Discussion

Shifts in the BMI distribution, 1977–2003

This study has provided a clearer and richer picture of the evolution of the obesity epidemic in New Zealand than has previously been available (Wilson et al 2001). It appears that the origins of the epidemic precede 1977, yet the epidemic did not really begin to accelerate until the late 1980s or early 1990s. Most likely it began first among middle-aged females. Even today, it remains largely confined to middle-aged groups (especially among males), although some spread into both older and younger age groups has already occurred (especially among females).

Possible explanations for the observed shift in BMI distribution over this 26-year period (14 years for Māori) include demographic trends, declining tobacco consumption, and changes in dietary and physical activity patterns.

Changes in the age structure of the population (especially the middle-aged group) have been relatively small over the study period, however, and can explain only a small proportion of the shift in the population's BMI distribution (although a slightly greater proportion for Māori). Changes in ethnic mix and socioeconomic conditions likewise account for little of the observed BMI distributional shifting for the New Zealand population as a whole.

Reduction in the prevalence and intensity of smoking over the 1980s and 1990s has also been shown to explain very little of the shift in population BMI distribution, both in this study and another (Simmons et al 1996).

Interestingly, this study shows that the secular trend in stature has continued in New Zealand even into the 1990s (in terms of decade of adult height attainment; ie, the 1970s birth cohort), similar to the experience in most European countries (Cole 2000) and the United States (Ogden et al 2004). Pooling over all ethnic groups and both genders, the mean adult stature of New Zealanders increased by just over 1 cm per decade over the 1970s, 1980s and 1990s. However, this trend again explains very little of the BMI distributional shift.

We are left with the conclusion that the major drivers of the epidemic have in fact been changing dietary and physical activity patterns, themselves largely reflections of an increasingly obesogenic environment.

Examination of the shifts in the BMI distribution, most readily achieved using the Tukey mean–difference plots, shows a fairly consistent pattern across decades, age groups and genders. The pattern that emerges is largely compatible with the mixed model, with little change occurring at the lower percentiles and most of the increase in BMI being concentrated at the higher percentiles (ie, a pattern of increasing skewness). This is mirrored in the rising prevalence of obesity (from 9 to 20 percent among males and 11 to 22 percent among females) accompanied by near stable prevalence of overweight (at approximately 42 percent among males and 27 percent among females). Although differing in detail, the overall pattern is similar for Māori (for the 1989–2003 period), among whom the prevalence of obesity rose from 19 to 28 percent (males) and from 20 to 28 percent (females), while the prevalence of overweight remained stable at 30 percent (males) or increased slightly (from 29 to 32 percent) (females).

In essence, much of the weight gain appears to have involved people who were already obese becoming even more obese (leading to a sharp rise in the number of people with extreme obesity – from less than 1 percent to almost 3 percent of the total adult population), together with a proportion of people who were already overweight moving up into the obese category, only to be replaced by a similar number of people moving up from the normal weight into the overweight category. So the proportion of the population in the obese category has increased sharply (more than doubled) while that in the normal weight category has decreased moderately (about one-fifth), and that in the overweight category has remained stable.

However, this general pattern disguises some differences between subgroups. In particular, the pattern, rate or extent of change has varied between genders, ethnic groups and socioeconomic categories at different times within the overall observation period.

Shifts in the BMI distribution, 1997–2003

This study provides the first indication that, while the epidemic has continued its relentless progress, the period of rapid acceleration from 1989 to 1997 may be coming to an end. Instead, the growth rate of the epidemic appears to have fallen from 1997 to 2003, especially among Māori and among females. Among non-Māori males the BMI distribution has continued to become increasingly skewed, but at a slightly slower rate than in the preceding decade (though the average growth rate in obesity prevalence for this subpopulation currently remains at approximately 5 percent per year, which still represents a very rapid growth rate). Among Māori males, however, the growth rate in obesity prevalence has declined to less than 1 percent per year, although the confidence interval around this point estimate is very wide. Even more dramatically, obesity prevalence is now increasing at a rate of 'only' 3 percent per year among non-Māori females and may already be declining (albeit very slowly) among Māori females. That is, the epidemic may already have peaked among the latter subpopulation, although again the wide confidence intervals prevent definitive conclusions being drawn as to trends in this ethnic group.

