

# CMAJ · JAMC

APRIL 10, 2007, VOL. 176, NO. 8 • LE 10 AVRIL 2007, VOL. 176, N<sup>o</sup> 8

## **2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children**

This complete set of guidelines consists of an executive summary and 26 chapters on specific aspects of obesity prevention and management. It is available online as a PDF at [www.cmaj.ca/cgi/content/full/176/8/S1/DC1](http://www.cmaj.ca/cgi/content/full/176/8/S1/DC1).

CMAJ 2007;176(8 SUPPL):ONLINE-1-117

# Executive summary

**David C.W. Lau, James D. Douketis, Katherine M. Morrison, Irene M. Hramiak, Arya M. Sharma, Ehud Ur, for members of the Obesity Canada Clinical Practice Guidelines Expert Panel**

**O**besity is now reaching epidemic proportions in both developed and developing countries and is affecting not only adults but also children and adolescents. Over the last 20 years, obesity has become the most prevalent nutritional problem in the world, eclipsing undernutrition and infectious disease as the most significant contributor to ill health and mortality. It is a key risk factor for many chronic and noncommunicable diseases.

In Canada, the prevalence of overweight and obesity has increased over recent decades among both children and adults in all areas of the country. According to the most recent estimates from the 2004 Canadian Community Health Survey,<sup>1</sup> 59% of the adult population is overweight (i.e., body mass index [BMI]  $\geq 25$  kg/m<sup>2</sup>) and 1 in 4 (23%) is obese (i.e., BMI  $\geq 30$  kg/m<sup>2</sup>). The sheer numbers of people who are overweight and obese highlight a pressing public health problem that shows no signs of improving in the near future. What is more alarming is the problem of obesity among children and adolescents in Canada, which is advancing at an even more rapid pace than obesity among adults. In 2004, 1 in 4 (26%) Canadian children and adolescents aged 2–17 years was overweight. The obesity rate has increased dramatically in the last 15 years: from 2% to 10% among boys and from 2% to 9% among girls.<sup>1,2</sup> This increase is cause for concern, since there is a tendency for obese children to remain obese as adults. Moreover, obesity-related health problems are now occurring at a much earlier age and continue to progress into adulthood. Given the recent temporal obesity trends among children and youth, the prevalence of obesity among adults will likely continue to increase as the current generation of children enters adulthood.

Obesity should no longer be viewed as a cosmetic or body-image issue. There is compelling evidence that overweight people are at increased risk of a variety of health problems, including type 2 diabetes, hypertension, dyslipidemia, coronary artery disease, stroke, osteoarthritis and certain forms of cancers. It has recently been estimated that about 1 in 10 premature deaths among Canadian adults 20–64 years of age is directly attributable to overweight and obesity. In addition to affecting personal health, the increased health risks translate into an increased burden on the health care system. The cost of obesity in Canada has been conservatively estimated to be \$2 billion a year or 2.4% of total health care expenditures in 1997.<sup>3</sup> Thus, the continuing epidemic of obesity in Canada is exacting a high toll on the health of the population.

The cause of obesity is complex and multifactorial. Within the context of environmental, social and genetic factors, at the simplest level obesity results from long-term positive energy balance — the interaction of energy intake and energy expenditure. The rapid increase in the prevalence of obesity over the past 20 years is a result of environmental and cultural influences rather than genetic factors. With progressive improvements in the standard of living in developed and developing countries, overnutrition and sedentary lifestyle have supplanted physical labour and regular physical activity, which has resulted in positive energy balance and overweight.

Considerable advances have been made in dietary, exercise, behavioural, pharmacologic and bariatric surgical approaches to successful long-term management of obesity. Lifestyle interventions remain the cornerstone of the treatment of obesity, but adherence is poor and long-term success is modest because of significant barriers both on the part of affected individuals and health care professionals responsible for the treatment. Pharmacotherapy and bariatric surgery are useful adjuncts for improving the health outcomes of overweight people, but, for a variety of reasons, these modalities of treatment are not widely adopted.

Despite steady progress in the management of obesity, its prevalence continues to rise. To date, population interventions have tended to focus on individual risk factors and have been largely ineffective. Hence, sweeping prevention and intervention strategies are required to slow, and hopefully reverse, the alarming increase in obesity prevalence in Canada and globally.

A number of clinical practice guidelines on the assessment and management of obesity have been published in the past. These have been largely based on consensus statements by expert panels. Moreover, most of these guidelines focus on individuals rather than on communities and the population as a whole. Recognizing these deficiencies, Obesity Canada — a not-for-profit organization founded in 1999 to improve the health of Canadians by decreasing the occurrence of obesity — convened a panel of experts to determine whether a comprehensive set of guidelines could be developed to address not only the management but also the prevention of obesity in both adults and children. Members of the Steering Committee and Expert Panel unanimously agreed on an evidence-based approach. Through the process of developing these guidelines, which began in the spring of 2004, members of the Steering Committee and Expert Panel identified major gaps in knowledge regarding obesity treat-

ment and prevention. Considerable research is required in many areas to optimize management and to prevent the increasing prevalence of overweight and obesity in Canada. The authors' recommendations range from the need for enhanced surveillance and population-based data to new research on the biological, social, cultural and environmental determinants of obesity as well as research on effective treatment strategies, policies and interventions.

Because obesity is increasingly viewed as a societal issue, members of the Steering Committee and Expert Panel unanimously agreed to include chapters on the prevention of obesity in children and adults at the population level, as well as implications of the guidelines for health policy-makers and other interested parties.

The challenges of disseminating and implementing these guidelines were acknowledged by the Steering Committee, and a dissemination strategy has been developed to ensure that they are translated into clinical practice.

Publication of these guidelines is not the end of this process; it is only the beginning. Ongoing evaluation and revision of the various chapters and recommendations will be undertaken, as appropriate. It is hoped that, with continuing new knowledge from research, regular updating of these guidelines will accord greater certainty to many of the recommendations and expected outcomes.

## Formulation of recommendations: an overview

We have attempted to use a rigorous, evidence-based approach to the development of the practice recommendations, while also acknowledging the breadth of topics to be assessed and the inherent limitations of the obesity literature on these topics.

In addition to making recommendations for treatment interventions, the most common application of clinical practice guidelines, we have also provided recommendations on interventions related to screening and prevention at the individual and population levels.

The recommendations are based on a prespecified process

**Table 1:** Criteria for assigning a level of evidence to recommendations

Level of evidence	Criteria
1	<ul style="list-style-type: none"> <li>Randomized controlled trials (or meta-analyses) <i>without</i> important limitations</li> </ul>
2	<ul style="list-style-type: none"> <li>Randomized controlled trials (or meta-analyses) <i>with</i> important limitations</li> <li>Observational studies (nonrandomized clinical trials or cohort studies) with overwhelming evidence</li> </ul>
3	<ul style="list-style-type: none"> <li>Other observational studies (prospective cohort studies, case-control studies, case series)</li> </ul>
4	<ul style="list-style-type: none"> <li>Inadequate or no data in population of interest</li> <li>Anecdotal evidence or clinical experience</li> </ul>

that was overseen by the Steering Committee. Specific chapters of the guidelines were delegated to a group of content experts within the Expert Panel, who performed a systematic literature review and were responsible for drafting the recommendations for each chapter. Recommendations were appraised by an independent Evidence-based Review Committee, members of which assessed whether the assigned level of evidence reflected the strength of the existing literature. The interactive process by which the recommendations were developed, reviewed and revised included 4 joint meetings of the Steering Committee and Expert Panel. The final draft of the guidelines was reviewed by the Steering Committee and by external stakeholders and experts, who included representatives from academia, industry and government and nongovernment officials.

The approach used to formulate the recommendations was based on the following conventions:

- A clear question or well-defined issue surrounding an obesity-related intervention was the starting point for review of the literature and formulation of recommendations.
- Each recommendation is evidence-based — arrived at through a systematic review of the literature — and reflects the consensus of the Steering Committee and relevant Expert Panel members.
- Each recommendation includes a level of evidence (1 to 4 [Table 1]) and a grade (A, B or C [Table 2]).
- The level of evidence informs the reader about the strength of the evidence in favour of (or against) the intervention and is based on prespecified objective criteria.
- The grade informs the reader about whether an intervention should (or should not) be implemented and reflects both the level of evidence supporting the recommendation and a consideration, where applicable, of the harms and costs of the intervention and its importance and value to the individual or population.
- The level of evidence assigned to an intervention is not necessarily linked to a corresponding grade. However, a high grade is less likely in the setting of low-quality evidence.

**Table 2:** Criteria for assigning a grade to recommendations

Grade	Criteria
A	<p>Strong recommendation (action can apply to most individuals in most circumstances)</p> <ul style="list-style-type: none"> <li>benefits clearly outweigh risks (or vice versa)</li> <li>evidence is level 1, 2 or 3</li> </ul>
B	<p>Intermediate recommendation (action may vary depending on the person's characteristics or other circumstances)</p> <ul style="list-style-type: none"> <li>unclear whether benefits outweigh risks</li> <li>evidence is level 1, 2 or 3</li> </ul>
C	<p>Consensus (weak) recommendation (alternative actions may be equally reasonable)</p> <ul style="list-style-type: none"> <li>unclear whether benefits outweigh risks</li> <li>evidence is level 3 or 4</li> </ul>

- The wording “we recommend” is used to express a grade A recommendation. The wording “we suggest” is used to express a grade B recommendation. The wording used for grade C recommendations varies but reflects the uncertainty surrounding the benefits and risks of the intervention.
- A consensus recommendation, which is classified as grade C, is a statement that provides a reasonable approach or guideline for that domain of study. A consensus recommendation reflects insufficient evidence to inform clinical practice or anecdotal evidence only.

## Research recommendations

Research recommendations were developed to identify gaps in existing knowledge and to establish priorities for future research. Research recommendations were formulated by content experts and were subjected to iterative reviews and modifications by the Steering Committee.

## Objectives and content of the guidelines

These Canadian guidelines on the prevention and management of obesity were developed to address the need for evidence-based recommendations for the management of obesity at the individual and population levels, to identify gaps in knowledge and to establish an agenda for future research in this area.

## Objectives

The specific objectives of the guidelines were:

- to establish a process for developing evidence-based guidelines for the screening, prevention and treatment of obesity in Canada
- to provide recommendations regarding application of the following interventions to individuals and populations:
  - screening for obesity (using BMI and waist circumference)
  - screening for obesity-related conditions (e.g., dyslipidemia, diabetes)
  - screening for psychosocial disorders (e.g., mood disorders, eating disorders)
  - prevention of obesity (individual and community-based interventions)
  - dietary intervention
  - physical exercise therapy
  - cognitive-behaviour therapy
  - pharmacotherapy
  - bariatric surgery
  - alternative or nontraditional therapy
  - health care team support
- to disseminate material to a broad spectrum of health care providers
- to assist in public health policy development
- to identify gaps in knowledge and suggest a research agenda.

## Population and special groups of interest

In developing these guidelines, we aimed to address a broad range of populations and patients, encompassing all age groups and subgroups (the latter classified according to comorbidity or ethnic background):

- children, adolescents and adults who are overweight or obese, or with an increased waist circumference
- population subgroups (e.g., people with type 2 diabetes, hypertension, dyslipidemia, severe obesity [BMI  $\geq 40$  kg/m<sup>2</sup>], and ethnic groups [e.g., Aboriginal people, Asian people]).

## Outcomes of interest

The guidelines identify several key outcomes of interest. These were considered, whenever possible, in the formulation of recommendations:

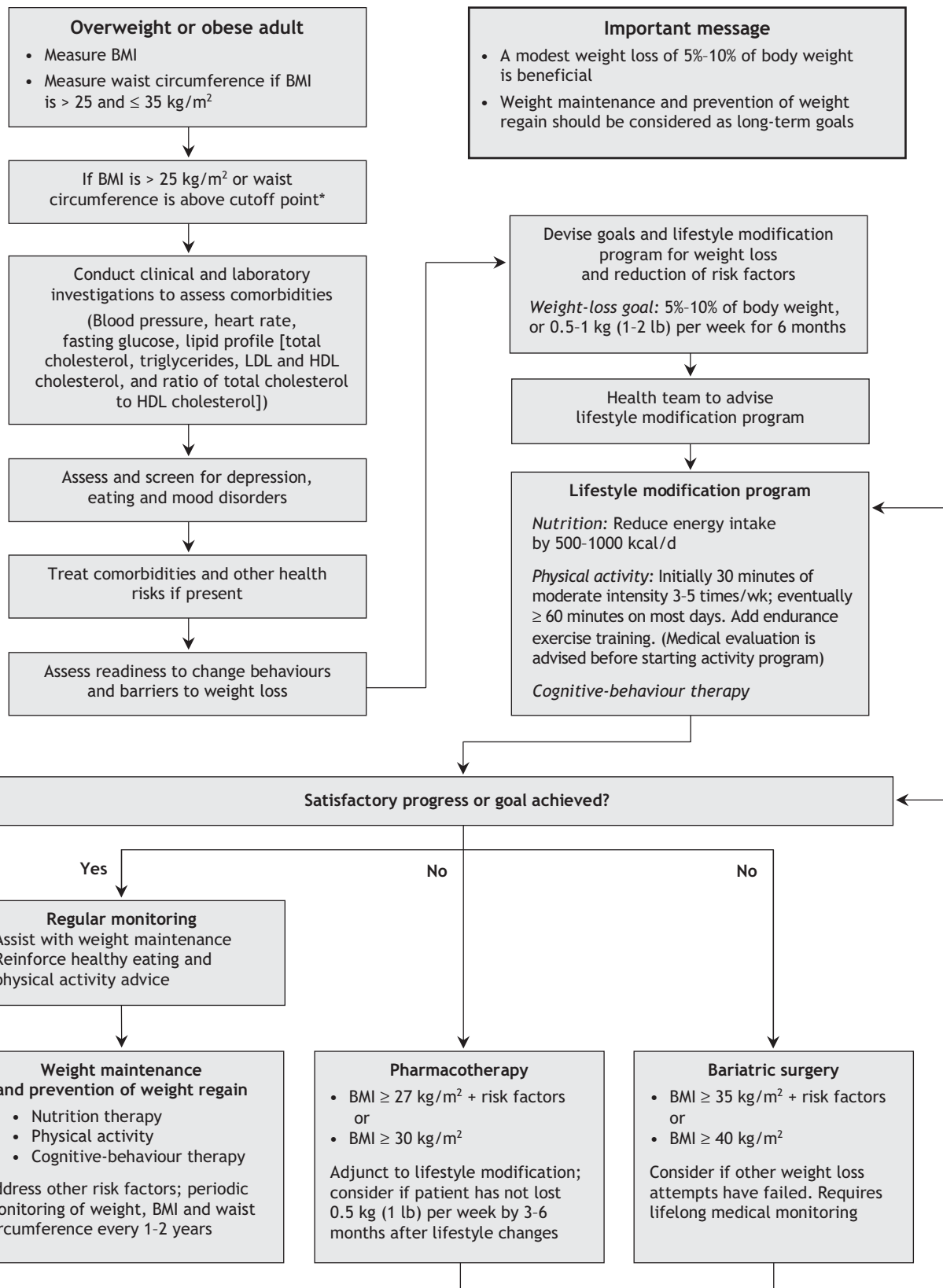
- *Anthropometric outcomes*: body weight, BMI and waist circumference
- *Biochemical or physical outcomes*: fasting glucose, total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, ratio of total cholesterol to HDL cholesterol, and systolic and diastolic blood pressure
- *Clinical outcomes*: cardiovascular disease and other morbidities, mortality and quality-of-life measures
- *Psychosocial outcomes*: depression, mood disorders and eating disorders

## Using the guidelines: an illustrative case scenario

These guidelines are intended as a practical guide that can be used by health care professionals in everyday clinical practice. When using the guidelines, the reader can refer to the list of recommendations (listed at the end of this summary) to find key points rapidly. Alternatively, the reader can refer to individual chapters for a more in-depth review of specific issues. Each of the 26 chapters contains the recommendations listed at the end of this summary that pertain to a specific issue along with an outline of the background and rationale for those recommendations.

With either approach, we suggest that the reader review chapter 1, which provides a rationale for the grades and levels of evidence assigned to the recommendations. Because there is no standardized method for developing clinical practice guidelines, the reader may be aided by a prior understanding of the approach used by the committees. In brief, the *level of evidence* is an objective assessment that reflects the quality of evidence in published studies. The *grade* is a subjective assessment that considers *both* the level of evidence and other factors (e.g., harms and costs) relevant to an intervention when applied at the level of the individual or population.

The following case scenario illustrates how the guidelines may be used in the management of a typical adult patient assessed in clinical practice. Fig. 1 summarizes the assessment



**Fig. 1:** Algorithm for the assessment and stepwise management of the overweight or obese adult. LDL = low-density lipoprotein, HDL = high-density lipoprotein. \*Body mass index (BMI) and waist circumference cutoff points are different for some ethnic groups; see Table 3 for ethnic-specific waist circumference cutoff points.

and stepwise management of the overweight or obese adult, which can be applied to the following case.

Ms. A is a 46-year-old woman who is married with 2 children and is seen for an initial primary care assessment. She has experienced some “weariness and increased irritability” in recent months but otherwise has been in good health. Her menses are regular, and she does not have any climacteric symptoms. She is not taking any prescription medications but occasionally takes an over-the-counter nonsteroidal anti-inflammatory drug (NSAID) for low back pain. She is a former smoker who works in a sedentary occupation and does not exercise regularly (because of lack of time). Her mother (age 72 years) has type 2 diabetes, and her father (age 75 years) has coronary artery disease.

On physical examination, Ms. A appears generally well. She weighs 89 kg and is 1.6 m tall. Blood pressure is 135/85 mm Hg, heart rate is 72 beats/min, chest is clear to auscultation and percussion, heart sounds are normal, no abdominal organomegaly is palpable, and there are no musculoskeletal or skin abnormalities. Her body habitus suggests a central (abdominal) pattern of obesity.

As the primary care physician, you are concerned that Ms. A is obese (with a central pattern of fat distribution) and is at risk of obesity-related diseases, in this case type 2 diabetes and hypertension. On further questioning, the patient states that she has poor eating habits and admits to frequent “snacking” on energy-dense, low-nutrient foods. She also has symptoms that suggest a depressive mood disorder, which is common in obese adults. She does not have any symptoms suggestive of obstructive sleep apnea, which is also associated with obesity.

**Table 3:** Ethnic-specific values for waist circumference

Country or ethnic group	Waist circumference* (as a measure of central obesity), cm	
	Men	Women
Europid*	≥ 94	≥ 80
South Asian, Chinese†	≥ 90	≥ 80
Japanese‡	≥ 85	≥ 90
South and Central American	Use South Asian cutoff points until more specific data are available	
Sub-Saharan African	Use European cutoff points until more specific data are available	
Eastern Mediterranean and Middle East (Arab)	Use European cutoff points until more specific data are available	

\*In future epidemiologic studies of populations of Europid origin, prevalence should be given using both European and North American cutoff points to allow better comparisons. In the United States, the NCEP-ATP III values (men > 102 cm, women > 88 cm) are likely to continue to be used for clinical purposes. However, it is strongly recommended that for epidemiologic studies and, wherever possible, for case detection, ethnic-specific cutoff points should be used for people of the same ethnic group wherever they are found. Thus, the criteria recommended for Japan would also be used in expatriate Japanese communities, as would those for South Asian men and women regardless of place and country of residence.

†Based on a Chinese, Malay and Asian Indian population.

‡Subsequent data analyses suggest that Asian values should be used for Japanese populations until more data are available.

Note: NCEP-ATP III = National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)

## Initial patient assessment and investigations

In chapters 3–8, issues relating to the initial assessment of overweight or obese patients are addressed. Based on a clinical assessment of Ms. A, supplemented by key points from relevant chapters, we recommend the following interventions.

### Measurement of BMI and waist circumference

**Rationale:** Measuring BMI and waist circumference is an essential first step to determine the level and distribution of adiposity and is a *grade A* recommendation when screening for overweight and obesity in individuals. These measures are straightforward and easy to perform. The measurement of BMI (weight divided by height squared) and waist circumference can be used to help determine a patient’s risk profile for cardiovascular disease and overall health risk; they also provide a reference point for monitoring BMI or waist circumference over time, especially if a weight management intervention is planned.

Ms. A’s BMI is 34.8 kg/m<sup>2</sup> (89 kg ÷ [1.6 m]<sup>2</sup>). Based on current guidelines for body weight classification (outlined in chapter 3), Ms. A is approximately at the cutoff point between class I obesity (BMI 30–34.9 kg/m<sup>2</sup>) and class II obesity (BMI 35–39.9 kg/m<sup>2</sup>). Her waist circumference, at 98 cm, is high for her sex (i.e., ≥ 80 cm), indicating a central (abdominal) pattern of obesity (see Table 3). The combination of these 2 measures of health risk puts this patient at “very high risk” of obesity-related diseases.

### Measurement of laboratory parameters

Laboratory parameters include fasting blood glucose level, total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, ratio of total cholesterol to HDL cholesterol, liver enzyme levels and urinalysis.

**Rationale:** Fasting blood glucose level and lipid profile are important components in the assessment of an overweight or obese person, and this is a *grade A* recommendation (chapters 6 and 8) because patients with obesity (especially central obesity) are at high risk of pre-diabetes (impaired fasting glucose or impaired glucose tolerance, or both), type 2 diabetes and dyslipidemia. Early recognition of such disorders may permit dietary and lifestyle changes that will mitigate the risk of disease development and progression. Assessment of liver enzymes and urinalysis is a *grade B* recommendation (chapters 6 and 8) because obese patients may be at increased risk of liver disease (nonalcoholic steatohepatitis) and impaired renal function.

For Ms. A, laboratory testing reveals the following: fasting glucose level 6.4 mmol/L, total cholesterol 5.75 mmol/L, LDL cholesterol 3.59 mmol/L, HDL cholesterol 1.26 mmol/L, triglycerides 1.98 mmol/L, ratio of total:HDL cholesterol 4.56, normal liver enzyme levels and normal urinalysis results.

### Assessment for depression and other mood disorders

**Rationale:** Major depression and other mood disorders are common in patients with obesity; they occur in 20%–60% of women aged 40 years or older with a BMI > 30 kg/m<sup>2</sup>, the

group into which Ms. A falls. Furthermore, the presence of a mood disorder may adversely affect adherence to weight management interventions. Screening for such disorders in appropriate individuals is a *grade B* recommendation (see chapter 7). Treatment of a major depressive disorder should be undertaken in concert with any planned weight management intervention, because some pharmacologic weight-loss treatments must be avoided in patients who are taking antidepressant medications.

Ms. A is considered to have a mild depressive disorder that appears to be linked, in part, to concerns and anxiety about her body image. At this time, a serotonin-specific re-uptake inhibitor (SSRI) and psychotherapy are considered.

### Developing the health care team for a weight management program

Based on the above clinical and laboratory findings, you conclude that Ms. A has impaired fasting glucose (and pre-diabetes), dyslipidemia and a mild depressive disorder. She is at risk of cardiovascular disease and increasing depressive symptoms. In addition, her blood pressure (135/85 mm Hg) requires monitoring because of the risk of hypertension.

Using chapter 9 as a guide, we recommend that Ms. A begin a weight management program. Before its initiation, discuss with her the need for a multidisciplinary health care team to help her carry out the program. The health care team will include a coordinating health professional (who may be a primary care physician, medical specialist or registered nurse), a dietary professional, an exercise professional and a clinical psychologist.

The coordinating health professional will engage the patient's family so that they are made to feel part of the health care team. The health professional will make specific recommendations to help the patient adhere to the weight management program and identify potential barriers to the changes in lifestyle that will become an integral part of the program. The health care professional will also discuss the need for reasonable weight management goals that are sustainable. As discussed in chapter 9, the use of a multidisciplinary, lifestyle-based intervention for the management of obese individuals is a *grade A* recommendation. Furthermore, discussion of the weight management program before its initiation among members of the health care team and the patient to establish management goals and review potential barriers to achieving these goals is a *grade B* recommendation.

### Initiating a weight management program

Based on chapters 10–16 of the guidelines, we recommend that Ms. A undertake a weight management strategy focusing on dietary therapy and lifestyle interventions. To initiate and allow ongoing follow-up of these interventions, refer the patient to appropriate health professionals, who will help to initiate the interventions and monitor the patient's progress over time. Implicit in all weight management programs is long-term monitoring of the patient's status, similar to the long-term monitoring undertaken in patients with other chronic

conditions, such as hypertension or diabetes. Follow-up visits are used to provide ongoing counselling about dietary and lifestyle management, education and, perhaps most important, ongoing support to the patient so that she maintains the dietary and lifestyle changes over the long-term.

### Dietary and lifestyle interventions

*Rationale:* Dietary and lifestyle interventions aimed at decreasing energy intake and increasing energy expenditure through a balanced dietary and exercise program are an essential component of all weight management programs. In chapter 12, we provide a *grade A* recommendation for a healthy diet and regular physical activity as the first-line treatment option for overweight or obese adults to attain clinically important weight loss and reduce obesity-related symptoms. We also give a *grade A* to the recommendation for diet and exercise therapy in overweight or obese adults with risk factors for type 2 diabetes, as is the case with Ms. A, who has pre-diabetes (with impaired fasting glucose) and a family history of type 2 diabetes.

### Assessment by a nutrition health professional

*Rationale:* To outline a dietary treatment plan and to provide adequate education requires counselling by a health professional with expertise in dietary management. Typically, many physicians do not have adequate time to devote to dietary management and may not have the expertise required to recommend a diet therapy plan. Using a qualified and experienced health professional (preferably a registered dietitian) for dietary counselling and to implement an optimal dietary plan for achieving and maintaining a healthy body weight is a *grade B* recommendation (chapter 11).

For Ms. A, the nutrition professional will focus on a dietary regimen tailored to the patient with impaired glucose tolerance or emerging type 2 diabetes.

### Assessment by an exercise health professional

*Rationale:* Physical exercise is an integral component of a weight management program, especially for weight maintenance. As with dietary treatment, many physicians do not have the time or expertise to advise patients on an exercise program that is tailored to individual needs and capabilities. Advice from an exercise health professional is required. For a typical patient, physical activity (30 minutes a day of moderate intensity, increasing, when appropriate, to 60 minutes a day) as part of a weight management program is a *grade B* recommendation (chapter 13).

For Ms. A, an exercise program is problematic because of time constraints related to balancing work-related and family-related duties. An exercise professional will help develop a program that is workable within these confines.

### Assessment by a clinical psychologist (or psychiatrist)

*Rationale:* In chapter 10, we give a *grade A* to the recommendation for 1 or more of behaviour modification, cognitive be-

havioural therapy, activity enhancement and dietary counselling as part of a comprehensive weight management intervention.

For Ms. A, who appears to have had mild depressive symptoms that may be related, in part, to body image perception and suboptimal eating habits, a psychologist (or psychiatrist) with expertise in the management of obese patients will aid in providing appropriate behaviour modification and, if required, psychotherapy and psychotropic medication.

### Long-term monitoring of a patient in a weight management program

Many patients who initiate a weight management program may experience initial health benefits characterized by weight loss and improvements in physical measures (symptom reduction), laboratory measures (glucose and lipid levels) and psychologic measures (mood improvement). However, many patients also experience a relapse or worsening of these measures over the long term.

Although specific patient management issues are beyond the scope of these guidelines, we do provide an assessment of treatment options to be considered after the first-line treatment relating to dietary and lifestyle interventions. These treatment options, which are intended to supplement the first-line treatment, are discussed in chapters 14–16. Based on individual patient characteristics that reflect baseline health risks and benefits accrued from first-line weight management therapy, a patient may be eligible to receive pharmacologic therapy for obesity, bariatric surgery or alternative treatments, which include herbal and other dietary supplements.

## RECOMMENDATIONS

### SECTION ONE: CLINICAL

#### Epidemiology of obesity

1. Because of the health impact of the rising prevalence and incidence of overweight and obesity in Canada, we recommend implementing strategies directed at the prevention and treatment of overweight and obesity in children, adolescents and adults [*grade A, level 3*].
2. Because of the lack of adequate information on the prevalence of obesity and related risk factors in Canada, particularly among subgroups of the population, we recommend the creation of a national surveillance system that incorporates, at a minimum, measurements of height, weight and waist circumference [*grade A, level 3*].

#### Classification of overweight and obesity in adults and children

3. We recommend measuring body mass index (BMI; weight in kilograms divided by height in metres

squared) in all adults [*grade A, level 3<sup>4–6</sup>*] and in all children and adolescents (aged 2 years and older). We recommend using the growth charts of the US Centers for Disease Control and Prevention for BMI to screen children and adolescents for overweight ( $\geq 85$ th to  $< 95$ th percentile) and obesity ( $\geq 95$ th percentile) [*grade A, level 3<sup>7</sup>*].

4. We recommend measuring waist circumference in all adults to assess obesity-related health risks [*grade A, level 3<sup>5,6</sup>*].

### Assessment of overweight and obesity in adults and children

#### Assessment of readiness to change

5. We suggest that health care professionals assess readiness and barriers to change before an individual implements a healthy lifestyle plan for weight control or management [*grade B, level 3<sup>8,9</sup>*].

#### Clinical and laboratory assessment of overweight and obese adults, adolescents and youth

6. We recommend that the clinical evaluation of overweight and obese adults<sup>4,10–12</sup> and children<sup>13,14</sup> include a history and a general physical examination to exclude secondary (endocrine or syndrome-related) causes of obesity and obesity-related health risks and complications [*grade A, level 3*].
7. We recommend measuring fasting plasma glucose level and determining lipid profile, including total cholesterol, triglycerides, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol and ratio of total cholesterol to HDL cholesterol, as screening tests in overweight and obese adults [*grade A, level 3<sup>10,15</sup>*], and we suggest that these screening tests be done in children aged 10 years and older [*grade B, level 3<sup>13,16</sup>*]. We suggest repeating these tests at regular intervals as needed [*grade C, level 4*].
8. We suggest additional investigations, such as liver enzyme tests, urinalysis and sleep studies (when appropriate), to screen for and exclude other common obesity-related health problems [*grade B, level 3<sup>17–19</sup>*].
9. We suggest that the health care professional screen the overweight or obese adult for eating disorders, depression and psychiatric disorders, as appropriate [*grade B, level 3<sup>20–31</sup>*].

### Management of obesity in adults and children

#### Role of health care team professionals in obesity counselling and management

10. We recommend a comprehensive healthy lifestyle intervention for overweight and obese people [*grade A, level 1*]. We suggest that members of the health care team discuss with those willing to participate in weight manage-



ment programs appropriate education, support and therapy as adjuncts to lifestyle interventions [*grade B, level 2*].

11. Primary care health professionals are encouraged to work with other health care team members to develop a comprehensive weight management program for the overweight or obese person to promote and maintain weight loss [*grade C, level 3*].
12. Primary care health professionals are encouraged to create a nonjudgmental atmosphere when discussing weight management [*grade C, level 4*].
13. Health care professionals are encouraged to consider the barriers people might have concerning obesity and its management [*grade C, level 4*].

### Lifestyle intervention

14. We recommend an energy-reduced diet and regular physical activity as the first treatment option for overweight and obese adults<sup>32,33</sup> and children<sup>34-36</sup> to achieve clinically important weight loss and reduce obesity-related symptoms [*grade A, level 2*].
15. In children, we recommend ongoing follow-up by health professionals for a minimum of 3 months [*grade A, level 2*<sup>34-37</sup>].
16. We recommend diet and exercise therapy for overweight and obese people with risk factors for type 2 diabetes [*grade A, level 1*<sup>38-40</sup>] and cardiovascular disease [*grade A, level 2*<sup>41,42</sup>].
17. We suggest that individuals willing to participate in weight management programs be provided with education and support in behaviour modification techniques as an adjunct to other interventions [*grade B, level 2*<sup>43-45</sup>].
18. We recommend comprehensive lifestyle interventions (combining behaviour modification techniques, cognitive behavioural therapy, activity enhancement and dietary counselling) for all obese adults [*grade A, level 1*<sup>39,46-48</sup>].
19. When treating obesity in children, we suggest using family-oriented behaviour therapy [*grade B, level 1*<sup>49-53</sup>].

### Dietary intervention

20. We suggest that the optimal dietary plan for achieving healthy body weight and dietary counselling for adults, adolescents and children be developed with a qualified and experienced health professional (preferably a registered dietitian) together with the individual and family to meet their needs [*grade B, level 2*<sup>54-56</sup>].
21. We recommend that a nutritionally balanced diet (designed to reduce energy intake) be combined with other supportive interventions to achieve a healthy body weight in overweight and obese people of all ages and to ensure the maintenance of growth in adolescents and youth [*grade C, level 4*].
22. We suggest a high-protein or a low-fat diet (within acceptable macronutrient distribution ranges indicated in the Dietary Reference Intakes) as a reasonable short-term (6-12 months) treatment option for obese adults as part of a weight-loss program [*grade B, level 2*<sup>57,58</sup>].

23. Meal replacements may be considered as a component of an energy-reduced diet for selected adults interested in commencing a dietary weight-loss program [*grade C, level 2*<sup>59,60</sup>].

### Physical activity

#### Adults

24. All those considering initiating a vigorous exercise program are encouraged to consult their physician or health care team professionals [*grade C, level 4*].
25. We suggest long-term, regular physical activity, which is associated with maintenance of body weight or a modest reduction in body weight for all overweight and obese people [*grade B, level 2*<sup>61,62</sup>].
26. Physical activity and exercise should be sustainable and tailored to the individual. We recommend that the total duration be increased gradually to maximize the weight-loss benefits [*grade A, level 2*<sup>63-65</sup>].
27. We suggest physical activity (30 minutes a day of moderate intensity, increasing, when appropriate, to 60 minutes a day) as part of an overall weight-loss program [*grade B, level 2*<sup>62,63</sup>].
28. Endurance exercise training may reduce the risk of cardiovascular morbidity in healthy postmenopausal women, and we suggest its use for adults with an increased BMI [*grade B, level 2*].

#### Children and adolescents

29. We recommend that the primary care physician or health care team encourage children and adolescents to reduce sedentary pursuits and “screen time” (i.e., television, video games) [*grade A, level 2*<sup>67-69</sup>].
30. We recommend that activity prescribed for children be fun and recreational, with lifestyle activities tailored to the relative strengths of the individual child and family [*grade A, level 2*<sup>70</sup>]. Health professionals are encouraged to emphasize the short-term benefits of physical activity rather than the long-term health benefits to children [*grade C, level 4*].

### Pharmacotherapy

#### Adults

31. We suggest the addition of a selected pharmacologic agent for appropriate overweight or obese adults, who are not attaining or who are unable to maintain clinically important weight loss with dietary and exercise therapy, to assist in reducing obesity-related symptoms [*grade B, level 2*<sup>71-76</sup>].
32. We suggest the addition of a selected pharmacologic agent for overweight or obese adults with type 2 diabetes, impaired glucose tolerance or risk factors for type 2 diabetes, who are not attaining or who are unable to maintain clinically important weight loss with dietary and exercise therapy, to improve glycemic control and reduce their risk of type 2 diabetes [*grade B, level 2*<sup>71,72,77-82</sup>].

### Children and adolescents

33. We suggest that orlistat be considered to aid in weight reduction and weight maintenance when added to a regimen of lifestyle intervention among adolescents [*grade B, level 1*].
34. Because of lack of data for prepubertal children, the use of pharmacologic agents in this group should be considered only within the context of a supervised clinical trial [*grade C, level 4*].

### Bariatric surgery

35. We suggest that adults with clinically severe obesity (BMI  $\geq 40$  kg/m<sup>2</sup> or  $\geq 35$  kg/m<sup>2</sup> with severe comorbid disease) may be considered for bariatric surgery when lifestyle intervention is inadequate to achieve healthy weight goals [*grade B, level 2*<sup>83</sup>].
36. We suggest that bariatric surgery in adolescents be limited to exceptional cases and performed only by experienced teams [*grade C, level 4*].
37. We suggest that a minimally invasive approach be considered for weight loss surgery when an appropriately trained surgical team and appropriate resources are available in the operating theatre [*grade C, level 3*<sup>84</sup>].

### Alternative therapies

38. There is insufficient evidence to recommend in favour of or against the use of herbal remedies, dietary supplements or homeopathy for weight management in the obese person [*grade C, level 4*<sup>85-98</sup>].

### Prevention of obesity in adults and children

39. The use of surveillance systems and measurement tools is encouraged to determine the effectiveness and efficacy of obesity prevention programs and interventions. The development of a comprehensive, coordinated and rigorous surveillance plan with strong links among program developers, advocates, policy-makers and other stakeholders is encouraged as a key component in obesity prevention [*grade C, level 4*].
40. Obesity prevention should take a multisector approach — similar to that used for tobacco control in Canada. Prevention efforts should invest in and target all age groups and span life from infancy to old age. Innovative ways to provide access and programs to less economically viable citizens should be developed [*grade C, level 4*].
41. Programs that combine a low-fat or energy-reduced diet and endurance exercise have not been shown to be more effective than programs using either component alone for obesity prevention; both approaches should be considered [*grade B, level 3*].
42. We suggest that individual and small-group counselling for dietary interventions be considered for the prevention of obesity in adults [*grade B, level 2*]. Counselling by telephone, counselling by mail and financial incentives do

not appear to be effective, and we do not encourage their use [*grade C, level 3*].

43. There is insufficient evidence to recommend in favour of or against broad community interventions aimed at cardiovascular disease risk reduction for the prevention of obesity [*grade C, level 3*].
44. Discussion of the prevention of childhood obesity with the pregnant mother is encouraged [*grade C, level 4*].
45. Exclusive breast-feeding of infants is encouraged until at least 6 months of age to prevent later obesity [*grade C, level 4*].
46. Discussion of limiting consumption of energy-dense snack foods high in sugar and fat during childhood and adolescence is encouraged [*grade C, level 4*].
47. We suggest limiting “screen time” (i.e., watching television, playing video or computer games) to no more than 2 hours a day to encourage more activity and less food consumption, and to limit exposure to food advertising [*grade B, level 3*].
48. The role of schools as pivotal settings for the promotion of healthy active living and school-based prevention programs to reduce the risk of childhood obesity is encouraged, as are interventions to increase daily physical activity through physical education class time and opportunities for active recreation [*grade C, level 4*].
49. The development of programs in multiple settings targeting behaviour change with parental and family involvement is encouraged [*grade C, level 4*].

## SECTION TWO:

## RESEARCH, POLICY, EDUCATION

### Research and policy

50. Normative data from representative samples of the Canadian population should be collected to allow the development of Canadian-specific growth curves for BMI and waist circumference. Research efforts should be directed at developing reference data that are based on health-related criteria or outcomes rather than being merely representative of the population [*grade C, level 4*].
51. Population-based research is needed to help establish ethnic-specific cutoff values for waist circumference, with optimal sensitivity and specificity for discriminating clinical events [*grade C, level 4*].
52. Future research should be directed at determining the clinical utility of waist circumference in the identification of health risk among children and youth, independently or in combination with BMI [*grade C, level 4*].
53. For population surveillance of overweight and obesity in children, we recommend that the BMI thresholds of the International Obesity Task Force be used to classify children and youth as overweight and obese. Where possible, we recommend that prevalence be presented using both the US Centers for Disease Control and Prevention thresholds and the International Obesity Task Force cutoff points to facilitate international comparisons [*grade C, level 4*].

54. Future research should be directed at understanding the impact of gender, biological maturation, nutrition, physical activity levels, sociocultural milieu, built environments, ethnic background, biological factors, psychological factors and genetics on obesity and obesity-related health risk in the context of the Canadian population [grade C, level 4].
55. The screening criteria for obesity-related health consequences should be assessed in the clinical setting for sensitivity, specificity and clinical value in improving the health of children with obesity [grade C, level 4].
56. Investigation of the prevalence of the health consequences of obesity in childhood should be undertaken in diverse populations and should include longitudinal studies to examine the prognosis of hyperinsulinemia, impaired glucose tolerance, impaired fasting glucose level and the clustering of cardiovascular risk factors [grade C, level 4].
57. Studies to determine optimal intervention strategies for children with established health consequences related to obesity are urgently required [grade C, level 4].
58. Randomized clinical trials are needed to examine the effect of the treatment of depression on outcomes of treatment for obesity. Further studies with improved diagnostic methods and prospective designs are also needed to delineate the association between obesity and major depressive disorder [grade C, level 4].
59. Research should be undertaken to develop and evaluate the organization of care for overweight and obese people and to determine the cost-effectiveness of these strategies [grade C, level 4].
60. Long-term, randomized controlled trials of nutritional therapy for obesity treatment in the pediatric population are urgently required. Such studies should consider different ethnic groups and ages, should be with and without energy restriction and should address dietary efficacy, acceptability, long-term compliance, impact of the diet on overweight status, and physical and psychological health risks [grade C, level 4].
61. Research should be undertaken to further validate models and tools for assessing intention to change as well as the effectiveness of interventions to improve readiness to change [grade C, level 4].
62. Research to find drugs that do not cause weight gain in treatment areas such as psychiatry is needed [grade C, level 4].
63. Funding of all types of research at all levels to address knowledge gaps and answer outstanding questions in the area of obesity is a high priority. Research is needed to develop, test and refine effective policies and interventions (best practices) in obesity prevention to enhance the evidence base for future public health interventions. Specific emphasis must be placed on translating research into policy, programs and practice [grade C, level 4].

## Education

64. Undergraduate curricula and education for graduate

health practitioners should be improved to fulfill knowledge, skills and attitude goals with respect to management and prevention of obesity [grade C, level 4].

65. Continuing education activities that provide physicians and health professionals with the skills they need to counsel people confidently in healthy weight management should be developed [grade C, level 4].

## Implementation of the guidelines

66. Dissemination of the guidelines can be orchestrated by a central organization, but implementation should be carried out locally by individuals or local organizations [grade C, level 4].
67. The transfer of information into clinical practice should focus on establishing weight reduction and weight control as an important secondary prevention strategy for diabetes and cardiovascular disease [grade C, level 4].
68. More research is needed to improve understanding of the mechanisms of clinical practice guidelines implementation [grade C, level 4<sup>99</sup>].
69. The guidelines should be disseminated in a simple, clear format that will be well received and accepted [grade C, level 4].
70. A network of local key opinion leaders should be developed as an important component of a successful dissemination and implementation strategy [grade C, level 4<sup>100</sup>].
71. A multifaceted global dissemination and implementation plan should involve a sequence of events, including publication in peer-reviewed and non-peer-reviewed journals [grade C, level 4<sup>101</sup>].
72. To ensure continual quality improvement, a committee should be created to measure outcomes, then monitor the effectiveness of the implementation program [grade C, level 4].

The recommendations were developed, reviewed and revised by the Expert Panel and the Steering Committee. The final draft of the guidelines was reviewed by the Steering Committee and by external stakeholders and experts, who included representatives from academia, industry and government and nongovernment officials.

See Appendix 1 for a complete list of committees and contributors to this work.

**Contributors:** David Lau and James Douketis were the principal coauthors of the executive summary. All of the authors contributed substantially to the conception and design and to the interpretation of the guideline findings, revised the manuscript for important intellectual content and approved the final version to be published.

**Competing interests:** None declared for James Douketis and Katherine Morrison. David Lau owns common shares in GlaxoSmithKline and Eli Lilly. He is a consultant to Abbott Laboratories, Ltd., AstraZeneca Canada Inc., Merck Frosst Canada Inc., Bristol-Myers Squibb Canada, Eli Lilly Canada Inc., Oryx Pharmaceuticals Inc., Pfizer Canada Inc., sanofi-aventis Canada Inc., Servier Canada Inc. and Solvay Pharma Inc.; and has received speaker fees from Abbott Laboratories, Ltd., AstraZeneca Canada Inc., GlaxoSmithKline, Merck Frosst Canada Inc., Merck/Schering, Eli Lilly Canada Inc., sanofi-aventis Canada Inc. and Novo Nordisk Canada Inc.; research grants from AstraZeneca Canada Inc., Bristol Myers Squibb, Dainippon Pharmaceuticals, GlaxoSmithKline, Pfizer Canada Inc., and sanofi-aventis Canada Inc.; and travel assistance to attend international meetings from Abbott Laboratories, Ltd., AstraZeneca Canada Inc. and sanofi-aventis Canada Inc. Irene Hramiak is a consultant to GlaxoSmithKline Inc. and is on a National Advisory Board for Abbott Laboratories Ltd., Eli Lilly, Novo Nordisk, sanofi-aventis Canada Inc. and GlaxoSmithKline Inc. She has received honoraria for speaking engagements from Merck Frosst Canada

Ltd., GlaxoSmithKline Inc. and Novo Nordisk and has received a travel grant from Novo Nordisk. Arya Sharma is a consultant to Abbott Laboratories Ltd., Boehringer Ingelheim, Novartis, sanofi-aventis Canada Inc. and Merck Frosst Canada Ltd. He has received speaker fees from Abbott Laboratories Ltd., Boehringer Ingelheim, AstraZeneca Canada Inc., Novartis and Merck Frosst Canada Ltd. and travel assistance from Abbott Laboratories Ltd., Boehringer Ingelheim, Merck Frosst Canada Ltd., Novartis and sanofi-aventis Canada Inc. Ehud Ur has received speaker fees from sanofi-aventis Canada Inc., Abbott Laboratories, Ltd., GlaxoSmithKline and Novo Nordisk; research grants from GlaxoSmithKline and Novo Nordisk; and travel assistance from sanofi-aventis Canada Inc., Abbott Laboratories, Ltd.

**Acknowledgements:** Financial assistance for the development of these guidelines was generously provided in the form of arm's-length grants-in-aid from Abbott Laboratories Ltd., AstraZeneca Canada Inc., GlaxoSmithKline Inc., Merck Frosst Canada Ltd., Pfizer Canada Inc., Hoffmann-La Roche Ltd., Johnson and Johnson Medical Products, sanofi-aventis Canada Inc. and Unilever Canada Inc. Sponsors were not involved in any aspect of the guidelines development, the literature interpretation, the decision to publish or any other aspect of publication of the guidelines. The funds were used for conference calls and for organization of and travel to meetings for the members of the Steering Committee and Expert Panel Committee. None of the members of these committees received any financial or in-kind remuneration for their contribution to this work.

We extend special thanks to Core Health Services Inc., Toronto, Ont., for providing logistic assistance in organizing meetings and conference calls and for distribution of the guidelines; Barbara Kermod-Scott, medical writer, for writing and editing services; and Valerie Crosbie for her editorial assistance in the preparation of these guidelines.

## REFERENCES

- Shields M. Measured obesity: overweight Canadian children and adolescents. In: *Nutrition: findings from the Canadian Community Health Survey*; issue 1; 2005 (cat no 82-620-MWE2005001). Available: [www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf](http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf) (accessed 2007 Jan 9).
- Willms JD, Tremblay MS, Katzmarzyk PT. Geographic and demographic variation in the prevalence of overweight Canadian children. *Obes Res* 2003;11:668-73.
- Birmingham CL, Muller JL, Palepu A, et al. The cost of obesity in Canada. *CMAJ* 1999;160:483-8.
- The practical guide: identification, evaluation, and treatment of overweight and obesity in adults*. Bethesda Md.: National Institutes of Health; 2000. Publ. no. 00-4084. Available: [www.nhlbi.nih.gov/guidelines/obesity/prctgd\\_b.pdf](http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_b.pdf) (accessed 2006 Dec 27).
- Dickey RA, Bartuska DG, Bray GW, et al. ACCE/ACE position statement on the prevention, diagnosis, and treatment of obesity. *Endocr Pract* 1998;4(5):297-330. Available: [www.aace.com/pub/pdf/guidelines/obesityguide.pdf](http://www.aace.com/pub/pdf/guidelines/obesityguide.pdf) (accessed 2007 Jan 20).
- Lyznicki JM, Young DC, Riggs JA, et al. Obesity: assessment and management in primary care. *Am Fam Physician* 2001;63(11):2185-96.
- Kuczmariski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat* 11 2002;(246):1-190.
- McTigue KM, Harris R, Hemphill B, et al. Screening and interventions for obesity in adults: summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med* 2003;139:933-49.
- Leiter LA, Abbott D, Campbell NR, et al. Lifestyle modifications to prevent and control hypertension. 2. Recommendations on obesity and weight loss. Canadian Hypertension Society, Canadian Coalition for High Blood Pressure Prevention and Control, Laboratory Centre for Disease Control at Health Canada, Heart and Stroke Foundation of Canada. *CMAJ* 1999;160(9 Suppl):S7-12.
- Genest J, Frohlich J, Fodor G, et al. Recommendations for the management of dyslipidemia and the prevention of cardiovascular disease: summary of the 2003 update. *CMAJ* 2003;169:921-4.
- Allison DB, Mentore JL, Heo M, et al. Antipsychotic-induced weight gain: a comprehensive research synthesis. *Am J Psychiatry* 1999;156:1686-96.
- Wetterling T. Body weight gain with atypical antipsychotics. A comparative review. *Drug Saf* 2001;24:59-73.
- Daniels SR, Arnett DK, Eckel RH, et al. Overweight in children and adolescents: pathophysiology, consequences, prevention, and treatment. *Circulation* 2005;111:1999-2012.
- Australian National Health and Medical Research Council. *Clinical practice guidelines for the management of overweight and obesity in children and adolescents*. Canberra, Commonwealth of Australia: Australian National Health and Medical Research Council; 2003. Available: [www.health.gov.au/internet/wcms/Publishing.nsf/Content/obesityguidelines-guidelines-children.htm/\\$FILE/children.pdf](http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/obesityguidelines-guidelines-children.htm/$FILE/children.pdf) (accessed 2006 Dec 27).
- Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. Canadian Diabetes Association 2003 clinical practice guidelines for the prevention and management of diabetes in Canada. *Can J Diabetes* 2003;27(Suppl 2):S1-152.
- Kavey RE, Daniels SR, Lauer RM, et al. American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood. *Circulation* 2003;107:1562-6.
- Chagnac A, Weinstein T, Herman M, et al. The effects of weight loss on renal function in patients with severe obesity. *J Am Soc Nephrol* 2003;14:1480-6.
- Kambham N, Markowitz GS, Valeri AM, et al. Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int* 2001;59:1498-509.
- Van den Brandt PA, Spiegelman D, Yaun SS, et al. Pooled analysis of prospective cohort studies on height, weight, and breast cancer risk. *Am J Epidemiol* 2000;152:514-27.
- Roberts RE, Kaplan GA, Shema SJ, et al. Are the obese at greater risk for depression? *Am J Epidemiol* 2000;152:163-70.
- Roberts RE, Deleger S, Strawbridge WJ, et al. Prospective association between obesity and depression: evidence from the Alameda County Study. *Int J Obes Relat Metab Disord* 2003;27:514-21.
- Pine DS, Goldstein RB, Wolk S, et al. The association between childhood depression and adulthood body mass index. *Pediatrics* 2001;107:1049-56.
- Goodman E, Whitaker RC. A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics* 2002;110:497-504.
- DiPietro L, Anda RF, Williamson DF, et al. Depressive symptoms and weight change in a national cohort of adults. *Int J Obes Relat Metab Disord* 1992;16:745-53.
- Richardson LP, Davis R, Poulton R, et al. A longitudinal evaluation of adolescent depression and adult obesity. *Arch Pediatr Adolesc Med* 2003;157:739-45.
- Ziegelstein RC, Fauerbach JA, Stevens SS, et al. Patients with depression are less likely to follow recommendations to reduce cardiac risk during recovery from a myocardial infarction. *Arch Intern Med* 2000;160:1818-23.
- DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for non-compliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. *Arch Intern Med* 2000;160:2101-7.
- Linde JA, Jeffery RW, Levy RL, et al. Binge eating disorder, weight control self-efficacy, and depression in overweight men and women. *Int J Obes Relat Metab Disord* 2004;28:418-25.
- Sherwood NE, Jeffery RW, Wing RR. Binge status as a predictor of weight loss treatment outcome. *Int J Obes Relat Metab Disord* 1999;23:485-93.
- Unutzer J, Katon W, Williams JW Jr, et al. Improving primary care for depression in late life: the design of a multicenter randomized trial. *Med Care* 2001;39:785-99.
- Williams JW Jr, Katon W, Lin EH, et al. The effectiveness of depression care management on diabetes-related outcomes in older patients. *Ann Intern Med* 2004;140:1015-24.
- Sikand G, Kondo A, Foreyt JP, et al. Two-year follow-up of patients treated with a very-low-calorie diet and exercise training. *J Am Diet Assoc* 1988;88:487-8.
- Pavlou KN, Krey S, Steffee WP. Exercise as an adjunct to weight loss and maintenance in moderately obese subjects. *Am J Clin Nutr* 1989;49(5 Suppl):1115-23.
- Eliakim A, Kaven G, Berger I, et al. The effect of a combined intervention on body mass index and fitness in obese children and adolescents — a clinical experience. *Eur J Pediatr* 2002;161:449-54.
- Saelens BE, Sallis JF, Wilfley DE, et al. Behavioral weight control for overweight adolescents initiated in primary care. *Obes Res* 2002;10:22-32.
- Nemet D, Barkan S, Epstein Y, et al. Short- and long-term beneficial effects of a combined dietary-behavioral-physical activity intervention for the treatment of childhood obesity. *Pediatrics* 2005;115:e443-9.
- Epstein LH, McCurley J, Wing RR, et al. Five-year follow-up of family-based behavioral treatments for childhood obesity. *J Consult Clin Psychol* 1990;58:661-4.
- Torjesen PA, Birkeland KI, Anderssen SA, et al. Lifestyle changes may reverse development of the insulin resistance syndrome. The Oslo Diet and Exercise Study: a randomized trial. *Diabetes Care* 1997;20:26-31.
- Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393-403.
- Lindstrom J, Eriksson JG, Valle TT, et al. Prevention of diabetes mellitus in subjects with impaired glucose tolerance in the Finnish diabetes prevention study: results from a randomized clinical trial. *J Am Soc Nephrol* 2003;14(Suppl 2):S108-13.
- Wood PD, Stefanick ML, Williams PT, et al. The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. *N Engl J Med* 1991;325:461-6.
- Stefanick ML, Mackey S, Sheehan M, et al. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *N Engl J Med* 1998;339:12-20.
- Wing RR, Jeffery RW. Outpatient treatments of obesity: a comparison of methodology and clinical results. *Int J Obes Relat Metab Disord* 1979;3:261-79.
- Bennett GA. Behaviour therapy for obesity: a quantitative review of the effects of selected treatment characteristics on outcome. *Behav Ther* 1986;17:554-62.
- Summerbell CD, Ashton V, Campbell KJ, et al. Interventions for treating obesity in children. *Cochrane Database Syst Rev* 2003;(3):CD001872.
- Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343-50.

