Clinical Guidelines for Weight Management in New Zealand Adults

The guideline represents a statement of best practice based on the available evidence and expert consensus (at the time of submission to the Ministry of Health). The guideline is not intended to replace the practitioner's judgement and in each case care decisions should consider the person's clinical state, age, and co-morbidities; the person's and their family/whānau preferences; and the most recent evidence.

Citation: Ministry of Health, Clinical Trials Research Unit. 2009. *Clinical Guidelines for Weight Management in New Zealand Adults*. Wellington: Ministry of Health.

Published in December 2009 by the
Ministry of Health
PO Box 5013, Wellington, New Zealand

ISBN 978-0-478-33987-1
HP 5014

This document is available on the Ministry of Health's website:
http://www.moh.govt.nz

Contents

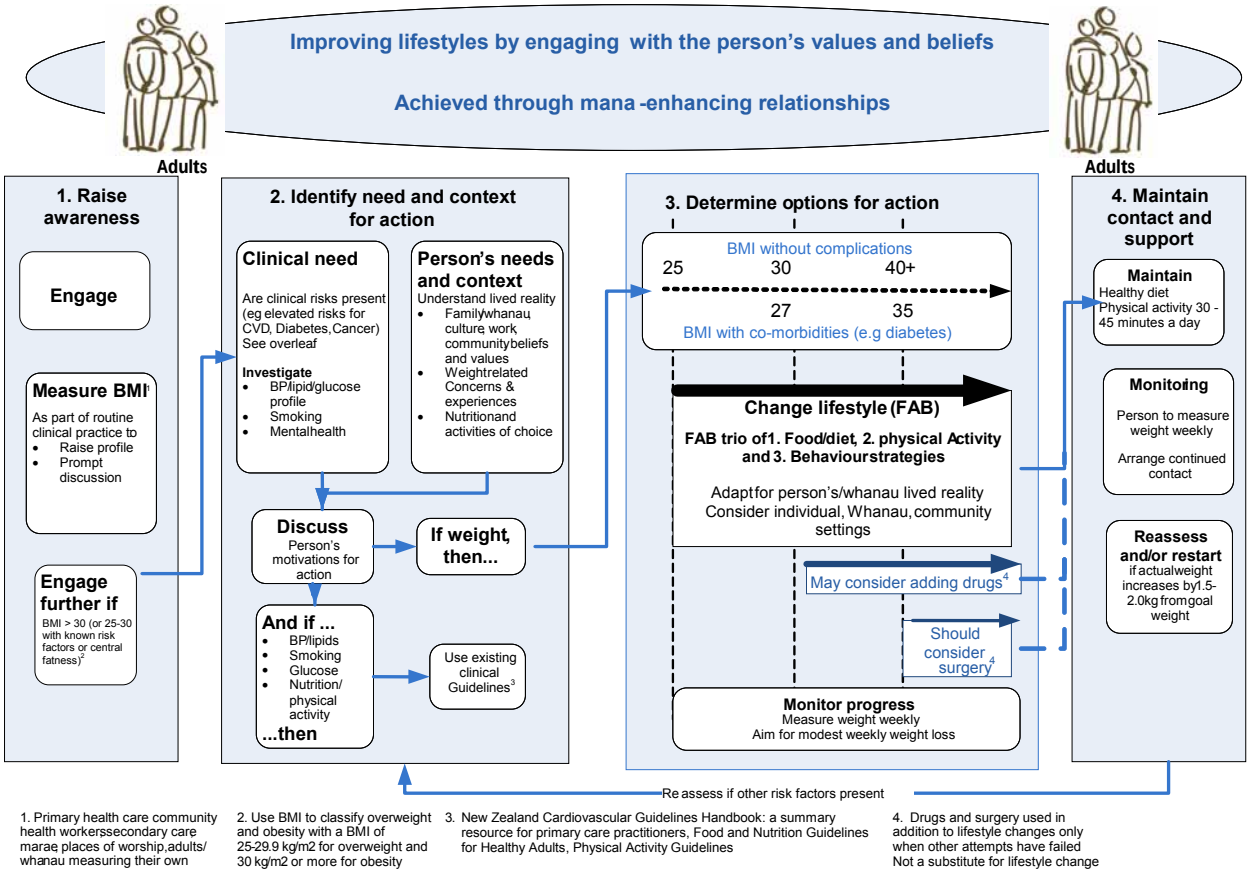
Appendices

References

List of Tables

List of Figures

Algorithm and Key Messages



The high rates of obesity in New Zealand are driven by socioeconomic determinants of health and the obesogenic environment. However, providing support for individuals and their whānau to reduce the risks of excess weight by changing their lifestyle and behaviour is also important.

- Overweight and obesity increase the risk of mortality and morbidity, particularly from cardiovascular disease, some cancers, type 2 diabetes, as well as other co-morbidities.
- Reducing the risks of excess weight is largely about changing lifestyle and behaviour.
- Behavioural change is best effected by working with people's beliefs and values and lived realities through mana-enhancing relationships.

Step 1: Engage and raise awareness

- Engage with the person.
- Measure body mass index (BMI) as part of routine practice for estimate of risk (use the table below).

Classification	Body mass index kg/m^2	Disease risk	
		Waist ♂ 94–102 cm ♀ 80–88 cm	**Waist ♂ > 102 cm ♀ > 88 cm**
Normal	18.5–24.9	–	–
Overweight	25.0–29.9	+	++
Obese I	30.0–34.9	++	+++
Obese II	35.0–39.9	+++	+++
Obese III	40.0+	++++	++++

+ Increased risk; ++ High risk; +++ Very high risk; ++++ Extremely high risk. BMI may not be as accurate in highly muscular people or in ethnic groups with smaller body stature. (Therefore, in South Asians, for example, consider lowering the treatment threshold in the presence of central fatness or additional risk factors.)

Step 2: Identify need and context for action

If the person is in a high-risk category, assess the person's lived realities and clinical need.

- Consider the person's:
  - family/whānau, culture, work, and community, beliefs and values
  - weight-related concerns and previous experiences with weight loss
  - nutrition and activities of choice
  - age, sex, and ethnicity (Māori, Pacific and South Asian population groups)
  - family history of cardiovascular disease
  - smoking status
  - blood pressure and lipid profile
  - common co-morbidities (eg, diabetes)
  - psychiatric history and use of anti-psychotics or mood stabilisers.
- Discuss risks and motivations for action and use other guidelines as required.

Step 3: Determine options for action

- The most effective approach to weight loss uses three key interventions in combination: changes to **f**ood/diet, increased physical **a**ctivity, and **b**ehavioural strategies (called 'the FAB approach').
- The only effective approach to weight management is a permanent change to how people live their lives.
- A realistic target for weight loss varies by individual. Benefits start to accrue when 5–10% of initial body weight is lost. Aim for a modest weekly weight loss.
- Consider referral to professional and community providers.

Diet

- Low energy, low glycaemic index/load, and modified macronutrient approaches are similarly effective. Consider sustainability and the individual's and their family/whānau's preference for diet. Do not use fad diets.
- Very low energy diets require close supervision.

Physical activity

- For weight loss aim to increase periods of physical activity to at least 60 minutes every day. Start with small achievable goals (eg, 5 minutes per day) and build up to target.
- Reduce screen time (eg, watching television, videos and DVDs, and using the computer).

Behavioural strategies

- Include the person's partner and family/whānau in the person's weight management to increase weight loss.
- Identify the changes the person/whānau wishes to work on first. Use problem-solving and goal-setting strategies to achieve changes.

Pharmaceuticals

- Consider anti-obesity drugs when BMI ≥ 30 kg/m^2 or ≥ 27 kg/m^2 (with significant co-morbidities, eg, type 2 diabetes and sleep apnoea).
- Note that anti-obesity drugs such as orlistat and sibutramine must be used in conjunction with lifestyle changes. Counsel a low-fat diet when considering orlistat. Monitor blood pressure with sibutramine use.

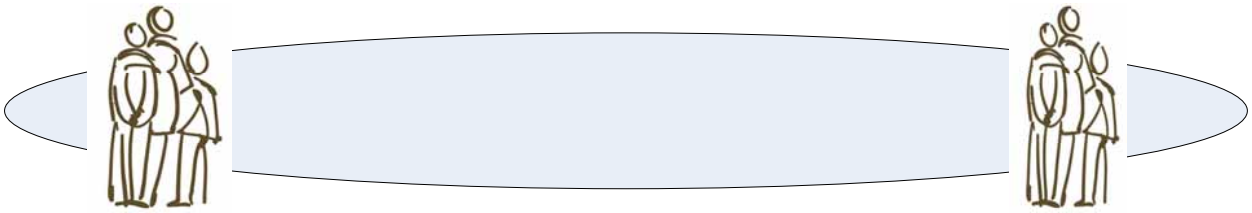
Surgery

- Consider referral for bariatric surgery when BMI ≥ 40 kg/m^2 or ≥ 35 kg/m^2 with significant co-morbidities.

Step 4: Arrange ongoing contact and support (once reach goal weight)

- Make arrangements to reinforce lifestyle change through regular brief contact (eg, ongoing clinical, family/whānau or community contact).
- Encourage person to continue to weigh regularly (eg, weekly) and has strategies to manage weight regain.
- Encourage person to maintain healthy diet and do at least 30–45 minutes physical activity every day.
- Restart weight management programme immediately, if person's weight regain increases 1.5–2.0 kg over goal weight.
- Consider anti-obesity drugs for weight-loss maintenance.

Improving equity of outcomes of adults



- Improving weight management outcomes for Māori, Pacific and South Asian populations is a priority for this guideline. Good practice that reflects the rights, needs, culture and context of priority populations can improve the uptake and impact of guideline-based interventions. The guideline emphasises the importance of involving others as appropriate (eg, spouse, family/whānau) and achieving mana-enhancing relationships through a responsive health system at all stages of the algorithm. Mana-enhancing relationships are ones where there is genuine respect for the person with weight-related risks and a sense of collaboration to connect with those deeper values and beliefs to achieve behaviour change.
- Understand the values and beliefs of the person and others as appropriate (eg, spouse, family/whānau and communities) to support healthy lifestyles.
- The following good practice points may improve service responsiveness and outcomes for Māori, Pacific and South Asian people.
  - Develop mana-enhancing relationships that empower people through respect, trust and mutual ownership by seeking to understand and acknowledge **their** "lived realities", including social determinants, cultural imperatives and socioeconomic circumstances.
  - Assess the clinical needs of the person.
  - Identify opportunities **with** the person to address their clinical needs **within** the context of their lived realities.
  - Identify **with** the person options for action that are realistic and **aligned** to their lived realities.
  - Maintain contact and support **with** the person in a way that is consistent with their lived realities.

Improvi

Abbreviations

AGREE	Appraisal of Guidelines for Research & Evaluation tool
BMI	body mass index
CHD	coronary heart disease
CVD	cardiovascular disease
FPG	fasting plasma glucose
d/L	decilitre
GBP	gastric bypass
GI	glycaemic index
GRADE	Grading of Recommendations, Assessment Development & Evaluation
GTAG	Guideline Technical Advisory Group
HDL	high density lipoproteins
IHD	ischaemic heart disease
kKcal	kilocalories
kg	kilogram
kg/m^2	kilogram per square metre, unit of BMI
kJ	kilojoule
LAGB	laparoscopic adjustable gastric band
LDL	low density lipoproteins
LGB	laparoscopic gastric bypass
LRYGB	laparoscopic Roux-en-y gastric bypass
LSG	laparoscopic sleeve gastrectomy
mg	milligram
mmHg	millimetres mercury, unit of blood pressure
mmol/L	millimols per litre
n	number
NICE	National Institute for Health and Clinical Excellence
NNH	number needed to harm
p	probability, usually p value
RCT	randomised controlled trial
RYGB	Roux-en-y gastric bypass
RR	relative risk or risk ratio
SD	standard deviation
SMD	standardised mean difference
SPARC	Sport and Recreation New Zealand
TE	total energy

WC	waist circumference
WHO	World Health Organization
WMD	weighted mean difference
95%CI	95% confidence interval

About this Guideline

Aim of this guideline

The aim of this guideline is to provide evidence-based guidance for weight management in adults. It is expected that this guideline will be used principally in primary care and community-based initiatives.

Primary prevention of overweight and obesity, although vitally important, is outside the contracted scope of this guideline.

This guideline stands alongside a guideline developed for weight management in children and young people.

Need for a guideline

Since the late 1980s, the prevalence of obesity has increased in New Zealand adults, rising from approximately 10% in adults in 1989 (Ministry of Health 2004b) to 25% in 2006/07 (Ministry of Health 2008c). The most recent estimates of health impact found that a high body mass index (BMI) accounted for about 3200 deaths in New Zealand in 1997 (Ministry of Health and University of Auckland 2003). The direct health-care costs of obesity in New Zealand were estimated at approximately $460 million in 2004 (Ministry of Health 2008a).

Obesity disproportionately affects Māori and Pacific population groups. About 43% percent of Māori adults were obese in 2006/07 compared with 23% of the combined New Zealand European and Other adult group (ie, the group of adults who are not Māori, Pacific, or Asian) (Ministry of Health 2008c). About 65% of Pacific adults were obese in 2006/07 compared with 23% of the combined New Zealand European and Other adult group, an increase from 55% of Pacific adults in 1997 (Ministry of Health 2008c).

Māori and Pacific population groups are considered priority populations for the purposes of this guideline. Although Māori and Pacific population groups represented 14.6% and 6.9% of the total New Zealand population in 2006 (Statistics New Zealand), they represented about 24% and 17% respectively of people with a BMI over 30 kg/m^2 and about 26% and 23% respectively of people with a BMI over 35 kg/m^2.

Māori and Pacific population groups are disproportionately represented in indices of social deprivation (White et al 2008), and the costs associated with weight management interventions may be a barrier to access to these interventions. For instance, Hill et al (2007) found fewer Māori and Pacific population groups than New Zealand Europeans were prescribed the anti-obesity drug sibutramine in 2001–2004. Organisation of service providers may also pose a barrier to Māori and Pacific people. For these reasons, the Māori and Pacific populations are a significant focus of this guideline.

The prevalence of overweight and obesity was also reported for Asian people in the 2006/07 New Zealand Health Survey (Ministry of Health 2008c). However, the term 'Asian' includes a wide variety of ethnic groups, including population groups originating from South Asia, South-east Asia, and East Asia (Rasanathan et al 2006). The diversity of these groups suggests the term 'Asian' is unhelpful when discussing health profiles, because it disguises health disparities between the different groups (Workshop Organising Team 2005). Therefore, we have chosen to use the term 'population groups' (eg, South Asia population groups) in this guideline.

The combined group of South Asian population groups is a priority population group in this guideline. However, the prevalence of obesity in South Asian population groups in New Zealand is uncertain. This guideline uses the term 'South Asian' to refer to population groups originating from India (including Fijian Indians), Pakistan, Bangladesh, Sri Lanka, Nepal, Afghanistan, Bhutan, and the Maldives. (Note that population differences also occur within this group.) The New Zealand Health Survey does not report data by subgroups within the category Asian. The Asian Health Chart Book 2006 estimated the prevalence of obesity from 2002/03 New Zealand Health Survey data (Ministry of Health 2006), but used 25 kg/m^2 as a threshold for obesity and included all respondents over the age of 15. (Therefore, the information does not enable comparison of this group with other population groups where the threshold for obesity is 30 kg/m^2.

Risks associated with obesity

The increasing prevalence of overweight and obesity is of considerable concern. The associations between excess body weight and mortality from cardiovascular disease and some cancers, and the incidence of type 2 diabetes (Willett et al 1999, James et al 2004), are well established. The association between BMI and many of these diseases appears to be continuous, starting from BMI levels of about 20–21 kg/m^2. Excess body weight is also associated with sleep apnoea, asthma, insulin resistance, non-alcoholic fatty liver disease, gout, polycystic ovaries, impaired fertility, musculoskeletal problems, as well as other morbidities (Willett et al 1999, James et al 2004).

Benefits of losing weight

Evidence suggests weight reduction interventions can confer important health benefits. Quite modest weight loss can reduce blood pressure. The Trial of Hypertension Prevention found a 2 kg weight loss over a six-month period resulted in decreases of about 4 mmHg in systolic and 3 mmHg in diastolic blood pressure (Stevens et al 2001). A systematic review of trials estimated that each 1 kg loss in body weight equates to an average reduction in blood pressure of 1 mmHg (Neter et al 2003). Modest weight loss (5–10 kg) can also prevent the onset of type 2 diabetes (Knowler et al 2002). Weight loss also has a positive impact on glucose and lipid control: Avenell et al (2004) found that every 10 kg of weight loss reduced blood glucose 0.04 mmol/L and low density lipoprotein (LDL) cholesterol by 0.25 mmol/L. Intentional weight loss in adults with type 2 diabetes also appears to reduce their risk of mortality by 25% (Poobalan et al 2007). Significant weight loss in obese people has resulted in improvements or resolution of co-morbidities, such as diabetes, hypertension, and sleep apnoea (Sjostrom et al 2004, Grunstein et al 2007).

Can evidence from randomised controlled trials deliver the same level of benefits to clinical practice? Even with pragmatic trial designs, the intervention is controlled and whether the outcomes from trials can be translated into clinical practice is uncertain. However, a naturalistic evaluation of the Counterweight programme in primary care suggests it is possible to achieve similar levels of weight loss in primary care as is obtained by clinical trials (Counterweight Project Team 2008). Mean weight loss was -3.0 kg at 12 months and -2.3 kg at 24 months, which correspond to findings in clinical trials (National Heart Lung Blood Institute 1998). Of those who remained attending the Counterweight programme, about one in three maintained a 5% or larger body weight loss at 12 months (one in 10 of all enrolled people).

Structure of this guideline

Each section in this guideline is structured in the following way.

- An overview of the evidence.
- One or more graded recommendations that are, wherever appropriate, congruent with the recommendations or guidance of other organisations, such as Sport and Recreation New Zealand.
- Good practice points that provide additional guidance, reflecting the expert opinion of the Guideline Technical Advisory Group and are backed up by testing, augmented literature reviews, and consultation with Māori, Pacific, and South Asian population groups.
- A more detailed narrative that summarises the evidence tables.
- A description of evidence that might provide useful information about the priority population groups
- Evidence statements that summarise the evidence and give a quality assessment of the evidence.

How to interpret the recommendations

The evidence was assessed using the Grading of Recommendations, Assessment Development and Evaluation (GRADE) system (Guyatt et al 2008b), a system for developing recommendations that incorporate the values of the Guideline Technical Advisory Group (Swiglo et al 2008). The recommendations were graded as **strong** or **weak**. The strength of the recommendation reflects the confidence the Guideline Technical Advisory Group has that patients receiving the recommended intervention will be better off than they would be if they did not receive that intervention and that the desirable effects of the recommended intervention outweigh the undesirable effects (Swiglo et al 2008). If, in the Guideline Technical Advisory Group's view, the trade-off was sufficiently clear that most practitioners and/or patients would choose the recommended intervention, then the recommendation was graded as strong. The recommendation was graded as weak when the trade-off was less clear and it was considered likely that the practitioner and/or patient might not choose the recommended intervention (Guyatt et al 2008a). See Appendix 1 for further discussion of this grading system.

Guideline implementation and training

Implementation of the guideline and the training needs of guideline users have been considered as part of full implementation planning. They will align with the overall roll-out and implementation of *New Zealand Cardiovascular Guidelines Handbook* (New Zealand Guidelines Group 2009) and the upcoming revised diabetes guidelines.

Treaty of Waitangi and Māori Development

The Government recognises the Treaty of Waitangi as the founding document of New Zealand. The Government envisages all New Zealanders moving forward together with a shared commitment to the future. Since the late 1980s, Māori have become a strong, vibrant force in our society, buoyed by the renaissance of Māori culture and language. However, too many are being left behind. The Government is committed to ensuring all Māori enjoy a brighter, more prosperous future. To do this, the Government is nurturing strong families/whānau and communities. In particular, the Government is seeking significant outcomes in whānau ora by eliminating poverty, advocating for social justice, and advancing Māori social, cultural, economic, and community development in the best interests of the nation.

Policy statement – He Korowai Oranga: The Māori Health Strategy

He Korowai Oranga: The Māori Health Strategy (Ministry of Health 2002) provides a framework for improving Māori health and, in particular, for informing weight management in Māori. At the heart of He Korowai Oranga is the goal of whānau ora: realising Māori potential. The four key pathways to achieve whānau ora are:

- whānau, hapū, iwi, and community development
- Māori participation in the health and disability sector (eg, supporting effective Māori health providers and a highly skilled Māori workforce)
- effective health and disability services
- working across sectors to address the broader determinants of health (eg, social development, education, and housing).

Translating the Treaty of Waitangi, Māori development, and He Korowai Oranga into a pragmatic framework for weight management for Māori

To contribute to the pragmatic implementation of the Treaty of Waitangi and Māori development, while working within the parameters of He Korowai Oranga: The Māori Health Strategy, three strategic actions for Māori have been identified in the area of weight management for Māori.

- Accelerate the development of a culturally competent sector that understands the lived realities of Māori and the importance of mana-enhancing relationships.
- Ensure effective health services for Māori are provided by non–Māori-led and Māori-led providers.
- Promote the ongoing development of Māori-led providers and the Māori workforce.

Evidence of need in Māori

Obesity is disproportionately prevalent in Māori compared with the combined New Zealand European and Other group (Ministry of Health 2008c). About 43% of Māori adults were obese in 2006/07 compared with 23% of New Zealand European and Other adults. The use of anti-obesity drugs may be disproportionately lower in Māori than in New Zealand Europeans (Hill et al 2007). Hill et al (2007) found fewer Māori than New Zealand Europeans were prescribed sibutramine in 2001–2004, and the usage was proportionately lower in Māori women than in New Zealand European women (5.8 per 1000 compared with 6.9 per 1000 population). Usage was similar in both Māori and New Zealand European men (1.2 per 1000 compared with 1.0 per 1000 population).

There are no published data on bariatric surgery in Māori. Only the Wakefield Gastroenterology Centre appears to be regularly publishing New Zealand outcome data (Dhabuwala et al 2000, He and Stubbs 2004, White et al 2005, Wickremesekera et al 2005, Stubbs et al 2006), but it does not publish data about the ethnicity of the patients. Data from the New Zealand Health Information Service suggest a disparity in access to publicly funded bariatric procedures between Māori and the combined New Zealand European and Other group. For instance, 149 procedures were funded in 2007/08 and the rate was 25.9 per 100,000 population with a BMI over 35 kg/m^2 in Māori compared with 51.9 per 100,000 population with a BMI over 35 kg/m^2 in New Zealand Europeans and Others.

Limited evidence base for Māori

We found no direct evidence on the effectiveness of weight management interventions with Māori. We also found no randomised controlled trials of weight-loss interventions with Māori.

Making the guideline work for Māori

The lack of evidence pertinent to Māori in the area of weight-loss interventions required us to make other efforts to identify appropriate good practice points, so we commissioned an augmented review from Dr Cindy Kiro (Kiro 2009).

The goal of the recommendations and good practice points in this guideline is to contribute to the pragmatic implementation of the Treaty of Waitangi and Māori development and to support the weight management sector to respond to the unmet need for effective interventions for Māori. Given the desire to ensure the guideline works for Māori despite the limited evidence base, detailed good practice points and advice on solutions are integrated into the remainder of the document. More detailed information on processes and frameworks are in Appendices 1 and 2.

Making the guideline work for Pacific population groups

Evidence of need in Pacific population groups

Obesity is disproportionately prevalent in Pacific population groups compared with the combined New Zealand European and Other group (Ministry of Health 2008c). Approximately 64% of Pacific populations were obese in the New Zealand Health Survey 2006/07 compared with approximately 23% of the combined New Zealand European and Other group. The prevalence of obesity in Pacific men and women has increased from approximately 55% in the 1997 National Nutrition Survey (Russell et al 1999) and 60% in the 2002/03 New Zealand Health Survey (Ministry of Health 2004a).

Use of anti-obesity drugs may be disproportionately lower in the Pacific population than in New Zealand Europeans (Hill et al 2007). Hill et al (2007) found fewer Pacific people were prescribed sibutramine during 2001–2004 compared with New Zealand Europeans, and the usage was proportionately lower in Pacific women and men than in New Zealand European women and men (2.4 and 0.4 per 1000 compared with 6.9 and 1.0 per 1000 respectively).

There are no published data about bariatric surgery in Pacific populations. Only the Wakefield Gastroenterology Centre appears to be regularly publishing New Zealand outcome data (Dhabuwala et al 2000, He and Stubbs 2004, White et al 2005, Wickremesekera et al 2005, Stubbs et al 2006), but it does not publish data on the ethnicity of the patients. Data from the New Zealand Health Information Service suggests disparity in Pacific access to publicly funded bariatric procedures; for instance, 149 procedures were funded in 2007/08 and the rate was 18.7 per 100,000 population with a BMI over 35 kg/m^2 in the Pacific population compared with 51.9 per 100,000 population with a BMI over 35 kg/m^2 in the combined New Zealand European and Other group.

Limited evidence base for Pacific population groups

We found no randomised controlled trials of interventions for weight loss with Pacific population groups, although there have been two non-randomised controlled studies of church-based interventions: one with Samoan churches (Bell et al 2001) and one with Samoan and Tongan churches (Simmons et al 2004).

In Simmons et al (2004), the programme was supervised by church committees who adapted and enhanced the programme materials. The interventions were tailored to the communities by changing language, the approaches to presentations, foods and cooking methods, kinds of exercise, and the order of sessions. Healthy food policies were introduced for church celebrations and the pastor and bishops continuously encouraged participation in the programmes. Additional adaptations were made by the individual churches. At two years there was significantly less weight gain in participants from the Samoan intervention church compared with participants in the Samoan control church, and there was no significant difference between participants from either of the two Tongan churches. Participants' readiness to change might have influenced the results, because the Samoan intervention church had support structures and resources in place before the trial started.

Bell et al (2001) used three Samoan churches, two being intervention churches and one the control church. A health committee was established at intervention church to promote low-fat diets and increased leisure-time physical activity. Sessions were conducted by Pacific Islands Heartbeat, with demonstrations and culturally appropriate foods. Weekly aerobics sessions were built into church activities. Newsletters and diabetes support groups were used to support the nutrition and activity sessions, and church leaders were trained to become leaders of nutrition and physical activity sessions. Participants in the intervention churches lost weight whereas members of the control church gained weight (-0.4 kg compared with 1.3 kg, p = 0.04).

The lack of evidence pertinent to Pacific population groups required further effort to identify appropriate good practice points for this priority population. Therefore, we commissioned an augmented review from Dr Jemaima Tiatia for Pacific population groups (Tiatia 2009).

Making the guideline work for South Asian population groups

Evidence of need in South Asian population groups

Note that even within the combined South Asian population group there are population group differences that may be important for clinical practice. For instance, evidence from a large cohort of South Asian people in the United Kingdom suggested that people who self-identified as Bangladeshi (n = 2482) had a higher risk of developing cardiovascular disease and diabetes, despite being on average shorter, less obese, and having a lower mean blood pressure than Pakistani (n = 4068) or Indian populations (n = 7328) (Hippisley-Cox et al 2008). Therefore, it is important to consider the presenting individual.

The 2006/07 New Zealand Health Survey found a lower prevalence of obesity among Asian populations (11%), but the survey did not publish more information about the prevalence of overweight and obesity in specific Asian ethnic groups (Ministry of Health 2008c).

The Asian Health Chart Book used 2002/03 New Zealand Health Survey data to estimate the prevalence of obesity in Indian males and females at 34% and 53% respectively (Ministry of Health 2006). The subgroups categorised as 'Indian' were Indian, Bengali, Fijian Indian, Gujarati, Tamil, Punjabi, Sikh, Anglo-Indian, and Other Indian. The Asian Health Chart Book defined obesity as a BMI of 25.0 kg/m^2 or more, but recognised that such a threshold might not be appropriate.

Using World Health Organization (WHO) thresholds, the Asian Health Chart Book reported the prevalence of overweight for Indian men and women at 27% and 34% (95%CIs 15–39% and 26–51%), while the prevalence of obesity in Indian men and women was 7% and 15% respectively (95%CIs 1–14% and 8–22%). These data compare with the age-standardised prevalence of overweight in the total population in the 2002/03 New Zealand Health Survey of 40% in men and 27% in women, with the prevalence of obesity at 19% for men and 21% for women (Ministry of Health 2004a). However, the data from the Asian Health Chart Book was not standardised for age and sex, included respondents aged 15 and over, and had very wide confidence intervals

around the point estimates. Therefore, there is considerable uncertainty about the prevalence of overweight and obesity in South Asian population groups in New Zealand, and it is not possible to determine potential inequalities about prevalence until accurate data are available.

Lower BMI thresholds for overweight and obesity have been suggested for Asian populations based on evidence of a higher percentage of body fat at a given BMI and a higher prevalence of co-morbid disease in cross-sections of Asian populations at a lower BMI than in European populations (National Institute for Health and Clinical Excellence 2006, Huxley et al 2007). However, the most recent WHO Expert Consultation noted that the evidence does not offer clear guidance on accurate BMI thresholds for Asian ethnic groups, so the WHO Expert Consultation retained the WHO BMI thresholds of 25 kg/m^2 for overweight and 30 kg/m^2 for obesity for all adults.

Information is lacking on the use of anti-obesity drugs and bariatric surgery by South Asian population groups (Hill et al 2007). Hill et al (2007) reported aggregated data for sibutramine usage in Asian populations from data from the Intensive Medicines Monitoring Programme in New Zealand between 2001 and 2004. Only 2% of the 17,298 people dispensed sibutramine were Asian, which was lower than the proportion of the total resident population in New Zealand that was Asian at the time (6.3%).