Possible explanations for the apparent slowing in the growth rate of the New Zealand obesity epidemic over the late 1990s and early 2000s include artefact, chance, saturation, and genuine public health response (or some combination of these).

Artefactual explanations warrant serious consideration because changes in survey design, sample frames and sampling strategies, response rates, calculation of sample weights and confidence intervals, choice and calibration of instruments for measuring height and weight, standardisation of measurement method (including removal of clothing), and adjustment for seasonality have occurred between all four surveys. Of most relevance is the comparison of the 1997 National Nutrition Survey (NNS) with the 2003 NZHS (although comparison of the 1989 with the 1997 survey is also potentially problematic).

However, a range of statistical tests and substantive analyses suggest that artefact is at most only a partial explanation for the observed differences in BMI distributions between the 1997 and 2003 surveys. For example, comparison of the results obtained using the unweighted instead of the weighted sample are very similar, reducing the possibility of bias being introduced through the weighting process. Differences between the two surveys vary for different genders and age groups, suggesting again the lack of a systematic bias between them. Differences in self-reported diabetes prevalence between the 1996/97 NZHS (which was linked to the 1997 NNS)

and the 2003 NZHS are consistent with the observed BMI distributional shift. Nevertheless, the possibility of selection or information biases between the 1997 NNS and the 2003 NZHS cannot be completely excluded as a possible explanation for the apparent slowing of the epidemic in the most recent period. Or it may be that differences between the 1989 and 1997 surveys created an artefactual impression of rapid acceleration during the early to mid-1990s, and the apparent slowing in the late 1990s to early 2000s simply reflects this.

Chance may have also exaggerated the apparent variations in AAPC across the observation period, despite the differences in BMI distributions being statistically significant at the conventional 95 percent level. For example, the point estimate for obesity prevalence (pooling age groups) may have been biased upwards in 1997 and downwards in 2003 as a result of random variation in the respective samples, thus inflating the AAPC estimate for 1989–1997 while underestimating it for 1997–2003.

Saturation (exhaustion of susceptibles, ie all people susceptible to becoming obese have already done so) must inevitably occur if the high-risk model is true – as our results suggest may be the case (although our results are more compatible with a mixed model). If so, saturation should be seen first among that subgroup of the population among whom the epidemic is found to have begun the earliest and to have already advanced the furthest – Māori females. This is precisely what we observe. Nevertheless, it is surprising that this (sub)population should begin to display exhaustion of susceptibles at a time when 'only' one-quarter of the group is obese and a further one-third are overweight – although this leaves only approximately one-quarter of this subpopulation categorised as being of 'normal' weight; for all other subpopulations, exhaustion of susceptibles would be even less likely. While the prevalence at which saturation becomes manifest is not understood, surveys in several Pacific islands suggest that it is possible for as much as 90 percent of a population to exceed 'normal' BMI limits (with as much as 70 percent or more meeting the conventional criterion of BMI > 30 for 'obese') (Davis et al 2004), although the saturation threshold may well be lower in other ethnic groups.

Is it possible that a proportion of the apparent slowing in the growth rate of the obesity epidemic is in fact due to community *response to the public health message* about healthy weight? While physical activity and nutrition promotion has been actively pursued for some years, there has as yet been relatively little modification to the obesogenic environment (eg, the food environment or opportunities to be more physically active).

However, public health success without intervention (ie, without a formal or structured campaign or other deliberately organised and managed intervention) is not unknown. For example, in New Zealand, a time series (ARIMA) analysis of SIDS incidence found that mothers changed their infants' sleep position in response to an article in a women's magazine (reporting on a case control study that identified sleep position as a critical modifiable risk factor for this condition), *six months before* a major public health campaign was mounted for this purpose (Tobias, unpublished). A similar phenomenon has been described in relation to HIV/AIDS, where high-risk groups changed their behaviour once the risk became known to them, and in advance of any extensive public health interventions to promote and support such behaviour change (De Angelis et al 1998).

Given the extensive media coverage of the obesity epidemic and its risks to health in recent years (especially the past decade), it is conceivable that at least some individuals have indeed

been able to change their dietary and physical activity patterns despite the absence of any major reduction in the obesogenicity of the environment.

