Introduction

Objective

The objective of this work is to apply graphical methods to monitor changes in the body mass index (BMI) distribution of New Zealand adults from 1977 to 2003 by age, gender, ethnicity and socioeconomic position, using measured BMI from serial nationally representative cross-sectional prevalence surveys.

Background

Excess body weight is one of the most important modifiable risk factors for a number of important diseases, including type 2 diabetes mellitus, ischaemic heart disease (IHD), ischaemic stroke and several common cancers (WHO 2000). The impact of excess body weight on these diseases operates, at least in part, through its effects on insulin resistance, blood glucose, blood lipids and blood pressure.

Energy intake is determined by food and beverage consumption. Energy expenditure has three main components: basal metabolic rate, dietary thermogenesis (ie, energy expended converting food to nutrients), and physical activity (WHO 2000). The most variable component of energy expenditure is physical activity, which contributes approximately 30 percent of energy expenditure in sedentary adults and approximately 50 percent in adults involved in heavy manual work.

Excess body weight is the result of a positive energy balance; that is, a chronic excess of energy intake over energy expenditure. Currently it is not known whether a large positive energy balance on some days or a small positive energy balance on most days produces the larger increase in body weight.

Although some people are more genetically susceptible to weight gain than others, the rapid increase in mean BMI and in the prevalence of overweight and obesity during the last two decades has occurred too quickly to be explained by genetic or demographic changes (WHO 2000). Instead, the dramatic shift in population BMI distribution is thought to be the result of exposure to an 'obesogenic' environment, which promotes sedentary lifestyles and overconsumption of energy-dense foods and beverages (Swinburn et al 1999).

Body mass index

Body mass index is the anthropometric measure that provides the most useful population-level indicator of excess body weight. BMI is a measure of weight adjusted for height, and is calculated by dividing weight in kilograms by the square of height in metres (kg/m^2). For this report, adults were classified as overweight or obese according to the following criteria (Table 1).

Table 1: Classification of overweight and obesity according to BMI (kg/m^2)

Classification	European and Other	Māori and Pacific peoples
Overweight	25.0–29.9	26.0–31.9
Obese	≥ 30.0	≥ 32.0
Extreme obesity	≥ 40.0	≥ 40.0

Higher BMI cut-offs have been used to classify overweight and obesity (but not extreme obesity) in Māori and Pacific peoples. These higher cut-offs have been recommended because at any given BMI, Māori and Pacific peoples have a lower level of body fat than Europeans (Swinburn 1998). However, the relationship between these higher cut-offs and the health risk experienced by these ethnic groups is unclear. *Lower* BMI cut-offs have been proposed for Asian ethnic groups (WHO, IASO, IOT 2000), yet these are even less well established and are not used in this report. The use of ethnic-specific cut-offs remains a controversial topic, and the choice of cut-offs should be guided by the objectives of the analysis (WHO Expert Consultation 2004).

For all ethnic groups, BMI cut-offs are ultimately arbitrary rather than representing true risk-based thresholds. In fact, the health risks associated with increasing BMI are continuous and graded, and begin at a BMI below 25 (Asia Pacific Cohort Studies Collaboration 2004). For example, the association between BMI and type 2 diabetes is continuous (log linear) down to BMI values as low as 19 or 20 kg/m^2 (Willett et al 1999). Therefore, attention should be focused on (shifts in) the whole BMI distribution, not just on BMI categories, however discretised.

A full analysis of the mortality burden attributable to New Zealand's current BMI distribution (as well as mortality potentially avoidable in the future under different BMI distribution scenarios) has been reported elsewhere (Ministry of Health and University of Auckland 2003). In contrast, this report attempts to describe the population's changing BMI distribution over the past quarter century (1977 to 2003) in order to gain insight into the evolution of the 'obesity epidemic'[1] in New Zealand.

Describing BMI distributions

The simplest way to examine changes in a population's BMI distribution between two points in time is simply to show both distributions as relative frequency histograms in the same chart (a relative frequency histogram plots the proportion of the population with each level of BMI against the BMI). Here the histogram is smoothed over the BMI intervals using kernel smoothing in order to visualise the whole distribution more clearly. This process gives kernel densities (Figure 5).