There are no published data about the use of bariatric surgery by South Asian population groups in New Zealand (Dhabuwala et al 2000, He and Stubbs 2004, White et al 2005, Wickremesekera et al 2005, Stubbs et al 2006). One publicly funded bariatric procedure was performed on an Indian patient in 2007/08, but it is not possible to determine any disparity given the lack of data on population prevalence in South Asian populations.

Limited evidence base for South Asian population groups

There is no direct evidence on the effectiveness of interventions with South Asian population groups. We found no randomised controlled trials of interventions for weight loss specifically with South Asian groups. However, there is limited indirect evidence in asthma management and diabetes from international trials, which included evidence from South Asian populations (Hawthorne et al 2008, Bailey et al 2008). Although, it is not possible to directly extrapolate from this evidence to the priority population groups in New Zealand, the evidence does point to culturally tailored approaches possibly being more effective than non-tailored approaches.

The lack of evidence pertinent to South Asian population groups required further effort to identify appropriate responses for these priority populations. Therefore, a detailed review of the guidelines was undertaken by Ruth De Souza for South Asian population groups.

Improving the health of Māori, Pacific, and South Asian population groups

The starting point for the development of this guideline and its associated implementation and training considerations was He Korowai Oranga: The Māori Health Strategy. The goal of the guideline for the priority populations is to help realise whānau ora: supporting the whānau/family and communities to make healthy lifestyle choices that enhance the health and well-being of the whānau. Whānau ora requires health services to be co-ordinated around the needs and realities of whānau. Services also need to incorporate cultural views, beliefs, and practices and be guided by Māori, Pacific, and other models of health.

For the overweight and obesity guidelines to be responsive and relevant to the priority populations, they must be implemented in a way that meets the social and cultural realities of family/whānau and communities, for example, income levels and geographic location (eg, living in a rural area can negatively influence health outcomes for the priority populations). The provider's cultural competence is integral to understanding these lived realities. Cultural competence requires practitioners "to provide patient-centred care by adjusting their [own and organisational] attitudes and behaviours to the needs and desires of different patients, including accounting for the impact of emotional, cultural, social, and psychological issues on the main biomedical ailment" (American Medical Association 1999, p154). Cultural competence, therefore, requires practitioners move beyond simply developing their awareness to acting to overcome the barriers to whānau ora.

The starting point for achieving whānau ora lies with the quality of the relationship between the provider and the family/whānau. The relationship must be one that enhances trust, respect, and mutual ownership of the goals. For the purposes of this guideline, we have defined such relationships as 'mana-enhancing relationships'. Mana-enhancing relationships are interactions where there is mutual respect and a sense of collaboration to connect with the deeper values and beliefs of the person and their family/whānau to change behaviour. Such relationships acknowledge and work with people's lived realities – the cultural, social, and socioeconomic context – to empower change. Through the platform of mana-enhancing relationships and lived realities, the more technical aspects of the guideline can be implemented. The good practice points below may assist providers to realise this vision.

Good practice points for providers

- Develop proactively your cultural competence to facilitate effective communication.
- Develop your relationship with the person before doing anything else to ensure you can empathise with the person's cultural, social, and socioeconomic circumstances.
- Develop your relationship by:
  - assessing factors such as cultural engagement, the role of the person's family/whānau (immediate and extended) and wider community, and the person's employment situation, living circumstances, health literacy, values, and beliefs

  - recognising that differences in views of health and wellbeing may mean you need to work alongside other providers (eg, marae-based workers or church ministers) to improve health outcomes.

- Work with the person to understand their needs within their context (ie, within their lived reality) by:
  - identifying the person's needs and establishing realistic goals
  - assessing and helping the person to overcome the barriers (eg, costs) to meeting goals
  - working with the person's family/whānau, not just with the individual person
  - exploring the meaning of food and opportunities for adapting the person's diet by considering:
    - the types of food available in different contexts (eg, on marae or at church)
    - foods of cultural significance and their seasonal availability
  - exploring physical activity options and opportunities for adapting traditional and customary activities (eg, hunting) for exercise and physical activity
  - considering group-based activities and other activities the person enjoys
  - being aware of and referring to local community options for people interested in weight loss (eg, marae-based, healthy hapū programmes, and church-based or local community group options).
  - being sensitive to the person's communication needs and providing resource material and information in the person's and their family/whānau's preferred language, if possible.

Measurement and Classification of Overweight and Obesity

BMI and waist circumference are two simple indirect measures that can be used to identify people whether a person is overweight or obese (Table 1).

BMI is weight in kilograms divided by height in metres squared (kg/m^2). WHO recommends people with a BMI of 25–29.9 kg/m^2 be considered overweight and a BMI of 30 kg/m^2 or more be considered obese. However, the risk of mortality from cardiovascular disease and cancer and the risk of becoming diabetic start increasing from a BMI of about 21 kg/m^2 with excess abdominal fat (as indicated by waist circumference), which suggests a higher risk than that suggested by BMI alone in people with a BMI under 35 kg/m^2.

Recommendations

Use BMI to classify overweight and obesity with thresholds of 25–29.9 kg/m^2 for overweight and 30 kg/m^2 or more for obesity for all adults.

Use waist circumference to provide useful additional information, especially if excess intra-abdominal fat is suspected in people without a high BMI.

Good practice points

- If using only one measure of overweight and obesity, use BMI as part of routine clinical practice. However, BMI may not be accurate as a discriminator in adults who are highly muscular, in ethnic groups with a smaller body size (eg, South Asian population groups), and in older people.
- Be aware of cultural sensitivities to body parts and body image when measuring height and weight.
- Use your clinical judgment when considering whether people above or below the BMI thresholds are at increased risk and could benefit from weight loss.
- Once a BMI has been calculated, use weight for monitoring an overweight or obese patient's progress towards their goal weight.
- Measure waist circumference at the midpoint between the lowest rib and the iliac crest.

Table 1: Classification of disease risks by World Health Organization body mass index and waist circumference thresholds

Classification	Body mass index (kg/m^2)	Class	Disease risk* relative to normal weight and waist circumference (WC)	
			Men WC = 94–102 cm	Men WC > 102 cm
			Women WC = 80–88 cm	Women WC > 88 cm
Normal weight+	18.5–24.9		–	–
Overweight	25.0–29.9		Increased	High
Obese				
Mild	30.0–34.9	I	High	Very high
Moderate	35.0–39.9	II	Very high	Very high
Extreme	≥ 40.0+	III	Extremely high	Extremely high

* Disease risk for type 2 diabetes, hypertension, and cardiovascular disease.

\+ Increased waist circumference can also be a marker for increased risk even in people of normal weight.

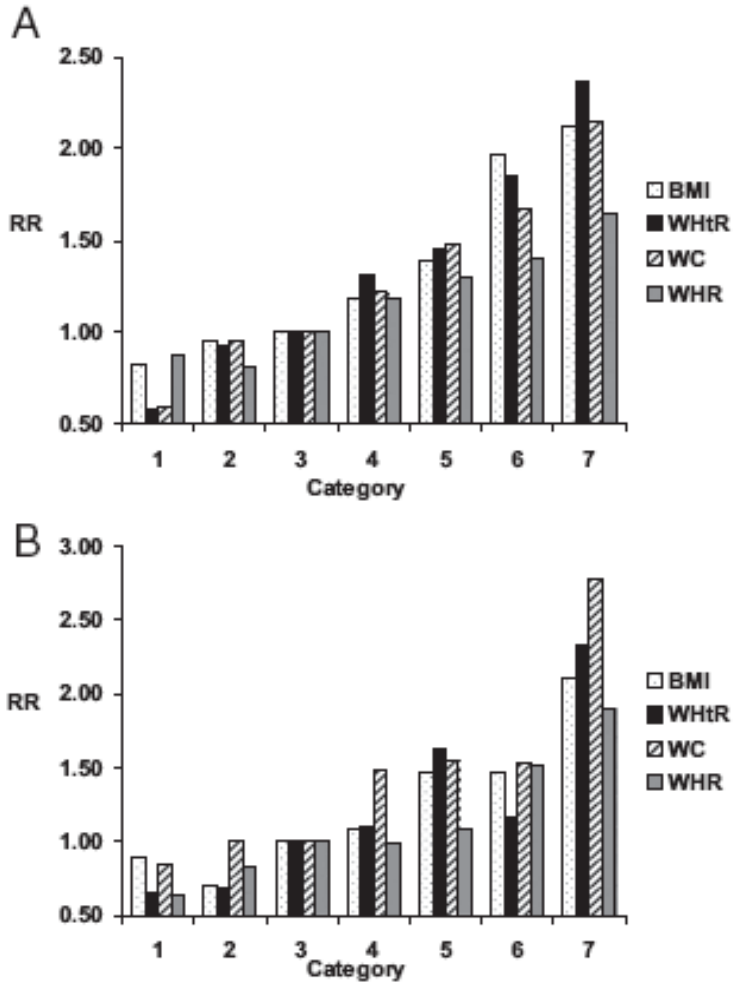
Body mass index

Rationale for using body mass index to measure adiposity

Direct measures of the extent a person's fat tissue, or adiposity, are not practical or affordable in most healthcare settings. Therefore, indirect measures such as BMI, waist circumference, waist-to-hip ratio, and waist-to-height ratio are used. These indirect measures are similarly effective at determining risk of future disease (Figure 1) with all being highly correlated with each other (correlation co-efficient approximately 0.8) and with incident cardiovascular disease (Gelber et al 2008). Waist-to-height ratio had significantly stronger associations with cardiovascular disease than the other measures, but the difference was unlikely to be clinically significant.

The measurement of waist-to-height and waist-to-hip ratios requires training in a standardised technique, which limits their use in clinical practice. In contrast, BMI is simple to measure and calculate with minimal training and is widely accepted as a valid measure in adults (National Institute for Health and Clinical Excellence 2006, Lau et al 2006, World Health Organization 2000).

Figure 1: Relative risk of cardiovascular disease in men (panel A) and women (panel B) according to anthropomorphic indexes



Notes:

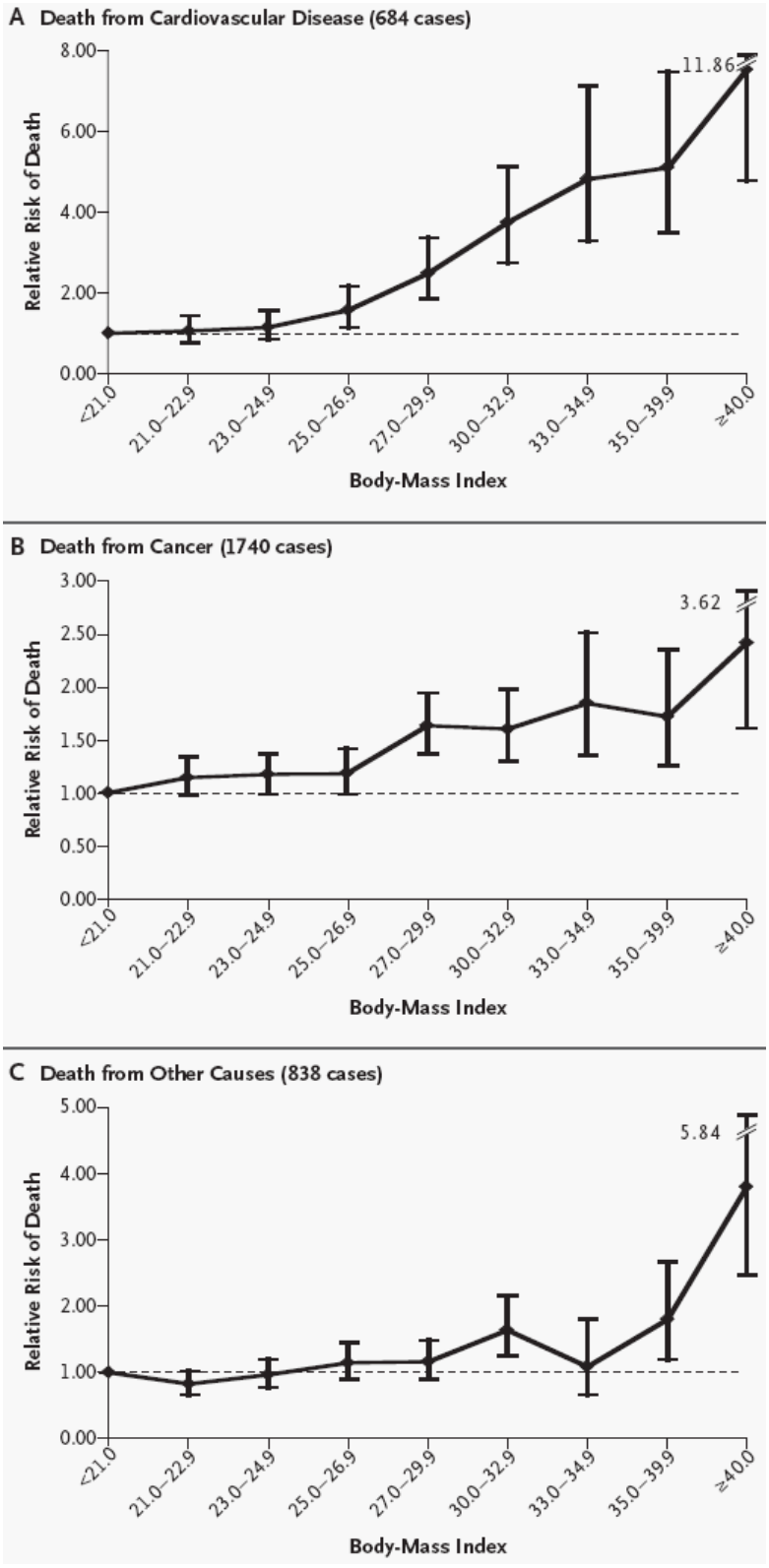
Panel A (men): Body mass index (BMI) categories: <20, 20–22.4, 22.5–24.9, 25.0–27.4, 27.5–29.9, 30.0–34.9, and ≥35 kg/m^2. Waist-to-height ratio (WHtR) categories: < 0.45, 0.45–<0.49, 0.49–<0.53, 0.53–<0.58, 0.58–<0.62, 0.62–<0.69, and ≥0.69. Waist circumference (WC) categories: 22.0–31.25, 31.5–34.25, 34.5–37.25, 37.5–40.75, 41.0–43.5, 43.75–48.0, and 48.25–62 inches. Waist-to-hip ratio (WHR) categories: <0.83, 0.83–<0.89, 0.89–<0.94, 0.94–<0.99, 0.99–<1.03, 1.03–<1.11, and ≥1.11.

Panel B (women): BMI categories are the same as for men. Women's WHtR categories: <0.42, 0.42–<0.47, 0.47–<0.52, 0.52–<0.57, 0.57–<0.61, 0.61–<0.68, and ≥0.68. Women's WC categories: 20.0–27.0, 27.25–30.0, 30.25–33.25, 33.5–36.5, 36.75–38.75, 39.0–43.75, and 44.0–55.0 inches. Women's WHR categories: <0.72, 0.72–<0.77, 0.77–0.82, 0.82–<0.86, 0.86–<0.89, 0.89–<0.95, and ≥0.95.

Source: Gelber et al (2008).

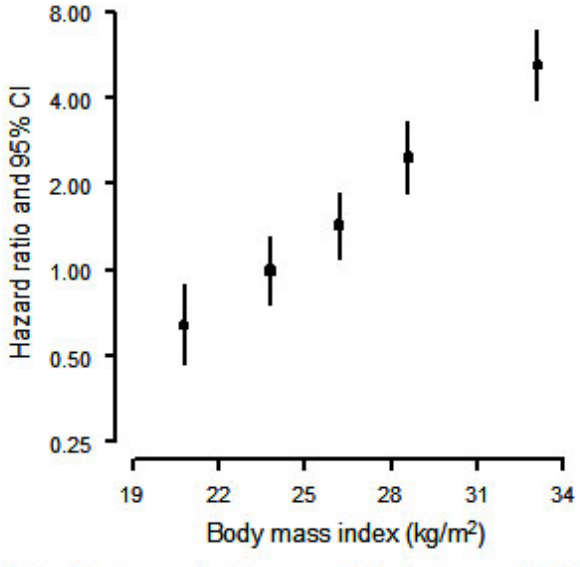
Overweight and obesity, as classified by BMI, have been linked in large cohort studies and meta-analyses of cohort studies to increased risk of cardiovascular disease (including ischaemic heart disease and ischaemic stroke), type 2 diabetes, some cancers (including colorectal, post-menopausal breast, and endometrial cancers), and osteoarthritis (Willett et al 1999, James et al 2004). The association of BMI with many of these diseases appears continuous from BMI levels of about 20–21 kg/m^2 (Figures 2 and 3).

Figure 2: Multivariate relative risks of death from cardiovascular disease (Panel A), cancer (Panel B), and other causes (Panel C) according to body mass index among women who have never smoked



Source: Hu et al (2004).

Figure 3: Association between incidence of diabetes and body mass index



Note: CI = confidence interval; kg/m^2 = kilogram per square metre.

Source: Ni Mhurchu et al (2006).

Limitations to using body mass index in clinical practice

BMI has three limitations in clinical practice.

- BMI does not distinguish between fat and lean mass.
- BMI hides any ethnic differences in the ratio of fat to lean mass.
- BMI does not provide information about the distribution of body fat.

BMI does not distinguish between fat and lean mass: Individuals with the same BMI may have different proportions of body fat (World Health Organization 2000, World Health Organization Expert Consultation 2004, Rush et al 2004, Depres and Tchernof 2007). For instance, athletes and sports people with BMIs greater than 25 kg/m^2 will be categorised as overweight even though they have normal, or even low, body fat content. Women have also been shown to have more fat than men at equivalent BMIs, and with age, men and women lose lean tissue, so an older person will have more fat than a younger person with the same BMI (James et al 2004, National Institute for Health and Clinical Excellence 2006).

There may be ethnic differences in the ratio of fat to lean mass: The ratio of fat to lean mass may be relatively higher in South Asian population groups than in other population groups, with Chinese population groups falling between Indian and European groups. In contrast, Māori and Pacific populations tend to have a higher proportion of lean body mass (Swinburn et al 1999, Deurenberg et al 2002). This means individuals with similar BMIs who are from different population subgroups may have different levels of adiposity.

BMI does not provide information about the distribution of body fat: People with central (or abdominal) fat distribution represent a subgroup with the highest risk of type 2 diabetes and cardiovascular disease (Depres and Tchernof 2007). Central deposition of fat and decreased muscle mass with age may lead to no overall change in weight or BMI, but an increase in health risk. Some ethnic groups may also be more prone to visceral or subcutaneous fat accumulation at any given BMI (James et al 2004), which may be detected using waist circumference.

Despite the three limitations, BMI is the main measure used in other international obesity guidelines (National Heart Lung Blood Institute 2000, National Institute for Health and Clinical Excellence 2006, Lau et al 2006) and is recommended by WHO for classifying overweight and obesity (World Health Organization 2000). BMI is also the measure that is linked with the broadest range of health states, can be used to estimate relative risk of in most people, and is acceptable and easy to use in clinical practice.

Body mass index thresholds for overweight and obesity

This guideline uses BMI thresholds to identify populations with higher risk of some co-morbid diseases, such as cardiovascular disease, cancer, and diabetes, not to classify 'fatness'. WHO recommends the cut-off thresholds for classification of weight shown in Table 2.

Table 2: Principal World Health Organization body mass index thresholds

Classification	Body mass index (kg/m^2)	Risk of health conditions
Normal weight	18.5–24.9	Average risk
Overweight	25.0–29.9	Increased risk
Obese		Substantially increased risk
Class I	30.0–34.9	Moderate risk
Class II	35.0–39.9	Severe risk
Class III	≥ 40.0	Very severe risk

It has been suggested higher BMI thresholds be used for Māori and Pacific populations, based on evidence that Māori and Pacific populations have lower levels of body fat compared with the non-Māori and non-Pacific populations at equivalent BMIs (Swinburn et al 1999, Rush et al 2004). For the New Zealand Health Survey, the Ministry of Health initially used higher thresholds for Māori and Pacific, but is now using the same WHO thresholds for all participants (Ministry of Health 2008c). The rationale for the change was that health risk is continuous and begins below the BMI threshold of 25 kg/m^2, and there is no evidence to show that lower levels of body fat in different groups might translate into lower levels of risk. The opposite is more likely as a comparison of Australian, New Zealand, and Asian cohorts with North American and European cohorts all showed similar strengths of association for ischaemic heart disease despite different ethnicities (Ministry of Health and University of Auckland 2003, Asia Pacific Cohort Studies Collaboration 2004). Similarly, some evidence suggests Māori and Pacific adults still have a higher risk of diabetes after adjusting for BMI (Sundborn et al 2007).

The most recent WHO Expert Consultation re-examined the use of different thresholds in different ethnic groups, especially with respect to Asian populations. Again different (lower) BMI thresholds have been suggested for Asian populations based on evidence of a higher percentage of body fat at a given BMI and higher prevalence of co-morbid disease in Asian populations at a lower BMI than in European populations (National Institute for Health and Clinical Excellence 2006, Huxley et al 2007). However, the evidence is drawn from cross-sectional studies with all Asian population groups (South Asian, South East Asian, Chinese, Japanese, and Iranian) grouped together, and the WHO Expert Consultation noted that possible BMI thresholds varied across Asian ethnicities. The lack of consistency is supported by findings from prospective cohort studies in specific Asian ethnicities. For instance, mortality from cardiovascular disease was lowest in those with a BMI of 23–25 kg/m^2 and increased from a BMI of 25 kg/m^2 or more in a Japanese cohort of men and women (Hozawa et al 2008), whereas the relationship between BMI and mortality from ischaemic heart disease was linear from a BMI of 20 kg/m^2 in a cohort Chinese men (Chen et al 2006). The same cohort also reported that stroke mortality only increased from a BMI over 25 kg/m^2 (Zhou et al 2008). Epidemiological research on South Asian population groups in developed countries and their specific health risks is lacking.

The evidence does not offer clear guidance on BMI thresholds for different Asian ethnicities. Consequently, the WHO Expert Consultation has retained the WHO BMI thresholds of 25 kg/m^2 (overweight) and 30 kg/m^2 (obesity) for all adults (Table 2).

Waist circumference

Rationale for using waist circumference to measure adiposity

The amount of intra-abdominal or visceral fat has been associated with a range of abnormalities and diseases (James et al 2004), particularly type 2 diabetes and cardiovascular disease (Depres and Tchernof 2007, National Guideline Clearinghouse 2005 (revised 2007).). Waist circumference is a reasonable indicator of abdominal fat (James et al 2004). It differs from other measures of abdominal fat, such as the waist-to-hip ratio, in that it is an index of the absolute amount of abdominal fat. Other measures provide an estimate of the relative accumulation of abdominal fat (Depres and Tchernof 2007), so a loss of abdominal fat might not be reflected by a change in such measures whereas a loss of abdominal fat will be reflected by a change in waist circumference.

A systematic review of 120 studies found that the association between waist circumference and cardiovascular disease and diabetes was not dependent on the measurement protocol (Ross et al 2008). Therefore use the WHO criteria, where waist circumference is measured using the midpoint (ie, the midpoint between the lowest rib and the iliac crest) as boney reference points.

Waist circumference thresholds for overweight and obesity

WHO has suggested thresholds for waist circumference (Table 3). Body composition studies indicate that some ethnic groups are more prone to abdominal fat accumulation. Higher ratios of abdominal fat have been found in Indian males in New Zealand compared with Pacific and Pākehā males (Rush et al 2004), which supports findings in studies of South Asian population groups in other developed countries (National Institute for Health and Clinical Excellence 2006). Therefore, it has been suggested that different thresholds for waist circumference be used for Asian ethnic groups. Most recently, a systematic review of 21 cross-sectional studies found that BMI might may not be as good a discriminator of prevalent diabetes and hypertension as waist circumference in Asian populations (Huxley et al 2007) and suggested that the optimal thresholds for waist circumference should be lower for Asian populations than those suggested by WHO. However, all Asian population groups (South Asian, South East Asian, Chinese, Japanese, and Iranian) were grouped together because there was not enough data to produce ethnic-specific thresholds. More importantly perhaps, the association between measures of weight and the development of disease over time could not be examined. This limitation is particularly relevant as diabetes itself may influence body weight, thus confounding assessments of risk developed from cross-sectional data.

Table 3: World Health Organization waist circumference thresholds

Risk of metabolic complications	Waist circumference threshold	
	Men	Women
Average risk	< 94 cm	< 80 cm
Increased risk	94–102 cm	80–88 cm
Substantially increased risk	> 102 cm	> 88 cm

WHO has suggested that an individual's relative risk may be more accurately classified using both BMI and waist circumference (World Health Organization 2000). A systematic review of 32 cohort studies found similar associations between body weight as measured by BMI and waist circumference and incident diabetes (Vazquez et al 2007), but other research has also suggested that waist circumference may be a better predictor of ischaemic heart disease than BMI (James et al 2004). One review concluded that although waist circumference is a valid measure of abdominal mass and disease risk in individuals with a BMI less than 35 kg/m^2, it adds little to the absolute measure of risk provided by BMI at a BMI of 35 kg/m^2 or higher. (National Health & Medical Research Council 2003).

Evidence for Māori, Pacific, and South Asian population groups

The 2006/07 New Zealand Health Survey found obesity had increased from the 1997 National Nutrition Survey for both men and women (Table 4), although the rate of increase in the total population may be slowing (Ministry of Health 2008c). More women than men were obese.

Table 4: Age-standardised prevalence of overweight and obesity in adults, 1997, 2002/03, 2006/07

Population	1997		2002/03		2006/07	
	Overweight (%)	Obese (%)	Overweight (%)	Obese (%)	Overweight (%)	Obese (%)
Total	33.9	18.6	34.3	24.1	34.8	25.4
Men	39.5	16.6	40.7	23.4	40.6	24.8
Women	28.6	20.6	27.9	24.8	29.0	26.0
Māori	31.5	40.7	31.2	42.1	31.7	43.2
Men	32.4	41.0	33.1	42.6	34.4	43.2
Women	30.6	40.4	29.4	41.5	29.2	43.2
Pacific	32.0	54.5	28.7	59.6	24.5	65.1
Men	42.1	49.0	31.8	58.6	27.0	63.9
Women	22.6	59.7	25.7	60.7	21.9	66.3

Note: Data from the 1997 National Nutrition Survey and 2002/03 New Zealand Health Survey used ethnic-specific body mass index thresholds. The 2006/07 New Zealand Health Survey adjusted data from the previous surveys to single body mass index thresholds to allow comparison with 2006/07 data.

Pacific men and women had the highest overall prevalence of obesity (65%) in the 2006/07 New Zealand Health Survey. Classes II and III obesity (BMI of 35–39.9 kg/m^2 and ≥ 40 kg/m^2, respectively – Table 2) showed the greatest increases in Pacific men and women between 1997 and 2006/07 (Table 5). Although the prevalence of overweight is declining in Pacific populations, overweight is shifting into the obese range and the prevalence of obesity is increasing. The decline in overweight is not explained by an increased proportion of the Pacific population being in a healthy weight range; the proportion has remained stable over the decade for Pacific men (about 8%) and declined in Pacific women (from 18% to 11%).

Table 5: Age-standardised prevalence of Classes I, II, and III obesity in adults, 1997, 2002/03, 2006/07

Adult population	1997† (%)	2002/03‡ (%)	2006/07‡ (%)
Total			
Class I obese*	13.0	16.4	16.2
Class II obese*	3.6	5.0	5.9
Class III obese*	2.0	2.7	3.3
Māori			
Class I obese*	23.7	23.1	23.9
Class II obese*	11.4	10.8	11.4
Class III obese*	5.6	7.8	7.7
Pacific			
Class I obese*	29.8	29.2	31.0
Class II obese*	12.0	16.0	18.1
Class III obese*	12.7	14.5	16.0

* Class I body mass index (BMI) 30–34.9 kg/m^2; Class II BMI 35–39.9 kg/m^2; Class III BMI ≥ 40 kg/m^2.

Māori men and women also had a high prevalence of obesity (43%) and were 1.7 times more likely to be obese than the total population in 2006/07. There was no significant increase in obesity in Māori men or women between 1997 and 2006/07 (men 41–43%; women 40–43%), although there was a small increase in Class III obesity (BMI ≥ 40 kg/m^2). The prevalence of overweight has remained relatively stable over the same period (about 32%).

The most deprived neighbourhoods had a higher prevalence of obesity than the least deprived neighbourhoods. The proportion of men and women who were obese was similar in the top four quintiles of neighbourhood deprivation (men, 22–25%; women, 20–27%) but increased in the lowest quintile, especially for women (men, 35%; women, 40%).

The prevalence of obesity increases with age, and the highest rates for the total population were observed in the men aged 55–64 and women aged 65–74.

The New Zealand Health Survey 2006/07 found a lower prevalence of obesity among Asian populations (11%) but the survey does not provide more specific information about the prevalence of overweight and obesity in specific Asian ethnicities (Ministry of Health 2008c). The Asian Health Chart Book, using 2002/03 New Zealand Health Survey data, estimated the prevalence of obesity in Indian males and females at 34% and 53% respectively (Ministry of Health 2006). The subgroups categorised as 'Indian' were Indian, Bengali, Fijian Indian, Gujarati, Tamil, Punjabi, Sikh, Anglo-Indian, and Other Indian. However, this report defined obesity as a BMI of 25.0 kg/m^2 or higher, and recognised such a threshold might not be appropriate for Indian ethnic groups. Using WHO thresholds, the Asian Health Chart Book reported the prevalence of overweight for Indian men and women was 27% and 34% respectively (95%CIs 15–39% and 26–51%), while the prevalence of obesity in Indian men and women was 7% and 15% respectively (95%CIs 1–14% and 8–22%). These data compare with age-standardised prevalence of overweight in the total population in the 2002/03 New Zealand Health Survey of 40% and 27% in men and women respectively, while the prevalence of obesity was 19% and 21% (Ministry of Health 2008c). However, the data from the Asian Health Chart Book were not standardised for age and sex, included respondents aged 15 and older, and had very wide confidence intervals around the point estimates. Therefore, there is considerable uncertainty about the prevalence of overweight and obesity in South Asian population groups in New Zealand.