A reasonable working hypothesis is that some degree of slowing in the growth rate of the obesity epidemic has occurred over the past five or so years (albeit probably not among non-Māori males), and that this is most likely due to a mix of genuine public health response to the barrage of media publicity on this topic in recent years, together with early saturation among some groups. Yet the extent of the slow-down may have been exaggerated through a combination of chance variation and technical differences between the surveys (especially between the 1997 and 2003 surveys, or possibly the 1989 and 1997 surveys). Clearly, the BMI data from the next wave of health and nutrition surveys (scheduled for 2005–07) will be critical in confirming or refuting this apparent trend.

Policy implications

What policy implications fall out of this detailed description of the shifts in population BMI distribution over the past quarter century? Firstly, we now have clear evidence that the epidemic – while its origins may go back several decades – only really gathered steam in the late 1980s or early 1990s. Middle-aged adults remain the group most heavily affected. Worryingly, however, some spread into younger age groups has already occurred. In the 1990s (but not the 1980s), young people (15–24 years) already had an obesity prevalence of 9 percent (as estimated in this report from 1997 NNS data), similar to the 10 percent prevalence among children (5–14 years) found in the 2002 National Children's Nutrition Survey (Ministry of Health 2003b). Also, spread into older age groups has occurred, particularly among females.

The greater involvement of females, in terms of obesity prevalence, can be expected to dissipate in future as males 'catch up' to their female counterparts. Similarly, ethnic differences should also narrow over the next decade. However, it is possible that the socioeconomic inequality in BMI distribution demonstrated in this report will strengthen and become more graded among males, while persisting or possibly weakening among females.

Our study provides suggestive evidence that the phase of rapid epidemic advance may already be over among females and the epidemic may now be growing more slowly than it did during the early to mid-1990s in this gender; indeed, among Māori women it may already have peaked. However, artefact cannot be excluded as an explanation for this welcome if unexpected trend – at least in part. If not simply an artefact of technical differences between the surveys, this finding of slowing down in the rate of growth of the epidemic (at least among some population groups) may reflect in part the exhaustion of susceptibles (saturation) and in part individual, family and community responses to public health messages about healthy weight.

Even if the latter explanation (ie, a genuine slowing in the epidemic growth rate among non-Māori females and possibly among Māori of both genders) is true, this provides no justification for complacency. Firstly, slowing has not yet been clearly shown for non-Māori males, and even for non-Māori females the epidemic continues to grow, albeit more slowly than formerly. Secondly, the possibility that the progress of the epidemic may in fact be less relentless than previously thought should re-invigorate public health efforts and lead us to set more ambitious targets – and then redouble our efforts to achieve them. Instead of aiming merely to slow down the rate of growth of the epidemic, which is all that was previously considered by many public health workers and researchers to be possible, we may instead be able to realistically

contemplate restoring the population BMI distribution of the 1970s or early 1980s. While communication of the healthy weight and other healthy eating and active lifestyle messages should of course continue to be a part of this, it remains likely that any *sustainable* change will require policy intervention to reduce the obesogenicity of the environment, both with regard to childhood and adulthood obesity.

This is so despite our finding that the pattern of BMI distributional shifting over the past quarter century in New Zealand more closely resembles that of a mixed than a universal model. Even if a substantial minority of the population is in some way resistant to the obesogenic environment, this resistance is unlikely to be absolute. Furthermore, a majority of each birth cohort is likely to remain susceptible to exposures in the social environment that encourage overconsumption of food and avoidance of physical activity. Environmental modification would address the problem for these susceptibles, and for all succeeding cohorts.

A full discussion of policy and other interventions to control, or possibly reverse, the obesity epidemic is beyond the scope of this report. Here, it is sufficient to note that reducing obesity, improving nutrition and increasing physical activity are all included among the priority objectives of the New Zealand Health Strategy (Minister of Health 2000). These three objectives have been combined into the Healthy Eating – Healthy Action Strategy (Ministry of Health 2003a), a high-level framework involving action not only by the health sector but by other sectors as well – including education, physical activity, transport, food and agriculture, and local government. An implementation plan for this strategy has recently been developed by the Ministry in partnership with other central government agencies (eg, SPARC), health-related non-government organisations, academia and industry (Ministry of Health 2004). This plan should be consulted for details of the wide range of interventions potentially available to reduce the obesogenicity of the social and built environments.

