Diabesity

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Almost 30% of Western Australian adolescents are overweight, very soon 80% of all American adults will be overweight and there are now more overweight people worldwide than underweight. There seems to be an acceptance; as we get older we get fatter, our blood pressure rises, our lipids go up and our glucose tolerance deteriorates. The term "diabesity" has been coined to describe the linkage between obesity and diabetes. Although obesity is not a prerequisite for development of Type 2 diabetes, there is an exceptionally strong relationship between both. Even at a body mass index (BMI) of 26 kg/m^2, you have between a 2 to 8-fold increased risk of getting Type 2 diabetes; at BMIs of 35+, the increased risk is 42-fold for men or 93-fold in women. These enormous relative risks demonstrate that obesity is driving this epidemic of diabetes.

Measureable predictors explained

In patients with diabetes, hypertension or dyslipidaemia obesity is an independent predictor of worse clinical outcomes; it also closely associates with insulin resistance.

A simple effective way to detect insulin resistance, and therefore the likehood of metabolic syndrome that predicts future cardiovascular disease (CVD) and diabetes, is to measure waist circumference (see table). BMI is an indirect measure of body fat which in general correlates well with, but does not characterise body fat. Adipotoxicity is the term given to central visceral fat that promotes increased free fatty acids and cytokine release and decreased adiponectin; these all promote atherogenesis and the related cardiovascular complications.

Identification of high risk patients by screening allows a targeted therapeutic approach. Of interest is a distinct population of obese yet 'metabolically normal' individuals who do not develop diabetes or CVD; instead, osteoarthritic complications may dominate.

The degree of health impairment is therefore determined by three factors:

1) the amount of fat,

2) the distribution of fat, and

3) the presence of other risk factors.

Why is diabesity so widespread?

Diabesity is a disease of recent times. Why? Undoubtedly, it is the change in the environment that is responsible for this health problem we are facing today.

Consistent differences, even if very minor, between energy intake and energy expenditure can lead to large changes in body fat mass over time.

There is clear evidence that portion sizes are increasing. The more food on the plate, the more we eat.

In a wider context however social factors contribute to a progressively more obesogenic environment: reliance on cars, abundance of energy dense food. An environment that promotes overeating and undermines activity makes weight management a constant challenge.

Survival genes may predispose humans to develop obesity although genome wide association studies have failed to demonstrate any significant genetic contributors in adult onset obesity. Nonetheless, genetic research continues to offer insights into pathogenesis and may provide future novel therapeutic options.

Of course, reversible causes of obesity and resultant poor glycaemic control are considered when investigating diabesity, such as hypothyroidism and Cushing's syndrome. Thin skin, easy bruising, proximal myopathy are useful clinical discriminators between diabesity and Cushing's syndrome.

Approach to management

The combination of poor glycaemic control and obesity is challenging as some anti-diabetic medications might contribute to weight gain. The novel GLP-1 analogues may however confer weight loss.

Disappointment and scepticism about the effectiveness of obesity therapies may explain the lack of established obesity health services. Lifestyle change is the cornerstone of therapy and management is for the long term. Short-term obesity therapy does not result in long-term weight loss – one of the biggest hurdles patients experience is weight regain. Simple recommendations include sustained consumption of less than 25% of energy intake as fat, total energy intake of1200-1800 kcal/day and 150 minutes of aerobic exercise per week.

Drugs may be complimentary to lifestyle even though there are risks of abuse and side effects. Current anti-obesity medications are; Phenteremine, Orlistat and Sibutramine. They are essentiality similar in the ability to lose 5-10% of bodyweight although differ in side effect profiles. Orlistat may be helpful in patients with dyslipidaemia and diabetes whereas Sibutramine may offer help if mood disturbance is present. Weight loss of 5-10 % is required to achieve meaningful metabolic benefits, such as a 1% reduction in glycosylated haemoglobin.

A progressively popular opinion is that bariatric surgery, predominantly laparoscopic adjustable gastric banding (LAGB), is the only effective therapy for obesity. Superior weight loss can be achieved with LAGB versus comprehensive lifestyle changes including pharmacotherapy. Furthermore, at two years, diabetes remission occurred in 73% of patients who underwent LAGB compared to only 13% in the conventional treatment arm. Information on long term weight loss, diabetes remission and safety are needed to determine the place of LAGB in the prevention and treatment of Type 2 diabetes.

The public health response

With a worldwide epidemic of diabesity coming our way, policy decisions at a government level will have the largest impact. Increased awareness across all levels of society is needed.

Health care professionals have an opportunity to assist patients in setting realistic personal goals, in reducing the effect of the obesogenic environment and choosing the best medical therapies. An effective weight loss program combining diet therapy, physical activity, and behavior therapy can result in meaningful metabolic improvements.

Bariatric surgery is the most effective available weight loss therapy, but is associated with the highest risk of complications. Let's start somewhere simple: think smart, eat well and move more! ■

References available on request

Table: Waist circumference and risk of metabolic complications

Men		
	Increased risk	**Substantially increased risk**
	≥ 94cm	≥ 102cm
Women		
	Increased risk	**Substantially increased risk**
	≥ 80cm	≥ 88cm

(Caucasian values, different cut-offs exist depending on ethnicity)