47. Stevens VJ, Obarzanek E, Cook NR, et al. Long-term weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, phase II. *Ann Intern Med* 2001;134:1-11.
48. Kuller LH, Simkin-Silverman LR, Wing RR, et al. Women's Healthy Lifestyle Project: a randomized clinical trial: results at 54 months. *Circulation* 2001;103:32-7.
49. Wonderlich SA, de Zwaan M, Mitchell JE, et al. Psychological and dietary treatments of binge eating disorder: conceptual implications. *Int J Eat Disord* 2003;34:S58-73.
50. Pendleton VR, Goodrick GK, Poston WS, et al. Exercise augments the effects of cognitive-behavioral therapy in the treatment of binge eating. *Int J Eat Disord* 2002;31:172-84.
51. Gorin AA, Le Grange D, Stone AA. Effectiveness of spouse involvement in cognitive behavioral therapy for binge eating disorder. *Int J Eat Disord* 2003;33:421-33.
52. Agras WS, Telch CF, Arnow B, et al. One-year follow-up of cognitive-behavioral therapy for obese individuals with binge eating disorder. *J Consult Clin Psychol* 1997;65:343-7.
53. Eldredge KL, Stewart Agras W, Arnow B, et al. The effects of extending cognitive-behavioral therapy for binge eating disorder among initial treatment nonresponders. *Int J Eat Disord* 1997;21:347-52.
54. Brehm BJ, Seeley RJ, Daniels SR, et al. A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *J Clin Endocrinol Metab* 2003;88:1617-23.
55. Astrup A, Grunwald GK, Melanson EL, et al. The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obes Relat Metab Disord* 2000;24:1545-52.
56. Due A, Toubro S, Skov AR, et al. Effect of normal-fat diets, either medium or high in protein, on body weight in overweight subjects: a randomised 1-year trial. *Int J Obes Relat Metab Disord* 2004;28:1283-90.
57. Ashley JM, St Jeor ST, Perumean-Chaney S, et al. Meal replacements in weight intervention. *Obes Res* 2001;9:312S-20S.
58. Heaney RP, Davies KM, Barger-Lux MJ. Calcium and weight: clinical studies. *J Am Coll Nutr* 2002;21:152S-5S.
59. Bravata DM, Sanders L, Huang J, et al. Efficacy and safety of low-carbohydrate diets: a systematic review. *JAMA* 2003;289:1837-50.
60. Raben A. Should obese patients be counselled to follow a low-glycaemic index diet? No. *Obes Rev* 2002;3:245-56.
61. Jakicic JM, Marcus BH, Gallagher KI, et al. Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial. *JAMA* 2003;290:1323-30.
62. Pritchard JE, Nowson CA, Wark JD. A worksite program for overweight middle-aged men achieves lesser weight loss with exercise than with dietary change. *J Am Diet Assoc* 1997;97:37-42.
63. Garrow JS, Summerbell CD. Meta-analysis: effect of exercise, with or without dieting, on the body composition of overweight subjects. *Eur J Clin Nutr* 1995;49:1-10.
64. Miller WC, Koceja DM, Hamilton EJ. A meta-analysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. *Int J Obes Relat Metab Disord* 1997;21:941-7.
65. Fogelholm M, Kukkonen-Harjula K. Does physical activity prevent weight gain — a systematic review. *Obes Rev* 2000;1:95-111.
66. Slentz CA, Duscha BD, Johnson JL, et al. Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRRIDE — a randomized controlled study. *Arch Intern Med* 2004;164:31-9.
67. Reilly JJ, McDowell ZC. Physical activity interventions in the prevention and treatment of paediatric obesity: systematic review and critical appraisal. *Proc Nutr Soc* 2003;62:611-9.
68. Epstein LH, Valoski AM, Vara LS, et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. *Health Psychol* 1995;14:109-15.
69. Epstein LH, Paluch RA, Gordy CC, et al. Decreasing sedentary behaviors in treating pediatric obesity. *Arch Pediatr Adolesc Med* 2000;154:220-6.
70. Epstein LH, Wing RR, Koeske R, et al. A comparison of lifestyle exercise, aerobic exercise, and calisthenics on weight loss in obese children. *Behav Ther* 1985;16:345-56.
71. Torgerson JS, Hauptman J, Boldrin MN, et al. XENical in the prevention of Diabetes in Obese Subjects (XENDOS) study: a randomized study of orlistat as an adjunct to lifestyle changes for the prevention of type 2 diabetes in obese patients. *Diabetes Care* 2004;27:155-61.
72. Hanefeld M, Sachse G. The effects of orlistat on body weight and glycaemic control in overweight patients with type 2 diabetes: a randomized, placebo-controlled trial. *Diabetes Obes Metab* 2002;4:415-23.
73. Finer N, James WP, Kopelman PG, et al. One-year treatment of obesity: a randomized, double-blind, placebo-controlled, multicentre study of orlistat, a gastrointestinal lipase inhibitor. *Int J Obes Relat Metab Disord* 2000;24:306-13.
74. Davidson MH, Hauptman J, DiGirolamo M, et al. Weight control and risk factor reduction in obese subjects treated for 2 years with orlistat: a randomized controlled trial. *JAMA* 1999;281:235-42.
75. Sjostrom L, Rissanen A, Andersen T, et al. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group. *Lancet* 1998;352:167-72.
76. James WP, Astrup A, Finer N, et al. Effect of sibutramine on weight maintenance after weight loss: a randomised trial. STORM Study Group. Sibutramine Trial of Obesity Reduction and Maintenance. *Lancet* 2000;356:2119-25.
77. Bakris G, Calhoun D, Egan B, et al. Orlistat improves blood pressure control in obese subjects with treated but inadequately controlled hypertension. *J Hypertens* 2002;20:2257-67.
78. Kelley DE, Bray GA, Pi-Sunyer FX, et al. Clinical efficacy of orlistat therapy in overweight and obese patients with insulin-treated type 2 diabetes: a 1-year randomized controlled trial. *Diabetes Care* 2002;25:1033-41.
79. Miles JM, Leiter L, Hollander P, et al. Effect of orlistat in overweight and obese patients with type 2 diabetes treated with metformin. *Diabetes Care* 2002;25:1123-8.
80. Hollander PA, Elbein SC, Hirsch IB, et al. Role of orlistat in the treatment of obese patients with type 2 diabetes. A 1-year randomized double-blind study. *Diabetes Care* 1998;21:1288-94.
81. McNulty SJ, Ur E, Williams G. A randomized trial of sibutramine in the management of obese type 2 diabetic patients treated with metformin. *Diabetes Care* 2003;26:125-31.
82. Sanchez-Reyes L, Fanghanel G, Yamamoto J, et al. Use of sibutramine in overweight adult hispanic patients with type 2 diabetes mellitus: a 12-month, randomized, double-blind, placebo-controlled clinical trial. *Clin Ther* 2004;26:1427-35.
83. Sjostrom L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med* 2004;351:2683-93.
84. Nguyen NT, Goldman C, Rosenquist CJ, et al. Laparoscopic versus open gastric bypass: a randomized study of outcomes, quality of life, and costs. *Ann Surg* 2001;234:279-89.
85. Paranjpe P, Patki P, Patwardhan B. Ayurvedic treatment of obesity: a randomised double-blind, placebo-controlled clinical trial. *J Ethnopharmacol* 1990;29:1-11.
86. Karlsson C, Stenlof K, Johannsson G, et al. Effects of growth hormone treatment on the leptin system and on energy expenditure in abdominally obese men. *Eur J Endocrinol* 1998;138:408-14.
87. Lacey JM, Tershakovec AM, Foster GD. Acupuncture for the treatment of obesity: a review of the evidence. *Int J Obes Relat Metab Disord* 2003;27:419-27.
88. Mhurchu CN, Poppitt SD, McGill AT, et al. The effect of the dietary supplement, Chitosan, on body weight: a randomised controlled trial in 250 overweight and obese adults. *Int J Obes Relat Metab Disord* 2004;28:1149-56.
89. Bahadori B, Wallner S, Schneider H, et al. Effect of chromium yeast and chromium picolinate on body composition of obese, non-diabetic patients during and after a formula diet. *Acta Med Austriaca* 1997;24:185-7.
90. Boozer CN, Daly PA, Homel P, et al. Herbal ephedra/caffeine for weight loss: a 6-month randomized safety and efficacy trial. *Int J Obes Relat Metab Disord* 2002;26:593-604.
91. Pasmán WJ, Westerterp-Plantenga MS, Saris WH. The effectiveness of long-term supplementation of carbohydrate, chromium, fibre and caffeine on weight maintenance. *Int J Obes Relat Metab Disord* 1997;21:1143-51.
92. Johannsson G, Marin P, Lonn L, et al. Growth hormone treatment of abdominally obese men reduces abdominal fat mass, improves glucose and lipoprotein metabolism, and reduces diastolic blood pressure. *J Clin Endocrinol Metab* 1997;82:727-34.
93. Albert SG, Mooradian AD. Low-dose recombinant human growth hormone as adjuvant therapy to lifestyle modifications in the management of obesity. *J Clin Endocrinol Metab* 2004;89:695-701.
94. Herrmann BL, Berg C, Vogel E, et al. Effects of a combination of recombinant human growth hormone with metformin on glucose metabolism and body composition in patients with metabolic syndrome. *Horm Metab Res* 2004;36:54-61.
95. Pittler MH, Abbot NC, Harkness EF, et al. Randomized, double-blind trial of chitosan for body weight reduction. *Eur J Clin Nutr* 1999;53:379-81.
96. Bray GA, Greenway FL. Current and potential drugs for treatment of obesity. *Endocr Rev* 1999;20:805-75.
97. Ernst E, Pittler MH. Chitosan as a treatment for body weight reduction: a meta-analysis. *Perfusion* 1998;11:461-5.
98. Jordan J, Sharma AM. Potential for sibutramine-yohimbine interaction? *Lancet* 2003;361:1826.
99. Gross PA, Greenfield S, Cretin S, et al. Optimal methods for guideline implementation: conclusions from Leeds Castle meeting. *Med Care* 2001;39(Suppl 2):1185-92.
100. Chalmers J. Implementation of guidelines for management of hypertension. *Clin Exp Hypertens* 1999;21:647-57.
101. Connor H, Annan F, Bunn E, et al. The implementation of nutritional advice for people with diabetes. *Diabet Med* 2003;20:786-807.

**Correspondence to:** Dr. David C.W. Lau, Departments of Medicine, Biochemistry and Molecular Biology, Julia McFarlane Diabetes Research Centre, Diabetes and Endocrine Research Group, University of Calgary, 2521-3330 Hospital Dr. NW, Calgary AB T2N 4N1; dcwlu@ucalgary.ca

---

**Appendix 1: Committee members and coauthors who contributed to the 2006 Canadian clinical practice guidelines on the management and prevention of obesity in adults and children**

---

**Program chair**

David C.W. Lau, President, Obesity Canada, Professor of Medicine, Biochemistry and Molecular Biology, Julia McFarlane Diabetes Research Centre, and Chair, Diabetes and Endocrine Research Group, University of Calgary, Calgary, Alta.

**Steering Committee**

Irene M. Hramiak, Professor of Medicine, University of Western Ontario, Medical Director of the Lawson Diabetes Centre, St. Joseph's Health Care, London, Ont.; James D. Douketis, Associate Professor of Medicine, McMaster University, St. Joseph's Healthcare, Hamilton, Ont.; Katherine M. Morrison, Assistant Professor of Pediatrics, McMaster University, and active staff, McMaster Children's Hospital, Hamilton, Ont.; Arya M. Sharma, Professor of Medicine, Canada Research Chair for Cardiovascular Obesity Research and Management, McMaster University, Hamilton, Ont.; Ehud Ur, Professor of Medicine, Dalhousie University, and Head, Division of Endocrinology and Metabolism, Capital Health, Halifax, NS

**Expert Panel**

Oded Bar-Or (deceased), Professor Emeritus of Pediatrics, McMaster University, and Director of the Children's Exercise and Nutrition Centre, Chedoke Hospital, Hamilton Health Sciences, Hamilton, Ont.; Daniel Birch, Associate Professor, Department of Surgery, University of Alberta, Edmonton, Alta.; Simon Biron, Professor of Surgery and Director, General Surgery Division, Laval University, and Head, Department of Surgery, Laval Hospital, Laval, Que.; Robert R.M. Dent, Director, Weight Management Clinic, Ottawa Hospital, Ottawa, Ont.; Jean-Pierre Després, Chair and Professor of Human Nutrition, Department of Sciences of Food and Nutrition, Laval University, and Director of Research, Cardiology, Québec Heart Institute, Laval Hospital Research Centre, Sainte-Foy, Que.; Denis Drouin, Clinical Professor of Family Medicine, Faculty of Medicine, Laval University, Québec, Que.; Diane T. Finegood, Scientific Director, Canadian Institutes of Health Research – Institute of Nutrition, Metabolism and Diabetes, and Professor of Kinesiology, School of Kinesiology, Simon Fraser University, Burnaby, BC; Dominique R. Garrel, Professor of Medicine and Director, Department of Nutrition, Université de Montréal, Hôtel-Dieu – Centre hospitalier de l'Université de Montréal, Montréal, Que.; Linda Gillis, Children's Exercise and Nutrition Centre, McMaster Children's Hospital, Hamilton, Ont.; Michael Grace, Professor Emeritus of Surgery, University of Western Ontario, London, Ont.; Rhona Hanning, Associate Professor, Department of Health Studies and Gerontology, Faculty of Applied Health Sciences, University of Waterloo, Waterloo, Ont.; Peter T. Katzmarzyk, Associate Professor, School of Kinesiology and Health Studies and Department of Community Health and Epidemiology, Queen's University, Kingston, Ont.; Claire LeBlanc, Associate Professor of Pediatrics, University of Ottawa, Children's Hospital of Eastern Ontario, Ottawa, Ont.; Lawrence A. Leiter, Professor of Medicine and Nutritional Sciences, University of Toronto, and Head, Division of Endocrinology and Metabolism, St. Michael's Hospital, Toronto, Ont.; Rena Mendelson, Professor of Nutrition, Department of Nutritional Sciences, Ryerson University, Toronto, Ont.; Linda McCargar, Professor, Department of Agriculture, Food and Nutritional Science, Faculty of Agriculture, Forestry and Home Economics, and Director, Human Nutrition Research Centre, University of Alberta, Edmonton, Alta.; Terri L. Paul, Assistant Professor of Medicine, University of Western Ontario, and endocrinologist, St. Joseph's Health Centre, London, Ont.; Denis Prud'homme, Dean, Faculty of Health Sciences, University of Ottawa, and Behavioural and Metabolic Research Unit, Montfort Hospital, Ottawa, Ont.; Kim Raine, Director and Professor, Centre for Health Promotion Studies, University of Alberta, Edmonton, Alta.; Bruce A. Reeder, Professor and Head, Department of Community Health and Epidemiology, University of Saskatchewan, and staff, Department of Family Medicine, Royal University Hospital, Saskatoon, Sask.; Elizabeth A.C. Sellers, Assistant Professor of Pediatrics and Child Health, Section of Pediatric Endocrinology, University of Manitoba, Winnipeg Children's Hospital, Winnipeg, Man.; Mark S. Tremblay, Professor, College of Kinesiology, University of Saskatchewan, and Senior Scientific Director on Health Measurement, Statistics Canada, Ottawa, Ont.; Michael Vallis, Associate Professor of Psychology, Dalhousie University, and psychologist, Queen Elizabeth II Health Sciences Centre, Halifax, NS; Vivienne A. Vance, Department of Health Studies and Gerontology, Faculty of Applied Health Sciences, University of Waterloo, Waterloo, Ont.; Elinor Wilson, Chief Executive Officer, Canadian Public Health Association, Ottawa, Ont.

**Coauthors**

Geoff Ball, Director, Pediatric Centre for Weight and Health, and Adjunct Professor, Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, Alta.; Bobbi N. Barbarich, dietitian, Pediatric Centre for Weight and Health, Stollery Children's Hospital, Edmonton, Alta.; Glenn Berall, Assistant Professor of Pediatrics, University of Toronto, Chief of Pediatrics, North York General Hospital, and active staff, Division of Gastroenterology and Nutrition, Hospital for Sick Children, Toronto, Ont.; Hany Bissada, Director, Regional Centre for Eating Disorders, University of Ottawa, Ottawa, Ont.; Jean-Pierre Chanoine, Clinical Professor and Head, Endocrinology and Diabetes Unit, British Columbia's Children's Hospital, University of British Columbia, Vancouver, BC; Carol Clarke, registered dietitian, Dietitians of Canada Diabetes, Obesity and Cardiovascular Network; Virginia Desantadina, Division of Medical Nutrition, Department of Pediatrics, North York General Hospital, Toronto, Ont., and pediatrician, Buenos Aires, Argentina; Isabelle Dionne, Professeure agrégée, Faculté d'éducation physique et sportive, Université de Sherbrooke, Sherbrooke, Que.; Eric Doucet, Assistant Professor, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ont.; Judith A. Francis, psychologist, Cardiac Rehabilitation and Secondary Prevention Program, London Health Sciences Centre, London, Ont.; Rami Habib, Assistant Professor of Psychiatry, University of Ottawa, and psychiatrist, Mood Disorders Program, Royal Ottawa Hospital, Ottawa, Ont.; Tracy Hussey, dietitian, McMaster Children's Hospital, Hamilton Health Sciences, Hamilton, Ont.; Alison Irving, research consultant, QEI Research Services, Ottawa, Ont.; Ian Janssen, Assistant Professor, Department of Community Health and Epidemiology, School of Physical and Health Education, Queen's University, Kingston, Ont.; Jacquie Jumpsen, Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, Alta.; Cathy Kubrak, Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, Alta.; Julie Lenk, Department of Agricultural, Food and Nutritional Science, University of Alberta, Edmonton, Alta.; Roselle Martino, Dietitians of Canada Diabetes, Obesity and Cardiovascular Network, Toronto, Ont.; Karen Mornin, dietitian, St. Paul's Hospital, Vancouver, BC; Robert Ross, Professor, School of Physical and Health Education, Faculty of Medicine, Queen's University, Kingston, Ont.; Louis Soucy, Assistant Professor of Psychiatry, University of Ottawa and Royal Ottawa Hospital, Ottawa, Ont.; Andre Tchernof, Assistant Professor, Department of Molecular Endocrinology and Oncology Research Centre, Laval University, Québec, Que.; Dana Whitham, registered dietitian, St. Michael's Hospital, Toronto, Ont.; Melodie Yong, registered dietitian, St. Paul's Hospital, Vancouver, BC

**Evidence-based Review Committee**

Roman Jaeschke, Clinical Professor, Department of Medicine; Dereck Hunt, Associate Professor, Department of Medicine; James Douketis, Associate Professor, Departments of Medicine and of Clinical Epidemiology and Biostatistics, McMaster University, Hamilton, Ont.

---

# 1. Evaluation of evidence-based literature and formulation of recommendations

James D. Douketis

There is no universally accepted method for developing consensus guidelines and practice recommendations, and practices vary.<sup>1-7</sup> Recent methods proposed to formulate recommendations involve a simplified “clear benefits versus risks” or “unclear benefits versus risks” approach that reflects not only the quality of evidence in favour of or against an intervention, but also the potential harms, costs, burdens and importance or value of an intervention to an individual or population.<sup>8-10</sup>

## Process for formulating recommendations

Development of the recommendations in these guidelines was based on a prespecified process overseen by the Steering Committee. Specific domains of study were delegated to groups of content experts who performed systematic literature reviews and were responsible for drafting provisional recommendations for each section.

Recommendations were based on a systematic review of the literature. The following data acquisition, literature review and summation process was used:

1. Determine the question for which a recommendation will be made — The question had to be specific and applicable in everyday clinical practice at the individual level or applicable by governing-administrative-public health groups at the population level.
2. Carry out a systematic review of the literature — Electronic databases (MEDLINE, EMBASE, Cochrane Controlled Clinical Trials Register and HealthSTAR) were searched from time of inception of the database until the end of the review period, identifying all studies (English and non-English) with an English-language abstract. In addition, a manual review of systematic reviews or meta-analyses was conducted to identify studies that may not have been found in the database searches.

Studies were selected if they were randomized controlled trials (RCTs), prospective cohort studies, case-control studies or retrospective cohort studies and if populations or patients were overweight (body mass index [BMI] 25–29.9 kg/m<sup>2</sup>), obese (BMI ≥ 30 kg/m<sup>2</sup>) or at risk for becoming overweight. The study had to include a population or patient follow-up period of at least 1 year (at least 6 months if only limited data were available). Data were abstracted from source studies or from updates of high-quality systematic reviews and meta-analyses. Studies were separated into groups depending on their design. Areas in which relevant literature was lacking were identi-

fied, as they would require consensus agreement in the formulation of recommendations.

3. Determine the level of the evidence for the recommendation — The level of evidence for a recommendation was based on an objective appraisal of the literature according to a prespecified scale as reflected by the study design and study quality. For RCTs, prespecified criteria were used to assess quality.<sup>4</sup> The level of evidence ranged from 1 (highest) to 4 (lowest). (See Table 1, in the Summary chapter.)
4. Determine the grade of recommendation — The grade for a recommendation reflected the level of evidence and several additional factors, including one or more of the following: benefits and risks of an intervention, magnitude of the effects, cost of the intervention, and value (or importance) of an intervention to an individual or population. The grade may be A, B or C (see Table 2, in the Summary chapter).
5. Review of the literature review and recommendations — The first draft of the literature review and accompanying recommendations in each domain of study was subjected to an iterative review process. The provisional recommendations were appraised by an Evidence-based Review Committee, which assessed the appropriateness of each recommendation. Subsequent versions of the recommendations were based on feedback from all members of the Expert Panel, during face-to-face meetings, and on feedback from members of the Steering Committee. The penultimate version of the practice guidelines was reviewed by the Steering Committee and, subsequently, by external stakeholders and experts (including representatives from academia, industry and government and non-government officials). This final draft of the guidelines reflects the feedback of the external reviewers.

## Principles used for formulating recommendations

The approach used to formulate recommendations was based on the following principles:

- A clear question or well-defined issue surrounding an obesity-related intervention was the starting point for review of the literature and formulation of recommendations. (Note: in this context, “intervention” is not confined to therapy, but also includes screening, prevention, dissemination and implementation.)
- Each recommendation is evidence-based — arrived at through a systematic review of the literature — and reflects consensus of the Steering Committee and relevant

Expert Panel members. (For domains of study where evidence is lacking or based on anecdotal experience, a recommendation is based on consensus alone.)

- Each recommendation includes a level of evidence (1 to 4 [see Table 1 in Summary]) and a grade (A, B or C [see Table 2 in Summary]).
- The level of evidence informs the reader about the strength of the evidence in favour of (or against) the intervention and is based on prespecified objective criteria.
- The grade informs the reader about whether an intervention should (or should not) be implemented and reflects both the level of evidence supporting the recommendation *and* a consideration of the harms and costs of the intervention and its importance and value to the individual or population.
- The level of evidence assigned to an intervention is not necessarily linked to a corresponding grade. However, a high grade is less likely in the setting of low-quality evidence.
- A consensus recommendation, which is classified as grade C, is a statement that provides a reasonable approach or guideline for that domain of study. A consensus recommendation reflects insufficient evidence to inform clinical practice or anecdotal evidence only.

## Some considerations regarding the levels of evidence and grades of recommendations

### Why is an evidence-based approach used for all recommendations?

The rationale for a systematic, evidence-based approach for all domains of study is 2-fold. First, it allows a consistent approach to formulating recommendations, allowing application of the same criteria (relating to levels of evidence and grades of recommendations) to be applied with the minimum of bias. Second, a systematic evidence-based approach identifies gaps in knowledge and provides a basis for future research agendas. Furthermore, it challenges investigators to address research questions by using study methods of the highest possible quality.

### What is the rationale for the scheme used to assign a level to evidence?

The approach used to assign levels of evidence is based on 2 premises. First, there is a hierarchy of study design that places greater value on properly done RCTs and less value on observational studies. Second, there is an acknowledgement of the importance of study quality, irrespective of study design.

For example, evidence from RCTs can be classified as level 1 or level 2 depending on their quality, which is determined according to prespecified criteria. Similarly, evidence from observational studies can be classified as level 2 or level 3, depending on the study quality and the magnitude of the effect observed. Finally, level 4 is assigned to evidence that is sparse or anecdotal. This can be considered a default level of evidence and indicates the need for future research in this domain of study.

### What is the rationale for the scheme used to assign a grade to a recommendation?

The approach used to assign a grade to a recommendation is essentially dichotomous: grade A is a strong recommendation in favour of (or against) an intervention, and grade B is an intermediate recommendation in favour of (or against) an intervention. Grade C is a weak, or consensus, recommendation developed largely as a practical consideration to allow expert commentary on domains of study in which evidence is sparse or nonexistent and for which grade A or B would not be feasible.

In the recommendations, the wording “we recommend” is used to express a grade A recommendation, whereas the wording “we suggest” is used to express a grade B recommendation. The wording used for grade C recommendations varies but reflects the uncertainty surrounding the benefits and risks of the intervention.

### Why is there no link between the level of evidence and grade of recommendation?

In some consensus guidelines, the level of evidence dictates the strength of the recommendation. This approach has the advantage that a strong recommendation for an intervention can be made only in the face of the highest level of evidence. On the other hand, this approach has potential drawbacks. It precludes making strong recommendations in domains of study in which RCTs (which provide the highest level of evidence) are not feasible. The second drawback is that it may not allow consideration of factors other than an efficacy assessment of an intervention, such as harms, costs and value to the individual and population.

For example, there are unlikely to be randomized trials to assess the prognostic importance of risk factors for obesity-related diseases or to assess the accuracy of various anthropometric measures of obesity against a reference standard. However, the importance of identifying disease risk factors in overweight or obese people and the benefits of undertaking screening for obesity in populations may be such that a strong recommendation would be warranted. Equally, an intervention that is proven to be efficacious, based on evidence from high-quality randomized trials, should not automatically receive a strong recommendation, because potential harms may not be established, the ratio of benefits to burdens may be small and costs may be prohibitive when considered at the population level (Table 4).

It is likely that an intervention supported by high-quality evidence (level 1) will be accompanied by a strong recommendation (grade A) and that an intervention supported by lower quality evidence (level 2 or 3) will be accompanied by an intermediate recommendation (grade B). However, the approach used allows the consideration of factors beyond that of the quality of evidence (often relating only to efficacy) in developing recommendations. These additional factors include the intervention’s harms, costs and value to the individual or population. Consequently, an intervention associated with high-quality evidence (level 1) may be accompanied



by an intermediate recommendation (grade B) if there are other concerns about the intervention when applied to the individual or population. Conversely, an intervention associated with lower-quality evidence (level 2 or 3) may be accompanied by a strong recommendation (grade A) if the intervention has clear benefits when applied to the individual or population.

## Summary

The development of consensus guidelines for obesity is complex. It involves recommending treatment interventions (the most common application of practice guidelines) and interventions related to screening (at the individual and population levels) and prevention. In formulating these guidelines,

**Table 4:** Interpreting assigned levels of evidence and grades of recommendations

Level of evidence	Grade of recommendation		
	Grade A (strong)	Grade B (intermediate)	Grade C (weak or consensus)
Level 1	<p>The intervention is based on randomized trials without important limitations <i>and</i> has a favourable profile regarding benefits, risks, costs and values</p> <p>Can be applied to most people under most circumstances <i>and</i> is based on the highest level of evidence</p> <p><i>Example:</i> Dietary modification to prevent diabetes in obese adults with impaired glucose tolerance</p>	<p>The intervention has level 1 evidence (to support its efficacy), but other factors may downgrade the strength of recommendation, such as safety, costs or uncertainty about value to the individual or population</p> <p>Can be applied to some people, but this may vary depending on the characteristics of the individual or other circumstances</p> <p><i>Example:</i> Family-oriented behaviour therapy for the treatment of obesity in children</p>	<p>Not applicable (level 1 evidence will confer a grade A or B on a recommendation)</p>
Level 2	<p>The intervention is based on randomized trials with important limitations or observational studies with overwhelming evidence <i>and</i> has a favourable profile regarding benefits, risks, costs and values</p> <p>Can be applied to most people under most circumstances</p> <p>Although not based on the highest level of evidence (because attaining this may not be feasible), other factors make it important to be applied to most people</p> <p><i>Example:</i> Reduction in television watching in children to promote healthy weight</p>	<p>The intervention is based on randomized trials with important limitations or (less commonly) on observational studies with overwhelming findings</p> <p>Can be applied to some people, but this may vary depending on the characteristics of the individual or other circumstances</p> <p><i>Example:</i> Low-carbohydrate dietary interventions for weight loss in adults</p>	<p>Not applicable (level 1 evidence will confer a grade A or B on a recommendation)</p>
Level 3	<p>The intervention is based on observational studies (case-control or cohort studies)</p> <p>Can be applied to most people under most circumstances</p> <p>Although not based on the highest level of evidence (because attaining this may not be feasible), other factors make it important to be applied to most people</p> <p><i>Example:</i> Screening for overweight and obesity in populations</p>	<p>The intervention is based on observational studies (case-control or cohort studies)</p> <p>Can be applied to some people, but this may vary depending on the characteristics of the individual or other circumstances</p> <p><i>Example:</i> Bariatric surgery for weight loss in adults</p>	<p>The intervention is based on sparse evidence (e.g., case series)</p> <p>Carried out with discretion; alternative interventions may be equally reasonable</p> <p><i>Example:</i> Pharmacotherapy for weight loss in adolescents</p>
Level 4	<p>Not applicable (level 4 evidence will confer only a grade C on a recommendation)</p> <p><i>Example:</i> None applicable</p>	<p>Not applicable (level 4 evidence will confer only a grade C on a recommendation)</p> <p><i>Example:</i> None applicable</p>	<p>The intervention is based on anecdotal evidence</p> <p>Carried out with discretion; alternative interventions may be equally reasonable</p> <p><i>Example:</i> Herbal remedies for weight loss</p>

we have attempted to use an evidence-based approach consistently across all domains of study while allowing flexibility so that practical commentary can be made about domains of interest relevant to the practising clinician where evidence is currently lacking. The scheme for developing levels of evidence and grades of recommendations is, to a large extent, tailored to the diversity of subject domains in which commentary is provided and the variability of evidence available to assess these domains. Finally, we acknowledge that the scheme developed here is implicitly imperfect and will require ongoing modification over time.

From the Department of Medicine, McMaster University, St. Joseph's Healthcare, Hamilton, Ont.

**Competing interests:** None declared.

---

## REFERENCES

1. The periodic health examination. Canadian Task Force on the Periodic Health Examination. *CMAJ* 1979;121(9):1193-254.

2. Goldbloom R, Battista RN. The periodic health examination: 1. Introduction. *CMAJ* 1986;134(7):721-3.
3. Douketis JD, Feightner JW, Attia J, et al. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. Canadian Task Force on Preventive Health Care. *CMAJ* 1999;160(4):513-25.
4. US Preventive Services Task Force. Screening for obesity in adults: recommendations and rationale. *Ann Intern Med* 2003;139(11):930-2.
5. Gyorkos TW, Tannenbaum TN, Abrahamowicz M, et al. An approach to the development of practice guidelines for community health interventions. *Can J Public Health* 1994;85:S8-13.
6. Atkins D, Fink K, Slutsky J; Agency for Healthcare Research and Quality, North American Evidence-based Practice Centers. Better information for better health care: the Evidence-based Practice Center program and the Agency for Healthcare Research and Quality. *Ann Intern Med* 2005;142(12 pt 2):1035-41.
7. Eccles M, Clapp Z, Grimshaw J, et al. North of England evidence based guidelines development project: methods of guideline development. *BMJ* 1996;312:760-2.
8. Schunemann HJ, Munger H, Brower S, et al. Methodology for guideline development for the Seventh American College of Chest Physicians Conference on Antithrombotic and Thrombolytic Therapy: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. *Chest* 2004;126(3 Suppl):174S-8S.
9. Grades of Recommendations, Assessment, Development, and Evaluation (GRADE) Working Group. Grading quality of evidence and strength of recommendations: grades of recommendations, assessment, development and evaluation. *BMJ* 2004;328:1490-4.
10. Guyatt G, Gutterman D, Baumann MH, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American college of chest physicians task force. *Chest* 2006;129:174-81.

## 2. Epidemiology of obesity

Peter T. Katzmarzyk

The prevalence of overweight and obesity has increased in Canada over recent decades in both children and adults in all areas of the country.<sup>1-7</sup> According to the most recent estimates from the nutrition component of the 2004 Canadian Community Health Survey,<sup>6</sup> approximately 59% of the adult population is overweight (BMI  $\geq 25$  kg/m<sup>2</sup>) and 23.4% is obese (BMI  $\geq 30$  kg/m<sup>2</sup>). The sheer numbers of people who are overweight and obese highlight a pressing public health problem that shows no signs of improving in the near future.

### Temporal trends and current status of obesity in Canada

The increase in obesity that has been observed in Canada in recent years has mirrored that in the United States to some extent (Fig. 2). In Canada, the prevalence of adult obesity increased from approximately 11% in 1972 to 24% (age-adjusted) in 2005;<sup>6,17</sup> in the United States, it increased from approximately 15% in 1976-80 to 32% in 2003-04.<sup>18,19</sup>

Although the overall prevalence of overweight and obesity is high in Canada, some groups may be at particularly high risk. The relation between socioeconomic status and obesity is extremely complex, and there has been little research on this issue in Canada. According to self-reported data from the 2000/01 Canadian Community Health Survey (CCHS),<sup>20</sup> the likelihood of being overweight increases with income among men but decreases with income among women<sup>21</sup>; however, this relation was not as clear when data from the 2004 CCHS were examined.<sup>6,22</sup> One study has demonstrated that Canadian low-income children are more likely than those living in higher-income families to be overweight. For every \$10 000 increase in family income, a child's risk of being overweight decreases by 3%, and for each additional year of paternal education the risk decreases by 4%.<sup>5</sup> In western Canada, men and women living in rural areas are significantly more likely than their urban counterparts to be obese, although this observation does not hold for other regions of the country.<sup>23</sup>

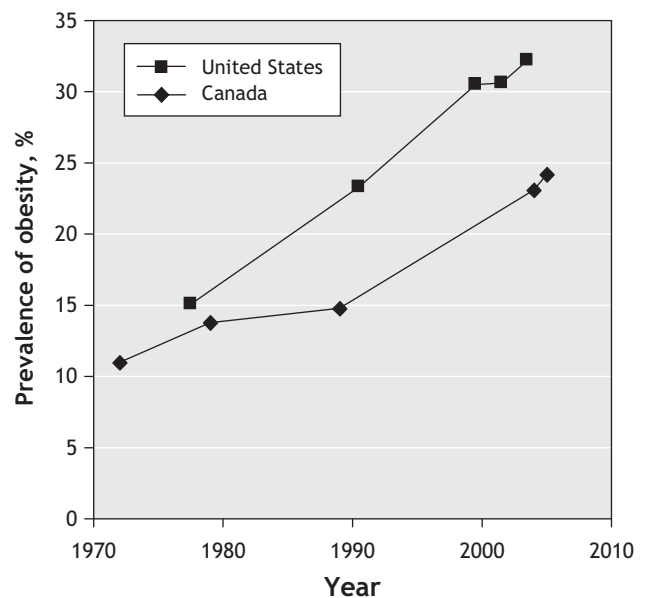
Unfortunately, little information on the prevalence of obesity among different ethnic groups is available. Self-reported data from the CCHS indicate that, among the major ethnic groups in Canada, Aboriginal people (living off reserve) have the highest prevalence of both overweight and obesity.<sup>24</sup> High prevalence of obesity among Aboriginal people has also been documented in other Canadian studies.<sup>25,26</sup>

Geographic variation in prevalence of obesity also occurs (Fig. 3). Of particular note is the high prevalence ( $\geq 20\%$ ) in

eastern and northern Canada. The geographic distribution of obesity in adults is similar to that of overweight in children and adolescents.<sup>5,7</sup>

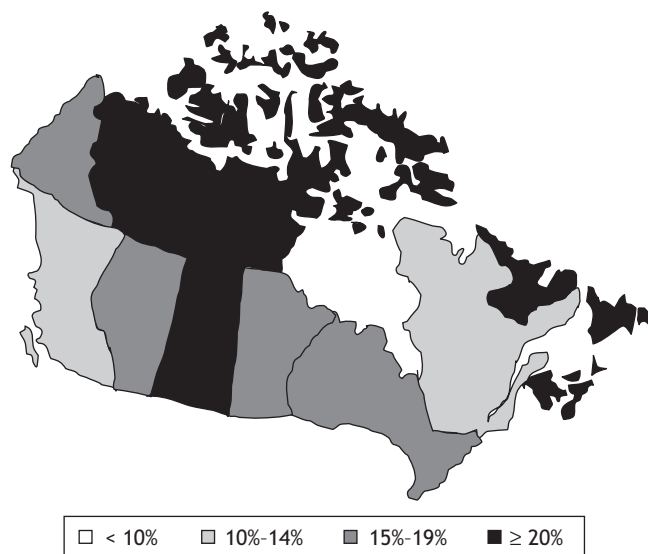
Although the prevalence of obesity has increased among adults in Canada, the increases observed among children and adolescents are particularly troubling (Fig. 4). The prevalence of obesity increased from approximately 3% among boys and girls in 1978/79 to 8% in 2004 based on international BMI cutoff points.<sup>28</sup> This dramatic increase is cause for concern, since there is a tendency for obese children to remain obese as adults.<sup>29</sup> Given these trends among children and adolescents, it is likely that the prevalence of obesity among adults will continue to increase as the current generation of children enters adulthood.

Although current surveillance of obesity in Canada relies solely on BMI calculation, waist circumference is a useful an-

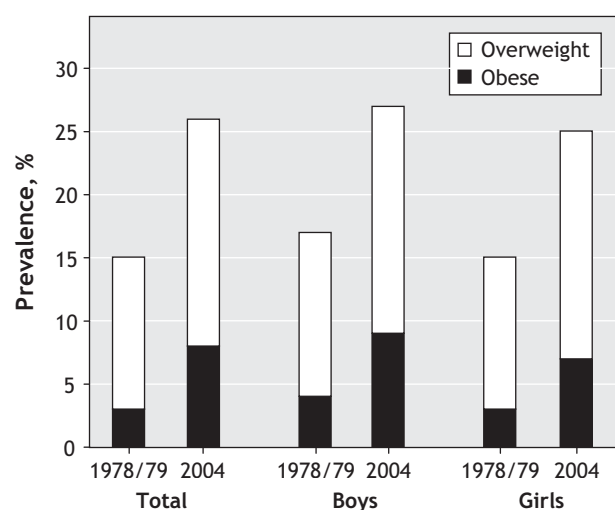


**Fig. 2:** Temporal trends in the prevalence of measured obesity in Canada and the United States. Sources: US National Health and Nutrition Examination Surveys,<sup>8-12</sup> 1972 Canada Nutrition Survey,<sup>13</sup> Canada Health Survey, 1978-1979,<sup>14</sup> Canadian Heart Health Survey, 1986-1992<sup>15</sup> and Canadian Community Health Surveys.<sup>6,17</sup> The estimates for Canada are age-standardized to the 2004 Canadian Community Health Survey,<sup>6</sup> and the estimates for the United States are age-standardized to the 2000 population census.<sup>16</sup>

thropometric marker for identifying those at increased health risk due to abdominal obesity.<sup>30,31</sup> Unfortunately, waist circumference is not regularly measured in national health surveys. Obesity surveillance in Canada has relied mainly on self-reporting of height and weight, either over the telephone or in face-to-face interviews. Given that people tend to over-report their height and under-report their weight,<sup>32,33</sup> which leads to underestimates of average BMI,<sup>34</sup> estimates of the prevalence of overweight and obesity that rely on these data are conservative. For example, in the United States, self-reported data from the 2000 Behavioural Risk Factor Surveillance System indicated that the prevalence of obesity was



**Fig. 3:** Geographic variation in the prevalence of self-reported obesity among adults in 2005.<sup>27</sup>



**Fig. 4:** Changes in the prevalence of obesity among Canadian children (aged 2-17 years), 1978/79 to 2004. Source: Adapted from Shields.<sup>7</sup>

19.8%,<sup>35</sup> whereas direct measurements from the 1999/2000 National Health Examination Survey III (NHANES III) indicated that the prevalence of obesity was 30.5%.<sup>36</sup> This discrepancy highlights the importance of measuring height, weight and waist circumference. It should be noted that heights and weights were measured directly in subsamples of both the 2004 and 2005 CCHS, and a planned Canadian Health Measures Survey will include measurement of height, weight and waist circumference.

## Public health impact of obesity

There is clear evidence that obese people are at increased risk of health problems, including type 2 diabetes, coronary artery disease, stroke, osteoarthritis and certain cancers.<sup>37</sup> In addition to affecting personal health, these increased risks translate into an increased burden on the health care system.<sup>38,39</sup> Approximately 1 in 10 premature deaths among Canadian adults 20-64 years of age may be directly attributable to overweight and obesity.<sup>40</sup> Thus, the continuing epidemic of obesity in Canada is exacting a toll on population health. Sweeping prevention and intervention strategies are required to slow or reverse the current trends.

## Recommendations

1. Because of the health impact of the rising prevalence and incidence of overweight and obesity in Canada, we recommend implementing strategies directed at the prevention and treatment of overweight and obesity in children, adolescents and adults [*grade A, level 3*].
2. Because of the lack of adequate information on the prevalence of obesity and related risk factors in Canada, particularly among subgroups of the population, we recommend the creation of a national surveillance system that incorporates, at a minimum, measurements of height, weight and waist circumference [*grade A, level 3*].

From the Department of Community Health and Epidemiology, School of Kinesiology and Health Studies, Queen's University, Kingston, Ont.

**Competing interests:** None declared.

## REFERENCES

1. Torrance GM, Hooper MD, Reeder BA. Trends in overweight and obesity among adults in Canada (1970-1992): evidence from national surveys using measured height and weight. *Int J Obes Relat Metab Disord* 2002;26:797-804.
2. Tremblay MS, Katzmarzyk PT, Willms JD. Temporal trends in overweight and obesity in Canada, 1981-1996. *Int J Obes Relat Metab Disord* 2002;26:538-43.
3. Katzmarzyk PT. The Canadian obesity epidemic, 1985-1998. *CMAJ* 2002;166:1039-40.
4. Katzmarzyk PT. The Canadian obesity epidemic: an historical perspective. *Obes Res* 2002;10:666-74.
5. Willms JD, Tremblay MS, Katzmarzyk PT. Geographic and demographic variation in the prevalence of overweight Canadian children. *Obes Res* 2003;11:668-73.
6. Tjepkema M. Measured obesity: adult obesity in Canada — measured height and weight. In: *Nutrition: findings from the Canadian Community Health Survey* 2004; issue 1. Cat no 82-620-MWE2005001. Available: [www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf](http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf) (accessed 2007 Jan 17).
7. Shields M. Measured obesity: overweight Canadian children and adolescents. In: *Nutrition: findings from the Canadian Community Health Survey* 2004; issue 1. Cat no 82-620-MWE2005001. Available: [www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf](http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf) (accessed 2007 Jan 17).
8. National Health and Nutrition Examination Survey: NHANES II data files docu-

- mentation codebook, 1976–80. [Web site of the US Centers for Disease Control and Prevention]. Available: [www.cdc.gov/nchs/about/major/nhanes/nhanesi.htm](http://www.cdc.gov/nchs/about/major/nhanes/nhanesi.htm) (accessed 2007 Jan 21).
9. National Health and Nutrition Examination Survey: NHANES III data files. [Web site of the US Centers for Disease Control and Prevention]. Available: [www.cdc.gov/nchs/about/major/nhanes/nh3data.htm](http://www.cdc.gov/nchs/about/major/nhanes/nh3data.htm) (accessed 2007 Jan 21).
  10. National Health and Nutrition Examination Survey: NHANES 1999–2000. [Web site of the US Centers for Disease Control and Prevention]. Available: [www.cdc.gov/nchs/about/major/nhanes/nhanes99\\_00.htm](http://www.cdc.gov/nchs/about/major/nhanes/nhanes99_00.htm) (accessed 2007 Jan 21).
  11. National Health and Nutrition Examination Survey: NHANES 2001–2002. [Web site of the US Centers for Disease Control and Prevention]. Available: [www.cdc.gov/nchs/about/major/nhanes/nhanes01-02.htm](http://www.cdc.gov/nchs/about/major/nhanes/nhanes01-02.htm) (accessed 2007 Jan 21).
  12. National Health and Nutrition Examination Survey: NHANES 2003–2004. [Web site of the US Centers for Disease Control and Prevention]. Available: [www.cdc.gov/nchs/about/major/nhanes/nhanes2003-2004/nhanes03\\_04.htm](http://www.cdc.gov/nchs/about/major/nhanes/nhanes2003-2004/nhanes03_04.htm) (accessed 2007 Jan 21).
  13. Nutrition Canada. 1970–1972 Canada Nutrition Survey. Microdata — codebook. Available: <http://prod.library.utoronto.ca:8090/datalib/codebooks/nac/g0000577/nutrition70.htm> (accessed 2007 Mar 5).
  14. Statistics Canada. Canada Health Survey, 1978–1979. Data users guide. Available: [http://prod.library.utoronto.ca:8090/datalib/codebooks/cst/health/chs78\\_79.pdf](http://prod.library.utoronto.ca:8090/datalib/codebooks/cst/health/chs78_79.pdf) (accessed 2007 Mar 5).
  15. MacLean DR, Petrasovits A, Nargundkar M, et al; Canadian Heart Health Surveys Research Group. Canadian heart health surveys: a profile of cardiovascular risk. Survey methods and data analysis. *CMAJ* 1992;146:1969-74.
  16. Population Division; US Census Bureau. Table 1: Annual estimates of the population by sex and five-year age groups for the United States: April 1, 2000 to July 1, 2005. 2006 May. NC-EST2005-01. Available: [www.census.gov/popest/national/asrh/NC-EST2005/NC-EST2005-01.xls](http://www.census.gov/popest/national/asrh/NC-EST2005/NC-EST2005-01.xls) (accessed 2006 Dec 27).
  17. Health indicators. Ottawa: Statistics Canada. Cat no 82-221-XIE. Available: [www.statcan.ca/english/freepub/82-221-XIE/82-221-XIE2006001.htm](http://www.statcan.ca/english/freepub/82-221-XIE/82-221-XIE2006001.htm) (accessed 2007 Jan 17).
  18. Flegal KM, Carroll MD, Kuczmarski RJ, et al. Overweight and obesity in the United States: prevalence and trends, 1960–1994. *Int J Obes Relat Metab Disord* 1998;22:39-47.
  19. Ogden CL, Carroll MD, Curtin LR, et al. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA* 2006;295:1549-55.
  20. Canadian Community Health Survey, cycle 1.1 (2000–2001). Ottawa: Statistics Canada; 2001. Available: [www.statcan.ca/english/concepts/health/index.htm](http://www.statcan.ca/english/concepts/health/index.htm) (accessed 2007 Feb 23).
  21. *Improving the health of Canadians*. Ottawa: Canadian Institute for Health Information; 2004. Available: [secure.cih.ca/cihiweb/dispPage.jsp?cw\\_page=PG\\_39\\_E&cw\\_topic=39&cw\\_rel=AR\\_322\\_E](http://secure.cih.ca/cihiweb/dispPage.jsp?cw_page=PG_39_E&cw_topic=39&cw_rel=AR_322_E) (accessed 2007 Jan 21).
  22. *Improving the health of Canadians: promoting healthy weights*. Ottawa: Canadian Institute for Health Information; 2006. Available: [www.cih.ca/cihiweb/dispPage.jsp?cw\\_page=PG\\_470\\_E&cw\\_topic=470&cw\\_rel=AR\\_1217\\_E](http://www.cih.ca/cihiweb/dispPage.jsp?cw_page=PG_470_E&cw_topic=470&cw_rel=AR_1217_E) (accessed 2007 Jan 21).
  23. Reeder BA, Chen Y, Macdonald SM, et al; Canadian Heart Health Surveys Research Group. Regional and rural-urban differences in obesity in Canada. *CMAJ* 1997;157(Suppl 1):S10-6.
  24. Tremblay MS, Perez C, Ardern CI, et al. Obesity, overweight and ethnicity. *Health Rep* 2005;16:23-34.
  25. Anand SS, Yusuf S, Jacobs R, et al. Risk factors, atherosclerosis, and cardiovascular disease among Aboriginal people in Canada: the Study of Health Assessment and Risk Evaluation in Aboriginal Peoples (SHARE-AP). *Lancet* 2001;358:1147-53.
  26. Katzmarzyk PT, Malina RM. Obesity and relative subcutaneous fat distribution among Canadians of First Nation and European ancestry. *Int J Obes Relat Metab Disord* 1998;22:1127-31.
  27. Obesity among Canadian adults: 2005 Canadian Community Health Survey. Ottawa: Statistics Canada; 2006. Health Indicators. Vol 2006, issue 1. Cat no 82-221-XIE.
  28. Cole TJ, Bellizzi MC, Flegal KM, et al. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000;320:1240-3.
  29. Whitaker RC, Wright JA, Pepe MS, et al. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med* 1997;337:869-73.
  30. Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference, and health risk: evidence in support of current National Institutes of Health guidelines. *Arch Intern Med* 2002;162:2074-9.
  31. Ardern CI, Katzmarzyk PT, Janssen I, et al. Discrimination of health risk by combined body mass index and waist circumference. *Obes Res* 2003;11:135-42.
  32. Rowland ML. Self-reported weight and height. *Am J Clin Nutr* 1990;52:1125-33.
  33. Paltal M, Prineas RJ, Berman R, et al. Comparison of self-reported and measured height and weight. *Am J Epidemiol* 1982;115:223-30.
  34. Paccard F, Wietlisbach V, Rickenbach M. Body mass index: comparing mean values and prevalence rates from telephone and examination surveys. *Rev Epidemiol Sante Publique* 2001;49:33-40.
  35. Mokdad AH, Bowman BA, Ford ES, et al. The continuing epidemics of obesity and diabetes in the United States. *JAMA* 2001;286:1105-200.
  36. Flegal KM, Carroll MD, Ogden CL, et al. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA* 2002;288:1723-7.
  37. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults — the evidence report. *Obes Res* 1998;6(Suppl 2):51-209S.
  38. Birmingham CL, Muller JL, Palepu A, et al. The cost of obesity in Canada. *CMAJ* 1999;160:483-8.
  39. Katzmarzyk PT, Janssen I. The economic costs associated with physical inactivity and obesity in Canada: an update. *Can J Appl Physiol* 2004;29:90-115.
  40. Katzmarzyk PT, Ardern CI. Overweight and obesity mortality trends in Canada, 1985–2000. *Can J Public Health* 2004;95:16-20.

### 3. Classification of overweight and obesity in adults

Jean-Pierre Després, Andre Tchernof

**O**besity, which is characterized by an excess amount of body fat, results from complex interactions between environmental and genetic factors. Obesity is associated with increased morbidity and mortality, and considerable evidence suggests a graded relation between the latter and body fat content. Because of the worldwide recognition that obesity adversely affects health, the National Institutes of Health<sup>1</sup> and the North American Association for the Study of Obesity,<sup>2</sup> among other organizations, have published guidelines for the assessment and management of obesity. Other documents have also been released,<sup>3-5</sup> all emphasizing the need to assess and treat obesity and its related complications.

To avoid misclassification or any potential harm arising from systematic obesity screening, only obese patients at the highest risk of morbidity and mortality should be identified, and the presence of comorbidities should guide clinical management. In this regard, several factors must be considered in the classification of overweight and obesity in adults.

#### Strengths and limitations of body mass index as a measure of total adiposity

By far the most widely recognized indicator of obesity is BMI (i.e., weight divided by height squared [ $\text{kg}/\text{m}^2$ ]). The World Health Organization (WHO) has published international standards for classifying overweight and obesity, which have been

endorsed in the United States and Canada<sup>2,6,7</sup> (Table 5). Overweight is defined as a BMI between  $25 \text{ kg}/\text{m}^2$  and just below  $30 \text{ kg}/\text{m}^2$ , whereas obesity is defined as a BMI of  $30 \text{ kg}/\text{m}^2$  and above. Among patients with BMI values above  $30 \text{ kg}/\text{m}^2$ , several subclasses of obesity are proposed based on the severity of the condition. A BMI of  $40 \text{ kg}/\text{m}^2$  and above defines severe or extreme obesity. Patients with extreme obesity may be genetically vulnerable to a permissive environment and may have a distinct form of obesity. Such severe forms of obesity may be more difficult to treat using environmental approaches and may even justify antiobesity surgery (see chapter 15).<sup>1,2</sup>

Although BMI is a simple and convenient measure, it has some limitations. First, it is a crude index of body composition, especially in the  $25\text{--}30 \text{ kg}/\text{m}^2$  range. For example, physically active people who regularly perform muscle-resistance exercise (e.g., football players) may sometimes be considered overweight or even obese even though they have normal, or even low, body fat content.

Second, BMI fails to provide information about the distribution of body fat. The way in which excess body fat accumulates differs widely among people, especially with respect to location of fat stores in the body. Considerable evidence from prospective observational and metabolic studies shows that overweight or obese people with an excess of abdominal fat — central or abdominal obesity — represent a subgroup at highest risk of type 2 diabetes and cardiovascular disease (CVD).<sup>8-11</sup> Thus, in clinical practice, it is of paramount impor-

**Table 5:** Classification of overweight and obesity by body mass index (BMI) and associated disease risk

Category	BMI, $\text{kg}/\text{m}^2$	Obesity class	Disease risk* (relative to normal weight and waist circumference [WC])	
			Men, WC $\leq 102 \text{ cm}\dagger$ Women, WC $\leq 88 \text{ cm}$	Men, WC $> 102 \text{ cm}\dagger$ Women, WC $> 88 \text{ cm}$
Underweight	$< 18.5$		—	—
Normal‡	$18.5\text{--}24.9$		—	—
Overweight	$25.0\text{--}29.9$		Increased	High
Obese				
Mild	$30.0\text{--}34.9$	I	High	Very high
Moderate	$35.0\text{--}39.9$	II	Very high	Very high
Severe or extreme	$\geq 40$	III	Extremely high	Extremely high

\*For type 2 diabetes, hypertension and cardiovascular disease.

†Waist circumference cutoff points may be lower in some populations (e.g., older individuals, Asian population), especially in the presence of features of the metabolic syndrome (e.g., hypertriglyceridemia).

‡Increased waist circumference can be a marker for increased risk even in individuals of normal weight.

Source: Adapted from World Health Organization.<sup>1,6</sup>

tance to measure an index of abdominal fat in addition to BMI.

A common example of central obesity is the selective deposition of visceral abdominal fat that occurs with age.<sup>12,13</sup> This can be accompanied by decreased muscle mass, leading to no change in weight or BMI. Women going through menopause may also experience selective deposition of abdominal visceral fat and loss of muscle mass. These changes would not necessarily be revealed by body weight, which may be stable, despite an increase in diabetogenic and atherogenic visceral adipose tissue.<sup>14-16</sup> In addition, some ethnic groups may be especially prone to visceral or subcutaneous abdominal fat accumulation at any given BMI value. Thus, although it is important to measure BMI as an initial step in clinical practice, it is also important to follow up with an assessment of abdominal fat accumulation.

### Abdominal obesity and the metabolic syndrome

The notion of abdominal obesity is recognized in current obesity guidelines.<sup>1,2,6,17,18</sup> The guidelines of the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (NCEP-ATP III, Adult Treatment Panel III) state that abdominal obesity is the most common cause of a cluster of metabolic abnormalities — the metabolic syndrome — which includes a specific atherogenic dyslipidemia, insulin resistance, impaired fibrinolysis and an inflammatory profile.<sup>17</sup> The NCEP-ATP III has proposed the use of simple clinical tools to identify those likely to have metabolic syndrome (Table 6). For example, an abdominally obese person with increased triglyceride levels and reduced high-density lipoprotein (HDL) cholesterol concentration would have metabolic syndrome according to the NCEP-ATP III criteria. Such a person would, therefore, be considered at increased risk of CVD.

The important concept behind the NCEP-ATP III criteria is

**Table 6:** Clinical criteria recommended by the NCEP-ATP III to identify people with metabolic syndrome<sup>17</sup>

Risk factor	Defining level
Abdominal obesity (waist circumference), cm*	
Men	> 102
Women	> 88
Triglycerides, mmol/L	≥ 1.69
HDL cholesterol, mmol/L	
Men	< 1.03
Women	< 1.29
Blood pressure, mm Hg	≥ 130/≥ 85
Fasting glucose, mmol/L	≥ 5.6 <sup>19</sup>

Note: NCEP-ATP III = National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III).

\*Waist circumference cutoff points may be lower in some populations and subgroups.

the identification of abdominal obesity as the most prevalent form of the metabolic syndrome. NCEP-ATP III guidelines also imply that an increased waist circumference alone is insufficient to identify high-risk abdominally obese patients, as it must be accompanied by markers of increased risk of type 2 diabetes and CVD (i.e., elevated triglyceride levels, low HDL cholesterol concentration, increased glycemia and elevated blood pressure). From a therapeutic standpoint (in addition to treating traditional CVD risk factors when present), the criteria identify waist circumference as a key target.

Although publication of the NCEP-ATP III guidelines was a major breakthrough, because they emphasize the importance of assessing abdominal obesity in clinical practice, several issues will have to be addressed to refine the concept and help clinicians in their practice. The 5 criteria and cutoff values proposed by the NCEP-ATP III to diagnose metabolic syndrome reflect the consensus of experts. They have not been validated regarding their ability to provide optimal discrimination of individuals with both metabolic syndrome and a related increase in risk of chronic heart disease. As a consequence, since their publication, clinicians have often confused the definition of metabolic syndrome with the NCEP-ATP III criteria. The criteria are simple surrogate variables to identify, in clinical practice, high-risk patients with the cluster of abnormalities of the metabolic profile. These people are likely to be characterized by abdominal obesity, insulin resistance and an atherogenic dyslipidemic state, as well as a prothrombotic, inflammatory profile. This profile may or may not be accompanied by hyperglycemia and hypertension. Findings from the Quebec Cardiovascular Study<sup>20</sup> and from the Botnia Study<sup>21</sup> have revealed that some features of the metabolic syndrome not included in the current list of established CVD risk factors substantially increase disease risk, even in the absence of such classic predictors as elevated low-density lipoprotein (LDL) cholesterol, smoking, diabetes and hypertension. Further work is clearly warranted to quantify the added value of considering abdominal obesity and the metabolic syndrome in assessing global CVD risk.

### Assessing abdominal obesity: waist circumference or waist-to-hip ratio

Although guidelines recommend consideration of abdominal obesity in the identification of high-risk overweight or obese patients,<sup>1,2,17,18</sup> for any given BMI value, there is considerable variation in waist circumference, and an increased waist circumference predicts increased morbidity and mortality beyond those indicated by BMI alone.<sup>22,23</sup> Recently published data from the INTERHEART case-control study<sup>24</sup> clearly showed that, for any given BMI quintile, an elevated waist-to-hip ratio is predictive of an increased risk of myocardial infarction. These results provide further support to the notion that it is important to consider body fat distribution in the assessment of the risk of myocardial infarction.

However, several points must be addressed regarding the use of either waist circumference or waist-to-hip ratio in clinical practice. First, waist circumference is an index of the *absolute* amount of abdominal fat, whereas waist-to-hip ratio

provides an estimate of the *relative* accumulation of abdominal fat.<sup>25</sup> Therefore, in clinical practice, 2 people with markedly different BMI values might have similar waist-to-hip ratios, but strikingly different amounts of visceral adipose tissue associated with substantial variation in their metabolic risk profile. However, these 2 people would have markedly different waist circumferences and, therefore, substantial differences in total abdominal fat.

Results of the INTERHEART study emphasizing the relation between waist-to-hip ratio and the risk of myocardial infarction irrespective of BMI are consistent with some previously published prospective studies<sup>26,27</sup> with ischemic heart disease as the end point. However, the general literature indicates that both BMI and the amount of abdominal fat are important factors in predicting CVD events and risk of type 2 diabetes.<sup>10,22,28–32</sup> In the INTERHEART study, blood samples were not collected when patients were in a fasting state. Thus, the investigators had to rely on the ratio of apolipoprotein B to apolipoprotein AI, which is less affected by eating than lipoprotein lipids, to detect the presence of an atherogenic dyslipidemia. It remains to be seen whether waist-to-hip ratio would be superior to the measurement of waist circumference combined with simple clinical markers of the metabolic syndrome, such as elevated triglyceride and reduced HDL cholesterol concentrations, in predicting the risk of myocardial infarction.

Finally, the loss of abdominal fat resulting from weight loss will be reflected by a decrease in waist circumference, but not necessarily by a change in waist-to-hip ratio. In addition, the waist-to-hip ratio measurement doubles the potential for error. Waist circumference may be of more limited value in assessing extremely obese patients, but changes in this variable over time may represent a better index of change in abdominal fat than a change in waist-to-hip ratio.

An increased waist circumference alone is not sufficient to identify high-risk patients with excess visceral adiposity and related metabolic abnormalities. In this regard, the NCEP-ATP III<sup>17</sup> and the International Diabetes Federation guidelines<sup>33</sup> have suggested that both increased waist circumference and simple indices of metabolic disorder are necessary to identify high-risk people with abdominal obesity and those with metabolic syndrome. Thus, on the basis of international consensus on the need to measure waist circumference as a first step in evaluating the risk associated with a given BMI<sup>17,33</sup> and considering the importance of simple markers that can be measured in clinical practice (level of triglycerides in fasting patients, levels of HDL-cholesterol and glucose and blood pressure), we recommend that waist circumference be measured as the best simple index of abdominal obesity and that cutoff points for waist circumference be used in clinical practice.

