gradual loss will mean that the feeling of hunger is less acute.

General strategies for losing weight

Sustained weight loss is achieved through attention to both the quantity and the quality of food intake, preferably associated with an increase in exercise – inactivity is believed to increase health risks more than obesity.[21]

Sudden loss, while potentially producing dramatic changes, will provoke more hunger pangs and be more difficult to sustain. The body needs to get used to new fat levels and other considerations, such as the change in the bacterial profile of the biome, need to ▶

CARDIOVASCULAR DISEASE

Eye care practitioners are well distributed throughout communities, including those with an ethnicity bias to groups known to have a higher risk of developing cardiovascular disease and who may not necessarily attend their local medical practice without any symptoms. They will go to their optometrist when vision continues to change in their 40s and beyond and this underlines further the potential usefulness of the optometrist in screening for insidious disease processes.

The term cardiovascular disease (CVD) may refer to any disorder of the heart, blood vessels and blood circulation. CVD includes coronary heart disease (around 50 per cent of CVD), stroke (around 25 per cent) and circulatory problems ranging from vessel shut down, to inflammatory disease and problems with the blood itself. Underlying changes to vessels and circulatory system tissues predispose to these diseases.

Arteriosclerosis refers to a group of disorders that cause thickening and loss of elasticity of artery walls. Arteriosclerosis occurs to some degree in most adults with increasing age though the presence of arteriosclerotic vessel changes in the retinal vasculature may be evidence of a long-standing cardiovascular problem such as systemic hypertension. The loss of elasticity of the vessel wall results in raised blood pressure. The main type of arteriosclerosis is atherosclerosis. Narrowing of the blood vessels is due to the development of raised patches or plaques in the inner lining of arteries consisting of a substance called atheroma. Atheroma (from the Greek word athere meaning 'porridge') is a mixture of low density lipoproteins, decaying muscle cells, fibrous tissue, platelets, cholesterol and sometimes calcium.

Atheromatous plaques will tend to form in regions of turbulent blood flow such as the origin of the internal and external carotid arteries and may slough off emboli that may lead to vessel occlusion.

Risk factors for atherosclerosis include:

- Cigarette smoking
- Male gender
- Obesity
- Physical inactivity
- Diabetes
- Family history
- Type 1 personality profile

KEY POINTS

- Based on the current data, obesity is likely associated with cataract. However, there is still controversy regarding which type of cataract is related to obesity.
- There is considerable evidence from clinical studies to support an association between obesity and higher IOP. However, current evidence of a direct association between obesity and glaucomatous neuropathy is weak.
- There is considerable evidence that supports an association between obesity and age-related maculopathy. The nature of this association with the different types of ARM and the role of weight loss in preventing the development or slowing the progression of ARM, however, remains to be determined
- Lifestyle changes, such as weight loss, has been advocated as a key factor in helping prevent diabetes and to delay diabetic complications including retinopathy in susceptible patients.
- Obesity has been recognised as a significant risk factor for retinal vein occlusion in a very limited number of studies
- Other ophthalmic conditions may also be associated with obesity. For example, it has been suggested that obesity may be related to oculomotor nerve palsy. Obesity has also been associated with recurrent lower eyelid entropion. Obstructive sleep apnoea syndrome, a common co-morbid condition related to obesity, has been associated with papilloedema and floppy eyelid syndrome. Moreover, obesity is also a risk factor for benign intracranial hypertension.
- Prader-Willi syndrome, an obesity-associated condition, was found to be related with a number of ocular abnormalities including myopia, astigmatism, amblyopia, strabismus and exotropia.

Based on the review paper by Cheung 2007[10]

be given time to happen. A kilogram a week is plenty.

Highly processed food, such as white rice, white bread and sugar, need to be replaced with whole foods with lower glycaemic indices. Less refined foods also have a higher proportion of indigestible fibre which adds bulk and helps the feeling of satiety without actually adding calories.

Reducing the amount of cooked food in the diet and replacing it with raw food reduces accessibility to calories as the raw food is more difficult to digest. It should not be overdone though as switching to a fully raw-food diet can cause complications including amenorrhoea.[22] Drinking half a litre of water up to 30 minutes before eating has been found to result in a lower energy intake, and helps to keep weight off once it has been lost.[23] Avoiding eating late in the evening allows digestion to take place when we are awake and burning more calories – a good guideline is to stop eating three hours before going to bed, and to eat something bulky and filling, but with low access to calories such as a cabbage salad.

If a lot of weight needs to be lost, a consultation with the family doctor or a professional nutritionist is advisable before starting.

Conclusion

Concerns about the rise in obesity in the UK should be a concern to all eye care practitioners. Not only is there an association between obesity, general health and ocular health, but the place of eye care practitioners in the community with repeat access to the public make them ideally positioned to understand the challenges of tackling obesity and offer useful and relevant advice. Prevention is always better than cure. I hope this article has been able to offer a good background in understanding the basic science between obesity and its control.

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