

Obesity: Genetics is no excuse

Obesity is a chronic, relapsing condition. As with smoking, drinking too much alcohol and using illicit drugs, if you have that predisposition to addictive or harmful behaviour – such as eating too much – you may well have it forever. In this respect, it is easy to see why some people tend to put weight back on even after a successful weight loss intervention. In fact, one could argue that eating too much is the worst addiction to have because we cannot totally abstain from food, and so we are continually teasing ourselves with something that we often abuse in excess. However, what is the true cause of obesity? Is it a genetic predisposition that leads us to eat too much, or is it the obesogenic environment in which we live? The Foresight Report (Government Office for Science, 2007) suggested that there are many factors that are responsible for obesity which may, in part, explain why the obesity epidemic has been so difficult to manage or conquer.

We now have evidence to suggest there is a “greedy gene”. However, does this mean that there is no hope for overweight or obese people, and because it is “in my genes” it is pointless wasting valuable NHS resources on weight management programmes? We have known for some time that obesity is linked with an increased prevalence of many common and serious conditions, such as heart disease and diabetes, and that relatively small amounts of weight loss, such as 10 kg, correlate to a significant reduction in morbidity and mortality (Jung, 1997). In one study of 2436 people in Copenhagen it was found that body weight was increasing by approximately 1 kg each year, and that even weight maintenance in this obesogenic environment would convey health benefits compared with allowing people to let their BMI and waist circumference increase (Heitmann and Garby, 1999).

In a study involving 2726 Scottish children, the fat mass and obesity-related gene variant was linked with increased energy intake and may be present in over half of participants (Cecil et al, 2008). It appears to have a role in the control of food intake and food choice rather than the regulation of energy expenditure. The

suggestion is that having the gene may lead children to eat around 100 extra calories at every meal, and possibly more in adults. It may even encourage those with the gene to target more fatty or sugary, calorie-dense foods in preference to healthy options. In reality, it might be responsible for 10 kg of weight in a morbidly obese person of 170 kg, but it does not explain the other 160 kg. This is more likely to be due to poor dietary choices, inadequate education, or physical inactivity through choice or the social environment in which we live.

Is it really useful to an individual to know that there is an obesity-related gene? Does it not give them a further excuse for over-indulgence? We have had a generation of people believing that it is “my glands”, or they are “big boned”, or it is “all muscle”. Now we see people who insist that they eat just one lettuce leaf a week, and in the absence of any proven metabolic problems, say their weight must be due to their genes. When put on a calorie-deficit diet, in combination with an exercise programme, or with appropriate pharmacotherapy, they still lose weight.

Our species has not significantly changed genetically for millions of years, and yet the obesity epidemic has started to spiral out of control over the past generation (Zaninotto et al, 2006). This cannot be explained by an obesity gene alone. It would seem too coincidental that studies have shown a direct correlation between the rise in obesity prevalence with the increasing use of the motor car and other labour saving devices (Prentice and Jebb, 1995).

It would be foolish to dispute a genetic effect, but most obesity is likely to be associated with rather weak genetic tendencies that are modifiable by diet and exercise. Regular exercise and a healthy, calorie-controlled diet remain the best way to control and lose weight, despite any possible genetic causes. In the long term, society needs to address its obesogenic tendencies and perhaps we need to identify, and ultimately learn how to manipulate, the control system for body weight so that any solutions work in harmony with, as opposed to against, our genes. ■



Matthew Capehorn

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Matthew Capehorn is a Board Member of the National Obesity Forum (NOF), Chairman of NOF for Yorkshire and the Humber, and Clinical Manager, Rotherham Institute for Obesity, Clifton Medical Centre, Rotherham.