Waist circumference should be measured while the patient is in the standing position. The measuring tape should be positioned in a horizontal plane at the level of the top of the iliac crest, which is used as a landmark to standardize measurement. The person should be standing with their feet 25–30 cm apart and arms hanging naturally at the sides. The measurer should stand to the side of the patient and fit the tape snugly around the waist, horizontal to the floor. The circumference

should be measured to the nearest 0.5 cm, with the patient's abdominal muscles relaxed at the end of a normal expiration.

## Cutoff values for waist circumference

Although there is a graded relation between waist girth and the prevalence of comorbidities, cutoff values have been proposed for waist circumference, as for the BMI. For example, the WHO Obesity Task Force has proposed a classification system based on both BMI and waist circumference. This was later endorsed by the National Heart, Lung, and Blood Institute expert panel on the identification, evaluation and treatment of overweight and obesity<sup>6</sup> (Table 5).

Although the measurement of waist circumference is recommended in several guidelines, the cutoff values proposed to define abdominal obesity in men (102 cm) and women (88 cm) are those corresponding to BMI values of 30 kg/m<sup>2</sup> in each sex. These values have not been validated for their ability to discriminate clinical events. They are also likely to differ in various populations (e.g., men v. women, different age groups, different ethnic populations). Indeed, there are population differences in susceptibility to visceral fat deposition and in vulnerability to develop complications for any given level of abdominal visceral fat. Lower cutoff values will possibly be proposed for Asian and South Asian populations, since complications occur at much lower BMI values in these groups.<sup>34,35</sup> On the other hand, black people are less prone than white people to visceral adipose tissue accumulation for any given level of total body fat or waist circumference,<sup>36–39</sup> and this may be why they have lower triglyceride and apolipoprotein B levels than white people have.<sup>36</sup> Thus, cutoff values defining abdominal obesity in various populations will have to be based on clinical events rather than on consensual agreement. Considerable population-based research is needed in this area.

Recently, the International Diabetes Federation put together a consensus committee to revise WHO guidelines to identify patients with the metabolic syndrome in an attempt to align these recommendations with the NCEP-ATP III guidelines. It was recognized that the most prevalent form of the metabolic syndrome was found in patients with an expanded waistline. On the basis of this notion, increased waist circumference was recommended as an obligatory criterion to identify individuals likely to have the metabolic syndrome (Box 1). Under the International Diabetes Federation guidelines, diagnosis of the metabolic syndrome requires an increased waist circumference combined with 2 of the 4 additional NCEP-ATP III clinical criteria. However, the International Diabetes Federation committee felt that further work was needed to validate the proposed waist circumference cutoff points. Given the evidence that in other parts of the world, such as South Asia, lower waist circumference values are associated with increased risk of complications,<sup>34,35</sup> the International Diabetes Federation has recommended population-specific waist circumference values. These are provided in Table 7. Again, these waist circumference values have not been fully validated against the development of clinical events, an objective that will require further work.



Irrespective of the population considered, an increased waist circumference combined with increased fasting triglyceride levels with or without additional metabolic abnormalities<sup>29</sup> could represent initial identification of a subgroup of people who require further investigation for the presence of an altered CVD and metabolic disease risk profile. For example, the combination of a waist circumference > 90 cm and a

**Box 1: International Diabetes Federation criteria for the diagnosis of the metabolic syndrome<sup>33</sup>**

For a person to be classified as having the metabolic syndrome they must have:

*Central obesity:* waist circumference  $\geq 94$  cm for Europid men and  $\geq 80$  cm for Europid women; ethnicity-specific values for other groups

plus any 2 of the following 4 criteria:

- *Raised triglyceride level:*  $\geq 1.7$  mmol/L or specific treatment for this lipid abnormality
- *Reduced HDL cholesterol:*  $< 1.03$  mmol/L in males and  $< 1.29$  mmol/L in females or specific treatment for this lipid abnormality
- *Raised blood pressure:* systolic BP  $\geq 130$  mm Hg or diastolic BP  $\geq 85$  mm Hg, or treatment of previously diagnosed hypertension
- *Raised fasting plasma glucose level:*  $\geq 5.6$  mmol/L or previously diagnosed type 2 diabetes. (If above 5.6 mmol/L, oral glucose tolerance test is strongly recommended, but not necessary to define presence of the syndrome.)

Note: BP = blood pressure, HDL = high-density lipoprotein.

**Table 7: Ethnic-specific values for waist circumference**

Country or ethnic group	Waist circumference* (as a measure of central obesity), cm	
	Men	Women
Europid*	$\geq 94$	$\geq 80$
South Asian, Chinese†	$\geq 90$	$\geq 80$
Japanese‡	$\geq 85$	$\geq 90$
South and Central American	Use South Asian cutoff points until more specific data are available	
Sub-Saharan African	Use European cutoff points until more specific data are available	
Eastern Mediterranean and Middle East (Arab)	Use European cutoff points until more specific data are available	

\*In future epidemiologic studies of populations of Europid origin, prevalence should be given using both European and North American cutoff points to allow better comparisons. In the United States, the NCEP-ATP III values (men > 102 cm, women > 88 cm) are likely to continue to be used for clinical purposes. However, it is strongly recommended that for epidemiologic studies and, wherever possible, for case detection, ethnic-specific cutoff points should be used for people of the same ethnic group wherever they are found. Thus, the criteria recommended for Japan would also be used in expatriate Japanese communities, as would those for South Asian men and women regardless of place and country of residence.

†Based on a Chinese, Malay and Asian Indian population.

‡Subsequent data analyses suggest that Asian values should be used for Japanese populations until more data are available.

fasting triglyceride level > 2 mmol/L may be sufficient to identify the subgroup of middle-aged men with the features of the metabolic syndrome.<sup>40</sup> Recently published evidence<sup>40-48</sup> suggests that this “hypertriglyceridemic waist” phenotype may indeed represent a useful clinical marker of the metabolic syndrome. In addition, the patient’s history as well as a family history of type 2 diabetes and CVD should be investigated. Global risk of chronic heart disease should also be assessed, based on the presence of classic risk factors. In this regard, better risk assessment algorithms incorporating traditional CVD risk factors and emerging markers associated with abdominal obesity are also much needed. In Canada, the Working Group on Hypercholesterolemia and Other Dyslipidemias<sup>49</sup> recommends the calculation of a global chronic heart disease risk score to guide clinical management. This score is helpful to identify obese patients at highest risk of chronic heart disease. The presence of the metabolic syndrome may bring the patient into a higher risk category than that predicted by the Framingham global score (i.e., from low to moderate or from moderate to high).

## Conclusions

In summary, because of its critical role in the etiology of the metabolic syndrome, abdominal obesity must be assessed in clinical practice. Both BMI and waist circumference should be measured in all patients. The presence of the metabolic syndrome should not automatically put the patient in the highest risk category for chronic heart disease (i.e., 10-year risk  $\geq 20\%$ ). In the absence of classic risk factors, a patient with the metabolic syndrome would be considered to be at moderately elevated risk of chronic heart disease. Numerous issues should be addressed in future studies to improve the assessment and management of abdominally obese patients with the metabolic syndrome (Box 2). Waist circumference and the presence of comorbidities or of obesity-related risk factors should also be assessed when using a BMI of 30 kg/m<sup>2</sup> or greater as a simple and convenient index of obesity. Physi-

**Box 2: Metabolic syndrome and risk of chronic heart disease (CHD) – issues for research**

- Impact of abdominal obesity and the metabolic syndrome on CHD risk in patients with type 2 diabetes
- Impact of abdominal obesity and the metabolic syndrome on CHD risk in the nondiabetic population
- Critical markers (and cutoff values) for identification and quantification of related CHD risk
- Identification of critical targets for the prevention of type 2 diabetes and CHD in high-risk patients
- Susceptibility of various populations to the metabolic syndrome
- Population differences in susceptibility to deposition of visceral adipose tissue
- Population differences in susceptibility to complications (type 2 diabetes, CHD) for any given excess of visceral adipose tissue

cians should consider that a high BMI alone does not necessarily reflect increased disease risk. Also, a high waist circumference value observed in isolation does not always indicate high-risk abdominal obesity. Patients with abdominal obesity and CVD risk factors (including type 2 diabetes or metabolic syndrome), require therapeutic support to reduce weight and waist circumference. Large intervention studies have shown that lifestyle intervention programs or pharmacotherapy (inducing weight loss and reduction in waist circumference) could substantially decrease the risk of type 2 diabetes among high-risk people with impaired glucose tolerance<sup>20,21</sup> or with obesity.<sup>40</sup> Finally, population differences in susceptibility to deposition of visceral adipose tissue and to complications should be investigated.

## Recommendations

1. We recommend measuring body mass index (BMI; weight in kilograms divided by height in metres squared) in all adults [*grade A, level 3<sup>2-4</sup>*].
2. We recommend measuring waist circumference in all adults to assess obesity-related health risks [*grade A, level 3<sup>3,4</sup>*].
3. Population-based research is needed to help establish ethnic-specific cutoff values for waist circumference, with optimal sensitivity and specificity for discriminating clinical events [*grade C, level 4*].

From the Québec Heart Institute, Laval Hospital Research Centre, Sainte-Foy, Que. (Després); and Molecular Endocrinology and Oncology Research Centre, Laval University Medical Centre, Québec, Que. (Tchernof)

**Competing interests:** None declared for Andre Tchernof. Jean-Pierre Després has received honoraria from the following pharmaceutical companies as a consultant or lecturer: Abbott Laboratories (speaker), AstraZeneca (speaker), Fournier Pharma Inc./Solvay Pharma (speaker), GlaxoSmithKline (speaker, research funding), MSD (advisory board), Pfizer Canada Inc./Pharmacia Corporation (speaker), sanofi-aventis (speaker, advisory board, research funding) and Novartis (advisory board); furthermore, his laboratory has received research grants from some of the above companies and also from the Canadian Diabetes Association and the Canadian Institutes of Health Research.

## REFERENCES

1. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults — the evidence report. *Obes Res* 1998;6(Suppl 2):51-209S.
2. *The practical guide: identification, evaluation, and treatment of overweight and obesity in adults*. Bethesda (MD): National Institutes of Health; 2000. Publ no 00-4084. Available: [www.nhlbi.nih.gov/guidelines/obesity/prctgd\\_b.pdf](http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_b.pdf) (accessed 2006 Dec 27).
3. Dickey RA, Bartuska DG, Bray GW, et al. ACEE/ACE position statement on the prevention, diagnosis, and treatment of obesity. *Endocr Pract* 1998;4(5):297-330. Available: [www.aace.com/pub/pdf/guidelines/obesityguide.pdf](http://www.aace.com/pub/pdf/guidelines/obesityguide.pdf) (accessed 2007 Jan 20).
4. Lyznicki JM, Young DC, Riggs JA, et al. Obesity: assessment and management in primary care. *Am Fam Physician* 2001;63:2185-96.
5. Overweight, obesity, and health risk. National Task Force on the Prevention and Treatment of Obesity. *Arch Intern Med* 2000;160:898-904.
6. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* 2000;894:i-xii:1-253.
7. Douketis JD, Paradis G, Keller H, et al. Canadian guidelines for body weight classification in adults: application in clinical practice to screen for overweight and obesity and to assess disease risk. *CMAJ* 2005;172:995-8.
8. Kissebah AH, Krakower GR. Regional adiposity and morbidity. *Physiol Rev* 1994;74:761-811.
9. Krotkiewski M, Bjorntorp P, Sjostrom L, et al. Impact of obesity on metabolism in men and women. Importance of regional adipose tissue distribution. *J Clin Invest* 1983;72:1150-62.
10. Ohlsson LO, Larsson B, Svarsdudd K, et al. The influence of body fat distribution on the incidence of diabetes mellitus. 13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes* 1985;34:1055-8.
11. Despres JP, Moorjani S, Lupien PJ, et al. Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis* 1990;10:497-511.
12. Lemieux S, Prud'homme D, Tremblay A, et al. Anthropometric correlates to changes in visceral adipose tissue over 7 years in women. *Int J Obes Relat Metab Disord* 1996;20:618-24.
13. Enzi G, Gasparo M, Biondetti PR, et al. Subcutaneous and visceral fat distribution according to sex, age, and overweight, evaluated by computed tomography. *Am J Clin Nutr* 1986;44:739-46.
14. Kotani K, Tokunaga K, Fujioka S, et al. Sexual dimorphism of age-related changes in whole-body fat distribution in the obese. *Int J Obes Relat Metab Disord* 1994;18:207-12.
15. Guthrie JR, Dennerstein L, Taffe JR, et al. The menopausal transition: a 9-year prospective population-based study. The Melbourne Women's Midlife Health Project. *Climacteric* 2004;7:375-89.
16. Tremolieres FA, Pouilles JM, Ribot CA. Relative influence of age and menopause on total and regional body composition changes in postmenopausal women. *Am J Obstet Gynecol* 1996;175:1594-600.
17. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 2001;285:2486-97.
18. Grundy SM, Brewer HB Jr, Cleeman JI, et al. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation* 2004;109:433-8.
19. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation* 2005;112:2735-52.
20. Lamerche B, Tchernof A, Mauriege P, et al. Fasting insulin and apolipoprotein B levels and low-density lipoprotein particle size as risk factors for ischemic heart disease. *JAMA* 1998;279:1955-61.
21. Isomaa B, Almgren P, Tuomi T, et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care* 2001;24:683-9.
22. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in women. *JAMA* 1998;280:1843-8.
23. Janssen I, Katzmarzyk PT, Ross R. Waist circumference and not body mass index explains obesity-related health risk. *Am J Clin Nutr* 2004;79:379-84.
24. Yusuf S, Hawken S, Ounpuu S, et al. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 2005;366(9497):1640-9.
25. Despres JP, Lemieux I, Prud'homme D. Treatment of obesity: need to focus on high risk abdominally obese patients. *BMJ* 2001;322(7288):716-20.
26. Larsson B, Svarsdudd K, Welin L, et al. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *Br Med J (Clin Res Ed)* 1984;288:1401-4.
27. Lapidus L, Bengtsson C, Larsson B, et al. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow up of participants in the population study of women in Gothenburg, Sweden. *Br Med J (Clin Res Ed)* 1984;289:1257-61.
28. Li TY, Rana JS, Manson JE, et al. Obesity as compared with physical activity in predicting risk of coronary heart disease in women. *Circulation* 2006;113:499-506.
29. Rimm EB, Stampfer MJ, Giovannucci E, et al. Body size and fat distribution as predictors of coronary heart disease among middle-aged and older US men. *Am J Epidemiol* 1995;141:1117-27.
30. Folsom AR, Stevens J, Schreiner PJ, et al. Body mass index, waist/hip ratio, and coronary heart disease incidence in African Americans and whites. Atherosclerosis Risk in Communities Study Investigators. *Am J Epidemiol* 1998;148:1187-94.
31. Wang Y, Rimm EB, Stampfer MJ, et al. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *Am J Clin Nutr* 2005;81:555-63.
32. Wei M, Gaskill SP, Haffner SM, et al. Waist circumference as the best predictor of noninsulin dependent diabetes mellitus (NIDDM) compared to body mass index, waist/hip ratio and other anthropometric measurements in Mexican Americans — a 7-year prospective study. *Obes Res* 1997;5:16-23.
33. Alberti KG, Zimmet P, Shaw J. The metabolic syndrome — a new worldwide definition. *Lancet* 2005;366:1059-62.
34. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004;363:157-62.
35. Tan CE, Ma S, Wai D, et al. Can we apply the National Cholesterol Education Program Adult Treatment Panel definition of the metabolic syndrome to Asians? *Diabetes Care* 2004;27:1182-6.
36. Despres JP, Couillard C, Gagnon J, et al. Race, visceral adipose tissue, plasma lipids, and lipoprotein lipase activity in men and women: the Health, Risk Factors, Exercise Training, and Genetics (HERITAGE) family study. *Arterioscler Thromb Vasc Biol* 2000;20:1932-8.
37. Lovejoy JC, de la Bretonne JA, Klempner M, et al. Abdominal fat distribution and metabolic risk factors: effects of race. *Metabolism* 1996;45:1119-24.
38. Albu JB, Murphy L, Frager DH, et al. Visceral fat and race-dependent health risks in obese nondiabetic premenopausal women. *Diabetes* 1997;46:456-62.
39. Conway JM, Chanetsa FF, Wang P. Intraabdominal adipose tissue and anthropometric surrogates in African American women with upper- and lower-body obe-

- sity. *Am J Clin Nutr* 1997;66:1345-51.
40. Lemieux I, Pascot A, Couillard C, et al. Hypertriglyceridemic waist: a marker of the atherogenic metabolic triad (hyperinsulinemia; hyperapolipoprotein B; small, dense LDL) in men? *Circulation* 2000;102:179-84.
  41. St-Pierre J, Lemieux I, Vohl MC, et al. Contribution of abdominal obesity and hypertriglyceridemia to impaired fasting glucose and coronary artery disease. *Am J Cardiol* 2002;90:15-8.
  42. Bos G, Dekker JM, Heine RJ. Non-HDL cholesterol contributes to the "hypertriglyceridemic waist" as a cardiovascular risk factor: the Hoorn study. *Diabetes Care* 2004;27:283-4.
  43. Solati M, Ghanbarian A, Rahmani M, et al. Cardiovascular risk factors in males with hypertriglyceridemic waist (Tehran Lipid and Glucose Study). *Int J Obes Relat Metab Disord* 2004;28:706-9.
  44. Hiura Y, Acklin F, Newman J, et al. Hypertriglyceridemic waist as a screening tool for CVD risk in indigenous Australian women. *Ethn Dis* 2003;13(1):80-4.
  45. LaMonte MJ, Ainsworth BE, DuBose KD, et al. The hypertriglyceridemic waist phenotype among women. *Atherosclerosis* 2003;171(1):123-30.
  46. Kahn HS, Valdez R. Metabolic risks identified by the combination of enlarged waist and elevated triacylglycerol concentration. *Am J Clin Nutr* 2003;78:928-34.
  47. Lemieux I, Almeras N, Mauriege P, et al. Prevalence of 'hypertriglyceridemic waist' in men who participated in the Quebec Health Survey: association with atherogenic and diabetogenic metabolic risk factors. *Can J Cardiol* 2002;18:725-32.
  48. Blackburn P, Lamarche B, Couillard C, et al. Postprandial hyperlipidemia: another correlate of the "hypertriglyceridemic waist" phenotype in men. *Atherosclerosis* 2003;171:327-36.
  49. Genest J, Frohlich J, Fodor G, et al. Recommendations for the management of dyslipidemia and the prevention of cardiovascular disease: summary of the 2003 update. *CMAJ* 2003;169:921-4.

## 4. Classification of overweight and obesity in children and adolescents

Peter T. Katzmarzyk, Ian Janssen, Katherine M. Morrison, Mark S. Tremblay

The problem of obesity in Canada is increasing at a more rapid pace among children and adolescents than among adults.<sup>1,2</sup> Between 1978/79 and 2004 the prevalence of obesity increased from approximately 4% to 9% among boys and from 3% to 7% among girls 2–17 years of age.<sup>2</sup> Given that obese children and adolescents have a tendency to remain obese as adults,<sup>4</sup> these trends suggest that the epidemic of obesity among adults will accelerate in future years.

It is more challenging to recognize overweight and obesity among children and adolescents than it is among adults. In children and adolescents, body composition<sup>4,5</sup> and simple anthropometric indicators commonly used to identify obesity (e.g., BMI and waist circumference<sup>6,7</sup>) change quite dramatically with normal growth and maturation. Thus, age-specific and sex-specific thresholds are required to classify adiposity status correctly in children and adolescents.

### Body mass index

BMI is the most commonly used index of adiposity among children and adolescents, since height and weight are readily measured in the clinical setting. The BMI of children and adolescents is associated with other measures of body fatness,<sup>8,9</sup> traditional cardiovascular disease risk factors,<sup>7,10–14</sup> metabolic syndrome phenotypes,<sup>15,16</sup> social and psychological problems<sup>17,18</sup> and general health-related quality of life.<sup>19,20</sup> Furthermore, childhood and adolescent BMI is associated with the risk of adulthood obesity<sup>3,21</sup> and long-term mortality risk.<sup>22,23</sup>

The use of BMI-for-age to evaluate overweight and obesity in children and adolescents has been recommended by several expert committees.<sup>24–30</sup>

Two main approaches have been used to classify children and adolescents using BMI: a distributional approach (percentiles, norm-referenced), and a linking of childhood BMI cutoff points with adult overweight and obesity thresholds (BMI of 25 kg/m<sup>2</sup> and 30 kg/m<sup>2</sup>, quasi-criterion referenced). Although BMI is related to health outcomes in childhood and adolescence, there is a paucity of data available on the sensitivity and specificity of specific BMI thresholds across a range of ages and maturational status.

Classification of overweight and obesity in children and adolescents is commonly based on a distributional approach. For example, the 85th and 95th percentiles of BMI from nationally representative samples have typically been used to identify children as “overweight” and “obese,” respectively, or “at risk of overweight” and “overweight,” respectively. The

US Centers for Disease Control and Prevention (CDC) has developed BMI-for-age growth charts using data from several American sources collected from 1963 to 1994 (Fig. 5 and Fig. 6).<sup>31</sup> It should be noted that, given recent temporal increases in BMI, more than 15% of youth are now overweight and 5% are obese.

Although the CDC thresholds are widely used for clinical practice in the United States, no published Canadian BMI growth charts are currently available. However, the ethnic make-up of the Canadian population is arguably quite similar to that of the United States, since 80.5% and 86.6% of the US and Canadian populations, respectively, are white (i.e., not part of a visible minority group).<sup>32,33</sup> Furthermore, many Canadian clinicians currently use the CDC growth charts for stature and body mass. Thus, for clinical use, we recommend that the CDC 85th and 95th percentiles be used to classify children and adolescents as overweight ( $\geq$  85th to  $<$  95th percentile) and obese ( $\geq$  95th percentile). Note: although the CDC uses the terms “at risk of overweight” and “overweight,” the terms preferred by the International Obesity Task Force (IOTF) — “overweight” and “obese” — are used throughout this document.

The second approach to identifying overweight and obese children and adolescents is the method of Cole and colleagues.<sup>6</sup> In it, the percentile levels corresponding to a BMI of 25 kg/m<sup>2</sup> (overweight) and 30 kg/m<sup>2</sup> (obese) at age 18 were identified and projected backward into childhood using a large international sample of children and youth. The development of these thresholds was endorsed by an expert committee convened by the IOTF.<sup>24</sup> These thresholds are often referred to as the IOTF cutoff points (Table 8). The IOTF cutoff points are anchored to the adult thresholds for overweight and obesity and are, therefore, a reflection of the relation between BMI and health risks in adulthood rather than the relation between BMI and health in childhood per se.

Recently, the World Health Organization (WHO) released new international child growth “standards” that are based on how children should grow<sup>34</sup> in contrast with data that illustrate how children grow when exposed to different environments. The new WHO growth curves were generated from the WHO Multicentre Growth Reference Study,<sup>35</sup> which gathered data on children 5 years of age and younger from 6 countries (Brazil, Ghana, India, Norway, Oman and the United States). All children were exposed to healthy living conditions (e.g., breast-fed, transition to good diet, nonsmoking mother, adequate access to health care and immunizations). The application of these new cutoff points to populations will result in dif-

**2 to 20 years: Boys**  
**Body mass index-for-age percentiles**

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_

Date	Age	Weight	Stature	BMI*	Comments

**\*To Calculate BMI:** Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000  
 or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703

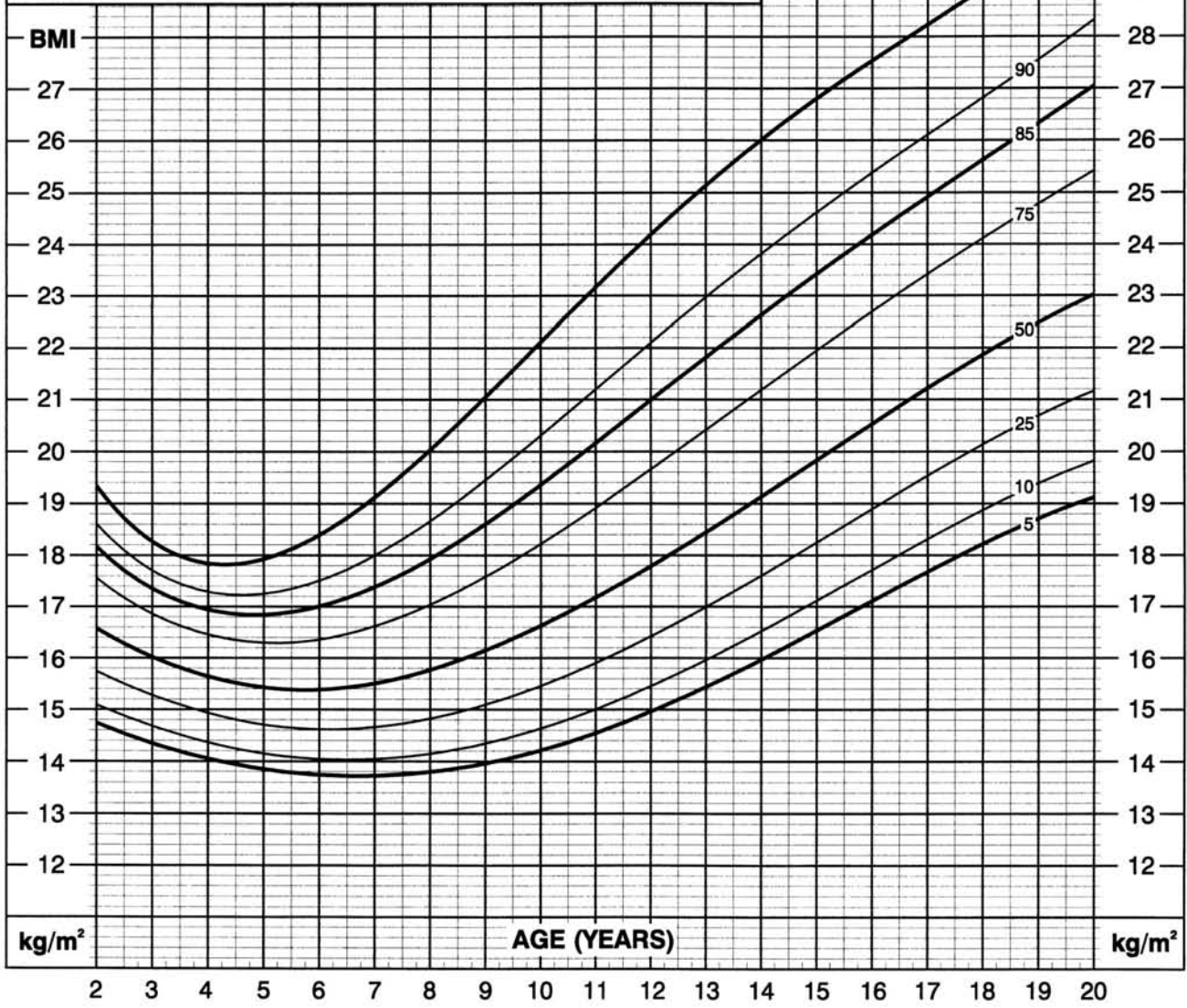


Fig. 5: Growth chart: BMI-for-age percentiles for boys aged 2–20 years. Source: US Centers for Disease Control and Prevention, National Center for Health Statistics ([www.cdc.gov/growthcharts](http://www.cdc.gov/growthcharts)), 2000 May 30.

**2 to 20 years: Girls**  
**Body mass index-for-age percentiles**

NAME \_\_\_\_\_

RECORD # \_\_\_\_\_

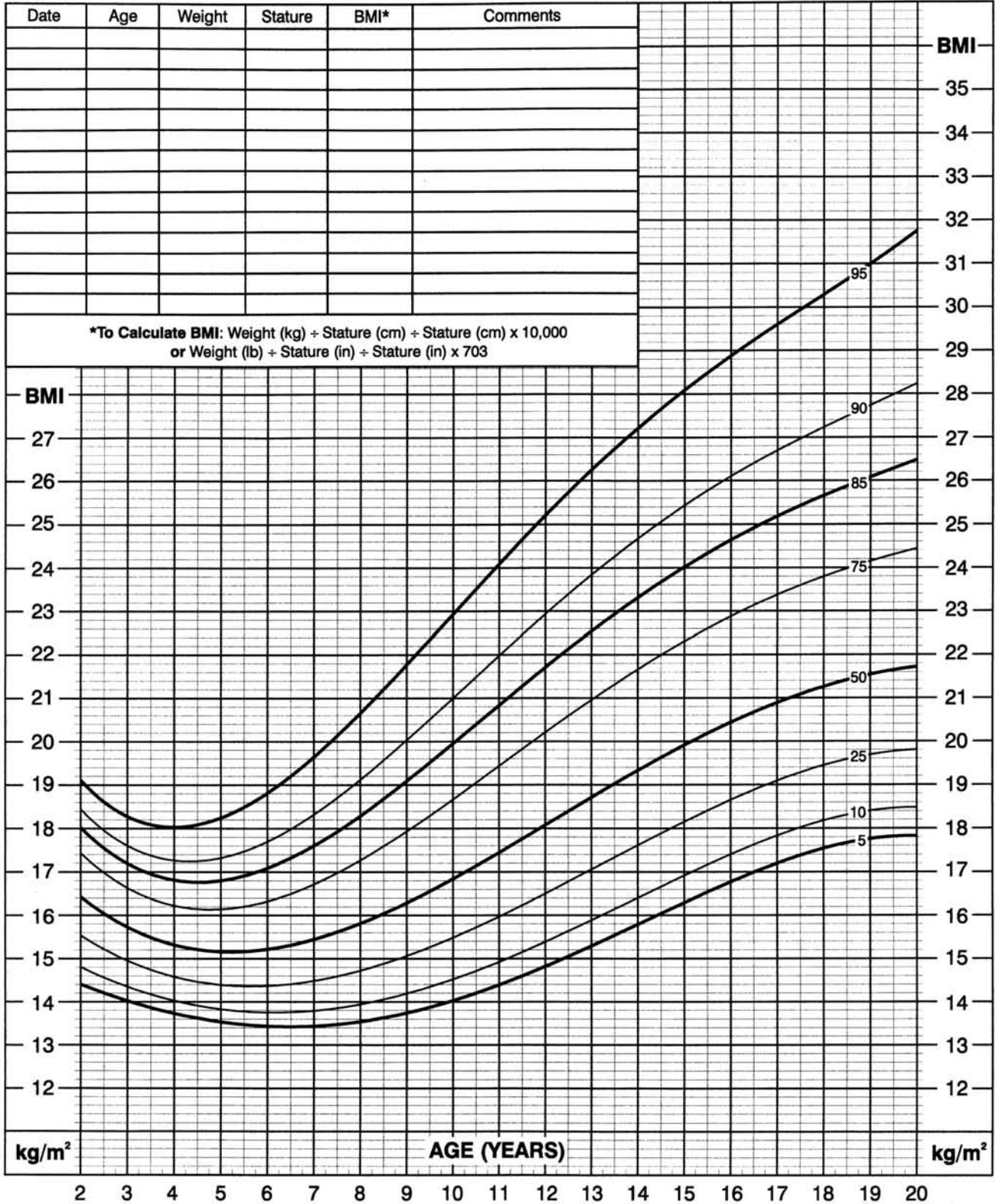


Fig. 6: Growth chart: BMI-for-age percentiles for girls aged 2–20 years. Source: US Centers for Disease Control and Prevention, National Center for Health Statistics ([www.cdc.gov/growthcharts](http://www.cdc.gov/growthcharts)), 2000 May 30.

ferent prevalences of overweight and obesity, which are believed to result in better predictions of future health outcomes and burden. Further research in this area is required, especially on the application of criterion-based growth curves through the childhood and adolescent years. The clinical utility of the WHO growth curves should be studied as they become integrated into practice over the coming years.

The use of different approaches to classify obesity over time (e.g., norm-references, data collection methods) makes the study of temporal trends in obesity difficult and leads to potential problems of misinterpretation. Further, the use of

**Table 8:** International Obesity Task Force cutoff points for the identification of overweight and obesity in children and adolescents using body mass index (kg/m<sup>2</sup>)

Age, yr	Overweight		Obesity	
	Males	Females	Males	Females
2	18.41	18.02	20.09	19.81
2.5	18.13	17.76	19.80	19.55
3	17.89	17.56	19.57	19.36
3.5	17.69	17.40	19.39	19.23
4	17.55	17.28	19.29	19.15
4.5	17.47	17.19	19.26	19.12
5	17.42	17.15	19.30	19.17
5.5	17.45	17.20	19.47	19.34
6	17.55	17.34	19.78	19.65
6.5	17.71	17.53	20.23	20.08
7	17.92	17.75	20.63	20.51
7.5	18.16	18.03	21.09	21.01
8	18.44	18.35	21.60	21.57
8.5	18.76	18.69	22.17	22.18
9	19.10	19.07	22.77	22.81
9.5	19.46	19.45	23.39	23.46
10	19.84	19.86	24.00	24.11
10.5	20.20	20.29	24.57	24.77
11	20.55	20.74	25.10	25.42
11.5	20.89	21.20	25.58	26.05
12	21.22	21.68	26.02	26.67
12.5	21.56	22.14	26.43	27.24
13	21.91	22.58	26.84	27.76
13.5	22.27	22.98	27.25	28.20
14	22.62	23.34	27.63	28.57
14.5	22.96	23.66	27.98	28.87
15	23.29	23.94	28.30	29.11
15.5	23.60	24.17	28.60	29.29
16	23.90	24.37	28.88	29.43
16.5	24.19	24.54	29.14	29.56
17	24.46	24.70	29.41	29.69
17.5	24.73	24.85	29.70	29.84
18	25	25	30	30

Source: Adapted from Cole et al.<sup>6</sup>

different BMI thresholds in different countries makes cross-national comparisons problematic. For example, the CDC and IOTF cutoff points are not equivalent. In general, the “overweight” thresholds are similar, but the IOTF’s “obesity” cutoff point is higher than that of the CDC for most ages. This results in consistently higher obesity prevalence based on the CDC cutoff points compared with the IOTF cutoff points, with these differences being particularly large in the younger age ranges.<sup>36</sup> For the surveillance of overweight and obesity in the research setting, we recommend that the IOTF thresholds be used to facilitate international comparisons. Where possible, prevalences should be presented using both IOTF and CDC thresholds to allow greater comparability.

## Waist circumference

Waist circumference is the best anthropometric predictor of visceral adiposity in adults,<sup>37,38</sup> and a large waist circumference is associated with increased risk of chronic disease,<sup>39–41</sup> premature death<sup>42,43</sup> and high health care costs.<sup>44</sup>

Among children and adolescents, waist circumference is a good predictor of other measures of adiposity<sup>45–48</sup> and risk level for heart disease.<sup>7,49–53</sup> Reference data for waist circumference in children and adolescents have been developed for several countries, including the United States<sup>54</sup> and Canada.<sup>55</sup> Although waist circumference is significantly related to risk factor levels in children, further research is required to determine its clinical utility and association with health risks independent of BMI.<sup>56</sup> At this time, the lack of appropriate national reference data for waist circumference across childhood and adolescence and the lack of standardization of measurement protocols preclude recommending its widespread use in clinical practice. An important area of future research is to develop health-related waist circumference measurement protocols and classification criteria for children and adolescents within the Canadian context.

## Measurement of body mass index

BMI is a composite measure of weight and height (weight in kilograms divided by height in metres squared).<sup>57</sup> Alternatively, BMI can be calculated in pounds and inches ([weight in pounds divided by height in inches squared] × 703). Height should be measured to the nearest centimetre using a stadiometer. The patient should look straight ahead, stand as tall as possible and take a deep breath while the measurement is taken. Weight should be measured to the nearest 0.1 kg with an accurate, well-maintained physician’s scale. The patient should be weighed in light clothing, without footwear. Accurate measurement of height and weight is particularly important in children, as small errors can lead to large percentile shifts in BMI.

## Other considerations and limitations

The measurement and interpretation of BMI in the general health assessment of children and youth is complicated and must be placed in context. Factors that must be taken into

consideration when assessing obesity include stage of biologic maturation, the presence of comorbidities, ethnic origin, level of habitual physical activity, somatotype and frame size.

The reference data for BMI and waist circumference for children and adolescents that are currently available are based mainly on distributional percentile cutoff points rather than on relations to health outcomes. The IOTF BMI cutoff points are linked with the adult overweight and obese thresholds of 25 kg/m<sup>2</sup> and 30 kg/m<sup>2</sup>. Thus, the pediatric cutoff points are indirectly tied to adult health outcomes, but not health outcomes in childhood and adolescence per se. Limited attempts have been made to develop waist circumference thresholds in children and adolescents based on relations to risk factors.<sup>51,58</sup> However, a priority for future research is the investigation of the clinical utility of existing waist circumference percentiles and the development of health-related BMI and waist circumference thresholds.

The currently recommended strategies to identify overweight and obesity in children and adolescents employ age-specific and sex-specific values. However, incorporation of indicators of biologic maturation may improve the identification of obesity-related health risk. BMI values differ across pubertal stages of development among adolescents of the same age.<sup>59</sup> Although incorporating an index of maturation into the assessment of obesity may be beneficial, a recent study has shown no improvement in the identification of obese adolescents.<sup>60</sup> Thus, more research is required to evaluate the utility of including simple indices of biologic maturation, such as those based on secondary characteristics of pubic hair and breast development, as developed by Tanner,<sup>61</sup> or more recent research using simple anthropometric measures<sup>62</sup> in the assessment of obesity. Given that BMI is an index of weight and height, it cannot distinguish between lean and fat tissues. Thus, clinical judgment must always be used in identifying children and adolescents who are truly over“fat” as opposed to over“weight.” Along the same lines, physical activity levels are an important consideration. It has recently been shown that physically fit children have lower levels of central adiposity for a given BMI.<sup>63</sup>

Among adults, obesity-related health risks seem to accumulate at lower BMI levels among Asian people than among people in other ethnic groups.<sup>64-66</sup> Thus, a BMI cutoff point for obesity of 30 kg/m<sup>2</sup> among adult Asian people may be too high to capture those at elevated health risk, and it should be lowered.<sup>67</sup> It is currently unclear what role ethnic origin plays in the identification of obesity in children and adolescents. This represents an important avenue of future research.

Future research should consider the potential harmful effects of screening for overweight and obesity among children and adolescents and the potential beneficial effects of focusing on promoting positive behaviours. A recent review by the US Preventive Services Task Force found no direct evidence of harm associated with overweight screening in children; however, the lack of screening studies meant inferences had to depend on other sources.<sup>68</sup>

The available BMI reference data are based on representative US populations. An effort should be made to collect Canadian normative or criterion-referenced data for the deri-

vation of growth curves for the purpose of clinical assessment of obesity-related health risk.

## Recommendations

1. We recommend measuring BMI in all children and adolescents (aged 2 years and older). We recommend using the growth charts of the US Centers for Disease Control and Prevention for BMI to screen children and adolescents for overweight ( $\geq 85$ th to  $< 95$ th percentile) and obesity ( $\geq 95$ th percentile) [*grade A, level 3*].<sup>30</sup>
2. Normative data from representative samples of the Canadian population should be collected to allow the development of Canadian-specific growth curves for BMI and waist circumference. Research efforts should be directed at developing reference data that are based on health-related criteria or outcomes rather than being merely representative of the population [*grade C, level 4*].
3. Future research should be directed at determining the clinical utility of waist circumference in the identification of health risk among children and youth, independently or in combination with BMI [*grade C, level 4*].
4. For population surveillance of overweight and obesity in children, we recommend that the BMI thresholds of the International Obesity Task Force be used to classify children and youth as overweight and obese. Where possible, we recommend that prevalence be presented using both the US Centers for Disease Control and Prevention thresholds and the International Obesity Task Force cutoff points to facilitate international comparisons [*grade C, level 4*].
5. Future research should be directed at understanding the impact of gender, biologic maturation, nutrition, physical activity levels, sociocultural milieu, built environments, ethnic background, biological factors, psychological factors and genetics on obesity and obesity-related health risk in the context of the Canadian population [*grade C, level 4*].

From the School of Kinesiology and Health Studies and the Department of Community Health and Epidemiology, Queen's University, Kingston, Ont. (Katzmarzyk, Janssen); the Department of Pediatrics, McMaster University, Hamilton, Ont. (Morrison); and the College of Kinesiology, University of Saskatchewan, and Statistics Canada, Ottawa, Ont. (Tremblay)

**Competing interests:** None declared.

## REFERENCES

1. Tremblay MS, Katzmarzyk PT, Willms JD. Temporal trends in overweight and obesity in Canada, 1981-1996. *Int J Obes Relat Metab Disord* 2002;26:538-43.
2. Shields M. Measured obesity: overweight Canadian children and adolescents. In: *Nutrition: findings from the Canadian Community Health Survey*; issue 1; 2005 (cat no 82-620-MWE2005001). Available: [www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf](http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf) (accessed 2007 Jan 9).
3. Whitaker RC, Wright JA, Pepe MS, et al. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med* 1997;337:869-73.
4. Guo SS, Chumlea WC, Roche AF, et al. Age- and maturity-related changes in body composition during adolescence into adulthood: the Fels Longitudinal Study. *Int J Obes Relat Metab Disord* 1997;21:1167-75.
5. Malina RM, Bouchard C, Bar-Or O. *Growth, maturation and physical activity*. 2nd ed. Champaign (IL): Human Kinetics; 2004.
6. Cole TJ, Bellizzi MC, Flegal KM, et al. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000;320:1-6.
7. Freedman DS, Serdula MK, Srinivasan SR, et al. Relation of circumferences and skinfold thicknesses to lipid and insulin concentrations in children and adolescents: the Bogalusa Heart Study. *Am J Clin Nutr* 1999;69:308-17.



8. Deurenberg P, Weststrate JA, Seidell JC. Body mass index as a measure of body fatness: age- and sex-specific prediction formulas. *Br J Nutr* 1991;65:105-14.
9. Roche AF, Sievogel RM, Chumlea WC, et al. Grading body fatness from limited anthropometric data. *Am J Clin Nutr* 1981;34:2831-8.
10. Chu NF, Rimm EB, Wang DJ, et al. Clustering of cardiovascular disease risk factors among obese schoolchildren: the Taipei Children Heart Study. *Am J Clin Nutr* 1998;67:1141-6.
11. Morrison JA, Sprecher DL, Barton BA, et al. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr* 1999;135:458-64.
12. Morrison JA, Barton BA, Biro FM, et al. Overweight, fat patterning and cardiovascular disease risk factors in black and white boys. *J Pediatr* 1999;135:451-7.
13. Katzmarzyk PT, Tremblay A, Perusse L, et al. The utility of the international child and adolescent overweight guidelines for predicting coronary heart disease risk factors. *J Clin Epidemiol* 2003;56:456-62.
14. Paradis G, Lambert M, O'Loughlin J, et al. Blood pressure and adiposity in children and adolescents. *Circulation* 2004;110:1832-8.
15. Cook S, Weitzman M, Auinger P, et al. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. *Arch Pediatr Adolesc Med* 2003;157:821-7.
16. de Ferranti SD, Gauvreau K, Ludwig DS, et al. Prevalence of the metabolic syndrome in American adolescents: findings from the Third National Health and Nutrition Examination Survey. *Circulation* 2004;110:2494-7.
17. Goldfield A, Chrisler JC. Body stereotyping and stigmatization of obese persons by first graders. *Percept Mot Skills* 1995;81:909-10.
18. Janssen I, Craig WM, Boyce WF, et al. Associations between overweight and obesity with bullying behaviors in school-aged children. *Pediatrics* 2004;113:1187-94.
19. Schwimmer JB, Burwinkle TM, Jarni JW. Health-related quality of life of severely obese children and adolescents. *JAMA* 2003;289:1813-9.
20. Williams J, Wake M, Hesketh K, et al. Health-related quality of life of overweight and obese children. *JAMA* 2005;293:70-6.
21. Guo SS, Huang C, Maynard LM, et al. Body mass index during childhood, adolescence and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. *Int J Obes Relat Metab Disord* 2000;24:1628-35.
22. Engeland A, Bjorge T, Sogaard AJ, et al. Body mass index in adolescence in relation to total mortality: 32-year follow-up of 227,000 Norwegian boys and girls. *Am J Epidemiol* 2003;157:1517-23.
23. Engeland A, Bjorge T, Tverdal A, et al. Obesity in adolescence and adulthood and the risk of adult mortality. *Epidemiology* 2004;15:79-85.
24. Bellizzi MC, Dietz WH. Workshop on childhood obesity: summary of the discussion. *Am J Clin Nutr* 1999;70(1):173S-175S.
25. Himes JH, Dietz WH. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. The Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services. *Am J Clin Nutr* 1994;59:307-16.
26. International Obesity Task Force. Assessment of obesity: Which child is fat? *Obes Rev* 2004;5(Suppl):10-5.
27. Barlow SE, Dietz WH. Obesity evaluation and treatment: Expert Committee recommendations. The Maternal and Child Health Bureau, Health Resources and Services Administration and the Department of Health and Human Services. *Pediatrics* 1998;102:E29.
28. Krebs NF, Jacobson MS. Prevention of pediatric overweight and obesity. *Pediatrics* 2003;112:424-30.
29. Australian National Health & Medical Research Council. *Clinical practice guidelines for the management of overweight and obesity in children and adolescents*. Canberra: Australian National Health and Medical Research Council; 2003. Available: [www.health.gov.au/internet/wcms/Publishing.nsf/Content/obesityguidelines-guidelines-children.htm/\\$FILE/children.pdf](http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/obesityguidelines-guidelines-children.htm/$FILE/children.pdf) (accessed 2007 Jan 24).
30. Dietitians of Canada, Canadian Paediatric Society, College of Family Physicians of Canada, et al. The use of growth charts for assessing and monitoring growth in Canadian infants and children. *Can J Diet Pract Res* 2004;65:22-32.
31. Kuczumarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat* 11 2002;(246):1-190.
32. *Profile of citizenship, immigration, birthplace, generation status, ethnic origin, visible minorities and Aboriginal peoples, for Canada, provinces, territories, census divisions, census subdivisions, and dissemination areas. 2001 Census*. Ottawa: Statistics Canada; 2003. Cat no 95F0489XCB2001002.
33. *Annual estimates of the population by race alone and Hispanic or Latino origin for the United States and States: July 1, 2003*. Washington (DC): US Census Bureau; 2004. Available: [www.census.gov/popest/states/asrh/tables/SC-EST2003-04.pdf](http://www.census.gov/popest/states/asrh/tables/SC-EST2003-04.pdf) (accessed 2007 Jan 24).
34. *WHO child growth standards: length/height-for-age, weight-for-age, weight-for-height and body mass index-for-age: methods and development*. Geneva: World Health Organization, Department of Nutrition for Health and Development; 2006.
35. De Onis M, Garza C, Victora CG, et al. The WHO Multicentre Growth Reference Study (MGRS): rationale, planning, and implementation. *Food Nutr Bull* 2004;25(Suppl 1):S3-S84.
36. Flegal KM, Ogden CL, Wei R, et al. Prevalence of overweight in US children: comparison of US growth charts from the Centers for Disease Control and Prevention with other reference values for body mass index. *Am J Clin Nutr* 2001;73:1086-93.
37. Pouliot MC, Despres JP, Lemieux S, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *Am J Cardiol* 1994;73:460-8.
38. Rankinen T, Kim SY, Perusse L, et al. The prediction of abdominal visceral fat level from body composition and anthropometry: ROC analysis. *Int J Obes Relat Metab Disord* 1999;23:801-9.
39. Chan JM, Rimm EB, Colditz GA, et al. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care* 1994;17:961-9.
40. Folsom AR, Kushi LH, Anderson KE, et al. Associations of general and abdominal obesity with multiple health outcomes in older women: the Iowa Women's Health Study. *Arch Intern Med* 2000;160:2117-28.
41. Rexrode KM, Buring JE, Manson JE. Abdominal and total adiposity and risk of coronary heart disease in men. *Int J Obes Relat Metab Disord* 2001;25:1047-56.
42. Bigaard J, Tjonneland A, Thomsen BL, et al. Waist circumference, BMI, smoking, and mortality in middle-aged men and women. *Obes Res* 2003;11:895-903.
43. Katzmarzyk PT, Craig CL, Bouchard C. Adiposity, adipose tissue distribution and mortality rates in the Canada Fitness Survey follow-up study. *Int J Obes Relat Metab Disord* 2002;26:1054-9.
44. Cormier MA, Tate CW, Grunwald GK, et al. Relationship between waist circumference, body mass index, and medical care costs. *Obes Res* 2002;10:1167-72.
45. Daniels SR, Khoury PR, Morrison JA. Utility of different measures of body fat distribution in children and adolescents. *Am J Epidemiol* 2000;152:1179-84.
46. Goran MI, Gower BA, Treuth M, et al. Prediction of intra-abdominal and subcutaneous abdominal adipose tissue in healthy pre-pubertal children. *Int J Obes Relat Metab Disord* 1998;22:549-58.
47. Himes JH. Agreement among anthropometric indicators identifying the fattest adolescents. *Int J Obes Relat Metab Disord* 1999;23:518-21.
48. Sarria A, Moreno LA, Garcia-Llora LA, et al. Body mass index, triceps skinfold and waist circumference in screening for adiposity in male children and adolescents. *Acta Paediatr* 2001;90:387-92.
49. Flodmark CE, Sveger T, Nilsson-Ehle P. Waist measurement correlates to a potentially atherogenic lipoprotein profile in obese 12-14-year-old children. *Acta Paediatr* 1994;83:941-5.
50. Maffei C, Pietrobelli A, Grezzani A, et al. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes Res* 2001;9:179-87.
51. Moreno LA, Pineda I, Rodriguez G, et al. Waist circumference for the screening of the metabolic syndrome in children. *Acta Paediatr* 2002;91:1307-12.
52. Rodriguez G, Moreno LA, Blay MG, et al. Body composition in adolescents: measurements and metabolic aspects. *Int J Obes Relat Metab Disord* 2004;28:S54-8.
53. Savva SC, Tornaritis M, Sava ME, et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *Int J Obes Relat Metab Disord* 2000;24:1453-8.
54. Fernandez JR, Redden DT, Pietrobelli A, et al. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. *J Pediatr* 2004;145:439-44.
55. Katzmarzyk PT. Waist circumference percentiles for Canadian youth 11-18y of age. *Eur J Clin Nutr* 2004;58(7):1011-5.
56. Janssen I, Katzmarzyk PT, Srinivasan SR, et al. Combined influence of body mass index and waist circumference on coronary artery disease risk factors among children and adolescents. *Pediatrics* 2005;115:1623-30.
57. *Canadian guidelines for body weight classification in adults*. Ottawa: Health Canada; 2003. Cat no H49-179/2003E. Available: [www.hc-sc.gc.ca/fn-an/alt\\_formats/hpfb-dgpsa/pdf/nutrition/weight\\_book-livres\\_des\\_poids\\_e.pdf](http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/weight_book-livres_des_poids_e.pdf) (accessed 2007 Jan 24).
58. Katzmarzyk PT, Srinivasan SR, Chen W, et al. Body mass index, waist circumference, and clustering of cardiovascular disease risk factors in a biracial sample of children and adolescents. *Pediatrics* 2004;114:e198-205.
59. Bini V, Celi F, Berlioli MG, et al. Body mass index in children and adolescents according to age and pubertal stage. *Eur J Clin Nutr* 2000;54:214-8.
60. Taylor RW, Falorni A, Jones IE, et al. Identifying adolescents with high percentage body fat: a comparison of BMI cutoffs using age and stage of pubertal development compared with BMI cutoffs using age alone. *Eur J Clin Nutr* 2003;57:764-9.
61. Tanner JM. *Growth at adolescence*. 2nd ed. Oxford: Blackwell Scientific; 1962.
62. Mirwald RL, Baxter-Jones AD, Bailey DA, et al. An assessment of maturity from anthropometric measurements. *Med Sci Sports Exerc* 2002;34:689-94.
63. Nassif GP, Psarra G, Sidossis LS. Central and total adiposity are lower in overweight and obese children with high cardiorespiratory fitness. *Eur J Clin Nutr* 2005;59:137-41.
64. Kim Y, Suh YK, Choi H. BMI and metabolic disorders in South Korean adults: 1998 Korea National Health and Nutrition Survey. *Obes Res* 2004;12:445-53.
65. Li G, Chen X, Jang Y, et al. Obesity, coronary heart disease risk factors and diabetes in Chinese: an approach to the criteria of obesity in the Chinese population. *Obes Res* 2002;3:167-72.
66. Moon OR, Kim NS, Jang SM, et al. The relationship between body mass index and the prevalence of obesity-related diseases based on the 1995 National Health Interview Survey in Korea. *Obes Res* 2002;10:191-6.
67. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004;363:157-63.
68. Whitlock EP, Williams SB, Gold R, et al. Screening and interventions for childhood overweight: a summary of evidence for the US Preventive Services Task Force. *Pediatrics* 2005;116:e125-44.

## 5. Assessment of readiness to change

Michael Vallis

Although behavioural change is essential for achieving and maintaining weight loss,<sup>1,2</sup> it has proven difficult to sustain for a number of reasons.<sup>3</sup> Motivational readiness to change is an important aspect of the change process. Readiness is not general; rather, it is specific to individual behaviours<sup>4</sup> and can be influenced by a host of temporal, environmental and social factors. Assessing readiness to change and supporting an individual's motivation to sustain healthy behaviours over time is an important component in the treatment of obesity.

Motivational readiness involves intention (or stage), self-efficacy, decisional balance, barriers and temptations, and processes of change.<sup>5</sup> One way to assess stage is to assign distinct categories, contextualized by intention to change within a specific time frame (either immediately or within 6 months).<sup>6-8</sup> The first 3 categories are referred to as the preaction stages.

- *Precontemplation*: At this stage, the person is not even thinking about change and does not intend to change in the foreseeable future. Interventions should be targeted at getting him or her to consider change, in a nondefensive manner.
- *Contemplation*: At this stage, the person is not prepared to change behaviour immediately but is considering change within the next 6 months. Interventions should be targeted at helping him or her make a commitment and develop a realistic plan to change.
- *Preparation*: At this stage, the person is actively considering changing behaviour within the next month. Although the person has not actually changed behaviour, he or she is taking steps toward change. Interventions should be targeted at providing support and encouragement.
- *Action*: At this stage, the person has begun to change his or her behaviour, but the changes are not well established (i.e., within 6 months of change). Interventions should focus on reinforcing the change and planning for setbacks.

- *Maintenance*: At this stage, the person has changed his or her behaviour and has maintained that change for 6 months or longer. Interventions should be targeted at supporting self-efficacy and preventing relapse.

Self-efficacy refers to confidence in one's ability to perform a specific behaviour (or behaviours) at a specific time. Self-efficacy can be assessed by obtaining a subjective numerical rating on a visual analogue scale (Fig. 7) or a 7-point Likert scale. Self-efficacy increases as a person moves from the preaction to action stages.

Decisional balance refers to the pros and cons of performing the healthy behaviour. Scales exist that provide numerical scores for pros, cons and the difference, but clinically a list is obtained and discussed. Pros are low in preaction stages and increase in action stages, whereas the cons of the healthy behaviour are higher in preaction than in action stages.

Finally, specific barriers and temptations are assessed by direct enquiry into the person's experience. Barriers and temptations are better managed (using problem-solving techniques) as he or she moves to action.

A number of behavioural and experiential processes that are seen to help mediate stage are also identified in the model. There are no predictions about the relation between specific processes and stage, other than an overall increase in the use of processes as a person moves from preaction to action stages. The validity of the model of stages of change, or readiness to change, has been tested by examining the relation between stages and such factors as healthy diet, activity level and BMI. Studies have consistently found that the predictions, as stated above, have been confirmed.

### Assessing intention to change

A number of specific studies have supported the validity of the stages of change by demonstrating an association between stage of healthy eating and an independent measure of healthy

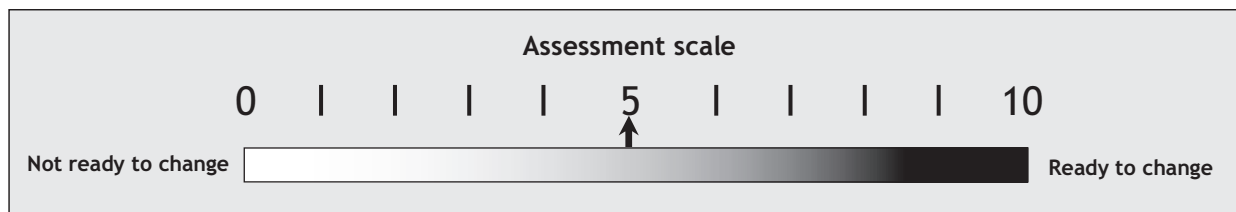


Fig. 7: Example of a visual analogue scale used to assess a person's intent to change behaviour.

behaviour, such as percentage of fat in diet or number of fruit and vegetable servings eaten daily.<sup>9-13</sup> Large study populations have been assessed and categorized using readiness-to-change algorithms. The stages are then compared in terms of measures of healthy eating. A similar method has been applied to physical activity, with similar results.<sup>4,14-22</sup> Overall, individuals who are in the action and maintenance stages of change exhibit healthier behaviours than those in the preaction stages.

Power, design and statistical analyses in these noninterventional studies are more than adequate. However, positive results are tempered by several factors. First, outcomes such as healthy eating or increased activity level do not vary in a stepwise fashion across stages, but more as a function of preaction versus action stages; that is, healthier behaviours are demonstrated by individuals in the action stages. Second, most of the data are limited to cross-sectional study designs. Longitudinal designs are lacking (although see McCann and colleagues<sup>9</sup>). Third, studies tend to rely on self-reported measures of the healthy behaviour, such as a food frequency questionnaire or a self-report exercise scale (although see Dannecker and colleagues<sup>21</sup> and Krummel and colleagues<sup>23</sup>).

Stage can be assessed by using a behavioural algorithm, which results in categorizing into the stages listed above.<sup>7,11</sup> Stage can also be validly assessed by obtaining a numerical rating (e.g., 1 to 10) of intention to perform a specific behaviour.<sup>24</sup> Using the behavioural algorithm involves presentation of a specific behavioural outcome (e.g., performing exercise at least 3 times a week for at least 30 minutes at a level that results in heavy breathing or sweating) for which respondents choose one of the following responses:

- No, and I do not intend to in the next 6 months (precontemplation stage)
- No, but I intend to in the next 6 month (contemplation stage)
- No, but I intend to in the next 30 days (preparation stage)
- Yes, I have been for less than 6 months (action stage)
- Yes, I have been for more than 6 months (maintenance stage).