The main issues revealed by an inequalities analysis are the higher prevalence of obesity in Pacific and Māori populations, areas of high neighbourhood deprivation, women, and older age age groups. A further issue is the absence of accurate data on the prevalence of overweight and obesity in South Asian population groups.

Assessment

Overweight and obesity are associated with increased risk of several co-morbidities. These co-morbidities have the potential to adversely affect almost every body system. Table 6 summarises many of the co-morbidities of obesity. The disease outcomes documented as the most important in terms of morbidity and mortality are those related to cardiovascular disease and diabetes. Obese individuals have two to three times the relative risk of cardiovascular disease and diabetes than individuals with a normal bodyweight. Also important is the increased risk of specific cancers. Therefore, recommendations for clinical assessment and investigations in these guidelines are aligned with those in the *New Zealand Cardiovascular Guidelines Handbook* (New Zealand Guidelines Group 2009). However, clinical judgement should guide the assessment of individuals who fall below the BMI thresholds. For example, a person's abdominal girth may suggest a high ratio of fat to lean body mass (and therefore greater health risk) despite their BMI being under 25 kg/m^2.

Recommendations

Ensure every clinical assessment includes:

- confirmation of age, sex, ethnicity, and smoking status
- the family history of cardiovascular disease, type 2 diabetes, and genetic lipid disorder
- BMI and waist circumference
- the average of two seated blood pressure measures and pulse.

Give adults a cardiovascular disease risk assessment and diabetes screen, if they:

- are Māori, Pacific, or from the Indian subcontinent: at age 35 for men and 45 for women
- have known cardiovascular disease risk factors or are at high risk of diabetes: from age 35 for men and 45 for women
- are asymptomatic and have no known risk factors: at age 45 for men and 55 for women
- have diabetes mellitus, in which case cardiovascular disease risk is assessed annually.

Use, to assess cardiovascular disease risk and diabetes status, the laboratory tests:

- fasting lipid profile (total cholesterol, triglycerides, low density lipoprotein (LDL) cholesterol, high density lipoprotein (HDL) cholesterol, total cholesterol-to-HDL cholesterol ratio)
- fasting glucose (as discussed in *New Zealand Cardiovascular Guidelines Handbook*) and a glucose tolerance test if indicated (see Table 7).

Assess cardiovascular disease risk by determining the five-year cardiovascular disease risk using the absolute risk approach and recommendations for repeat risk assessment in *New Zealand Cardiovascular Guidelines Handbook*. If the person's five-year cardiovascular disease risk is:

- 15% or higher or the person is diabetic or on lipid-lowering or blood pressure medication, assess the person's cardiovascular disease risk every year
- 10–15%, assess the person's cardiovascular disease risk again in two years' time
- 5–10%, assess the person's cardiovascular disease risk again in five years' time
- under 5%, assess the person's cardiovascular disease risk again in 10 years' time.

Good practice points

- Develop proactively your cultural competence to facilitate effective communication.
- Develop your relationship with the person before doing anything else to ensure you can empathise with the person's cultural, social, and socioeconomic circumstances.
- Develop your relationship with the person by:
  - assessing factors such as cultural engagement, the role of the person's family/whānau (immediate and extended) and wider community, and the person's employment, living circumstances, health literacy, values, and beliefs
  - recognising that differences in views of health and wellbeing may mean you need to work alongside other providers (eg, marae-based workers or church ministers) to improve health outcomes.

Clinical judgement

- Include additional tests or assess factors in addition to those listed above based on the patient's history.
- Base your decision to undertake additional tests on your clinical judgement of each case, rather than performing them routinely.

Physical examination

- Examine the person's head and neck, including the thyroid if indicated.
- Make a full cardiovascular examination, looking for evidence of cardiomegaly and vascular bruits.
- Examine the person's abdomen for hepatomegaly and abdominal pannus.
- Examine the person's extremities for oedema, joint deformities, cellulitis, and osteoarthritic changes.
- Examine the person's breasts, especially in post-menopausal women.

Laboratory tests and radiological procedures

- Liver function blood tests and, if there is a suspicion of liver disease, an ultrasound.
- A mammography or colonoscopy, if there is a suspicion of post-menopausal breast cancer or colon cancer.
- An exercise electrocardiogram to investigate ischaemic cardiovascular disease.
- A sleep study for sleep apnoea, if the person is a regular snorer.

Table 6: Risk factors and co-morbidities of overweight and obesity

Body system	Risk factor or co-morbidity
Cardiovascular	Hypertension and hyperlipidaemia Coronary heart disease, including coronary artery disease Congestive heart failure and left ventricular hypertrophy Thrombophlebitis and deep venous thrombosis Various veins and venous stasis ulcers Pulmonary embolism
Endocrine	Insulin resistance and type 2 diabetes Polycystic ovary syndrome
Gastrointestinal and hepatobiliary	Abdominal hernia Gastro-oesophageal reflux disease Gallstones Non-alcoholic fatty liver disease Colon cancer
Genito-urinary	Stress urinary incontinence Urinary tract infections Prostate cancer
Musculoskeletal	Cellulitis and carbuncles Carpal tunnel syndrome Degenerative joint disease (eg, osteoarthritis) Gout
Neurologic and psychiatric	Anxiety and depression Idiopathic intra-cranial hypertension Stroke
Obstetric and gynaecologic	Infertility Miscarriage Gestational diabetes
Pulmonary	Dyspnoea Asthma Obesity hypoventilation syndrome Obstructive sleep apnoea

Source: Brethauer et al (2006), Haslam et al (2006), and Hramiak et al (2007).

Table 7: What to do following fasting plasma glucose test results

Fasting plasma glucose result	Action	Interpretation
≤ 5.4 mmol/L	If risk factors for diabetes present, retest in five years or earlier	Result normal
5.5–6.0 mmol/L	Request an oral glucose tolerance test in high-risk groups[†]	Result may be normal, but some patients show diabetes or impaired glucose tolerance in an oral glucose tolerance test
6.1–6.9 mmol/L	Request an oral glucose tolerance test	Diagnostic of impaired fasting glucose. Diabetes or impaired glucose tolerance have not been excluded
≥ 7 mmol/L	Repeat fasting plasma glucose test	Twice on two different days[‡] diagnostic of diabetes

Note: mmol/L = millimol per litre.

[†] Being of non-European ethnicity, having first-degree relatives with diabetes, or having a history of gestational diabetes.

[‡] Unless there is unequivocal hyperglycaemia with acute metabolic decompensation or obvious symptoms of thirst or polyuria, a diagnosis of diabetes should always be confirmed by repeating the fasting plasma glucose test.

Note: The assessment of cardiovascular risk is explained in greater detail in *New Zealand Cardiovascular Guidelines Handbook* (New Zealand Guidelines Group 2003b, New Zealand Guidelines Group 2009). The handbook includes recommendations for assessing cardiovascular risk and diabetes screening and managing cardiovascular risk factors (including type 2 diabetes and smoking cessation).

Rationale for inclusion in assessment

Cardiovascular disease

Excess body weight is associated with high levels of blood pressure, cholesterol, and glucose. These factors are all important causes of cardiovascular disease (Haslam et al 2006, Hramiak et al 2007, Ishikawa-Takata et al 2002, James et al 2004, Pi-Sunyer 1993, Williams et al 2007). Therefore, all these factors should be considered in an assessment of people who are overweight or obese.

Observational data have demonstrated increases in blood pressure with increasing BMI that persist after controlling for potential confounders. In addition, weight loss is associated with a reduction in blood pressure. It is difficult to quantify this effect from trials, because the weight-loss interventions usually involve dietary changes that could also influence blood pressure in addition to weight loss alone. However, it has been suggested that a 1 kg loss in weight could lower systolic blood pressure by 0.5–1 mmHg, or a 3–9% reduction in body weight may result in an average systolic blood pressure reduction of 3 mmHg (Ministry of Health and University of Auckland 2003, Mulrow et al 2008).

Excess body weight is also associated with high blood lipid levels. Trials have demonstrated a reduction in total cholesterol and a change in cholesterol subfractions with weight loss. A meta-analysis of weight-loss trials demonstrated that for every 1 kg of weight lost, total cholesterol fell 0.05 mmol/L (Dattilo and Kris-Etherton 1992). However, the trials involved multiple dietary changes, so the cholesterol reduction due to weight loss alone is likely to have been smaller (Dattilo and Kris-Etherton 1992, Haslam et al 2006, Ministry of Health and University of Auckland 2003).

In addition to the associations between excess weight and important cardiovascular risk factors, research has also focused on the association between BMI and cardiovascular disease endpoints. A meta-analysis of cohort data in the Asia–Pacific region that included over 2 million person-years of follow-up suggested that a reduction in BMI of one unit was associated with about a 9% lower risk of ischaemic heart disease in adults aged 45–59. There were similar reductions in risk of death from ischaemic stroke (Asia Pacific Cohort Studies Collaboration 2004, Ministry of Health and University of Auckland 2003). These results concur with another systematic review of cohort studies, predominantly from North America and Europe, which demonstrated that in most studies, a one-unit decrease in BMI was associated with a 5–10% reduction in rates of ischaemic heart disease (Whitlock et al 2002).

The association between BMI and the risk of cardiovascular disease is mediated, at least in part, through risk factors such as elevated blood pressure and cholesterol. However, it is less clear whether an independent component of cardiovascular risk is mediated through BMI itself. This point is important in determining whether excess risk of disease persists for obese people after optimal treatment for hypertension and hypercholesterolemia. The information, therefore, contributes to determining whether people still need to control their weight if their blood pressure and cholesterol levels are not high.

Data from 17,500 participants in the prospective Chicago Heart Association Detection Project suggested that obese people had significantly higher risks of hospitalisation and mortality from cardiovascular disease or coronary heart disease compared with people of normal weight even when they had similar profiles for cardiovascular risk factors such as smoking, high blood pressure, and/or serum total cholesterol level (Yan et al 2006). A meta-analysis of data from 21 cohort studies involving over 300,000 participants worldwide and 18,000 coronary heart disease events indicated that adverse effects of overweight on blood pressure and cholesterol levels could account for about 45% of the increased risk of coronary heart disease. Therefore, a significantly increased risk of coronary heart disease was independent of these effects (Bogers et al 2007). However, these analyses could not control for blood glucose level, which is also associated with excess weight. Analysis of data from the NHANES I epidemiologic follow-up study demonstrated an increase in risk of coronary heart disease death among obese people compared with lean people among both diabetics and non-diabetics (Mann et al 2006).

In contrast, data from a Korean cohort showed that the association between overweight and death from atherosclerotic cardiovascular causes was attenuated to statistically non-significant levels after adjustment for blood pressure, cholesterol level, and blood glucose level (Jee et al 2006). However, the people included in this cohort had relatively low BMI levels compared with most of the ‘Western’ cohorts studied.

In summary, a clear association exists between BMI and cardiovascular risk factors such as elevated blood pressure, lipids, and diabetes. These associations contribute to the increased risk of cardiovascular disease experienced by those who are overweight and obese. It is less clear how much of an effect excess weight has on cardiovascular disease risk independent of its effects on these established risk factors.

Diabetes

Excess weight has been identified as a particularly important risk factor for developing high blood glucose and type 2 diabetes, which are associated with several adverse health outcomes, including cardiovascular disease (Carey et al 1997, Chan et al 1994, Grinker et al 2000, Haslam et al 2006, Hramiak et al 2007, James et al 2004). The development of insulin resistance is seen as the dominant mechanism whereby weight gain leads to diabetes. Weight-loss trials have also demonstrated marked improvements in diabetic status or even a return to normal glucose tolerance with weight loss (James et al 2004).

Data from eight years of follow-up of over 43,500 women in the United States (US) Nurses' Health Study (Carey et al 1997) demonstrated that after adjusting for age, family history of diabetes, smoking, exercise, and several dietary factors, the relative risk of type 2 diabetes for the 90th percentile of BMI (29.9 kg/m^2) compared with the 10th percentile (20.1 kg/m^2) was 11.2 (95% confidence interval (95%CI) 7.9–15.9). Data over a similar time-frame from over 51,500 US male health professionals found that men with a BMI of 35 kg/m^2 or more had a multivariate relative risk of type 2 diabetes of 42.1 (95%CI 22.0–80.6) compared with men with a BMI of less than 23.0 kg/m^2 (Chan et al 1994).

The association between BMI and type 2 diabetes is continuous from BMI values as low as 21 kg/m^2 (Willett et al 1999) (ie, well below the accepted cut-off values for overweight and obesity). Many non-experimental studies have indicated that a one-unit increase in BMI is associated with an increased risk of developing diabetes of approximately 20–30% (James et al 2004).

Cancer

Obese people are at increased risk of specific cancers such as endometrial, post-menopausal breast, colon, kidney, prostrate, and gallbladder cancer (Bergstrom et al 2001, Calle et al 2003, Hramiak et al 2007, James et al 2004). Of this group of cancers, the cancers that are most important in terms of their contribution to mortality are colon, post-menopausal breast, and endometrial cancer.

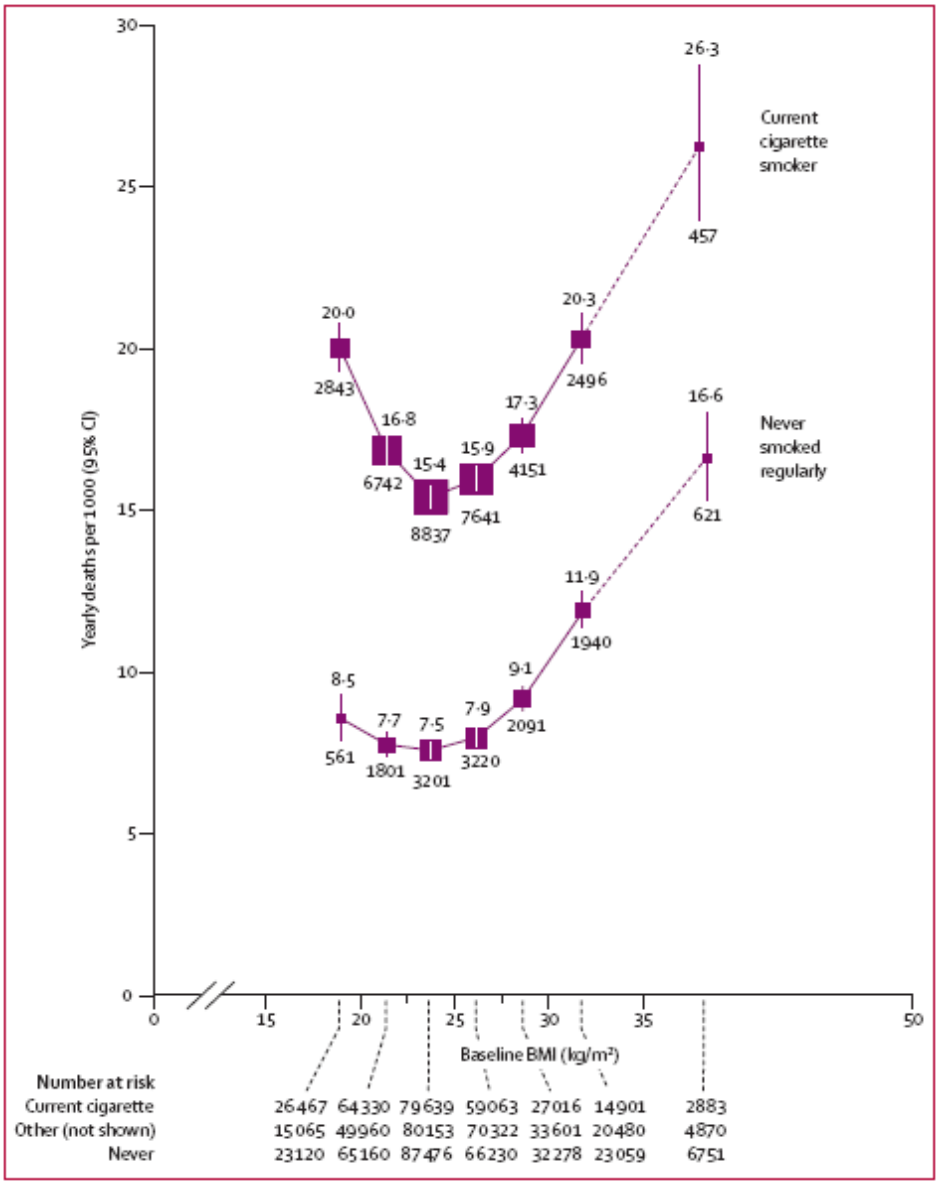
Studies indicate that the risk of both post-menopausal breast cancer and colon cancer increases about 3% with a one-unit increase in BMI, while the risk of endometrial cancer appears to increase about 10% with a one-unit increase in BMI (Bergstrom et al 2001).

While these increases are significant, they are not of the same magnitude as the increased risk of cardiovascular disease and diabetes with high BMI.

Smoking

Another important modifiable cardiovascular risk factor relevant to assessing risk in people who are overweight and obese is tobacco smoking (Kuller et al 1991, Ueshima et al 2004, Woodward et al 2005). The risk of coronary heart disease and stroke for smokers is more than double that of non-smokers (Woodward et al 2005). This increase in risk is also additive to BMI. The Prospective Studies Collaboration found current cigarette smokers have approximately double the absolute risk of mortality across the spectrum of BMI compared with people who had never smoked regularly (Prospective Studies Collaboration 2009) (Figure 4).

Figure 4: All cause mortality at ages 35–79 compared with a body mass index (BMI) of 15–50 kg/m^2, by smoking status



Note: CI = confidence interval; kg/m^2 = kilogram per square metre.

Source: Prospective Studies Collaboration (2009).

Other co-morbidities

Other adverse health outcomes associated with overweight and obesity are summarised in Table 6. Of these outcomes, obstructive sleep apnoea has a high risk of adverse outcome as it can lead to cardiac arrhythmias, nocturnal hypoxia, heart failure, pulmonary hypertension, and systemic hypertension (Haslam et al 2006, Hramiak et al 2007, James et al 2004).

Conditions such as osteoarthritis, gallstones, stress incontinence, and gynaecological abnormalities, while more frequent in people who are overweight and obese, are not generally life-threatening. However, obesity is associated with increased risk of Caesarean section, and maternal haemorrhage and infection (Chu et al 2007, Heslehurst et al 2008).

Clinical assessment

The assessment of people considered to be overweight and obese should focus on assessing risk. The greatest risks to health for overweight and obese individuals are cardiovascular disease with its associated risk factors (elevated blood pressure, lipids, and smoking), type 2 diabetes, and some cancers. Although other co-morbidities are associated with excess weight, they represent less serious risks to health.

The risk assessment determines the path of clinical management and the intensity of risk factor modification that is required. Because cardiovascular disease and diabetes are the most important co-morbidities caused by overweight and obesity, clinical assessment should initially focus on determining the presence of these illnesses. Therefore, recommendations for physical examination and laboratory investigations in these guidelines are aligned with those in the *New Zealand Cardiovascular Guidelines Handbook* (New Zealand Guidelines Group 2009). The *New Zealand Cardiovascular Guidelines Handbook* summarises guidelines on the assessment and management of cardiovascular risk (New Zealand Guidelines Group 2003a), the management of type 2 diabetes (New Zealand Guidelines Group 2003b), and smoking cessation (Ministry of Health 2007). Therefore, the *New Zealand Cardiovascular Guidelines Handbook* should be incorporated into the assessment and management of overweight and obese individuals with these conditions.

Assessment of cardiovascular risk in overweight and obesity

The *New Zealand Cardiovascular Guidelines Handbook* (New Zealand Guidelines Group 2009, New Zealand Guidelines Group 2003b) recommends that people be selected for cardiovascular disease risk assessment based on their age, sex, ethnicity, and the presence of known cardiovascular disease risk factors. Cardiovascular disease risk is then determined using an absolute risk approach and New Zealand cardiovascular risk charts. Accordingly, information should be recorded on age, sex, ethnicity, family history of cardiovascular disease, smoking status, blood pressure, fasting lipid profile with the ratio of total cholesterol to HDL, and an assessment of whether an individual has diabetes.

Individuals with a history of pre-existing cardiovascular disease (eg, previous myocardial infarction), genetic lipid disorders or diabetic nephropathy (urinary albumin

≥ 200 mg/L or albumin-to-creatinine ratio ≥ 30 mg/mmol) should be placed at highest absolute risk of a subsequent cardiovascular event with a five-year cardiovascular disease risk greater than 20% (New Zealand Guidelines Group 2009).

The cardiovascular disease risk charts should be used in assessing all other individuals to calculate their absolute risk level in terms of five-year cardiovascular disease risk as high (15–20%), moderate (10–15%), or mild (< 10%). As indicated in the New Zealand Cardiovascular Guidelines Handbook (New Zealand Guidelines Group 2009), people with an isolated, elevated, single risk factor will have a five-year risk of more than 15%. These single risk factors include total cholesterol above 8 mmol/L, total cholesterol-to-HDL ratio over 8, or blood pressure consistently over 170/100 mmHg. In addition, the Framingham equation may underestimate risk in some people. This underestimation means certain groups should be moved up one risk category (5%). These groups are people: with a family history of premature cardiovascular disease in a first-degree relative; who are Māori, Pacific, or from the Indian subcontinent; who have diabetes and microalbuminuria; have had diabetes for more than 10 years or HbA1c consistently over 8%.

Assessment for diabetes or impaired glucose tolerance in overweight and obesity

Management of Type 2 Diabetes: Best practice, evidence-based guideline recommends screening for diabetes or impaired glucose tolerance using a fasting plasma glucose test (New Zealand Guidelines Group 2003b). For an interpretation of the results and recommendations for further investigation after using the fasting plasma glucose test, see Table 7.

Lifestyle (FAB) Approach

Lifestyle approaches involve combined (eg, **f**ood, **a**ctivity and **b**ehavioural based approaches – the FAB approach) rather than single factor approaches to weight loss. Lifestyle approaches increased weight loss by between 2.5 kg and 3.5 kg in overweight and obese people compared to usual care or single factor approaches. Dietary changes involved either reduction to total energy intake through energy restriction or decreasing consumption of total and saturated fats, while increased physical activity involved moderate intensity exercise such as brisk walking three to five days per week. Where identified behavioural strategies included the standard elements discussed in behavioural approaches (eg, self-monitoring, stimulus control, problem solving). Evaluation of obesity management in primary care suggests sustained weight loss can be achieved at similar levels to that achieved in randomized trials in specialist services.

Once target weight loss is achieved, weight maintenance can be achieved with brief ongoing support in addition to maintaining dietary changes and 30–45 minutes of moderate intensity physical activity per day, five days per week. Periodic return to weight loss strategies may be required.

Recommendations

Comprehensive lifestyle approaches that incorporate diet, physical activity, and behavioural strategies (the FAB approach) should be the first treatment option for weight loss and sustained for weight maintenance.
Strength of recommendation: Strong

After successful weight loss, continued contact with advice provider maintains initial weight loss when combined with dietary change and increased physical activity.
Strength of recommendation: Strong

Good practice points

- Providers should proactively develop their cultural competence to facilitate effective communication.
- Work to develop the relationship first to ensure an empathy with the cultural, social and socioeconomic circumstances of the person.
  - Assess such factors as cultural engagement, role of family/whānau (both the immediate, extended and wider community), employment, living circumstances, health literacy, values and beliefs.
  - Recognise differences in views of health and well-being may lead to the need to work alongside other providers (eg, marae-based workers or church ministers) to improve health outcomes.
- Work with the person to understand the needs within their context ('lived realities'):
  - Identify their needs and establish realistic goals.
  - Assess and overcome barriers (eg, costs) to meeting goals.
  - Work with wider whānau/family, not just the individual.

- Be aware of and refer to local community options especially for Māori, Pacific and South Asian populations interested in weight loss, eg, marae-based healthy hapu programmes, church-based or local community group options. Consider private providers that provide FAB-based approaches through mana enhancing relationships.
- Use information resources for people interested in healthy lifestyles (in person's preferred language where possible), especially for Māori, Pacific and South Asian populations.

Weight loss

- Aim for the person to achieve a modest weekly weight loss. Target weight loss will vary by individual. For some, it may be more realistic and positive to reflect the value of maintaining weight) – health benefits start to accrue when 5–10% of initial body weight lost.
- Identify lifestyle changes person would like to start with. Encourage small changes initially to increase confidence and chance of successful change, eg, for those not regularly active suggest they start with 5 or 10 minutes a day and work up to more time each week.

Diet

(For more detail see page 40)

- Explore the meaning of food and opportunities for adapting diet, especially with Māori, Pacific and South Asian populations. Consider:
  - types of food available in different contexts, eg, marae and church-based
  - consider foods of cultural significance and seasonal availability.
- Be aware of and refer to the community options for Māori, Pacific and South Asian populations interested in weight loss, eg, marae-based healthy hapu programmes, church-based or local community group options.
- People should be encouraged to choose a dietary change they feel most able to make first.
  - Appropriate portion size is the person's cupped hand size.
  - Promote increased daily intake of fruit and vegetables.
  - For carbohydrate rich foods, encourage those that are high in fibre and have lower glycaemic index (≤ 55), eg, wholegrain or rye bread, brown or basmati rice, rolled oats.
  - Encourage smaller portion sizes, particularly of energy dense foods.
  - Reduce fat intake, especially saturated fats (eg, ghee). Saturated fats can be reduced by:
    - eating low-fat dairy products
    - eating lean meat cuts and avoiding all manufactured meat products
    - substituting margarine for butter and lard
    - using low fat cooking methods, such as grilling and baking
    - removing skin from chicken and trimming fat from meat

- eating mono- and poly-unsaturated fats instead of saturated fats such as vegetable oils (except coconut oil and palm oil), margarine, avocado
- raw nuts and nut spreads; and fish and fish oils
- reduce as much as possible and try to avoid energy dense takeaways, fast foods and fried foods
- reduce as much as possible and try to avoid energy dense snacks, eg, potato chips, snack bars, buttered popcorn, biscuits, cake, and ice cream
- reduce as much as possible and try to avoid sugary drinks, eg, fizzy drinks, fruit juice, sports drinks, and flavoured drinks/cordials.

Physical activity and exercise

(For more detail see page 52)

- Those with medical conditions or health concerns that may be exacerbated by physical activity should consult with a health care professional. For those that have not been regularly active:
  - They may start with 5 or 10 minutes a day and work up to more time each week.
  - Physical activity can be split into smaller bouts (eg, walking for 10 minutes after each meal) to reach daily activity target instead of all at once.
- For weight loss, do at least 60 minutes of moderate intensity aerobic physical activity (eg, brisk walking) on most if not all days of the week (300 minutes per week).
- Reduce screen time (watching television, videos, DVDs, playing or working on the computer or playing inactive electronic games, eg, video gaming).
- Be active in as many ways as possible (work, leisure, travel, etc) in activities the person enjoys. Consider group-based activities. Encourage using every opportunity to add physical activity into daily life, eg, climbing stairs instead of using lifts and escalators.
- Explore physical activity options and opportunities for adapting traditional and customary activities (eg, hunting). Consider group-based activities and activities the person enjoys.
- Include muscle-strengthening activities on two or more days of the week. Note that muscle-strengthening activities may result in initial weight gain due to changes in muscle mass.
- Consider referral to existing exercise or physical activity programmes (such as Green Prescription).

Behavioural strategies

(For more detail see page 57)

- Families, whānau and especially spouses, should be recruited into supporting the weight loss strategy (may also include wider whānau/family and community support).
- Identify what changes the person wishes to work on first, eg, increase physical activity.
- Use problem solving and incremental goal setting strategies to set realistic targets, eg, walking five minutes per day for first week rising to 10 minutes in second week.

- Identify activities people find enjoyable.
- Establish appropriate rewards for meeting goal (rewards need to be appropriate to persons' lived realities).
- Treat unmet goals as opportunities to learn more about the person's lived reality and the barriers they face.

Weight-loss maintenance

- Ensure people have a plan for dealing with weight regain (eg, restart the weight management programme immediately after a weight regain increases 1.5–2.0 kg over the goal weight).
- Monitor the person's weight regularly (eg, weekly).
- Encourage the person to maintain a healthy diet.
- Encourage the person to maintain at least 30–45 minutes physical activity every day.

Definition of lifestyle approach

The expression 'lifestyle approach' does not have a formal definition. In this guideline, two sources of evidence informed this section.

First, combined two or more interventions for weight loss compared with a no-treatment or an information-only control. For instance, diet and exercise when compared with a no-treatment or information-only control was considered a lifestyle approach.

Second, multifactorial approach, usually a three-factor intervention, compared with a single factor approach. For instance, approaches that included diet, exercise, and behavioural interventions compared with diet were considered lifestyle approaches. However, combined approaches that tested the isolated effect of an added intervention are not considered here, but are considered in the section addressing the effect of the isolated factor. Thus, diet and exercise compared with diet alone is considered elsewhere (see physical activity).

Evidence for lifestyle approaches

Weight loss

Evidence on the effectiveness of lifestyle approaches was obtained from one systematic review (Galani and Schneider 2007) and seven trials not included in the review that were extracted from the National Institute for Health and Clinical Excellence (NICE) guidelines (Ost and Gotestam 1976, Wood et al 1991, Djuric et al 2002, Jalkanen 1991, Bacon et al 2002, Mayer-Davis et al 2004, Blonk et al 1994).