The present report, together with its companion report on the health impact of higher-than-optimal population BMI distributions (Ministry of Health and University of Auckland 2003), should provide a useful input to the further development and evaluation of the strategy and its associated implementation plan.

Monitoring implications

The results reported here also have implications for ongoing monitoring of the obesity epidemic. The unanticipated possibility that the growth rate of the epidemic may already be slowing, at least among women, and possibly even reversing among Māori women, necessitates repetition of the NZHS within the next two years (as planned), to confirm that this is in fact the case and not merely an artefact of one particular survey. The next (and all subsequent) surveys should make every effort to maintain high response rates for anthropometric measures and standardise methodology to minimise method drift, and so ensure the integrity of the BMI time series. Repetition of the next National Nutrition Survey, scheduled for 2006–07, will also be critical, by allowing trends in BMI to be related to trends in dietary intakes and participation in physical activity.

Future surveys should also have sufficient power to permit analysis of ethnic-specific BMI distributions beyond only a Māori/non-Māori split, and be repeated at frequent and regular intervals (preferably two- to three-yearly). Anthropometric measures need not be restricted to BMI but should include waist circumference as well (as has been collected in recent surveys).

In addition to monitoring trends in the population BMI distribution, the causes and consequences of such trends should also be monitored. The former information domain would involve improving current survey assessments of energy and macronutrient intake, together with physical activity levels and participation rates.[11] The latter information domain would involve ongoing monitoring of attributable burdens of fatal outcomes related to BMI, and extension of impact assessment to non-fatal conditions such as musculoskeletal, mental and reproductive health outcomes.

Features of the obesogenic environment, ranging from the role of advertising in shaping food preferences to the affordability and availability of public transport, should also be brought under surveillance, along with evaluation of specific nutrition and physical activity policies and programmes funded or co-ordinated under the Healthy Eating – Healthy Action strategy. Monitoring of the recently launched Food Industry Accord should also be included.

This study of the evolution of the obesity epidemic in New Zealand, together with its planned regular updates, will also provide a means for projecting future BMI distributions, and modelling impacts of actual or potential interventions on both the BMI distribution itself and on its health consequences. Such simulations could provide a valuable input to evidence-informed policy and so contribute to minimising the extent and duration of this '21st century' epidemic.

Summary of key policy and monitoring implications

- Mean BMI and obesity prevalence are continuing to increase, although possibly less rapidly now than in the 1990s, at least among non-Māori females and Māori of both genders.

- The apparent slowing of the 'epidemic' in some population groups could be artefactual and still needs to be confirmed in future surveys (especially for Māori, for whom the results are based on relatively small numbers of survey participants).

- Even if the slowing in the epidemic growth rate is confirmed, this gives no reason for complacency. Rather, this finding should invigorate intersectoral control efforts and encourage the setting of more ambitious targets.

- The pattern of shifting of the BMI distribution is compatible with a mixed rather than a universal model – suggesting that strategies aimed at reducing the obesogenicity of the environment could usefully be complemented with targeted strategies aimed at high-risk groups.

- BMI distributional shifting began earliest and has advanced furthest among middle-aged women, but now involves both sexes, Māori and non-Māori, and has spread to younger and older age groups.

- Increased efforts to monitor and control childhood obesity are critical for the health of future generations of adults (the 2002 National Children's Nutrition Survey indicates that 10 percent of school age children are now obese). Thus any slowing in the growth rate of the epidemic among adults could be temporary and may reverse as the current generation of children reach adulthood.

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[11] A joint Ministry of Health and SPARC project has developed an improved physical activity instrument, the NZPAQ, for use in future surveys of population physical activity levels.

- Socioeconomic inequality in the distribution of BMI is marked among females and beginning to emerge among males. Strategies tailored to the needs of lower socioeconomic groups are needed to reverse this trend.

- Monitoring of BMI distributional shifting provides a basis for the projection of future BMI-related burden. This information can also be used to assess the effectiveness of intervention strategies and to model the potential impact of different policy options.

- Such information may not only be of use at the national policy level, but may assist District Health Boards and primary health organisations in designing and evaluating their own obesity prevention and control strategies, programmes and services.

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