There is also sufficient evidence to support clinical assessment of self-efficacy, decisional balance, and barriers and temptations when promoting weight loss behaviours.<sup>4,5,25-31</sup> These studies show that increased weight loss is associated with higher levels of self-efficacy, greater “pros” for weight loss and lower levels of barriers or temptations. As with the stages results, these studies are cross-sectional and rely primarily on self-reporting and correlational designs.

Once the health care professional has assessed readiness, decisional balance, self-efficacy, and barriers and temptations, the results are used to establish a plan for success at weight management. The health care professional should inform individuals that readiness to change is modifiable and can be increased by assessing and manipulating the other constructs of the model. For instance, the health care professional should ask the person to list the reasons why he or she wants to change and the reasons why change is likely to be hard. A frank and open discussion about the disadvantages of change can lead to a realistic plan and to a commitment to change. Similarly, by identifying barriers and temptations,

the health care professional can engage in standard problem-solving to reduce the person’s chances of failure. Finally, open discussion of increasing confidence can be helpful.

The health care professional is better able to help when he or she adopts what has come to be known as the motivational interviewing approach.<sup>32</sup> Motivational interviewing is guided by the following ingredients (summarized by the acronym FRAMES — feedback, responsibility, advice, menu, empathy, self-efficacy):

- Feedback on personal risk or impairment. The health care professional should spend time helping the person understand the risks he or she currently faces.
- Emphasis on personal responsibility for change. Sensitive discussion of the importance of the person taking responsibility for his or her behaviour, with the support of the health care professional, is reinforced over time.
- Clear advice to change.
- A menu of change options. Listing alternatives, including diet, activity, behaviour modification and social supports, is recommended.
- Therapist empathy. It is important for the health care professional to communicate in such a way that the person feels understood and supported.
- Facilitation of client self-efficacy or optimism. Building confidence and providing hope and encouragement can be very helpful in maintaining motivation for change.

## Conclusions

Health care professionals should assess a person’s readiness or intention to change a specific behaviour before that person implements a healthy lifestyle plan. This assessment can be made by obtaining a rating of level of intent (e.g., on a 10-point visual analogue scale or by determining stage of change based on the 5-step algorithm presented above).

Training opportunities to provide continuing education in assessing readiness should be offered to nonbehavioural health care professionals.

In addition to overall intention to change, a person should be asked to judge his or her current level of confidence in performing the specific behaviour in question; to identify, before beginning a behaviour change intervention, the specific personal advantages and disadvantages of change as well as the advantages and disadvantages of staying the same; and to identify specific, individual situations and circumstances that serve as either barriers to performing the healthy behaviour or as temptations to not perform the healthy behaviour.

Once this assessment is complete, the information can be used to discuss change strategies. Educating the person to the fact that these factors can be altered and that altering them will improve his or her willingness to change is recommended.

## Recommendations

1. We suggest that health care professionals assess readiness and barriers to change before an individual implements a healthy lifestyle plan for weight control or management [*grade B, level 3*].

2. Research should be undertaken to further validate models and tools for assessing intention to change as well as the effectiveness of interventions to improve readiness to change [*grade C, level 4*].
3. Continuing education activities that provide physicians and health professionals with the skills they need to counsel people confidently in healthy weight management should be developed [*grade C, level 4*].

From Dalhousie University, Halifax, NS

**Competing interests:** None declared.

## REFERENCES

1. McTigue KM, Harris R, Hemphill B, et al. Screening and interventions for obesity in adults: summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med* 2003;139(11):933-49.
2. Leiter LA, Abbott D, Campbell NR, et al. Lifestyle modifications to prevent and control hypertension. 2. Recommendations on obesity and weight loss. Canadian Hypertension Society, Canadian Coalition for High Blood Pressure Prevention and Control, Laboratory Centre for Disease Control at Health Canada, Heart and Stroke Foundation of Canada. *CMAJ* 1999;160(9 Suppl):S7-12.
3. Vallis TM. Psychological traps in diabetes management: To be forewarned is to be forearmed. *Can J Diabetes Care* 2001;25(Suppl 2):31-8.
4. Boudreaux ED, Wood KB, Mehan D, et al. Congruence of readiness to change, self-efficacy, and decisional balance for physical activity and dietary fat reduction. *Am J Health Promot* 2003;17:329-36.
5. Prochaska J, Norcross JC, DiClemente CC. *Changing for good: a revolutionary six-stage program for overcoming bad habits and moving your life forward positively*. New York: Avon Books; 1994.
6. Glanz K, Patterson RE, Kristal AR, et al. Stages of change in adopting healthy diets: fat, fiber, and correlates of nutrient intake. *Health Educ Q* 1994;21:499-519.
7. Greene GW, Rossi SR, Reed GR, et al. Stages of change for reducing dietary fat to 30% of energy or less. *J Am Diet Assoc* 1994;94:1105-10.
8. Ruggiero L, Prochaska JO. Readiness for change: application of the transtheoretical model to diabetes. *Diabetes Spectrum* 1993;6:21-60.
9. McCann BS, Bowjerg VE, Curry SJ, et al. Predicting participation in a dietary intervention to lower cholesterol among individuals with hyperlipidemia. *Health Psychol* 1996;15:61-4.
10. Ni Mhurchu C, Margetts BM, Speller VM. Applying the stages-of-change model to dietary change. *Nutr Rev* 1997;55(1 pt 1):10-6.
11. Greene GW, Rossi SR, Rossi JS, et al. Dietary applications of the stages of change model. *J Am Diet Assoc* 1999;99:673-8.
12. Ling AM, Horwath C. Defining and measuring stages of change for dietary behaviors: readiness to meet fruit, vegetable, and grain guidelines among Chinese Singaporeans. *J Am Diet Assoc* 2000;100:898-904.
13. Hawkins DS, Hornsby PP, Schorling JB. Stages of change and weight loss among rural African American women. *Obes Res* 2001;9:59-67.
14. Hellman EA. Use of the stages of change in exercise adherence model among older adults with a cardiac diagnosis. *J Cardiopulm Rehabil* 1997;17:145-55.
15. Nigg CR, Courneya KS. Transtheoretical model: examining adolescent exercise behavior. *J Adolesc Health* 1998;22:214-24.
16. Laforge RG, Rossi JS, Prochaska JO, et al. Stage of regular exercise and health-related quality of life. *Prev Med* 1999;28:349-60.
17. Miiunpalo S, Nupponen R, Laitakari J, et al. Stages of change in two modes of health-enhancing physical activity: methodological aspects and promotional implications. *Health Educ Res* 2000;15:435-48.
18. Dunnagan T, Haynes G, Smith V. The relationship between the stages of change for exercise and health insurance costs. *Am J Health Behav* 2001;25:447-59.
19. Sarkin JA, Johnson SS, Prochaska JO, et al. Applying the transtheoretical model to regular moderate exercise in an overweight population: validation of a stage of change measure. *Prev Med* 2001;33:462-9.
20. Rodgers WM, Courneya KS, Bayduza AL. Examination of the transtheoretical model and exercise in 3 populations. *Am J Health Behav* 2001;25:33-41.
21. Dannecker EA, Hausenblas HA, Connaughton DP, et al. Validation of stages of exercise change questionnaire. *Res Q Exerc Sport* 2003;74:236-47.
22. Kim YH. Korean adolescents' exercise behavior and its relationship with psychological variables based on stages of change model. *J Adolesc Health* 2004;34:523-30.
23. Krummel DA, Semmens E, Boury J, et al. Stages of change for weight management in postpartum women. *J Am Diet Assoc* 2004;104:1102-8.
24. Kraft P, Sutton S, Reynolds HM. The transtheoretical model of behaviour change: are the stages qualitatively different? *Psychol Health* 1999;14:433-50.
25. Martin PD, Dutton GR, Brantley PJ. Self-efficacy as a predictor of weight change in African-American women. *Obes Res* 2004;12:646-51.
26. Edell BH, Edington S, Herd B, et al. Self-efficacy and self-motivation as predictors of weight loss. *Addict Behav* 1987;12:63-6.
27. Clark MM, Abrams DB, Niaura RS, et al. Self-efficacy in weight management. *J Consult Clin Psychol* 1991;59:739-44.
28. Marcus BH, Selby VC, Niaura RS, et al. Self-efficacy and the stages of exercise behavior change. *Res Q Exerc Sport* 1992;63:60-6.
29. Prochaska JO, Velicer WF, Rossi JS, et al. Stages of change and decisional balance for 12 problem behaviors. *Health Psychol* 1994;13:39-46.
30. Zabinski MF, Saelens BE, Stein RJ, et al. Overweight children's barriers to and support for physical activity. *Obes Res* 2003;11:238-46.
31. Prochaska JO, Velicer WF. The transtheoretical model of health behavior change. *Am J Health Promot* 1997;12:38-48.
32. Rollnick S. Behaviour change in practice: targeting individuals. *Int J Obes Relat Metab Disord* 1996;20(Suppl 1):S22-6.

## 6. Assessment of obesity and its complications in adults

Irene Hramiak, Lawrence Leiter, Terri L. Paul, Ehud Ur

With the high prevalence of obesity among all age groups in North America, the medical practitioner must be able to identify those at greatest risk. Anthropometric measurements and BMI assessment confirm the obese phenotype. Obese people are at high risk of developing significant comorbidities. Those with metabolic syndrome, characterized by increased visceral fat and the insulin resistance, are at risk of type 2 diabetes mellitus and cardiovascular disease.<sup>1-3</sup> Obesity also predisposes to endothelial dysfunction, carotid intimal medial thickening and the development of early coronary artery disease.<sup>4</sup> Obese people typically have hyperlipidemia with low levels of HDL cholesterol and high levels of triglycerides. Hypertension is more common in obese people and is a consequence of insulin resistance, increased aldosterone levels and salt sensitivity.<sup>4</sup>

Clearly one of the most serious comorbidities of obesity is type 2 diabetes. In both men and women with a BMI above 30 kg/m<sup>2</sup>, there is a linear relation between BMI and the risk of diabetes<sup>5</sup> (Fig. 8). Many factors, including insulin resistance, increased levels of free fatty acids and increased hepatic glucose output, lead to systemic hyperinsulinemia. Over time,

beta-cell production of insulin fails, hyperglycemia is ongoing, and frank type 2 diabetes develops.<sup>6</sup> In women, insulin resistance is integral to the polycystic ovarian syndrome, with its characteristic oligomenorrhea, hirsutism, androgenic alopecia, acne and acanthosis nigricans. Patients with polycystic ovarian syndrome are known to be at increased risk of heart disease and metabolic abnormalities.

Obstructive sleep apnea is an underrecognized complication of obesity. In the Swedish Obese Subjects Study,<sup>7</sup> among patients with a BMI greater than 35 kg/m<sup>2</sup>, 50% of men and 35% of women reported snoring and apnea. Obstructive sleep apnea can lead to cardiac arrhythmias, nocturnal hypoxia, heart failure and pulmonary hypertension.

Obese people are at increased risk of endometrial, breast, colon and prostate cancer.<sup>8</sup> Obesity is associated with a spectrum of liver abnormalities, referred to as nonalcoholic fatty liver disease. Patients with fatty liver disease have moderately elevated liver enzymes, 1-4 times the upper limit of normal; elevation of the alanine aminotransferase level is usually higher than that of aspartate aminotransferase. Bilirubin, albumin and prothrombin abnormalities may develop in later stages. The natural history varies among patients. Hepatic

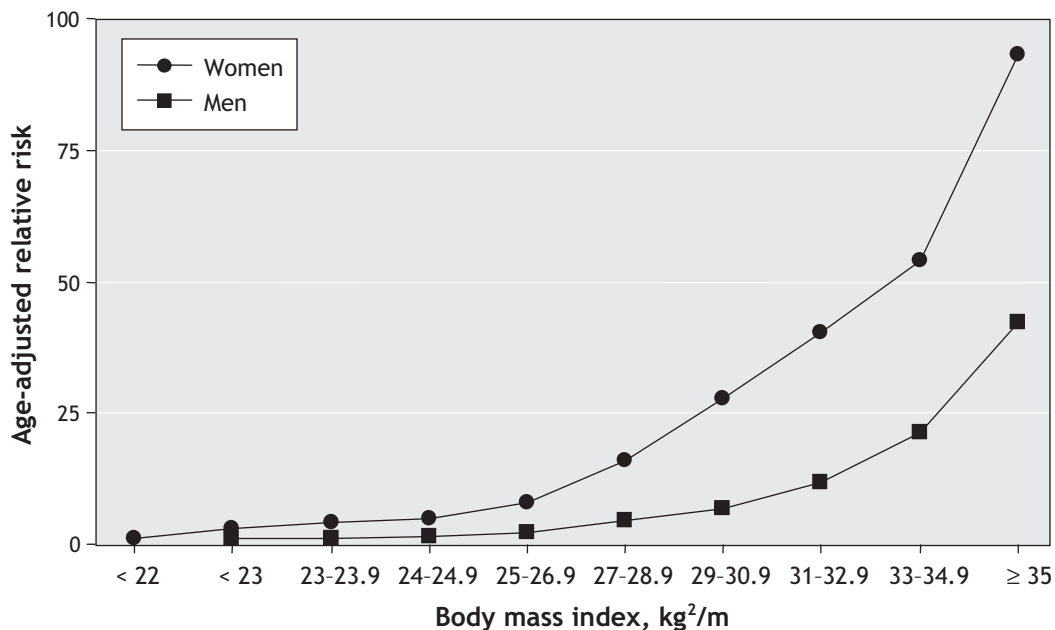


Fig. 8: Relationship between body mass index and risk of type 2 diabetes mellitus. Sources: Chan J et al (*Diabetes Care* 1994;17:961) and Colditz G et al (*Ann Intern Med* 1995;122:481).

steatosis is frequently characterized by a benign course without histologic progression. Nonalcoholic steatohepatitis (NASH), however, may become associated with increasing fibrosis and ultimately cirrhosis.<sup>9</sup> In addition, the obese person is at increased risk of osteoarthritis, gallbladder disease and skin changes, as well as neurologic changes such as benign intracranial hypertension (Box 3).

## Questions to be considered during history taking

### General weight history

The pattern of weight gain and weight loss over an obese person's lifetime may be significant. For instance, a person who weighed over 4500 g at birth and whose mother had diabetes during the pregnancy is at increased risk of obesity. Being overweight during childhood and adolescence indicates lifelong obesity. Lifelong obesity is associated with a lower risk of comorbidities than weight gained solely during adult life.<sup>10–13</sup> In women, weight gain at times of hormonal change (menarche, pregnancy and menopause) is common.

### Readiness for weight loss

It is essential to assess the patient's readiness to lose weight. For a person to succeed in a weight loss program, he or she must be motivated; free of major life crises, depression, substance abuse and eating disorders; and have sufficient time to devote to weight control. If the patient is not ready, the health care professional should explore the barriers to weight reduction and work at the prevention of future weight gain.

Other questions whose answers might help in the management of the patient include: Why is the patient seeking attention for the weight problem at this particular time? Why have previous attempts at weight loss failed? What are the patient's expectations with regard to weight loss and weight maintenance?

### Patient enquiry

It is important to obtain a medication history, not only for treatment of the comorbid conditions of obesity, but also to determine whether any medications are causing weight gain (Table 9). Recently, novel antipsychotics have been reported to result in significant weight gain.<sup>14–16</sup> Any previous surgery for obesity, gallbladder disease or malignant disease should be noted.

### Physical examination

A thorough general physical examination is warranted for all patients with obesity. Appropriate tools (e.g., appropriately sized blood pressure cuffs) are necessary.

- An accurate height in metres and weight in kilograms should be determined, and BMI should be calculated.<sup>17–19</sup>
- We recommend measuring waist circumference in all

adults to assess obesity-related health risks.<sup>18,19</sup> Waist circumference should be measured while the patient is in the standing position. The tape should be positioned in a horizontal plane at the level of the top of the iliac crest, which is used as a landmark to standardize measurement position. The patient's abdominal muscles should be relaxed and the measure should be performed after normal expiration.<sup>20</sup>

- In some populations, such as Asian and Middle Eastern people, the waist-to-hip ratio may be more predictive of heart disease than the waist circumference. The hip circumference is measured at the level of the widest diameter

#### Box 3: Complications associated with obesity

##### Cardiovascular system

Coronary artery disease  
Hypertension  
Pulmonary embolism  
Varicose veins

##### Gastrointestinal system

Cholelithiasis  
Gastroesophageal reflux disease  
Colon cancer  
Hepatic steatosis  
Hernias  
Nonalcoholic fatty liver disease

##### Integumental system

Cellulitis  
Carbuncles  
Hygiene problems  
Intertrigo  
Venous stasis of legs

##### Musculoskeletal system

Immobility  
Low back pain  
Osteoarthritis

##### Genitourinary system

Hypogonadism  
Prostate cancer  
Urinary stress incontinence

##### Neurologic system

Idiopathic intracranial hypertension  
Meralgia paresthetica  
Stroke

##### Psychosocial

Depression and suicidal ideation  
Social and employment discrimination  
Work disability  
Low self-esteem

##### Respiratory system

Dyspnea and fatigue  
Obesity hypoventilation syndrome (Pickwickian syndrome)  
Obstructive sleep apnea

around the buttocks. The calculated waist-to-hip ratio is high if it is greater than 0.83 in women and 0.9 in men across all populations.<sup>2</sup>

- Blood pressure should be determined, using a wide cuff where appropriate. The width of the cuff's inflatable bladder should be at least 40% of the arm's circumference, and the length of the bladder should be at least 80% of the arm's circumference (Table 10).<sup>21-23</sup> Readings are taken with the patient seated on each of 2 or more office visits.
- Examination of the head and neck should include a thyroid examination and funduscopy. If obstructive sleep apnea is suspected, the ears, nose and throat should be examined. The greatest risk factor for obstructive sleep apnea is large neck circumference adjusted for height.<sup>24-26</sup>
- Respiratory examination should elicit any signs of congestive heart failure.
- Cardiovascular examination, in addition to the usual practice, should focus on any evidence of cardiomegaly and vascular bruits.
- Abdominal examination should include an assessment for hepatomegaly and the abdominal pannus.

**Table 9:** Areas of patient enquiry in the assessment of obesity

Condition	Symptoms
Coronary artery disease	Shortness of breath at rest or on exertion, orthopnea, pedal edema, chest pain
Hypertension	Any history of elevated blood pressure
Diabetes	Polydipsia, polyuria, visual blurring, poorly healing infections
Obstructive sleep apnea	Fatigue, excessive daytime somnolence, morning headaches, snoring, night-time apnea
Polycystic ovarian syndrome	Hirsutism, alopecia, acne, menstrual irregularity, any abnormal vaginal bleeding
Malignant disease	Blood in the stool, prostatic hypertrophy, abnormal vaginal bleeding, breast masses
Benign intracranial hypertension	Headache, visual abnormalities, tinnitus, sixth nerve palsy
Musculoskeletal problems	Joint swelling, pain and redness
Psychiatric problems	Depression or eating disorders

**Table 10:** Appropriate size of cuff for measuring blood pressure

Arm circumference, cm	Size of cuff, cm
18-26	9 × 18 (child)
26-33	12 × 33 (standard)
33-41	15 × 33 (large, obese)
> 41	18 × 36 (extra large, obese)

Source: Canadian Hypertension Education Program (CHEP) 2006 recommendations [blood measurement slide kit].<sup>23</sup>

- Examination of the extremities should reveal any evidence of edema, joint deformities or decreased range of motion associated with osteoarthritic changes.
- A thorough breast examination should be performed, particularly in the postmenopausal woman who is at greater risk of breast cancer. In addition, gynecologic examination for ovarian or uterine masses and cervical assessment should be performed.
- In men, a digital rectal examination may be warranted.
- The skin should be closely examined for the presence of acanthosis nigricans, xanthomas, venous stasis changes, intertrigo, hirsutism and ulceration.

## Investigations in obesity

Laboratory tests are recommended to screen for diabetes, dyslipidemia and gout. They should include gram glucose challenge if the fasting glucose level is above 6.0 mmol/L (or 5.7 mmol/L with another risk factor for diabetes)<sup>27</sup> and fasting lipid profile (total cholesterol, triglycerides, HDL cholesterol and calculated LDL cholesterol).<sup>28</sup> Liver function studies, in particular aspartate aminotransferase and alanine aminotransferase levels, should be measured if nonalcoholic fatty liver disease is suspected. Diagnosis of nonalcoholic fatty liver disease can be confirmed by imaging (ultrasonography or computed tomography). Confirmation of the diagnosis may require a liver biopsy. Urinalysis should be undertaken annually to assess proteinuria.<sup>29-31</sup>

In obese women with symptoms suggestive of polycystic ovarian syndrome, hormonal studies should be undertaken when the patient has been off the birth control pill for at least 6 weeks. Levels of testosterone, luteinizing hormone, follicle-stimulating hormones, prolactin, dehydroepiandrosterone (DHEAS) and 17-hydroxyprogesterone should be measured. For patients with polycystic ovarian syndrome, a lipid profile and fasting blood glucose level should also be obtained to screen for diabetes.

Routine screening to rule out metabolic causes of obesity (e.g., hypothyroidism, acromegaly, Cushing's disease) should be discouraged unless clinically indicated.

Any additional testing should be based on clinical parameters and may include electrocardiogram stress testing, sleep study, joint assessment by radiography, mammography, colonoscopy and endometrial biopsy.

There are currently no specific recommendations for screening for malignant disease in obese individuals beyond those suggested for the general population.<sup>32,33</sup>

## Recommendations

1. We recommend measuring BMI (weight in kilograms divided by height in meters squared) in all adults [*grade A, level 3*<sup>17-19</sup>].
2. We recommend measuring waist circumference in all adults to assess obesity-related health risks [*grade A, level 3*<sup>18,19</sup>].
3. We recommend that the clinical evaluation of overweight and obese adults include a history and a general physical examination to exclude secondary (endocrine or syn-

drome-related) causes of obesity and obesity-related health risks and complications [grade A, level 3<sup>10,16,27,33</sup>].

4. We recommend measuring fasting plasma glucose level and determining lipid profile, including total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol and ratio of total cholesterol to HDL cholesterol, as screening tests in overweight and obese adults [grade A, level 3<sup>16,28</sup>]. We suggest repeating these tests at regular intervals as needed [grade C, level 4].
5. We suggest additional investigations, such as liver enzyme tests, urinalysis and sleep studies (when appropriate), to screen for and exclude other common obesity-related health problems [grade B, level 3<sup>29-31</sup>].

From the Department of Medicine, University of Western Ontario, London, Ont. (Hrarniak, Paul); the Departments of Medicine and Nutritional Sciences, University of Toronto, Toronto, Ont. (Leiter); and the Department of Medicine, Dalhousie University, Halifax, NS (Ur)

**Competing interests:** None declared for Terri Paul. Irene Hrarniak is a consultant to GlaxoSmithKline Inc. and is on a National Advisory Board for Abbott Laboratories Ltd., Eli Lilly, Novo Nordisk, sanofi-aventis Canada Inc. and GlaxoSmithKline Inc. She has received honoraria for speaking engagements from Merck Frosst Canada Ltd., GlaxoSmithKline Inc. and Novo Nordisk and has received a travel grant from Novo Nordisk. Lawrence Leiter has ongoing paid consultancy with Abbott Laboratories Ltd. and sanofi-aventis Canada Inc., has received an honorarium from Roche Canada and sanofi-aventis Canada Inc. and has received speaker fees or educational grants from sanofi-aventis Canada Inc. Ehud Ur has received speaker fees from sanofi-aventis Canada Inc., Abbott Laboratories Ltd., GlaxoSmithKline and Novo Nordisk; research grants from GlaxoSmithKline and Novo Nordisk; and travel assistance from sanofi-aventis Canada Inc. and Abbott Laboratories Ltd.

## REFERENCES

1. Isomaa B, Almgren P, Tuomi T, et al. Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care* 2001;24:683-9.
2. Yusuf S, Hawken S, Ounpuu S, et al. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 2005;366:1640-9.
3. Alberti KG, Zimmet P, Shaw J; IDF Epidemiology Task Force Consensus Group. The metabolic syndrome — a new worldwide definition. *Lancet* 2005;366:1059-62.
4. Poirier P, Giles TD, Bray GA, et al. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation* 2006;113:898-918.
5. Willett WC, Manson JE, Stampfer MJ, et al. Weight, weight change, and coronary heart disease in women. Risk within the 'normal' weight range. *JAMA* 1995;273:461-5.
6. Kopelman PG. Obesity as a medical problem. *Nature* 2000;404:635-43.
7. Grunstein RR, Stenlof K, Hedner J, et al. Impact of obstructive sleep apnea and sleepiness on metabolic and cardiovascular risk factors in the Swedish Obese Subjects (SOS) Study. *Int J Obes Relat Metab Disord* 1995;19:410-8.
8. Pan SY, Johnson KC, Ugnat AM, et al. Association of obesity and cancer risk in Canada. *Am J Epidemiol* 2004;159:259-68.
9. Jeffcoate W. Obesity is a disease: food for thought. *Lancet* 1998;351:903-4.
10. National Institutes of Health. *Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults: the evidence report*. Bethesda (MD): National Institutes of Health; 1998. NIH no 98-4083. Available: [www.nhlbi.nih.gov/guidelines/obesity/ob\\_gdlns.pdf](http://www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.pdf) (accessed 2007 Jan 30).
11. McTigue KM, Harris R, Hemphill B, et al. Screening and interventions for obesity in adults: summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med* 2003;139:933-49.
12. Muscelli E, Camastra S, Gastaldelli A, et al. Influence of duration of obesity on the insulin resistance of obese non-diabetic patients. *Int J Obes Relat Metab Disord* 1998;22:262-7.
13. Brochu M, Tchernof A, Dionne JJ, et al. What are the physical characteristics associated with a normal metabolic profile despite a high level of obesity in postmenopausal women? *J Clin Endocrinol Metab* 2001;86:1020-5.
14. Allison DB, Casey DE. Antipsychotic-induced weight gain: a review of the literature. *J Clin Psychiatry* 2001; 62(Suppl 7):22-31.
15. Russell JM, Mackell JA. Body weight gain associated with atypical antipsychotics: epidemiology and therapeutic implications. *CNS Drugs* 2001;15:537-51.
16. Wetterling T. Body weight gain with atypical antipsychotics. A comparative review. *Drug Saf* 2001;24:59-73.
17. National Institutes of Health. *The practical guide: identification, evaluation, and treatment of overweight and obesity in adults*. Bethesda (MD): National Institutes of Health; 2000. NIH no 00-4084. Available: [www.nhlbi.nih.gov/guidelines/obesity/prctgd\\_b.pdf](http://www.nhlbi.nih.gov/guidelines/obesity/prctgd_b.pdf) (accessed 2007 Jan 30).
18. Dickey RA, Bartuska DG, Bray GW, et al. AACE/ACE position statement on the prevention, diagnosis, and treatment of obesity. *Endocr Pract* 1998;4(5):297-330. Available: [www.aace.com/pub/pdf/guidelines/obesityguide.pdf](http://www.aace.com/pub/pdf/guidelines/obesityguide.pdf) (accessed 2007 Jan 31).
19. Lyznicki JM, Young DC, Riggs JA, et al. Obesity: assessment and management in primary care. *Am Fam Physician* 2001;63:2185-96.
20. Health Canada. *Canadian guidelines for body weight classification in adults*. Ottawa: Health Canada; 2003. Cat no H49-179/2003E. Available: [www.hc-sc.gc.ca/fn-an/alt\\_formats/hpfb-dgpsa/pdf/nutrition/weight\\_book-livres\\_des\\_poids\\_e.pdf](http://www.hc-sc.gc.ca/fn-an/alt_formats/hpfb-dgpsa/pdf/nutrition/weight_book-livres_des_poids_e.pdf) (accessed 2007 Jan 31).
21. Douketis JD, Feightner JW, Attia J, et al. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. Canadian Task Force on Preventive Health Care. *CMAJ* 1999;160:513-25.
22. Khan NA, McAlister FA, Campbell NRC, et al; Canadian Hypertension Education Program. The 2004 Canadian recommendations for the management of hypertension: Part II — Therapy. *Can J Cardiol* 2004;20:41-54.
23. Canadian Hypertension Education Program (CHEP). 2006 Canadian Hypertension Education Program recommendations: Blood pressure assessment, hypertension diagnosis and follow-up [supplementary slide kit]. Available: [www.hypertension.ca/chep/docs/CHEP\\_2006\\_BP\\_Measure.ppt](http://www.hypertension.ca/chep/docs/CHEP_2006_BP_Measure.ppt) (accessed 2007 Mar 13).
24. Davies RJ, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnoea syndrome. *Eur Respir J* 1990;3:509-14.
25. Wolk R, Shamsuzzaman AS, Somers VK. Obesity, sleep apnea, and hypertension. *Hypertension* 2003;42:1067-74.
26. Stradling JR, Crosby JH. Predictors and prevalence of obstructive sleep apnoea and snoring in 1001 middle aged men. *Thorax* 1991;46:85-90.
27. Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. Canadian Diabetes Association 2003 clinical practice guidelines for the prevention and management of diabetes in Canada. *Can J Diabetes* 2003;27(Suppl 2):S1-152.
28. Genest J, Frohlich J, Fodor G, et al. Recommendations for the management of dyslipidemia and the prevention of cardiovascular disease: summary of the 2003 update. *CMAJ* 2003;169:921-4.
29. Kasiske BL, Crosson JT. Renal disease in patients with massive obesity. *Arch Intern Med* 1986;146:1105-9.
30. Chagnac A, Weinstein T, Herman M, et al. The effects of weight loss on renal function in patients with severe obesity. *J Am Soc Nephrol* 2003;14:1480-6.
31. Kambham N, Markowitz GS, Valeri AM, et al. Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int* 2001;59:1498-509.
32. Van den Brandt PA, Spiegelman D, Yaun SS, et al. Pooled analysis of prospective cohort studies on height, weight, and breast cancer risk. *Am J Epidemiol* 2000;152:514-27.
33. Trentham-Dietz A, Newcomb PA, Storer BE, et al. Body size and risk of breast cancer. *Am J Epidemiol* 1997;145:1011-9.



# 7. Psychiatric issues in the management of obesity

Robert Dent, Rami Habib, Louis Soucy, Hany Bissada

**W**e conducted a systematic review and evaluation of major depressive disorder, its association with obesity and its influence on outcomes in the treatment of obesity; binge-eating disorder, its association with overweight and obesity and its influence on obesity treatment outcomes; and psychotropic drug-induced weight gain and the association of psychotropic drugs with weight gain. This choice was based on our experience in treating obesity, the availability of pertinent literature and avoidance of duplication of topics reviewed in other chapters.

The following electronic databases were searched: PubMed, MEDLINE, EMBASE and the Cochrane Controlled Clinical Trials Register. Inclusion criteria were: prospective clinical trials (randomized and nonrandomized); retrospective trials in large populations; and systematic reviews and meta-analyses. The keywords for the searches were: obesity and major affective disorder, seasonal affective disorder, depression, depression and adherence, binge-eating behaviour, binge-eating disorder, bipolar affective disorder, schizophrenia, obesity or weight gain and tricyclic antidepressants, clomipramine, monoamine oxidase inhibitors, clozapine, quetiapine, moclobemide, fluoxetine, fluvoxamine, sertraline, citalopram, paroxetine, venlafaxine, sibutramine, lithium, topiramate, haloperidol, valproate, lamotrigine, ziprasidone, olanzapine and risperidone. Studies were selected according to the standard methods for formulating the evidence-based recommendations of this project. Studies with a follow-up of less than a year were accepted for inclusion if longer-term studies were unavailable and the results were deemed clinically useful.

## Major depressive disorder and obesity

### Is there an association between major depressive disorder and obesity?

The literature review revealed 11 studies that met project criteria and used standard diagnostic criteria for major depressive disorder: 2 were large cross-sectional studies,<sup>1,2</sup> 7 were prospective studies,<sup>3-9</sup> and 2 were systematic reviews.<sup>10,11</sup> Four of the prospective studies showed that major depressive disorder predicted obesity later in life.<sup>6-9</sup> One prospective study showed that obesity in childhood was predictive of future development of depression.<sup>5</sup> An increased prevalence of depression was associated with a lower BMI among women than among men (BMI 30 kg/m<sup>2</sup> v. BMI 40 kg/m<sup>2</sup> respectively).<sup>1</sup> The prospective studies indicated that, although there was an association, the 2 conditions may not coexist in any 1 patient

at the same time. Three studies linked major depressive disorder to factors relevant to obesity or metabolic syndrome.<sup>12-14</sup> These studies provide strong evidence for an association between depression and obesity.

### Does major depressive disorder affect obesity treatment outcomes?

We found no study dealing with obesity per se, but 4 studies that met project criteria showed that major depressive disorder did affect outcomes of treatment of other chronic medical conditions. One case series<sup>15</sup> showed that major depressive disorder was associated with decreased adherence to diet, exercise and medication use in patients after a myocardial infarction. One meta-analysis<sup>16</sup> showed that major depressive disorder was associated with poor adherence to diet (4 studies) and to medication use (5 studies) in patients with diabetes and end-stage renal disease. Two 12-month prospective studies<sup>17,18</sup> showed that major depressive disorder was associated with poor treatment outcomes.

### Does treating major depressive disorder improve obesity treatment outcomes?

Two studies meeting project criteria were found.<sup>19,20</sup> Both were RCTs of 1-year duration with large samples, but neither dealt with obesity treatment per se. Both showed improvement in treatment outcomes — decreased health care costs among elderly patients<sup>19</sup> and increased exercise among people with diabetes<sup>20</sup> — with treatment of major depressive disorder.

## Binge-eating disorder and obesity

It is particularly important for health professionals to consider binge-eating disorder in the management of obese and overweight individuals. In the past, there has been a concern that dieting might cause binge-eating disorder or increase the tendency to binge. Binge eating is often cited as a barrier to weight management<sup>18</sup>; it is strongly associated with overweight and obesity<sup>21</sup> and with psychiatric comorbidities, especially depression.<sup>21</sup>

An expert panel<sup>22</sup> that reviewed articles evaluating the relations among dieting, obesity treatment, weight cycling, eating disorders and psychological functioning in overweight and obese adults<sup>23</sup> concluded that non-dieting approaches to the treatment of eating disorders seem to lead to improvements in mood and self-esteem but typically to minimal reduction in

body weight. The panel also found that empirical studies did not support the concern that dieting induces eating disorders or other psychological dysfunction in overweight and obese adults. Such concerns should not preclude attempts to reduce energy intake and increase physical activity to achieve modest weight loss or prevent additional weight gain.

### **What is the prevalence of binge-eating disorder among obese patients?**

Three studies on this topic met project criteria: a cross-sectional study involving 1632 people,<sup>17</sup> a case series of 159 people<sup>24</sup> and a systematic review.<sup>21</sup> The prevalence of binge-eating disorder was found to be about 20% among obese men (27% in studies using questionnaires<sup>17</sup> and 9%–19% in studies based on structured interviews<sup>24</sup>). Overall, binge-eating disorder occurred in 10%–20% of overweight and obese individuals. We found no studies meeting project criteria that showed the prevalence of binge-eating disorder among people who were not overweight or obese.

### **Are obese people with binge-eating disorder more likely to have major depressive disorder?**

Five studies met project criteria.<sup>18,25–28</sup> Three<sup>25–27</sup> reported a 50% prevalence of major depressive disorder, and the other 2 reported that prevalence of major depressive disorder among patients with binge-eating disorder was twice that in comparison groups.<sup>18,28</sup> Woodside and colleagues<sup>28</sup> found that, among individuals with binge-eating disorder, men had rates of major depressive disorder 2–3-fold higher than rates among women. One review questioned the possibility that the major depressive disorder found in those with binge-eating disorder might account for the increased prevalence of major depressive disorder in the obese population. Evidence for an association between binge-eating disorder in overweight and obese people and major depressive disorder was stronger than for any other area of exploration in this section. Significant contributors to the definition of this condition have asked whether binge-eating disorder might be less important as a disorder than as a marker of psychiatric comorbidities such as major depressive disorder.<sup>21</sup>

### **Is binge-eating disorder associated with poorer outcomes in weight management programs?**

Only 2 studies<sup>17,18</sup> examined an association between binge-eating disorder and outcomes in weight management programs. Both reported little impact on weight loss. Both also showed that comorbid major depressive disorder might have a greater impact on weight management outcomes than binge-eating disorder.

### **Does treating binge-eating disorder improve weight management outcomes?**

The 3 studies meeting project criteria showed that individuals with binge-eating disorder responded to behavioural treat-

ments with the result of reduced bingeing but minimal weight loss.<sup>29–31</sup> Although there were 6 reports of RCTs of drug treatment of binge-eating disorder with such drugs as fluoxetine, fluvoxamine, sertraline, topiramate and sibutramine, none met study criteria because of their short duration, but they did show promise of improvements in both binge eating and weight status.

### **Weight gain and pharmacotherapy for major depressive disorder and psychiatric conditions**

Psychotropic-associated weight gain affects compliance with drug use, which results in relapse and longer hospital stays.<sup>32–35</sup> Weight gain is associated with lower quality of life and social withdrawal<sup>32,34</sup> as well as an increased risk of diabetes and other health disorders. In a large group of people with schizophrenia, those taking atypical antipsychotic agents were 9% more likely than those receiving typical neuroleptics to have diabetes.<sup>36</sup> Fontaine and colleagues<sup>37</sup> estimated that the decreased risk of suicide with the use of antipsychotic agents may be offset by the increased mortality associated with antipsychotic-induced weight gain.

We examined the association of weight gain with commonly used psychotropic drugs: tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs), serotonin noradrenalin reuptake inhibitors (SNRIs), atypical antidepressants, monoamine oxidase inhibitors (nonselective and selective), mood stabilizers, conventional antipsychotics and novel antipsychotics. Table 11 shows the weight gain potential of these classes of psychotropics.<sup>37–94</sup> Because of the lack of availability of long-term studies, especially for the older drugs, we accepted controlled or comparative trials with a duration of 4 weeks or more if they were clinically relevant and if longer-term trials were not available. We reviewed articles for each class of drug and, wherever possible, classified drugs as “associated with weight loss,” “weight neutral” or “associated with weight gain.” We chose not to give estimates for amount of weight gain because:

- Most of the patients in these studies were already taking psychotropics, which cause weight gain. Consequently, the reported weight gain would be relative to the comparison group and would likely be an underestimate of the true weight-gain potential of the drug.
- Most of the studies could not distinguish between weight gain caused by the drug used to treat the psychiatric condition and weight changes associated with the onset or improvement of the psychiatric condition.

Allison and others<sup>33,61</sup> compared the weight gain associated with antipsychotic medications in the first 10 weeks of treatment and ranked them in ascending order according to weight-gain potential as follows: placebo, ziprasidone, fluphenazine, haloperidol, risperidone, chlorpromazine, thioridazine, olanzapine and clozapine. Tandon and colleagues<sup>95</sup> ranked quetiapine between risperidone and olanzapine.

Table 11: Ranking of psychotropic agents in terms of their potential effect on weight			
Class of drug	Associated with weight loss	Weight neutral	Associated with weight gain
Monoamine oxidase inhibitors (nonselective, irreversible)		Tranylcypromine <sup>32,38,39</sup>	Phenelzine <sup>32,38-40</sup>
Monoamine oxidase inhibitors (reversible)		Moclobemide <sup>32,41,42</sup>	
Mood stabilizers/anticonvulsants	Topiramate <sup>43-47</sup>	Carbamazepine <sup>48</sup> Lamotrigine <sup>47,49</sup>	Gabapentin <sup>47,50</sup> Lithium <sup>38,47-56</sup> Valproate <sup>47,51,57-60</sup>
Typical antipsychotics			Chlorpromazine <sup>32,33,61,62</sup> Fluphenazine <sup>58,63</sup> Haloperidol <sup>58,63</sup> Thioridazine <sup>32,33,61,62</sup>
Atypical antipsychotics		Ziprasidone <sup>*33,64-67</sup>	Clozapine <sup>33,49,68-71</sup> Olanzapine <sup>33,49,57,58,63,68-77</sup> Quetiapine <sup>33,49,68-71</sup> Risperidone <sup>33,49,68-71,73,78,79</sup>
Anxiolytics		Chlorodiazepoxide <sup>80</sup> Lormetazepam <sup>81</sup> Nitazepam <sup>81</sup>	
Tricyclic antidepressants			Amitriptyline <sup>40,82-85</sup> Chlomipramine <sup>42,86,87</sup> Desipramine <sup>82</sup> Imipramine <sup>40</sup> Nortriptyline <sup>38,82</sup> Trimipramine <sup>40</sup>
Selective serotonin reuptake inhibitors		Citalopram <sup>88</sup> Fluoxetine <sup>88,89</sup> Fluvoxamine <sup>88</sup> Sertraline <sup>88</sup>	Paroxetine <sup>88,90,91</sup>
Serotonin noradrenalin reuptake inhibitors	Sibutramine	Venlafaxine <sup>32</sup>	
Miscellaneous antidepressants		Bupropion <sup>32,35,85,87</sup> Trazodone <sup>84</sup>	Mirtazepine <sup>92-94</sup>

\*Not available in Canada.

## Conclusions

There is evidence to link paroxetine, mirtazepine, lithium, valproate, gabapentin, tricyclic antidepressants, typical antipsychotics and atypical antipsychotics (with the possible exception of ziprasidone) with weight gain. Although there is sufficient evidence to link the use of certain psychotropics with weight gain, it is not adequate to make evidence-based recommendations for treating the problem. Drug selection is the best strategy for the prevention and treatment of drug-associated weight gain, in addition to the standard methods for weight management. Table 11 provides direction for choosing classes of psychotropics and drugs within the classes that are less likely to be associated with weight gain. The ranking of antipsychotic drugs mentioned in the previous paragraph is intended to provide further assistance in choosing drugs in this class.

## Recommendations

1. We suggest that the health care professional screen the overweight or obese adult for eating disorders, depression

and psychiatric disorders, as appropriate [*grade B, level 3*<sup>3,4,6-9,15-20</sup>].

2. Randomized clinical trials are needed to examine the effect of the treatment of depression on outcomes of treatment for obesity. Further studies with improved diagnostic methods and prospective designs are also needed to delineate the association between obesity and major depressive disorder [*grade C, level 4*].
3. Research to find drugs that do not cause weight gain in treatment areas such as psychiatry is needed [*grade C, level 4*].

From the Weight Management Clinic, Ottawa Hospital (Dent); the Mood Disorders Program, Royal Ottawa Hospital (Habib); the University of Ottawa and Royal Ottawa Hospital (Soucy); the Regional Center for the Treatment of Eating Disorders, University of Ottawa (Bissada), Ottawa, Ont.

**Competing interests:** None declared for Hany Bissada, Rami Habib and Louis Soucy. Robert Dent has ongoing paid consultancies with AstraZeneca Advisory Board for Galida (now ended) and Abbott Advisory Board for Meridia. He has received speaker fees and educational grants from Abbott Laboratories Ltd., GlaxoSmithKline and Novartis, and travel assistance from Abbott Laboratories Ltd. to attend a meeting.

## REFERENCES

- Carpenter KM, Hasin DS, Allison DB, et al. Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: results from a general population study. *Am J Public Health* 2000;90:251-7.
- Onyike CU, Crum RM, Lee HB, et al. Is obesity associated with major depression? Results from the Third National Health and Nutrition Examination Survey. *Am J Epidemiol* 2003;158:1139-47.
- Roberts RE, Kaplan GA, Shema SJ, et al. Are the obese at greater risk for depression? *Am J Epidemiol* 2000;152:163-70.
- Roberts RE, Deleger S, Strawbridge WJ, et al. Prospective association between obesity and depression: evidence from the Alameda County Study. *Int J Obes Relat Metab Disord* 2003;27:514-21.
- Mustillo S, Worthman C, Erkanli A, et al. Obesity and psychiatric disorder: developmental trajectories. *Pediatrics* 2003;111:851-9.
- Pine DS, Goldstein RB, Wolk S, et al. The association between childhood depression and adulthood body mass index. *Pediatrics* 2001;107:1049-56.
- Goodman E, Whitaker RC. A prospective study of the role of depression in the development and persistence of adolescent obesity. *Pediatrics* 2002;110:497-504.
- DiPietro L, Anda RF, Williamson DF, et al. Depressive symptoms and weight change in a national cohort of adults. *Int J Obes Relat Metab Disord* 1992;16:745-53.
- Richardson LP, Davis R, Poulton R, et al. A longitudinal evaluation of adolescent depression and adult obesity. *Arch Pediatr Adolesc Med* 2003;157:739-45.
- McElroy SL, Kotwal R, Malhotra S, et al. Are mood disorders and obesity related? A review for the mental health professional. *J Clin Psychiatry* 2004;65:634-51.
- Faith MS, Calamaro CJ, Dolan MS, et al. Mood disorders and obesity. *Curr Opin Psychiatry* 2004;17:9-13.
- Ladwig KH, Marten-Mittag B, Lowel H, et al. Influence of depressive mood on the association of CRP and obesity in 3205 middle aged healthy men. *Brain Behav Immun* 2003;17:268-75.
- Raikkonen K, Matthews KA, Kuller LH. The relationship between psychological risk attributes and the metabolic syndrome in healthy women: Antecedent or consequence? *Metabolism* 2002;51:1573-7.
- McCaffery JM, Niaura R, Todaro JF, et al. Depressive symptoms and metabolic risk in adult male twins enrolled in the National Heart, Lung, and Blood Institute twin study. *Psychosom Med* 2003;65:490-7.
- Ziegelstein RC, Fauerbach JA, Stevens SS, et al. Patients with depression are less likely to follow recommendations to reduce cardiac risk during recovery from a myocardial infarction. *Arch Intern Med* 2000;160:1818-23.
- DiMatteo MR, Lepper HS, Croghan TW. Depression is a risk factor for noncompliance with medical treatment: meta-analysis of the effects of anxiety and depression on patient adherence. *Arch Intern Med* 2000;160:2101-7.
- Linde JA, Jeffery RW, Levy RL, et al. Binge eating disorder, weight control self-efficacy, and depression in overweight men and women. *Int J Obes Relat Metab Disord* 2004;28:418-25.
- Sherwood NE, Jeffery RW, Wing RR. Binge status as a predictor of weight loss treatment outcome. *Int J Obes Relat Metab Disord* 1999;23:485-93.
- Unutzer J, Katon W, Williams JW Jr, et al. Improving primary care for depression in late life: the design of a multicenter randomized trial. *Med Care* 2001;39:785-99.
- Williams JW Jr, Katon W, Lin EH, et al. The effectiveness of depression care management on diabetes-related outcomes in older patients. *Ann Intern Med* 2004;140:1015-24.
- Stunkard AJ, Allison KC. Binge eating disorder: Disorder or marker? *Int J Eat Disord* 2003;34:S107-16.
- Overweight, obesity, and health risk. National Task Force on the Prevention and Treatment of Obesity. *Arch Intern Med* 2000;160:898-904.
- National Task Force on the Prevention and Treatment of Obesity. Dieting and the development of eating disorders in overweight and obese adults. *Arch Intern Med* 2000;160:2581-9.
- Cargill BR, Clark MM, Pera V, et al. Binge eating, body image, depression, and self-efficacy in an obese clinical population. *Obes Res* 1999;7:379-86.
- Mussell MP, Mitchell JE, de Zwaan M, et al. Clinical characteristics associated with binge eating in obese females: a descriptive study. *Int J Obes Relat Metab Disord* 1996;20:324-31.
- Bulik CM, Sullivan PF, Kendler KS. Medical and psychiatric morbidity in obese women with and without binge eating. *Int J Eat Disord* 2002;32:72-8.
- Telch CF, Stice E. Psychiatric comorbidity in women with binge eating disorder: prevalence rates from a non-treatment-seeking sample. *J Consult Clin Psychol* 1998;66:768-76.
- Woodside DB, Garfinkel PE, Lin E, et al. Comparisons of men with full or partial eating disorders, men without eating disorders, and women with eating disorders in the community. *Am J Psychiatry* 2001;158:570-4.
- Wilfley DE, Welch RR, Stein RI, et al. A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Arch Gen Psychiatry* 2002;59:713-21.
- Gladis MM, Wadden TA, Vogt R, et al. Behavioral treatment of obese binge eaters: Do they need different care? *J Psychosom Res* 1998;44:375-84.
- Tasca GA, Ritchie K, Conrad G, et al. Attachment scales predict outcome in a randomized controlled trial of two group therapies for binge eating disorder: an aptitude by treatment interaction. *Psychother Res* 2006;16:106-21.
- Masand PS. Weight gain associated with psychotropic drugs. *Expert Opin Pharmacother* 2000;1:377-89.
- Allison DB, Casey DE. Antipsychotic-induced weight gain: a review of the literature. *J Clin Psychiatry* 2001;62(Suppl 7):22-31.
- Kurzthaler I, Fleischhacker WW. The clinical implications of weight gain in schizophrenia. *J Clin Psychiatry* 2001;62(Suppl 7):32-7.
- Aronne LJ, Segal KR. Weight gain in the treatment of mood disorders. *J Clin Psychiatry* 2003;64(Suppl 8):22-9.
- Sernyak MJ, Leslie DL, Alarcon RD, et al. Association of diabetes mellitus with use of atypical neuroleptics in the treatment of schizophrenia. *Am J Psychiatry* 2002;159:561-6.
- Fontaine KR, Heo M, Harrigan EP, et al. Estimating the consequences of antipsychotic induced weight gain on health and mortality rate. *Psychiatry Res* 2001;101:277-88.
- Garland EJ, Remick RA, Zis AP. Weight gain with antidepressants and lithium. *J Clin Psychopharmacol* 1988;8:323-30.
- Cantu TG, Korek JS. Monoamine oxidase inhibitors and weight gain. *Drug Intell Clin Pharm* 1988;22:755-9.
- Harris B, Young J, Hughes B. Comparative effects of seven antidepressant regimes on appetite, weight and carbohydrate preference. *Br J Psychiatry* 1986;148:590-2.
- Fitton A, Faulds D, Goa KL. Moclobemide. A review of its pharmacological properties and therapeutic use in depressive illness. *Drugs* 1992;43:561-96.
- Guelfi JD, Payan C, Fermanian J, et al. Moclobemide versus clomipramine in endogenous depression. A double-blind randomised clinical trial. *Br J Psychiatry* 1992;160:519-24.
- McElroy SL, Suppes T, Keck PE, et al. Open-label adjunctive topiramate in the treatment of bipolar disorders. *Biol Psychiatry* 2000;47:1025-33.
- Astrup A, Caterson I, Zelissen P, et al. Topiramate: long-term maintenance of weight loss induced by a low-calorie diet in obese subjects. *Obes Res* 2004;12:1658-69.
- Wilding J, Van Gaal L, Rissanen A, et al. A randomized double-blind placebo-controlled study of the long-term efficacy and safety of topiramate in the treatment of obese subjects. *Int J Obes Relat Metab Disord* 2004;28:1399-410.
- Astrup A, Toubro S. Topiramate: a new potential pharmacological treatment for obesity. *Obes Res* 2004;12:167S-73S.
- Nemeroff CB. Safety of available agents used to treat bipolar disorder: focus on weight gain. *J Clin Psychiatry* 2003;64:532-9.
- Coxhead N, Silverstone T, Cookson J. Carbamazepine versus lithium in the prophylaxis of bipolar affective disorder. *Acta Psychiatr Scand* 1992;85:114-8.
- Keck PE, McElroy SL. Bipolar disorder, obesity, and pharmacotherapy-associated weight gain. *J Clin Psychiatry* 2003;64:1426-35.
- Baulac M, Cavalcanti D, Semah F, et al. Gabapentin add-on therapy with adaptable dosages in 610 patients with partial epilepsy: an open, observational study. The French Gabapentin Collaborative Group. *Seizure* 1998;7:55-62.
- Bowden CL, Calabrese JR, McElroy SL, et al. A randomized, placebo-controlled 12-month trial of divalproex and lithium in treatment of outpatients with bipolar I disorder. Divalproex Maintenance Study Group. *Arch Gen Psychiatry* 2000;57:481-9.
- Peselow ED, Dunner DL, Fieve RR, et al. Lithium carbonate and weight gain. *J Affect Disord* 1980;2:303-10.
- Elmslie JL, Mann JI, Silverstone JT, et al. Determinants of overweight and obesity in patients with bipolar disorder. *J Clin Psychiatry* 2001;62:486-91.
- Vestergaard P, Amdisen A, Schou M. Clinically significant side effects of lithium treatment. A survey of 237 patients in long-term treatment. *Acta Psychiatr Scand* 1980;62:193-200.
- Chen Y, Silverstone T. Lithium and weight gain. *Int Clin Psychopharmacol* 1990;5:217-25.
- Burgess S, Geddes J, Hawton K, et al. Lithium for maintenance treatment of mood disorders. *Cochrane Database Syst Rev* 2001;(3):CD003013.
- Tohen M, Chengappa KN, Suppes T, et al. Relapse prevention in bipolar I disorder: 18-month comparison of olanzapine plus mood stabiliser v. mood stabiliser alone. *Br J Psychiatry* 2004;184:337-45.
- Rendell JM, Gijsman HJ, Keck P, et al. Olanzapine alone or in combination for acute mania. *Cochrane Database Syst Rev* 2003;(3):CD004040.
- Bowden CL, Valproate. *Bipolar Disord* 2003;5:189-202.
- Biton V, Levisohn P, Hoyler S, et al. Lamotrigine versus valproate monotherapy-associated weight change in adolescents with epilepsy: results from a post hoc analysis of a randomized, double-blind clinical trial. *J Child Neurol* 2003;18:133-9.
- Allison DB, Mentore JL, Heo M, et al. Antipsychotic-induced weight gain: a comprehensive research synthesis. *Am J Psychiatry* 1999;156:1686-96.
- Blin O, Micallef J. Antipsychotic-associated weight gain and clinical outcome parameters. *J Clin Psychiatry* 2001;62(Suppl 7):11-21.
- Lieberman JA, Tollefson G, Tohen M, et al. Comparative efficacy and safety of atypical and conventional antipsychotic drugs in first-episode psychosis: a randomized, double-blind trial of olanzapine versus haloperidol. *Am J Psychiatry* 2003;160:1396-404.
- Arato M, O'Connor R, Meltzer HY, et al. Ziprasidone: efficacy in the prevention of relapse and in the long-term treatment of negative symptoms of chronic schizophrenia [abstract]. *Eur Neuropsychopharmacol* 1997;(Suppl 2):S214.
- Arato M, O'Connor R, Meltzer HY. A 1-year, double-blind, placebo-controlled trial of ziprasidone 40, 80 and 160 mg/day in chronic schizophrenia: the Ziprasidone Extended Use in Schizophrenia (ZEUS) study. *Int Clin Psychopharmacol* 2002;17:207-15.
- Bagnall A, Lewis RA, Leitner ML. Ziprasidone for schizophrenia and severe mental illness. *Cochrane Database Syst Rev* 2000;(4):CD001945.
- Simpson GM, Glick ID, Weiden PJ, et al. Randomized, controlled, double-blind multicenter comparison of the efficacy and tolerability of ziprasidone and olanzapine in acutely ill inpatients with schizophrenia or schizoaffective disorder. *Am J*

- Psychiatry* 2004;161:1837-47.
68. Czobor P, Volavka J, Sheitman B, et al. Antipsychotic-induced weight gain and therapeutic response: a differential association. *J Clin Psychopharmacol* 2002;22:244-51.
  69. McIntyre RS, Trakas K, Lin D, et al. Risk of weight gain associated with antipsychotic treatment: results from the Canadian National Outcomes Measurement Study in Schizophrenia. *Can J Psychiatry* 2003;48:689-94.
  70. Wirshing DA, Wirshing WC, Kysar L, et al. Novel antipsychotics: comparison of weight gain liabilities. *J Clin Psychiatry* 1999;60:358-63.
  71. Duggan L, Fenton M, Rathbone J, et al. Olanzapine for schizophrenia. *Cochrane Database Syst Rev* 2005;(2):CD001359.
  72. Corya SA, Andersen SW, Detke HC, et al. Long-term antidepressant efficacy and safety of olanzapine/fluoxetine combination: a 76-week open-label study. *J Clin Psychiatry* 2003;64:1349-56.
  73. Conley RR, Mahmoud R. A randomized double-blind study of risperidone and olanzapine in the treatment of schizophrenia or schizoaffective disorder. *Am J Psychiatry* 2001;158:765-74.
  74. Tohen M, Ketter TA, Zarate CA, et al. Olanzapine versus divalproex sodium for the treatment of acute mania and maintenance of remission: a 47-week study. *Am J Psychiatry* 2003;160:1263-71.
  75. Rosenheck R, Perlick D, Bingham S, et al. Effectiveness and cost of olanzapine and haloperidol in the treatment of schizophrenia: a randomized controlled trial. *JAMA* 2003;290:2693-702.
  76. Tran PV, Tollefson GD, Sanger TM, et al. Olanzapine versus haloperidol in the treatment of schizoaffective disorder. Acute and long-term therapy. *Br J Psychiatry* 1999;174:15-22.
  77. Buchanan RW, Ball MP, Weiner E, et al. Olanzapine treatment of residual positive and negative symptoms. *Am J Psychiatry* 2005;162:124-9.
  78. Martin A, Scahill L, Anderson GM, et al. Weight and leptin changes among risperidone-treated youths with autism: 6-month prospective data. *Am J Psychiatry* 2004;161:1125-7.
  79. Gilbody SM, Bagnall AM, Duggan L, et al. Risperidone versus other atypical antipsychotic medication for schizophrenia. *Cochrane Database Syst Rev* 2000;(3):CD002306.
  80. Bjertnaes A, Block JM, Hafstad PE, et al. A multicentre placebo-controlled trial comparing the efficacy of mianserin and chlordiazepoxide in general practice patients with primary anxiety. *Acta Psychiatr Scand* 1982;66:199-207.
  81. Oswald I, Adam K. Benzodiazepines cause small loss of body weight. *BMJ* 1980;281:1039-40.
  82. Fernstrom MH, Kupfer DJ. Antidepressant-induced weight gain: a comparison study of four medications. *Psychiatry Res* 1988;26:265-71.
  83. Kupfer DJ, Coble PA, Rubinstein D. Changes in weight during treatment for depression. *Psychosom Med* 1979;41:535-44.
  84. Hecht Orzack M, Cole JO, Friedman L, et al. Weight changes in antidepressants: a comparison of amitriptyline and trazodone. *Neuropsychobiology* 1986;15(Suppl 1):28-30.
  85. Chouinard G. Bupropion and amitriptyline in the treatment of depressed patients. *J Clin Psychiatry* 1983;44:121-9.
  86. Malina RM, Bouchard C, Bar-Or O. *Growth, maturation and physical activity*. 2nd ed. Champaign (IL): Human Kinetics; 2004.
  87. Feighner J, Hendrickson G, Miller L, et al. Double-blind comparison of doxepin versus bupropion in outpatients with a major depressive disorder. *J Clin Psychopharmacol* 1986;6:27-32.
  88. Maina G, Albert U, Salvi V, et al. Weight gain during long-term treatment of obsessive-compulsive disorder: a prospective comparison between serotonin reuptake inhibitors. *J Clin Psychiatry* 2004;65:1365-71.
  89. Michelson D, Amsterdam JD, Quitkin FM, et al. Changes in weight during a 1-year trial of fluoxetine. *Am J Psychiatry* 1999;156:1170-6.
  90. Fava M, Judge R, Hoog SL, et al. Fluoxetine versus sertraline and paroxetine in major depressive disorder: changes in weight with long-term treatment. *J Clin Psychiatry* 2000;61:863-7.
  91. Sussman N, Ginsberg DL, Bikoff J. Effects of nefazodone on body weight: a pooled analysis of selective serotonin reuptake inhibitor- and imipramine-controlled trials. *J Clin Psychiatry* 2001;62:256-60.
  92. Thase ME, Nierenberg AA, Keller MB, et al. Efficacy of mirtazapine for prevention of depressive relapse: a placebo-controlled double-blind trial of recently remitted high-risk patients. *J Clin Psychiatry* 2001;62:782-8.
  93. Versiani M, Moreno R, Ramakers-van Moorsel CJ, et al. Comparison of the effects of mirtazapine and fluoxetine in severely depressed patients. *CNS Drugs* 2005;19:137-46.
  94. Benkert O, Szegedi A, Kohnen R. Mirtazapine compared with paroxetine in major depression. *J Clin Psychiatry* 2000;61:656-63.
  95. Tandon R, Harrigan E, Zorn S. Ziprasidone: a novel antipsychotic with unique pharmacology and therapeutic potential. *J Serotonin Res* 1997;4:159-77.