Galani and Schneider (2007) found 30 randomised controlled trials (n = 11,579) of lifestyle interventions to prevent or treat obesity in overweight (BMI = 25–29.9 kg/m^2) or obese (BMI > 30 kg/m^2) adults. The trials had to have a minimum follow-up of one year and aim to prevent overweight participants transitioning to obesity or to treat obesity in obese participants. The search was restricted to trials published from 1995 to 2005. The review was a moderate quality systematic review funded by the Swiss Federal Office of Health with a clear objective, search strategy, and inclusion criteria. Brief tables of study characteristics were published, although these did not include adequate information to facilitate independent assessment of study quality. However, the studies were quality assessed using a modified Jadad score, with a high score of three. Six trials scored three (high quality), 16 trials scored two (moderate quality) and eight trials scored one (low quality). The additional seven trials from the NICE guideline were generally of low quality, with data reported for only completers, inadequate reporting of methods (randomisation, allocation concealment, and management of missing data), and often no baseline data included for non-completers. Data from these trials were combined with trials from Galani and Schneider (2007) using RevMan 5.

One trial to prevent obesity (Wood et al 1991) was added to the trials from Galani and Schneider (2007). Mean weight loss in the 11 trials of lifestyle interventions to prevent the transition from overweight to obesity was significantly greater in those treated with a lifestyle approach after at least a 12-month follow-up (weighted mean difference (WMD) -2.5 kg, 95%CI -3.4 kg to -1.6 kg, n = 2577, I^2 = 79%). The data were combined using a random effects model. The range of effect in the 11 trials was -6.4 kg lost to 0.4 kg gained. All but one trial reported weight loss favouring the lifestyle group. In nine trials the effects were statistically significant.

Six trials to treat obesity (Ost and Gotestam 1976, Wood et al 1991, Djuric et al 2002, Jalkanen 1991, Bacon et al 2002, Mayer-Davis et al 2004, Blonk et al 1994) were added to the trials from Galani and Schneider (2007). Mean weight loss in the 18 trials of lifestyle interventions to treat obesity was significantly greater in those treated with a lifestyle approach after at least a 12-month follow-up (WMD -3.4 kg, 95%CI -4.5 kg to -2.1 kg, n = 6416, I^2 = 92%). The data was combined using a random effects model. The range of treatment effect in the 18 trials was -11 kg lost to 1.7 kg gained. The effects were statistically significant in 12 trials.

One trial (n = 191) investigated the effect of group-based and individual-based lifestyle approaches (Ash et al 2006). Weight loss did not significantly differ between the two approaches (-2.9 kg compared with -1.8 kg, p > 0.05).

Weight-loss maintenance

Two trials investigated the effect of different approaches to preventing regain following an initial weight loss (Wing et al 2007, Svetkey et al 2008). Both compared face-to-face support with internet-based support and an information-only control arm.

Wing et al (2007) recruited 314 participants and found face-to-face support was more effective at preventing regain (mean difference -2.4 kg, 95%CI -10.8 kg to -0.002 kg, p = 0.05). Face-to-face contact was via regular monthly meetings. There was no difference between internet and control groups or face-to-face and internet groups. The programme involved regular self-monitoring, and adjustments in energy balance behaviours were indicated by a traffic light colour zone. Weekly weight was submitted using an automated telephone system. Weight maintenance was described as a weight gain of 1.4 kg or less. People meeting this target were in the 'green zone', and received immediate positive reinforcement and advice to stay with what they were doing. Participants with a weight gain of 1.4–2.2 kg were in the 'yellow zone' and were instructed to use problem-solving skills to bring their weight back to the green zone. Participants with a weight gain of 2.3 kg or more were in the 'red zone' and encouraged to restart active weight-loss efforts using either their initial approach to weight loss or a standard low calorie, low fat diet and increased physical activity.

Svetkey et al (2008) recruited 1032 participants and found brief 5–15 minute monthly face-to-face meetings were more effective than either an interactive internet and automated telephone system (-1.2 kg, 95%CI -0.4 kg to -2.0 kg, p = 0.008) or the information-only control (-1.5 kg, 95%CI -0.7 kg to -2.3 kg, p = 0.001). Face-to-face support was in addition to continued adherence to a recommended dietary pattern (Dietary Approaches to Stop Hypertension) and increasing physical activity to 225 minutes per week. Personal contact consisted of 5–15 minutes of monthly telephone contact with a trained interventionist each month except for every fourth month where participants had a 45–60 minute face-to-face interview. Each personal contact session began with self-reported weight and a review of progress since last contact, including the number of days a food diary was kept, the frequency of weighing, the average number of minutes of exercise, and progress on additional goals and action plans. Each contact provided support from the interventionist, accountability for commitments made at previous contact, and opportunities to discuss barriers to weight loss and plans for overcoming barriers.

Evidence for Māori, Pacific and South Asian population groups

None of the trials was based in or recruited from New Zealand populations. There is no direct evidence of effectiveness in Māori, Pacific, or South Asian population groups. However, Mayer-Davis et al (2004) modified a lifestyle intervention developed from the Diabetes Prevention Program trial (Diabetes Prevention Program (DPP) Research Group 2005) to address the needs of rural African American populations. Weight loss was greater among the lifestyle group than the usual care group at the 12 month follow-up, although the difference was not statistically significant (WMD -1.90 kg, 95%CI -4.31 kg to +0.51 kg).

Whether lifestyle interventions are as effective in minority populations as in majority populations has been the subject of discussion. The Trials of Hypertension Prevention trials, which were included in Galani and Schneider (2007), suggested untailored weight-loss programmes are less effective for African Americans than for the majority population – 23% of White and 13% of African Americans lost 10% of their body weight after six months, and weight loss after 36 months was lower in both African American men and women than in White American men and women. However, data from the

Trial of Non-Pharmacologic Interventions in the Elderly (also included in Galani and Schneider 2007) found African Americans had lost significantly less weight at six months compared with White Americans, although this pattern was not present after six months. Svetkey et al (2008), whose recruited about 40% African Americans in their study, found no significant difference between effects in African Americans and the majority population. Although it is not appropriate to directly extrapolate evidence from other minority population groups to Māori, Pacific, and South Asian population groups in New Zealand, the above discussion provides a useful insight that lifestyle interventions can be tailored to different population groups. The main issue is the lack of evidence relating to the lifestyle approaches for New Zealand Māori, Pacific, and South Asian population groups.

Evidence statements

Lifestyle approaches (dietary change, exercise ± behavioural interventions) increase weight loss by 2.5 kg (95%CI 1.6 kg to 3.4 kg) in overweight people (BMI = 25–29.9) at one year or more.

Quality of evidence	**Moderate**
Study limitations	Some limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision‡

* Methods not reported in Galani and Schneider (2007), but scored for quality using modified Jadad scale; median score with 2 out of 3, and more than 20% loss in two studies.

‡ Random effects model used for analysis due to heterogeneity.

Lifestyle approaches (dietary change, exercise ± behavioural interventions) increase weight loss by 3.5 kg (95%CI 2.4 kg to 4.5 kg) in obese persons (BMI ≥ 30) at one year or more.

Quality of evidence	**Moderate**
Study limitations	Some limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision‡

* Methods not reported in Galani and Schnieder (2007), but scored for quality using modified Jadad scale; median score with 2 out of 3, and more than 20% loss in two studies.

‡ Random effects model used for analysis due to heterogeneity.

A group-based lifestyle approach did not significantly decrease weight loss at one year or more compared to an individual-based intervention (-2.9 compared with -1.8 kg, $p > 0.05$).

Quality of evidence	**Low**
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Imprecision likely‡

* Unclear allocation concealment, differential loss, not analysed using intention to treat.

‡ One trial.

After successful weight loss, monthly brief face-to-face support in addition to a comprehensive lifestyle programme reduced weight regain by -1.5 kg (95%CI -0.7 kg to -2.3 kg) at 30 months compared with an information-only approach and by -1.2 kg (95%CI -0.4 kg to -2.0 kg, $p = 0.008$) compared with an internet-based approach.

Quality of evidence	**High**
Study limitations	No important limitations
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision‡

‡ Unable to incorporate trials into meta-analysis.

Dietary Approaches

Dietary approaches to weight loss involve nutrition advice with recommendations made to:

- reduce total energy intake (by decreasing the overall consumption of foods and beverages)
- modify the types of foods consumed (eg, by reducing the proportion of fat in the diet).

Over 12 months nutrition advice increased weight loss by approximately 5 kg in overweight and obese people compared with usual care or no dietary advice. All types of diets (low energy, very low energy, low glycaemic index, and modified macronutrient) produced similar weight losses (about 4 kg). However, the amount of weight lost will depend on the individual, and may range from weight maintenance to larger weight losses (over 10 kg). The type of diet recommended should be tailored to the individual and their family/whānau, taking into account co-morbidities, income, and access to advice. The effect of the different dietary approaches on the long-term maintenance of weight loss could not be determined, although once weight loss is achieved, brief ongoing dietary advice may prevent weight regain.

Recommendation

Nutrition advice for weight loss should be offered to people who are overweight or obese. The following diets (low energy, very low energy, low glycaemic index, and modified macronutrient) are similarly effective for weight loss provided they result in reduced energy intake. Type of diet should be tailored to the individual and family preference.
Strength of recommendation: Strong

Good practice points

- Encourage a lifestyle (FAB) approach. Monitor very low energy diets frequently to ensure nutritional adequacy.
- Remember that high protein and low carbohydrate diets containing meal replacements may be expensive.
- Remember that very low and very high carbohydrate diets (eg, the Atkins and Ornish diets) are difficult to adhere to over the long term.

Types of dietary approaches

Dietary approaches are not standardised into a formal taxonomy. Analysis of dietary trials can be complicated by this lack of a taxonomy; for instance, the Atkins diet has been described as both a high fat and a high protein diet, making its allocation into a single dietary category difficult. Studies in this guideline were categorised according to their best fit with one of the definitions in Table 8.

Table 8: Categorisation of studies in this guideline

Category	Definition of studies included in category
1 Nutrition advice	Studies comparing nutrition advice with control (no nutrition advice, usual care, or minimal intervention).
2 Dietitian or nutritionist compared with non-expert delivery of dietary advice	Studies comparing the same nutrition advice delivered by people with different levels of expertise: a dietitian or nutritionist; and a trained non-expert (such as a nurse or general practitioner).
3 Low energy diets	Studies comparing a low energy diet (1000–1600 kcal or 4200–6720 kJ per day) with control (no energy restriction, usual care, or minimal intervention).
4 Very low energy diets	Studies comparing a very low energy diet (< 1000 kcal or < 4200 kJ per day), which may include meal replacements, with a non-active control.
5 Low glycaemic index/load diets	Studies comparing a low glycaemic index/load diet with a high glycaemic index/load diet or with a non-active control. A low glycaemic index is defined as 55 or under; medium as 56–69, and high as 70 or higher.
6 Modified macronutrient diets	Studies comparing one type of modified macronutrient diet with another type of modified macronutrient diet or a non-active control. Altered macronutrient diets were defined as differing substantially from the acceptable distribution ranges in New Zealand: 50–55% total energy (TE) from carbohydrate, 20–35% from total fat, and 15–25% from protein. Five subcategories were defined as: • low carbohydrate: ≤ 40% TE carbohydrate • low fat: ≤ 10% TE fat • high protein: ≥ 35% TE protein • high carbohydrate: ≥ 65% TE carbohydrate • New Zealand Healthy Eating Diet: 50–55% TE from carbohydrate, 20–35% from total fat, and 15–25% from protein (with no specific energy restriction).
7 Head-to-head studies of type of diet	Studies comparing one type of diet with another (ie, across categories 3 to 6); for example, a very low energy diet compared with a low glycaemic index diet.

Evidence for dietary approaches

Nutrition advice

Evidence about the effectiveness of nutrition advice was obtained from one systematic review (Dansinger et al 2007) and one trial (Anderssen et al 1996).

Dansinger et al (2007) included 46 trials (n = 11,853). The included trials were diverse in terms of methodology and participants. Outcomes were body weight and BMI. Interventions focused on lowering fat or energy intake and lasted 12 weeks or longer. Follow-up ranged from six to 60 months (five years). The authors described two-thirds of the trials as being of a fair quality.

Anderssen et al (1990) compared dietary advice with delayed intervention control in a predominantly male population (90%) aged 41–50. Follow-up was at 12 months (the intervention lasted nine months). Quality was moderate, but the trial was small (n = 98).

Results for Dansinger et al (2007) and Anderssen et al (1990) were consistent, but they could not be combined in a meta-analysis due to their different outcome measures. Dansinger et al (2007) found BMI decreased significantly in groups receiving nutrition advice compared with usual care groups. Mean net changes were -1.88 kg/m^2 (95%CI -2.29 kg/m^2 to -1.46 kg/m^2) at 12 months; -0.63 kg/m^2 (95%CI -1.99 kg/m^2 to +0.83 kg/m^2) at 24 months; and -1.41 kg/m^2 (95%CI -1.99 kg/m^2 to -0.83 kg/m^2) at 48 months. Similarly, Anderssen et al (1990) found the decrease in body weight was significantly more in the nutrition advice group than in the control group at 12 months (mean difference -5.1 kg, 95%CI -6.7 kg to -3.5 kg). Dansinger et al (2007) also assessed weight maintenance during maintenance phases (six to 60 months). BMI increased +0.03 kg/m^2 per month more in nutrition advice groups than in control groups. This rate was calculated to return participants to their baseline weight after approximately 5.5 years.

Nutrition advice may need to account for whether a person has had previous, unsuccessful weight-loss attempts or weight loss with subsequent weight regain. Previous successes in weight loss are likely to be repeated because the rate of weight loss on repeated attempts to lose weight has been shown to be similar to that on the first attempt (Li et al 2007). Weight cycling (repeated bouts of weight loss and regain) has been linked to increased risk of all-cause mortality and mortality from coronary heart disease (Brownell and Rodin 1994, Williamson 1996). However, a systematic review on the impact of intentional weight loss and mortality among initially healthy participants could not conclude whether intentional weight loss increased or decreased mortality (Simonsen et al 2008). Two of the nine included studies found mortality decreased, three found mortality increased, and four found no clear association. Successful weight loss has been associated with seven factors: having greater initial weight loss; reaching a self-determined goal weight; having a physically active lifestyle; having a regular meal pattern, including breakfast; and following healthier eating, control of over-eating, and self-monitoring behaviours (Elfhag and Rossner 2005).

Dietitian or nutritionist compared with non-expert delivery of dietary advice

Evidence about the effectiveness of nutrition advice delivered by a dietitian or nutritionist compared with advice delivered by a trained practice nurse or general practitioner was obtained from two trials (Pritchard et al 1999, Dale 2007).

Pritchard et al (1999) compared dietitian/nutritionist advice with dietitian/nutritionist advice plus general practitioner support (n = 273), and followed-up at 12 months. The trial quality was low.

Dale (2007) compared dietitian/nutritionist advice with nurse advice and followed-up at 24 months. Two-hundred participants who had lost 5% or more of their bodyweight over the previous six months were included. The quality of the study was moderate to high.

Pritchard et al (1999) assessed weight loss and Dale (2007) assessed weight maintenance, so the outcomes between the studies differed. Therefore, these trials were not combined in meta-analyses. Pritchard et al (1999) found no significant difference between groups for percentage of body weight lost at 12 months (mean difference +0.7%, 95%CI -0.42% to +1.82%). Dale (2007) found no significant difference between groups for the maintenance of body weight lost at 24 months (mean difference 0.0 kg, 95%CI -1.8 kg to +1.8 kg, p = 0.98).

Low energy diets

Evidence about the efficacy of low energy diets was obtained from two trials (Wood et al 1988, Jehn et al 2006).

Wood et al (1988) was a moderate quality trial where participants were individually counselled to reduce energy intake, but not to increase physical activity.

Jehn et al (2006) was a moderate quality trial that used a low energy version of the DASH diet (a tailored low energy diet focusing on healthy eating) with all food supplied for the first nine weeks.

The results from the two trials were combined in a meta-analysis using RevMan 5. There was no significant difference between low energy diet and control groups, although the direction of effect favoured the low energy diet (WMD -4.41 kg, 95%CI -10.29 kg to +1.47 kg, n = 148, I^2 = 98%, random effects).

Very low energy diets (including meal replacements)

The evidence about very low energy diets (including meal replacements) was obtained from one systematic review (Franz et al 2007) and one trial (Stenius-Aarniala et al 2000).

The trials in the review compared very low energy diets with other weight-loss diets consisting of regular food. Franz et al (2007) included seven trials in the meal replacement/very low energy section (n = 802), and was current to September 2004. Trials were included in the study if the primary outcome was weight loss in overweight or obese adults and there was at least 12 months of follow-up. Included trials had to use meal replacements for at least two meals per day as an adjunct to a very low energy diet (providing ≤ 800 kcal per 3360 kJ per day). Interventions lasted 12–40 weeks, follow-up was 12–27 months, and trials were low quality.

Stenius-Aarniala et al (2000) compared a very low energy diet with an attention control. This very low quality trial that evaluated the effect of weight loss in 38 obese people with asthma, and compared a very low energy diet (420 kcal or 1764 kJ per day), including meal replacements, with education about asthma and allergy (contact was equal for both groups).

Franz et al (2007) did not provide enough data to combine the trials in a meta-analysis with Stenius-Aarniala et al (2000). However, the direction of the effect was consistent; Franz et al found participants using very low energy diets lost significantly more weight than those in control groups (-3.8 kg ± 6.3 kg, $p < 0.05$, random effects). Stenius-Aarniala et al also found those in the very low energy diet group lost significantly more weight than those in the control group (-13.40 kg, 95%CI -18.43 kg to -8.37 kg).

Low glycaemic index/load diets

There was no direct evidence for the effect of low glycaemic index diets, because no studies compared a low glycaemic index diet with a control. Evidence about the efficacy of low glycaemic index/load diets was obtained from one systematic review (Thomas et al 2007) and one trial (Das et al 2007).

Thomas et al (2008) included trials that compared a low glycaemic index/load diet with a higher glycaemic index diet. Intervention periods had to last at least two weeks, and populations with diabetes were excluded. Intervention periods ranged from five weeks to six months, and follow-up from five weeks to 12 months. The low glycaemic index diet was 20% lower in carbohydrate and 10% higher in fat and protein than the high glycaemic index diet, although the biggest difference between diets was reported to be glycaemic load (45.4 compared with 118.3). Included trials were small (four trials; n = 163) and of low quality.

Das et al (2007) compared a low glycaemic load diet to a high glycaemic load diet. All food was provided for the first six months, followed by six months of dietary intervention with self-selected food from an individualised plan. Both diets were approximately 30% energy reduced relative to baseline energy requirements. The trial was small (n = 34) and of low quality.

Thomas et al (2008) and Das et al (2007) were combined in a meta-analysis. Participants using low glycaemic index diets lost significantly more weight (WMD -0.98 kg, 95%CI -0.11 kg to -1.85 kg, n = 197, $I^2 = 0\%$) at 12 months. The degree of weight loss with the low glycaemic index diet was 13 kg over 12 months in Thomas et al (2008); Das et al (2007) did not provide this information.

Modified macronutrient diets

Each type of modified macronutrient diet (low carbohydrate, low fat, high protein, high carbohydrate, and the Healthy Eating diet) was compared with all other types of modified macronutrient diets combined in a meta-analysis using RevMan 5. We found all types of modified macronutrient diets appeared to be effective over the long term and produce weight losses of 2–5 kg over 12 months, but no single modified macronutrient diet appeared more effective than any other. More extreme types of modified macronutrient diets (eg, the Atkins and Ornish diets) may be difficult to adhere to (Dansinger et al 2005).

Low carbohydrate compared with other modified macronutrient diets: The effectiveness of low carbohydrate diets compared with other modified macronutrient diets was obtained from one systematic review (Nordmann et al 2006) and three trials (Gardner et al 2007, McAuley et al 2006, Dansinger et al 2005).

Nordmann et al (2006) included three trials with 12 months follow-up that compared low carbohydrate with low fat diets The trials were small (total n = 195) and of low quality.

Gardner et al (2007), McAuley et al (2006), and Dansinger et al (2005) compared low carbohydrate diets with Healthy Eating diets. The trials were of moderate quality. The trials were combined in a meta-analysis. There was no significant difference between a low carbohydrate diet and any other modified macronutrient diet (WMD -0.53 kg, 95%CI -2.37 kg to +1.30 kg, n = 494, I^2 = 55%, random effects).

Low fat compared with other modified macronutrient diets: The effectiveness of low fat diets compared with other modified macronutrient diets combined was obtained from two systematic reviews (Nordmann et al 2006, Pirozzo et al 2008).

Nordmann et al (2006) compared low fat with low carbohydrate diets, and included three low to moderate quality trials with 12 months follow-up. The trials were small (total n = 275). Pirozzo et al (2008) included five trials that compared low fat with other predominantly low carbohydrate diets at 12 months. The trials were also low quality and small (median n = 106). The trials were combined in a meta-analysis. There was no significant difference in weight loss between the low fat diet and any another modified macronutrient diet (WMD +1.06 kg, 95%CI -0.73 kg to +2.84 kg, n = 587, I^2 = 65%, random effects).

High protein compared with other modified macronutrient diets: The effectiveness of high protein diets compared with other modified macronutrient diets was obtained from four trials (Clifton et al 2008, Gardner et al 2007, McAuley et al 2006, Dansinger et al 2005).

Clifton et al (2008) compared a high protein diet with a high carbohydrate diet, was small (n = 100), and was of low quality. Gardner et al (2007), McAuley et al (2006), and Dansinger et al (2005) were all moderate quality trials that compared a high protein diet to a Healthy Eating diet, but had small sample sizes, recruiting 158, 62, and 80 participants respectively. Dansinger et al (2005) also had poor adherence to the diets. The trials were combined in a meta-analysis. There was no significant difference in weight loss between high protein and other modified macronutrient diets (WMD -1.93 kg, 95%CI -4.98 kg to +1.11 kg, n = 400, I^2 = 86%, random effects).

High carbohydrate compared with other modified macronutrient diets: The effectiveness of high carbohydrate diets compared with other modified macronutrient diets combined was obtained from three trials (Clifton et al 2008 2, Gardner et al 2007, Dansinger et al 2005).

Clifton et al (2008) compared a high carbohydrate diet with a high protein diet, was small (n = 100), and was of low quality. Gardner et al (2007) and Dansinger et al (2005) were moderate quality trials and compared high carbohydrate diets with Healthy Eating diets, but were small, recruiting 155 and 80 participants respectively. Dansinger et al (2005) also had poor adherence to the diets. The three trials were combined in a meta-analysis. There was no significant difference in weight loss between the high carbohydrate and other modified macronutrient diets (WMD -0.19 kg, 95%CI -1.46 kg to +1.08 kg, n = 335, I^2 = 0%).

Healthy Eating diet compared with other modified macronutrient diets: The effectiveness of the Healthy Eating diet compared with other modified macronutrient diets was obtained from three trials (Gardner et al 2007, McAuley et al 2006, Dansinger et al 2005).

The trials were of moderate quality. Gardner et al (2007) compared a Healthy Eating diet with a low carbohydrate diet, McAuley et al (2006) compared a Healthy Eating diet with a high protein diet, and Dansinger et al (2007) compared a Healthy Eating diet with a high carbohydrate diet. The trials were small, recruiting 156, 62, and 80 participants respectively. Dansinger et al (2005) also had poor adherence to the diets. The trials were combined in a meta-analysis. There was no significant difference in weight loss between the Healthy Eating Diet and any other modified macronutrient diet (WMD -0.52 kg, 95%CI -6.00 kg to +4.95 kg, n = 298, I^2 = 93%, random effects).

Diets across dietary categories

To make sense of the evidence about head-to-head studies that compared one type of diet (low energy, very low energy, low glycaemic index/load and modified macronutrient diets) with another type of diet, studies were grouped together where the same diets were compared. Where possible, a meta-analysis was carried out for each group using RevMan 5. We found all types of diets appear effective for adult weight loss over the long term. However, the amount of weight lost ranged from weight maintenance to a loss of more than 10 kg over 12 months. Also, the more extreme types of modified macronutrient diets (eg, the Atkins and Ornish diets) may be difficult to adhere to (Dansinger et al 2005).

Very low energy compared with low energy diets: The effectiveness of very low energy diets compared with low energy diets was obtained from one systematic review (Tsai and Wadden 2006) and two trials (Viegener et al 1990, Simonen et al 2000).

The six trials included in Tsai and Wadden (2006) were of low quality and small (sample sizes of 17–58). Viegener et al (1990) was also of low quality and small (n = 85). Simonen et al (2000) was of a moderate quality trial, but very small (n = 16). The trials included in Tsai and Wadden could not be pooled with the other trials because inadequate data were published in the review. However, Viegener et al and Simonen et al could be combined. These studies showed no significant difference in body weight lost between those on the very low energy diets and those on the low energy diets (WMD -0.67 kg, 95%CI -3.43 kg to +2.09 kg, n = 101, I^2 = 24%). Tsai and Wadden reported similar results, with no significant difference in percentage of body weight lost (WMD 1.3% ± 5.1%, $p > 0.20$).

Modified macronutrient compared with very low energy diets: The effectiveness of modified macronutrient diets compared with very low energy diets was obtained from a systematic review (Avenell et al 2004), with one trial with 18 months' follow-up. The trial was of low quality. Participants in the modified macronutrient diet group (low fat diet) had not lost significantly more or less weight than those in the very low energy group at 18 months (WMD -4.70 kg, 95%CI -11.79 kg to +2.39 kg, n = 160).

Modified macronutrient compared with low energy diets: The effectiveness of modified macronutrient diets compared with low energy diets was obtained from one systematic review (Avenell et al 2004) and five trials (Stern et al 2004, Brinkworth et al 2004, Due et al 2004, Azadbakht et al 2007, McManus et al 2001).

Avenell et al (2004) compared low carbohydrate diets with low energy diets (seven trials, n = 150). The quality of the seven trials was low.

Of the five trials, Stern et al (2004) compared a low carbohydrate diet with a low energy diet in a low quality trial (n = 132); Brinkworth et al (2004) compared a high protein diet with a low energy diet in a low quality trial (n = 58); Due et al (2004) compared a high protein diet with a low energy diet in a very low quality trial (n = 50); Azadbakht et al (2007) compared a low fat diet with a low energy diet in a low quality trial (n = 100); and McManus et al (2004) compared a low fat diet with a low energy diet in a very low quality trial (n = 101).

Avenell et al (2004) did not provide enough data to combine their trials with the new trials in a meta-analysis, but the new trials could be combined. There was no significant difference in weight loss (WMD -2.0 kg, 95%CI -4.92 kg to +0.92 kg, n = 441, I^2 = 90%, random effects). Avenell et al (2004) also found no significant difference in weight loss between modified macronutrient and low energy diets at 12, 18, 24, 36, and 60 months.

Modified macronutrient compared with low glycaemic index/load diets: The effectiveness of modified macronutrient diets compared with low glycaemic index/load diets was obtained from two trials (Ebbeling et al 2007, Dale 2007).

The trial by Ebbeling et al (2007) was of moderate quality. The trial by Dale (2007) was of a moderate to high quality. Ebbeling et al recruited 73 participants compared with the 200 Dale recruited. The trials could not be combined in a meta-analysis because the outcomes measured were different. However, the results were consistent. Ebbeling et al found no significant difference (p = 0.50) in body fat percentage between low and high carbohydrate diets at 18 months (-1.5% ± 0.40% compared with -1.1% ± 0.40, p = 0.50). Similarly, Dale (2007) found no significant difference in change in body weight between groups at 24 months (mean difference +0.70 kg, 95%CI -1.1 kg to +2.5 kg, p = 0.15).

Evidence for Māori, Pacific and South Asian population groups

Two trials were completed in New Zealand (Dale 2007, McAuley et al 2006). One trial did not report ethnicity (McAuley et al 2006), and the other (Dale 2007) reported a predominantly New Zealand European study population. It is likely the former trial was also predominantly New Zealand European, because it recruited from the Dunedin community (as did the other trial). Therefore, no dietary interventions have been principally tested in Māori, Pacific, or South Asian population groups.

Three trials in US populations reported including a large proportion of minority ethnic groups (Gardner et al 2007, Dansinger et al 2005, Ebbeling et al 2007), but the trials did not analyse results by ethnic group.

The main problem is the lack of evidence relating to the acceptability and effectiveness of different types of diets for Māori, Pacific, and South Asian population groups.

Evidence statements

Nutrition advice increases weight loss in overweight and obese people by -5.1 kg (95%CI -6.7 to -3.5) and -1.9 kg/m^2 (95%CI -2.3 to 1.5) over 12 months.

Quality of evidence	**Low to moderate**
Study limitations	Some limitations*
Inconsistency	No inconsistency
Directness	Direct
Precision	Some imprecision‡

* Data in Anderssen et al (1996) and 92% of data in Dansinger et al (2007) were analysed using completers only. Allocation concealment, blinding, and baseline comparability of trials in Dansinger et al (2007) not reported.

‡ Random effects model used for analysis of Dansinger et al (2007) due to heterogeneity.

When nutrition advice stops, weight regain is likely to occur at a rate of +0.03 kg/m^2 per month, up to 48 months and possibly longer (at this rate of regain the baseline weight would be reached after about five years).

Quality of evidence	**High**
Study limitations	Some limitations*
Inconsistency	No inconsistency
Directness	Direct
Precision	Some imprecision‡

* Ninety-two percent of data of Dansinger et al (2007) were analysed using a completers-only analysis and allocation concealment, blinding, and baseline comparability of included trials was not reported.

‡ Random effects model used for analysis of Dansinger et al (2007) due to heterogeneity.

Nutrition advice delivered by a dietitian or nutritionist is not more effective for weight loss in overweight and obese people than nutrition advice delivered by a trained nurse or general practitioner.

Quality of evidence	**Moderate**
Study limitations	Some serious limitations*
Inconsistency	No inconsistency
Directness	Direct
Precision	Imprecision likely‡

* Real possibility of disclosure of allocation sequence reported for Prichard (1999). Randomisation, follow-up, blinding, and baseline comparability not reported for Prichard (1999). Dale (2007) was a moderate to high quality.

‡ Two trials, one comparing a doctor- and dietitian-led group with a dietitian-led group and the other comparing a nurse-led group with a dietitian-led group.