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[1] The term 'obesity epidemic' is used loosely in this report to refer to the observed shifts in BMI distribution rather than specifically to the increase in obesity prevalence *per se*.
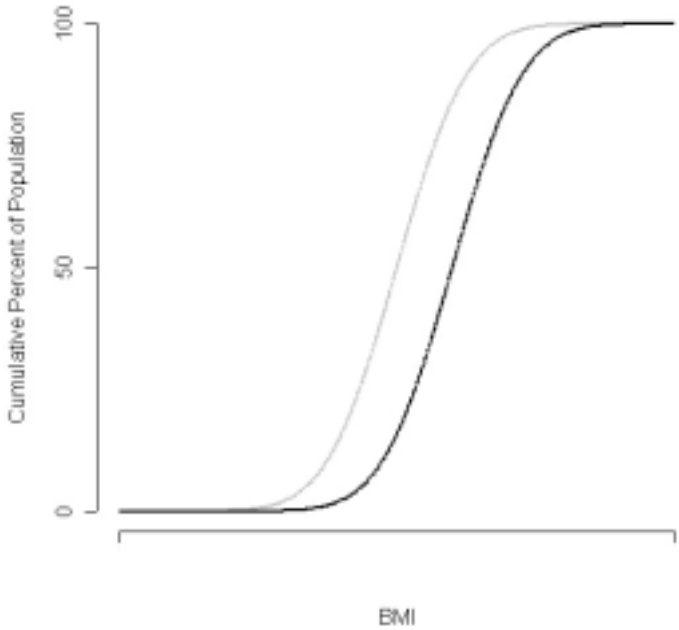
Figure 5: BMI distribution (kernel densities)
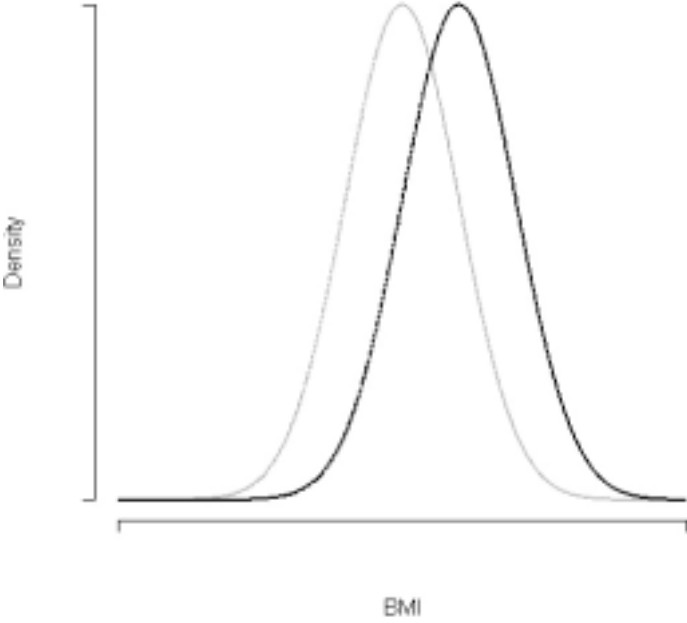
Key: ——— population at time t1 ——— population at time t2

Another graphical method often used to compare two distributions is the cumulative distribution. The cumulative distribution shows, for each of the populations, the cumulative percent of the population at each BMI value (Figure 6).

Figure 6: Cumulative BMI distribution

Key: ——— population at time t1 ——— population at time t2

It is easier to visualise the difference between the two distributions using the cumulative distribution than the simple relative frequency histogram (kernel densities), but it is still difficult to see changes at the extremes of the distributions, and to quantify the distributional shift. A method that overcomes both of these limitations is the Tukey mean–difference (m–d) plot (Cleveland 1993).

The m–d plots show the difference between the two distributions at each percentile against the mean of the two percentiles. For example, if the BMI value of the 50th percentile was 26.0 for one distribution and 25.0 for the other, then the m–d plot would show an x-axis value of 25.5 and a y-axis value of 1.0 (and so on for all other percentiles) (Figure 7).

Figure 7: Tukey mean–difference plot (single percentile only shown)
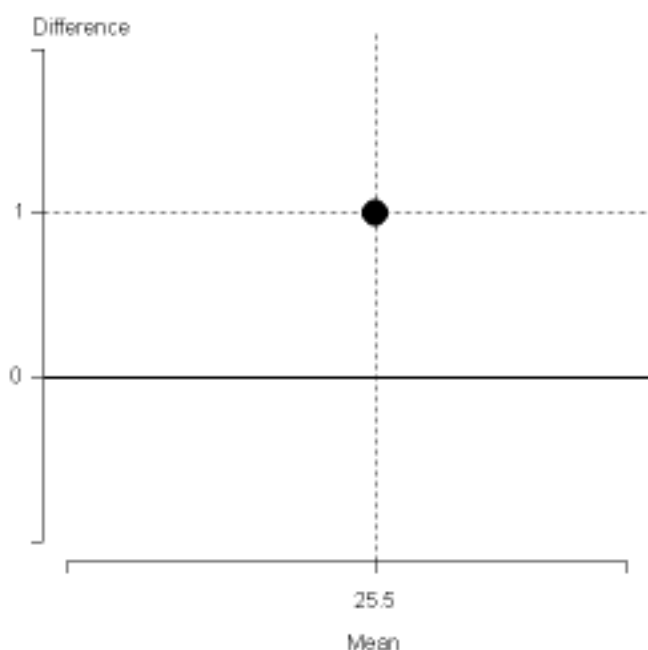
Note that if no shift had occurred at this percentile, the y-axis value would have been zero (and the x-axis value would have remained 25.5). So no shift at any percentile (identical distributions) is represented in an m–d plot as a horizontal line at zero.