## 8. Clinical evaluation of obese children and adolescents

Katherine M. Morrison, Jean-Pierre Chanoine

The initial assessment of the overweight child or adolescent has several components: identification of risk factors for the development of obesity; exclusion of a secondary cause of obesity; and determination of obesity-related comorbidities. Overweight is defined as a BMI between the 85th and 95th percentiles, and obesity as a BMI in the 95th percentile or higher for age and sex, based on the growth charts of the US Centers for Disease Control and Prevention (see Figs. 5 and 6 in chapter 4).<sup>1</sup>

### Identification of risk factors for the development of obesity

The assessment of an obese child requires a complete history and physical examination. A number of risk factors for obesity development have been identified in these guidelines, and enquiry relating to these should be included in the history of an obese child. Family history of obesity and obesity-related disorders, pregnancy history (maternal diabetes, pregnancy exposures, low birth weight) and infant feeding history should be ascertained. Patterns of physical activity (more than 2 hours per day of television or computer and video games, and low participation in physical activities) and nutritional intake (especially high sugared-drink intake, low fruit and vegetable intake, and disordered eating patterns) should be identified.

### Exclusion of a secondary cause of obesity

Obesity or overweight is a description of a phenotype rather than a diagnosis. Although endocrine and genetic causes of obesity are known, these are uncommon, and the vast majority of children will not have an identifiable endocrine or genetic cause of obesity. Endocrine causes of obesity in childhood are associated with attenuated linear growth (hypothyroidism, Cushing's disease, growth hormone deficiency) or a history of central nervous system (CNS) injury (hypopituitarism). Most children with idiopathic obesity are tall for their age and genetic expectations. Testing for endocrine disorders is unlikely to be useful unless the child has demonstrated reduced growth velocity, is shorter than expected based on the family background or has a history of CNS injury.

Obesity associated with a genetic syndrome is usually of early onset, is often associated with neurodevelopmental delay and may be associated with dysmorphic features (e.g., Prader-Willi syndrome and Laurence-Moon syndrome). Referral to a geneticist for the evaluation of an obese child is

usually not required unless there is marked obesity of early onset and associated abnormalities.

Progress in the understanding of appetite control has led to the identification of monogenic disorders resulting in obesity. The most common of these is caused by a mutation of the melanocortin-4-receptor (MC4R) and may be present in up to 10% of children with extreme obesity of early onset.<sup>2</sup> Causal therapy for this disorder is not yet available. Leptin deficiency is the only monogenic disorder for which a specific treatment is available (recombinant leptin replacement), but this deficiency is exceedingly rare (11 cases reported).<sup>3,4</sup> These syndromes are both associated with early-onset obesity and with hyperphagia that may include food craving, waking at night to eat and food-stealing behaviours.

Obesity may arise from the use of some medications, including glucocorticoids and newer antipsychotic drugs (e.g., risperidone and olanzapine).<sup>5,6</sup>

### Determination of obesity-related comorbidities

Many of the obesity-related comorbidities recognized in adulthood begin to develop in childhood:

- cardiovascular (hypertension)
- metabolic (dyslipidemia, dysglycemia, type 2 diabetes)
- psychosocial (poor self-esteem, depression)
- orthopedic (slipped capital femoral epiphysis, tibia vara [Blount disease], musculoskeletal discomfort)
- respiratory (obstructive sleep apnea)
- gastrointestinal (nonalcoholic fatty liver disease, cholelithiasis, gastroesophageal reflux)
- reproductive (polycystic ovary syndrome)
- renal (focal segmental glomerulosclerosis)

Although these disorders are prevalent in childhood, research into screening strategies remains scant or nonexistent. Furthermore, there is a lack of information on optimal interventions in children who have been identified with these comorbidities. Guidelines for the assessment of obesity-related health consequences are presented in Table 12. Guidance on screening for diabetes in obese children is provided in Box 4.

### Cardiovascular

Cardiovascular risk factors cluster in overweight and obese children<sup>7</sup> and persist from childhood into adulthood. Furthermore, cardiovascular risk factors in childhood are related to the development of atherosclerosis in adolescence.<sup>8</sup> Child-

hood obesity is also related to increased carotid intima-media thickness (a noninvasive measure of atherosclerosis)<sup>9,10</sup> and coronary artery disease<sup>11</sup> in adulthood.

Hypertension is a risk factor for atherosclerotic heart disease in adults and adolescents. Isolated systolic hypertension is the predominant form of hypertension associated with obesity.<sup>12</sup> Because blood pressure in childhood varies with age,

sex and height, hypertension is currently defined as systolic or diastolic blood pressure, measured repeatedly with an appropriate size of cuff (see Table 10 in chapter 6), that is at or above the 95th percentile for age, sex and height.<sup>13</sup> Population- and school-based studies show that blood pressure increases with increased BMI<sup>14-19</sup> in children and adolescents 5-18 years of age. In a recent review of 8 studies in the United States, the prevalence of systolic hypertension among the heaviest children (top quintile for BMI) was 6.6%-7.7% among children 5-12 years of age and 10.5%-11.5% among adolescents 13-17 years of age.<sup>17</sup> The relative risk for hypertension, confirmed with multiple measurements, among obese children in the United States was 3.26 (95% confidence interval [CI] 2.50-4.24),<sup>20</sup> comparable to earlier findings in a Quebec study in which the relative risk was 3.6 (95% CI 1.4-9.1) and 3.2 (95% CI 1.1-9.4) among boys and girls, respectively.<sup>21</sup>

Among obese children in clinic populations, 25%-32% have elevated systolic blood pressure and 4%-17% have elevated diastolic pressure.<sup>22,23</sup> Hypertension is often associated with other cardiovascular risk factors and with obstructive sleep apnea.<sup>19</sup> Focal segmental glomerulosclerosis has been reported in obese adolescents, and improvement with weight loss has been described.<sup>24,25</sup> However, insufficient studies are available to make a recommendation for screening.

## Metabolic

### Dyslipidemia

Dyslipidemia is reported with increasing frequency among obese children and adolescents. Low levels of high-density lipoprotein (HDL) cholesterol and elevated fasting triglyceride levels are the most common lipid abnormalities identified in obese children. Both are associated with the development of atherosclerosis and with adult cardiovascular disease, particularly when clustered with other cardiovascular risk factors. In population-based studies involving adolescents, low HDL cholesterol levels were noted in 39%-69% of obese children (the wide range is likely a result of variation in

**Table 12:** Assessment of obesity-related health consequences

Obesity-related health consequence	Proposed assessment
<b>Cardiovascular</b>	
• Hypertension*	• Serial blood pressure measurements performed in standardized manner
<b>Metabolic</b>	
• Dysglycemia or type 2 diabetes	If $\geq 10$ years of age:
• Dyslipidemia	
	• Fasting plasma glucose or oral glucose tolerance test if criteria met (see Box 4)
	• Fasting levels of total cholesterol, HDL cholesterol, triglycerides and LDL cholesterol (calculated)
<b>Respiratory</b>	
• Obstructive sleep apnea	• History: snoring; sleep-disordered breathing; early morning headaches; excess daytime fatigue
	• Consider sleep study if history consistent
<b>Gastrointestinal</b>	
• Nonalcoholic fatty liver disease	• History
	• Physical examination
• Gastroesophageal reflux	• Consider measuring ALT and AST levels; alkaline phosphatase; albumin as appropriate
• Gallstones	
<b>Orthopedic</b>	
• Slipped capital femoral epiphysis	• History
	• Physical examination
• Tibia vara (Blount disease)	
• Spondylolisthesis	
• Axial arthritis	
<b>Reproductive</b>	
• Polycystic ovary syndrome	• History: menstrual irregularity, secondary amenorrhea
	• Physical examination: hirsutism, acne
	• Consider measuring levels of free testosterone, luteinizing hormone and follicle stimulating hormone; abdominal ultrasound
<b>Psychosocial</b>	
• Depression	• History
• Low self-esteem	
• Binge-eating disorder	

Note: HDL = high-density lipoprotein; LDL = low-density lipoprotein, ALT = alanine aminotransferase, AST = aspartate aminotransferase.

\*Defined as systolic or diastolic blood pressure  $\geq 95$ th percentile based on age, sex and height.<sup>13</sup>

#### Box 4: Canadian Diabetes Association criteria for screening for diabetes in obese children

Obese children  $\geq 10$  years of age should be considered for screening for type 2 diabetes every 2 years using a fasting plasma glucose test if they meet 2 of the following criteria:

- Member of a high-risk ethnic group
- Family history of type 2 diabetes, especially if the child was exposed to diabetes in utero
- Acanthosis nigricans
- Polycystic ovarian syndrome
- Hypertension
- Dyslipidemia

An oral glucose tolerance test may also be considered as a screening test

Source: Canadian Diabetes Association 2003 clinical practice guidelines.<sup>38</sup>

the definition of “low”).<sup>26–28</sup> Children in obesity clinics have been found to have low HDL cholesterol levels in 10%–22% of cases (although the criteria for diagnosis are more stringent than those used in the population-based studies described earlier in this chapter).<sup>22,23</sup> The relative risk of low HDL cholesterol was higher among obese girls than among obese boys (odds ratio [OR] 9.1 [95% CI 3.5–23.8] and 2.8 [95% CI 1.1–7.5]), respectively, compared with normal-weight children.<sup>21</sup> Elevated levels of low-density lipoprotein (LDL) cholesterol have been observed more commonly in obese than in normal-weight children<sup>21,23</sup> (OR 2.8 [95% CI 1.1–7.5] and 5.6 [95% CI 2.1–15.0] for boys and girls, respectively).

Given the increased prevalence of dyslipidemia among obese children and its association with subsequent atherosclerosis, screening obese children over 10 years of age should be considered.<sup>29</sup> Consideration may be given to screening younger children with a family history of premature coronary artery disease (in male relatives < 55 years and in female relatives < 65 years).<sup>30</sup> Although lipid indices vary somewhat with age, for simplification, cutoff points that indicate abnormal levels for HDL cholesterol (< 0.9 mmol/L), fasting triglycerides (< 1.69 mmol/L) and LDL cholesterol (2.85 mmol/L) have been recommended for boys and girls up to 18 years of age.<sup>29</sup> These values are consistent with the 5th–10th percentiles for HDL cholesterol, the 95th percentile for triglycerides and the 90th–95th percentiles for LDL cholesterol levels according to the Lipid Research Clinics’ database.<sup>31</sup> Although the ideal frequency of repeat screening is unknown, given the influence of puberty on lipid levels, repeat testing in 2 years should be considered if obesity persists.

### Abnormalities of glucose metabolism

Identifiable disturbances in glucose metabolism include abnormal fasting plasma glucose level, disturbed response to a glucose load (impaired glucose tolerance) and type 2 diabetes. All of these conditions are more common in obese children. In addition, hyperinsulinemia, consistent with insulin resistance, has been identified in a large proportion of obese children in population-based studies<sup>14</sup> and in up to 40% of children in a clinic-based study.<sup>22</sup>

Type 2 diabetes can present in children and adolescents and is particularly prevalent in specific ethnic groups (Aboriginal, African, Hispanic and Asian people) in the United States. Increased prevalence among Canadian First Nations children and adolescents<sup>32–34</sup> has been identified, but comprehensive studies in other ethnic groups in Canada are not available.

Dysglycemia includes either impaired fasting glucose or impaired glucose tolerance and is associated with an increased risk of type 2 diabetes in adults. Longitudinal follow-up studies involving children with dysglycemia are not yet available. Impaired fasting glucose (fasting plasma glucose level 5.6–7.0 mmol/L) is present in 7.0% of US adolescents and in 17.8% of obese adolescents.<sup>35</sup> Impaired glucose tolerance, measured during a 2-hour glucose tolerance test, has been examined largely in clinical settings. Among the children tested, impaired glucose tolerance (2-hour glucose level

7.8–11.0 mmol/L) was much more common than impaired fasting glucose (fasting plasma glucose level 6.1–6.9 mmol/L) (12%–27% v. 0%–1%).<sup>22,23</sup> Impaired glucose tolerance was more likely among children with additional risk factors for abnormalities in glucose metabolism (36%).<sup>36,37</sup>

Currently, the Canadian Diabetes Association 2003 clinical practice guidelines<sup>38</sup> and the American Diabetes Association position statement<sup>39</sup> recommend measuring the fasting plasma glucose level to screen for type 2 diabetes in obese children 10 years of age or older who have 2 of the following risk factors: high-risk ethnic group; family history of type 2 diabetes, especially if in utero exposure; acanthosis nigricans; polycystic ovary syndrome; hypertension; and dyslipidemia. However, impaired fasting glucose level may be insufficient to identify dysglycemia. Longitudinal studies to examine the usefulness of hyperinsulinemia and the prognosis of dysglycemia in childhood or adolescence are not yet available to assist in understanding the clinical significance of these disorders. In adults, dysglycemia predicts increased risk for type 2 diabetes and cardiovascular disease, and intervention can reduce rates of progression to type 2 diabetes.

### Clustering of cardiovascular risk factors in childhood obesity

Criteria for “metabolic syndrome” in children and adolescents have not been established, because normal values for all elements of the metabolic syndrome vary with age and sex. Therefore, it is perhaps not surprising that, of the 13 studies that examined metabolic syndrome in youth, identical criteria were used in only 2: NHANES III<sup>26</sup> and NHANES 1999/2000.<sup>28</sup>

In studies that used the presence of 3 of 5 criteria, metabolic syndrome was present in 4.2%–6.4% of US adolescents and in 19.5%–32.1% of obese adolescents.<sup>26,28</sup> Using similar criteria (although somewhat less stringent for dyslipidemia), about 13% of a representative sample of Quebec adolescents had metabolic syndrome.<sup>40</sup> Children and adolescents in obesity clinics typically had a greater prevalence of metabolic syndrome: 30% and 49.7% of children in the United Kingdom and United States, respectively. The rates increased from 38.7% to 49.7% with increasing degree of obesity (BMI z-score > 2.0 and 2.5, respectively) in the United States,<sup>36</sup> but not in the United Kingdom.<sup>22</sup>

It remains clear that, as clustering of cardiovascular risk factors occurs in obese children, it is important to identify comorbidities in these children.

### Other health consequences of childhood obesity

Obstructive sleep apnea may be 4–5 times more likely to occur in obese children than in lean children, although this estimate is based on limited data.<sup>41–43</sup> Although no proven effective screening method has been established, recent data suggest that a history of snoring, daytime somnolence and learning disability may predict sleep apnea in obese children.<sup>44</sup>

Gastrointestinal morbidities associated with childhood obesity include nonalcoholic fatty liver disease, cholestatic



liver disease and gastroesophageal reflux. Although the prevalence of nonalcoholic fatty liver disease is difficult to determine, magnetic resonance imaging and ultrasound studies suggest that it might be as high as 45% among obese adolescents.<sup>45-47</sup> Liver transaminase levels are not elevated in all children with nonalcoholic fatty liver disease, but they are usually elevated in those with severe disease.<sup>45</sup> Polycystic ovary syndrome often accompanies insulin resistance and is seen in obese adolescent females. The presence of polycystic ovary syndrome should be considered in adolescent females with dyslipidemia, dysglycemia and irregular menses or clinical evidence of hyperandrogenemia.<sup>48</sup> Orthopedic problems in obese children include slipped capital femoral epiphysis, coxa vara, Blount disease (tibia vara), Legg-Calvé-Perthes disease and back pain.

Psychosocial disturbances, including low self-esteem<sup>49</sup> and depression, in children and adolescents with obesity have been reported, but the community prevalence has not been well described. Clinic-based studies suggest that obese children may have compromised peer relationships<sup>50</sup> and may suffer from social isolation and depression. However, 2 recent reviews suggest that obese children seeking treatment have poorer psychological well-being than community samples, which may be an important reason for presentation to a clinic.<sup>51,52</sup> Further discussion of these aspects is included in chapter 7.

## Recommendations

1. We recommend that the clinical evaluation of overweight and obese children include a history and a general physical examination to exclude secondary (endocrine or syndrome-related) causes of obesity and obesity-related health risks and complications [*grade A, level 3*<sup>53,54</sup>].
2. We recommend measuring fasting plasma glucose level and determining lipid profile, including total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol and ratio of total cholesterol to HDL cholesterol in children aged 10 years and older [*grade B, level 3*<sup>29,53</sup>]. We suggest repeating these tests at regular intervals as needed [*grade C, level 4*].
3. The screening criteria for obesity-related health consequences should be assessed in the clinical setting for sensitivity, specificity and clinical value in improving the health of children with obesity [*grade C, level 4*].
4. Investigation of the prevalence of the health consequences of obesity in childhood should be undertaken in diverse populations and should include longitudinal studies to examine the prognosis of hyperinsulinemia, impaired glucose tolerance, impaired fasting glucose level and the clustering of cardiovascular risk factors [*grade C, level 4*].
5. Studies to determine optimal intervention strategies for children with established health consequences related to obesity are urgently required [*grade C, level 4*].

From the Department of Pediatrics, McMaster University, Hamilton, Ont. (Morrison); and the Department of Pediatrics, University of British Columbia, Vancouver, BC (Chanoine)

**Competing interests:** None declared.

## REFERENCES

1. Kuczmarski RJ, Ogden CL, Guo SS, et al. 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat 11* 2002;(246):1-190.
2. Farooqi IS, Keogh JM, Yeo GS, et al. Clinical spectrum of obesity and mutations in the melanocortin 4 receptor gene. *N Engl J Med* 2003;348:1085-95.
3. Farooqi IS, Jebb SA, Langmack G, et al. Effects of recombinant leptin therapy in a child with congenital leptin deficiency. *N Engl J Med* 1999;341:879-84.
4. Montague CT, Farooqi IS, Whitehead JP, et al. Congenital leptin deficiency is associated with severe early-onset obesity in humans. *Nature* 1997;387:903-8.
5. Aman MG, Arnold LE, McDougle CJ, et al. Acute and long-term safety and tolerability of risperidone in children with autism. *J Child Adolesc Psychopharmacol* 2005;15:869-84.
6. Vieweg WV, Sood AB, Pandurangi A, et al. Newer antipsychotic drugs and obesity in children and adolescents. How should we assess drug-associated weight gain? *Acta Psychiatr Scand* 2005;111:177-84.
7. Srinivasan SR, Bao W, Wattigney WA, et al. Adolescent overweight is associated with adult overweight and related multiple cardiovascular risk factors: the Bogalusa Heart Study. *Metabolism* 1996;45:235-40.
8. Berenson GS, Srinivasan SR, Bao W, et al. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *N Engl J Med* 1998;338:1650-6.
9. Davis PH, Dawson JD, Riley WA, et al. Carotid intimal-medial thickness is related to cardiovascular risk factors measured from childhood through middle age: the Muscatine Study. *Circulation* 2001;104:2815-9.
10. Li S, Chen W, Srinivasan SR, et al. Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa Heart Study. *JAMA* 2003;290:2271-6.
11. Must A, Jacques PF, Dallal GE, et al. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. *N Engl J Med* 1992;327:1350-5.
12. Sorof JM, Poffenbarger T, Franco K, et al. Isolated systolic hypertension, obesity, and hyperkinetic hemodynamic states in children. *J Pediatr* 2002;140:660-6.
13. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics* 2004;114(2 Suppl):555-76.
14. Freedman DS, Dietz WH, Srinivasan SR, et al. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics* 1999;103:1175-82.
15. Morrison JA, Sprecher DL, Barton BA, et al. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *J Pediatr* 1999;135:458-64.
16. Paradis G, Lambert M, O'Loughlin J, et al. Blood pressure and adiposity in children and adolescents. *Circulation* 2004;110:1832-8.
17. Rosner B, Prineas R, Daniels SR, et al. Blood pressure differences between blacks and whites in relation to body size among US children and adolescents. *Am J Epidemiol* 2000;151:1007-19.
18. Sorof J, Daniels S. Obesity hypertension in children: a problem of epidemic proportions. *Hypertension* 2002;40:441-7.
19. Marcus CL, Greene MG, Carroll JL. Blood pressure in children with obstructive sleep apnea. *Am J Respir Crit Care Med* 1998;157:1098-103.
20. Sorof JM, Lai D, Turner J, et al. Overweight, ethnicity, and the prevalence of hypertension in school-aged children. *Pediatrics* 2004;113:475-82.
21. Katzmarzyk PT, Tremblay A, Perusse L, et al. The utility of the international child and adolescent overweight guidelines for predicting coronary heart disease risk factors. *J Clin Epidemiol* 2003;56:456-62.
22. Viner N, Segal TY, Lichtarowicz-Krynska E, et al. Prevalence of the insulin resistance syndrome in obesity. *Arch Dis Child* 2005;90:10-4.
23. Invitti C, Guzzaloni G, Gilardini L, et al. Prevalence and concomitants of glucose intolerance in European obese children and adolescents. *Diabetes Care* 2003;26:118-24.
24. Adelman RD, Restaino IG, Alon US, et al. Proteinuria and focal segmental glomerulosclerosis in severely obese adolescents. *J Pediatr* 2001;138:481-5.
25. Mochizuki H, Joh K, Matsuyama N, et al. Focal segmental glomerulosclerosis in a patient with Prader-Willi syndrome. *Clin Nephrol* 2000;53:212-5.
26. Cook S, Weitzman M, Auinger P, et al. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. *Arch Pediatr Adolesc Med* 2003;157:821-7.
27. Goodman E, Daniels SR, Morrison JA, et al. Contrasting prevalence of and demographic disparities in the World Health Organization and National Cholesterol Education Program Adult Treatment Panel III definitions of metabolic syndrome among adolescents. *J Pediatr* 2004;145:445-51.
28. Duncan GE, Li SM, Zhou XH. Prevalence and trends of a metabolic syndrome phenotype among U.S. Adolescents, 1999-2000. *Diabetes Care* 2004;27:2438-43.
29. Kavey RE, Daniels SR, Lauer RM, et al. American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood. *Circulation* 2003;107:1562-6.
30. American Academy of Pediatrics. Committee on Nutrition. Cholesterol in childhood. *Pediatrics* 1998;101:141-7.
31. Lipid Research Clinics Population Studies Program. *The Lipid Research Clinics Population Studies data book, volume 1: the prevalence study*. Bethesda (MD): US Department of Health and Human Services; 1980.
32. Dean HJ, Mundy RL, Moffatt M. Non-insulin-dependent diabetes mellitus in Indian children in Manitoba. *CMAJ* 1992;147(1):52-7.

33. Delisle HF, Ekoe JM. Prevalence of non-insulin-dependent diabetes mellitus and impaired glucose tolerance in two Algonquin communities in Quebec. *CMAJ* 1993; 148:41-7.
34. Harris SB, Perkins BA, Whalen-Brough E. Non-insulin-dependent diabetes mellitus among First Nations children. New entity among First Nations people of north western Ontario. *Can Fam Physician* 1996;42:869-76.
35. Williams DE, Cadwell BL, Cheng YJ, et al. Prevalence of impaired fasting glucose and its relationship with cardiovascular disease risk factors in US adolescents, 1999–2000. *Pediatrics* 2005;116:1122-6.
36. Weiss R, Dziura J, Burgert TS, et al. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med* 2004;350:2362-74.
37. Wiegand S, Maikowski U, Blankenstein O, et al. Type 2 diabetes and impaired glucose tolerance in European children and adolescents with obesity — a problem that is no longer restricted to minority groups. *Eur J Endocrinol* 2004;151:199-206.
38. Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. Canadian Diabetes Association 2003 clinical practice guidelines for the prevention and management of diabetes in Canada. *Can J Diabetes* 2003;27(Suppl 2):S1-152.
39. American Diabetes Association. Screening for type 2 diabetes [position statement]. *Diabetes Care* 2004;27(Suppl 1):S11-4.
40. Lambert M, Paradis G, O'Loughlin J, et al. Insulin resistance syndrome in a representative sample of children and adolescents from Quebec, Canada. *Int J Obes Relat Metab Disord* 2004;28:833-41.
41. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med* 2002;165:1217-39.
42. Redline S, Tishler PV, Schluchter M, et al. Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. *Am J Respir Crit Care Med* 1999;159:1527-32.
43. Marcus CL, Curtis S, Koerner CB, et al. Evaluation of pulmonary function and polysomnography in obese children and adolescents. *Pediatr Pulmonol* 1996;21: 176-83.
44. Goodwin JL, Kaemingk KL, Mulvaney SA, et al. Clinical screening of school children for polysomnography to detect sleep-disordered breathing: The Tucson Children's Assessment of Sleep Apnea Study (TuCASA). *J Clin Sleep Med*. 2005;1: 247-54.
45. Fishbein MH, Miner M, Mogren C, et al. The spectrum of fatty liver in obese children and the relationship of serum aminotransferases to severity of steatosis. *J Pediatr Gastroenterol Nutr* 2003;36:54-61.
46. Arslan N, Buyukgebiz B, Ozturk Y, et al. Fatty liver in obese children: prevalence and correlation with anthropometric measurements and hyperlipidemia. *Turk J Pediatr* 2005;47:23-7.
47. Guzzaloni G, Grugni G, Minocci A, et al. Liver steatosis in juvenile obesity: correlations with lipid profile, hepatic biochemical parameters and glycemic and insulinemic responses to an oral glucose tolerance test. *Int J Obes Relat Metab Disord* 2000;24:772-6.
48. Kent SC, Legro RS. Polycystic ovary syndrome in adolescents. *Adolesc Med* 2002; 13:73-88, vi.
49. Strauss RS. Childhood obesity and self-esteem. *Pediatrics* 2000;105:e15.
50. Strauss RS, Pollack HA. Social marginalization of overweight children. *Arch Pediatr Adolesc Med* 2003;157:746-52.
51. Flodmark CE. The happy obese child. *Int J Obes (Lond)* 2005;29(Suppl 2):S31-3.
52. Wardle J, Cooke L. The impact of obesity on psychological well-being. *Best Pract Res Clin Endocrinol Metab* 2005;19:421-40.
53. Daniels SR, Arnett DK, Eckel RH, et al. Overweight in children and adolescents: pathophysiology, consequences, prevention, and treatment. *Circulation* 2005;111: 1999-2012.
54. *Clinical practice guidelines for the management of overweight and obesity in children and adolescents*. Canberra, Australia: National Health and Medical Research Council; 2003. Available: [www.health.gov.au/internet/wcms/Publishing.nsf/Content/obesityguidelines-guidelines-children.htm/\\$FILE/children.pdf](http://www.health.gov.au/internet/wcms/Publishing.nsf/Content/obesityguidelines-guidelines-children.htm/$FILE/children.pdf) (accessed 2007 Jan 31).

# 9. Role of the health care team in the evaluation and management of obesity

Robert Dent, Michael Vallis, Irene Hramiak, Judith A. Francis

**O**besity is a chronic medical condition requiring long-term intervention of 3 main types: lifestyle modification, medication and surgery. Some physicians believe they lack the time, confidence or expertise to provide ongoing obesity counselling and monitoring in addition to medical care. In such cases, a multidisciplinary approach to the management of the obese person may be especially beneficial. A multidisciplinary health care team can assist the overweight or obese person with both medical and behavioural approaches to weight management.

The team approach can be used with individuals or with groups. There is some evidence that attendance in group activities reinforces behaviour and commitment to weight loss and maintenance and, ultimately, to success. Forming a team or being part of a team may be a benefit in the ongoing care of a large group of obese patients.<sup>1,2</sup> The most successful treatment programs appear to include the following elements:

- Screening and assessment of medical risk, targeting people with obesity-related diseases<sup>3</sup>
- Treatment with lifestyle modification (education in decreasing food intake, eating a healthy diet and increasing physical activity; behaviour modification; stimulus control techniques; and techniques for increasing motivation); medication; and surgery to modify gastrointestinal physiology to reduce capacity for food intake and nutrient absorption
- Long-term monitoring and ongoing follow-up

## Physicians' barriers to weight management

Research suggests that few physicians are discussing weight matters with their patients, let alone implementing a treatment program that contains these components. A literature search suggests that only about 40% of obese people receive recommendations from their physicians regarding weight loss and weight-loss maintenance even if they have comorbidities (e.g., diabetes and hypertension).<sup>4-9</sup> Such recommendations are made even less frequently for overweight people, even if they have diabetes and hypertension.<sup>4-9</sup> In addition, the medical profession is failing to counsel young, disease-free adults and those in lower socioeconomic groups. We are missing important opportunities for primary prevention. Furthermore, we are making little progress in this regard. Data gathered for large, cross-sectional studies (the Behavioural Risk Factor Surveillance System) for 1990, 1996 and 2000 suggest that there was no increase in the proportion of obese patients receiving recommendations regarding weight loss during the 1990s.

## Attitudes toward overweight and obesity

Obese people are subject to negative bias by members of society in general and the medical profession in particular. A comparison of survey studies before and after 1999 suggests that this bias is not decreasing, at least among health care professionals. Bias, prejudice and negative attitudes of health professionals toward obesity remain a major barrier in the care of obese individuals. Overweight people may be reluctant to seek health care because of fear of scolding or humiliation. Three studies meeting project criteria showed that obese women delayed seeking health care and important screening examinations. Adams and colleagues<sup>10</sup> found that obese women were less likely than non-obese women to have routine pelvic examinations, and Fontaine and coworkers<sup>11</sup> found that they were less likely to have breast examinations. This is especially disturbing, since breast and uterine cancers are more prevalent among obese women. Olson and others<sup>12</sup> reported that obese women were about 4 times more likely to delay or cancel health care visits because of weight concerns.

Between 1969 and 1987, a number of studies revealed that both physicians and medical students viewed obese people as unintelligent, unsuccessful, inactive and weak-willed.<sup>13-16</sup> Physicians lacked compassion for obese people and preferred not to treat them. Health practitioners tended to blame the person for the obesity, considering it a self-induced disease. According to surveys, physicians, medical students, nurses and dietitians also showed overt bias against the overweight and obese person. More recent studies involving health professionals and patients reported much less overt bias, but bias was still present.<sup>17-27</sup>

Researchers have also investigated patient reports of practitioner attitudes.<sup>28-31</sup> In 1990, Rand and Macgregor<sup>28</sup> found that nearly 80% of bariatric surgery patients felt that they had been treated disrespectfully by members of the medical profession. Ten years later, Anderson and colleagues<sup>31</sup> used a similar questionnaire to ask bariatric surgery patients about their care and concluded that, although doctor-patient interactions concerning weight had improved in the last decade, there was still much room for improvement.

## Inadequate skills and training

A number of studies surveying physicians, nurses, dietitians and students majoring in exercise have shown that health professionals' education regarding obesity and its management is deficient. During the last decade, 8 studies revealed

that health practitioners had inadequate or even no training in the causes and treatment of obesity. Kushner<sup>32</sup> found that lack of training in counselling and lack of knowledge about nutrition were major barriers preventing American primary care physicians from providing nutrition counselling to patients. Kristeller and Hoerr<sup>21</sup> investigated physician beliefs, attitudes and practices regarding obesity in relation to medical risk, management, interest in training and other resources for 6 specialties (family practice, internal medicine, gynecology, endocrinology, cardiology and orthopedics). Only 4.5% of family practitioners and 36.4% of endocrinologists reported “any specialty training related to obesity.” Price and coworkers<sup>33</sup> found a need for improved education for pediatricians. Among internal medicine residents in 2 university-based residency training programs, Block and others<sup>34</sup> found good knowledge of obesity risks, but poor skills, attitude and knowledge about obesity. They concluded that residency training programs must address these issues. Similar results have been found in Israel<sup>18</sup> and Australia.<sup>19,20</sup> Furthermore, in its guidelines for obesity assessment and management in primary care, the American Medical Association has identified inadequate training and lack of training mechanisms for physicians in the medical management of obesity as barriers to obesity management.<sup>35</sup>

### **Lack of reimbursement for providing treatment**

The American Medical Association has also identified lack of payment by most health insurance and managed-care plans for obesity-related treatment programs as a barrier to obesity management.<sup>35</sup> There is less coverage for the treatment of obesity than for the care of other chronic medical conditions.

It is critical that all physician barriers to counselling are identified and addressed.<sup>6-8</sup>

### **Patient barriers to initiating weight treatment**

In addition to physician barriers, a number of patient-related barriers might make it difficult for the physician to initiate and implement weight-loss counselling.

Individuals may be reluctant to seek medical attention. As noted earlier, obese women tend to delay health care visits and important screening examinations,<sup>10-12</sup> especially breast and pelvic examinations.

Overweight people may have depression and other psychosocial barriers to seeking, initiating or continuing treatment for obesity.

Overweight and obese people, especially those with binge-eating disorder, lack self-efficacy. Self-efficacy refers to confidence in one's ability to do what is required to produce the desired outcome. In the context of weight management, this involves confidence in one's ability to resist food in high-risk situations and to proceed with physical activity in adverse conditions. Self-efficacy has been shown to predict outcomes in the context of a variety of addictive behaviours (e.g., smoking) and is thought to play an important

role in weight management. Three studies meeting project criteria<sup>36-38</sup> found low self-efficacy ratings among people with binge-eating disorder.

### **The health professional's role: making recommendations**

Studies have shown that the simplest thing a physician or other health professional can do to help a person initiate lifestyle modifications is to recommend that the person do so.<sup>4,39-44</sup> Evidence suggests that most people would be more likely to engage in health promotion behaviours if their physician recommended them. Even simple health promotion recommendations about weight have been shown to be effective in motivating patients to start healthier diets and exercise to achieve weight loss and weight-loss maintenance. For instance, 77.5% of people who reported receiving medical advice in the previous year to lose weight said they were trying to lose weight, compared with 33.4% of those who did not receive advice on weight management.<sup>4</sup> Significant improvements have also been shown in losing weight, undertaking regular exercise and decreasing alcohol intake 1 year after brief preventive intervention by primary care physicians.<sup>41</sup> Written personalized exercise prescriptions,<sup>40</sup> physician recommendations for exercise<sup>6</sup> and tailored nutrition messages<sup>42</sup> have all been found to improve weight treatment behaviour in the short term.

Although some people may be able to make significant lifestyle changes on their own following a simple recommendation to lose weight from their physician, others may require more information, structure, ongoing monitoring and support. In such cases, tools are available to help the physician implement a treatment program consisting of the components outlined earlier.

### **Motivational interviewing**

Motivational interviewing, pioneered by Miller and Rollnick,<sup>45</sup> has its roots in alcohol abuse counselling. It is an approach to counselling that is geared toward increasing an individual's motivation, or buy-in, to the work that needs to be done to reduce substance dependence. Miller and Rollnick offered the approach as a brief intervention (hence some confusion with the term minimal intervention). Motivational interviewing is guided by the 6 “mediators” or “ingredients” of change (referred to with the mnemonic FRAMES):

- Feedback of personal risk or impairment
- Emphasis on personal Responsibility for change
- Clear Advice to change
- A Menu of alternative change options
- Therapist Empathy
- Facilitation of client Self-efficacy or optimism

These ingredients are delivered by the clinician using the following principles: express empathy, develop discrepancy, avoid argumentation, roll with resistance and support self-efficacy. Research evaluating motivational interviewing suggests that it might improve outcomes for people attempting weight loss and maintenance.<sup>46-48</sup>

Attempts to evaluate the efficacy of training counsellors in the use of motivational interviewing has taken place mainly within the addictions counselling area. Results have been mixed, with reports of both marginal and substantial improvements in motivational interviewing skills among health professionals receiving training.<sup>49-51</sup>

Physicians may be able to provide effective behavioural and lifestyle modification counselling using a structured treatment manual, such as the LEARN Program for Weight Control ([www.LearnEducation.com](http://www.LearnEducation.com)).<sup>52</sup> Topics covered in this manual include behaviour modification and stimulus control techniques, increasing programmed and lifestyle activities, nutritional information and relationship issues that may affect weight loss and maintenance. The LEARN program is an active approach that requires patients to complete regular homework assignments as well as monitor their food intake and exercise.

## Role of multidisciplinary team members in managing obesity

### Medical practitioner

The medical practitioner may be a nurse, family physician or medical specialist. His or her role includes assessing the person's health status and risk factors. This medical practitioner assesses clinical parameters, such as blood pressure, waist circumference, weight and BMI. If medications are being used, the practitioner could be responsible for assessing response and any side effects. If bariatric surgery is indicated, he or she would be responsible for the decision, referral or performance of the surgery and follow-up. The medical practitioner will be involved in goal setting (small achievable goals for weight, activity and health status). He or she will follow clinical parameters and assess laboratory values over time to manage risk factors.

### Psychologist

Psychologists play a role in the management of obesity by assessing a person's motivation to engage in weight loss. They will then tailor treatment strategies to the person's readiness to change. Psychologists help people challenge the attitudes and beliefs that hinder weight loss and maintenance. They introduce behaviour modification techniques and help individuals modify their environment to facilitate weight loss and maintenance. Psychologists are also qualified to diagnose and treat mental health problems (e.g., depression, anxiety, eating disorders) that may interfere with weight loss and maintenance. In addition, psychologists help individuals identify and work through relationship issues that may be connected to unhealthy eating habits.

### Dietitian

Dietitians provide an individualized diet based on principles of healthy eating. This diet takes into account the person's

unique life circumstances, including cultural preferences, age, socioeconomic status and overall health. Dietitians provide information about nutritional and energy content of various foods. They teach the person about portion control, cooking techniques and food purchasing. They encourage the person to monitor his or her food intake, then assess intake and barriers to weight loss during ongoing assessment.

### Exercise professionals

Exercise professionals include kinesiologists (certified by the provincial kinesiology association and with a bachelor's degree in human kinetics or kinesiology) and personal trainers (certified by Can-Fit-Pro, the American Council on Exercise or the American College of Sports Medicine). Exercise professionals play a role in the management of obesity by undertaking an initial fitness assessment, then providing ongoing guidance, supervision and support to individuals as they work toward increasing their level of physical activity. In addition, these professionals provide practical advice on how to incorporate activity into daily life and prevent injury.

Other models for the multidisciplinary approach to obesity management have also been described.<sup>53</sup>

## Recommendations

1. We recommend a comprehensive healthy lifestyle intervention for overweight and obese people [*grade A, level 1*]. We suggest that members of the health care team discuss with those willing to participate in weight management programs appropriate education, support and therapy as adjuncts to lifestyle interventions [*grade B, level 2*].
2. Primary care health professionals are encouraged to work with other health care team members to develop a comprehensive weight management program for the overweight or obese person to promote and maintain weight loss [*grade C, level 3*].
3. Primary care health professionals are encouraged to create a nonjudgmental atmosphere when discussing weight management [*grade C, level 4*].
4. Health care professionals are encouraged to consider the barriers people might have concerning obesity and its management [*grade C, level 4*].
5. Research should be undertaken to develop and evaluate the organization of care for overweight and obese people and to determine the cost-effectiveness of these strategies [*grade C, level 4*].
6. Undergraduate curricula and education for graduate health practitioners should be improved to fulfill knowledge, skills and attitude goals with respect to management and prevention of obesity [*grade C, level 4*].

From the Weight Management Clinic, Ottawa Hospital, Ottawa, Ont. (Dent); the Queen Elizabeth II Health Sciences Centre, Dalhousie University, Halifax, NS (Vallis); the Department of Medicine, University of Western Ontario, and the Lawson Diabetes Centre, St. Joseph's Health Care, London, Ont. (Hramiak); and the Cardiac Rehabilitation and Secondary Prevention Program, London Health Sciences Centre, London, Ont. (Francis)

**Competing interests:** None declared for Judith Francis and Michael Vallis. Robert Dent has ongoing paid consultancies with AstraZeneca Advisory Board for Galida (now ended) and Abbott Advisory Board for Meridia. He has received speaker fees and educational grants from Abbott Laboratories Ltd., GlaxoSmithKline Inc. and Novartis, and travel assistance from Abbott Laboratories Ltd. to attend a meeting. Irene Hramiak is a consultant to GlaxoSmithKline Inc. and is on a National Advisory Board for Abbott Laboratories Ltd., Eli Lilly, Novo Nordisk, sanofi-aventis Canada Inc. and GlaxoSmithKline Inc. She has received honoraria for speaking engagements from Merck Frosst Canada Ltd., GlaxoSmithKline Inc. and Novo Nordisk and has received a travel grant from Novo Nordisk.

## REFERENCES

- The obesity epidemic: a mandate for the multidisciplinary approach. Proceedings of a roundtable. Boston, Massachusetts, USA. October 27, 1997. *J Am Diet Assoc* 1998;98(10 Suppl 2):S1-61.
- Heshka S, Greenway F, Anderson JW, et al. Self-help weight loss versus a structured commercial program after 26 weeks: a randomized controlled study. *Am J Med* 2000;109:282-7.
- Douketis JD, Feightner JW, Attia J, et al. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. Canadian Task Force on Preventive Health Care. *CMAJ* 1999;160:513-25.
- Sciamanna CN, Tate DF, Lang W, et al. Who reports receiving advice to lose weight? Results from a multistate survey. *Arch Intern Med* 2000;160:2334-9.
- Friedman C, Brownson RC, Peterson DE, et al. Physician advice to reduce chronic disease risk factors. *Am J Prev Med* 1994;10:367-71.
- Galuska DA, Will JC, Serdula MK, et al. Are health care professionals advising obese patients to lose weight? *JAMA* 1999;282:1576-8.
- Mokdad AH, Bowman BA, Ford ES, et al. The continuing epidemics of obesity and diabetes in the United States. *JAMA* 2001;286:1195-200.
- Bramlage P, Wittchen HU, Pittrow D, et al. Recognition and management of overweight and obesity in primary care in Germany. *Int J Obes Relat Metab Disord* 2004;28:1299-308.
- Wee CC, McCarthy EP, Davis RB, et al. Physician counseling about exercise. *JAMA* 1999;282:1583-8.
- Adams CH, Smith NJ, Wilbur DC, et al. The relationship of obesity to the frequency of pelvic examinations: do physician and patient attitudes make a difference? *Women Health* 1993;20:45-57.
- Fontaine KR, Faith MS, Allison DB, et al. Body weight and health care among women in the general population. *Arch Fam Med* 1998;7:381-4.
- Olson CL, Schumaker HD, Yawn BP. Overweight women delay medical care. *Arch Fam Med* 1994;3:888-92.
- Maddox GL, Liederma V. Overweight as a social disability with medical implications. *J Med Educ* 1969;44:214-20.
- Blumberg P, Mellis LP. Medical students' attitudes toward the obese and morbidly obese. *Int J Eat Disord* 1985;4(2):169-75.
- Klein D, Najman J, Kohrman AF, et al. Patient characteristics that elicit negative responses from family physicians. *J Fam Pract* 1982;14:881-8.
- Price JH, Desmond SM, Krol RA, et al. Family practice physicians' beliefs, attitudes, and practices regarding obesity. *Am J Prev Med* 1987;3:339-45.
- Hoppe R, Ogden J. Practice nurses' beliefs about obesity and weight related interventions in primary care. *Int J Obes Relat Metab Disord* 1997;21:141-6.
- Fogelman Y, Vinker S, Lachter J, et al. Managing obesity: a survey of attitudes and practices among Israeli primary care physicians. *Int J Obes Relat Metab Disord* 2002;26:1393-7.
- Campbell K, Crawford D. Management of obesity: attitudes and practices of Australian dietitians. *Int J Obes Relat Metab Disord* 2000;24:701-10.
- Campbell K, Engel H, Timperio A, et al. Obesity management: Australian general practitioners' attitudes and practices. *Obes Res* 2000;8:459-66.
- Kristeller JL, Hoerr RA. Physician attitudes toward managing obesity: differences among six specialty groups. *Prev Med* 1997;26:542-9.
- Teachman BA, Brownell KD. Implicit anti-fat bias among health professionals: Is anyone immune? *Int J Obes Relat Metab Disord* 2001;25:1525-31.
- Chambliss HO, Finley CE, Blair SN. Attitudes toward obese individuals among exercise science students. *Med Sci Sports Exerc* 2004;36:468-74.
- Harvey EL, Glenny A, Kirk SF, et al. Improving health professionals' management and the organisation of care for overweight and obese people. *Cochrane Database Syst Rev* 2001; (2):CD000984.
- Maiman LA, Wang VL, Becker MH, et al. Attitudes toward obesity and the obese among professionals. *J Am Diet Assoc* 1979;74:331-6.
- Oberrieder H, Walker R, Monroe D, et al. Attitude of dietetics students and registered dietitians toward obesity. *J Am Diet Assoc* 1995;95:914-6.
- McArthur LH, Ross JK. Attitudes of registered dietitians toward personal overweight and overweight clients. *J Am Diet Assoc* 1997;97:63-6.
- Rand CS, Macgregor AM. Morbidly obese patients' perceptions of social discrimination before and after surgery for obesity. *South Med J* 1990;83:1390-5.
- Wadden TA, Anderson DA, Foster GD, et al. Obese women's perceptions of their physicians' weight management attitudes and practices. *Arch Fam Med* 2000;9:854-60.
- Hebl MR, Xu J, Mason MF. Weighing the care: patients' perceptions of physician care as a function of gender and weight. *Int J Obes Relat Metab Disord* 2003;27:269-75.
- Anderson C, Peterson CB, Fletcher L, et al. Weight loss and gender: an examination of physician attitudes. *Obes Res* 2001;9:257-63.
- Kushner RF. Barriers to providing nutrition counseling by physicians: a survey of primary care practitioners. *Prev Med* 1995;24:546-52.
- Price JH, Desmond SM, Ruppert ES, et al. Pediatricians' perceptions and practices regarding childhood obesity. *Am J Prev Med* 1989;5:95-103.
- Block JP, DeSalvo KB, Fisher WP. Are physicians equipped to address the obesity epidemic? Knowledge and attitudes of internal medicine residents. *Prev Med* 2003;36:669-75.
- Lyznicki JM, Young DC, Riggs JA, et al. Obesity: assessment and management in primary care. *Am Fam Physician* 2001;63:2185-96.
- Linde JA, Jeffery RW, Levy RL, et al. Binge eating disorder, weight control self-efficacy, and depression in overweight men and women. *Int J Obes Relat Metab Disord* 2004;28:418-25.
- Cargill BR, Clark MM, Pera V, et al. Binge eating, body image, depression, and self-efficacy in an obese clinical population. *Obes Res* 1999;7:379-86.
- Miller PM, Watkins JA, Sargent RG, et al. Self-efficacy in overweight individuals with binge eating disorder. *Obes Res* 1999;7:552-5.
- Harris L. *Health maintenance: a nationwide survey of the barriers toward better health and ways of overcoming them*. Newport Beach (CA): Pacific Mutual Life Insurance Company; 1978.
- Swinburn BA, Walter LG, Arroll B, et al. The green prescription study: a randomized controlled trial of written exercise advice provided by general practitioners. *Am J Public Health* 1998;88:288-91.
- Logsdon DN, Lazaro CM, Meier RV. The feasibility of behavioral risk reduction in primary medical care. *Am J Prev Med* 1989;5:249-56.
- Campbell MK, DeVellis BM, Strecher VJ, et al. Improving dietary behavior: the effectiveness of tailored messages in primary care settings. *Am J Public Health* 1994;84:783-7.
- Lewis BS, Lynch WD. The effect of physician advice on exercise behavior. *Prev Med* 1993;22:110-21.
- Butler C, Rollnick S, Stott N. The practitioner, the patient and resistance to change: recent ideas on compliance. *CMAJ* 1996;154:1357-62.
- Miller WR, Rollnick S. *Motivational interviewing: preparing people for change*. New York: Guilford Press; 2002.
- McCambridge J, Strang J. The efficacy of single-session motivational interviewing in reducing drug consumption and perceptions of drug-related risk and harm among young people: results from a multi-site cluster randomized trial. *Addiction* 2004;99:39-52.
- Smith DE, Heckemeyer CM, Kratt PP, et al. Motivational interviewing to improve adherence to a behavioral weight-control program for older obese women with NIDDM: a pilot study. *Diabetes Care* 1997;20:52-4.
- Wilson GT, Schlam TR. The transtheoretical model and motivational interviewing in the treatment of eating and weight disorders. *Clin Psychol Rev* 2004;24:361-78.
- Rubel EC, Sobell LC, Miler WR. Do continuing education workshops improve participants' skills? Effects of a motivational interviewing workshop on substance abuse counselors' skills and knowledge. *Behavior Therapist* 2000;23:73-7.
- Moyers TB, Martin T, Catley D, et al. Assessing the integrity of motivational interviewing interventions: Reliability of the motivational interviewing skills code. *Behav Cogn Psychother* 2003;31:177-84.
- Miller WR, Yahne CE, Moyers TB, et al. A randomized trial of methods to help clinicians learn motivational interviewing. *J Consult Clin Psychol* 2004;72:1050-62.
- Womble LG, Wadden TA, McGuckin BG, et al. A randomized controlled trial of a commercial internet weight loss program. *Obes Res* 2004;12:1011-8.
- Frank A. A multidisciplinary approach to obesity management: the physician's role and team care alternatives. *J Am Diet Assoc* 1998;98(10 Suppl 2):S44-8.

# 10. Behaviour therapy

Michael Vallis

**B**ehaviour therapy refers to a wide collection of specific interventions, generally derived from behavioural theories and targeted specifically at modifying various aspects of a person's behaviour. Behaviour therapy methods have been used for decades and have undergone a number of transformations. It is reasonable to make distinctions between behaviour modification techniques, behaviour therapy for stress management and behaviour modification in the context of lifestyle modification as they apply to obesity management. In contemporary contexts, behaviour modification techniques are seldom used in isolation but are often combined with social support, nutrition and physical activity interventions. As well, behaviour techniques are usually presented in combination, which allows patients and health care professionals to focus on specific techniques that have relevance to the situation or the individual.

## Behaviour modification techniques

Standard behaviour modification techniques include self-monitoring and goal-setting, modifying specific eating behaviours (e.g., slowing the rate of eating, controlling where eating occurs, delaying gratification), stimulus control and reinforcement management.<sup>1</sup> Self-monitoring involves keeping detailed records of situations where unhealthy behaviours occur. Details of the precipitants, consequences and moderating factors are recorded and analyzed to determine goals for change. Stimulus control involves identifying the stimuli (e.g., situations, times, people, emotions) that elicit unhealthy behaviour. These stimuli are then modified to alter the behaviour (e.g., reducing the availability of tempting high-fat, energy-dense foods in a person's home, buying only small amounts of a tempting food, avoiding certain social situations that elicit excess eating). Reinforcement management is the technique whereby the person gives him- or herself a reinforcement for completing a specific behaviour (e.g., buying new clothes after exercising 3 times a week for 1 month).

Many studies have examined the effect of these techniques on eating and weight loss; most were small scale and short term. Three large-scale reviews (2 involving adults, 1 involving children) of well over 100 studies evaluating behaviour modification techniques support the effectiveness of these techniques in promoting weight loss, at least over the short term. Wing and Jeffery<sup>2</sup> reviewed 48 independent empirical studies that included a total of 995 participants. They re-

ported a mean weight loss of 5 kg over an average of 11.7 weeks of treatment. They also noted that behaviour therapy studies, relative to current medications of the time, exercise and diet, resulted in the best maintenance of weight loss. The short duration of these studies is a weakness but is consistent with the methods of behaviour studies at the time. Bennett<sup>3</sup> reviewed 105 studies involving a total of 6121 patients (follow-up data were available for 5453 patients). He reported a mean weight loss of 5.64 kg attributable to behaviour therapy, with a long-term (1-year) weight loss of 5.38 kg. Summerbell and colleagues<sup>4</sup> conducted a Cochrane review of the efficacy of lifestyle interventions for obese children; 13 of 18 RCTs focused on behaviour modification techniques. The authors concluded that, notwithstanding methodological problems, behaviour therapy aimed at children is effective. However, interventions aimed at the parents might be more effective. Collectively, these data support the use of behaviour modification techniques.

## Behaviour therapy for stress management

Several types of behaviour therapy for stress management in the treatment of obesity have been evaluated. The rationale for this treatment is that stress contributes to obesity by eliciting unhealthy behaviours (e.g., eating when upset) or by interfering with healthy behaviour (e.g., stress leading to avoidance of exercise). Cognitive-behaviour therapy addresses the specific thoughts and feelings associated with unhealthy and healthy behaviours. Stress management requires training in techniques to reduce identified stresses that are associated with unhealthy behaviours. Problem-solving techniques are ways to identify potential solutions to a specific problem (usually identified by self-monitoring). Brainstorming methods are used to generate possible solutions that are then systematically tested. Evidence from several RCTs of sufficient methodological rigour to warrant their inclusion in this review support the use of stress-management interventions as adjunctive treatments when there is evidence that stress or cognitions are playing a role in unhealthy behaviours.<sup>5-7</sup> In an RCT, Munsch and colleagues<sup>5</sup> demonstrated the effectiveness of cognitive-behaviour therapy when implemented by general practitioners. All of these studies were limited to a 1-year follow-up period. Collectively, they support implementing stress-management interventions when stress issues are identified. Such interventions are likely to be most useful as an adjunct to other interventions, since they may not be relevant to a majority of patients.

## Lifestyle modification

A number of large-scale RCTs have demonstrated the efficacy of behaviourally focused lifestyle interventions in treating obesity and obesity-related medical problems. These interventions combine behaviour modification techniques, stress management and cognitive-behaviour therapy with nutrition counselling and physical activity. Important large-scale studies include the Trials of Hypertension Prevention,<sup>8</sup> the Diabetes Prevention Program,<sup>9</sup> the Finnish Diabetes Prevention Study<sup>10</sup> and the Women's Healthy Lifestyle Project.<sup>11</sup> All provide strong evidence for the efficacy of lifestyle modification and all used rigorous methods and included reasonable follow-up periods. These studies provide strong evidence that behavioural change through intense lifestyle modification will result in weight loss.

## Additional evidence of the efficacy of behaviour therapy

Further evidence of the efficacy of behaviour therapy can be found in long-term studies that evaluated the behavioural characteristics of people successful at maintaining weight loss. For instance, Westenhoefer and coworkers<sup>12</sup> reported that successful weight loss at 3 years was related to specific eating behaviours, including a flexible approach to eating, avoidance of snacking and high-fat foods, and taking time to eat with no competing or distracting activity. In addition, the ability to cope with stress and maintain regular physical activity predicted long-term successful weight loss. McGuire and colleagues<sup>13</sup> reported that weight-loss maintainers were distinguished by the ongoing use of behavioural strategies, physical activity and weight monitoring. Maintenance of behaviour change was also found to be associated with long-term success in analyses of the National Weight Control Registry.<sup>13-17</sup>

Although there is some controversy over the value of recognizing binge-eating disorder as a separate psychiatric diagnostic category in the DSM-IVR,<sup>18,19</sup> a number of studies have shown that cognitive-behaviour therapy is effective in reducing binge-eating behaviour. However, cognitive-behaviour therapy is not necessarily effective in helping people achieve appreciable weight loss.<sup>20-24</sup> Further research is needed to validate both the construct of binge-eating disorder and effective treatment approaches.

## Conclusions

Individuals willing to participate in weight-management programs should be provided with education and support in behaviour modification techniques as an adjunct to other interventions.<sup>2-4</sup>

Individual progress should be closely monitored during intervention. If behaviour change proves difficult, the role of stress and cognitions in interfering with behaviour change should be explored through direct enquiry. If there is any evidence that stress or cognitions play a role in eating or resistance to change, cognitive-behaviour therapy, problem-solving

therapy and stress management should be considered as adjunctive interventions.<sup>5-7</sup>

Comprehensive lifestyle interventions that include one or more of behaviour modification, cognitive-behaviour therapy, activity enhancement and dietary counselling should be considered, where possible, for all obese people.<sup>8-11</sup>

Cognitive-behaviour therapy or interpersonal psychotherapy should be considered as a treatment adjunct in obese people with binge-eating disorder.<sup>20-24</sup>

## Recommendations

1. We suggest that individuals willing to participate in weight management programs be provided with education and support in behaviour modification techniques as an adjunct to other interventions [*grade B, level 2<sup>2-4</sup>*].
2. We recommend comprehensive lifestyle interventions (combining behaviour modification techniques, cognitive-behaviour therapy, activity enhancement and dietary counselling) for all obese adults [*grade A, level 1<sup>8-11</sup>*].
3. When treating obesity in children, we suggest using family-oriented behaviour therapy [*grade B, level 1<sup>20-24</sup>*].

