

# Some insights into the epidemiology of obesity



**Dr Steve Robson**  
FRANZCOG

**One of the tyrannies of distance facing Australians is the long-haul flight required to attend overseas conferences. Few things make travel more miserable than finding a morbidly obese passenger in the seat next to you.**

If you suspect that this is happening more and more often, there are data to support you. Dannenberg and colleagues, from the Centers for Disease Control in the United States, have estimated that American airlines used an additional 1.3 billion litres of jet fuel in 2000 carrying the additional weight of passengers than they would have in 1990.<sup>1</sup> That accounts for an extra 3.8 million tonnes of CO<sub>2</sub> emissions than would have been emitted carrying the same number of passengers a decade before!

*'...although the incidence of overweight and obesity are increasing through the developed world, the same populations are living longer than ever despite the detrimental effects of excessive weight.'*<sup>7</sup>

The Australian Bureau of Statistics (ABS) describes the increasing prevalence of overweight and obesity as an epidemic, posing major risks to long-term health.<sup>2</sup> The ABS estimates that over 20 billion dollars are spent in Australia as a result of this epidemic. As clinicians, we notice that patients who are overweight or obese are more prone to obstetric and gynaecological illness, are more difficult to examine, assess and image, and are more challenging to manage. As was noted in a recent issue of *The Australian and New Zealand Journal of Obstetrics and Gynaecology*: 'In the 1970s, it was rare to see a pregnant woman of 35 with obesity, hypertension and type 2 diabetes, but this is a common daily observation in many antenatal clinics today.'<sup>3</sup>

## What is obesity?

Obesity is excessive adipose deposition on the body and the usual definitions use the body mass index [BMI: mass (kg)/height (m)<sup>2</sup>] of 25 to 30 kg/m<sup>2</sup> for overweight and more than 30 kg/m<sup>2</sup> for obese. While this is useful for epidemiological purposes, this definition is perhaps overly simplistic as it does not take into account the distribution of body fat. This may be more accurately described by the waist:hip ratio (WHR). It is recognised that a primarily truncal distribution of body fat (the 'apple' shape) where the WHR is more than 0.85 in women and 1.0 in men, might be a better predictor of associated morbidities (especially diabetes) than BMI alone.<sup>3</sup>

So-called 'gynaecoid' fat distribution, primarily below the umbilicus, (the 'pear' shape) appears to be less of a risk. The presence of aromatase in adipocytes allows metabolism of androgens to estrogens in fatty tissue. As well, truncal adipose tissue is associated with cardiovascular pathologies and metabolic syndrome.

## How common are overweight and obesity?

Every few years, the ABS conducts National Health Surveys (NHS) of Australians aged 18 years and over. The most recent NHS, conducted during 2004-5, estimated that 53 per cent of all adults (or 7.4 million people) were either overweight or obese, up from 44 per cent in 1995 after adjustment for age differences.<sup>2</sup> While the rate of overweight increased from 32 per cent to 35 per cent over that time period, the prevalence of obesity went from 12 per cent to 18 per cent. For women, the increased prevalence was very similar across all age groups, whereas the greatest increase was in men aged 35 to 44 years.

According to the survey, adults with a tertiary educational qualification are less likely to be obese than those without post-school qualifications. While equal proportions of people in high and low income households had a BMI greater than 25, those in low income households were more likely to be obese rather than overweight (21 per cent versus 15 per cent).

Data from the New Zealand Ministry of Health show that one in five New Zealand adults are obese and one in three are overweight.<sup>5</sup> This is a particular issue for Maori and Pacific Islanders, with 27 per cent of Maori adults obese and 47 per cent of Pacific Island women obese.

An epidemic of weight disorder is also affecting children. Studies from New Zealand suggest that of school-age children, almost one third are overweight or obese. The problem is particularly acute for children of Pacific Island (62 per cent) or Maori (41 per cent) background.<sup>6</sup>

## Why the epidemic?

It is very interesting to note that although the incidence of overweight and obesity are increasing through the developed world, the same populations are living longer than ever despite the detrimental effects of excessive weight.<sup>7</sup> These problems have puzzled evolutionary epidemiologists for some time. Obesity and diabetes have a large genetic component and on the surface it seems difficult to explain how natural selection would favour conditions with such unequivocally negative impacts.<sup>8</sup> In an attempt to explain this, Neel proposed the 'thrifty gene' hypothesis.<sup>9</sup> The 'thrifty gene', which is very efficient in the intake and utilisation of

food, might have been advantageous in ancient times to assist the human species through times where famine was a severe selective pressure. In times of plenty, these adaptations allow rapid laying down of fat as a bulwark against the next famine. Individuals with the adaptation thus have a survival advantage, since increased body weight would also favour fertility. It is only in the modern era where food is plentiful that this genetic strategy backfires.