There was no significant difference between low energy diet and control groups (WMD -4.4 kg, 95%CI -10.3 kg to 1.5 kg) over 12 months compared with the control.

Quality of evidence	**High**
Study limitations	Some limitations*
Inconsistency	Some inconsistency**
Directness	Direct
Precision	Imprecision likely‡

* For both trials, analysis was for completers only. Allocation concealment was not reported for Wood (1988).

** The Wood (1988) trial found low energy diets more effective than the control diet. Jehn et al (2006) found no difference between low energy and control groups.

‡ Two trials; interventions differed. One offered general low energy advice, and the other the DASH low energy diet.

Very low energy diets (including those incorporating meal replacements) increase weight loss in overweight and obese people by -3.8 kg (standard deviation 6.3 kg) over 12 months compared with the control.

Quality of evidence	**Low to moderate**
Study limitations	Major limitations*
Inconsistency	No inconsistency
Directness	Direct
Precision	Some imprecision‡

* Franz et al (2007) was of moderate quality but included trials were of a low quality: Allocation concealment, randomisation methods, and blinding were not reported; analysis was for completers only; 69% follow-up overall. Stenius-Aarniala et al (2000) did not report allocation concealment, follow-up, blinding, or baseline comparability. Analysis was for completers only.

‡ Random effects model used in Franz et al (2007) as studies were heterogeneous.

Low glycaemic index/load diets increase weight loss in overweight and obese people by -1.0 kg (95%CI -0.1 kg to -1.9 kg) compared with high glycaemic index diets. Low glycaemic index/load diets produce weight losses of -4 kg to -12 kg over 12 months.

Quality of evidence	**Low to moderate**
Study limitations	Some limitations*
Inconsistency	Some inconsistency**
Directness	Indirect
Precision	Possible imprecision‡

* Thomas et al (2008) and Das et al (2007) did not report allocation concealment methods. Analysis in Das et al (2007) was for completers only, and study numbers were very small (n = 34).

** Thomas et al (2008) found low glycaemic index diets more effective for weight loss than a high glycaemic index diet; Das (2007) found no difference between diets.

‡ A fixed-effects model was used in Thomas et al (2008) as four included trials were homogeneous. The glycaemic index of the intervention diets differed between studies in the review and the Das et al trial, and the Das et al trial limited BMI to 25–30 kg/m^2.

All types of modified macronutrient diets (low carbohydrate, low fat, high protein, high carbohydrate, and the New Zealand Healthy Eating diet) are similarly effective for weight loss. All modified macronutrient diets produce weight losses of about 4 kg over 12 months.

Quality of evidence	**Low to moderate**
Study limitations	Some limitations*
Inconsistency	No important inconsistency
Directness	Indirect
Precision	Some imprecision‡

* Pirozzo et al (2008) and Nordman et al (2007) included low quality trials; for Pirozzo et al two out of six included studies analysed results for completers only; Nordman et al (2007) included trials with high attrition rates (15–50%) not reported by group. Adherence to the diet was poor in Dansinger et al (2005). Uneven losses by group in McAuley et al (2006).

‡ Some heterogeneity of trials in Nordmann et al (2006) and Pirozzo et al (2008).

All types of diets (low energy, very low energy, low glycaemic index/load, and modified macronutrient) are similarly effective for weight loss and produce weight losses of about 4 kg over 12 months. However, weight lost ranged from weight maintenance to a loss of more than 10 kg over 12 months.

Quality of evidence	Low to moderate
Study limitations	Some limitations*
Inconsistency	No important inconsistency
Directness	Indirect
Precision	Imprecision possible‡

* Most trials in Tsai and Wadden (2006) and Avenell et al (2004) analysed completers only. Small number of trials in each review (n = 5 and n = 7). Completers-only analysis in Viegener et al (1990) and Azadbakht et al (2007). Small study numbers in Simonen et al (2000). Uneven losses in Due et al (2004) and McManus et al (2001). Most trials did not report randomisation and allocation concealment methods.

‡ Results of two reviews and nine trials were included. Trials differed by follow-up period and nature of intervention.

Physical Activity and Exercise

Physical activity is any body movement produced by skeletal muscles that expends energy. This broad definition includes virtually all types of activity (eg, walking, cycling, waka ama, kapa haka, dance, traditional games, gardening, housework, sports, and intentional exercise). Intentional exercise is planned, structured, and repetitive activity. The terms physical activity and exercise are often used inter-changeably. The following information is based on evidence that assessed primarily exercise interventions. However, the good practice points below incorporate the broader definition of physical activity.

Exercise alone does not significantly increase weight loss after 12 months. Exercise combined with diet results in an average increased weight loss of 1.4 kg at 12 months, while the combination of exercise, diet, and behavioural strategies results in greater weight loss (2.1 kg) in comparison to exercise and diet at 12 months or more. Exercise is an important component of weight-loss programmes, but should be considered in combination with diet and some form of behavioural support.

Recommendation

Increased exercise should be incorporated into a weight-loss regimen only in combination with other strategies.
Strength of recommendation: Strong

Good practice points

- Encourage a lifestyle approach (the FAB approach).
  Be aware of and refer to local community options, especially for Māori, Pacific, and South Asian populations interested in weight loss (eg, marae-based, healthy hapū programmes and church-based or local community group options).
- Use information resources prepared for Māori, Pacific, and South Asian populations interested in physical activity that are in the person's preferred language, where possible.
- Encourage the person, for health benefits (including weight maintenance), to do at least 30 minutes of moderate-intensity aerobic physical activity (eg, brisk walking) on most or all days of the week (150 minutes each week).
- Encourage the person, for extra health benefits (including weight loss), to do at least 60 minutes of moderate-intensity aerobic physical activity (eg, brisk walking) on most or all days of the week (300 minutes each week).

What is physical activity and how much is required?

Physical activity is any body movement produced by skeletal muscles that expends energy. This broad definition involves virtually all types of activity, for example:

- sport and recreation
- active transport (eg, walking to work)

- incidental activity (eg, walking to do errands)
- exercise (physical activity that is planned, structured, and repetitive).

In common usage physical activity and exercise are used interchangeably. Physical activity is conventionally described by the four dimensions:

- type (eg, aerobic, muscle-strengthening, and flexibility)
- frequency (ie, the number of times)
- duration (ie, the amount of time)
- intensity (eg, light, moderate, or vigorous).

Aerobic physical activity is activity in which the body's large muscles move in a rhythmic manner for a sustained period. Aerobic activity, also called endurance activity, improves cardiorespiratory fitness. Examples of aerobic activity are waka ama, walking, kapa haka, running, swimming, and bicycling. Muscle-strengthening activity (eg, strength training, resistance training, and muscular strength and endurance exercises) is physical activity that increases skeletal muscle strength, power, endurance, and mass.

In this guideline the goal of physical activity is to increase a person's energy expenditure and their resting metabolic rate to achieve weight loss. To obtain health benefits, the New Zealand physical activity guidelines recommend that adults do at least 30 minutes of moderate-intensity aerobic physical activity on most if not all days of the week. This can also involve shorter sessions of 10 minutes three times each day. Vigorous and muscle-strengthening physical activities are also encouraged. The 2008 *Physical Activity Guidelines for Americans* released by the Centres for Disease Control and Prevention recommended 150 minutes of moderate-intensity aerobic physical activity or 75 minutes of vigorous intensity or a combination of both each week and muscle-strengthening activities on two or more days of the week. For even greater health benefits, the Centres for Disease Control and Prevention recommend 300 minutes of moderate-intensity aerobic activity or 150 minutes of vigorous activity or a combination of both each week and muscle-strengthening activities on two or more days of the week.

Physical activities that contribute to meeting physical activity guidelines

Moderate-intensity to vigorous physical activities of an aerobic nature in the sport and recreation, active transport, incidental, and exercise settings contribute to meeting the physical activity guidelines to obtain health benefits. Examples of moderate-intensity and vigorous aerobic physical activities are shown in Table 9.

Table 9: Examples of moderate-intensity, vigorous and muscle-strengthening physical activities

Moderate-intensity physical activity	Vigorous physical activity	Muscle-strengthening physical activity
Walking briskly (4.8 km/h or faster, but not race walking) Water aerobics Bicycling (slower than 16 km/h) Tennis (doubles) Ballroom dancing Light gardening	Race walking Jogging Running Swimming laps Waka ama Tennis (singles) Kapa haka Bicycling (16 km/hour or faster) Dancing Skipping Heavy gardening (continuous digging or hoeing, with heart rate increases) Hiking uphill or with a heavy backpack	Resistance training (eg, weight training or strength training) using weights, resistance (elastic) bands or plastic tubes Circuit-training Callisthenics (using body weight as resistance, eg, doing press-ups or sit-ups) Carrying heavy loads Doing heavy gardening)

Muscle-strengthening activities

Muscle-strengthening activities use the resistance to muscular contraction to build the strength, anaerobic endurance, and size of skeletal muscles. When properly performed, muscle-strengthening activities provide benefits that cannot be achieved with aerobic activity. These benefits include increased bone, muscle, tendon, and ligament strength and toughness; improved joint function; reduced potential for injury; increased bone density; and improved cardiac function.

Muscle-strengthening activities make muscles do more work than they are used to doing by progressively overloading the muscles. Muscle-strengthening activities are primarily anaerobic activities, although some proponents have adapted them into circuit training to provide the benefits of aerobic exercise. Examples of muscle strengthening activities are shown in Table 9.

Overweight and obese people need to be careful with certain types of muscle-strengthening activities, especially upper body activities, because they have an increased risk of heart attack. They first need to increase their aerobic fitness. It is important to remember that muscle-strengthening activities may result in an initial weight gain as muscles change (primarily increase).

Flexibility exercises

Flexibility refers to a person's ability to move his or her joints through a full range of motion. Greater flexibility in one's muscles allows for more movement around the joints. Flexibility can be improved with specific stretching exercises. Warming up with gentle

stretching for 5–10 minutes prepares a person for aerobic activities such as walking or swimming, and reduces the chance of injury during other activities.

Evidence for physical activity and exercise

One trial compared high-intensity physical activity (exercise goal of 2500 kcal/week) with low-intensity physical activity (exercise goal of 1000 kcal/week) (Tate et al 2007). One Cochrane systematic review (Shaw et al 2006) and nine trials were extracted from the NICE guideline (Donnelly et al 2003, Foreyt et al 1993, Messier et al 2004, Pavlou et al 1989, Sikand et al 1988, Wadden et al 1998, Wing et al 1988, Wing et al 1998). Pavlou et al (1989) reported on two trials.

Three trials compared the outcome of diet and exercise with that of diet alone (Donnelly et al 2003, Pavlou et al 1989). Six trials combined behavioural therapy, activity, and diet and compared the outcome from them with that from behavioural therapy and diet (Foreyt et al 1993, Messier et al 2004, Sikand et al 1988, Wadden et al 1998, Wing et al 1988, Wing et al 1998). Five trials involved walking as the primary activity (Donnelly et al 2003, Foreyt et al 1993, Sikand et al 1988, Wing et al 1988, Wing et al 1998). Four trials included aerobic and strength training exercises (Pavlou et al 1989, Messier et al 2004, Wadden et al 1998). Overall, the trials in Shaw et al (2006) were generally small and of a low quality. The trials extracted from the NICE guideline were of very low to moderate quality.

Shaw et al (2006) included trials with less than 12 months' follow-up. Therefore, only the following trials were included in meta-analyses (Anderssen et al 1996, Kiernan et al 2001, Pritchard et al 1997, Stefanick et al 1998, Wood et al 1988, Wood et al 1991). These trials were combined in meta-analyses with the trials extracted from the NICE guideline.

The mean weight loss in the two trials that compared exercise with a no-treatment control was not statistically significant at 12 months (WMD -2.81 kg, 95%CI, -6.19 to +0.57 kg, n = 270, I^2 = 93%) (Stefanick et al 1998, Wood et al 1988). Due to heterogeneity, a random effects method was used to combine these two trials.

The mean weight loss in the three trials that compared exercise to diet significantly favoured diet (WMD -2.70 kg, 95%CI, -3.43 to -1.96 kg, n = 313, I^2 = 13%) after 12 months follow up (Pritchard et al 1997, Stefanick et al 1998, Wood et al 1988).

The mean weight loss in the five trials that compared exercise and diet to diet alone significantly favoured the combination of diet and exercise for weight loss (WMD -1.42 kg 95%CI -2.16 to -0.68 kg, n = 543, I^2 = 0%) after 12 months follow up (Donnelly et al 2003, Pavlou et al 1989, Stefanick et al 1998, Wood et al 1991).

Two trials compared diet and exercise to diet alone, but reported the outcome only as a change in BMI (Anderssen et al 1996, Kiernan et al 2001). The mean BMI change in these trials significantly favoured the combination of diet and exercise (WMD -0.66 kg, 95%CI, -1.09 to -0.22 kg, n = 269, I^2 = 0%) after 12 months' follow up.

The mean weight loss in the six trials that compared exercise and diet and behaviour therapy to diet and behavioural therapy significantly favoured the addition of exercise to the combination (WMD -2.09 kg, 95%CI, -3.78 to -0.41 kg, n = 314, I^2 = 2%) after 12 months follow up (Foreyt et al 1993, Messier et al 2004, Sikand et al 1988, Wadden et al 1998, Wing et al 1988, Wing et al 1998).

Overall, strategies that combined exercise with other approaches found greater weight loss than the isolated effect of exercise alone.

Shaw et al (2006) found no significant difference in mean weight loss in the seven trials that compared high-intensity exercise with low-intensity exercise at up to 24 months (WMD -0.08 kg, 95%CI -1.20 to +1.04 kg, n = 224, I^2 = 33%). These trials were generally small (median sample size of 29) and of low quality. The trial in Tate et al (2007) could not be included in a meta-analysis with these trials, but it recruited 202 participants and was of a moderate quality. Tate et al found a significant effect in favour of the lower intensity intervention of -0.9 kg for the low-intensity exercise goal (1000 kcal/wk) compared with a weight gain of 2.86 kg in the high-intensity group (exercise goal of 2500 kcal/wk) (mean difference -2.96 kg, p<0.01).

Evidence for Māori, Pacific and South Asian population groups

No trials have been conducted with Māori, Pacific, or South Asian population groups.

One trial used targeted recruitment strategies to enhance the recruitment of African American participants (Messier et al 2004). Twenty-four percent of participants were African American, but no subgroup analysis was conducted.

Evidence statements

Combining exercise and dietary strategies increases weight loss by 1.4 kg up to one year.

Quality of evidence	**Low to moderate**
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* Unclear allocation concealment in all studies. Unclear whether analysed using intention to treat in all studies. Blinding not reported in any studies.

Adding behavioural support to exercise and dietary strategies increases weight loss by 2.1 kg up to one year.

Quality of evidence	**High**

Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* Management of missing data not reported. Loss by group not reported (overall loss low). Unclear allocation concealment in all studies. Unclear whether analysed using intention to treat in all studies. Blinding reported in two studies only.

Behavioural Strategies

Behavioural support involves strategies to reinforce changes in lifestyle change, particularly in diet and physical activity. Behavioural strategies increase weight loss by 2–8 kg at up to one year. More-intensive programmes produce greater weight loss at up one year than less-intensive programmes, but there is no clear difference between group-based or individual approaches. Family-based programmes may increase weight loss by up to 2 kg compared with individual programmes. Family-based programmes usually mean the person has partner support, and involve, at a minimum, the person attending weight-loss meetings with their partner. Adding motivational interviewing to standard behavioural support increases weight loss and prevents weight regain, but ongoing support may be crucial to maintaining an effect in minority populations.

Recommendation

Behavioural support is incorporated into weight-loss strategies.
Strength of recommendation: Strong

Good practice points

- Encourage the lifestyle approach (the FAB approach).
  Be aware of and refer to local community options, especially for Māori, Pacific, and South Asian populations interested in weight loss (eg, marae-based, healthy hapū programmes and church-based or local community group options).

Types of behavioural strategies

Behavioural strategies draw on the principles of learning theory. The goal of behavioural support is to alter the eating and activity habits of the overweight or obese person. Unless the person acquires new eating and physical activity habits, their ability to maintain their weight loss over the long term is unlikely.

The process of behavioural support involves identifying and specifying problem behaviours and the circumstances under which they occur. Then specific, measurable, and modest goals can be established with the person, and revised as they make progress. Target behaviours are monitored, usually by the person, to obtain a record of behavioural change. Cognitive strategies are often included to help modify the person's thoughts, which may be barriers to change.

Behavioural support includes seven strategies to help overcome the barriers to adherence to lifestyle changes. These tools are:

- self-monitoring
- stimulus control
- stress management
- problem solving
- contingency management

- cognitive restructuring
- social support.

Self-monitoring

Self-monitoring involves recording food intake and physical activity. Self-monitoring is a key step in behavioural approaches because it leads to increased awareness of patterns that can then be adjusted. Records can be paper-based diaries or electronic diaries on the internet. Records should include at least the amount and types of food eaten.

Stimulus control

Stimulus control helps to modify a person's eating and physical activity behaviours, by helping people to limit their exposure to high-risk situations. Examples of stimulus control are:

- learning to shop for healthier foods by understanding nutrition information panels
- keeping energy-dense foods out of the house
- clearing high-energy foods out of kitchen cupboards and the fridge
- substituting high-energy foods with healthier options
- limiting the times and places of eating
- consciously avoiding situations in which overeating occurs
- using the stairs not lifts and escalators
- removing energy-saving devices (eg, television remote controls).

Stress management

Stress can trigger poor eating patterns. Stress management can help defuse situations that lead to overeating. Coping strategies, meditation, and relaxation techniques have been successfully used to reduce stress.

Problem solving

Problem-solving skills involve a person correcting difficult areas related to their eating and physical activity. Approaches to problem solving include identifying weight-related problems (eg, eating cheese and crackers while watching television), and then generating or brainstorming possible solutions and choosing one (eg, buying only grated cheese or cottage cheese, or having low energy snacks available). Problem solving involves identifying, planning, and implementing the healthier alternative, and then evaluating the outcome of possible changes in behaviour. Setbacks in behavioural change should be viewed as opportunities to learn rather than as times to punish. The person should be encouraged to ask, "What did I learn from this attempt?". Answering this question is more useful than adding to poor self-esteem.

Contingency management

Contingency management involves the planned use of rewards for specific activities that are positive for weight loss or weight maintenance. Rewards can come from others or from the person themselves. Rewards may be gained for spending more time doing physical activity (eg, walking or gardening) or for meeting a weight-loss goal (eg, losing 500 g). Oral as well as tangible rewards can be useful, particularly for adults.

It is worth noting that a recent review on monetary incentives for weight loss found no effect for monetary incentives. Therefore, rewards should be intrinsically valuable to the person attempting to lose weight (Paul-Ebhohimhen and Avenell 2008) (eg, a fun family activity or going to a movie).

Cognitive restructuring

Unrealistic goals and inaccurate beliefs about weight loss and body image need to be modified to help change self-defeating thoughts and feelings that undermine weight-loss efforts. Rational responses designed to replace negative thoughts are encouraged. For example, the thought, "I blew my diet this morning by eating that pie, so I may as well eat what I like for the rest of the day", could be replaced by a more adaptive thought such as, "Well, I ate that pie this morning, but I can still eat in a healthy manner at lunch and dinner".

Social support

Strong systems of social support are helpful in weight loss. Family/whānau, friends, and colleagues can all help to maintain a person's motivation and provide positive reinforcement. Some people may benefit by entering a weight-reduction support group. Starting a lunch-time walking group at work is another means of providing support and encouraging fun with physical activity. Partners should be involved whenever possible.

Evidence for behavioural strategies

Evidence about the effectiveness of behavioural intervention was obtained from a Cochrane systematic review (Shaw et al 2005), a systematic review conducted for the NHS Health Technology Assessment (Avenell et al 2004), and two trials conducted since the NICE guideline was completed (Gold et al 2007, West et al 2007).

The trials in Shaw et al (2005) were generally of a low to moderate quality (36 trials, n = 3495) and few could be included in meta-analyses. The mean weight loss in the two trials that compared behavioural therapies to no therapy was -2.0 kg (95%CI -2.7 kg to -1.3 kg, n = 1254, I^2 = 0%) after more than 12 months. When behavioural interventions were added to diet and exercise, the mean weight loss was greater in the group also receiving behavioural interventions (WMD -4.7 kg, 95%CI -4.9 kg to -4.4 kg, n = 467, I^2 = 87%) at no more than 12 months. However, only two studies had a 12-month follow-up, and when a random effects model was used for reporting the results the findings were not significant. More-intensive approaches were associated with greater weight loss (WMD -2.3 kg, 95%CI -3.3 kg to -1.4 kg, n = 306, I^2 = 29%) at no more than 12 months, but did not increase weight loss over longer periods (WMD -0.2 kg, 95%CI -.3.8 kg to 3.3 kg, n = 58).

The trials in Avenell et al (2004) were small and of a poor quality. Three trials provided 12-month data, one 18-month data, and one 36- and 60-month data. Behavioural interventions when added to diet were associated with significant weight loss at 12 months (WMD -7.7 kg, 95%CI -12 kg to -3.4 kg, n = 50) and at 18 months (WMD -4.2 kg, 95%CI -8.3 kg to 0.04 kg, n = 31) but not at 36 or 60 months. Adding behavioural interventions to sibutramine and diet resulted in increased weight loss (WMD -10.7 kg, 95%CI -14.2 kg to -7.2 kg, n = 72) at 12 months.

Gold et al (2007) could not be added to either review. The trial tested face-to-face behavioural support compared with internet-based meetings, so might have usefully added data to the meta-analysis in Shaw et al (2005), which compared more intensive to less intensive support. However, the poor follow-up rate would not have qualified the trial for inclusion in this review. Face-to-face support result in less weight lost than internet-based support (mean difference 1.6 kg, p = 0.15, n = 123).

One trial identified tested the effect of 12 months of motivational interviewing added to a standard behavioural intervention for weight loss in women with type 2 diabetes (West et al 2007). The trial was of a high quality and found the addition of motivational interviewing (a process of interviewing, assessing, and actively motivating people) increased weight loss compared with the standard behavioural intervention after 18 months (mean difference -1.6 kg, p = 0.04). Weight loss was the focus of the first six months of the intervention, and the following 24 months focused on maintaining the initial weight loss.

Six trials were extracted from the NICE guideline to assess the effectiveness of family interventions compared with individual interventions (Black and Lantz 1984, Cousins et al 1992, Murphy et al 1982, Pearce et al 1981, Wing et al 1991, Wing and Jeffery 1999). These trials were put into meta-analyses using RevMan 5. Family-based interventions – usually limited to including the spouse in the intervention – increased weight loss (WMD 1.85 kg, 95%CI 0.34 kg to 3.35 kg, n = 236, I^2 = 15%). The spouse attended weight-loss meetings with the participant. In some studies, the spouse had an active role in monitoring, but in other studies did not actively engage in behavioural strategies.

Four trials were extracted from the NICE guideline to assess the effectiveness of group-based interventions compared with individual interventions (Hakala et al 1993, Jones et al 1986, Long et al 1983, Straw and Terre 1983). These trials were put into meta-analyses using RevMan 5. Group-delivered interventions did not significantly increase weight loss (WMD 1.23 kg, 95%CI -1.19 kg to 3.64 kg, n = 107, I^2 = 36%).

Evidence for Māori, Pacific and South Asian population groups

No trials have been conducted with Māori, Pacific, or South Asian population groups.

Two trials with minority populations were identified (Cousins et al 1992, West et al 2007), but it is not appropriate to directly extrapolate this evidence to Māori, Pacific, and South Asian population groups in New Zealand.

The main issue revealed by the inequalities analysis is the absence of evidence for Māori, Pacific, and South Asian population groups.

Evidence statements

Including behavioural strategies will increase weight loss at up to one year. More-intensive approaches increase weight loss compared with less-intensive approaches.

Quality of evidence	**Low to moderate**
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision‡

* Unclear allocation concealment in all but one study. More than a 20% loss in a few studies. Unclear whether analysed using intention to treat in all but one study.

‡ Random effects model used for analysis of behaviour therapies plus diet and exercise due to heterogeneity.

Adding motivational interviewing to standard behavioural strategies will increase weight loss and decrease regain at 18 months. Continued long-term support may be more important to minority populations than majority populations.

Quality of evidence	**High**
Study limitations	Minor limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision‡

* Management of missing data not reported, nor loss by group (overall loss low).

Family involvement increases weight loss, but group-based interventions are not more or less effective than individual interventions.

Quality of evidence	**Low**
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision‡

* Unclear allocation concealment in all studies. More than a 20% loss in four out of nine studies, not analysed using intention to treat in all studies.

‡ Effect of family interventions borderline significant.

Pharmacotherapies (Weight-Loss Drugs)

The drugs considered for inclusion in the guideline were orlistat (Xenical), sibutramine (Reductil), phentermine (Duromine), and fluoxetine (Fluox, Plinzene, and Prozac). The drugs registered for use in weight loss in New Zealand are orlistat, sibutramine, and phentermine. There is no evidence to assess the one-year effect of phentermine. Fluoxetine is not effective for weight loss. Diuretics should not be used for weight loss. se of orlistat or sibutramine may lead to increased weight loss of 3–4 kg. These drugs may also be used to maintain an initial weight loss when larger amounts have been lost.

Drug therapy is not a substitute for a lifestyle change and changes to diet and exercise are still necessary to assist with weight maintenance once medication use ceases.

Recommendations

Orlistat may be used in conjunction with a low-fat diet and increased physical activity to produce additional weight loss and to maintain weight loss in overweight or obese adults.
Strength of recommendation: Strong

Sibutramine may be used in conjunction with dietary modifications and increased physical activity to produce additional weight loss and to maintain weight loss in overweight or obese adults.
Strength of recommendation: Strong

Good practice points

- Provide information in people's preferred language, if possible.
- Become familiar with new drug information as it is released; safety is an evolving area.

Orlistat

- Consider treatment with orlistat for weight loss only in people who have not reached their target weight loss or have reached a plateau with dietary, activity, and behavioural changes
- Remember that gastrointestinal side effects, including faecal incontinence, are likely if a person eats high-fat meals when taking orlistat.
- Tell the person to take orlistat in conjunction with a low-fat diet.
- Ensure the person's drug therapy continues beyond three months only if the person has lost at least 5% of their initial body weight since starting drug therapy (although note the rate of weight loss may be slower in people with diabetes).
- Consider orlistat for long-term use for weight maintenance once the initial weight is lost.

Sibutramine

- Remember that sibutramine is contraindicated in people aged under 18 or over 65 and in people with inadequately controlled hypertension. Caution is required in people with some psychiatric illness. Check for other contraindications.
- Consider treatment with sibutramine for weight loss only in people who have not reached their target weight loss or have reached a plateau with dietary, activity, and behavioural changes.
- Continue drug therapy beyond three months only if the person has lost at least 5% of their initial body weight since starting drug therapy.
- Monitor the person's blood pressure to observe for any hypertensive effect of the medication. Do not prescribe sibutramine unless there are adequate arrangements for monitoring adverse effects (specifically pulse and blood pressure). MedSafe data sheets suggest fortnightly monitoring for the first three months.
- MedSafe data sheets support treatment for 12 months, but trials suggest considering longer-term use, especially for maintaining weight loss.

Types of weight-loss drugs

The modes of action of the three registered drugs are as follows.

- Orlistat is a gastrointestinal lipase inhibitor. It binds with lipase in the stomach or small intestine, and thereby prevents dietary fat from being broken down and digested.
- Sibutramine is a serotonin and noradrenaline reuptake inhibitor. By inhibiting reuptake more serotonin and noradrenalin are available to act on receptors, thereby increasing satiety and increasing energy expenditure.
- Phentermine is a sympathomimetic amine. Phentermine is a stimulant that acts on the central nervous system and suppresses appetite.
- Rimonabant was considered but not included because it is unlikely to be registered in New Zealand. Fluoxetine is not registered for weight loss.

Evidence for weight-loss drugs

No studies of phentermine met the 12-month inclusion criteria for this guideline. Two systematic reviews were identified (Haddock et al 2002, Li et al 2007). The second review summarised the first review's findings with respect to phentermine. Nine trials were reported in the review and an additional trial has since been published (Kim et al 2006). The maximum period of follow up in the 10 trials was 24 weeks, but it was not possible to add the new evidence to the findings of the review. However, use of phentermine significantly increased weight loss in these short-term studies.

Li et al (2007) included trials of fluoxetine for weight loss. Six of the trials reported 12-month outcomes, but one trial investigated the effect of fluoxetine in weight gain in people with depression rather than for treating overweight or obesity. Four of the remaining trials had very small samples (range n = 19–45), but one trial was large (n = 458). The authors considered the trial findings too heterogeneous to summarise in a pooled analysis. The findings of the small trials ranged from 0.7 kg to 14.8 kg greater weight lost in the fluoxetine-treated people. However, the large trial found a non-significant difference of +0.4 kg (95%CI -1.7 kg to +2.5 kg) between the groups with more weight gain in the group treated with fluoxetine (Li et al 2007).

Evidence regarding the effectiveness of weight-loss drugs was largely obtained from two systematic reviews: a Cochrane review on anti-obesity drugs (Padwal et al 2008) and a head-to-head meta-analysis of weight-loss drugs (Neovius et al 2008). Further research on orlistat published since the inclusion date of these reviews was also assessed (Poston et al 2006, Richelsen et al 2007), but did not alter the findings.

Padwal et al (2008) included 30 studies with 19,619 participants. Sixteen trials (n = 10,631) evaluated orlistat, and 10 trials (n = 2,623) evaluated sibutramine. Four trials (n = 6,635) evaluated rimonabant, but are not discussed here because rimonabant is unlikely to be registered in New Zealand. Neovius et al (2008) included eight trials with 885 participants, but only two trials had a 12-month follow-up. These trials compared the effects of orlistat with those of sibutramine in 254 men and women with type 2 diabetes (n = 141) or hypertension (n = 113).