From Queen Elizabeth II Health Sciences Centre, Dalhousie University, Halifax, NS

Competing interests: None declared.

## REFERENCES

1. Wadden TA. The treatment of obesity: an overview. In: Stunkard AJ, Wadden TA, editors. *Obesity: theory and therapy*. 2nd ed. New York: Raven Press; 1993. p. 197-217.
2. Wing RR, Jeffery RW. Outpatient treatments of obesity: a comparison of methodology and clinical results. *Int J Obes* 1979;3:261-79.
3. Bennett GA. Behaviour therapy for obesity: a quantitative review of the effects of selected treatment characteristics on outcome. *Behav Ther* 1986;17:554-62.
4. Summerbell CD, Ashton V, Campbell KJ, et al. Interventions for treating obesity in children [Cochrane review]. *Cochrane Database Syst Rev* 2003;(3):CD001872.
5. Munsch S, Biedert E, Keller U. Evaluation of a lifestyle change programme for the treatment of obesity in general practice. *Swiss Med Wkly* 2003;133:148-54.
6. Wilfley DE, Welch RR, Stein RI, et al. A randomized comparison of group cognitive-behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. *Arch Gen Psychiatry* 2002;59:713-21.
7. Perri MG, Nezu AM, McKelvey WF, et al. Relapse prevention training and problem-solving therapy in the long-term management of obesity. *J Consult Clin Psychol* 2001;69:722-6.
8. Stevens VJ, Obarzanek E, Cook NR, et al. Long-term weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, phase II. *Ann Intern Med* 2001;134:1-11.
9. Knowler WC, Barrett-Connor E, Fowler SE, et al; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393-403.
10. Tuomilehto J, Lindstrom J, Eriksson JG, et al; Finnish Diabetes Prevention Study Group. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343-50.
11. Kuller LH, Simkin-Silverman LR, Wing RR, et al. Women's Healthy Lifestyle Project: a randomized clinical trial: results at 54 months. *Circulation* 2001;103:32-7.
12. Westenhoefer J, von Falck B, Stellfeldt A, et al. Behavioural correlates of successful weight reduction over 3 y. Results from the Lean Habits Study. *Int J Obes Relat Metab Disord* 2004;28:334-5.
13. McGuire MT, Wing RR, Klem ML, et al. Behavioral strategies of individuals who have maintained long-term weight losses. *Obes Res* 1999;7:334-41.
14. Klem ML, Wing RR, McGuire MT, et al. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *Am J Clin Nutr* 1997;66:239-46.
15. Wyatt HR, Grunwald GK, Mosca CL, et al. Long-term weight loss and breakfast in subjects in the National Weight Control Registry. *Obes Res* 2002;10:78-82.
16. Shick SM, Wing RR, Klem ML, et al. Persons successful at long-term weight loss and maintenance continue to consume a low-energy, low-fat diet. *J Am Diet Assoc* 1998;98:408-13.



17. McGuire MT, Wing RR, Klem ML, et al. Long-term maintenance of weight loss: Do people who lose weight through various weight loss methods use different behaviors to maintain their weight? *Int J Obes Relat Metab Disord* 1998;22:572-7.
18. Devlin MJ, Goldfein JA, Dobrow I. What is this thing called BED? Current status of binge eating disorder nosology. *Int J Eat Disord* 2003;34:S2-18.
19. Cooper Z, Fairburn CG. Refining the definition of binge eating disorder and non-purging bulimia nervosa. *Int J Eat Disord* 2003;34:S89-95.
20. Wonderlich SA, de Zwaan M, Mitchell JE, et al. Psychological and dietary treatments of binge eating disorder: conceptual implications. *Int J Eat Disord* 2003;34:S58-73.
21. Pendleton VR, Goodrick GK, Poston WS, et al. Exercise augments the effects of cognitive-behavioral therapy in the treatment of binge eating. *Int J Eat Disord* 2002;31:172-84.
22. Gorin AA, Le Grange D, Stone AA. Effectiveness of spouse involvement in cognitive behavioral therapy for binge eating disorder. *Int J Eat Disord* 2003;33:421-33.
23. Agras WS, Telch CF, Arnow B, et al. One-year follow-up of cognitive-behavioral therapy for obese individuals with binge eating disorder. *J Consult Clin Psychol* 1997;65:343-7.
24. Eldredge KL, Stewart Agras W, Arnow B, et al. The effects of extending cognitive-behavioral therapy for binge eating disorder among initial treatment nonresponders. *Int J Eat Disord* 1997;21:347-52.

# 11. Dietary intervention for the treatment of obesity in adults

Rena Mendelson, Roselle Martino, Carol Clarke, Karen Mornin, Dana Whitham, Melodie Yong

**W**eight gain and weight loss are a result of an imbalance between energy expenditure and intake. However, effective dietary treatment of obesity continues to elude most patients and health care professionals.<sup>1</sup> Numerous experimental protocols have been developed to test the relative impact of dietary manipulation on appetite and food intake. These are often performed on healthy individuals over a short period. They may include such dietary modifications as increasing fibre intake, adding foods with a low-glycemic index, increasing protein intake or modifying the macronutrient distribution.<sup>2</sup> In addition, various meal replacements have been developed and tested to see whether they will produce sustainable weight loss. Although the results of these may offer guidance on potential dietary interventions, the most rigorous data come from RCTs of at least 1 year's duration. The purpose of this investigation was to review current dietary interventions and to recommend optimal dietary treatment to promote sustainable weight loss for individuals with obesity.

## Dietary interventions

Reduced energy intake (by about 500 kcal/d) will promote gradual weight loss over time at the expected rate of 1–2 kg per month. Currently there is insufficient evidence to conclude that manipulation of the macronutrient distribution to achieve either a low carbohydrate intake or a low fat intake offers any significant advantages.

### Low-carbohydrate diets

Early reports from studies of the Atkins-style diet indicated that a low-carbohydrate diet (less than 30 g/d) offered advantages over a low-fat diet (25% of energy intake) with respect to weight loss and serum triglyceride and HDL cholesterol levels. However, the short duration of these studies (6 months or less) is a limitation.<sup>3</sup> Two long-term trials (1-year duration) demonstrated similar results in that weight loss was significantly greater with a low-carbohydrate diet than with a low-fat diet at 3 months<sup>4</sup> and 6 months<sup>4,5</sup>; however, the difference between study groups at 12 months was no longer significant.<sup>4,6</sup> Improvements in serum triglyceride levels persisted. The authors attributed the diminishing impact on weight loss at 1 year to reduced adherence to the low-carbohydrate diet. A systematic review examining the efficacy of low-carbohydrate diets concluded that there was insufficient evidence to recommend for or against their use.<sup>7</sup> Since long-term alterations in

macronutrient intake offer advantages only in the pattern of weight loss and not in the overall amount of weight loss, the use of long-term (> 6 months) low-carbohydrate diets is not recommended.

### Low-fat diets

Astrup and colleagues<sup>8</sup> undertook a meta-analysis of the impact of low-fat diets, analyzing the results of 16 clinical feeding trials conducted over the previous 30 years. They concluded that low-fat interventions (reducing fat intake by 10% without restricting energy intake) resulted in an average weight loss of 3.2 kg after 1 year. However, these studies were not designed to promote weight loss and therefore may underestimate the effect of low-fat diets as a treatment for obesity. A systematic review<sup>9</sup> found that low-fat diets produced significant weight loss for up to 3 years when compared with usual care. Addressing the question of whether weight loss was a result of low-fat or low-energy diets, 2 reviews concluded that there was no benefit in using a low-fat diet over an energy-restricted diet to achieve sustainable weight loss.<sup>10,11</sup>

### High-protein diets

Two RCTs measuring the effect of increased protein intake on weight loss met the criteria for inclusion in this study. Raising protein levels to 30% rather than 15% of energy intake had no significant effect on weight loss after 1 year.<sup>12</sup> In a comparison of diets based on 25% v. 12% of energy from protein, the group eating a higher protein diet had a greater weight loss at 6 months, but at 1 year the difference was not significant.<sup>13</sup> However, the high-protein group had a greater decrease in waist circumference, waist-to-hip ratio and abdominal fat mass at 1 year. In a systematic review<sup>14</sup> to assess the impact of higher protein intake, the authors concluded that there was convincing evidence that higher protein intake increases satiety compared with diets with lower protein content, but the effects on weight loss have been inconsistent.

### Low-glycemic index, high-fibre diets

In a systematic review<sup>15</sup> of dietary interventions to assess the effects of high-glycemic index foods and low-glycemic index foods on appetite and energy balance, no differences were found in 14 trials. Minor losses were associated with high-glycemic index foods in 2 trials and with low-glycemic index foods in 4 trials. However, none of the trials exceeded

6 months' duration. A review<sup>16</sup> of 22 studies on the impact of fibre on appetite suppression and weight loss concluded that the addition of 10–14 g of dietary fibre may promote a weight loss of 1.3–1.9 kg over 3 months.

## Meal replacements

A few RCTs have tested the effectiveness of meal replacements in decreasing energy intake and promoting weight loss. In one RCT,<sup>17</sup> the experimental group consumed 2 liquid meal replacements each day, as instructed by a registered dietitian, along with a low-energy dinner. The control group was instructed to follow a traditional low-energy diet (approximately 1200 kcal/d). At 1 year, the experimental group ( $n = 26$ ) lost 7.7 kg, compared with 3.5 kg lost by the control group. The small sample and high dropout rates were problems with this study. In another trial,<sup>18</sup> 3 liquid meal replacements combined with fruits and vegetables proved more effective than a traditional weight-loss diet after 1 year (weight loss 6.4 kg v. 1.3 kg). When liquid meal replacements were used 4 times a day (160 kcal per serving) along with a low-energy dinner totalling 1000 kcal/d, participants lost significantly more weight at 20 weeks than did those consuming a balanced low-energy diet (1200–1500 kcal/d). After week 20, both groups consumed a traditional low-energy diet of 1200–1500 kcal/d, and the differences in weight loss disappeared in weeks 40–65, probably as a result of both groups then consuming a comparable energy intake.<sup>19</sup> A meta-analysis<sup>20</sup> of 6 RCTs designed to test the effectiveness of meal replacements in restricting energy intake concluded that, at 1 year, weight loss was significantly higher in the group consuming the meal replacements than in the control group (7%–8% v. 3%–7%).

## Calcium

A meta-analysis of 5 clinical studies to assess the relation between calcium intake and body weight indicated that calcium supplements resulted in a greater weight loss than placebo over a 4-year period.<sup>21</sup> In a review of 3 RCTs and 6 studies, the same authors argued that each 300-mg/d increase in calcium intake was associated with a decrease in body weight of 2.5–3.0 kg.<sup>22</sup> However, a more recent review<sup>23</sup> of 9 RCTs and 7 other studies indicated that more research is needed to address several unanswered questions (e.g., effects and dosage) regarding the effect of calcium supplements or dairy products on weight loss, regulation and maintenance.

## Conclusions

Energy intake is typically reduced to 1200–1500 kcal/d in weight-loss intervention trials. There is evidence to confirm that such dietary interventions will cause a loss of body weight and adipose tissue. However, maintenance of weight loss through dietary intervention alone is a significant challenge. It has been shown that high proportions of participants do not complete the studies or do not adhere to the dietary requirements after 1 year. Ongoing efforts to lose weight are fre-

quently met with modest levels of weight loss<sup>24</sup> or loss followed by weight gain.<sup>25</sup>

A low-carbohydrate diet offers some advantages in terms of weight loss in the first 6 months, and additional advantages at 1 year with respect to serum triglyceride and HDL levels only. A low-fat diet is more effective than a balanced diet in promoting weight loss at 1 year. Similarly, a high-protein diet may promote weight loss through increased satiety resulting in reduced energy intake. However, there is insufficient evidence to promote one form of macronutrient distribution over another or the use of the glycemic index in planning weight-loss diets to treat obesity in the long term.

Meal replacements may offer some advantages over traditional energy-reduced diets for individuals who are able to comply with their use, but more evidence is needed before recommendations for dietary treatment can be made. Calcium and dairy products may have some benefits; again, more evidence is needed to make recommendations with respect to daily food servings and supplement dosage.

No single dietary approach is especially effective, and there is considerable variability in the response of individuals to dietary intervention.

## Recommendations

1. We suggest that the optimal dietary plan for achieving healthy body weight and dietary counselling for adults be developed with a qualified and experienced health professional (preferably a registered dietitian) together with the individual and family to meet their needs [*grade B, level 2*<sup>3,8,13</sup>].
2. We recommend that a nutritionally balanced diet (designed to reduce energy intake) be combined with other supportive interventions to achieve a healthy body weight in overweight and obese people of all ages [*grade A, level 2*].
3. We suggest a high-protein or a low-fat diet (within acceptable macronutrient distribution ranges indicated in the Dietary Reference Intakes) as a reasonable short-term (6–12 months) treatment option for obese adults as part of a weight-loss program [*grade B, level 2*<sup>17,22</sup>].
4. Meal replacements may be considered as a component of an energy-reduced diet for selected adults interested in commencing a dietary weight-loss program [*grade C, level 2*<sup>7,15</sup>].

From the School of Nutrition, Ryerson University, Toronto, Ont. (Mendelson); Dietitians of Canada: Diabetes, Obesity and Cardiovascular Network (Martino, Clarke); St. Paul's Hospital, Vancouver, BC (Mornin, Yong); and St. Michael's Hospital, Toronto, Ont. (Whitham)

**Competing interests:** None declared for Carol Clarke, Roselle Martino, Karen Mornin, Dana Whitham and Melodie Yong. Rena Mendelson has received speaker fees from sanofi-aventis Canada Inc. and fees to contribute to an educational CD-ROM "Cardiovascular and metabolic complications of obesity" sponsored by sanofi-aventis Canada Inc.

## REFERENCES

1. Jeffery RW, Drewnowski A, Epstein LH, et al. Long-term maintenance of weight loss: current status. *Health Psychol* 2000;19(1 Suppl):5-16.
2. Kumanyika SK, Van Horn L, Bowen D, et al. Maintenance of dietary behavior change. *Health Psychol* 2000;19(1 Suppl):42-56.
3. Brehm BJ, Seeley RJ, Daniels SR, et al. A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardio-

- vascular risk factors in healthy women. *J Clin Endocrinol Metab* 2003;88:1617-23.
4. Foster GD, Wyatt HR, Hill JO, et al. A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med* 2003;348:2082-90.
  5. Samaha FF, Iqbal N, Seshadri P, et al. A low-carbohydrate as compared with a low-fat diet in severe obesity. *N Engl J Med* 2003;348:2074-81.
  6. Stern L, Iqbal N, Seshadri P, et al. The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. *Ann Intern Med* 2004;140:778-85.
  7. Bravata DM, Sanders L, Huang J, et al. Efficacy and safety of low-carbohydrate diets: a systematic review. *JAMA* 2003;289:1837-50.
  8. Astrup A, Grunwald GK, Melanson EL, et al. The role of low-fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *Int J Obes Relat Metab Disord* 2000;24:1545-52.
  9. Avenell A, Brown TJ, McGee MA, et al. What are the long-term benefits of weight reducing diets in adults? A systematic review of randomized controlled trials. *J Hum Nutr Diet* 2004;17:317-35.
  10. Pirozzo S, Summerbell C, Cameron C, et al. Advice on low-fat diets for obesity. *Cochrane Database Syst Rev* 2002;(2): CD003640.
  11. Wilkinson DL, McCargar L. Is there an optimal macronutrient mix for weight loss and weight maintenance? *Best Pract Res Clin Gastroenterol* 2004;18:1031-47.
  12. Brinkworth GD, Noakes M, Keogh JB, et al. Long-term effects of a high-protein, low-carbohydrate diet on weight control and cardiovascular risk markers in obese hyperinsulinemic subjects. *Int J Obes Relat Metab Disord* 2004;28:661-70.
  13. Due A, Toubro S, Skov AR, et al. Effect of normal-fat diets, either medium or high in protein, on body weight in overweight subjects: a randomised 1-year trial. *Int J Obes Relat Metab Disord* 2004;28:1283-90.
  14. Halton TL, Hu FB. The effects of high protein diets on thermogenesis, satiety and weight loss: a critical review. *J Am Coll Nutr* 2004;23:373-85.
  15. Raben A. Should obese patients be counselled to follow a low-glycaemic index diet? No. *Obes Rev* 2002;3(4):245-56.
  16. Howarth NC, Saltzman E, Roberts SB. Dietary fiber and weight regulation. *Nutr Rev* 2001;59:129-39.
  17. Ashley JM, St Jeor ST, Perumean-Chaney S, et al. Meal replacements in weight intervention. *Obes Res* 2001;9:312S-20S.
  18. Rothacker DQ, Staniszewski BA, Ellis PK. Liquid meal replacement vs. traditional food: a potential model for women who cannot maintain eating habit change. *J Am Diet Assoc* 2001;101:345-7.
  19. Wadden TA, Foster GD, Sarwer DB, et al. Dieting and the development of eating disorders in obese women: results of a randomized controlled trial. *Am J Clin Nutr* 2004;80:560-8.
  20. Heymsfield SB, van Mierlo CA, van der Knaap HC, et al. Weight management using a meal replacement strategy: meta and pooling analysis from six studies. *Int J Obes Relat Metab Disord* 2003;27:537-49.
  21. Davies KM, Heaney RP, Recker RR, et al. Calcium intake and body weight. *J Clin Endocrinol Metab* 2000;85:4635-8.
  22. Heaney RP, Davies KM, Barger-Lux MJ. Calcium and weight: clinical studies. *J Am Coll Nutr* 2002;21:152S-5S.
  23. Barr SI. Increased dairy product or calcium intake: is body weight or composition affected in humans? *J Nutr* 2003;133:245S-8S.
  24. Dansinger ML, Gleason JA, Griffith JL, et al. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA* 2005;293:43-53.
  25. Anderson JW, Konz EC, Frederich RC, et al. Long-term weight-loss maintenance: a meta-analysis of US studies. *Am J Clin Nutr* 2001;74:579-84.

# 12. Combined diet and exercise therapy for the treatment of obesity in adults

Vivienne A. Vance, Rhona Hanning, Linda McCargar

**T**he alarming increase in obesity, its impact on the health of Canadians and the fact that it has proved to be highly resistant to change suggest a critical need for the development of evidence-based recommendations for treatment. Although the causes of obesity are complex and multiple, interventions to date have tended to focus on single risk factors and have been largely ineffective.

## Methods

Studies were selected based on a systematic review of RCTs of combined diet and exercise therapy for the treatment of overweight and obesity in adults. Published reviews<sup>1-5</sup> were used to corroborate findings and interpretation and to identify additional studies. In addition to the criteria listed in chapter 1 of these guidelines, a study's intervention had to include both a diet and exercise component.

Weight-loss programs for patients with diabetes or significant risk factors for diabetes and cardiovascular disease were included in the review. Outcomes of interest included weight, BMI, waist circumference, percentage body fat, indicators of glucose tolerance, total cholesterol, LDL cholesterol, HDL cholesterol and triglyceride levels, blood pressure, diabetes incidence and medication use. A total of 237 studies were identified, 42 of which met the inclusion criteria.

Dietary interventions generally included moderate energy restriction (by about 500 kcal/d), reduced total and saturated fats (< 30% and < 10% of energy, respectively), high fibre (increased whole grains, fruits and vegetables) balanced diet plans designed to produce a weight loss of 0.5–1.0 kg per week. Very-low-calorie diets designed to produce more rapid weight loss were also included. Exercise interventions included aerobic exercise (primarily moderate-intensity walking) and, in some cases, resistance training.

## Weight loss, body composition and weight maintenance

Long-term RCTs have produced mixed results concerning the effects of combined diet and exercise therapy compared with diet or exercise alone on weight loss and body composition. Several studies have found that, although diet and exercise therapy produced significant weight loss, the effects over a 1-year follow-up period were not significantly greater than those achieved by diet alone.<sup>6-9</sup> For example, in a group of overweight and obese adults, Foreyt and colleagues<sup>6</sup> found that a reduced-energy diet combined with 45 minutes of brisk

walking 3–5 times a week produced a mean weight loss of 8.9 kg over 1 year, compared with 8.0 kg in the diet-only group. Wadden and coworkers<sup>7</sup> found that the combination of diet plus aerobic exercise or strength training, or both, produced significant weight loss (13.7–17.2 kg) after 48 weeks of treatment. However, this weight loss was not significantly different from that in the diet-only group (14.4 kg). In 3 studies that included an exercise-only control group,<sup>6,9,10</sup> diet plus exercise produced significantly greater weight loss than exercise-only interventions. Although the addition of exercise to dietary intervention may not provide additional weight-loss benefits in the short term (< 1 year), combined diet and exercise therapy has consistently proven to produce significantly better long-term weight maintenance.<sup>8,11,12</sup>

Comparing the effects of a very-low-calorie diet, with or without aerobic exercise, Sikand and colleagues<sup>8</sup> reported no significant difference in weight loss between the diet-only and the diet-plus-exercise groups after 4 months of treatment. However, they found that after 2 years the diet-only group had regained 95.8% of their weight loss, compared with 58.2% in the diet-plus-exercise group. In a study involving obese men, Pavlou and coworkers<sup>13</sup> found that significant weight loss (7.1–13.2 kg) was achieved in both the diet-plus-exercise and the diet-only groups at 8 weeks. This weight loss was maintained over 18 months by those who combined dietary intervention with regular exercise, whereas participants who did not incorporate regular exercise regained 60%–92% of their weight loss during the same period. Skender and others<sup>11</sup> found that exercise alone resulted in a smaller weight loss at 1 year than did diet alone and diet plus exercise. However, weight regain in the diet groups over the subsequent 12 months resulted in similar overall weight loss in the exercise-only and diet-plus-exercise groups compared with the diet-only group over a 2-year follow-up period. A recent evidence-based review of obesity treatments in adults<sup>1</sup> concluded that the combination of diet and exercise therapy produced a 1.5–3.0 kg greater weight loss over 2 years than diet alone.

In addition to the positive effects of physical activity on long-term weight maintenance, 2 systematic reviews found good evidence to suggest that the addition of exercise to dietary intervention aids in the preservation of lean tissue during weight loss.<sup>2,3</sup> It should be noted, however, that not all studies have supported the added benefits of exercise over diet alone on body composition. Wadden and colleagues<sup>7</sup> found that, although diet and diet-plus-exercise interventions produced favourable changes in body composition (percent-

age loss from fat, preservation of fat-free mass), differences between the diet-only and diet-plus-exercise groups were not significant. However, diet-plus-exercise interventions produced smaller reductions in resting energy expenditure over 48 weeks.

## Type, intensity and duration of physical activity

Studies designed to assess the effects of type, intensity and duration of exercise combined with dietary intervention<sup>7,14-16</sup> suggest that the duration of activity is an important predictor of weight loss. There is insufficient evidence, however, to support an association between type and intensity of exercise and weight loss. A recent study<sup>16</sup> revealed that weight loss was significantly greater among women reporting 200 minutes per week or more of exercise than among those reporting less than 150 minutes per week or inconsistent exercise patterns, but it showed no effect of exercise intensity. Comparing the effects of intermittent short-bout (10 minutes) exercise sessions versus a continuous long-bout (20-40 minutes) exercise session of equal total duration, Jakicic and colleagues<sup>15</sup> found that, although weight loss over 18 months was positively associated with exercise duration, it did not differ significantly between the 2 groups of women. A study designed to investigate the effects of diet plus aerobic or strength training, or both, found no significant differences in weight loss or changes in body composition as a function of the type of physical activity.<sup>7</sup> There is some evidence to suggest that home-based exercise programs versus structured supervised programs,<sup>17</sup> and moderate versus more intense exercise (2-3 hours per week v. 4-6 hours per week),<sup>18</sup> may increase exercise adherence and improve long-term weight loss and maintenance for obese individuals.

## Type 2 diabetes

The pooled results of several long-term diabetes prevention programs suggest a significant positive effect of diet and exercise therapy on weight loss, glycemic control and reduction of diabetes incidence among overweight and obese people with impaired glucose tolerance.<sup>10,19-32</sup> For example, the Diabetes Prevention Program, which was designed to assess the effects of intensive lifestyle interventions,<sup>19,20</sup> found that a reduced-energy, low-fat diet combined with a walking program (150 minutes per week) produced a significantly greater mean weight loss (5.6 kg) than did either standard care plus metformin (2.1 kg) or standard care plus placebo (0.1 kg) over an average follow-up of 2.8 years. The incidence of metabolic syndrome was reduced by 41% with the combined diet and exercise compared with standard care. The Finnish Diabetes Prevention Study,<sup>21-25</sup> which compared the effects of diet and exercise therapy with standard care, reported significantly greater weight loss and improvements in fasting glucose levels at 1 year among participants assigned to the lifestyle intervention group. Modest weight loss (3.5 kg) was maintained among those in the intervention group at 3 years, with 41.8% of the intervention group versus 12% of the control group

achieving a weight reduction of at least 5 kg. Both the Diabetes Prevention Program and the Finnish Diabetes Prevention Study reported a 58% reduction in diabetes incidence over 3 years among participants enrolled in an intensive lifestyle intervention program. The Oslo Diet and Exercise Study,<sup>10,26</sup> which included diet-only, exercise-only, combined therapy and control groups, found that the combination of diet plus exercise produced significantly greater weight loss compared with the exercise-only and control groups. Combined diet and exercise was also most effective in improving glucose tolerance and reversing the development of insulin resistance syndrome.

Long-term data on the effects of combined diet and exercise therapy among obese patients with type 2 diabetes are sparse. Findings from 2 RCTs<sup>12,33</sup> and 1 nonrandomized clinical trial<sup>34</sup> suggest that, compared with diet alone, diet plus exercise may produce greater weight loss, improvements in glycemic control and reductions in medication use among people with type 2 diabetes.

## Cardiovascular disease

Combined diet and exercise therapy resulted in significant improvement in cardiovascular disease risk factors compared with either treatment alone. The Oslo Diet and Exercise Study,<sup>35,36</sup> for example, revealed that the combination therapy produced more favourable outcomes in terms of triglyceride, HDL cholesterol and plasma leptin levels than with either diet or exercise alone. Diet and diet plus exercise were equally effective in reducing blood pressure among patients with mild hypertension.

Over a 12-month follow-up period, Wood and colleagues<sup>37</sup> found significantly greater weight loss, a lower percentage of body fat, significant improvements in the ratio of abdomen-to-hip girth and in triglyceride, apolipoprotein A-I and HDL cholesterol levels among men assigned to diet and exercise than among those assigned to diet only. A significant reduction in the ratio of total cholesterol to HDL cholesterol was seen among women assigned to combined treatment. The addition of exercise to dietary intervention prevented the lowering of HDL cholesterol, which often results from a low-fat diet, in both men and women. Substantial positive effects on the ratio of LDL cholesterol to HDL cholesterol and the ratio of apolipoprotein B to apolipoprotein A-I, and a significantly reduced estimated risk of coronary artery disease were observed among men and women in the diet-and-exercise group compared with those in the diet-only group. Diet plus exercise produced significant reductions in systolic and diastolic blood pressure, although the effect was not significantly better than with diet only.

Comparing the effects of diet, aerobic exercise and a combination of the 2 on plasma lipoproteins, Stefanick and coworkers<sup>38</sup> reported no significant differences in HDL cholesterol and triglyceride levels or in the ratio of total cholesterol to HDL cholesterol at 1 year. However, they found that reductions in LDL cholesterol were not significant among men or women without the addition of exercise to the dietary intervention. Other studies comparing diet and exercise ther-

apy versus usual care or no treatment<sup>39-45</sup> have revealed significant improvements in several cardiovascular disease risk factors among participants assigned to the combined treatment. These risk factors included body weight, waist-to-hip ratio, blood pressure, fibrinolysis, and levels of triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol, plasma leptin and C-reactive protein.

## Conclusions

Interpreting the results of lifestyle interventions in the treatment of obesity is limited because of the loss of participants to follow-up and their poor adherence to dietary and exercise protocols. The absence of a diet-only or exercise-only control group in some trials makes it difficult to identify independent versus combined effects of treatment. However, despite these methodological limitations, the results of long-term RCTs provide sufficient evidence to recommend diet and exercise therapy for overweight and obese patients with or without comorbidities.

The combination of diet and exercise is associated with improved long-term weight-loss maintenance compared with either intervention alone. Adding exercise to dietary intervention may also provide some conservation of fat-free mass during weight loss.

Although it is uncertain whether there is an optimal exercise prescription for weight loss, increased exercise duration produces greater weight loss. Home-based programs and the prescription of moderate exercise may improve adherence and long-term weight loss. Although the reported long-term weight loss achieved by diet and exercise therapy is moderate, a recent evidence-based review<sup>1</sup> revealed that even modest weight loss (defined as up to 10% of body weight) produced significant health benefits.

Intensive lifestyle intervention for the prevention of type 2 diabetes has consistently demonstrated that diet and exercise therapy promotes modest reductions in body weight, improved glycemic control and a significant reduction in diabetes risk among overweight men and women with impaired glucose tolerance. People with type 2 diabetes assigned to combined therapy may experience greater weight loss, improvements in glycemic control and a decreased need for oral medications compared with those assigned to diet-only interventions.

Although diet and diet plus exercise seem to be equally effective in reducing high blood pressure, the addition of exercise to dietary intervention produces significant improvement in several other cardiovascular disease risk factors beyond those achieved with diet alone. Plasma lipid levels, in particular, seem to respond more favourably to combined treatment.

There is strong evidence that diet and exercise therapy produces weight loss and many related health benefits. However, long-term adherence to diet and exercise treatment is often poor. Weight regain, although significantly less in those exposed to combined treatment, is common. Future research should be focused on the development of strategies to improve diet and exercise compliance to produce sustainable weight loss.

## Recommendations

1. We recommend an energy-reduced diet and regular physical activity as the first treatment option for overweight and obese adults to achieve clinically important weight loss and reduce obesity-related symptoms [*grade A, level 2<sup>B,13</sup>*].
2. We recommend diet and exercise therapy for overweight and obese people with risk factors for type 2 diabetes [*grade A, level 1<sup>0,19,25</sup>*] and cardiovascular disease [*grade A, level 2<sup>37,38</sup>*].

From the Faculty of Applied Health Sciences, University of Waterloo, Waterloo, Ont. (Vance, Hanning); and the Faculty of Agriculture, Forestry and Home Economics, University of Alberta, Edmonton, Alta. (McCaragar)

**Competing interests:** None declared.

## REFERENCES

1. Orzano AJ, Scott JG. Diagnosis and treatment of obesity in adults: an applied evidence-based review. *J Am Board Fam Pract* 2004;17:359-69.
2. Thorogood M. Combining diet with physical activity in the treatment of obesity. *J Hum Nutr Diet* 1998;11:239-42.
3. Little P, Margetts B. The importance of diet and physical activity in the treatment of conditions managed in general practice. *Br J Gen Pract* 1996;46:187-92.
4. Avenell A, Broom J, Brown TJ, et al. Systematic review of the long-term effects and economic consequences of treatments for obesity and implications for health improvement. *Health Technol Assess* 2004;8:1-182.
5. Norris SL, Zhang X, Avenell A, et al. Long-term effectiveness of weight-loss interventions in adults with pre-diabetes: a review. *Am J Prev Med* 2005;28:126-39.
6. Foreyt JP, Goodrick GK, Reeves RS, et al. Response of free-living adults to behavioral treatment of obesity: attrition and compliance to exercise. *Behav Ther* 1993;24:659-69.
7. Wadden TA, Vogt RA, Andersen RE, et al. Exercise in the treatment of obesity: effects of four interventions on body composition, resting energy expenditure, appetite, and mood. *J Consult Clin Psychol* 1997;65:269-77.
8. Sikand G, Kondo A, Foreyt JP, et al. Two-year follow-up of patients treated with a very-low-calorie diet and exercise training. *J Am Diet Assoc* 1988;88:487-8.
9. Miller GD, Nicklas BJ, Davis CC, et al. Is serum leptin related to physical function and is it modifiable through weight loss and exercise in older adults with knee osteoarthritis? *Int J Obes Relat Metab Disord* 2004;28:1383-90.
10. Torjesen PA, Birkeland KI, Anderssen SA, et al. Lifestyle changes may reverse development of the insulin resistance syndrome. The Oslo Diet and Exercise Study: a randomized trial. *Diabetes Care* 1997;20:26-31.
11. Skender ML, Goodrick GK, Del Junco DJ, et al. Comparison of 2-year weight loss trends in behavioral treatments of obesity: diet, exercise, and combination interventions. *J Am Diet Assoc* 1996;96:342-6.
12. Wing RR, Epstein LH, Paternostro-Bayles M, et al. Exercise in a behavioural weight control programme for obese patients with type 2 (non-insulin-dependent) diabetes. *Diabetologia* 1988;31:902-9.
13. Pavlou KN, Krey S, Steffee WP. Exercise as an adjunct to weight loss and maintenance in moderately obese subjects. *Am J Clin Nutr* 1989;49(5 Suppl):1115-23.
14. Fogelholm M, Kukkonen-Harjula K, Nenonen A, et al. Effects of walking training on weight maintenance after a very-low-energy diet in premenopausal obese women: a randomized controlled trial. *Arch Intern Med* 2000;160:2177-84.
15. Jakicic JM, Winters C, Lang W, et al. Effects of intermittent exercise and use of home exercise equipment on adherence, weight loss, and fitness in overweight women: a randomized trial. *JAMA* 1999;282:1554-60.
16. Jakicic JM, Marcus BH, Gallagher KI, et al. Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial. *JAMA* 2003;290:1323-30.
17. Perri MG, Martin AD, Leermakers EA, et al. Effects of group- versus home-based exercise in the treatment of obesity. *J Consult Clin Psychol* 1997;65:278-85.
18. Fogelholm M, Kukkonen-Harjula K, Oja P. Eating control and physical activity as determinants of short-term weight maintenance after a very-low-calorie diet among obese women. *Int J Obes Relat Metab Disord* 1999;23:203-10.
19. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393-403.
20. Orchard TJ, Temprosa M, Goldberg R, et al. The effect of metformin and intensive lifestyle intervention on the metabolic syndrome: the Diabetes Prevention Program randomized trial. *Ann Intern Med* 2005;142:611-9.
21. Eriksson J, Lindstrom J, Valle T, et al. Prevention of Type II diabetes in subjects with impaired glucose tolerance: the Diabetes Prevention Study (DPS) in Finland. Study design and 1-year interim report on the feasibility of the lifestyle intervention programme. *Diabetologia* 1999;42:793-801.

22. Uusitupa M, Louheranta A, Lindstrom J, et al. The Finnish Diabetes Prevention Study. *Br J Nutr* 2000;83:S137-42.
23. Tuomilehto J, Lindstrom J, Eriksson JG, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343-50.
24. Lindstrom J, Louheranta A, Mannelin M, et al. The Finnish Diabetes Prevention Study (DPS): Lifestyle intervention and 3-year results on diet and physical activity. *Diabetes Care* 2003;26:3230-6.
25. Lindstrom J, Eriksson JG, Valle TT, et al. Prevention of diabetes mellitus in subjects with impaired glucose tolerance in the Finnish diabetes prevention study: results from a randomized clinical trial. *J Am Soc Nephrol* 2003;14(Suppl 2):S108-13.
26. Anderssen SA, Hjermann I, Urdal P, et al. Improved carbohydrate metabolism after physical training and dietary intervention in individuals with the "atherothrombotic syndrome." Oslo Diet and Exercise Study (ODES). A randomized trial. *J Intern Med* 1996;240:203-9.
27. Mensink M, Feskens EJ, Saris WH, et al. Study on Lifestyle Intervention and Impaired Glucose Tolerance Maastricht (SLIM): preliminary results after one year. *Int J Obes Relat Metab Disord* 2003;27:377-84.
28. Mensink M, Blaak EE, Corpeleijn E, et al. Lifestyle intervention according to general recommendations improves glucose tolerance. *Obes Res* 2003;11:1588-96.
29. Mensink M, Blaak EE, Vidal H, et al. Lifestyle changes and lipid metabolism gene expression and protein content in skeletal muscle of subjects with impaired glucose tolerance. *Diabetologia* 2003;46:1082-9.
30. Liao D, Asberry PJ, Shofer JB, et al. Improvement of BMI, body composition, and body fat distribution with lifestyle modification in Japanese Americans with impaired glucose tolerance. *Diabetes Care* 2002;25:1504-10.
31. Wing RR, Venditti E, Jakicic JM, et al. Lifestyle intervention in overweight individuals with a family history of diabetes. *Diabetes Care* 1998;21:350-9.
32. Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* 1997;20:537-44.
33. Uusitupa MI. Early lifestyle intervention in patients with non-insulin-dependent diabetes mellitus and impaired glucose tolerance. *Ann Med* 1996;28:445-9.
34. Paisey RB, Frost J, Harvey P, et al. Five year results of a prospective very low calorie diet or conventional weight loss programme in type 2 diabetes. *J Hum Nutr Diet* 2002;15:121-7.
35. Anderssen S, Holme I, Urdal P, et al. Diet and exercise intervention have favourable effects on blood pressure in mild hypertensives: the Oslo Diet and Exercise Study (ODES). *Blood Press* 1995;4:343-9.
36. Reseland JE, Anderssen SA, Solvoll K, et al. Effect of long-term changes in diet and exercise on plasma leptin concentrations. *Am J Clin Nutr* 2001;73:240-5.
37. Wood PD, Stefanick ML, Williams PT, et al. The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. *N Engl J Med* 1991;325:461-6.
38. Stefanick ML, Mackey S, Sheehan M, et al. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *N Engl J Med* 1998;339:12-20.
39. Ratner R, Goldberg R, Haffner S, et al. Impact of intensive lifestyle and metformin therapy on cardiovascular disease risk factors in the diabetes prevention program. *Diabetes Care* 2005;28:888-94.
40. Haffner S, Temprosa M, Crandall J, et al. Intensive lifestyle intervention or metformin on inflammation and coagulation in participants with impaired glucose tolerance. *Diabetes* 2005;54:1566-72.
41. Lindahl B, Nilsson TK, Jansson JH, et al. Improved fibrinolysis by intense lifestyle intervention. A randomized trial in subjects with impaired glucose tolerance. *J Intern Med* 1999;246:105-12.
42. Esposito K, Pontillo A, Di Palo C, et al. Effect of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial. *JAMA* 2003;289:1799-804.
43. Jalkanen L. The effect of a weight reduction program on cardiovascular risk factors among overweight hypertensives in primary health care. *Scand J Soc Med* 1991;19:66-71.
44. Stevens VJ, Corrigan SA, Obarzanek E, et al. Weight loss intervention in phase I of the Trials of Hypertension Prevention. The TOHP Collaborative Research Group. *Arch Intern Med* 1993;153:849-58.
45. Stevens VJ, Obarzanek E, Cook NR, et al. Long-term weight loss and changes in blood pressure: results of the Trials of Hypertension Prevention, phase II. *Ann Intern Med* 2001;134:1-11.



# 13. Physical activity and exercise therapy — adults

Denis Prud'homme, Eric Doucet, Isabelle Dionne, Robert Ross

**P**hysical activity is any movement produced by the contraction of skeletal muscle that substantially increases energy expenditure, whereas exercise is a subset of physical activity. The “dose” of physical activity needed to bring about a particular health benefit can be described in terms of frequency, duration, intensity and type of activity. Finally, the gross cost of a physical activity is total energy expenditure, which depends on resting metabolic rate, the cost of the activity itself and the cost of the thermic effect of food if the activity is postprandial.<sup>1</sup>

## Benefits of physical activity

It is widely accepted that regular physical activity reduces all-cause mortality, and the incidence of fatal and nonfatal cardiovascular disease and coronary artery disease. It is also associated with a reduction in the incidence of type 2 diabetes mellitus, colon cancer and osteoporosis. Further benefits include improvement in several cardiovascular risk factors (blood pressure, lipid and lipoprotein levels, coagulation and hemostatic factors).<sup>1</sup> Regular physical activity is also associated with the prevention of weight gain over time.<sup>2</sup>

Increasing one's level of cardiorespiratory fitness has been shown to reduce risk of chronic disease and total mortality independent of BMI and percentage of body fat.<sup>3,4</sup> The US Centers for Disease Control and Prevention and the American College of Sport Medicine recommend a minimum of 30 minutes of moderate-intensity activity on most days of the week to improve health (150 minutes per week),<sup>5</sup> whereas the Institute of Medicine recommends a minimum of 60 minutes of exercise on most days of the week to control body weight (420 minutes per week).<sup>6</sup>

The objective of this section is to review long-term ( $\geq 1$  year) RCTs that have investigated the effectiveness of physical activity on weight loss or weight maintenance.

## Physical activity and weight loss

Past meta-analyses<sup>7–10</sup> have concluded that physical activity is an important component of behavioural interventions, along with dietary control, for short-term weight loss among overweight and obese adults. Physical activity is also among the best predictors of long-term weight loss maintenance. In an evidence-based systematic review<sup>11</sup> of the dose–response relation between physical activity and total and regional body fat loss, weight loss was greater with short-term physical activity interventions ( $\leq 16$  weeks) than with long-term interventions

( $> 26$  weeks) (0.18 kg per week v. 0.06 kg per week). This effect could be explained in part by the higher energy expenditure (2200 kcal per week v. 1000 kcal per week) with short-term regimens. A dose–response relation was also observed between the amount of physical activity, expressed as energy expended per week, and the reduction of total adiposity in short-term interventions. With regard to regional body fat, the authors concluded that there was insufficient evidence to determine whether an increase in physical activity is associated with a reduction in abdominal obesity.

## Current status of knowledge

We searched PubMed for articles published from 1990 to 2004 using the key words exercise, physical activity, obesity, weight loss and weight maintenance. The reference lists of the identified articles were then reviewed for additional studies. In addition to the criteria described in chapter 1 (prospective RCT, duration  $\geq 1$  year and participants overweight or obese), a study had to meet the following conditions:

- Measurements of body weight, adiposity or abdominal obesity were available and established with validated methods, such as BMI, waist circumference and computed tomography (CT).
- Participants in the physical activity group were instructed not to change their eating habits or reduce their daily energy intake to 1200–1500 kcal (e.g., similar to the control or the diet group); studies with a more severe restrictive diet were excluded. Thus, we could assume that the negative energy balance was induced primarily by the increase in physical activity.
- Authors reported the energy expenditure associated with the physical activity or provided the information required to estimate the dose (e.g., maximum oxygen consumption, intensity, duration and frequency).

Nine RCTs<sup>12–20</sup> of physical activity for the treatment of obesity met the duration criterion. Four additional RCTs<sup>21–24</sup> of shorter duration ( $\geq 32$  weeks) were also considered because of the paucity of longer term studies. The selected studies included 1389 overweight and obese people (70% women and 30% men). Their age and BMI varied from 17 to 75 years and from 25 to 36 kg/m<sup>2</sup>, respectively. Most of the studies involved aerobic physical activity (e.g., walking, jogging) combined with a cognitive-behaviour intervention program (e.g., self-monitoring, stimulus control).

The mean weight change following the physical activity intervention ranged from  $-5.2$  to  $0.6$  kg ( $-0.09$  and  $0.01$  kg

per week in the most and least successful trials) among men<sup>11,12,16,17</sup> and from -6.24 to 0.6 kg (-0.13 to 0.01 kg per week) among women.<sup>12,14,17,20,22-24</sup> When physical activity was combined with dietary recommendations, the mean weight loss among men<sup>17</sup> was 2.6-4.2 kg (0.05-0.08 kg per week) and among women<sup>13,15,19</sup> 3.1-10.1 kg (0.06-0.19 kg per week). Large inter-individual variations in weight loss were often observed in these studies.

The evidence suggested a dose-response relation between the duration of physical activity and weight loss among overweight people.<sup>13,14,19,21</sup> For sedentary people, reported weight loss was significantly greater among those who engaged in physical activity for 200 minutes or more per week compared with those whose physical activity lasted less than 150 minutes per week or was inconsistent.<sup>13,14</sup> On the other hand, no dose-response relation was observed for intensity of activity. Long-term weight loss achieved with short bouts of physical activity was as important as weight loss achieved with long bouts.<sup>19,20</sup> Compared with a supervised program, a home-based program resulted in a higher rate of participation and adherence to treatment as well as significantly greater weight loss at 3 months follow-up (11.7 [standard deviation (SD) 9.0] v. 7.0 [SD 8.0] kg,  $p = 0.05$ ).<sup>15</sup>

Although the reported effects of physical activity on weight loss are modest, they are nonetheless encouraging.

Only 4 RCTs<sup>12,14,18,20</sup> addressing the effect of physical activity on amount of abdominal fat met the 1-year duration criterion; as a result, 2 additional RCTs<sup>21,22</sup> of shorter duration ( $\geq 32$  weeks) were also included. These 6 studies included 543 overweight and obese people (80% women and 20% men). Their age and BMI ranged from 17 to 75 years and from 25 to 36 kg/m<sup>2</sup>, respectively. Measurement with CT scanning in men was reported in only 1 study; waist circumference was the most frequent measure of abdominal obesity. In most of the studies, aerobic physical activity (e.g., walking, jogging) was combined with a cognitive-behaviour intervention program (e.g., self-monitoring, stimulus control). The mean reduction in waist circumference with physical activity ranged from 0.2 to 2.9 cm (0.28-0.60 mm per week) among women<sup>14,20</sup> and from 1.1 to 3.2 cm (0.34-1.00 mm per week) among men and women combined.<sup>21</sup> When exercise was combined with dietary recommendations, mean waist reductions among women were 3.6-7.2 cm (0.5-0.10 mm per week).<sup>21</sup>

Two long-term RCTs<sup>12,14</sup> used CT scanning to measure abdominal fat. In the first, Donnelly and colleagues<sup>12</sup> reported a significant decrease in visceral fat (mean 22.5 [SD 21.4] cm<sup>2</sup>), subcutaneous fat (mean 51.4 [SD 54.4] cm<sup>2</sup>) and total abdominal fat area (mean 73.9 [SD 68.9] cm<sup>2</sup>) at L4-L5 vertebral space among young overweight men assigned to a 16-month exercise program. However, the difference in abdominal fat between the intervention and control groups was not significant. Women who exercised showed no significant changes in subcutaneous, visceral or total abdominal fat. However, the control group had significant increases in subcutaneous and total abdominal fat compared with the exercise group. In the second RCT, a study involving overweight and obese postmenopausal women,<sup>14</sup> an aerobic physical activity program (e.g., brisk walking) resulted in a

significant reduction in visceral (8.6 g/cm<sup>2</sup>) and subcutaneous abdominal fat (28.8 g/cm<sup>2</sup>). Large inter-individual variations in abdominal fat changes were observed in this study. Irwin and colleagues<sup>14</sup> and Slentz and others<sup>21</sup> reported a dose-response relation between the amount of physical activity and changes in abdominal obesity measured by CT<sup>14</sup> and waist circumference<sup>21</sup> respectively in overweight and obese people. No dose-response relation was observed between physical activity intensity and concomitant reduction in abdominal fat.<sup>21</sup>

In short-term and nonrandomized trials, physical activity, with or without weight loss, was associated with a marked reduction in abdominal obesity in men and women, an effect that seems to have been associated with the initial level of visceral fat accumulation.<sup>11</sup> However, given the limited evidence, it is not possible to suggest that long-term regular physical activity is associated with a reduction in abdominal obesity.

## Weight regain

Weight regain is frequently observed during or following a weight-management intervention. There is some evidence that exercise may be useful in the prevention of weight regain, although its benefit over dietary education remains unclear.<sup>25</sup> In a recent systematic review, Fogelholm and Kukkonen-Harjula<sup>2</sup> reported that the benefits of regular physical activity as a part of weight maintenance strategy were unclear. As indicated in data from the National Weight Control Registry<sup>26</sup> and other studies,<sup>19,27</sup> a higher level of physical activity of moderate intensity (280-450 minutes per week, which is equivalent to walking 45 km per week) may be needed to prevent weight regain.

Despite the lack of clear evidence for an association between physical activity and weight maintenance, the beneficial effects of regular physical activity in terms of physical fitness<sup>3,4</sup> and reduction in several cardiovascular risk factors and risk of death from cardiovascular disease<sup>1,28,29</sup> justify its inclusion as part of an active lifestyle for obese people.<sup>2</sup>

## Conclusions

Obese patients should be informed that regular physical activity, in association with a healthy diet, represents an important element of their treatment plan. Sedentary obese people should engage in 30 minutes a day of moderate-intensity aerobic exercise (e.g., walking, jogging) and, when appropriate, progress to 60 minutes a day for body weight control or health benefits.

## Recommendations

1. All those considering initiating a vigorous exercise program are encouraged to consult their physician or health care team professionals [*grade C, level 4*].
2. We suggest long-term, regular physical activity, which is associated with maintenance of body weight or a modest reduction in body weight, for all overweight and obese people [*grade B, level 2*<sup>13,16</sup>].

3. Physical activity and exercise should be sustainable and tailored to the individual. We recommend that the total duration be increased gradually to maximize the weight-loss benefits [grade A, level 2<sup>2,8,9</sup>].
4. We suggest physical activity (30 minutes a day of moderate intensity, increasing, when appropriate, to 60 minutes a day) as part of an overall weight-loss program [grade B, level 2<sup>2,21</sup>].
5. Endurance exercise training may reduce the risk of cardiovascular morbidity in healthy postmenopausal women, and we suggest its use for adults with an increased BMI [grade B, level 2].

From the Faculty of Health Sciences, School of Human Kinetics, University of Ottawa, Ottawa, Ont. (Prud'homme, Doucet); the Faculté d'éducation physique et sportive, Université de Sherbrooke, Sherbrooke, Que. (Dionne); and the Faculty of Medicine, School of Physical and Health Education, Queen's University, Kingston, Ont. (Ross)

**Competing interests:** None declared.

## REFERENCES

1. Kesaniemi YK, Danforth E Jr, Jensen MD, et al. Dose-response issues concerning physical activity and health: an evidence-based symposium. *Med Sci Sports Exerc* 2001;33(6 Suppl):S351-8.
2. Fogelholm M, Kukkonen-Harjula K. Does physical activity prevent weight gain? A systematic review. *Obes Rev* 2000;1:95-111.
3. Blair SN, Kampert JB, Kohl HW III, et al. Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *JAMA* 1996;276:205-10.
4. Lee CD, Blair SN, Jackson AS. Cardiorespiratory fitness, body composition, and all-cause and cardiovascular disease mortality in men. *Am J Clin Nutr* 1999;69:373-80.
5. Pate RR, Pratt M, Blair SN, et al. Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. *JAMA* 1995;273:402-7.
6. Food and Nutrition Board, Institute of Medicine of the National Academies. *Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. Washington (DC): National Academies Press; 2002.
7. Ballor DL, Keeseey RE. A meta-analysis of the factors affecting exercise-induced changes in body mass, fat mass and fat-free mass in males and females. *Int J Obes* 1991;15:717-26.
8. Garrow JS, Summerbell CD. Meta-analysis: effect of exercise, with or without dieting, on the body composition of overweight subjects. *Eur J Clin Nutr* 1995;49:1-10.
9. Miller WC, Koceja DM, Hamilton EJ. A meta-analysis of the past 25 years of weight loss research using diet, exercise or diet plus exercise intervention. *Int J Obes Relat Metab Disord* 1997;21:941-7.
10. Wing RR. Physical activity in the treatment of the adulthood overweight and obesity: current evidence and research issues. *Med Sci Sports Exerc* 1999;31(11 Suppl):S547-52.
11. Ross R, Janssen I. Physical activity, total and regional obesity: dose-response considerations. *Med Sci Sports Exerc* 2001;33(6 Suppl):S521-7.
12. Donnelly JE, Hill JO, Jacobsen DJ, et al. Effects of a 16-month randomized controlled exercise trial on body weight and composition in young, overweight men and women: the Midwest Exercise Trial. *Arch Intern Med* 2003;163:1343-50.
13. Jakicic JM, Marcus BH, Gallagher KI, et al. Effect of exercise duration and intensity on weight loss in overweight, sedentary women: a randomized trial. *JAMA* 2003;290:1323-30.
14. Irwin ML, Yasui Y, Ulrich CM, et al. Effect of exercise on total and intra-abdominal body fat in postmenopausal women: a randomized controlled trial. *JAMA* 2003;289:323-30.
15. Perri MG, Martin AD, Leermakers EA, et al. Effects of group- versus home-based exercise in the treatment of obesity. *J Consult Clin Psychol* 1997;65:278-85.
16. Pritchard JE, Nowson CA, Wark JD. A worksite program for overweight middle-aged men achieves lesser weight loss with exercise than with dietary change. *J Am Diet Assoc* 1997;97:37-42.
17. Stefanick ML, Mackey S, Sheehan M, et al. Effects of diet and exercise in men and postmenopausal women with low levels of HDL cholesterol and high levels of LDL cholesterol. *N Engl J Med* 1998;339:12-20.
18. Wing RR, Venditti E, Jakicic JM, et al. Lifestyle intervention in overweight individuals with a family history of diabetes. *Diabetes Care* 1998;21:350-9.
19. Jakicic JM, Winters C, Lang W, et al. Effects of intermittent exercise and use of home exercise equipment on adherence, weight loss, and fitness in overweight women: a randomized trial. *JAMA* 1999;282:1554-60.
20. Donnelly JE, Jacobsen DJ, Heelan KS, et al. The effects of 18 months of intermittent vs. continuous exercise on aerobic capacity, body weight and composition, and metabolic fitness in previously sedentary, moderately obese females. *Int J Obes Relat Metab Disord* 2000;24:566-72.
21. Slentz CA, Duscha BD, Johnson JL, et al. Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRRIDE — a randomized controlled study. *Arch Intern Med* 2004;164:31-9.
22. Binder EF, Birge SJ, Kohrt WM. Effects of endurance exercise and hormone replacement therapy on serum lipids in older women. *J Am Geriatr Soc* 1996;44:231-6.
23. Frey-Hewitt B, Vranizan KM, Dreon DM, et al. The effect of weight loss by dieting or exercise on resting metabolic rate in overweight men. *Int J Obes* 1990;14:327-34.
24. Kohrt WM, Ehsani AA, Birge SJ Jr. Effects of exercise involving predominantly either joint-reaction or ground-reaction forces on bone mineral density in older women. *J Bone Miner Res* 1997;12:1253-61.
25. Glenny AM, O'Meara S, Melville A, et al. The treatment and prevention of obesity: a systematic review of the literature. *Int J Obes Relat Metab Disord* 1997;21:715-37.
26. Klem ML, Wing RR, McGuire MT, et al. A descriptive study of individuals successful at long-term maintenance of substantial weight loss. *Am J Clin Nutr* 1997;66:239-46.
27. Schoeller DA, Shay K, Kushner RF. How much physical activity is needed to minimize weight gain in previously obese women? *Am J Clin Nutr* 1997;66:551-6.
28. Grundy SM, Blackburn G, Higgins M, et al. Physical activity in the prevention and treatment of obesity and its comorbidities. *Med Sci Sports Exerc* 1999;31(11 Suppl):S502-8.
29. Hu G, Tuomilehto J, Silventoinen K, et al. The effects of physical activity and body mass index on cardiovascular, cancer and all-cause mortality among 47 212 middle-aged Finnish men and women. *Int J Obes (London)* 2005;29:894-902.

# 14. Pharmacotherapy for obesity — adults

Arya M. Sharma, James D. Douketis

Pharmacotherapy may be considered for weight loss in selected individuals who are overweight (BMI  $\geq 27$  kg/m<sup>2</sup>) or obese (BMI  $\geq 30$  kg/m<sup>2</sup>) and in whom clinically important weight loss might result in health benefits. The vast majority of studies that have assessed pharmacologic weight-loss interventions are short-term studies, with a follow-up of 3–6 months.<sup>1,2</sup> However, obesity is a chronic condition. A life-long management strategy is required, as for diabetes or hypertension.<sup>3,4</sup> Thus, studies of at least 12 months' duration are required to assess the potential benefits and risks of pharmacotherapy in obese or overweight patients.

Many obese and overweight people will face challenges in attaining the level of weight loss that they consider to be esthetically pleasing. However, weight loss of 5%–10% of total body weight, or 5 kg in an obese person who weighs 100 kg and is 160 cm tall, is considered clinically important. Such relatively modest weight loss has the potential to improve glycemic control, blood pressure control and lipid levels, especially in people with type 2 diabetes or hypertension. Weight loss of 5%–10% of body weight might also help to reduce symptoms from other obesity-related conditions such as gastroesophageal reflux and osteoarthritis. Long-term maintenance of such modest weight loss may reduce adverse clinical outcomes, including myocardial infarction, stroke and cardiovascular-related death.<sup>3–7</sup> Studies with patient follow-up of at least 2 years (preferably 4–6 years) are needed to demonstrate the clinical benefits of weight-loss interventions. Such studies have been undertaken involving patients receiving pharmacotherapy for diabetes and hypertension.<sup>8,9</sup>

The objective of this section is to determine the effects of long-term (> 1 year) pharmacologic weight-loss interventions on weight-loss efficacy, markers of cardiovascular disease

risk (e.g., glycemic and blood pressure control) and clinical outcomes.

## Data sources

The studies included in this analysis were RCTs that had a follow-up period of at least 1 year; included overweight or obese adults (BMI  $\geq 25$  kg/m<sup>2</sup>); and investigated weight loss with orlistat or sibutramine. (These are the only drugs used primarily for weight loss that have been approved for extended [ $> 3$  months] clinical use in Canada.)

Orlistat is a gastrointestinal lipase inhibitor that reduces fat absorption by about 30%<sup>10</sup> (Table 13). It is approved for clinical use for up to 2 years. Sibutramine is a serotonin and noradrenalin reuptake inhibitor that induces weight loss through enhanced satiety and increased basal energy expenditure.<sup>11</sup> Sibutramine is approved for clinical use for up to 1 year.

## Main findings

The 21 trials of pharmacotherapy for obesity that were identified involved a total of 11 533 participants.<sup>12–32</sup> All of the participants received a nonspecific, nutritionally balanced, reduced-energy diet in addition to either drug therapy or placebo. In all studies, the effect of pharmacotherapy on weight loss and other outcomes was assessed only during ongoing drug therapy and not after drug therapy was stopped.