Interpreting m–d plots

While not containing any new information beyond the (cumulative) distributions, the m–d plot allows us to more readily quantify the shift and to specify the location of the shift in relation to the distributions being compared. Figures 8–10 show three possible patterns:

- universal, single population or Rose model
- high-risk subgroup, two-population or stepped person–environment interaction model
- mixed or continuous person–environment interaction model.

Universal model

The 'universal' or 'single-population' model implies that increases in the prevalence of obesity are related to changes in the distribution of BMI in the population as a whole. Because the whole population is both exposed to an increasingly obesogenic environment and responds to this exposure in a consistent way, the entire distribution undergoes a uniform shift to the right (Figure 8, top chart). Similarly, the cumulative distribution shows a uniform shift to the right across its entire length (Figure 8, middle chart). And the m–d plot shows data points falling on a horizontal line whose distance from zero indicates the size of the (uniform) shift (Figure 8, lower chart).

Figure 8:   Uniform upward shift (the universal model)

Key: ——— population at time t1   ——— population at time t2
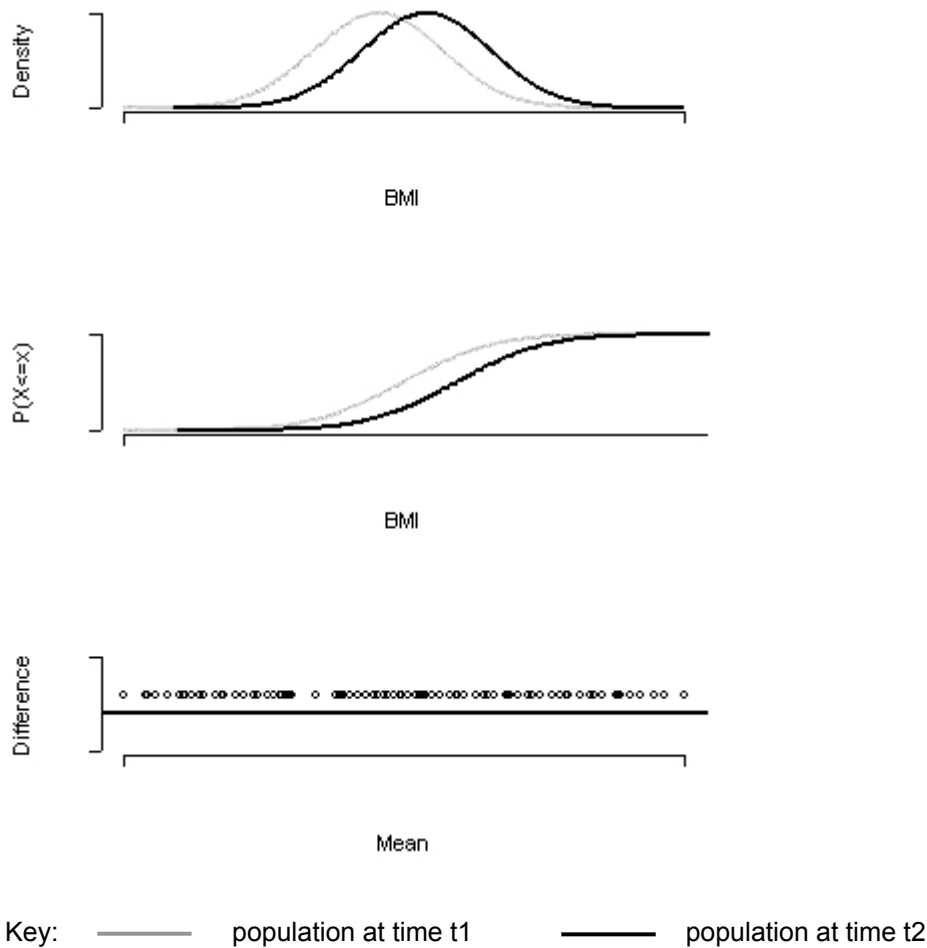
High-risk subgroup model