Orlistat

Padwal et al (2008) found that the use of orlistat in addition to a low fat diet and physical activity significantly increased weight loss (WMD -2.87 kg, 95%CI -3.21 kg to -2.53 kg, n = 9457, I^2 = 0%). A further study found no additional benefit of brief counselling on weight loss in people taking orlistat (Poston et al 2006). In people who had initially lost 14 kg, the use of orlistat for weight maintenance did not prevent weight regain over three years, but people receiving orlistat regained 2.4 kg less than those receiving a placebo (Richelsen et al 2007). Neovious et al (2008) in a head-to-head comparison of orlistat with sibutramine found no significant difference between the two drugs (WMD +0.4 kg, 95%CI -0.6 kg to +1.4 kg, n = 254, I^2 = 0%). However, the trials were in populations with type 2 diabetes and hypertension, and results may differ in people without these co-morbidities. There was increased risk of side effects associated with orlistat (absolute risk difference 24%, 95%CI 20–29%, n = 8938, I^2 = 77%, number needed to harm (NNH) = 4, random effects), including increased risk of faecal incontinence (absolute risk difference 6%, 95%CI 5–8%, n = 1639, I^2 = 0%, NNH = 17).

Sibutramine

Padwal et al (2008) found that the use of sibutramine in addition to lifestyle changes significantly increased weight loss compared with the control (-4.2 kg, 95%CI -4.8 kg to -3.6 kg, n = 1536, I^2 = 0%). Padwal et al also found sibutramine decreased weight regain after an initial weight loss (ie, sibutramine improved weight-loss maintenance after initial weight loss). Sibutramine significantly decreased weight regain (-4.0 kg, 95%CI -5.7 kg to -2.3 kg, n = 812, I^2 = 61%).

Sibutramine use was associated with increases in systolic (WMD +1.69 mmHg, 95%CI 0.11–3.28 mmHg, n = 1906, I^2 = 35%) and diastolic blood pressure (2.42 mmHg, 95%CI 1.51–3.32 mmHg, n = 1906, I^2 = 28%).

Evidence for Māori, Pacific and South Asian population groups

No trials have been conducted in Māori, Pacific, or South Asian population groups.

One trial (Swinburn et al 2005) in the Cochrane review (Padwal et al 2008) included New Zealand participants, but no data were provided on the ethnicity of participants. Small numbers of minority groups have been included in studies, but subgroup analyses by ethnicity have not been performed or reported. Therefore, there is no specific evidence on the effectiveness of drug therapy for indigenous or minority groups.

Although the overall use of weight-loss drugs in New Zealand has not been studied, the patterns of sibutramine usage in New Zealand between 2001 and 2004 have been examined through data from the Intensive Medicines Monitoring Programme (Hill et al 2007). In this programme, 17,298 people were dispensed sibutramine, 84% of whom were women. Seventy-six percent of the people were New Zealand European, 10% Māori, 1.7% Pacific, 2% Asian, and 10% Other. The mean age of men and women was 45 and 42 years respectively. Although the prescribing guidelines recommend using sibutramine in people aged 18–65, 398 people in this cohort were aged over 65. Sibutramine use was predominantly short term, with 48% of people using the drug for 60 days or less and 88% having one course of treatment. Usage was highest among New Zealand European women aged 30–59 (6.9 per 1000 population), followed by Māori women (5.8 per 1000 population), and was much lower among Pacific (2.4 per 1000) and Asian (1.8 per 1000) women. Similar patterns of usage are seen in men, although their overall use is much lower than it is for women. Given the higher prevalence of overweight and obesity in Māori and Pacific populations, sibutramine usage was proportionately under-represented in the cohort, although this may be accounted for in part by the higher prevalence of hypertension in Māori and Pacific populations (Ministry of Health 2008c) (people using sibutramine when they have hypertension must be monitored carefully). Another explanation is differential prescribing practices, with Māori and Pacific being under-prescribed.

The two main issues revealed by this inequalities analysis are the:

- absence of evidence in Māori, Pacific, and South Asian population groups
- disproportionately low use of the anti-obesity drug sibutramine by Māori and Pacific populations given the higher prevalence of obesity in these groups.

Evidence statements

Orlistat in combination with lifestyle change increases weight loss by around 3 kg in overweight and obese adults.

Quality of evidence	**Low to moderate**

Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* Unclear methods of randomisation and allocation concealment for most studies. Baseline comparability not reported for most studies. Attrition rates of up to 30%.

Orlistat in combination with lifestyle change decreases weight regain by 2.4 kg in people maintaining their initial weight loss.

Quality of evidence	**Moderate**
Study limitations	Some limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* Attrition rate of up to 35% with last observation carried forward for intention to treat analyses.

Sibutramine in conjunction with a lifestyle change increases weight loss by around 4 kg in overweight and obese adults.

Quality of evidence	**Low to moderate**
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* Unclear methods of randomisation and allocation concealment for most studies. Baseline comparability not reported for most studies. Attrition rates of up to 40%.

Sibutramine in conjunction with lifestyle change decreases weight regain after initial weight loss by around 4 kg in obese adults.

Quality of evidence	**Moderate to high**
Study limitations	Some limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* Attrition rates of 32–46%, with last value carried forward in two of three studies for intention to treat analyses.

Bariatric Surgery

Bariatric surgery is appropriate only if all other measures have been tried and failed. Bariatric surgery is substantially more effective than non-surgical interventions in achieving weight loss among obese people, but still requires lifestyle changes. Weight loss may reach 40–50 kg one-year after the surgery with a BMI reduction of 10–18 kg/m^2 and a percentage excess body weight loss of 50–80%. There is also evidence of substantial improvements in major co-morbidities such as blood pressure, lipid levels, diabetes, and other conditions such as obstructive sleep apnoea, gastro-oesophageal reflux, stress incontinence, and peripheral venous stasis.

Recommendation

Bariatric surgery should be considered for people with a BMI of 40 kg/m^2 or more or a BMI of 35–40 kg/m^2 if they have another significant disease (eg, type 2 diabetes, sleep apnoea, high blood pressure, or arthritis requiring joint replacement), if all appropriate non-surgical measures have been tried but failed.
Strength of recommendation: Strong

Good practice points

- Remind people considering bariatric surgery that it is not an alternative to a lifestyle change, so they still need to modify their dietary intake and increase their physical activity.
- Remind people that after bariatric surgery, they will require life-long nutritional monitoring.
- Remind people a person who has had bariatric surgery may need to have excess skin folds surgically removed.
- Remind people they will need to consider what bariatric procedure is required based on the surgeon's expertise, hospital facilities, and the person's and their family/whānau characteristics and lifestyle.
- Provide information about bariatric surgery in people's preferred language, if possible.

Types of bariatric surgery

The three bariatric procedures available in New Zealand are:

- adjustable gastric banding
- sleeve gastrectomy
- Roux-en-Y gastric bypass.

Adjustable gastric banding places an adjustable band around the person's upper stomach to create a pouch with 15–30 ml capacity. Saline in the band can be increased or decreased through a port under the skin of the abdomen. The added volume of the band suppresses the person's appetite, but intermittent refilling is required to maintain that feeling. Adjustable gastric banding can be reversed.

A sleeve gastrectomy removes the outer three-quarters of the person's stomach and turns the stomach into a long gastric tube or 'sleeve'. A sleeve gastrectomy cannot be reversed.

A Roux-en-Y gastric bypass involves changes to the person's stomach and small bowel. The operation creates a small pouch in the stomach with a narrow outlet. Although the pouch's capacity can vary, it is usually about 30 ml. A bypass of the small bowel is also created to reduce the absorption of food.

All three operations are usually performed by laparoscopy. The operations remove a person's excess appetite or urge to eat and limit the volume they are able to eat.

Perioperative death is very low after bariatric surgery but rates vary by procedure. Wound infections are one of the most common complications. Complications such as bowel leakage, bleeding, stenosis, ulcers, and other infections may occur (Stephenson and Hogan 2007, Brethauer et al 2006). With gastric bypasses, nutritional deficiencies may occur in the long term, in particular deficiencies of vitamin B12, folate, and iron. People who have had this surgery must have long-term monitoring.

Evidence for bariatric surgery

Six trials were extracted from the NICE guideline (Andersen et al 1984, Mingrone et al 2002, Lujan et al 2004, Nguyen et al 2004, Sundbom and Gustavasson 2004, Westling and Gustavsson 2001). The remaining trials were identified through new searches (Angrisani et al 2007, van Dielen et al 2005, Olbers et al 2006, O'Brien et al 2006, Puzziferri et al 2006, Skroubis et al 2006, Bessler et al 2007, Arceo-Olaiz et al 2008).

Data on the effectiveness of surgical therapy compared with non-surgical therapy for weight loss in obese adults comes from one cohort study conducted in Sweden (Sjostrom et al 2007) and three trials conducted in Australia, Denmark, and Italy (O'Brien et al 2006, Andersen et al 1984, Mingrone et al 2002).

The Swedish Obese Subjects study was a prospective matched-control study, included follow-up to 15 years, and involved 4047 participants (Sjostrom et al 2007). The outcome of surgical procedures (vertical banded gastroplasty 68%, gastric banding 18%, and gastric bypass 13%) was compared with the outcome from conventional treatment. Conventional treatment ranged from sophisticated lifestyle and behavioural interventions to no treatment. This study had substantial statistical power, but there was no randomisation and small differences existed between groups at baseline in age, weight, and BMI. However, these differences are unlikely to have had an effect on the final results. There did not appear to be any blinding. There was a loss to follow-up of 16% and 25% in the surgical and control groups at 10 years, and 34% and 13% respectively at 15 years. Significantly greater weight change occurred in all surgical groups compared with in the control. The control weight remained within plus or minus 2% of baseline throughout the observation period, whereas at years 1–2, 10, and 15 weight change was respectively 32%, 25%, and 27% for gastric bypass; 25%, 16%, and 18% for vertical banded gastroplasty; and 20%, 14% and 13% for banding.

The three trials compared the effects of laparoscopic adjustable gastric banding (O'Brien et al 2006), gastroplasty (Andersen et al 1984), and bilio-pancreatic diversion (Mingrone et al 2002) to non-surgical programmes including dietary advice. Method of randomisation was unclear in two of the three trials, but there does appear to be reasonable baseline comparability. Power calculations, concealment, and blinding were not reported or were unclear in all trials. Analyses were not by intention to treat, although there was a relatively low loss to follow-up (< 10%). The trials were all small; O'Brien et al (2006) recruited 80 participants, Anderson et al (1984) 57 participants, and Mingrone et al (2002) 79 participants. All three trials demonstrated greater weight loss in the surgical group than in the control. In Andersen et al (1984) and O'Brien et al (2006) the surgical group had lost approximately 15–20 kg more than the control group had lost at 24 months, and this was statistically significant. There was also higher weight loss at 12 months in Mingrone et al (2002), but these authors did not undertake any statistical testing. Women who had surgery had lost 28 kg more than the women on dietary restriction by 12 months. Men who had surgery had lost 43 kg more than men on dietary restriction at 12 months. Lower weight loss in the longer duration trials (Andersen et al 1984, O'Brien et al 2006) and over time in the Swedish study may be explained by the 'relapse' after the initial weight loss following surgery when some weight is regained.

Thirteen trials compared different bariatric surgical procedures in adults. These trials included six trials that compared different operations, six trials that compared laparoscopic compared with open approaches to operations, and one trial of different stapling techniques. The 13 trials involved:

- two trials of laparoscopic adjustable gastric band compared with laparoscopic Roux-en-Y gastric bypass surgery (Angrisani et al 2007, Olbers et al 2006, Olbers et al 2005)
- one trial of Roux-en-Y gastric bypass compared with a variant of bilio-pancreatic diversion surgery (Skroubis et al 2006)
- two trials of banded compared with unbanded laparoscopic Roux-en-Y gastric bypass surgery (Arceo-Olaiz et al 2008, Bessler et al 2007)
- one trial of laparoscopic Roux-en-Y gastric bypass surgery compared with laparoscopic sleeve gastrectomy (Karamanakos et al 2008)
- one trial of open vertical banded gastroplasty compared with laparoscopic adjustable gastric band surgery (van Dielen et al 2005)
- five trials of laparoscopic gastric bypass compared with open gastric bypass surgery (Puzziferri et al 2006, Lujan et al 2004, Nguyen et al 2004, Sundbom and Gustavasson 2004, Westling and Gustavsson 2001)
- one trial of different stapling techniques during a laparoscopic sleeve gastrectomy (Dapri et al 2007).

Nine of the 13 trials had sample sizes of less than 100 (Angrisani et al 2007, Arceo-Olaiz et al 2008, Bessler et al 2007, Karamanakos et al 2008, van Dielen et al 2005, Lujan et al 2004, Sundbom and Gustavasson 2004, Westling and Gustavsson 2001, Dapri et al 2007). No trials provided details of power calculations. The trials were conducted in Europe (Angrisani et al 2007, Olbers et al 2006, Olbers et al 2005, Skroubis et al 2006, Karamanakos et al 2008, van Dielen et al 2005, Lujan et al 2004, Sundbom and Gustavasson 2004, Westling and Gustavsson 2001, Dapri et al 2007) and the United States (Bessler et al 2007, Puzziferri et al 2006, Nguyen et al 2004). All trials were randomised and groups appeared to be comparable at baseline for age, sex, weight, and BMI. Concealment and blinding were not reported or were unclear in all trials. Details about intention to treat analyses were either absent or unclear in most studies. However, in 10 of the 13 trials there was complete case follow-up or less than 5% of cases lost to follow-up. The exceptions were Nguyen et al (2004), Olbers et al (2006, 2005) and Puzziferri et al (2006), with loss to follow-up of 63%, 28–30%, and 22–25% respectively.

All trials reported weight loss in both surgical intervention groups. Weight loss was reported as either a reduction in weight (approximately 40–43 kg at one year in four trials), a decrease in BMI (approximately 10–18 kg/m^2 at 12–24 months in seven trials), or the percentage excess weight loss (approximately 50–80% at 12–24 months in 10 trials). Six of the 13 trials found statistical differences between the comparison groups (Angrisani et al 2007, Olbers et al 2006, Olbers et al 2005, Skroubis et al 2006, Karamanakos et al 2008, van Dielen et al 2005, Nguyen et al 2004). Two of the trials reporting statistical differences were those with the highest loss to follow-up (Nguyen et al 2004, Olbers et al 2006, Olbers et al 2005), and another trial reported a statistical difference in the percentage excess weight loss at 12 months, but no statistical difference in weight loss or reduction in BMI (Karamanakos et al 2008). A surgical procedure (bilio-pancreatic diversion) in Skroubis et al (2006) is seldom used now due to high complication rates. Another two trials indicated greater weight loss in the laparoscopic Roux-en-Y gastric bypass and vertical banded gastroplasty groups (respectively) compared with the laparoscopic adjustable gastric band group (Angrisani et al 2007, van Dielen et al 2005). However, they were both relatively small trials and provide inconclusive evidence.

Studies of bariatric surgery have noted improvements in co-morbidities associated with obesity, such as diabetes and blood pressure. One review focused on the impact of bariatric surgery on the co-morbidities of obesity (Buchwald et al 2004). The review included 136 studies (5 randomised controlled trials, 28 non-randomised controlled trials or series, and 101 uncontrolled case series). Diabetes was improved or resolved in 86% of participants, hyperlipidemia improved in more than 70% of participants, hypertension resolved or improved in 79% of participants, and obstructive sleep apnoea improved or resolved in 84% of participants.

Three studies that compared surgical therapy to non-surgical therapy reported data on co-morbidities (Mingrone et al 2002, O'Brien et al 2006, Sjostrom et al 2004). The incidence of hypertriglyceridemia and diabetes was statistically lower in the surgically treated group than in the control group after two years and 10 years in Sjostrom et al (2004). Recovery from hypertension, diabetes, hypertriglyceridemia, a low HDL cholesterol level, and hyperuricemia was more frequent in the surgical group than in the control group, at two years and 10 years in Sjostrom et al (2004). O'Brien et al (2006) found significantly greater improvements in diasystolic blood pressure, fasting plasma glucose, the insulin sensitivity index, and HDL cholesterol at two years in the surgical group compared with in the controls. The net improvement was approximately 9 mmHg for DBP, 0.5 mmol/L for glucose, and 0.3 mmol/L for HDL cholesterol. Mingrone et al (2002) found statistically significant reductions in total cholesterol, and LDL cholesterol, triglycerides, and the total cholesterol-to-HDL ratio in the surgery group at 12 months and no significant changes in the controls.

In summary, these studies provide convincing evidence of greater weight loss from surgical than from non-surgical interventions, with many trials also demonstrating improvements in co-morbidities such as hypertension, lipids, and diabetes in both groups with all surgical procedures. However, these trials do not provide convincing evidence of greater weight loss with one given surgical procedure than with others.

Evidence for Māori, Pacific and South Asian population groups

No trials have been conducted in Māori, Pacific, or South Asian population groups.

The 18 studies of bariatric surgery were conducted in the US, Australia, Mexico, and several European countries. None of the trials was conducted in New Zealand and no data were provided on the ethnicity of participants.

Only the Wakefield Gastroenterology Centre appears to be regularly publishing New Zealand bariatric outcome data (Dhabuwala et al 2000, He and Stubbs 2004, White et al 2005, Wickremesekera et al 2005, Stubbs et al 2006), but no data on ethnicity have been published in the reports from this register (Dhabuwala et al 2000, He and Stubbs 2004, White et al 2005, Wickremesekera et al 2005, Stubbs et al 2006).

The main issue exposed by the inequalities analysis is the absence of evidence in Māori, Pacific, and South Asian population groups.

Evidence statements

Bariatric surgery increases weight loss compared with non-surgical interventions.

Quality of evidence	Moderate
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* One matched cohort study was not randomised but had reasonable baseline comparability and was of a substantial size to provide good statistical power. Methods of randomisation and concealment were unclear in the three trials, but had good baseline comparability and loss to follow-up was less than 10%.

Bariatric surgery is associated with resolution or improvements in significant co-morbidities.

Quality of evidence	Moderate
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* One matched cohort study was not randomised, but had reasonable baseline comparability and was of a substantial size to provide good statistical power. Methods of randomisation and concealment were unclear in the two trials, but they had good baseline comparability and loss to follow-up was less than 10%.

All surgical interventions are associated with weight loss, but there is insufficient randomised evidence that any one procedure is superior in achieving weight loss.

Quality of evidence	Low to moderate
Study limitations	Serious limitations*
Inconsistency	No important inconsistency
Directness	Direct
Precision	Some imprecision

* All trials were randomised, but details were unclear about concealment and blinding in most trials. Good baseline comparisons. Although details about intention to treat analyses were absent or unclear in most studies, 10 of the 13 trials had complete follow-up or less than 5% of cases lost to follow-up.

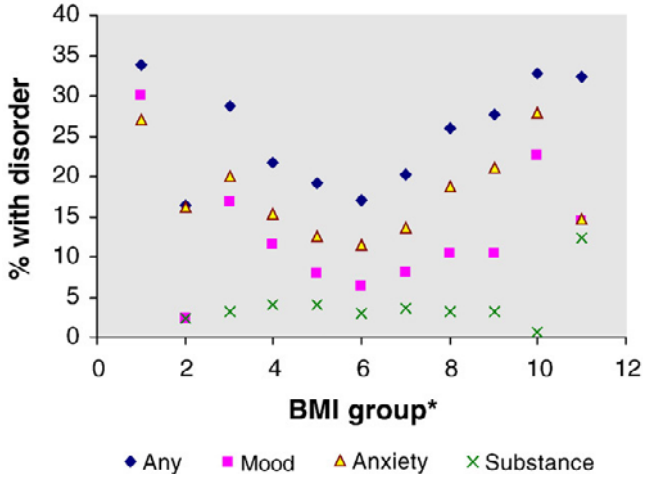
Mental Health

Te Rau Hinengaro, the New Zealand Mental Health Survey, was a nationally representative household survey in 2003–2004 to determine the prevalence of mental health disorders and associated co-morbidities (Oakley Browne et al 2006). The survey over-sampled Māori and Pacific populations to ensure the accuracy of estimates. A total of 12,992 participants aged 16 or over were assessed and their self-reported height and weight were obtained. Participants included 2595 Māori and 2374 Pacific people. The WHO's BMI thresholds were used to determine the prevalence of overweight and obesity. A limitation of this study was its use of self-reported weight, which may not be as reliable as more objective measures.

The prevalence of overweight and obesity were not reported separately. The combined prevalence of overweight and obesity (BMI ≥ 25 kg/m^2) in people with mental health disorders was approximately 52% (95%CI 50–54%). This prevalence is similar to the combined prevalence of overweight and obesity of 56% of participants reported in the 2002/03 New Zealand Health Survey (Ministry of Health 2004a). However, the picture differs when Te Rau Hinengaro participants' sex was considered. Prevalence of overweight and obesity was higher among females with a mental health disorder than among the total population (51.2% compared with 43.4%), but the same relationship did not hold for men (58.4% compared with 59.1%).

The prevalence of obesity (BMI ≥ 30 kg/m^2) in Te Rau Hinengaro participants has recently been reported (Scott et al 2008) (see Figure 5). The prevalence of obesity in people with any mental health disorder was 19.6% in males and 21.2% in females, which was similar to the prevalence reported in the 2002/03 New Zealand Health Survey (19.2% and 21.0%). The prevalence of obesity in Māori and Pacific Te Rau Hinengaro participants was 36.9% and 49.8% respectively, but the prevalence in association with mental health disorder was not reported by ethnicity.

Figure 5: Prevalence of mental health disorders by body mass index (BMI) group



* The BMI groups from 1 to 11 correspond to <15, 15–16.9, 17–18.49, 18.5–19.9, 20–24.9, 25–29.9, 30–34.9, 35– 39.9, 40–44.9, 45–49.9, and ≥ 50 kg/m^2.

Source: Scott et al (2008).

There is an association between body weight and mental health disorders. The odds ratio for any mood disorder when obese was 1.23 (95%CI 1.03–1.48) and for any anxiety disorder when obese was 1.46 (95%CI 1.23–1.72) when compared with those with a BMI of 18.5–29.9 kg/m^2. The mood disorders included in the study were major depression, dysthymia, bipolar disorder, and anxiety disorders (panic disorder, agoraphobia without panic, specific phobia, social phobia, general anxiety disorder, post-traumatic stress disorder, and obsessive–compulsive disorder). The prevalence of any mood and any anxiety disorder increased with BMI (Scott et al 2008).

Weight gain in people with mental health disorders is associated with medication. Systematic reviews of randomised controlled trials of medications compared with controls, with weight gain as an outcome or a side effect, are the most reliable method to determine the effect of drug treatments on weight. Weight gain, in the order of 2–7 kg, has been associated with (Leslie et al 2007):

- mood-stabilising medications, especially lithium and sodium valproate for the treatment of bipolar disorder
- atypical (new generation) antipsychotic medications, particularly olanzapine, clozapine, and risperidone for the treatment of psychosis, schizophrenia, schizo-affective disorder, bipolar disorder, borderline personality disorder, and alcohol dependence
- nortriptylline, doxepin, and amitryptylline for the treatment of depression

Simple drug switching may attenuate effects on the primary condition (Deshmuhk and Franco 2003). Other strategies have been evaluated in people with schizophrenia, but no trials of the management of overweight and obesity in populations with mental health disorders met the inclusion criteria of one year's follow-up for this guideline. However, the most recent systematic review of the short-term trials for managing weight in people treated with anti-psychotics explored the effect of non-drug strategies (Alvarez-Jimenez et al 2008). Alvarez-Jimenez et al found strategies to prevent and treat weight gain to be effective in people with schizophrenia (WMD -2.56 kg, 95%CI -3.20 kg to -1.92 kg, I^2 = 29%, 10 trials, n = 482). The intervention period ranged from eight weeks to six months, and follow-up after the intervention was between two and three months. The strategies included nutritional counselling, individual and group formats, and cognitive behavioural therapy. A meta-analysis of these strategies individually showed all to be effective, suggesting that the non-pharmacologic strategies outlined in this guideline can be adapted for people with mental health disorders.

Research Recommendations

We make 10 recommendations in relation to research into effective weight management strategies in adults.

1 Research on cohorts of Māori, Pacific, and South Asian population groups is needed to determine whether the association of BMI with risk of disease (cardiovascular, disease, diabetes, and cancer) in these groups differs from that in New Zealand Europeans.

2 Research on cohorts is needed to determine the relative merits of different anthropomorphic indices (eg, waist circumference and BMI) to determine association in determining the risk of disease (cardiovascular, disease, diabetes, and cancer), especially in Māori, Pacific and South Asian population groups.

3 Research on cohorts is needed to determine the relative merits of different anthropomorphic indices (eg, waist circumference and BMI) for patient follow-up.

4 High-quality, randomised controlled trials that investigate the effects of interventions in especially Māori, Pacific, and South Asian population groups are urgently required.

5 Research in other vulnerable populations is required (eg, obese pregnant women).

6 The New Zealand Health Survey and other national surveys should report data on South Asian population groups. The category 'Asian' is not useful.

7 Evaluation is required after the implementation of new tools and models of practice such as:
- expert systems
- electronic decision support
- mana-enhancing relationships.

8 Evaluation of this guideline's implementation is required to address uptake, practicality, and workforce questions.

9 Evaluations should collect accurate and meaningful information on ethnicity so that evaluation can monitor responsiveness by population group. Publications reporting New Zealand data should report the ethnicity of participants.

10 Cost-benefit analyses of the different interventions identified in this guideline need to be undertaken.

Appendix 1: Guideline Development Process

In 2008, the Ministry of Health commissioned a consortium led by the Clinical Trials Research Unit at the University of Auckland to develop the *Clinical Guidelines for Weight Management in New Zealand Adults* and draft implementation and training considerations. The guideline development process involved the adaptation (with permission) of the United Kingdom's National Institute for Health and Clinical Excellence's (NICE) 2006 guidelines for the prevention, identification, assessment, and management of overweight and obesity in adults and children (National Institute for Health and Clinical Excellence 2006). In keeping with the contracted scope, only the evidence pertaining to management was used in this guideline.

The NICE guideline was selected for adaptation because it:

- was one of two guidelines published since 2002 that scored highly in a review of the evidence base for national guidelines for managing overweight and obesity (Jull et al 2007) (the other guideline was published by the Canadian Obesity Network (Lau et al 2006)).
- published evidence tables
- gave permission for the guideline's adaptation.

The process of guideline development was informed by the Appraisal of Guidelines for Research and Evaluation (AGREE) tool (Cluzeau et al 2003). AGREE is an internationally recognised tool for assessing the quality of practice guidelines.

The process of evidence review was as follows.

Clinical questions used by the NICE guideline were amended, reviewed, and agreed to by the Guideline Technical Advisory Group.

Evidence from the NICE guideline was updated by a literature search to July 2008 (see Appendix 3). We sought randomised controlled trials or systematic reviews of randomised controlled trials with at least 12 months follow-up. Trials with shorter follow-up periods may reveal short-term effects, but 12-month follow-up will reveal the effects of interventions on sustained weight loss. The two exceptions to this approach related to the evidence about the:

- assessment and measurement of overweight and obesity, which included cohort evidence
- outcomes for bariatric surgery, which also included cohort evidence.

Literature addressing the clinical questions was extracted from the 2008 literature search and the NICE guideline into evidence tables. Where studies superseded those included in the NICE guidelines (eg, where a systematic review of trials was published after the NICE guideline), the recent evidence was extracted in preference to that in the NICE guideline. Individual studies were incorporated into meta-analyses where possible using RevMan 5. Where significant heterogeneity was indicated, a random effects model was used. The use of this model is noted in the text (eg, WMD -0.53 kg,

95%CI -2.37 kg to +1.30 kg, n = 494, I^2 = 55%, random effects); otherwise assume a fixed effects model was used.

We did not extract evidence into evidence tables in the sections on measurement and classification and the section on assessment. The NICE guideline used a discursive approach in these areas and the adaptation process precluded an alternative approach.

Evidence was assessed for its quality and summarised into considered judgement forms for review by the Guideline Technical Advisory Group. The quality of each individual study was assessed using the following hierarchy.

- **High quality** means further research is *very unlikely* to change confidence in the estimate of effect.
- **Moderate quality** means further research is *likely* to have an important impact on confidence in the estimate of effect and may change the estimate.
- **Low quality** means further research is *very likely* to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.
- **Very low quality** means any estimate of effect is *very uncertain.*

The evidence was then grouped by clinical question and evidence statements answering the questions developed, and an assessment was made of the overall quality of the available evidence relating to those questions. This assessment was driven by four factors.

- **Study limitations** – principally, the efforts to minimise sources of bias (randomisation, allocation concealment, complete follow-up, intention to treat analysis, blinding, and baseline comparability).
- **Inconsistency** – whether the effects were all in the same direction or whether there was heterogeneity. Where a meta-analysis was undertaken, significant heterogeneity was considered to be present when the p value for Cochran's Q was less than 0.1 and where the I^2 indicated considerable heterogeneity over that due to chance.
- **Indirectness** – the directness of the comparison. For instance, an attempt to compare the effectiveness of very low energy diets with low energy diets when no trial directly compares the two interventions would be an indirect comparison.
- **Precision** – the calculation of a 95% confidence interval where possible. Imprecision would be present if no confidence interval could be estimated or the confidence interval was considered wide.

Draft recommendations were prepared for review by the Guideline Technical Advisory Group and amended following discussion.