## Weight-loss efficacy

The efficacy of pharmacotherapy for obesity was assessed based on absolute weight loss (a crude measure of decreased

Table 13: Characteristics of drugs approved for the treatment of obesity

Drug	Class	Recommended dose	Mode of action	Adverse effects
Orlistat	Gastrointestinal lipase inhibitor	120 mg 3 times daily (during or up to 1 h after meal)	<ul style="list-style-type: none"><li>• Pancreatic lipase inhibitor</li><li>• Reduces fat absorption by 30%</li></ul>	<ul style="list-style-type: none"><li>• Abdominal bloating, pain and cramping</li><li>• Steatorrhea</li><li>• Fecal incontinence</li></ul>
Sibutramine	Serotonin and noradrenalin reuptake inhibitor	10–15 mg once daily (in morning)	<ul style="list-style-type: none"><li>• Reduces food intake by enhancing satiety</li><li>• May increase thermogenesis</li><li>• May prevent decline in energy expenditure with weight loss</li></ul>	<ul style="list-style-type: none"><li>• Xerostomia</li><li>• Constipation</li><li>• Dizziness</li></ul>

adiposity) after 1, 2 and 4 years of treatment; maintenance of weight loss; and the proportion of patients attaining clinically important weight loss ( $\geq 5\%$  of total body weight).

The pharmacotherapy trials showed that treatment with orlistat (120 mg 3 times daily) or sibutramine (5–15 mg once daily), when combined with a reduced-energy diet, was associated with weight loss. The long-term (1–2 years) weight loss with pharmacologic therapy was approximately 6–7 kg, compared with weight loss of about 2–3 kg with diet-only therapy (Table 14).

## Maintenance of weight loss

In a review of long-term trials (> 1-year treatment and follow-up) comparing orlistat therapy plus a reduced-energy diet with diet-only therapy, the combined therapy was found to be more effective than diet alone in maintaining weight loss and preventing weight regain.<sup>1</sup>

Five studies<sup>13,16,21–23</sup> assessed orlistat therapy for at least 2 years and up to 4 years. The weight loss attained by 1 year was, in general, better maintained over the subsequent 1–3 years in patients who had received ongoing drug therapy. For example, in one study,<sup>22</sup> ongoing treatment for 2 years with orlistat was associated with less regain of lost weight compared with diet-only therapy (32% v. 63%;  $p < 0.001$ ). In another 2-year study,<sup>25</sup> patients had 6 months of weight-loss induction using diet-only therapy, followed by combined sibutramine–diet therapy or diet–placebo therapy. Again, combined therapy was more effective than diet only in preventing weight regain. In this study, 80% or more of the weight loss was maintained in 43% of patients in the sibutramine group, compared with only 16% of those in the diet-only group.

## Clinically important weight loss

Overall, clinically important weight loss ( $\geq 5\%$  of total body weight) occurred in 40%–60% of patients given orlistat or sibutramine. A meta-analysis<sup>4</sup> that compared combined pharmacologic–diet therapy and diet-only therapy found that the combination therapy was associated with an almost 3-fold greater likelihood of attaining clinically important weight loss (odds ratio 2.9, 95% confidence interval 2.5–3.5).

## Effects of pharmacotherapy on markers of cardiovascular disease risk

Assessment of the effects of pharmacologic therapy on markers of cardiovascular disease risk should consider the patient population being studied. Healthier patients, without risk factors for cardiovascular disease, will be less likely than patients with obesity-related risk factors to demonstrate benefits of weight-loss interventions. Patients with such risk factors as dysglycemia (i.e., type 2 diabetes, impaired glucose tolerance), hypertension or dyslipidemia are more likely to derive benefits from weight loss because of higher baseline levels of cardiovascular disease risk indices (blood pressure, blood glucose and lipid levels).

### Orlistat

Orlistat therapy was found to have consistent effects on glycemic control in patients with or without dysglycemia. Treatment with orlistat achieved modest but significantly greater reductions in fasting blood glucose level (0.1–1.7 mmol/L) than diet-only therapy in most studies.<sup>12,15,17–20,22–24</sup> In one study,<sup>14</sup> there was no significant difference in glycemic control between orlistat therapy and diet-only therapy. Among patients with type 2 diabetes, orlistat therapy was associated with significantly improved glycemic control (lower fasting blood glucose level) in 3 studies<sup>18,19,24</sup> and a reduction in oral hyperglycemic drug therapy requirements in 2 studies<sup>18,19</sup> compared with diet only.

The effect of orlistat therapy on blood pressure was inconsistent across studies. There was a significant reduction in both systolic blood pressure (8.6–11.0 mm Hg v. 11.5–13.3 mm Hg) and diastolic blood pressure (6.2–9.2 mm Hg v. 8.1–11.4 mm Hg) in 3 studies,<sup>14,19,20</sup> significant reductions in either systolic or diastolic pressure in 4 studies<sup>12,13,22,23</sup> and no significant reduction in blood pressure in 2 studies.<sup>15,17</sup> In one study<sup>13</sup> that assessed the effects of orlistat in obese patients with hypertension, this treatment was associated with a significant reduction in diastolic but not systolic pressure, and it was not associated with a reduction in antihypertensive drug requirements. In a review,<sup>33</sup> pooled subgroups of obese patients with hypertension, who received orlistat and diet therapy, had greater mean reductions in systolic and diastolic blood pressure than hypertensive obese patients receiving diet therapy alone. Orlistat therapy was also associated with significantly greater reductions in LDL cholesterol levels (0.11–0.38 mmol/L) than diet-only therapy.<sup>12–24</sup> However, orlistat had no significant effects on triglyceride levels, except in one study,<sup>24</sup> and no significant effects on HDL cholesterol levels.

Sibutramine therapy was found to have no significant effect on glycemic control in patients with or without dysglycemia.<sup>25–29,31,32</sup>

### Sibutramine

Sibutramine therapy was found to have no significant effect on glycemic control in patients with or without dysglycemia.<sup>25–29,31,32</sup>

**Table 14:** Effect of orlistat and sibutramine on weight loss

Variable	Orlistat studies		Sibutramine studies	
	Orlistat + diet	Diet alone	Sibutramine + diet	Diet alone
Baseline weight, kg, mean (SD)	100.6 (3.5)	100.6 (3.5)	96.1 (10.9)	95.5 (10.0)
Weight loss, kg, mean (SD)				
After 1 yr	6.5 (2.7)	3.8 (1.9)	6.0 (1.7)	1.6 (1.7)
After 2 yr	6.7 (1.4)	3.5 (1.6)	8.9 (8.1)*	4.9 (5.9)*
After 4 yr	5.8 (0.4)*	3.0 (0.4)*	NA	NA

Note: SD = standard deviation, NA = not available.

\*Data are from a single study.

However, it did improve glycemic control (lower fasting blood glucose, improved hemoglobin A<sub>1c</sub> levels) in 2 studies involving patients with type 2 diabetes.<sup>26,30</sup> Sibutramine treatment was, in general, associated with modest increases in blood pressure (1–3 mm Hg) and resting heart rate (2–4 beats/min) compared with diet-only therapy. The increase in blood pressure was statistically significant in 2 studies<sup>25,27</sup> but not in 6 other studies.<sup>26,28–32</sup> There were no significant effects of sibutramine therapy compared with diet-only therapy on LDL cholesterol levels<sup>25–27,30–32</sup> and inconsistent effects on HDL cholesterol and triglyceride levels.<sup>25,26,29–32</sup>

### Effects of pharmacotherapy on clinical outcomes

Few studies have been adequately powered, in terms of sample size or duration of patient follow-up, to assess the effect of weight loss on clinical outcomes, such as myocardial infarction, stroke and death from cardiovascular disease. In the Xenical in the Prevention of Diabetes in Obese Subjects (XENDOS) trial,<sup>16</sup> patients received treatment with orlistat or placebo for up to 4 years. Compared with placebo, orlistat therapy was associated with a decreased risk of type 2 diabetes among patients with impaired glucose tolerance (18.8% v. 28.8%;  $p = 0.002$ ). However, it did not reduce the risk of impaired glucose tolerance among patients with normal glucose tolerance (27.6% v. 30.5%;  $p = 0.15$ ) or the risk of diabetes in patients with normal glucose tolerance (2.6% v. 2.7%).

The ongoing Sibutramine Cardiovascular Outcomes Trial (SCOUT) is assessing the effects of sibutramine in addition to a lifestyle intervention program compared with lifestyle intervention alone on morbidity and mortality among over 9000 obese people with cardiovascular disease risk factors. The results of these and other large long-term studies are urgently needed to inform clinical practice regarding the potential benefits and safety of long-term pharmacotherapy.

### Head-to-head comparative drug studies

There are no long-term studies, to our knowledge, that have compared the efficacy of orlistat versus sibutramine for weight loss. The only trials comparing orlistat and sibutramine were short (3–6 months)<sup>34–36</sup> and did not satisfy the inclusion criteria for our analysis. Although these 3 trials demonstrated that sibutramine was associated with a significantly greater weight loss than orlistat, their results should be interpreted with caution because of their small size (80, 86 and 150 patients respectively). Furthermore, in one study sibutramine was administered at a dose not recommended for clinical use (10 mg twice daily). A study that assessed combined sibutramine–orlistat versus single-drug (orlistat or sibutramine) therapy found no significant difference between the treatment groups.<sup>35</sup>

### Practical treatment considerations

When pharmacotherapy is being considered for weight loss, it should be part of a comprehensive program that is anchored

in dietary therapy. The choice of drug therapy should be based on several factors, including the person's cardiovascular disease risk profile, dietary habits and concomitant disease.

Individuals who consume large food portions or who have irregular eating habits (frequent snacking) may benefit from the satiety-enhancing and anorexic properties of sibutramine. Those who consume large quantities of fatty foods may benefit from orlistat to limit intake.

Orlistat and sibutramine are associated with adverse effects that may result in drug intolerance and are not approved for clinical use longer than 2 years. Sibutramine should be avoided in people with ischemic heart disease, congestive heart failure or other chronic cardiac disease, and orlistat should be avoided in people with inflammatory or other chronic bowel disease. Furthermore, as with other potentially long-term pharmacologic interventions, monitoring for compliance and adverse drug effects is warranted. Finally, combining orlistat and sibutramine therapy is not advocated for clinical use.

### Study limitations

The main limitation — found consistently among pharmacotherapy studies and resulting in level 2, rather than level 1, evidence for the recommendations in this chapter — was the high proportion of study participants lost to follow-up. The standard for randomized trials is that the proportion of patients lost to follow-up should not exceed 20% of the initial study population.<sup>37</sup> The mean proportion (and range) of those lost to follow-up in the treatment arms of active 1-year and 2-year pharmacotherapy studies for obesity were 42% (21%–53%) and 36% (20%–48%), respectively. This limits the applicability of findings to clinical practice, because reported outcomes were based on the “last-observation-carried-forward” method of data analysis. This method documents weight loss before participants leave the study and carries this weight loss forward along with patients who complete the study. This approach may overestimate weight loss, particularly if those who do not complete the study regain lost weight. The method may be relevant only to patients who demonstrate long-term drug compliance.

Another limitation is the lack of data on the effects of weight loss on noncardiovascular outcomes, such as gastroesophageal reflux, osteoarthritis, skin disease, and mood and other psychological disorders. There is also a paucity of data on the effects of weight loss on quality-of-life measures.

### Emerging treatments

Currently, a number of new molecular entities are being developed for the treatment of obesity.<sup>38</sup> In contrast to earlier formulations, these are being specifically targeted at receptors and metabolic processes that are deemed relevant to energy metabolism based on our current understanding of the complex biology of ingestive behaviour, metabolism and energy expenditure. Among these emerging strategies, cannabinoid type 1 receptor (CB-1) antagonists currently appear to be the most promising. Rimonabant, the lead compound in this

new class of antiobesity agents, has been shown to be effective in reducing body weight and waist circumference as well as improving metabolic risk factors in overweight and obese patients.<sup>39</sup> Although several new compounds addressing a range of molecular targets are expected to become available for the treatment of obesity, it may take some years for these treatments to emerge.

## Conclusions

Among overweight or obese patients, pharmacotherapy with orlistat or sibutramine, administered in combination with a nutritionally balanced, energy-reduced diet, is superior to diet-only therapy. Studies suggest that pharmacotherapy will help individuals attain clinically important weight loss ( $\geq 5\%$  of total body weight) and maintain weight loss over 1–4 years. Among patients with type 2 diabetes or hypertension, pharmacotherapy is effective in improving glycemic and blood pressure control. It also reduces requirements for antihyperglycemic drugs. If sustained over the long-term and complemented by other pharmacologic and lifestyle interventions that improve glycemic and blood pressure control, these effects have the potential to reduce cardiovascular outcomes, such as myocardial infarction and stroke. Ultimately, large, long-term studies, similar to XENDOS and SCOUT, are needed to assess the safety of long-term pharmacotherapy. Large, long-term, RCTs are also needed to determine whether sustained weight loss confers benefits in terms of quality of life, cardiovascular outcomes and other clinical outcomes (e.g., mood disorders, sleep apnea and joint disorders). Although several new compounds are currently under investigation for the treatment of obesity, it may take some years for these treatments to become available.

## Recommendations

1. We suggest the addition of a selected pharmacologic agent for appropriate overweight or obese adults, who are not attaining or who are unable to maintain clinically important weight loss with dietary and exercise therapy, to assist in reducing obesity-related symptoms [*grade B, level 2*<sup>16,17,21–23,25</sup>].
2. We suggest the addition of a selected pharmacologic agent for overweight or obese adults with type 2 diabetes, impaired glucose tolerance or risk factors for type 2 diabetes, who are not attaining or who are unable to maintain clinically important weight loss with dietary and exercise therapy, to improve glycemic control and reduce their risk of type 2 diabetes [*grade B, level 2*<sup>13,16–19,24,26,30</sup>].

From the Department of Medicine, McMaster University, Hamilton, Ont. (both authors)

**Competing interests:** None declared for James Douketis. Arya Sharma is a consultant to Abbott Laboratories Ltd., Boehringer Ingelheim, Novartis, sanofi-aventis Canada Inc. and Merck Frosst Canada Ltd. He has received speaker fees from Abbott Laboratories Ltd., Boehringer Ingelheim, Astra-Zeneca Canada Inc., Novartis and Merck Frosst Canada Ltd. and travel assistance from Abbott Laboratories Ltd., Boehringer Ingelheim, Merck Frosst Canada Ltd., Novartis and sanofi-aventis Canada Inc.

## REFERENCES

1. Douketis JD, Macie C, Thabane L, et al. Systematic review of long-term weight loss studies in obese adults: clinical significance and applicability to clinical practice. *Int J Obes (Lond)* 2005;29:1153–67.
2. Padwal R, Li SK, Lau DC. Long-term pharmacotherapy for overweight and obesity: a systematic review and meta-analysis of randomized controlled trials. *Int J Obes Relat Metab Disord* 2003;27:1437–46.
3. Jeffcoate W. Obesity is a disease: food for thought. *Lancet* 1998;351:903–4.
4. Rippe JM, Crossley S, Ringer R. Obesity as a chronic disease: modern medical and lifestyle management. *J Am Diet Assoc* 1998;98(Suppl 2):S9–15.
5. National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults — the evidence report. *Obes Res* 1998;6(Suppl 2):51S–209S.
6. Executive summary of the clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults. *Arch Intern Med* 1998;158:1855–67.
7. Oster G, Thompson D, Edelsberg J, et al. Lifetime health and economic benefits of weight loss among obese persons. *Am J Public Health* 1999;89:1536–42.
8. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). *Lancet* 1998;352:837–53.
9. Neal B, MacMahon S, Chapman N. Effects of ACE inhibitors, calcium antagonists, and other blood-pressure-lowering drugs: results of prospectively designed overviews of randomised trials. Blood Pressure Lowering Treatment Trialists' Collaboration. *Lancet* 2000;356:1955–64.
10. Guerciolini R. Mode of action of orlistat. *Int J Obes Relat Metab Disord* 1997;21: S12–23.
11. Finer N. Sibutramine: its mode of action and efficacy. *Int J Obes Relat Metab Disord* 2002;26:S29–33.
12. Broom I, Wilding J, Stott P, et al. Randomised trial of the effect of orlistat on body weight and cardiovascular disease risk profile in obese patients: UK Multimorbidity Study. *Int J Clin Pract* 2002;56:494–9.
13. Bakris G, Calhoun D, Egan B, et al. Orlistat improves blood pressure control in obese subjects with treated but inadequately controlled hypertension. *J Hypertens* 2002;20:2257–67.
14. Hauptman J, Lucas C, Boldrin MN, et al. Orlistat in the long-term treatment of obesity in primary care settings. *Arch Fam Med* 2000;9:160–7.
15. Lindgarde F. The effect of orlistat on body weight and coronary heart disease risk profile in obese patients: the Swedish Multimorbidity Study. *J Intern Med* 2000; 248:245–54.
16. Torgerson JS, Hauptman J, Boldrin MN, et al. XENical in the prevention of Diabetes in Obese Subjects (XENDOS) study: a randomized study of orlistat as an adjunct to lifestyle changes for the prevention of type 2 diabetes in obese patients. *Diabetes Care* 2004;27:155–61.
17. Hanefeld M, Sachse G. The effects of orlistat on body weight and glycaemic control in overweight patients with type 2 diabetes: a randomized, placebo-controlled trial. *Diabetes Obes Metab* 2002;4:415–23.
18. Kelley DE, Bray GA, Pi-Sunyer FX, et al. Clinical efficacy of orlistat therapy in overweight and obese patients with insulin-treated type 2 diabetes: a 1-year randomized controlled trial. *Diabetes Care* 2002;25:1033–41.
19. Miles JM, Leiter L, Hollander P, et al. Effect of orlistat in overweight and obese patients with type 2 diabetes treated with metformin. *Diabetes Care* 2002;25:1123–8.
20. Rossner S, Sjostrom L, Noack R, et al. Weight loss, weight maintenance, and improved cardiovascular risk factors after 2 years treatment with orlistat for obesity. European Orlistat Obesity Study Group. *Obes Res* 2000;8:49–61.
21. Finer N, James WP, Kopelman PG, et al. One-year treatment of obesity: a randomized, double-blind, placebo-controlled, multicentre study of orlistat, a gastrointestinal lipase inhibitor. *Int J Obes Relat Metab Disord* 2000;24:306–13.
22. Davidson MH, Hauptman J, DiGirolamo M, et al. Weight control and risk factor reduction in obese subjects treated for 2 years with orlistat: a randomized controlled trial. *JAMA* 1999;281:235–42.
23. Sjostrom L, Rissanen A, Andersen T, et al. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group. *Lancet* 1998;352:167–72.
24. Hollander PA, Elbein SC, Hirsch IB, et al. Role of orlistat in the treatment of obese patients with type 2 diabetes. A 1-year randomized double-blind study. *Diabetes Care* 1998;21:1288–94.
25. James WP, Astrup A, Finer N, et al. Effect of sibutramine on weight maintenance after weight loss: a randomized trial. STORM Study Group. Sibutramine Trial of Obesity Reduction and Maintenance. *Lancet* 2000;356:2119–25.
26. McNulty SJ, Ur E, Williams G. A randomized trial of sibutramine in the management of obese type 2 diabetic patients treated with metformin. *Diabetes Care* 2003; 26:125–31.
27. McMahon FG, Fujioka K, Singh BN, et al. Efficacy and safety of sibutramine in obese white and African American patients with hypertension: a 1-year, double-blind, placebo-controlled, multicenter trial. *Arch Intern Med* 2000;160:2185–91.
28. Smith IG, Goulder MA. Randomized placebo-controlled trial of long-term treatment with sibutramine in mild to moderate obesity. *J Fam Pract* 2001;50:505–12.
29. McMahon FG, Weinstein SP, Rowe E, et al. Sibutramine is safe and effective for weight loss in obese patients whose hypertension is well controlled with angiotensin-converting enzyme inhibitors. *J Hum Hypertens* 2002;16:5–11.
30. Sanchez-Reyes L, Fanghanel G, Yamamoto J, et al. Use of sibutramine in overweight adult Hispanic patients with type 2 diabetes mellitus: a 12-month, random-

- ized, double-blind, placebo-controlled clinical trial. *Clin Ther* 2004;26:1427-35.
31. Porter JA, Raebel MA, Conner DA, et al. The Long-term Outcomes of Sibutramine Effectiveness on Weight (LOSE Weight) study: evaluating the role of drug therapy within a weight management program in a group-model health maintenance organization. *Am J Manag Care* 2004;10:369-76.
  32. Hauner H, Meier M, Wendland G, et al. Study Group SA. Weight reduction by sibutramine in obese subjects in primary care medicine: the SAT Study. *Exp Clin Endocrinol Diabetes* 2004;112:201-7.
  33. Jacob S. The management of hypertension in the overweight and obese patient: Is weight reduction sufficient? *Drugs* 2004;64:1263.
  34. Aydin N, Topsever P, Kaya A, et al. Orlistat, sibutramine, or combination therapy: which performs better on waist circumference in relation with body mass index in obese patients? *Tohoku J Exp Med* 2004;202:173-80.
  35. Gokcel A, Gumurdulu Y, Karakose H, et al. Evaluation of the safety and efficacy of sibutramine, orlistat and metformin in the treatment of obesity. *Diabetes Obes Metab* 2002;4:49-55.
  36. Kaya A, Aydin N, Topsever P, et al. Efficacy of sibutramine, orlistat and combination therapy on short-term weight management in obese patients. *Biomed Pharmacother* 2004;58:582-7.
  37. US Preventive Services Task Force. Screening for obesity in adults: recommendations and rationale. *Ann Intern Med* 2003;139:930-2.
  38. Correia ML, Haynes WG. Emerging drugs for obesity: linking novel biological mechanisms to pharmaceutical pipelines. *Expert Opin Emerg Drugs* 2005;10:643-60.
  39. Van Gaal LF, Rissanen AM, Scheen AJ, et al. Effects of the cannabinoid-1 receptor blocker rimonabant on weight reduction and cardiovascular risk factors in overweight patients: 1-year experience from the RIO-Europe study. *Lancet* 2005;365:1389-97.



# 15. Surgical treatment of morbid obesity

Daniel W. Birch, Simon Biron, D. Michael Grace

The commonly held belief that obese people can ameliorate their condition by simply deciding to eat less and exercise more is at odds with compelling scientific evidence indicating that the propensity to obesity is, to a significant extent, genetically determined.<sup>1</sup> Surgical treatment for weight loss in morbidly obese adults is the most efficacious treatment option available. The goal of bariatric surgery should be to relieve the person from his or morbid body weight, cure or improve comorbidity and improve quality of life.

In an update to the Periodic Health Examination, Douketis and colleagues<sup>2</sup> concluded that surgery achieves a magnitude of weight loss greater than all other treatment options. Moreover, they showed that surgically induced weight loss is sustainable in most patients.<sup>3</sup> Christou and coworkers<sup>4</sup> described the positive impact of surgery for weight loss on long-term mortality, morbidity and demand on the health care system.

One of the most important guidelines for surgery remains the 1991 National Institutes of Health (NIH) consensus development conference statement on gastrointestinal surgery for severe obesity.<sup>5</sup> Despite being prepared over 15 years ago, this statement contains important guiding principles not only for the indications for surgery to achieve weight loss, but also for the health care environment that must accompany this aggressive management option. The key points from this statement that have set such a high standard for the surgical man-

agement of morbid obesity are summarized in Box 5. The specific indications for weight loss surgery that have also stood the test of time are listed in Box 6.

When endorsing bariatric surgery, no one should forget that morbidly obese people have failed at all other means of weight loss. Satisfactory long-term results (> 5 years) should be the goal.

The objectives of this chapter are to review the data on the efficacy of surgery for morbid obesity, its impact on comorbid disease and its role in sustaining both quality and quantity of life. In addition, special attention will be paid to studies that examine the long-term results of weight loss surgery.

## Study selection

Several data sources specifically related to the surgical management of morbid obesity were considered. These sources include important manuscripts in the pre-appraised literature. We searched MEDLINE for articles using the MeSH headings “obesity” or “obesity, morbid” or “bariatric surgery,” with the search limited by the following criteria: human, English, all adult: 19+ years, articles published in the last 10 years and either practice guideline, guideline, consensus development conference, NIH or meta-analysis. We also searched the Web sites of the National Guidelines Clearinghouse ([www.guideline.gov](http://www.guideline.gov)), the Cochrane Collaboration ([www.cochrane.org](http://www.cochrane.org))

### Box 5: Key points from the National Institutes of Health consensus development conference statement on gastrointestinal surgery for morbid obesity<sup>5</sup>

- Individuals seeking therapy for severe obesity for the first time should be considered for treatment in a nonsurgical program with integrated components of a dietary regimen, appropriate exercise and behavioural modification and support
- Bariatric surgery or bariatric surgical procedures could be considered for well-informed and motivated individuals who have acceptable operative risks
- People who are candidates for surgical procedures should be selected carefully after evaluation by a multidisciplinary team with medical, surgical, psychiatric and nutritional expertise
- The operation should be performed by a surgeon who has substantial experience in the appropriate procedures and who is working in a clinical setting with adequate support for all aspects of management and assessment
- Lifelong medical surveillance after surgical therapy is a necessity

### Box 6: Specific indications for considering bariatric surgery in individuals who are morbidly obese

- Individuals whose BMI exceeds 40 kg/m<sup>2</sup> are potential candidates for bariatric surgery if they strongly desire substantial weight loss because their obesity severely impairs their quality of life. They must understand, clearly and realistically, how their lives may change after the operation
- In certain instances, individuals with less severe obesity (BMI 35-40 kg/m<sup>2</sup>) may also be considered candidates for surgery. Such people include those with high-risk comorbid conditions such as life-threatening cardiopulmonary problems, severe sleep apnea, severe diabetes mellitus or physical problems interfering with lifestyle (e.g., joint disease treatable except for the obesity or body-size problems precluding or severely interfering with employment, family function and ambulation).

Source: National Institutes of Health consensus development conference statement on gastrointestinal surgery for morbid obesity.<sup>5</sup>

and the National Institute for Clinical Evidence ([www.nice.org.uk](http://www.nice.org.uk)) using relevant search terms. Abstracts were reviewed and multiple publications based on similar source documents were excluded. Documents were excluded if they were not clearly based on a systematic review of the existing literature and reflected only expert opinion. Bibliographies were also reviewed and known documents were included if not identified through search of the above sources.

Systematic reviews and evidence-based clinical practice guidelines were identified from print or Internet-based sources.<sup>6-17</sup> Other important studies contributed important data to this chapter, including RCTs<sup>18</sup> and long-term cohort studies.<sup>19</sup> RCTs may not be the only appropriate standard of evidence<sup>20</sup> for evaluating most surgical treatment, and of necessity evaluations must rely on observational<sup>21</sup> or cohort studies or on standards of practice that have empirically been proven safe and beneficial.

## Patient selection

Bariatric surgery is effective and safe for morbidly obese individuals.<sup>4</sup> Patients should be selected for surgery according to NIH and World Health Organization (WHO) criteria. The NIH<sup>5</sup> and WHO<sup>22</sup> have established that the surgical approach should be considered for patients with a BMI  $\geq 40$  kg/m<sup>2</sup> or  $\geq 35$  kg/m<sup>2</sup> with comorbid conditions. We focused on BMI because it is linked with the broadest range of health outcomes.<sup>23</sup> A detailed review of selection criteria is available,<sup>24</sup> but under some circumstances surgery for older<sup>16</sup> or younger<sup>25</sup> patients may be considered. All retained surgical series adhere to those criteria.

## Surgical options

Once the only bariatric technique available, jejunio-ileal bypass has been abandoned.<sup>7,26,27</sup> We consider it here of historical interest only.

Surgical procedures for morbid obesity are typically categorized by their impact on overall food intake (restrictive) or absorption (malabsorptive). The current mainstream surgical options for weight loss include vertical banded gastroplasty and adjustable gastric banding (restrictive procedures); biliopancreatic diversion (malabsorptive procedure); and roux-en-Y gastric bypass and biliopancreatic bypass with duodenal switch (combined procedures). Each of these can be completed using an open or laparoscopic approach.

In 2003, roux-en-Y gastric bypass was the most commonly performed procedure worldwide for weight loss, and the laparoscopic approach was used more frequently than the open approach.<sup>28</sup> This approach to the surgical management of obesity has flourished since Wittgrove and colleagues<sup>29</sup> reported the first series of patients undergoing laparoscopic gastric bypass for obesity in 1994. The evolution from open surgery to a minimally invasive approach has not been entirely empirical but is based primarily on data from large surgical series rather than on later data from RCTs that compare open and laparoscopic approaches. The laparoscopic approach has virtually eliminated the wound complications associated with surgery in the obese patient (wound infection and incisional hernia) and

thereby dramatically reduced the burden of illness.<sup>14,18</sup> Laparoscopy has also affected the surgical management of morbid obesity by driving the adjustable gastric band to the forefront of surgical management, based not only on its favourable clinical outcomes but also its technical simplicity and procedural efficiency.<sup>10,28</sup> In an RCT comparing laparoscopic and open gastric bypass surgery, Nguyen and colleagues<sup>18</sup> showed further important clinical advantages of the laparoscopic approach, including a shorter length of stay in hospital, earlier return to activities of daily living and work, and improved quality of life based on SF-36 (Medical Outcomes Study 36-item Short Form) scores 1 month following surgery.

## Effectiveness of different procedures for weight loss

Compared with conventional (nonoperative) strategies, surgery achieves dramatically improved weight loss.<sup>2,12</sup> The Swedish Obese Subjects Study<sup>19</sup> is the principle study comparing conventional, nonsurgical management with surgery for morbid obesity. This cohort study demonstrated for the first time that surgical management is more efficacious than medical management in terms of producing weight loss, improving lifestyle and dramatically ameliorating comorbid disease. At 10 years of follow-up, the surgical cohort had maintained a weight loss greater than 16.1% of their original body weight, compared with a weight gain of 1.6% in the conventionally managed cohort, for a difference of 16.3% (95% CI 14.9%–17.6%).

Existing data are insufficient to identify the surgical procedure for weight loss that is the most efficacious and safe. Although the biliopancreatic diversion seems to result in the greatest degree of weight loss compared with other procedures, this conclusion is based on limited data.<sup>9,14,17</sup> A systematic literature review<sup>10</sup> of results with laparoscopic adjustable gastric banding (LAGB) showed it to be effective, at least up to 4 years.

Data are now emerging on the comparable rate of weight loss between the laparoscopic roux-en-Y gastric bypass (LRNYGB) and LAGB at 8 years of follow-up. The favourable mortality and morbidity profile of LAGB and its limited nutritional and metabolic impact may lead to its application in a greater number of patients and possibly those perceived to be at greatest risk (i.e., older patients).<sup>14,16</sup>

According to a meta-analysis,<sup>14</sup> at 3 years of follow-up, patients had achieved a weight loss of 41.46 kg following gastric bypass (95% CI 37.36–45.56 kg), 32.03 kg following vertical banded gastroplasty (95% CI 27.67–36.38 kg), 34.77 kg following adjustable gastric banding (95% CI 29.47–40.07 kg) and 53.10 kg following biliopancreatic diversion (95% CI 47.36–58.84 kg).

In a more recent systematic review<sup>17</sup> of medium-term weight loss, a pooled analysis of all bariatric procedures showed a greater than 60% excess weight loss at 10 years. Because of the different characteristics of weight loss, including regain of weight over time, with gastric bypass, LAGB and biliopancreatic diversion, there were no statistically significant differences between these procedures in percentage excess weight loss at 8–10 years.

## Morbidity and mortality associated with surgery for morbid obesity

Although ordinarily safe and effective,<sup>4</sup> bariatric surgery is a major operation. Mortality ranges from 0.1% for gastric banding and 0.5% for gastric bypass to 1.1% for biliopancreatic bypass and duodenal switch.<sup>9</sup> Leakage and pulmonary embolism have been the main causes of death. Incisional hernias have been much more common after open than after laparoscopic procedures.<sup>18</sup>

Three systematic reviews and meta-analyses<sup>9,10,14</sup> have addressed the morbidity and mortality associated with surgery to induce weight loss. The data on early perioperative complications and mortality following surgery for morbid obesity suggest that LAGB has a more favourable safety profile than gastric bypass, vertical banded gastroplasty and biliopancreatic diversion. Maggard and colleagues,<sup>14</sup> who focused on pooled evidence of postoperative adverse events, found that postoperative gastrointestinal symptoms were experienced by 16.9% of patients following gastric bypass, 17.5% of patients following vertical banded gastroplasty, 7.0% of patients following LAGB and 37.7% of patients following biliopancreatic diversion (19% following biliopancreatic diversion with duodenal switch<sup>30</sup>); surgical complications occurred in 18.7% of patients following gastric bypass, 23.7% following vertical banded gastroplasty, 13.2% following LAGB and 5.9% following biliopancreatic diversion.

Early mortality following weight loss surgery ranges from 0.4% to 1%.<sup>9,10,14</sup> Recently, an important publication identified early and late mortality that were at odds with these data. Flum and others<sup>31</sup> showed that mortality following surgery for morbid obesity varied with patient age and the annual volume of related procedures carried out by the surgical team. They also demonstrated that deaths related to surgery and its complications continued to accrue over time and represented an important, but poorly defined, aspect of surgical management. These data were collected from Medicare recipients and may reflect a Canadian patient population (public health care system) more accurately than typical surgical series from high-volume US centres (private payer health care).

## Effects of surgery and weight loss on clinical outcomes and quality of life

Weight loss alleviates many of the comorbid conditions associated with obesity. Bariatric surgery dramatically improves quality of life because of the significant weight loss and modified physiology following surgery. Numerous reports have been published on the effects of bariatric surgery on diabetes,<sup>32</sup> liver disease,<sup>33</sup> pregnancy outcomes<sup>34–36</sup> and musculoskeletal pain.<sup>37</sup> Studies also have shown that, following bariatric surgery, obstructive sleep apnea is cured 95% of the time.

One systematic review<sup>9</sup> that analyzed the impact of weight loss following surgery on comorbid disease identified a dramatic improvement. The authors found that type 2 diabetes, hyperlipidemia, hypertension and obstructive sleep apnea was resolved in most patients (62%–86%) following surgery

for weight loss. This parallels earlier data on the resolution of comorbid disease following weight loss by any means.<sup>2</sup>

The only comparison of the prevalence of comorbid disease among morbidly obese patient treated conventionally or surgically is the Swedish Obese Subjects Study.<sup>19</sup> A series of reports from this study showed that weight loss induced by surgery will lead to clinically important, long-term resolution or prevention of comorbid disease. These data now extend to 10 years of follow-up.

## Treatment team and hospital

Bariatric surgery is carried out by general surgeons with training in open and laparoscopic abdominal surgery. Residency experience in obesity surgery, attendance at training courses or experience in an obesity surgery centre is needed.

The complex nature of obesity and its emergence as a true chronic disease suggest that successful medical or surgical management may be achieved only through long-term follow-up and multidisciplinary care. This underlies the 1991 NIH consensus statement on surgical management<sup>5</sup> but may present certain barriers to the development of bariatric surgery because of resource restrictions in the health care system.

We suggest that the treatment team include a dietitian (certified nutritionist), an internist, an anesthetist, a psychiatrist or psychologist, nurses, a respiratory physician, a physiotherapist and a social worker.<sup>24</sup> Support groups are also helpful. Any hospital with a bariatric surgery program must have a full range of surgical and medical specialties for consultation and management of complications. An intensive care unit is essential. Equipment for large patients, including scales, beds, wheelchairs and bathrooms, are needed.

The favourable outcomes associated with the minimally invasive approach to surgery for morbid obesity have caused the number of such procedures to increase relatively rapidly.<sup>7,28</sup> There are data to suggest the existence of a threshold volume for generating acceptable outcomes. Flum and others<sup>31</sup> have clearly identified an impact of surgeon volume on postoperative mortality.

Kelly and coworkers<sup>13</sup> produced a guideline statement containing specific recommendations that include details on support of a surgical team and credentialing of the operating surgeon. The advanced skills required for a minimally invasive approach to the surgical management of obesity (gastric bypass or adjustable gastric band) suggest that surgeons must seek appropriate proctoring or mentoring to achieve safe and efficacious surgical management.

## Innovative therapies

Several surgical treatment options for weight loss should be considered experimental because of insufficient data on their efficacy, safety, mechanism of action and indications for use.

The inflatable gastric balloon acts as a restrictive device and is purported to produce early satiety based on its occupation of space in the stomach. Several types of gastric balloon exist with technical differences and varying characteristics. Limited data on their efficacy, complications associated with

previously tested devices, cost and limited duration of action (the device typically must be removed after 6 months) have greatly limited the acceptance of this option.<sup>38</sup>

Gastric electrical stimulation with implanted electrodes may produce early satiety by an (as yet) undefined physiologic mechanism. Preliminary data have demonstrated intriguing but modest weight-loss characteristics associated with this device. Clinical trials are underway and may yield important information in the near future.

Emerging data suggest a possible role for surgery in patients with a BMI of less than 35 kg/m<sup>2</sup> who have severe obesity-related comorbid disease.<sup>32,39</sup> In a recent consensus statement on bariatric surgery,<sup>11</sup> this issue was highlighted as requiring further investigation.

## Conclusions

Bariatric surgery is efficacious and safe in appropriately selected patients who are managed according to the NIH consensus statement<sup>5</sup> and WHO criteria.<sup>22</sup>

The short- and long-term clinical outcomes of surgery for morbid obesity are favourable compared with those of any other treatment option currently available. The magnitude of weight loss following surgery for morbid obesity, its sustainability and the impact of surgery on comorbid disease all suggest that there will be a dramatic increase in the number of procedures performed in Canada in the future. Program initiation, human resource development and training of the surgical team may represent important barriers to the expansion of surgery for morbid obesity in Canada.

## Recommendations

1. We suggest that adults with clinically severe obesity (BMI  $\geq 40$  kg/m<sup>2</sup> or  $\geq 35$  kg/m<sup>2</sup> with severe comorbid disease) may be considered for bariatric surgery when lifestyle intervention is inadequate to achieve healthy weight goals [*grade B, level 2*<sup>19</sup>].
2. We suggest that a minimally invasive approach be considered for weight loss surgery when an appropriately trained surgical team and appropriate resources are available in the operating theatre [*grade C, level 3*<sup>18</sup>].

From the Department of Surgery, University of Alberta, Edmonton, Alta. (Birch); Department of Surgery, Laval University, Québec, Que. (Biron); Department of Surgery, University of Western Ontario, London, Ont. (Grace)

**Competing interests:** Simon Biron and D. Michael Grace declare no competing interests. Daniel Birch has received speaker fees and an honorarium from Johnson & Johnson and Ethicon Endo-Surgery.

## REFERENCES

1. Friedman JM. Modern science versus the stigma of obesity. *Nat Med* 2004;10:563-9.
2. Douketis JD, Feightner JW, Attia J, et al. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. Canadian Task Force on Preventive Health Care. *CMAJ* 1999;160:513-25.
3. Douketis JD, Macie C, Thabane L, et al. Systematic review of long-term weight loss studies in obese adults: clinical significance and applicability to clinical practice. *Int J Obes (Lond)* 2005;29:1153-67.
4. Christou NV, Sampalis JS, Liberman M, et al. Surgery decreases long-term mortality, morbidity, and health care use in morbidly obese patients. *Ann Surg* 2004;240:416-23.
5. Gastrointestinal surgery for severe obesity: National Institutes of Health consensus development conference statement. *Am J Clin Nutr* 1992;55(2 Suppl):615S-9S.
6. NIH conference. Gastrointestinal surgery for severe obesity. Consensus Development Conference Panel. *Ann Intern Med* 1991;115:956-61.
7. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: executive summary. Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults. *Am J Clin Nutr* 1998;68:899-917.
8. National Institute for Clinical Excellence. *Guidance on the use of surgery to aid in weight reduction for people with morbid obesity*. London (UK): National Institute for Clinical Excellence; 2002. Technology appraisal guidance 46. Available: www.nice.org.uk/pdf/Fullguidance-PDF-morbid.pdf (accessed 2007 Feb 14).
9. Buchwald H, Avidor Y, Braunwald E, et al. Bariatric surgery: a systematic review and meta-analysis. *JAMA* 2004;292:1724-37.
10. Chapman AE, Kiroff G, Game P, et al. Laparoscopic adjustable gastric banding in the treatment of obesity: a systematic literature review. *Surgery* 2004;135:326-51.
11. Buchwald H. Bariatric surgery for morbid obesity: health implications for patients, health professionals, and third-party payers. *J Am Coll Surg* 2005;200:593-604.
12. Colquitt J, Clegg A, Loveman E, et al. Surgery for morbid obesity [Cochrane review]. *Cochrane Database Syst Rev* 2005;(4):CD003641.
13. Kelly J, Tarnoff M, Shikora S, et al. Best practice recommendations for surgical care in weight loss surgery. *Obes Res* 2005;13:227-33.
14. Maggard MA, Shugarman LR, Suttorp M, et al. Meta-analysis: surgical treatment of obesity. *Ann Intern Med* 2005;142:547-59.
15. Snow V, Barry P, Fitterman N, et al. Pharmacologic and surgical management of obesity in primary care: a clinical practice guideline from the American College of Physicians. *Ann Intern Med* 2005;142:525-31.
16. Villareal DT, Apovian CM, Kushner RF, et al. Obesity in older adults: technical review and position statement of the American Society for Nutrition and NAAO, The Obesity Society. *Am J Clin Nutr* 2005;82:923-34.
17. O'Brien PE, McPhail T, Chaston TB, et al. Systematic review of medium-term weight loss after bariatric operations. *Obes Surg* 2006;16:1032-40.
18. Nguyen NT, Goldman C, Rosenquist CJ, et al. Laparoscopic versus open gastric bypass: a randomized study of outcomes, quality of life, and costs. *Ann Surg* 2001;234:279-89.
19. Sjostrom L, Lindroos AK, Peltonen M, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. *N Engl J Med* 2004;351:2683-93.
20. Kral JG, Dixon JB, Horber FF, et al. Flaws in methods of evidence-based medicine may adversely affect public health directives. *Surgery* 2005;137:279-84.
21. Hu X, Wright JG, McLeod RS, et al. Observational studies as alternatives to randomized clinical trials in surgical clinical research. *Surgery* 1996;119:473-5.
22. *Obesity: preventing and managing the global epidemic of obesity. Report of a WHO Consultation*. Geneva: World Health Organization; 1998. Technical report series, 894.
23. McTigue KM, Harris R, Hemphill B, et al. Screening and interventions for obesity in adults: summary of the evidence for the U.S. Preventive Services Task Force. *Ann Intern Med* 2003;139:933-49.
24. Grace DM. Patient selection for obesity surgery. *Gastroenterol Clin North Am* 1987;16:399-413.
25. Strauss RS, Bradley LJ, Brolin RE. Gastric bypass surgery in adolescents with morbid obesity. *J Pediatr* 2001;138:499-504.
26. Griffen WO Jr, Bivins BA, Bell RM. The decline and fall of the jejunoileal bypass. *Surg Gynecol Obstet* 1983;157:301-8.
27. Jorgensen S, Olesen M, Gudman-Hoyer E. A review of 20 years of jejunoileal bypass. *Scand J Gastroenterol* 1997;32:334-9.
28. Buchwald H, Williams SE. Bariatric surgery worldwide. *Obes Surg* 2004;14:1157-64.
29. Wittgrove AC, Clark GW, Tremblay LJ. Laparoscopic gastric bypass, roux-en-Y: preliminary report of five cases. *Obes Surg* 1994;4:353-7.
30. Marceau P, Biron S, Bourque RA, et al. Biliopancreatic diversion with a new type of gastrectomy. *Obes Surg* 1993;3:29-35.
31. Flum DR, Salem L, Elrod JA, et al. Early mortality among Medicare beneficiaries undergoing bariatric surgical procedures. *JAMA* 2005;294:1903-8.
32. Pories WJ, Swanson MS, MacDonald KG, et al. Who would have thought it? An operation proves to be the most effective therapy for adult-onset diabetes mellitus. *Ann Surg* 1995;222:339-50.
33. Kral JG, Thung SN, Biron S, et al. Effects of surgical treatment of the metabolic syndrome on liver fibrosis and cirrhosis. *Surgery* 2004;135:48-58.
34. Richards DS, Miller DK, Goodman GN. Pregnancy after gastric bypass for morbid obesity. *J Reprod Med* 1987;32:172-6.
35. Dixon JB, Dixon ME, O'Brien PE. Pregnancy after lap-band surgery: management of the band to achieve healthy weight outcomes. *Obes Surg* 2001;11:59-65.
36. Marceau P, Kaufman D, Biron S, et al. Outcome of pregnancies after biliopancreatic diversion. *Obes Surg* 2004;14:318-24.
37. Peltonen M, Lindroos AK, Torgerson JS. Musculoskeletal pain in the obese: a comparison with a general population and long-term changes after conventional and surgical obesity treatment. *Pain* 2003;104:549-57.
38. Genco A, Cipriano M, Bacci V, et al. BioEnterics IntraGastric Balloon (BIB): a short-term, double-blind, randomised, controlled, crossover study on weight reduction in morbidly obese patients. *Int J Obes (Lond)* 2006;30:129-33.
39. Rubino F, Gagner M. Potential of surgery for curing type 2 diabetes mellitus. *Ann Surg* 2002;236:554-9.

# 16. Alternative treatments for obesity

Dominique Garrel

**T**he lack of efficacious long-term treatments for obesity and the rapid increase in the incidence of this disease favour the use of various alternative approaches. A review was undertaken of trials of alternative therapies that used plant extracts, fibres, metabolic compounds, hormones, homeopathy and acupuncture in the treatment of obesity. Except for the studies of acupuncture, all of the studies reviewed were controlled, randomized and double-blinded. Meta-analyses and reviews were included if they presented only controlled, randomized and double-blinded studies.

Data sources were PubMed, MEDLINE and the Cochrane Controlled Clinical Trials Register. No studies of 1 year's duration and only a few of 6 months' duration were found; almost all of the studies reported trials of a few weeks' duration.

In the following analysis, the results of the literature review are described regardless of the duration of the studies. A secondary analysis addressed studies of at least 6 months' duration (Table 15).<sup>1-10</sup> The recommendations in this chapter are based on the latter analysis.

## Plant extracts and polymers

*Ayurvedic herbs:* One study<sup>11</sup> randomly assigned 90 participants to 4 groups: 1 group received placebo and the other 3 received an ayurvedic preparation combined with triphala for 3 months. (Triphala is an ayurvedic compound formulated with 3 fruits and used for digestion and constipation.) Those given the ayurvedic preparation lost significantly more weight than those who took the placebo ( $p < 0.05$ ). There were no reports of adverse events.

*Glucomannan* is a water-soluble dietary fibre derived from konjac root. It is a polysaccharide similar to guar gum. In one clinical study,<sup>12</sup> 20 participants were randomly assigned to receive placebo or glucomannan (1 g 3 times daily) for 8 weeks. Those in the treatment group lost 2.5 kg on average. No side effects were observed in the study participants. The placebo group did not show significant weight loss, and the difference in weight loss between the placebo and treated groups at the end of the study was significant.

*Guar gum*, a water-soluble paste made from the seeds of the guar plant, is a fibre used for weight management. A recent meta-analysis<sup>13</sup> of 20 RCTs pooled the results of 11 trials of guar gum for weight reduction. The authors concluded that guar gum was not effective in reducing weight. It was also associated with minor gastrointestinal side effects. The conclusions of this meta-analysis should be accepted with caution because 10 of the 11 trials examined did not have

weight reduction as a primary outcome. There is no evidence to date that guar gum can help people achieve weight loss.

*Psyllium*, another soluble fibre, was tested in 125 people with type 2 diabetes for 6 weeks.<sup>14</sup> There was no effect on body weight in the treatment group compared with the placebo group.

*Chitosan* is a polymeric molecule derived from the exoskeleton of crustaceans. It has been studied in 5 RCTs and 1 meta-analysis (which analyzed 5 additional double-blinded studies).<sup>1,2,15,16</sup> The duration of the longest study was 24 weeks. There was no consistent effect of this product on body weight. Gastrointestinal problems, such as flatulence and bloating, were the side effects most frequently reported. There are insufficient data to recommend in favour of or against the use of chitosan for weight management in the obese person. Caution is advised, and the person should be monitored.

*Garcinia cambogia and hydroxy citric acid:* Hydroxy citric acid is derived from the tropical plant *Garcinia cambogia*. Both have been tested as agents for weight loss. However, results were inconsistent, and the duration of the trials was short. Mild gastrointestinal side effects were reported.<sup>14</sup>

*Ephedra sinica* or ma-huang is an herb used alone or in combination with caffeine. Ephedrine is the active constituent of the plant. In a recent meta-analysis of 52 trials and 65 case reports,<sup>17</sup> ephedrine, alone or in combination with caffeine, promoted a modest weight loss (0.6–1 kg per month). However, it increases the risk of psychiatric disorders, palpitation and gastrointestinal symptoms 2.2- to 3.6-fold. These substances are under restrictive regulations in both the United States and Canada, and their use to treat obesity should be discouraged.

*Chromium picolinate* contains a chromium molecule linked to picolinic acid. It has been studied as an insulin sensitizer and as a “metabolic” agent that increases energy expenditure and lean body mass. A meta-analysis of 10 double-blind RCTs<sup>18</sup> suggested a modest effect of this substance on weight reduction (0.08–0.2 kg per week). Although no side effects were reported, chromium picolinate is mutagenic in rats and induces DNA damage through oxidative stress. There are insufficient data to recommend in favour of or against the use of chromium picolinate for weight management in the obese person. Caution is advised, and the person should be monitored.

*Yerba mate (Ilex paraguariensis)*, a tea-like beverage widely consumed in South America, is associated with numerous health claims. Its use in obesity has been described in association with 2 other plant products: guarana and damiana. In a 45-day study, 94 overweight individuals were randomly as-

**Table 15:** Evidence from long-term randomized clinical trials of the effect of various alternative interventions on weight loss

Study	Participants			Duration, wk	Mean change			
	Sex	BMI, kg/m <sup>2</sup>	Intervention (dose)		Weight, kg	BMI, kg/m <sup>2</sup>	Waist circumference, cm	% body fat
<b>Chitosan</b>								
Mhurchu et al <sup>1</sup>	44 M	35.5	Chitosan (250 mg 12 times daily)	24	-0.39	-0.17	-0.57	-0.85
	206 F		Placebo	24	0.17	0.05	0.07	-0.61
Zahorska-Markiewicz et al <sup>2</sup>	50 F	> 30	Chitosan (750 mg 6 times daily) + LCD*	24	-15.9†	ND	ND	ND
			Placebo + LCD*	24	-10.9			
<b>Chromium picolinate</b>								
Bahadori et al <sup>3</sup>	36	33.7	Chromium yeast (200 µg/d)	26	ND	-2.5	ND	-3.75
			Chromium picolinate (200 µg/d)	26		-2.1		0.81§
			Placebo	26		-2.0		-1.75
<b>Ephedra sinica</b>								
Boozer et al <sup>4</sup>	30 M	31.8	Ephedra (90 mg/d) + caffeine (192 mg/d)	24	-5.3¶	ND	-6**	-4.3††
	137 F		Placebo	24	-2.6		-2	-2.7
Quaade et al <sup>5</sup>	155 F 25 M	33.7	Ephedrine (60 mg/d)	24	-14.3	ND		
			Caffeine (60 mg/d)	24	-11.5			
			Ephedrine (60 mg/d) + caffeine (600 mg/d)	24	-16.6			
			Placebo	24	-13.2			
<b>Yohimbe</b>								
Sax <sup>6</sup>	47 M	ND	Yohimbine (peak dose 43 mg/d) + diet‡‡	24	-8.7	ND	-3.2	-6.2
			Placebo + diet‡‡	24	-9.4		-3.8	-6.1
<b>Growth hormone</b>								
Karlsson et al <sup>7</sup>	30 M	25-35	Growth hormone (9.5 µg/kg daily = 966 µg/d)	36	-1.0	32§§	-3.0¶¶	2.0
			Placebo	36	0.5	-12§§	-0.1	0.7
Johannsson et al <sup>8</sup>	30 M	31	Growth hormone (9.5 µg/kg daily = 966 µg/d)	36	0		-9.2%	2.0
			Placebo	36	0			0.7
Albert and Mooradian <sup>9</sup>	44 F	36.5	Growth hormone (F: 6.0 µg/kg = 600 µg M: 3.8 µg/kg = 400 µg)	24	-2.4***		-3.4†††	
	15 M		Placebo	24	-0.9		-1.5	
Herrmann et al <sup>10</sup>	25 M	33.4	Metformin (850 mg twice daily) + placebo	72			-2.7	
			Metformin + growth hormone (9.5 µg/kg per day)	72			-4.3	

Note: LCD = low-calorie diet, ND = not determined.

\*1000 kcal/d, physical activity and behaviour modification.

†Significantly different from placebo group ( $p < 0.05$ ).

‡8 weeks very-low-calorie diet plus 18 weeks maintenance diet.

§Significantly different from other 2 groups ( $p < 0.029$ ).

¶Significantly different from placebo group ( $p < 0.001$ ).

\*\*Significantly different from placebo group ( $p = 0.005$ ) and baseline ( $p < 0.001$ ).

††Significantly different from placebo group ( $p = 0.020$ ).

‡‡1800 kcal/d and < 25% fat.

§§Basal metabolic rate.

¶¶Significantly different from baseline level ( $p < 0.01$ ).

\*\*\*Significantly different from baseline level ( $p = 0.04$ ).

†††Significantly different from baseline level ( $p = 0.0001$ ).

signed to receive the plant preparation or a placebo.<sup>14</sup> A difference of 4.8-kg in weight loss between the 2 groups was seen. No side effects were reported. A delay in gastric emptying was noted. This plant deserves additional investigation.

*Yohimbe*, which is extracted from the bark of an African tree, is an alpha-2 receptor-antagonist. Results of 3 double-blind RCTs, 1 lasting 6 months, have been published.<sup>14</sup> There was no consistent effect on weight loss. Side effects included headache, nervousness and arthralgia. A possible dangerous interaction between yohimbine and sibutramine, a noradrenergic reuptake inhibitor, has been suggested.<sup>19</sup> There are insufficient data to recommend in favour of or against the use of yohimbe for weight management in the obese person. Caution is advised, and the person should be monitored.

## Hormones

Thyroid hormones have been used for weight loss because of their thermogenic effect. Severe cardiovascular complications have led to the cessation of this practice.

**Growth hormone:** The lipolytic and anabolic properties of growth hormone make this substance a good candidate for the treatment of obesity. Unfortunately, initial investigations were interrupted because administration of growth hormone decreased insulin sensitivity and caused arthritis. Recently, low doses of growth hormone (400 µg in men and 600 µg in women) were studied for 6 months.<sup>9</sup> Participants lost 2.4 kg on average. No changes in blood pressure or insulin sensitivity were noted.

Because of insufficient data, there is no recommendation in favour of or against the use of growth hormone (low-dose regimens) for weight management in the obese person. The use of recombinant human growth hormone in doses above those recommended for adults with growth deficiency (200–700 µg/d) is not recommended. Additional studies are needed to assess the effect of long-term low-dose administration of this hormone.

**Human chorionic gonadotrophin (hCG):** This hormonal preparation has been widely used to treat obesity with the claim that it has “lipolytic” effects, particularly in the gluteal region. Two extensive reviews of the literature<sup>20,21</sup> analyzed 16 uncontrolled studies and 8 controlled studies. Only one of these studies suggested that hCG was useful. All of the studies were performed in combination with very-low-calorie diets. From these data, it is concluded that hCG should not be used to treat obesity.

## Acupuncture

Seven observational studies of short duration have been reviewed.<sup>22</sup> However, methodological difficulties precluded adequate control and randomization, and the scientific merit of this method has not been evaluated.

## Homeopathy

One trial involving 166 participants receiving *Helianthus tuberosus* (Jerusalem artichoke) or a placebo for 12 weeks showed a greater weight loss with the product than with the

placebo (7.2 kg v. 4.7 kg).<sup>12</sup> However, there are insufficient data to recommend in favour of or against homeopathy for the treatment of obesity.

## Recommendation

There is insufficient evidence to recommend in favour of or against the use of herbal remedies, dietary supplements or homeopathy for weight management in the obese person [grade C, level 4<sup>1,3,4,7–11,15,16,19,20,22,23</sup>].

From the Department of Nutrition, University of Montréal, Montréal, Que.

**Competing interests:** Dominique Garrel is a member of advisory boards for Abbott Laboratories Ltd. and AstraZeneca Canada Inc. He has received an educational grant from AstraZeneca Canada Inc. and speaker fees from Abbott Laboratories Ltd.