The 'high-risk subgroup' or 'two-population' model implies that a subgroup of the population exists that is either more (or less) exposed or more (or less) susceptible to the obesogenic environment than the remainder of the population. Given universal exposure to the obesogenic environment, the most likely explanation is a gene–environment interaction, with 'susceptibles' undergoing weight gain while 'resistants' do not. Because only susceptibles undergo weight gain, the BMI distribution shows increasing skewness. At the lower (ie, lighter) end the two kernel density plots remain identical, but the peak of the second distribution falls relative to the first as its right-hand tail becomes thicker (Figure 9, top). Likewise, the cumulative distributions are superimposed at their lower ends, but the second skews increasingly to the right of the first at their upper ends (Figure 9, middle). And the m–d plot shows data points close to zero at lower percentiles, then rising progressively away from the horizontal at higher percentiles (Figure 9, bottom), clearly showing the increasing skewness.

Figure 9:   Increased skewness (the high-risk subgroup model)

Key: ———— population at time t1   ———— population at time t2
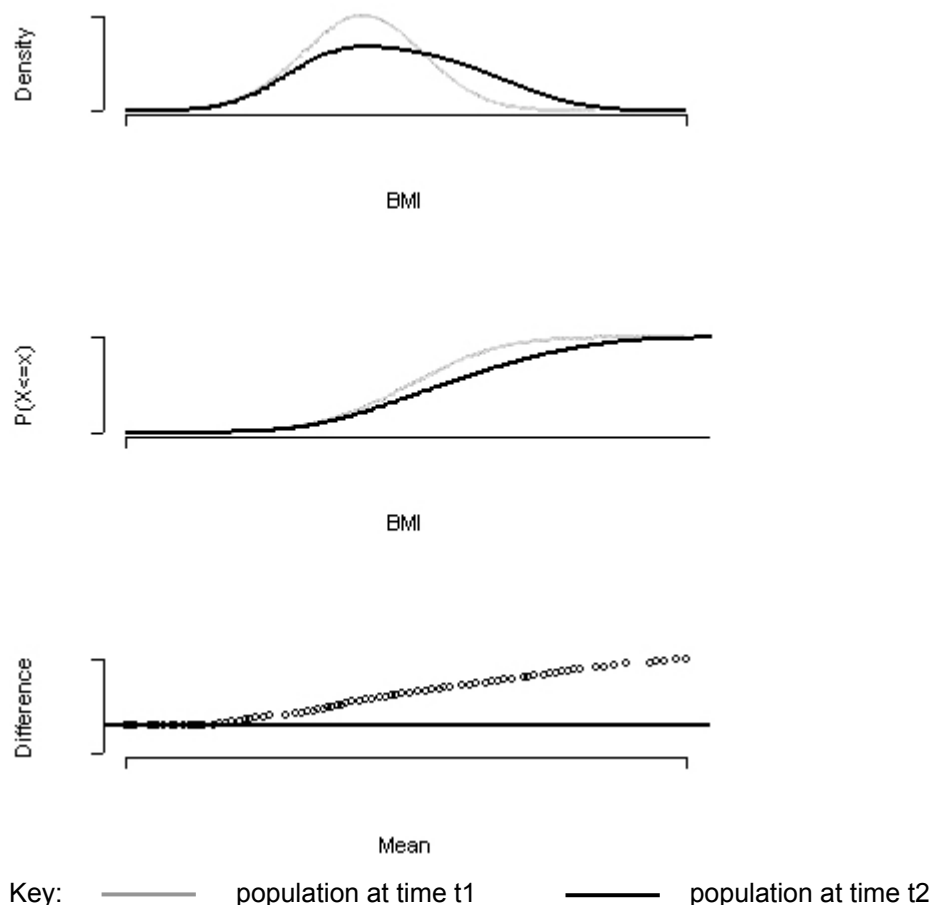
Mixed model

Finally, the 'mixed' or 'continuous person–environment interaction' model shows both a shift of the whole distribution coupled with increasing skewness at the upper end of the distribution. This reflects a situation in which the whole population is exposed and responds to the obesogenic environment with an upward shift of the distribution, but genetically more susceptible or behaviourally more exposed individuals (who are likely to be already heavier on average) respond more, leading to increased skewness of the distribution at its upper end (Figure 10). Thus this model differs from the high-risk subgroup model in that it does not posit a distinct resistant or unexposed population (ie, a stepped person–environment interaction), but instead proposes a gradient in susceptibility and/or exposure across the population as a whole (ie, a continuous person–environment interaction).

Figure 10: Upward shift with increased skewness (the mixed population model)