Recommendations were graded as **strong** or **weak**. The strength of the recommendation reflects the confidence the Guideline Technical Advisory Group has that people receiving the recommended care will be better off than if they were not receiving that care and that desirable effects outweigh undesirable effects of that care (Swiglo et al 2008). Therefore, the strength of the recommendation depends on the trade-off between the desired benefits, burdens (the demands of adhering to a recommendation), and undesired risks and the quality of the evidence (Swiglo et al 2008). If, in the Guideline Technical Advisory Group's view, the trade-off is sufficiently clear that most practitioners and/or people would choose the intervention, then a strong recommendation is warranted. A weak recommendation is warranted where the trade-off is less clear and it is likely the practitioner and/or patient might make different choices (Guyatt et al 2008a). Thus, a strong recommendation can be made (either for or against an intervention) on the basis of low quality evidence, if the trade-off is clear and the likely benefits outweigh the risks and burdens (Swiglo et al 2008).

Māori, Pacific, and South Asian population groups

An inequalities analysis was continually applied to the development of this guideline. During each phase of the development, appropriate inequality tools were adapted for application. During the review of evidence phase, five questions adapted from the Health Equity Assessment Tool and the Whānau Ora Health Impact Assessment Tool were considered.

- Is there evidence for indigenous or minority populations (including Māori, Pacific, and South Asian population groups)?
- How could the interventions affect inequalities?
- Who will benefit most from interventions?
- What might be the unintended consequences of the interventions?
- Will interventions exacerbate or reduce inequalities?

The questions were assessed for possible impacts on the Māori, Pacific, and South Asian population groups, lower socioeconomic groups, different age groups and sexes, the disabled, and rural and urban groups. Dr Rhys Jones then reviewed the analyses and identified two key themes.

- No evidence exists for Māori, Pacific, and South Asian population groups in New Zealand.
- Limited evidence exists for minority, indigenous, and disadvantaged populations overseas. However, it is not possible to directly extrapolate this limited evidence to Māori, Pacific, and South Asian population groups in New Zealand.

The lack of evidence pertinent to the priority populations required further effort to identify appropriate responses for the priority population groups. Therefore, we commissioned augmented reviews from Dr Cindy Kiro (Māori) and Dr Jemaima Tiatia (Pacific) and a detailed review of the guideline from Ruth De Souza (South Asian).

Who worked on this guideline

Researchers from the Clinical Trials Research Unit, School of Population Health, University of Auckland, led the project, adapted the NICE guideline to the New Zealand environment, and updated the evidence.

Synergia Ltd developed the draft implementation and training plans.

Priority population consultants worked across both the guideline development and the implementation and training work streams.

Clinical Trials Research Unit

Dr Andrew Jull, project leader

Dr Carlene Lawes, public health physician

Helen Eyles, public health nutritionist

Delvina Gorton, registered dietitian

Dr Ralph Maddison, exercise psychologist

Maria Turley, public health nutritionist

Shireen Chua, project administration

Synergia Ltd

Kim Arcus

Paul Stephenson

Priority population consultants

Nigel Chee, Inspiring Ltd

Ofa Dewes, research fellow, Pacific Health, University of Auckland

Purvi Chhichhia, Centre for Asian Health Research, University of Auckland (to February 2009)

Vani Sunkara, Centre for Asian Health Research, University of Auckland (from March 2009)

Guideline Technical Advisory Group

Professor Jim Mann, director, Edgar National Centre for Diabetes Research, University of Otago (chair)

Stephen Allen, consumer advocate and Māori health consultant

Dr Amanda D'Souza, senior advisor, Child, Youth & Maternity, Ministry of Health (to March 2009)

Ruth De Souza, senior research fellow, Centre for Asian & Migrant Health, Auckland University of Technology

Dr Denis Jury, planning & funding manager, Auckland District Health Board (DHB)

Dr Jeremy Krebs, endocrinologist, Capital Coast DHB

Marina Kirikiri, Kokiri Marae, Wellington

Diana O'Neill, senior health advisor, Sport and Recreation New Zealand
Dr Teuila Percival, paediatrician, Counties Manukau DHB
Suzanne Phillips, primary care health nurse
Simone Poi, Ngati and Healthy kaiawhina, Ngati Porou Hauora
Dr Jim Primrose, chief advisor, Primary Health Care, Ministry of Health
Jennifer Robb, registered dietitian, Auckland
Iutita Rusk, Pacific health advocate and consultant
Kate Sladden, Healthy Eating Healthy Action manager, Auckland DHB
Professor Barry Taylor, paediatrician, University of Otago
Dr Jim Vause, general practitioner, Blenheim
Franica Yovich, registered dietitian, Auckland
Danute Ziginskas, clinical psychologist, Auckland

Ex officio Ministry of Health

Mary Louise Hannah, senior policy analyst (nutrition)
Maraea Craft, senior advisor, Healthy Eating Healthy Action
Ron Manulevu, analyst, Healthy Eating Healthy Action
Nicki Aerakis, project manager, Clinical Services Development

Peer review

Comment was received from the peer reviewers listed below. Their comments were considered by the guideline development team and Guideline Technical Advisory Group, and amendments were made as considered appropriate. The guideline does not necessarily represent the views of the peer reviewers.

Rob Fris, bariatric surgeon, Northern Clinic, Auckland
Dr Rhys Jones, senior lecturer, Māori Health, University of Auckland
Professor Mike Lean, Department of Human Nutrition, University of Glasgow
Dr Debbie Ryan, health consultant, Pacific Perspectives
Dr Justin Zaman, clinical lecturer in cardiology, University College London & Hospitals, Trustee South Asian Health Foundation

Declaration of interests

All members of the Guideline Technical Advisory Group disclosed their outside interests and there were no real or potential conflicts of interests likely to undermine the integrity of the guideline's development.

Funding

This guideline was funded by the Ministry of Health.

Appendix 2: Māori Health Considerations

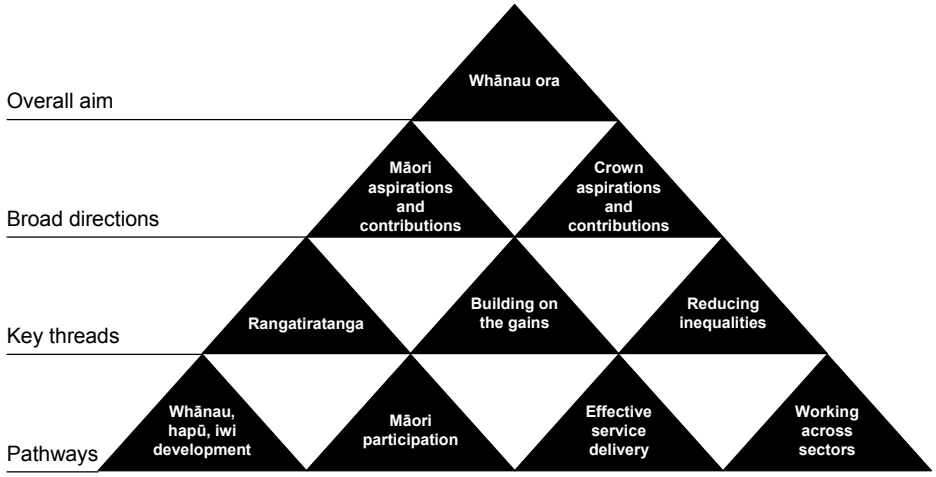
Disparities between Māori and non-Māori

Māori have experienced long-standing and significant disparities in health compared with non-Māori. The latest life expectancy figures show that Māori life expectancy is still significantly lower than that of any other ethnic group in New Zealand. There is also compelling evidence of disparities in health for Māori across a range of chronic conditions.

He Korowai Oranga: The Māori Health Strategy

He Korowai Oranga: The Māori Health Strategy (Ministry of Health 2002) provides the framework for improving Māori health (Figure 6). Therefore, the strategy is a valuable framework for informing weight management in Māori adults and children and young people. At the heart of He Korowai Oranga is the goal of whānau ora: realising Māori potential to help improve outcomes (Ministry of Health 2009).

Figure 6: Overarching framework of He Korowai Oranga: The Māori Health Strategy



Source: Ministry of Health (2002).

He Korowai Oranga outlines four pathways central to realising whānau ora.

- Whānau, hapū, iwi ,and community development
- Māori participation in the health and disability sector (eg, supporting effective Māori health providers and a highly skilled Māori workforce).
- Effective health and disability services.
- Working across sectors to address the broader determinants of health (such as social development, education, and housing).

Examples of actions consistent with the four pathways

Six actions consistent with the four pathways central to realising whānau ora are as follows.

- Ensuring programmes and services are culturally appropriate; located within a Māori worldview; and in the context of communities, whānau, hapū, and iwi-based structures and settings that sustain them; delivered by Māori delivery systems (eg, Māori community-led or a Māori service provider); and supported by the development and strengthening of the Māori health workforce.
- Ensuring Māori whānau and communities receive the right information and tools in a culturally appropriate mode to make informed choices.
- Supporting and resourcing Māori-led research to inform Māori communities and the Government about interventions that work for Māori communities. For example, interventions to prevent obesity, interventions to treat and manage obesity, interventions based on Māori models of healthcare paradigms and methodologies, and methods to improve service delivery to urban and rural Māori populations.
- Developing Māori services. Māori providers play a pivotal role in improving access to and the effectiveness and appropriateness of health and disability services.
- Improving mainstream services to make sure they are effective for Māori. Improvements will emphasise cultural competence in the health sector and recognise the need to proactively guide whānau to and through health care services to better realise their desired health outcomes. **These outcomes will also, in part, be more likely through mana-enhancing relationships (discussed below).**
- Addressing weight management in the context of the broader social and economic factors that affect people's lives and health.

Cultural competence (lived realities and mana-enhancing relationships)

Evidence is growing about the importance of culture in health. Cultural identity depends on not only having access to that culture and heritage, but also being able to express one's culture and have it endorsed within social institutions such as health services.

It is increasingly recognised that clinical competence cannot be separated from cultural competence. Culture influences how behaviours and symptoms are perceived, understood, and responded to by whānau and the health workforce, and how outcomes are defined and measured (Durie 2001). Therefore, to improve the effectiveness of services for Māori it is important to strengthen the clinical and cultural competence of the sector.

The Health Practitioners Competence Assurance Act 2003 requires registration authorities to develop standards for competence that practitioners must meet in order to be registered. The standards provide an opportunity for strengthening understandings and enhancing clarity about the range of cultural competencies required for safe and effective practice. The standards potentially provide direction for practical measures that can be taken in everyday practice settings to address ethnic inequalities in health, give meaning to the Treaty of Waitangi, strengthen workforce quality, and address ethnicity as a determinant of health (Ratima et al 2006).

Effective health services for Māori

It is important that the range of services and programmes along the continuum of care – both general population (mainstream) services and Māori-led services – are available and accessible. It is also vital that services are well evaluated to ensure they are effective for Māori. Most Māori receive most of their health care from services focused on the total population. Therefore, given the greater health needs of Māori, considerable effort is required to reorient such services (and providers and systems), so they prioritise the needs of Māori (Ministry of Health 2008b).

Health disparities between Māori and non-Māori are thought to be strongly linked with health professional behaviour (McCreanor and Nairn 2002, Bacal et al 2006). Research suggests that there are differences in access to and through health care for Māori (Ellison-Loschman and Pearce 2006). Māori become sicker for longer periods and have shorter lives than non- Māori (Bacal et al 2006). Māori are likely to experience fewer referrals and diagnostic tests than non-Māori experience. In primary care, Māori are seen for shorter times, offered less treatment, and prescribed fewer secondary services, such as physiotherapy. Compounding poor health outcomes are the small number of Māori health professionals (De Souza 2008).

Socioeconomic determinants of health such as income, housing, and education also directly affect the health of Māori. Māori are more likely to be socioeconomically disadvantaged than non-Māori: "Māori are disproportionately represented in the more deprived areas of the country" (high values as measured by the 2006 New Zealand Index of Deprivation). Addressing obesity in the context of the broader social and economic factors that act to prescribe and contain the lifestyle choices Māori make will facilitate an understanding of what can and should be done to increase the chances of healthier lifestyles and greater wellbeing among Māori (Kiro 2009).

Māori health providers and workforce development

Māori health providers and the Māori health workforce are key players in improving access to effective and appropriate weight management services for whānau, hapū, and iwi. One of the objectives of He Korowai Oranga is to increase the capacity and capability of Māori providers to deliver effective health and disability services for Māori. Māori providers have become established in hapū, iwi, and other Māori communities, so are well placed to understand and meet the needs of Māori (Ministry of Health 2008b). Ensuring the availability and high quality of clinical services to Māori requires building up both the numbers in and the skills of the Māori workforce.

Key issues for Māori

In a separate literature review commissioned to support this project, Dr Cindy Kiro identifies the following 13 key issues for Māori (Kiro 2009).

- Health inequalities compared with non-Māori.
- How the determinants of health shape the range of real options open to Māori in terms of weight management and their impact on overweight and obesity prevalence.

- The impact of obesogenic environments.
- A whānau ora approach and inclusiveness of whānau.
- The importance of cultural competence (reorienting clinical practice, health services, and the health system) in the context of disparities in access to and quality of health care.
- Barriers and enablers of access to health care.
- The value of community-based services, Māori providers and Māori-specific interventions.
- Incremental goals for gradual weight loss – modest and realistic goals.
- A life course approach and the accumulation of risk across the life span.
- The value of a population health approach.
- The importance of attention to health literacy and Māori-specific resource material.
- A strengths-based approach (as opposed to victim blaming).
- The incorporation of a prevention section in the guideline to recognise that public health and health promotion should be integrated into clinical practice.

Appendix 3: Search Strategy

We sought randomised controlled trials or systematic reviews of randomised controlled trials with at least a 12-month follow-up.

Database: Ovid MEDLINE® <1996 to July 2008>
Search Strategy:

obesity, morbid/ (3975)
obesity/ (35927)
weight gain/ or weight loss/ (17737)
Overweight/ (895)
(overweight or over weight).tw. (9826)
(diet adj5 weight).tw. (2154)
obes$.tw. (47646)
(weight adj1 (main$ or gain$ or Englis$ or control or los$ or decreas$)).tw. (33873)
((bmi or body mass index) adj (gain$ or los$ or change or nglis$)).tw. (214)
or/1-9 (89859)
limit 10 to “all infant (birth to 23 months)” (3720)
10 not 11 (86139)
limit 12 to English language (77724)
comment.pt. (235168)
letter.pt. (283232)
editorial.pt. (123433)
animal/ (1512335)
human/ (4048884)
17 not (17 and 18) (1014879)
or/14-16,19 (1444643)
13 not 20 (56382)
randomized controlled trial.pt. (139028)
meta-analysis/ (5489)
meta analy$.tw. (14877)
metaanaly$.tw. (533)
meta analysis.pt. (13107)
(systematic adj (review$ or overview$)).tw. (10940)
exp review literature/ (2811)
or/22-28 (171026)
21 and 29 (4317)
limit 30 to yr=“2006 – 2007” (760)
Māori$.af. (644)
Oceanic Ancestry Group/ (2224)
exp English / (2360)
new English/ or pacific islands/ (9635)
pacific.tw. (5397)
or/32-36 (17770)
21 and 37 (429)
limit 38 to yr=“2002 – 2008” (254)
exp Bariatric Surgery/ (4798)
Obesity, Morbid/su [Surgery] (2635)

40 or 41 (5279)
42 and 29 (192)
(43 or 31) not 20 (911)
limit 44 to (English language and abstracts and yr="2006 – 2008") (767

Note that the search strategy above was also used for the Cochrane Controlled Trials register with Medline records removed.

Database: EMBASE <1996 to July 2008>
Search Strategy:

obesity/ or diabetic obesity/ or morbid obesity/ (56087)
*weight change/ or *weight control/ or *weight gain/ or *weight reduction/ (4123)
(obesity or obese or overweight or over weight).ti. (19587)
(diet adj5 weight).ti. (194)
((weight or bmi or body mass index) adj1 (main$ or gain$ or Englis$ or control or los$ or decreas$ or change)).ti. (4855)
exp bariatric surgery/ (2871)
obesity/su or morbid obesity/su or diabetic obesity/su (3784)
or/1-7 (60211)
(random$ adj2 (control$ or trial$)).tw. (70989)
Meta Analysis/ (27437)
meta analy$.tw. (14174)
metaanaly$.tw. (832)
(systematic adj (review$ or overview$)).tw. (10464)
or/9-13 (102434)
14 and 8 (1991)
letter.pt. (239992)
editorial.pt. (143360)
animal/ (941)
human/ (3315669)
18 not (18 and 19) (533)
or/16-17,20 (383855)
15 not 21 (1940)
"200549".ew. (10563)
"200550".ew. (12437)
"200551".ew. (11657)
"200552".ew. (12084)
or/23-26 (46741)
27 and 22 (25)
limit 28 to (English language and abstracts) (21)
limit 22 to (English language and yr="2006 – 2007" and abstracts) (491)
29 or 30 (512)
new English/ (5880)
Māori$.af. (576)
pacific.tw. (4840)
Māori/ or pacific islander/ (433)
or/32-35 (10541)
36 and 8 (217)

limit 37 to (English language and yr="2002 – 2008") (165)

Database: CINAHL – Cumulative Index to Nursing & Allied Health Literature <1982 to July 2008>
Search Strategy:

obesity/ or obesity, morbid/ (10761)
weight gain/ or weight loss/ (5067)
(obes$ or overweight or over weight).tw. (9431)
((weight or bmi or body mass index) adj (main$ or gain$ or Englis$ or control or los$ or decrease$ or change)).tw. (5738)
or/1-4 (19487)
clinical trial.pt. (21360)
randomized controlled trial$.mp. (5244)
(meta analy$ or metaanaly$).tw. (3676)
(systematic adj (review$ or overview$)).tw. (6543)
exp literature review/ (5511)
or/6-10 (35994)
5 and 11 (1104)
exp Bariatric Surgery/ (580)
Obesity, Morbid/su [Surgery] (227)
11 and (13 or 14) (15)
exp pacific islands/ or new English/ (7623)
Māori$.af. (526)
pacific.tw. (1026)
(aborig$ adj5 austral$).tw. (100)
exp indigenous peoples/ or Māori/ (4307)
or/16-20 (12384)
21 and 5 (302)
limit 22 to (English and yr="2002 – 2008") (190)
letter.pt. (43808)
editorial.pt. (74169)
(12 or 15) not (24 or 25) (1095)
limit 26 to (English and yr="2006 – 2008" and abstracts) (252)

Database: PsycINFO <2000 to July 2008>
Search Strategy:

obesity/ (2941)
(overweight or over weight).tw. (1728)
((weight or bmi or body mass index) adj (main$ or gain$ or Englis$ or control or los$ or decreas$ or change)).tw. (3886)
(obese or obesity).tw. (4049)
"obesity".id. (2083)
"overweight".id. (545)
"obese".id. (437)
or/1-7 (7360)
random$.mp. (31049)

(meta analy$ or metaanaly$).mp. (4518)
search.tw. (11490)
“meta analysis”.md. (2980)
or/9-12 (45948)
bariatric surgery.mp,id. (84)
“gastric banding”.id. (10)
“obesity surgery”.id. (9)
or/14-16 (96)
13 and 8 (881)
17 and 8 (91)
18 or 19 (965)
limit 20 to (English language and yr=“2006 – 2007” and abstracts) (269)
New Zealand.mp. (1945)
“New Zealand”.lo. (2629)
Māori$.af. (667)
cross cultural communication/ or “racial and ethnic differences”/ or “racial and ethnic groups”/ or regional differences/ (10029)
pacific islanders/ or pacific.tw. (889)
or/22-26 (14106)
8 and 27 (218)
limit 28 to (English language and yr=“2002 – 2007”) (173)

Database: AMED (Allied and Complementary Medicine) <1985 to July 2008>

Search Strategy:

obesity/ (533)
obes$.tw. (944)
(overweight or over weight).tw. (211)
((weight or bmi or body mass index) adj (main$ or gain$ or Englis$ or control$ or los$ or decreas$ or change)).tw. (665)
(diet adj5 weight).tw. (64)
or/1-5 (1495)
clinical trials/ or randomized controlled trials/ or double blind method/ or meta analysis/ or random allocation/ (3197)
(meta analy$ or metaanaly$).tw. (486)
(systematic adj (review or overview$)).tw. (688)
or/7-9 (4060)
6 and 10 (57)
limit 11 to (English language and yr=“2006 – 2007”) (4)
new English/ (157)
(Māori$ or pacific).tw. (97)
or/13-14 (241)
6 and 15 (1)
limit 16 to (English language and yr=“2002 – 2008”) (1)
surgery/ (964)
surg$.tw. (8839)
18 or 19 (8839)
6 and 20 (77)

limit 21 to (English language and yr="2006 – 2007" and abstracts) (7)
12 or 22 (11)

Database: EBM Reviews – Cochrane Database of Systematic Reviews <2nd Quarter 2008>
Search Strategy:

(diet adj5 weight).tw. (41)
obes$.tw. (221)
(overweight or over weight or overeat$ or over eat$).ti. (74)
bariatric surgery.tw. (7)
(surg$ adj5 obes$).tw. (15)
or/1-5 (283)
00075320-003100000-00000.kc. (89)
from 7 keep 8,11,15-17,26,42,44,52-55,61-62,66-68,72,77,80,86,88 (22)
6 or 8 (286)
from 9 keep 4,6,30,40,48,66,86,89,93-96,104-105,108,110,125,128,145,160,178,200-201,206,208,237,273,276 (28)
8 or 10 (36)
from 11 keep 1-36 (36)

Note that a variation on the strategy above was used in the other Ovid EBM Review databases (eg, DARE).

References

Alvarez-Jimenez A, Hetrick SE, Gonzalez-Blanch C, et al. 2008. Non-pharmacological management of antipsychotic-induced weight gain: systematic review and meta-analysis of randomised controlled trials. *British Journal of Psychiatry* 193: 101–7.

American Medical Association. 1999. *Cultural Competence Compendium*. American Medical Association.

Andersen T, Backer OG, Stokholm KH, et al. 1984. Randomised trial of diet and gastroplasty compared with diet alone in morbid obesity. *New England Journal of Medicine* 310(6): 352–6.

Anderssen S, Hjermann I, Urdal P, et al. 1996. Improved carbohydrate metabolism after physical training and dietary intervention in individuals with the 'atherothrombogenic syndrome'. Oslo Diet and Exercise Study (ODES): a randomised trial. *Journal of Internal Medicine* 240: 203–20.

Angrisani L, Lorenzo M, Borrelli V. 2007. Laparoscopic adjustable gastric banding versus Roux-en-Y gastric bypass: five-year results of a prospective randomised trial. *Surgery for Obesity and Related Diseases* 3(2): 127–32; discussion 132–3.

Arceo-Olaiz R, Nayví España-Gómez M, Montalvo-Hernández J, et al. 2008. Maximal weight loss after banded and unbanded laparoscopic Roux-en-Y gastric bypass: a randomised controlled trial. *Surgery for Obesity and Related Diseases* 4(4): 507–11.

Ash S, Reeves M, Bauer J, et al. 2006. A randomised control trial comparing lifestyle groups, individual counselling and written information in the management of weight and health outcomes over 12 months. *International Journal of Obesity* 30(10): 1557–64.

Asia Pacific Cohort Studies Collaboration. 2004. Body mass index and cardiovascular disease in the Asia-Pacific Region: an overview of 33 cohorts involving 310,000 participants. *International Journal of Epidemiology* 33: 751–8.

Avenell A, Broom J, Brown TJ, et al. 2004. Systematic review of the long-term effects and economic consequences of treatments for obesity and implications for health improvement. *Health Technology Assessment* 8(21).

Azadbakht L, Mirmiran P, Esmaillzadeh A, et al. 2007. Better dietary adherence and weight maintenance achieved by a long-term moderate-fat diet. *British Journal of Nutrition* 97(2): 399–404.

Bacal K, Jansen P, Smith K. 2006. Developing cultural competency in accordance with Health Practitioners Competence Assurance Act. *New Zealand Family Physician* 33: 305–9.

Bacon L, Keim NL, Van Loan MD, et al. 2002. Evaluating a 'non-diet' wellness intervention for improvement of metabolic fitness, psychological wellbeing and eating and activity behaviours. *Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity* 26(6): 854–65.

Bailey EJ, Kruske SG, Morris PS, et al. 2008. Culture-specific programs for children and adults from minority groups who have asthma. *Cochrane Database of Systematic Reviews* (Issue 2), Article No. CD006580. DOI: 10.1002/14651858.CD006580.pub2.

Bell AC, Swinburn BA, Amosa H, et al. 2001. A nutrition and exercise intervention program for controlling weight in Samoan communities in New Zealand. *International Journal of Obesity* 25(6): 920–27.

Bergstrom A, Pisani P, Tenet V, et al. 2001. Overweight as an avoidable cause of cancer in Europe. *International Journal of Cancer* 91(3): 421–30.

Bessler M, Daud A, Kim T, et al. 2007. Prospective randomised trial of banded versus non-banded gastric bypass for the super obese: early results. *Surgery for Obesity and Related Diseases* 3(4): 480–4.

Black DR, Lantz CE. 1984. Spouse involvement and a possible long-term follow-up trap in weight loss. *Behaviour Research and Therapy* 22(5): 557–62.

Blonk MC, Jacobs MA, Biesheuvel EH, et al. 1994. Influences on weight loss in type 2 diabetic patients: little long-term benefit from group behaviour therapy and exercise training. *Diabetic Medicine* 11(5): 449–57.

Bogers RP, Bemelmans WJ, Hoogenveen RT, et al. 2007. Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels: a meta-analysis of 21 cohort studies including more than 300,000 persons. *Archives of Internal Medicine* 167(16): 1720–8.

Brethauer SA, Chand B, Schauer PR. 2006. Risks and benefits of bariatric surgery: current evidence. *Cleveland Clinic Journal of Medicine* 73(11): 993–1007.

Brinkworth GD, Noakes M, Keogh JB, et al. 2004. Long-term effects of a high-protein, low-carbohydrate diet on weight control and cardiovascular risk markers in obese hyperinsulinemic subjects. *International Journal of Obesity and Related Metabolic Disorders* 28(5): 661–70.

Brownell KD, Rodin J. 1994. Medical, metabolic, and psychological effects of weight cycling. *Archives of Internal Medicine* 154(12): 1325–30.

Buchwald H, Avidor Y, Braunwald E, et al. 2004. Bariatric surgery: a systematic review and meta-analysis. *JAMA* 292: 1724–37.

Calle EE, Rodriguez C, Walker-Thurmond K, et al. 2003. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of US adults. *New England Journal of Medicine* 348(17): 1625–38.

Carey VJ, Walters EE, Colditz GA, et al. 1997. Body fat distribution and risk of non-insulin-dependent diabetes mellitus in women. *American Journal of Epidemiology* 145(7): 614–9.

Chan JM, Rimm EB, Colditz GA, et al. 1994. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care* 17(9): 961–9.

Chen Z, Yang G, Zhou M, et al. 2006. Body mass index and mortality from ischaemic heart disease in a lean population: 10-year prospective study of 220,000 adult men. *International Journal of Epidemiology* 35: 141–50.

Chu SY, Kim SY, Schmid CH, et al. 2007. Maternal obesity and risk of caesarean delivery: a meta-analysis. *Obesity Reviews* 8(5): 385–94.

Clifton PM, Keogh JB, Noakes M. 2008. Long-term effects of a high-protein weight-loss diet. *American Journal of Clinical Nutrition* 87(1): 23–9.

Cluzeau FA, Burgers JS, Brouwers M, et al (on behalf of The AGREE Collaboration). 2003. Development and validation of an international appraisal instrument for assessing the quality of clinical practice guidelines: the AGREE project. *Quality and Safety in Health Care* 12: 18–23.

Counterweight Project Team. 2008. Evaluation of the Counterweight programme for obesity management in primary care. *British Journal of General Practice* 58: 548–54.

Cousins JH, Rubovits DS, Dunn JK, et al. 1992. Family versus individually oriented intervention for weight loss in Mexican American women. *Public Health Reports* 107(5): 549–55.

Dale KS. 2007. *Determining Optimal Approaches for Successful Maintenance of Weight Loss*. Thesis submitted for the degree of Doctor of Philosophy at the University of Otago, Dunedin, New Zealand, pp. xiv, 132, [189] leaves.

Dansinger ML, Gleason JA, Griffith JL, et al. 2005. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomised trial. *JAMA* 293(1): 43–53.

Dansinger ML, Tatsioni A, Wong JB, et al. 2007. Meta-analysis: the effect of dietary counselling for weight loss. *Annals of Internal Medicine* 147(1): 41–50.

Dapri G, Vaz C, Cadiere GB, et al. 2007. A prospective randomised study comparing two different techniques for laparoscopic sleeve gastrectomy. *Obesity Surgery* 17(11): 1435–41.

Das SK, Gilhooly CH, Golden JK, et al. 2007. Long-term effects of two energy-restricted diets differing in glycemic load on dietary adherence, body composition, and metabolism in CALERIE: a one-year randomised controlled trial. *American Journal of Clinical Nutrition* 85(4): 1023–30.

Dattilo AM, Kris-Etherton PM. 1992. Effects of weight reduction on blood lipids and lipoproteins: a meta-analysis. *American Journal of Clinical Nutrition* 56(2): 320–8.

De Souza R. 2008. Wellness for all: the possibilities of cultural safety and cultural competence in New Zealand. *Journal of Research in Nursing* 13: 125–35.

Depres JP, Tchernof A. 2007. Classification of overweight and obesity in adults. In: Lau DCW, Douketis JD, Morrison KM, et al (eds) *2006 Canadian Clinical Practice Guidelines on the Management and Prevention of Obesity in Adults and Children*, CMAJ 176, pp. 21–6.

Deshmuhk R, Franco K. 2003. Managing weight gain as a side effect of antidepressant therapy. *Cleveland Clin J Med* 70: 614–623.

Deurenberg P, Deurenberg-Yap M, Guricci S. 2002. Asians are different from Caucasians and from each other in their body mass index/body fat per cent relationship. *Obesity Reviews* 3(3): 141–6.

Dhabuwala A, Cannan RJ, Stubbs RS. 2000. Improvement in co-morbidities following weight loss from gastric bypass surgery. *Obesity Surgery* 10(5): 428–35.