*‘Perhaps research into messengers such as leptin and adipokinins will yield a “magic bullet” that will help re-regulate many peoples’ brains.’*

This hypothesis is certainly attractive and widely cited, but recently the idea of a ‘drifty gene’ has gained some favour. Speakman summarises the arguments against the ‘thrifty gene’ hypothesis in his paper.<sup>8</sup> In the first instance, since it is likely that modern humans evolved over the last two million years from hominid ancestors in Africa, this would mean that almost 100,000 generations have passed since then. Using reasonably cautious calculations, it would imply that almost everyone should have ‘thrifty’ genes, which is clearly not the case. Indeed, modelling of the likely spread of genes seems to make it almost impossible for the ‘thrifty gene’ hypothesis to fully explain the current epidemiology of obesity and resulting diabetes. Instead, it has been proposed that the current epidemic actually results from genetic ‘drift’ occurring because humans are under little or no selective pressure at all. Thus, random mutations that negatively affect genes controlling fat oxidation will increase in prevalence when food is plentiful. Newer modelling supports this hypothesis and it will be interesting to see where the argument runs.

It is assumed that obesity in the individual is caused by the imbalance between eating and physical activity. The National Health and Medical Research Council (NHMRC) guidelines for management of overweight and obesity state:

At the superficial level, the answers seem simple: eat less and move more. But in reality this is one of the most complex and difficult physiological problems to deal with in modern disease management.<sup>10</sup>

This is because regulation of body fat stores is controlled by multiple overlapping mechanisms, since the search for food and water is so important. It is likely that signals such as that from leptin provide regulatory centres in the brain with information about the volume of fat stored in the body. There is some evidence that obese individuals have an insensitivity to the action of leptin.<sup>11</sup> Thus, susceptible individuals find themselves in an ‘obesogenic’ environment. Unfortunately, once obesity occurs, few therapies are effective in long-term weight reduction.<sup>10,12</sup>

### The future

In some developed countries, the prevalence of obesity is doubling every decade.<sup>13</sup> Since there is no evidence that our lifespans are decreasing (yet), many people will live their long lives burdened with the morbidities of their condition. There is no doubt that overweight and obesity impose a massive burden on the economy, counted in the billions of dollars. Perhaps research into messengers such as leptin and adipokinins will yield a ‘magic bullet’ that will help re-regulate many peoples’ brains. Until then, we all have to look forward to more uncomfortable and environmentally disastrous flights to obesity conferences.

### References

1. Dannenberg AL, Burton DC, Jackson RJ. Economic and environmental costs of obesity. *Am J Preventive Med.* 2004; 27: 264.
2. Australian bureau of statistics. *Year Book Australia* 2008: 1301.0 (available from [www.abs.gov.au](http://www.abs.gov.au))
3. Ramachenderan J, Bradford J, McLean M. Maternal obesity and pregnancy complications: a review. *ANZJOG* 2008; 48: 228-235.
4. Klein S, Allison DB, Heymsfield SB, et al. Waist circumference and cardiometabolic risk: a consensus statement. *Am J Clin Nutr* 2007; 85: 1197-202.
5. Ministry of Health (NZ). Obesity in New Zealand Key facts: [www.moh.govt.nz/moh.nsf/indexmh/obesity-key-facts](http://www.moh.govt.nz/moh.nsf/indexmh/obesity-key-facts)
6. Utter J, Scragg R, Schaaf D, Fitzgerald E, Wilson N. Correlates of body mass index among a nationally representative sample of New Zealand children. *Int J Pediatr Obes.* 2007; 2: 104-13.
7. Lev-Ran A. Human obesity: an evolutionary approach to understanding our bulging waistline. *Diab Metab Res Rev.* 2001; 17: 347-362.
8. Speakman JR. Thrifty genes for obesity, and attractive but flawed idea, and an alternative perspective: the ‘drifty gene’ hypothesis. *Int J Obes.* 2008; e-pub. ahead of print.
9. Neel JV. Diabetes mellitus a ‘thrifty’ genotype rendered detrimental by ‘progress’? *Am J Hum Genet.* 1962; 14: 352-353.
10. NHMRC. *Clinical practice guidelines for the management of overweight and obesity in adults.* 2003.
11. Schwartz GJ. Biology of eating behaviour in obesity. *Obes. Res.* 2004; 12 (Suppl 2): 102S-6S.
12. Colquitt J, Clegg A, Loveman E, Royle P, Sidhu MK. Surgery for morbid obesity. *Cochrane Database of Systematic Reviews* 2005, Issue 4. Art. No: CD003641. DOI: 10.1002/14651858.CD003641.pub2.
13. Bongain A, Isnard V, Gillet J. Obesity in obstetrics and gynaecology. *Eu J Obstet Gynaecol Reprod Biol.* 1998; 77: 217-228.

## CPD Self-Education Activities

**Have you been involved in developing or reviewing guidelines and protocols?**

**Did you know you can claim CPD points in the self-education category?**



Download a form from the College website at:

[www.ranzcog.edu.au/fellows/cpdselfeducation.shtml](http://www.ranzcog.edu.au/fellows/cpdselfeducation.shtml)

If you have been further involved with the implementation and audit of the effectiveness of the guideline/protocol, you can claim this time spent in the PR&CRM category at the rate of one point per hour.