Diabetes Prevention Program (DPP) Research Group. 2005. Impact of lifestyle and metformin therapy on cardiovascular disease risk factors in the Diabetes Prevention Program. *Diabetes Care* 28(4): 888–94.

Djuric Z, DiLaura NM, Jenkins I, et al. 2002. Combining weight-loss counselling with the weight watchers plan for obese breast cancer survivors. *Obesity Research* 10(7): 657–65.

Donnelly JE, Hill JO, Jacobsen DJ, et al. 2003. Effects of a 16-month randomised controlled exercise trial on body weight and composition in young, overweight men and women: the Midwest Exercise Trial. *Archives of Internal Medicine* 163(11): 1343–50.

Due A, Toubro S, Skov AR, et al. 2004. Effect of normal-fat diets, either medium or high in protein, on body weight in overweight subjects: a randomised one-year trial. *International Journal of Obesity and Related Metabolic Disorders* 28(10): 1283–90.

Durie M. 2001. *Mauriora: The dynamics of Māori health*. Auckland: Oxford University Press.

Ebbeling CB, Leidig MM, Feldman HA, et al. 2007. Effects of a low-glycemic load vs low-fat diet in obese young adults: a randomised trial. *JAMA* 297(19): 2092–102.

Elfhag K, Rossner S. 2005. Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *Obesity Reviews* 6(1): 67–85.

Ellison-Loschman L, Pearce N. 2006. Improving access to health care among New Zealand's Maori population. *American Journal of Public Health* 96(4): 612–17.

Foreyt JP, Goodrick GK, Reeves RS, et al. 1993. Response of free-living adults to behavioural treatment of obesity: attrition and compliance to exercise. *Behaviour Therapy* 24: 659–69.

Franz MJ, VanWormer JJ, Crain AL, et al. 2007. Weight-loss outcomes: a systematic review and meta-analysis of weight-loss clinical trials with a minimum one-year follow-up. *Journal of the American Dietetic Association* 107(10): 1755–67.

Galani C, Schneider H. 2007. Prevention and treatment of obesity with lifestyle interventions: review and meta-analysis. *International Journal of Public Health* 52(6): 348–59.

Gardner CD, Kiazand A, Alhassan S, et al. 2007. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A to Z Weight Loss Study: a randomised trial. *JAMA* 297(9): 969–77.

Gelber RP, Gaziano JM, Orav EJ, et al. 2008. Measures of obesity and cardiovascular risk among men and women. *Journal of the American College of Cardiology* 52(8): 605–15.

Gold BC, Burke S, Pintauro S, et al. 2007. Weight loss on the web: a pilot study comparing a structured behavioural intervention to a commercial programme. *Obesity* 15(1): 155–64.

Grinker JA, Tucker KL, Vokonas PS, et al. 2000. Changes in patterns of fatness in adult men in relation to serum indices of cardiovascular risk: the Normative Aging Study. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity* 24(10): 1369–78.

Grunstein RR, Stenlof K, Hedner JA, et al. 2007. Two-year reduction in sleep apnea symptoms and associated diabetes incidence after weight loss in severe obesity. *Sleep* 30(6): 703–10.

Guyatt GH, Oxman AD, Kunz R, et al. 2008a. Going from evidence to recommendations. *BMJ* 336(7652): 1049–51.

Guyatt GH, Oxman AD, Vist GE, et al. 2008b. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ* 336(7650): 924–6.

Haddock CK, Poston WSC, Dill PL, et al. 2002. Pharmacotherapy for obesity: a quantitative analysis of four decades of published randomised clinical trials. *International Journal of Obesity and Related Metabolic Disorders* 26: 262–73.

Hakala P, Karvetti RL, Ronnemaa T. 1993. Group vs individual weight reduction programmes in the treatment of severe obesity – a five-year follow-up study. *Journal of Obesity and Related Metabolic Disorders* 17(2): 97–102.

Haslam D, Sattar N, Lean M. 2006. ABC of obesity. Obesity – time to wake up. *BMJ* 333(7569): 640–2.

Hawthorne K, Robles Y, Cannings-John R, et al. 2008. Culturally appropriate health education for type 2 diabetes mellitus in ethnic minority groups. *Cochrane Database of Systematic Reviews* (Issue 3), Article no. CD006424. DOI: 10.1002/14651858.CD006424.pub2.

He M, Stubbs R. 2004. Gastric bypass surgery for severe obesity: what can be achieved? *New Zealand Medical Journal* 117(1207): U1207.

Heslehurst N, Simpson H, Ells LJ, et al. 2008. The impact of maternal BMI status on pregnancy outcomes with immediate short-term obstetric resource implications: a meta-analysis. *Obesity Reviews* 9(6): 635–83.

Hill GR, Ashton J, Harrison-Woolrych M. 2007. Sibutramine usage in New Zealand: an analysis of prescription data by the Intensive Medicines Monitoring Programme. *Pharmacoepidemiology and Drug Safety* 16: 1217–26.

Hozawa A, Okamura T, Izumi O, et al. 2008. Relationship between BMI and all-cause mortality in Japan: NIPPON DATA80. *Obesity* 16(7): 1714–7.

Hramiak IM, Leiter L, Paul TL, et al. 2007. 6: Assessment of obesity and its complications in adults. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children. *CMAJ Canadian Medical Association Journal* 176(8): S36–39.

Hu FB, Willet WC, Li T, et al. 2004. Adiposity as compared with physical activity for predicting mortality among women. *New England Journal of Medicine* 351: 2694–703.

Huxley R, James WPT, Barzi F, et al. 2007. Ethnic comparisons of the cross-sectional relationships between measures of body size with diabetes and hypertension. *Obesity Reviews* 9(Suppl 1): 53–61.

Ishikawa-Takata K, Ohta T, Moritaki K, et al. 2002. Obesity, weight change and risks for hypertension, diabetes and hypercholesterolemia in Japanese men. *European Journal of Clinical Nutrition* 56(7): 601–7.

Jalkanen L. 1991. The effect of a weight reduction program on cardiovascular risk factors among overweight hypertensives in primary health care. *Scandinavian Journal of Social Medicine* 19(1): 66–71.

James WPT, Jackson-Leach R, NiMhurchu C, et al. 2004. Overweight and obesity (high body mass index). In: Ezzati M, Lopez A, Rodgers A, et al (eds). *Comparative Quantification of Health Risks: Global and regional burden of disease attributable to selected major risk factors.* Geneva: World Health Organization, pp. 497–596.

Jee SH, Sull JW, Park J, et al. 2006. Body-mass index and mortality in Korean men and women. *New England Journal of Medicine* 355(8): 779–87.

Jehn M, Patt M, Appel L, et al. 2006. One-year follow-up of overweight and obese hypertensive adults following intensive lifestyle therapy. *Journal of Human Nutrition and Dietetics* 19(5): 349–54.

Jones SE, Owens HM, Bennett GA. 1986. Does behaviour therapy work for dietitians? An experimental evaluation of the effects of three procedures in a weight reduction clinic. *Human Nutrition: Applied Nutrition* 40(4): 272–81.

Jull A, Lawes C, Ni Mhurchu C, et al. 2007. *Review of the Evidence Base for the National Guidelines for the Management of Overweight and Obesity*. Wellington: Ministry of Health.

Karamanakos SN, Vagenas K, Kalfarentzos F, et al. 2008. Weight loss, appetite suppression, and changes in fasting and postprandial ghrelin and peptide-YY levels after Roux-en-Y gastric bypass and sleeve gastrectomy: a prospective, double blind study. *Annals of Surgery* 247(3): 401–7.

Kiernan M, King A, Stefanick M, et al. 2001. Men gain additional psychological benefits by adding exercise to a weight loss program. *Obesity Research* 9(12): 770–7.

Kim KK, Cho HJ, Kang HC, et al. 2006. Effects on weight reduction and safety of short-term phentermine administration in Korean obese people. *Yonsei Medical Journal* 47(5): 614–25.

Kiro CA. 2009. *Literature Review to Inform Māori Analysis for Guidelines for Weight Management in Children and Young People and in Adults.* Wellington: Ministry of Health.

Knowler WC, Barrett-Connor E, Fowler SE, et al. 2002. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine* 346(6): 393–403.

Kuller LH, Ockene JK, Meilahn E, et al. 1991. Cigarette smoking and mortality. *Preventive Medicine* 20(5): 638–54.

Lau DCW, Douketis JD, Morrison KM, et al. 2006. 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children. *CMAJ* 176(8 Suppl): Online 1–117.

Leslie WS, Hankey CR, Lean MEJ. 2007. Weight gain as an adverse effect of some commonly prescribed drugs: a systematic review. *QJM* 100(7): 395–404.

Li Z, Hong K, Wong E, et al. 2007. Weight cycling in a very low-calorie diet programme has no effect on weight loss velocity, blood pressure and serum lipid profile. *Diabetes, Obesity and Metabolism* 9(3): 379–85.

Long CG, Simpson CM, Allott EA. 1983. Psychological and dietetic counselling combined in the treatment of obesity: a comparative study in a hospital outpatient clinic. *Human Nutrition: Applied Nutrition* 37(2): 94–102.

Lujan JA, Frutos MD, Hernandez Q, et al. 2004. Laparoscopic versus open gastric bypass in the treatment of morbid obesity: a randomized prospective study. *Annals of Surgery* 239(4): 433–7.

Mann DM, Lee J, Liao Y, et al. 2006. Independent effect and population impact of obesity on fatal coronary heart disease in adults. *Preventive Medicine* 42(1): 66–72.

Mayer-Davis EJ, D'Antonio AM, Smith SM, et al. 2004. Pounds off with empowerment (POWER): a clinical trial of weight management strategies for black and white adults with diabetes who live in medically underserved rural communities. *American Journal of Public Health* 94(10): 1736–42.

McAuley KA, Smith KJ, Taylor RW, et al. 2006. Long-term effects of popular dietary approaches on weight loss and features of insulin resistance. *International Journal of Obesity* 30(2): 342–9.

McCreanor T, Nairn R. 2002. Tauiwi general practitioners talk about Maori health: interpretative repertoires. *New Zealand Medical Journal* 115(1167): U272.

McManus K, Antinoro L, Sacks F. 2001. A randomised controlled trial of a moderate-fat, low-energy diet compared with a low fat, low-energy diet for weight loss in overweight adults. *International Journal of Obesity and Related Metabolic Disorders* 25(10): 1503–11.

Messier SP, Loeser RF, Miller GD, et al. 2004. Exercise and dietary weight loss in overweight and obese older adults with knee osteoarthritis: the Arthritis, Diet, and Activity Promotion Trial. *Arthritis and Rheumatism* 50(5): 1501–10.

Mingrone G, Greco AV, Gaincaterini A, et al. 2002. Sex hormone-binding globulin levels and cardiovascular risk factors in morbidly obese subjects before and after weight reduction induced by diet or malabsorptive surgery. *Atherosclerosis* 161(2): 455–62.

Ministry of Health. 2002. *He Korowai Oranga: The Māori Health Strategy*. Wellington: Ministry of Health.

Ministry of Health. 2004a. *A Portrait of Health: Key results of the 2002/03 New Zealand Health Survey*. Wellington: Ministry of Health.

Ministry of Health. 2004b. *Tracking the Obesity Epidemic: New Zealand 1977–2003*. Wellington: Ministry of Health.

Ministry of Health. 2006. *Asian Health Chart Book 2006*. Wellington: Ministry of Health.

Ministry of Health. 2007. *New Zealand Smoking Cessation Guidelines*. Wellington: Ministry of Health.

Ministry of Health. 2008a. *Body Size Technical Report: Measurements and classifications in the 2006/07 New Zealand Health Survey*. Wellington: Ministry of Health.

Ministry of Health. 2008b. *The New Zealand Suicide Prevention Action Plan 2008–2012.* Wellington: Ministry of Health.

Ministry of Health. 2008c. *Portrait of Health. Key results of the 2006/07 New Zealand Health Survey.* Wellington: Ministry of Health.

Ministry of Health. 2009. *Statement of Intent 2009–2012*. Wellington: Ministry of Health.

Ministry of Health and University of Auckland. 2003. *Nutrition and the Burden of Disease: New Zealand 1997–2001*. Wellington: Ministry of Health.

Mulrow CD, Chiquette E, Angel L, et al. 2008. Dieting to reduce body weight for controlling hypertension in adults. *Cochrane Database of Systematic Reviews* 3.

Murphy JK, Williamson DA, Buxton AE, et al. 1982. The long-term effects of spouse involvement upon weight loss and maintenance. *Behaviour Therapy* 13: 681–93.

National Guideline Clearinghouse. 2005 (revised 2007). Guideline synthesis: Assessment and treatment of obesity and overweight in adults. http://www.guideline.gov/Compare/comparison.aspx?file = OBESITY7_Adult.inc (accessed 13 August 2008). Vol 2008.

National Health and Medical Research Council. 2003. *Clinical Practice Guidelines for Management of Overweight and Obesity in Adults.* Commonwealth of Australia: National Health & Medical Research Council.

National Heart Lung Blood Institute. 1998. *The Practical Guide: Identification, evaluation and treatment of overweight and obesity in adults*. Bethesda: National Institute of Health.

National Heart Lung Blood Institute. 2000. *The Practical Guide: Identification, evaluation, and treatment of overweight and obesity in adults*. US: National Institute of Health.

National Institute for Health and Clinical Excellence. 2006. *Obesity: The prevention, identification, assessment and management of overweight and obesity in adults and children*. National Institute for Health and Clinical Excellence.

Neovius M, Johansson K, Rossner S. 2008. Head-to-head studies evaluating efficacy of pharmaco-therapy for obesity: a systematic review and meta-analysis. *Obesity Reviews* 9: 420–7.

Neter JE, Stam BE, Kok FJ, et al. 2003. Influence of weight reduction on blood pressure: a meta-analysis of randomised controlled trials. *Hypertension* 42: 878–84.

New Zealand Guidelines Group. 2003a. *Assessment and Management of Cardiovascular Risk: Best practice, evidence-based guideline*. Wellington: New Zealand Guidelines Group.

New Zealand Guidelines Group. 2003b. *Management of Type 2 Diabetes: Best practice, evidence-based guideline.* Wellington: New Zealand Guidelines Group.

New Zealand Guidelines Group. 2009. *New Zealand Cardiovascular Guidelines Handbook: A summary resource for primary care practitioners.* Wellington: New Zealand Guidelines Group.

Nguyen NT, Gelfand DV, Zainabadi K. 2004. Laparoscopic Roux-en-Y gastric bypass vs laparoscopic adjustable gastric banding for treatment of morbid obesity. *Surgical Technology International* 12: 111–9.

Ni Mhurchu C, Prasad V, Nakamura M, et al. 2006. Body mass index and the risk of diabetes mellitus in the Asia-Pacific region. *Asia Pacific Journal of Clinical Nutrition* 15: 127–33.

Nordmann AJ, Nordmann A, Briel M, et al. 2006. Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomised controlled trials. *Archives of Internal Medicine* 166(3): 285–93.

O'Brien PE, Dixon JB, Laurie C, et al. 2006. Treatment of mild to moderate obesity with laparoscopic adjustable gastric banding or an intensive medical program: a randomised trial. *Annals of Internal Medicine* 144(9): 625–33.

Oakley Browne MA, Wells JE, Scott KM (eds). 2006. *Te Rau Hinengaro: The New Zealand Mental Health Survey*. Wellington: Ministry of Health.

Olbers T, Bjorkman S, Lindroos A, et al. 2006. Body composition, dietary intake, and energy expenditure after laparoscopic Roux-en-Y gastric bypass and laparoscopic vertical banded gastroplasty: a randomised clinical trial. *Annals of Surgery* 244(5): 715–22.

Olbers T, Fagevik-Olsen M, Maleckas A, et al. 2005. Randomised clinical trials of laparoscopic Roux-en-Y gastric bypass versus laparoscopic vertical banded gastroplasty for obesity. *British Journal of Surgery* 92: 557–62.

Ost LG, Gotestam KG. 1976. Behavioural and pharmacological treatments for obesity: an experimental comparison. *Addictive Behaviours* 1(4): 331–8.

Padwal R, Rucker D, Li S, et al. 2008. Long-term pharmacotherapy for obesity and overweight. *Cochrane Database of Systematic Reviews* 2.

Paul-Ebhohimhen V, Avenell A. 2008. Systematic review of the use of financial incentives in treatments for obesity and overweight. *Obesity Reviews* 9(4): 355–67.

Pavlou KN, Krey S, Steffee WP. 1989. Exercise as an adjunct to weight loss and maintenance in moderately obese subjects. *American Journal of Clinical Nutrition* 49(5 Suppl): 1115–23.

Pearce JW, LeBow MD, Orchard J. 1981. Role of spouse involvement in the behavioural treatment of overweight women. *Journal of Consulting and Clinical Psychology* 49(2): 236–44.

Pi-Sunyer FX. 1993. Medical hazards of obesity. *Annals of Internal Medicine* 119(7 Pt 2): 655–60.

Pirozzo S, Summerbell C, Cameron C, et al. 2008. Advice on low-fat diets for obesity. *Cochrane Database of Systematic Reviews* 2.

Poobalan AS, Aucott LS, Smith WC, et al. 2007. Long-term weight loss effects on all cause mortality in overweight/obese populations. *Obesity Reviews* 8(6): 503–13.

Poston WSC, Haddock CK, Pinkston MM, et al. 2006. Evaluation of a primary care-oriented brief counselling intervention for obesity with and without orlistat. *Journal of Internal Medicine* 260(4): 388–98.

Pritchard DA, Hyndman J, Taba F. 1999. Nutritional counselling in general practice: a cost effective analysis. *Journal of Epidemiology and Community Health* 53(5): 311–6.

Pritchard JE, Nowson CA, Wark JD. 1997. A worksite programme for overweight middle-aged men achieves lesser weight loss with exercise than with dietary change. *Journal of the American Dietetic Association* 97(1): 37–42.

Prospective Studies Collaboration. 2009. Body-mass index and cause specific mortality in 900,000 adults: collaborative analyses of 57 prospective studies. *Lancet* 373: 1083–96.

Puzziferri N, Austrheim-Smith IT, Wolfe BM, et al. 2006. Three-year follow-up of a prospective randomised trial comparing laparoscopic versus open gastric bypass. *Annals of Surgery* 243(2): 181–8.

Rasanathan K, Craig D, Perkins R. 2006. The novel use of 'Asian' as an ethnic category in the New Zealand Health Sector. *Ethnicity and Health* 11(3): 211–27.

Ratima E, Waetford C, Wikaire E. 2006. Cultural competence of physiotherapists: reducing inequalities in health between Māori and non-Māori. *New Zealand Journal of Physiotherapy* 34(3): 153–9.

Richelsen B, Tonstad S, Rossner S, et al. 2007. Effect of orlistat on weight regain and cardiovascular risk factors following a very-low-energy diet in abdominally obese patients: a three-year randomised, placebo-controlled study. *Diabetes Care* 30(1): 27–32.

Ross R, Berentzen T, Bradshaw AJ, et al. 2008. Does the relationship between waist circumference, morbidity and mortality depend on measurement protocol for waist circumference? *Obesity Reviews* 9(4): 312–25.

Rush E, Plank L, Chandu V, et al. 2004. Body size, body composition, and fat distribution: a comparison on young New Zealand men of European, Pacific Island, and Asian ethnicities. *New Zealand Medical Journal* 117: U1203.

Russell D, Parnell W, Wilson N. 1999. *NZ Food NZ people: Key results of the 1997 National Nutrition Survey*. Wellington: Ministry of Health.

Scott KM, McGee MA, Wells JE, Oakley Browne MA. 2008. Obesity and mental disorders in the adult general population. *Journal of Psychosomatic Research* 64(1): 97–105.

Shaw K, Gennat H, O'Rourke P, et al. 2006. Exercise for overweight or obesity. *Cochrane Database of Systematic Reviews*, Issue 4, Article no. CD003817. DOI: 10.1002/14651858.CD003817.pub3.

Shaw K, O'Rourke P, Del Mar C, et al. 2005. Psychological interventions for overweight or obesity. *Cochrane Database of Systematic Reviews*, Issue 2, article no. CD003818. DOI: 10.1002/14651858.CD003818.pub2.

Sikand G, Kondo A, Foreyt JP, et al. 1988. Two-year follow-up of patients treated with a very-low-calorie diet and exercise training. *Journal of the American Dietetic Association* 88(4): 487–8.

Simmons D, Voyle JA, Fou F, et al. 2004. Tale of two churches: differential impact of a church-based diabetes control programme among Pacific Islands people in New Zealand. *Diabetic Medicine* 21(2): 122–8.

Simonen P, Gylling H, Howard AN, et al. 2000. Introducing a new component of the metabolic syndrome: low cholesterol absorption. *American Journal of Clinical Nutrition* 72(1): 82–8.

Simonsen MK, Hundrup YA, Obel EB, et al. 2008. Intentional weight loss and mortality among initially healthy men and women. *Nutrition Reviews* 66(7): 375–86.

Sjostrom L, Lindroos AK, Peltonen M, et al. 2004. Swedish Obese Subjects Study Scientific Group. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *New England Journal of Medicine* 351(26): 2683–93.

Sjostrom L, Narbro K, Sjostrom CD, et al. 2007. Effects of bariatric surgery on mortality in Swedish obese subjects. *New England Journal of Medicine* 357(8): 741–52.

Skroubis G, Anesidis S, Kehagias I, et al. 2006. Roux-en-Y gastric bypass versus a variant of biliopancreatic diversion in a non-superobese population: Prospective comparison of the efficacy and the incidence of metabolic deficiencies. *Obesity Surgery* 16(4): 488–95.

Stefanick M, Mackey S, Sheehan M, et al. 1998. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *New England Journal of Medicine* 339(1): 12–20.

Stenius-Aarniala B, Poussa T, Kvarnstrom J, et al. 2000. Immediate and long-term effects of weight reduction in obese people with asthma: randomised controlled study. *BMJ* 320(7238): 827–32.

Stephenson M, Hogan S. 2007. The safety, effectiveness and cost effectiveness of surgical and non-surgical interventions for patients with morbid obesity. *NZHTA Technical Brief* 6(8). Christchurch: New Zealand Health Technology Assessment.

Stern L, Iqbal N, Seshadri P, et al. 2004. The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomised trial. *Annals of Internal Medicine* 140(10): 778–85.

Stevens VJ, Obarzanek E, Cook NR, et al. 2001. Trials of Hypertension Prevention, phase II. *Annals of Internal Medicine* 134: 1–11.

Straw MK, Terre L. 1983. An evaluation of individualised behavioural obesity treatment and maintenance strategies. *Behaviour Therapy* 14(255–66).

Stubbs RS, O'Brien I, Jurikova L. 2006. What ring size should be used in association with vertical gastric bypass? *Obesity Surgery* 16(10): 1298–303.

Sundbom M, Gustavasson S. 2004. Randomised trial of hand-assisted laparoscopic versus open Roux-en-Y gastric bypass for treatment of morbid obesity. *British Journal of Surgery* 91(4): 418–23.

Sundborn G, Metcalf P, Scragg R, et al. 2007. Ethnic differences in the prevalence of new and known diabetes mellitus, impaired glucose tolerance, and impaired fasting glucose. Diabetes Heart and Health Survey (DHAH) 2002–2003, Auckland New Zealand. *New Zealand Medical Journal* 120(1257): U2607.

Svetkey LP, Stevens VJ, Brantley PJ, et al. 2008. Comparison of strategies for sustaining weight loss: the weight loss maintenance randomised controlled trial. *JAMA* 299(10): 1139–48.

Swiglo BA, Murad MH, Schunemann HJ, et al. 2008. A case for clarity, consistency, and helpfulness: state-of-the-art clinical practice guidelines in endocrinology using the grading of recommendations, assessment, development, and evaluation system. *Journal of Clinical Endocrinology and Metabolism* 93(3): 666–73.

Swinburn BA, Carey D, Hills AP, et al. 2005. Effect of orlistat on cardiovascular disease risk in obese adults. *Diabetes, Obesity and Metabolism* 7(3): 254–62.

Swinburn BA, Ley SJ, Carmichael HE, et al. 1999. Body size and composition in Polynesians. *International Journal of Obesity* 23: 1178–83.

Tate DF, Jeffery RW, Sherwood NE, et al. 2007. Long-term weight losses associated with prescription of higher physical activity goals. Are higher levels of physical activity protective against weight regain? *American Journal of Clinical Nutrition* 85(4): 954–9.

Thomas DE, Elliott EJ, Baur L. 2007. Low glycaemic index or low glycaemic load diets for overweight and obesity. *Cochrane Database of Systematic Reviews*, Issue 3, article no. CD005105. DOI: 10.1002/14651858.CD005105.pub2.

Tiatia J. 2009. *Literature Review for the Pacific Analysis of the New Zealand Guidelines for Weight Management in Adults and Children and Young People*. Auckland: Hibiscus Research Ltd.

Tsai AG, Wadden TA. 2006. The evolution of very-low-calorie diets: an update and meta-analysis. *Obesity* 14(8): 1283–93.

Ueshima H, Choudhury SR, Okayama A, et al. 2004. Cigarette smoking as a risk factor for stroke death in Japan: NIPPON DATA80. *Stroke* 35(8): 1836–41.

van Dielen FM, Soeters PB, de Brauw LM, et al. 2005. Laparoscopic adjustable gastric banding versus open vertical banded gastroplasty: a prospective randomised trial. *Obesity Surgery* 15(9): 1292–8.

Vazquez G, Duval S, Jacobs Jr DR, et al. 2007. Comparison of body mass index, waist circumference, and waist/hip ratio in predicting incident diabetes: a meta-analysis. *Epidemiologic Reviews* 29(1): 115–28.

Viegener BJ, Perri MG, Nezu AM, et al. 1990. Effects of an intermittent, low-fat, low-calorie diet in the behavioural treatment of obesity. *Behaviour Therapy* 21: 499–509.

Wadden TA, Vogt RA, Foster GD, et al. 1998. Exercise and the maintenance of weight loss: one-year follow-up of a controlled clinical trial. *Journal of Consulting and Clinical Psychology* 66(2): 429–33.

West DS, DiLillo V, Bursac Z, et al. 2007. Motivational interviewing improves weight loss in women with type 2 diabetes. *Diabetes Care* 30(5): 1081–7.

Westling A, Gustavsson S. 2001. Laparoscopic vs open Roux-en-Y gastric bypass: a prospective, randomised trial. *Obesity Surgery* 11(3): 284–92.

White P, Gunston J, Salmond C, et al. 2008. *Atlas of Socioeconomic Deprivation in New Zealand NZDep2006*. Wellington: Ministry of Health.

White S, Brooks E, Jurikova L, et al. 2005. Long-term outcomes after gastric bypass. *Obesity Surgery* 15(2): 155–63.

Whitlock G, Lewington S, Mhurchu CN. 2002. Coronary heart disease and body mass index: a systematic review of the evidence from larger prospective cohort studies. *Seminars in Vascular Medicine* 2(4): 369–81.

Wickremesekera K, Miller G, Naotunne TD, et al. 2005. Loss of insulin resistance after Roux-en-Y gastric bypass surgery: a time course study. *Obesity Surgery* 15(4): 474–81.

Willett WC, Dietz WH, Colditz GA. 1999. Guidelines for healthy weight. *New England Journal of Medicine* 341(6): 427–34.

Williams PT, Hoffman K, La I. 2007. Weight-related increases in hypertension, hypercholesterolemia, and diabetes risk in normal weight male and female runners. *Arteriosclerosis, Thrombosis and Vascular Biology* 27(8): 1811–9.

Williamson DF. 1996. "Weight cycling" and mortality: how do the epidemiologists explain the role of intentional weight loss? *Journal of the American College of Nutrition* 15(1): 6–13.

Wing RR, Epstein LH, Paternostro-Bayles M, et al. 1988. Exercise in a behavioural weight control programme for obese patients with type 2 (non-insulin-dependent) diabetes. *Diabetologia* 31(12): 902–9.

Wing RR, Jeffery RW. 1999. Benefits of recruiting participants with friends and increasing social support for weight loss and maintenance. *Journal of Consulting and Clinical Psychology* 67(1): 132–38.

Wing RR, Marcus MD, Epstein LH, et al. 1991. A "family-based" approach to the treatment of obese type II diabetic patients. *Journal of Consulting and Clinical Psychology* 59(1): 156–62.

Wing RR, Tate DF, Gorin AA, et al. 2007. "STOP regain": Are there negative effects of daily weighing? *Journal of Consulting and Clinical Psychology* 75(4): 652–6

Wing RR, Venditti E, Jakicic JM, et al. 1998. Lifestyle intervention in overweight individuals with a family history of diabetes. *Diabetes Care* 21(3): 350–9.

Wood P, Stefanick M, Dreon D, et al. 1988. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *New England Journal of Medicine* 319(18): 1173–9.

Wood PD, Stefanick ML, Williams PT, et al. 1991. The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. *New England Journal of Medicine* 325(7): 461–6.

Woodward M, Lam TH, Barzi F, et al. 2005. Smoking, quitting, and the risk of cardiovascular disease among women and men in the Asia-Pacific region. *International Journal of Epidemiology* 34(5): 1036–45.

Workshop Organising Team. 2005. *Issues and options paper: The use of the term 'Asian' in New Zealand and implications for research, policy development and community engagement*. Auckland: University of Auckland.

World Health Organization. 2000. Obesity: preventing and managing a health epidemic. Report of a WHO consultation. *World Health Organization Technical Report* 894: 898–904.

World Health Organization Expert Consultation. 2004. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 363: 157–63.

Yan LL, Daviglus ML, Liu K, et al. 2006. Midlife body mass index and hospitalisation and mortality in older age. *JAMA* 295(2): 190–8.

Zhou M, Offer A, Yang G, et al. 2008. Body mass index, blood pressure, and mortality from stroke: a nationally representative prospective study of 212,000 Chinese men. *Stroke* 39: 753–9.