## REFERENCES

1. Mhurchu CN, Poppitt SD, McGill AT, et al. The effect of the dietary supplement, chitosan, on body weight: a randomised controlled trial in 250 overweight and obese adults. *Int J Obes Relat Metab Disord* 2004;28:1149–56.
2. Zahorska-Markiewicz B, Krotkiewski M, Olszanecka-Glinianowicz M, et al. Effect of chitosan in complex management of obesity. *Pol Merkuriusz Lekarski* 2002;13:129–32.
3. Bahadori B, Wallner S, Schneider H, et al. Effect of chromium yeast and chromium picolinate on body composition of obese, non-diabetic patients during and after a formula diet. *Acta Med Austriaca* 1997;24:185–7.
4. Boozer CN, Daly PA, Homel P, et al. Herbal ephedra/caffeine for weight loss: a 6-month randomized safety and efficacy trial. *Int J Obes Relat Metab Disord* 2002;26:593–604.
5. Quaade F, Astrup A, Breum L, et al. The effect of an ephedrine/caffeine combination as a supplement to a weight-reducing diet. A randomized, placebo-controlled, double-blind trial. *Ugeskr Laeger* 1992;154:1258–63.
6. Sax L. Yohimbine does not affect fat distribution in men. *Int J Obes* 1991;15:561–5.
7. Karlsson C, Stenlof K, Johannsson G, et al. Effects of growth hormone treatment on the leptin system and on energy expenditure in abdominally obese men. *Eur J Endocrinol* 1998;138:408–14.
8. Johannsson G, Marin P, Lonn L, et al. Growth hormone treatment of abdominally obese men reduces abdominal fat mass, improves glucose and lipoprotein metabolism, and reduces diastolic blood pressure. *J Clin Endocrinol Metab* 1997;82:727–34.
9. Albert SG, Mooradian AD. Low-dose recombinant human growth hormone as adjuvant therapy to lifestyle modifications in the management of obesity. *J Clin Endocrinol Metab* 2004;89:695–701.
10. Herrmann BL, Berg C, Vogel E, et al. Effects of a combination of recombinant human growth hormone with metformin on glucose metabolism and body composition in patients with metabolic syndrome. *Horm Metab Res* 2004;36:54–61.
11. Paranjpe P, Patki P, Patwardhan B. Ayurvedic treatment of obesity: a randomised double-blind, placebo-controlled clinical trial. *J Ethnopharmacol* 1990;29:1–11.
12. Werk W, Galland F. *Helianthus tuberosus* in the therapy of obesity. Long term stabilization of weight reduction [in German]. *Therapiewoche* 1994;44:34–9.
13. Pittler MH, Ernst E. Guar gum for body weight reduction: meta-analysis of randomized trials. *Am J Med* 2001;110:724–30.
14. Pittler MH, Ernst E. Dietary supplements for body-weight reduction: a systematic review. *Am J Clin Nutr* 2004;79:529–36.
15. Pittler MH, Abbot NC, Harkness EF, et al. Randomized, double-blind trial of chitosan for body weight reduction. *Eur J Clin Nutr* 1999;53:379–81.
16. Ernst E, Pittler MH. Chitosan as a treatment for body weight reduction? A meta-analysis. *Perfusion* 1998;11:461–5.
17. Shekelle PG, Hardy ML, Morton SC, et al. Efficacy and safety of ephedra and ephedrine for weight loss and athletic performance: a meta-analysis. *JAMA* 2003;289:1537–45.
18. Pittler MH, Stevinson C, Ernst E. Chromium picolinate for reducing body weight: meta-analysis of randomized trials. *Int J Obes Relat Metab Disord* 2003;27:522–9.
19. Jordan J, Sharma AM. Potential for sibutramine–yohimbine interaction? *Lancet* 2003;361:1826.
20. Bray GA, Greenway FL. Current and potential drugs for treatment of obesity. *Endocr Rev* 1999;20:805–75.
21. Lijesen GK, Theeuwes I, Assendelft WJ, et al. The effect of human chorionic gonadotropin (hCG) in the treatment of obesity by means of the Simeons therapy: a criteria-based meta-analysis. *Br J Clin Pharmacol* 1995;40:237–43.
22. Lacey JM, Tershakovec AM, Foster GD. Acupuncture for the treatment of obesity: a review of the evidence. *Int J Obes Relat Metab Disord* 2003;27:419–27.
23. Pasmán WJ, Westerterp-Plantenga MS, Saris WH. The effectiveness of long-term supplementation of carbohydrate, chromium, fibre and caffeine on weight maintenance. *Int J Obes Relat Metab Disord* 1997;21:1143–51.

# 17. Dietary intervention for the treatment of pediatric obesity

Linda Gillis, Tracy Hussey, Julie Lenk, Geoff Ball, Jacquie Jumpsen, Rhona Hanning, Linda McCargar

**W**eight loss can be achieved when energy expended in activity is greater than energy consumed.<sup>1</sup> Various dietary approaches have been undertaken to lower energy intake to promote weight loss in the pediatric population. These include low-energy diets, very-low-calorie diets and other healthy eating approaches. Macronutrient dietary alterations (e.g., high-protein, high-fibre, low-glycemic-index or low-fat diets) have been attempted to reduce obesity. Alterations in micronutrients, such as calcium, have also been suggested. The purpose of this section is to review current dietary investigations and recommend optimal dietary treatment to promote healthy weights within the context of normal growth patterns for pediatric patients with obesity.

## Restricted diet

### Very-low-calorie diets

Very-low-calorie diets, such as the protein-sparing modified fast (PSMF), have been shown to be successful at promoting long-term weight loss in a few studies with a small number of participants. The PSMF diet involves a daily intake of 600–800 kcal, 1.5–2.5 g of protein per kilogram of ideal body weight and no more than 20–40 g of carbohydrates.<sup>2</sup> Although 8 studies have reported using the PSMF diet in the pediatric population,<sup>3–10</sup> only 3 had a follow-up period of 1 year,<sup>4,8,10</sup> and only 1 was an RCT.<sup>4</sup> In the 3 longer trials, the average decrease in body weight at 1 year was 27%, but the dropout rate was 29% on average. When Figueroa-Colon and others<sup>4</sup> randomly assigned participants to receive either the PSMF diet or a balanced very-low-calorie diet, the PSMF diet was more successful in reducing weight. However, given the paucity of data in this area, it is difficult to conclude that this diet is beneficial.

### Low-energy diets

Low-energy diets provide 500–1000 kcal a day less than normal requirements to promote a loss of 0.5–1 kg a week.<sup>2</sup> Various approaches are used, such as teaching general health principles, teaching about the Food Guide Pyramid or following the American Dietetic Association exchange program.<sup>11–15</sup> Many studies use 1 of these methods as a behavioural intervention. Low-energy diets are generally successful for weight loss, but it is difficult to compare studies because of variations in duration of the diets, length of follow-up and the use of inpatient versus outpatient treatment.

Only 1 study of low-energy diets was an RCT. Following 10 months of education on the Food Guide Pyramid in an inpatient setting, a 48% reduction in median-adjusted BMI was noted in the low-energy diet group, compared with a 6% reduction in the control group. It is not stated in the paper whether those in the control group continued with their normal intake. After 6 months, the median BMI increased 6% in the treatment group, with an additional increase of 4% at 14 months.<sup>13</sup>

## The Traffic Light Diet

The Traffic Light Diet (TLD), which is sometimes referred to as the Stop-Light Diet, was developed during the 1970s by Leonard Epstein and colleagues at the University of Pittsburgh. It has been used most often by Epstein and colleagues as the dietary component of a family-based treatment program for obese preschool (1–5 years old) and preadolescent (6–12 years old) children. Use of the TLD in the adolescent population is limited. We found only 1 study for this population; Johnson and colleagues<sup>16</sup> investigated the influence of nutrition (using the TLD) and exercise interventions on weight loss and lipid profiles of children and adolescents 8–17 years of age.

Based on the Food Guide Pyramid, the TLD is used to promote a balanced diet and decreased energy intake (900–1500 kcal/d). Foods in the TLD are grouped into categories corresponding to traffic light colours: green (go), yellow (caution) and red (stop). Generally, “green” foods contain 0–1.9 g of fat per serving, “yellow” foods contain 2.0–4.9 g of fat per serving and “red” foods contain 5 g or more of fat per serving.<sup>17</sup> The focus is on increasing the intake of green (low-energy/high-nutrient density) foods, particularly fruits and vegetables, and decreasing intake of red (high-energy/low-nutrient density) foods. Consumption of yellow (higher in energy/high-nutrient density) foods is encouraged to be consistent with the recommendations of the Food Guide Pyramid to ensure adequate nutrient intake.

Currently, no comparative studies have critically evaluated the TLD against another dietary strategy. However, when the TLD has been used as part of a comprehensive pediatric weight-management program (including behaviour change strategies, exercise and family components), it has resulted in improvements in overweight status at 5 and 10 years post-intervention.<sup>18–20</sup> Based on these data, it is unknown whether successful weight management (achieved, in part, through use of the TLD) led to concurrent changes in risk factors for



type 2 diabetes (e.g., insulin resistance) or cardiovascular disease (e.g., high blood pressure, low HDL cholesterol).

In addition to long-term improvements in weight, the TLD has led to improved nutrient density<sup>21</sup> and macronutrient composition.<sup>22,23</sup> Reductions in total energy intake<sup>21</sup> and in the number of “red” foods consumed have also been observed.<sup>22–24</sup> Studies by Duffy and colleagues<sup>22</sup> and Epstein and others<sup>23</sup> showed significant associations between the number of “red” foods consumed and a decrease in percentage overweight and weight loss, respectively.

Two studies assessed nutrient intake and growth of 17 obese children, ages 1–6 years,<sup>25</sup> and 12 obese children, ages 8–12 years,<sup>21</sup> who followed the TLD in a family-based weight-control program. Results were similar for all children: a decrease in energy and nutrient intake and an increase in nutrient density. More detailed analysis showed that children consumed an adequate intake of all nutrients based on the US Recommended Dietary Allowances (RDAs); consumption was above 100% of the RDA for most nutrients, except calcium, iron<sup>21,25</sup> riboflavin and thiamin.<sup>21</sup> These same studies revealed that long-term growth (assessed from height percentiles) was not compromised in children who followed a short-term, energy-restricted TLD that may have been low in some nutrients.<sup>21,25</sup> However, these results were derived from a small sample of obese children and may not represent growth and nutrient intake for all obese children who may follow the TLD.

As a component of the TLD, energy intake restricted to 900–1500 kcal/d, depending on the age and overweight status of the child, is recommended. Although the goal of the TLD is to increase consumption of “green” foods and decrease consumption of “red” foods, few studies have analyzed the number of “red” foods consumed or children’s adherence to the energy restrictions. This limit makes it difficult to determine whether the components of the TLD are affecting outcomes or whether the results are due to energy restriction or other strategies used in the family-based weight-control program.

We found no studies that critically evaluated the TLD compared with any other dietary strategy. Before recommendations on the use of the TLD for treatment of childhood obesity can be made, additional research is needed to determine its effectiveness compared with another dietary strategy, both with and without energy restriction. Further research on the TLD in diverse populations is also needed.

## Altered macronutrient intake

### High-protein diet

Eight studies investigated the efficacy of a high-protein diet for weight loss in a pediatric population. Of these, 4 are not discussed in detail because follow-up was only 2–5 months. Of the remaining 4, 1 study group constituted children<sup>4</sup> and the other 3 adolescents.<sup>10,26,27</sup> All 4 studies included follow-up assessments at 12 months (or later). Since 2 studies did not control for energy intake between the diet treatments,<sup>4,26</sup> no conclusions can be drawn regarding the influence of

macronutrient composition alone. When equal-energy diets were provided,<sup>27</sup> there were no differences in outcomes between the high-protein and balanced-diet groups. However, the levels of protein provided may have been too similar (15% and 19% of energy). Stallings and colleagues<sup>10</sup> implemented a high-protein modified fast for 3 months and a mixed diet for the remaining 9 months. The weight of 17 obese adolescents decreased from 154% to 125% of ideal body weight at 3 months, and the 12 who attended a final measurement session maintained the weight loss at 12 months. No comparison group was assessed. Thus, there is no strong evidence to recommend a high-protein diet for weight loss in children.

### High-fibre diet

Only 1 study was found<sup>28</sup> that investigated the effectiveness of high-fibre diets in treating pediatric obesity. In this randomized, double-blind, crossover study, either 15-g dietary fibre capsules or placebo were administered for 4 weeks. The resulting difference in weight loss — 0.336 kg in the treatment group v. 0.033 kg in the placebo group — was not significant. Because of the small sample ( $n=8$ ), poor compliance (4 of 8 participants did not take all the supplements) and short duration (4 weeks), no conclusions can be made regarding high-fibre diets.

### Low-glycemic-index diet

Although several studies have demonstrated that a diet emphasizing glycemic index and glycemic load (GI/GL) may improve health outcomes in overweight and obese adults,<sup>29–31</sup> there are insufficient data in the pediatric literature to arrive at a definitive conclusion. However, considering the results of adult studies and the few reports from children and adolescents, a low GI/GL diet might be a useful strategy in pediatric weight management. We found 3 studies that investigated the effect of a GI/GL diet in a pediatric population: 2 did not control for energy intake between the intervention and comparison groups,<sup>32,33</sup> and the third did not have a control group.<sup>34</sup> Additional research that includes overweight children and adolescents of different ethnic groups and ages with and without energy restriction is strongly encouraged. It is important to assess dietary efficacy, acceptability, long-term compliance, impact of the diet on overweight status and physical and psychosocial health risks.

### Low-fat diets

The definition of low fat in the literature is inconsistent, although it appears to refer to a fat intake of  $\leq 30\%$  of energy. This is a broad definition, however, since the range encompasses rates as low as 10%–15% and as high as 35%. Perhaps this reflects controversy with regard to safety, because it has been suggested that expressing dietary composition goals in terms of fat as a percentage of energy is inadequate if total energy intake is not specified as well.<sup>35</sup>

Many studies that reported BMI or other anthropometric

measurements indicated that the amount of fat in the diet was not related to these measures.<sup>36-44</sup> One study even reported the highest BMI in the low-fat intake group.<sup>45</sup>

In general, epidemiologic data do not suggest problems associated with a fat intake of 30% of energy. However, as Butte<sup>46</sup> points out, the metabolic consequences of varying fat content in the diet of infants and children has not been thoroughly studied. Some researchers suggest that more attention should be paid to the psychosocial effects of lowering fat intake,<sup>47</sup> or the possible long-term implications with respect to hormonal<sup>48</sup> or other changes not yet considered. At this point, we cannot say whether the benefits outweigh the harms.

In general, dietary factors associated with “health benefits” include the types of fats consumed and their ratios. When combined with carbohydrates derived mainly from whole-grain cereals, fruits and vegetables, pulses and nuts, moderately high fat intakes (30%–35% of total energy) have been associated with lower rates of obesity, diabetes and cardiovascular disease in adults.<sup>49</sup> Well-designed studies considering these dietary factors are required before the “optimal” amount and type of fat intake for children can be determined. Perhaps for some genetic predispositions, a “low-fat intake” of 30% of energy may be warranted, but a general recommendation is not supported.

## Altered micronutrient intake

In the pediatric literature, there are no intervention trials using calcium or dairy products to promote weight loss. In longitudinal studies, researchers have shown that high intakes of calcium and servings of dairy products are associated with less body fat.<sup>50-53</sup>

## Dieting and the development of eating disorders

It has been suggested that there is an association between dieting and the development of eating disorders; however, no direct causal relation has been established or reported. The association appears common in groups of “moderate to severe dieters”<sup>54</sup> or among those exhibiting “unhealthy” dieting behaviours.<sup>55</sup> Recent publications indicate that professionally administered weight-loss interventions in overweight children and adolescents pose a negligible risk of precipitating eating disorders.<sup>55-57</sup>

## Conclusions

The best treatment for obesity in the pediatric population has not yet been defined. Given the multifactorial origin of obesity, perhaps the focus should remain on targeting all modifiable factors. The type of fat consumed, rather than the amount of fat, may be more important in terms of disease risk. In addition, increasing physical activity and promoting adequate daily intakes of vegetables, fruits and whole grains have been convincingly shown to decrease the risk of all diet-related chronic diseases (including overweight and obesity).

## Recommendations

1. We suggest that the optimal dietary plan for achieving healthy body weight and dietary counselling for adolescents and children be developed with a qualified and experienced health professional (preferably a registered dietitian) together with the individual and family to meet their needs [*grade B, level 2*].
2. We recommend that a nutritionally balanced diet (designed to reduce energy intake) be combined with other supportive interventions to achieve a healthy body weight in overweight and obese people of all ages and to ensure the maintenance of growth in adolescents and youth [*grade C, level 4*].
3. Long-term, randomized controlled trials of nutritional therapy for obesity treatment in the pediatric population are urgently required. Such studies should consider different ethnic groups and ages, should be with and without energy restriction and should address dietary efficacy, acceptability, long-term compliance, impact of the diet on overweight status, and physical and psychological health risks [*grade C, level 4*].

From McMaster Children's Hospital, Hamilton, Ont. (Gillis, Hussey); the University of Alberta, Edmonton, Alta. (Ball, Lenk, McCargar); Capital Health, Edmonton, Alta. (Jumpsen); and the University of Waterloo, Waterloo, Ont. (Hanning)

**Competing interests:** None declared.

## REFERENCES

1. Epstein LH, Myers MD, Raynor HA, et al. Treatment of pediatric obesity. *Pediatrics* 1998;101:554-70.
2. Yanovski JA. Intensive therapies for pediatric obesity. *Pediatr Clin North Am* 2001; 48:1041-53.
3. Dietz WH Jr, Schoeller DA. Optimal dietary therapy for obese adolescents: comparison of protein plus glucose and protein plus fat. *J Pediatr* 1982;100:638-44.
4. Figueroa-Colon R, von Almen TK, Franklin FA, et al. Comparison of two hypocaloric diets in obese children. *Am J Dis Child* 1993;147:160-6.
5. Merritt RJ, Bistran BR, Blackburn GL, et al. Consequences of modified fasting in obese pediatric and adolescent patients. I. Protein-sparing modified fast. *J Pediatr* 1980;96:13-9.
6. Pencharz PB, Motil KJ, Parsons HG, et al. The effect of an energy-restricted diet on the protein metabolism of obese adolescents: nitrogen-balance and whole-body nitrogen turnover. *Clin Sci* 1980;59:13-8.
7. Pencharz PB, Clarke R, Archibald EH, et al. The effect of a weight-reducing diet on the nitrogen metabolism of obese adolescents. *Can J Physiol Pharmacol* 1988;66: 1469-74.
8. Sothorn. Udall JN, Jr., Suskind RM, Vargas A, Blecker U. Weight loss and growth velocity in obese children after very low calorie diet, exercise, and behavior modification. *Acta Paediatr* 2000;89:1036-43.
9. Stallings VA, Archibald EH, Pencharz PB. Potassium, magnesium, and calcium balance in obese adolescents on a protein-sparing modified fast. *Am J Clin Nutr* 1988;47:220-4.
10. Stallings VA, Archibald EH, Pencharz PB, et al. One-year follow-up of weight, total body potassium, and total body nitrogen in obese adolescents treated with the protein-sparing modified fast. *Am J Clin Nutr* 1988;48:91-4.
11. Becque MD, Katch VL, Rocchini AP, et al. Coronary risk incidence of obese adolescents: reduction by exercise plus diet intervention. *Pediatrics* 1988;81:605-12.
12. Braet C, Van Winckel M, Van Leeuwen K. Follow-up results of different treatment programs for obese children. *Acta Paediatr* 1997;86:397-402.
13. Braet C, Tanghe A, Bode PD, et al. Inpatient treatment of obese children: a multi-component programme without stringent calorie restriction. *Eur J Pediatr* 2003; 162:391-6.
14. Dao HH, Frelut ML, Oberlin F, et al. Effects of a multidisciplinary weight loss intervention on body composition in obese adolescents. *Int J Obes Relat Metab Disord* 2004;28:290-9.
15. Rocchini AP, Katch V, Anderson J, et al. Blood pressure in obese adolescents: effect of weight loss. *Pediatrics* 1988;82:16-23.
16. Johnson WG, Hinkle LK, Carr RE, et al. Dietary and exercise interventions for juve-

- nile obesity: long-term effect of behavioral and public health models. *Obes Res* 1997;5:257-61.
17. Epstein LH, Roemmich JN, Raynor HA. Behavioral therapy in the treatment of pediatric obesity. *Pediatr Clin North Am* 2001;48:981-93.
  18. Epstein LH, McCurley J, Wing RR, et al. Five-year follow-up of family-based behavioral treatments for childhood obesity. *J Consult Clin Psychol* 1990;58:661-4.
  19. Epstein LH, Valoski A, Wing RR, et al. Ten-year follow-up of behavioral, family-based treatment for obese children. *JAMA* 1990;264:2519-23.
  20. Epstein LH, Valoski A, Wing RR, et al. Ten-year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychol* 1994;13:373-83.
  21. Valoski A, Epstein LH. Nutrient intake of obese children in a family-based behavioral weight control program. *Int J Obes* 1990;14:667-77.
  22. Duffy G, Spence SH. The effectiveness of cognitive self-management as an adjunct to a behavioural intervention for childhood obesity: a research note. *J Child Psychol Psychiatry* 1993;34:1043-50.
  23. Epstein LH, Wing RR, Koeske R, et al. Child and parent weight loss in family-based behavior modification programs. *J Consult Clin Psychol* 1981;49:674-85.
  24. Graves T, Meyers AW, Clark L. An evaluation of parental problem-solving training in the behavioral treatment of childhood obesity. *J Consult Clin Psychol* 1988;56:246-50.
  25. Epstein LH, Valoski A, Koeske R, et al. Family-based behavioral weight control in obese young children. *J Am Diet Assoc* 1986;86:481-4.
  26. Sondike SB, Copperman N, Jacobson MS. Effects of a low-carbohydrate diet on weight loss and cardiovascular risk factor in overweight adolescents. *J Pediatr* 2003;142:253-8.
  27. Rolland-Cachera MF, Thibault H, Souberbielle JC, et al. Massive obesity in adolescents: dietary interventions and behaviours associated with weight regain at 2 y follow-up. *Int J Obes Relat Metab Disord* 2004;28:514-9.
  28. Gropper SS, Acosta PB. The therapeutic effect of fiber in treating obesity. *J Am Coll Nutr* 1987;6:533-5.
  29. Kelly S, Frost G, Whittaker V, et al. Low glycaemic index diets for coronary heart disease. *Cochrane Database Syst Rev* 2004; (4):CD004467.
  30. Opperman AM, Venter CS, Oosthuizen W, et al. Meta-analysis of the health effects of using the glycaemic index in meal-planning. *Br J Nutr* 2004;92:367-81.
  31. Pereira MA, Swain J, Goldfine AB, et al. Effects of a low-glycemic load diet on resting energy expenditure and heart disease risk factors during weight loss. *JAMA* 2004;292:2482-90.
  32. Ebbeling CB, Leidig MM, Sinclair KB, et al. A reduced-glycemic load diet in the treatment of adolescent obesity. *Arch Pediatr Adolesc Med* 2003;157:773-9.
  33. Spieth LE, Harnish JD, Lenders CM, et al. A low-glycemic index diet in the treatment of pediatric obesity. *Arch Pediatr Adolesc Med* 2000;154:947-51.
  34. Young PC, West SA, Ortiz K, et al. A pilot study to determine the feasibility of the low glycemic index diet as a treatment for overweight children in primary care practice. *Ambul Pediatr* 2004;4:28-33.
  35. Milner JA, Allison RG. The role of dietary fat in child nutrition and development: summary of an ASNS workshop. American Society for Nutritional Sciences. *J Nutr* 1999;129:2094-105.
  36. Boulton TJ, Magarey AM. Effects of differences in dietary fat on growth, energy and nutrient intake from infancy to eight years of age. *Acta Paediatr* 1995;84:146-50.
  37. Lagstrom H, Seppanen R, Jokinen E, et al. Influence of dietary fat on the nutrient intake and growth of children from 1 to 5 y of age: the Special Turku Coronary Risk Factor Intervention Project. *Am J Clin Nutr* 1999;69:516-23.
  38. Lapinleimu H, Viikari J, Jokinen E, et al. Prospective randomised trial in 1062 infants of diet low in saturated fat and cholesterol. *Lancet* 1995;345:471-6.
  39. Nicklas TA, Webber LS, Koschak M, et al. Nutrient adequacy of low fat intakes for children: the Bogalusa Heart Study. *Pediatrics* 1992;89:221-8.
  40. Niinikoski H, Viikari J, Ronnema T, et al. Regulation of growth of 7- to 36-month-old children by energy and fat intake in the prospective, randomized STRIP baby trial. *Pediatrics* 1997;100:810-6.
  41. Obarzanek E, Hunsberger SA, Van Horn L, et al. Safety of a fat-reduced diet: the Dietary Intervention Study in Children (Drosoph Inf ServC). *Pediatrics* 1997;100:51-9.
  42. Shea S, Basch CE, Stein AD, et al. Is there a relationship between dietary fat and stature or growth in children three to five years of age? *Pediatrics* 1993;92:579-86.
  43. Togo P, Osler M, Sorensen TI, et al. Food intake patterns and body mass index in observational studies. *Int J Obes Relat Metab Disord* 2001;25:1741-51.
  44. Ylönen K, Virtanen SM, Ala-Venna E, et al. Composition of diet in relation to fat intake of children aged 1-7 years. *J Hum Nutr Diet* 1996;9:207-18.
  45. Alexy U, Sichert-Hellert W, Kersting M, et al. Pattern of long-term fat intake and BMI during childhood and adolescence—results of the DONALD Study. *Int J Obes Relat Metab Disord* 2004;28:1203-9.
  46. Butte NF. Fat intake of children in relation to energy requirements. *Am J Clin Nutr* 2000;72(5 Suppl):1246S-52S.
  47. Satter E. A moderate view on fat restriction for young children. *J Am Diet Assoc* 2000;100:32-6.
  48. Dorgan JF, Hunsberger SA, McMahon RP, et al. Diet and sex hormones in girls: findings from a randomized controlled clinical trial. *J Natl Cancer Inst* 2003;95:132-41.
  49. Key TJ, Thorogood M, Appleby PN, et al. Dietary habits and mortality in 11,000 vegetarians and health conscious people: results of a 17 year follow up. *BMJ* 1996;313:775-9.
  50. Carruth BR, Skinner JD. The role of dietary calcium and other nutrients in moderating body fat in preschool children. *Int J Obes Relat Metab Disord* 2001;25:559-66.
  51. Novotny R, Daida YG, Acharya S, et al. Dairy intake is associated with lower body fat and soda intake with greater weight in adolescent girls. *J Nutr* 2004;134:1905-9.
  52. Phillips SM, Bandini LG, Cyr H, et al. Dairy food consumption and body weight and fatness studied longitudinally over the adolescent period. *Int J Obes Relat Metab Disord* 2003;27:1106-13.
  53. Skinner JD, Bounds W, Carruth BR, et al. Longitudinal calcium intake is negatively related to children's body fat indexes. *J Am Diet Assoc* 2003;103:1626-31.
  54. Patton GC, Selzer R, Coffey C, et al. Onset of adolescent eating disorders: population based cohort study over 3 years. *BMJ* 1999;318:765-8.
  55. Butryn ML, Wadden TA. Treatment of overweight in children and adolescents: does dieting increase the risk of eating disorders? *Int J Eat Disord* 2005;37:285-93.
  56. Braet C, Tanghe A, Decaluwe V, et al. Inpatient treatment for children with obesity: weight loss, psychological well-being, and eating behavior. *J Pediatr Psychol* 2004;29:519-29.
  57. Tanofsky-Kraff M, Faden D, Yanovski SZ, et al. The perceived onset of dieting and loss of control eating behaviors in overweight children. *Int J Eat Disord* 2005;38:112-22.

# 18. Physical activity and exercise therapy in children and adolescents

Oded Bar-Or

The importance of physical exercise for obese or overweight children and adolescents is similar to that for obese or overweight adults. However, there are age-related and maturation-related differences in the pattern of habitual physical activity and in the response to enhanced physical activity. The purpose of this chapter is to describe these differences and to propose recommendations that are appropriate to the pediatric population.

Chapter 19 of these guidelines addresses the combined effects of dietary changes and enhanced physical activity in the treatment of pediatric overweight and obesity. This chapter focuses on studies that compared different exercise treatments, or exercise with no exercise, although some of the studies also included dietary interventions.

## Data sources

The original intent was to include only RCTs with exercise interventions of at least 12 months' duration, in which the independent variables included BMI or percentage body fat. However, the results of the literature search were poor. The paucity of studies in this area was also reported in 2 Cochrane reviews<sup>1,2</sup> as well as in comprehensive reviews by the British National Health System Centre for Reviews and Dissemination.<sup>3-5</sup> Hence, this chapter addresses mainly RCTs that lasted less than 12 months. However, studies that did not address BMI or percentage body fat as dependent variables have been excluded.

## Habitual physical activity in children and adolescents

Various cross-sectional studies showed that obese children and adolescents were less habitually active than their non-obese peers.<sup>6,7</sup> Some studies suggested that the level of adiposity was inversely related to level of physical activity,<sup>7</sup> but others did not show such a relation.<sup>8,9</sup> However, on balance, there is enough evidence to conclude that, as a group, obese children and adolescents are less physically active than their nonobese peers.<sup>10,11</sup>

A relation between obesity and total daily energy expenditure is less clear. Although 1 study reported lower energy expenditure in obese preschool children,<sup>12</sup> another reported no difference in energy expenditure between obese and non-obese boys,<sup>13</sup> and another reported higher energy expenditure in obese than in nonobese adolescents.<sup>14</sup> One reason for such a discrepancy is the way in which energy expenditure is re-

ported. For example, in 1 study, absolute 24-hour energy expenditure, determined using doubly labelled water, was reported to be higher in obese adolescents than in their non-obese peers, but lower in the obese group corrected for body mass.<sup>14</sup>

## Effects of enhanced physical activity on body weight and composition

Enhanced physical activity does not seem to affect the body weight and composition of healthy, nonobese children and youth. However, there is sufficient indication to suggest that enhanced physical activity, aerobic exercise in particular, is beneficial to obese children and adolescents.<sup>4,5,10,11</sup> Most of the studies in this field compared the effects of diet-plus-exercise interventions with the outcomes of exercise alone.<sup>15-18</sup> Although the inclusion of diet (and, sometimes, behaviour modification) in both arms of the RCT does not allow a focus on the possible benefits of exercise alone, it does reflect real-life situations in which therapy is multidisciplinary. Few studies examine exercise as the only intervention. In these studies, the beneficial effect on body weight or composition is similar to that of the multidisciplinary intervention.<sup>19,20</sup>

Of major importance is the possible effect of enhanced physical activity on abdominal and visceral fat. Studies in adults have shown a beneficial effect. In children, 1 RCT<sup>20</sup> included 76 obese girls and boys (aged 7-11 years) who were randomly assigned to either a 4-month moderate-intensity aerobic program (45 minutes, 5 times per week) or a non-exercise regimen. The exercise group had a significant decrease in visceral fat and a significantly lower increase in subcutaneous abdominal fat. There are no data regarding the effect of cessation of the intervention on these variables.

## Effects of enhanced physical activity on comorbidities

In several small-scale RCTs, enhanced physical activity was shown to affect obesity-related comorbidities. However, it is not clear whether the beneficial effect was due to the exercise or was secondary to a reduction in adiposity.

## Insulin resistance

A crossover study<sup>21</sup> involving obese children (aged 7-11 years) was designed to assess the effects of enhanced physical activ-

ity and its cessation on fasting insulin levels as a surrogate measure of insulin resistance. A 4-month exercise program of moderate intensity (45 minutes, 5 times a week) induced a reduction in fasting plasma insulin levels. However, insulin levels reverted to pre-intervention values 4 months after cessation of the intervention.

## Hypertension

In some studies,<sup>22–24</sup> aerobic exercise was accompanied by a reduction in resting arterial blood pressure in obese adolescents, including those with hypertension. In contrast, an 8-week intervention of aerobic exercise did not result in a reduction in resting systolic or diastolic pressure in obese children<sup>25</sup> and adolescents.<sup>26</sup> Likewise, 4 months of aerobic exercise did not show changes in systolic blood pressure or in total peripheral resistance in obese children aged 7–11 years.<sup>27</sup> The reason for such inconsistency is unclear. It probably reflects differences in exercise intensity and duration, which may or may not have induced hemodynamic changes.

## Dyslipidemia

The effect of enhanced physical activity on plasma lipid levels is unclear. Although some studies reported a beneficial effect, others did not. For example, a 20-week diet-plus-exercise program resulted in a rise in HDL cholesterol, compared with no change in the diet-only group.<sup>22</sup> In another study, 4 months of aerobic exercise resulted in a decrease in the level of total triglycerides in obese children aged 7–11 years.<sup>21</sup> In contrast, a recent 12-month study<sup>28</sup> showed no difference in the effect on total cholesterol between the diet-plus-exercise and diet-only groups.

## Effects of reduced sedentary pursuits on obesity

An important area of research concerns the possibility that obese children can benefit from a reduction in sedentary activities, such as watching television and playing computer and video games. Two RCTs carried out by Epstein and colleagues<sup>15,29</sup> showed that a reduction in sedentary activities was more efficacious than an increase in physical activity (both groups were also given dietary education). In terms of design and execution, these 2 studies are considered of high quality.<sup>4</sup> They are among the few RCTs with a large enough sample (90 participants in the 2000 study and 61 in the 1995 study) to provide sufficient statistical power. Results from a 12-week randomized 2-arm pilot study involving 10 obese children<sup>30</sup> have suggested that contingent television watching (i.e., allowing children to watch television if they pedal a stationary cycle) can be highly successful in reducing overall television time, as well as in reducing body adiposity. This finding is in line with a school-based RCT<sup>31</sup> in which a reduction in television watching over a school year resulted in a decrease in age-adjusted BMI. To date, this is the only large-scale RCT that has documented a significant decrease in BMI in a general school population. The mechanism for the bene-

fit of reduced sedentary pursuits is unclear, because spending much time on such pursuits does not mean that the child's overall daily energy expenditure is reduced<sup>32</sup> or that energy consumption is increased.<sup>33</sup> Likewise, data regarding children's weight and amount of television viewing<sup>34</sup> or time spent on computer or video games are inconsistent.<sup>35,36</sup>

Despite the above uncertainties, it seems that an exercise-related prescription for obese children should include a reduction in time spent on sedentary pursuits, television watching in particular.

## Current knowledge regarding exercise prescription for obese children and youth

For adults, data are available regarding the type, optimal duration, frequency and intensity of prescribed exercise. These data are derived from studies that monitored morbidity-related and mortality-related end points (e.g., first myocardial infarction, survival after myocardial infarction, mortality). Such studies are not available for the pediatric population. Thus, even though abundant studies have examined the effects of enhanced physical activity on juvenile obesity, conclusive, evidence-based recommendations on optimal exercise prescription, including duration and frequency, cannot be made.

During the 1990s and early 2000s, several groups published guidelines for physical activity in the general population of children and adolescents. For example, in 1991 the US Department of Health and Human Services released *Healthy People 2000: National Health Promotion and Disease Prevention Objectives*,<sup>37</sup> a strategy for improving the health of Americans by the end of the century. It promotes 30 minutes or more a day of light to moderate exercise. In 1994 a consensus group published guidelines for adolescents that recommend 30 minutes or more of moderate-to-high intensity exercise 3 or more times a week.<sup>38</sup> In 1997 the US Centers for Disease Control and Prevention (CDC) published guidelines for children and youth.<sup>39</sup> Like the previous documents, these guidelines recommend 30 minutes a day of medium-to-high intensity exercise on most (preferably all) days of the week. In contrast, guidelines from the American College of Sports Medicine, the British Health Authority and, more recently, the CDC suggest that total daily activity last 60 minutes.

The problem with all of these guidelines is that they are not based on studies in which various exercise doses were compared. They were, by necessity, constructed through consensus of their authors.

Canada's physical activity guidelines for children and adolescents were published in 2002 through the combined effort of Health Canada and the Canadian Society for Exercise Physiology.<sup>40</sup> Other contributors were the Canadian Pediatric Society, the College of Family Physicians of Canada and the Canadian Physical Activity Unit of the Public Health Agency of Canada.

Unlike previous guidelines that specify a duration of physical activity, the Canadian guidelines recommend an *increment* to activities already performed by the child. The assumption is that our children and adolescents are insufficiently active, and

therefore any increment to their current activity pattern will be better than the status quo. Another important element in the Canadian guidelines is that any increase in the time devoted to exercise should be coupled with a decrease in “screen time” (i.e., watching television, playing computer or video games, participating in chat groups). This approach frees time for the child to become more physically active. Furthermore, the reduction in sedentary pursuits has its own merits, as discussed above.

## Conclusions

When explaining to a child the need for enhanced physical activity, health professionals should emphasize the tangible, short-term benefits, not the long-term health benefits. They should emphasize nonregimented lifestyle activities, such as games or a paper route, as these are more efficacious and induce better adherence than regimented aerobic activities. Physicians should also prescribe a reduction in sedentary pursuits, since this is a key element in exercise prescription.

Child-oriented prescription of exercise should always include fun and recreation.

Health professionals should emphasize activities that suit the individual’s relative strengths. Obese children are tall, buoyant in the water and often strong; thus, examples of suitable activities are basketball, lineman in football, shot-put and water-based games. It is important for health professionals not to prescribe activities in which the obese child is likely to fail (e.g., jumping, sprinting, rope climbing).

Health practitioners should consider including resistance or strength training, supervised by an adult, in the exercise prescription. The dividends of resistance training — higher fat-free mass and muscle strength — are visible within weeks. This speedy result increases the child’s motivation and adherence.

Health professionals should use the Canada physical activity guides for children and adolescents<sup>40</sup> to help increase activity time and decrease “screen time.”

## Recommendations

1. We recommend that the primary care physician or health care team encourage children and adolescents to reduce sedentary pursuits and “screen time” (i.e., television, video games) [*grade A, level 2<sup>4,15,29</sup>*].
2. We recommend that activity prescribed for children be fun and recreational, with lifestyle activities tailored to the relative strengths of the individual child and family [*grade A, level 2<sup>41</sup>*]. Health professionals are encouraged to emphasize the short-term benefits of physical activity rather than the long-term health benefits to children [*grade C, level 4*].

Oded Bar-Or is deceased. At the time of writing, he was with the Children’s Exercise and Nutrition Centre, Department of Pediatrics, McMaster University, Hamilton, Ont.

**Competing interests:** None declared.

## REFERENCES

1. Campbell K, Waters E, O’Meara S, et al. Interventions for preventing obesity in children. *Cochrane Database Syst Rev* 2002;(2):CD001871.
2. Wilson P, O’Meara S, Summerbell C, et al. The prevention and treatment of childhood obesity. *Qual Saf Health Care* 2003;12:65-74.
3. The prevention and treatment of childhood obesity. *Effective Health Care* 2002;7:1-12.
4. Reilly JJ, McDowell ZC. Physical activity interventions in the prevention and treatment of paediatric obesity: systematic review and critical appraisal. *Proc Nutr Soc* 2003;62:611-9.
5. Watts K, Jones TW, Davis EA, et al. Exercise training in obese children and adolescents: current concepts. *Sports Med* 2005;35:375-92.
6. Fontvieille AM, Kriska A, Ravussin E. Decreased physical activity in Pima Indian compared with Caucasian children. *Int J Obes Relat Metab Disord* 1993;17:445-52.
7. Pate R, Ross JG. The national children and youth fitness study II: factors associated with health-related fitness. *J Phys Ed Rec Dance* 1987;58:93-5.
8. Sallis JF, Prochaska JJ, Taylor WC. A review of correlates of physical activity of children and adolescents. *Med Sci Sports Exerc* 2000;32:963-75.
9. Maffei C, Talamini G, Tato L. Influence of diet, physical activity and parents’ obesity on children’s adiposity: a four-year longitudinal study. *Int J Obes Relat Metab Disord* 1998;22:758-64.
10. Bar-Or O, Baranowski T. Physical activity, adiposity, and obesity among adolescents. *Pediatr Exerc Sci* 1994;6:348-60.
11. Bar-Or O, Foreyt J, Bouchard C, et al. Physical activity, genetic, and nutritional considerations in childhood weight management. *Med Sci Sports Exerc* 1998;30:2-10.
12. Davies PS, Gregory J, White A. Physical activity and body fatness in pre-school children. *Int J Obes Relat Metab Disord* 1995;19:6-10.
13. Waxman M, Stunkard AJ. Caloric intake and expenditure of obese boys. *J Pediatr* 1980;96:187-93.
14. Bandini LG, Schoeller DA, Dietz WH. Energy expenditure in obese and nonobese adolescents. *Pediatr Res* 1990;27:198-203.
15. Epstein LH, Valoski AM, Vara LS, et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. *Health Psychol* 1995;14:109-15.
16. Epstein LH, Wing RR, Koeske R, et al. Effects of diet plus exercise on weight change in parents and children. *J Consult Clin Psychol* 1984;52:429-37.
17. Hills AP, Parker AW. Obesity management via diet and exercise intervention. *Child Care Health Dev* 1988;14:409-16.
18. Reybrouck T, Vinckx J, Van den Berghe G, et al. Exercise therapy and hypocaloric diet in the treatment of obese children and adolescents. *Acta Paediatr Scand* 1990;79:84-9.
19. Gutin B, Owens S, Riggs S, et al. Effect of physical training on cardiovascular health in obese children. In: Armstrong N, Kirby B, Welsman J, editors. *Children and exercise XIX: promoting health and well-being / XIXth International Symposium of Pediatric Work Physiology, Exeter, 1997*. London (UK): E & FN Spon; 1997. p. 382-9.
20. Owens S, Gutin B, Allison J, et al. Effect of physical training on total and visceral fat in obese children. *Med Sci Sports Exerc* 1999;31:143-8.
21. Ferguson MA, Gutin B, Le NA, et al. Effects of exercise training and its cessation on components of the insulin resistance syndrome in obese children. *Int J Obes Relat Metab Disord* 1999;23:889-95.
22. Becque MD, Katch VL, Rocchini AP, et al. Coronary risk incidence of obese adolescents: reduction by exercise plus diet intervention. *Pediatrics* 1988;81:605-12.
23. Brownell KD, Kelman JH, Stunkard AJ. Treatment of obese children with and without their mothers: changes in weight and blood pressure. *Pediatrics* 1983;71:515-23.
24. Rocchini AP, Katch V, Anderson J, et al. Blood pressure in obese adolescents: effect of weight loss. *Pediatrics* 1988;82:16-23.
25. Watts K, Beye P, Siafarikas A, et al. Effects of exercise training on vascular function in obese children. *J Pediatr* 2004;144:620-5.
26. Watts K, Beye P, Siafarikas A, et al. Exercise training normalizes vascular dysfunction and improves central adiposity in obese adolescents. *J Am Coll Cardiol* 2004;43:1823-7.
27. Humphries MC, Gutin B, Barbeau P, et al. Relations of adiposity and effects of training on the left ventricle in obese youths. *Med Sci Sports Exerc* 2002;34:1428-35.
28. Woo KS, Chook P, Yu CW, et al. Effects of diet and exercise on obesity-related vascular dysfunction in children. *Circulation* 2004;109:1981-6.
29. Epstein LH, Paluch RA, Gordy CC, Dorn J. Decreasing sedentary behaviors in treating pediatric obesity. *Arch Pediatr Adolesc Med* 2000;154:220-6.
30. Faith MS, Berman N, Heo M, et al. Effects of contingent television on physical activity and television viewing in obese children. *Pediatrics* 2001;107:1043-8.
31. Robinson TN. Reducing children’s television viewing to prevent obesity: a randomized controlled trial. *Jama* 1999;282:1561-7.
32. Montgomery C, Reilly JJ, Jackson DM, et al. Relation between physical activity and energy expenditure in a representative sample of young children. *Am J Clin Nutr* 2004;80:591-6.
33. Matheson DM, Wang Y, Klesges LM, et al. African-American girls’ dietary intake while watching television. *Obes Res* 2004;12:32S-37S.
34. Matheson DM, Killen JD, Wang Y, et al. Children’s food consumption during television viewing. *Am J Clin Nutr* 2004;79:1088-94.
35. Stettler N, Signer TM, Suter PM. Electronic games and environmental factors associated with childhood obesity in Switzerland. *Obes Res* 2004;12:896-903.
36. Vandewater EA, Shim MS, Caplovitz AG. Linking obesity and activity level with children’s television and video game use. *J Adolesc* 2004;27:71-85.

37. *Healthy people 2000: national health promotion and disease prevention objectives*. Washington (DC): US Department of Health and Human Services, Public Health Service; 1991.
38. Sallis JF. Physical activity guidelines for adolescents. *Pediatr Exerc Sci* 1994;6: 299-463.
39. Guidelines for school and community programs to promote lifelong physical activity among young people. Centers for Disease Control and Prevention. *MMWR Recomm Rep* 1997;46(RR-6):1-36.
40. *Canada's physical activity guides for children and youth*. Ottawa: Health Canada and the Canadian Society for Exercise Physiology; 2002. Available: [www.phac-aspc.gc.ca/pau-uap/paguide/child\\_youth/index.html](http://www.phac-aspc.gc.ca/pau-uap/paguide/child_youth/index.html) (accessed 2007 Feb 16).
41. Epstein LH, Wing RR, Koeske R, et al. A comparison of lifestyle exercise, aerobic exercise, and calisthenics on weight loss in obese children. *Behavior Therapy* 1985; 16:345-56.

# 19. Combined diet and exercise in the treatment of pediatric overweight and obesity

Bobbi N. Barbarich, Cathy Kubrak, Rhona Hanning, Linda McCargar

For children over 2 years of age, Health Canada recommends *Eating Well with Canada's Food Guide*,<sup>1</sup> which includes a range in the number of servings based on age and energy needs, and *Canada's Physical Activity Guides for Children and Youth*,<sup>2</sup> which recommends 30 minutes of physical activity a day over the initial 3–6 months, increasing after that to 90 minutes a day, while reducing sedentary activity by 60–90 minutes a day. Despite these recommendations, obesity among Canadian children remains a growing clinical concern.

## Data sources

Our objective was to review studies that have investigated the effects of diet therapy in combination with exercise in the treatment of childhood obesity. Key search terms included childhood obesity, diet, exercise, physical activity, treatment and nutritional therapy. Because few studies in the pediatric literature had a duration and follow-up of  $\geq 1$  year, we included those that lasted a minimum of 6 months.

## Current knowledge regarding the treatment of pediatric obesity

Evaluation of treatments is complicated by the lack of an international consensus on the definition of childhood obesity.<sup>3</sup> For the purposes of this section, the definition of obesity used in the studies examined was adopted.

Diet therapies and exercise regimens used in childhood obesity studies have varied greatly. Dietary interventions included nutrition counselling, various diets (e.g., the Traffic Light Diet), generic reduced-energy diets and specific reductions to induce weight loss at a desired weekly rate. Despite these differences, the main goal was to reduce energy intake while maintaining normal childhood growth. Exercise treatments included structured aerobic activity, assessed by percentage of maximum heart rate or maximum oxygen consumption, lifestyle exercises or calisthenics. The duration of physical activity ranged from 30 to 60 minutes and the frequency from twice weekly to daily, under observation or at the child's discretion. Thus, it was difficult to analyze specific effects of exercise or diet types.

## Results

We identified 31 studies of various types that addressed the treatment of pediatric obesity and how diet and exercise are

related to obesity in this population; 14 that met the inclusion criteria investigated diet and exercise in the pediatric population.<sup>4–17</sup> We found few RCTs. The age of children ranged from 3 to 17 years. All of the studies included both boys and girls.

The results of the studies were mixed. They did not consistently show that the combination of physical activity and diet treatment, with or without behaviour therapy, was a more effective treatment of childhood obesity than physical activity or diet treatment alone. They revealed that weight loss, blood pressure and BMI can be significantly reduced through each treatment method. Similarity in weight change suggests that decreased energy intake or altered metabolic rate through exercise can alter weight, blood pressure and BMI in obese children.<sup>6,9,17</sup> Epstein and colleagues<sup>9</sup> suggested that comparisons between diet plus exercise and diet alone are “complicated because the change in energy balance is more marked for diet than exercise, and the majority of the weight loss will be due to diet, rather than exercise.” However, some studies showed that the combination of physical activity and diet treatment (with or without behaviour therapy) was more effective than physical activity or diet treatment alone in reducing triglyceride and cholesterol levels, percentage of body fat, fat-free mass and skinfold measurements in obese children.<sup>6,8,10,17</sup> The long-term effects of these outcomes in obese children are unknown.

In recent years, studies examining the treatment of obesity have used a multidisciplinary approach, in which behavioural therapy addressing such concepts as self-esteem and coping mechanisms, physical activity and diet therapy were combined. Earlier studies examined the impact of diet, activity or behaviour interventions alone.<sup>10,18</sup> It is not within the scope of this chapter to review the effects of behavioural therapy on treatment success. However, it is impossible and unrealistic to separate behavioural treatment from physical activity or dietary intervention in the most recent studies.<sup>4,12,14</sup> The evidence is strong that multidisciplinary programs have been more successful than previous programs that applied one discipline alone in treating obesity and maintaining the results achieved during the course of the study. In these RCTs, the control group received a physician's or dietitian's recommendation to increase physical activity and reduce energy intake. The treatment groups participated in structured physical activity and continuing dietary guidance over 3–6 months. The treatment groups achieved significantly greater success than the control groups in enhancing endurance and reducing body weight or BMI. The dietary interventions involved reducing intake by 15%–20% below estimated total energy expendi-



ture<sup>4,12</sup> or an intake of 1200–1500 kcal/d based on the Traffic Light Diet.<sup>14</sup> Physical activity treatment included two 60-minute sessions a week. Children were encouraged to undertake an additional session of 30–45 minutes individually<sup>4,12</sup> or an additional 60 minutes, 5 times a week, monitored individually but regularly counselled via telephone calls with counsellors.<sup>14</sup> The intervention groups had significantly reduced BMI<sup>4,12,14</sup> and body weight,<sup>4,12</sup> whereas the control groups gained weight. At follow-up 3 months to 1 year after treatment, the treatment groups maintained their significantly lower BMIs in comparison with the control groups. These studies confirm the importance of a counsellor-guided, multidisciplinary program lasting at least 3 months in the treatment of obesity.

Parental participation is also important in the treatment of childhood obesity. Family-based treatment that includes parental participation is associated with sustained weight loss in obese children.<sup>6,8,11,19,20</sup> In the studies reviewed, family-based treatment included diet, exercise and behaviour modification. The main difference between control and treatment groups was in behaviour modification. Treatments that targeted both parent and child in terms of diet and exercise monitoring and behaviour modification resulted in greater weight loss over 5 and 10 years than those that targeted only the child or those with no specific target.<sup>8,20</sup>

## Other considerations and limitations

Research into the treatment of pediatric obesity using diet and exercise manipulation is evolving. Earlier research focused on changes in body weight and BMI, whereas more recent studies consider lipid profiles, fat-free mass changes and vascular function. However, data on the latter are limited, and recommendations regarding the type of exercise and diet combination to address these issues cannot be provided.

Pediatric obesity is a sensitive topic and must be addressed with care. There are many concerns regarding possible side effects and the child's functioning in response to treatment. In addition, it is not known what type of intervention promotes and maintains healthy attitudes toward exercise and eating. Solutions for pediatric obesity must be found. However, potential solutions cannot be recommended without consideration of well-being after treatment.

## Conclusions

There is an urgent need to treat childhood obesity. Despite this alarming problem, few data are available describing the optimum diet and exercise combinations for successful long-term treatment. Few well-designed, long-term studies have adequately defined the type of diet and activity that will promote growth and reduce fat mass while positively affecting markers

for chronic disease. Available research illustrates the need for professionally guided, family-oriented programs lasting several months and addressing diet, activity and behaviour.

## Recommendations

1. We recommend an energy-reduced diet and regular physical activity as the first treatment option for overweight and obese children to achieve clinically important weight loss and reduce obesity-related symptoms [*grade A, level 2<sup>4,12,14</sup>*].
2. We recommend ongoing follow-up by health professionals for a minimum of 3 months [*grade A, level 2<sup>4,6,12,14</sup>*].

From the Pediatric Centre for Weight and Health, Edmonton, Alta. (Barbarich); University of Alberta (Kubrak, McCargar); University of Waterloo (Hanning)

**Competing interests:** None declared.

## REFERENCES

1. *Eating well with Canada's food guide*. Ottawa: Health Canada; 2007. Available: [www.hc-sc.gc.ca/fn-an/food-guide-aliment/index\\_e.html](http://www.hc-sc.gc.ca/fn-an/food-guide-aliment/index_e.html) (accessed 2007 Feb 16).
2. *Canada's physical activity guides for children and youth*. Ottawa: Health Canada and the Canadian Society for Exercise Physiology; 2002. Available: [www.phac-aspc.gc.ca/pau-uap/paguide/child\\_youth/index.html](http://www.phac-aspc.gc.ca/pau-uap/paguide/child_youth/index.html) (accessed 2007 Feb 16).
3. Ball GD, Willows ND. Definitions of pediatric obesity. *CMAJ* 2005;172:309-10.
4. Eliakim A, Kaven G, Berger I, et al. The effect of a combined intervention on body mass index and fitness in obese children and adolescents – a clinical experience. *Eur J Pediatr* 2002;161:449-54.
5. Epstein LH, Koeske R, Zidanssek J, et al. Effects of weight loss on fitness in obese children. *Am J Dis Child* 1983;137:654-7.
6. Epstein LH, McCurley J, Wing RR, et al. Five-year follow-up of family-based behavioral treatments for childhood obesity. *J Consult Clin Psychol* 1990;58:661-4.
7. Epstein LH, Valoski A, Wing RR, et al. Ten-year outcomes of behavioral family-based treatment for childhood obesity. *Health Psychol* 1994;13:373-83.
8. Epstein LH, Wing RR, Koeske R, et al. Long-term effects of family-based treatment of childhood obesity. *J Consult Clin Psychol* 1987;55:91-5.
9. Epstein LH, Wing RR, Valoski A. Childhood obesity. *Pediatr Clin North Am* 1985;32:363-79.
10. Epstein LH, Wing RR, Koeske R, et al. A comparison of lifestyle exercise, aerobic exercise, and calisthenics on weight loss in obese children. *Behav Ther* 1985;16:345-56.
11. Flodmark CE, Ohlsson T, Ryden O, et al. Prevention of progression to severe obesity in a group of obese schoolchildren treated with family therapy. *Pediatrics* 1993;91:880-4.
12. Nemet D, Barkan S, Epstein Y, et al. Short- and long-term beneficial effects of a combined dietary-behavioral-physical activity intervention for the treatment of childhood obesity. *Pediatrics* 2005;115:e443-9.
13. Rolland-Cachera MF, Thibault H, Souberbielle JC, et al. Massive obesity in adolescents: dietary interventions and behaviours associated with weight regain at 2 y follow-up. *Int J Obes Relat Metab Disord* 2004;28:514-9.
14. Saelens BE, Sallis JF, Wilfley DE, et al. Behavioral weight control for overweight adolescents initiated in primary care. *Obes Res* 2002;10:22-32.
15. Schwingshandl J, Sudi K, Eibl B, et al. Effect of an individualised training programme during weight reduction on body composition: a randomised trial. *Arch Dis Child* 1999;81:426-8.
16. Valoski A, Epstein LH. Nutrient intake of obese children in a family-based behavioral weight control program. *Int J Obes* 1990;14:667-77.
17. Woo KS, Chook P, Yu CW, et al. Effects of diet and exercise on obesity-related vascular dysfunction in children. *Circulation* 2004;109:1981-6.
18. Epstein LH, Wing RR, Penner BC, et al. Effect of diet and controlled exercise on weight loss in obese children. *J Pediatr* 1985;107:358-61.
19. Epstein LH. Family-based behavioural intervention for obese children. *Int J Obes Relat Metab Disord* 1996;20:S14-21.
20. Epstein LH, Wing RR, Koeske R, et al. Effects of diet plus exercise on weight change in parents and children. *J Consult Clin Psychol* 1984;52:429-37.

## 20. Pharmacotherapy and bariatric surgery for the treatment of obesity in children and adolescents

Elizabeth A.C. Sellers

**O**besity in children and adolescents has increased markedly over the last 20 years,<sup>1</sup> along with the comorbid conditions associated with obesity.<sup>2</sup> These conditions include type 2 diabetes, non-alcoholic fatty liver disease, dyslipidemia, hypertension, obstructive sleep apnea, orthopedic problems and social and psychological concerns. Obesity established in childhood and adolescence is a known risk factor for obesity in the adult years.<sup>3</sup> Conventional treatments of obesity involving lifestyle modification have had mixed results.<sup>3</sup> There is a need to consider adjunctive therapy (pharmacotherapy or surgery or both) in this group, especially in those with or at high risk of comorbid conditions.

### Data sources

Studies selected for review in this section satisfied the following criteria: they were prospective randomized or nonrandomized clinical trials or case series; the study population comprised overweight (BMI z-score  $\geq 1.04$  to  $1.64$ ) or obese (BMI z-score  $\geq 1.64$ ) children or adolescents;  $\leq 18$  years of age; duration of follow-up was at least 6 months (12 months was preferred); the period of study was 1966 to the present.

A systematic review of MEDLINE, EMBASE and the Cochrane Controlled Clinical Trials Register was undertaken. This review was limited to English-language journals. Syndromes associated with obesity (e.g., Prader-Willi syndrome) were excluded. Studies involving pharmacologic agents and surgical techniques that have been shown to have significant side effects and are no longer in use were also excluded.

### Pharmacotherapy for obesity in children and adolescents

There are limited studies of pharmacologic intervention for the treatment of obesity in children and youth. Three studies involving the use of orlistat and 2 studies using sibutramine form the basis for the following discussion. They were reviewed on an individual basis given the limited number of studies available to guide recommendations.

#### Orlistat

Orlistat, a pancreatic and gastric lipase inhibitor, decreases the hydrolysis of dietary triglyceride and thus prevents the absorption of cholesterol and free fatty acids from the gastrointestinal tract. Systemic absorption of the drug itself is limited.

The dose of orlistat in each of the 3 studies reviewed was 120 mg by mouth 3 times a day.

McDuffie and colleagues<sup>4</sup> reported an uncontrolled prospective trial of orlistat in 20 adolescents (12–17 years of age). Participants also took part in a 12-week behavioural modification program. Over the 6-month study, the participants' mean BMI (and standard deviation [SD]) decreased from 42.7 (SD 1.3) kg/m<sup>2</sup> to 40.7 (SD 1.4) kg/m<sup>2</sup> and mean waist circumference decreased from 112.2 (SD 1.1) cm to 109.6 (SD 1.2) cm. The mean total cholesterol level also decreased, from 4.49 (SD 0.03) mmol/L to 3.92 (SD 0.12) mmol/L, as did the LDL cholesterol level, from 3.04 (SD 0.03) mmol/L to 2.56 (SD 0.03) mmol/L. No changes in HDL cholesterol or triglyceride levels or in the ratio of total to HDL cholesterol were observed. Insulin resistance, as estimated using the homeostasis model assessment of insulin resistance (HOMA-IR), and fasting insulin levels decreased, from 5.54 (SD 1.80) to 3.94 (SD 1.68) ( $p = 0.002$ ) and from 172.2 (SD 12.9) mmol/L to 127.7 (SD 12.2) mmol/L, respectively. Although this study demonstrated a modest weight loss and modest improvement in some cardiovascular risk factors, the contribution of the drug therapy compared with that of the psychoeducational program cannot be determined. No abnormalities in fat-soluble vitamin levels (on supplementation) were observed.

Ozkan and coworkers<sup>5</sup> performed a prospective, open-controlled study in adolescents with severe exogenous obesity. Outcomes reported were limited to weight loss. The 22 participants in the treatment group received 120 mg of orlistat 3 times a day in addition to conventional treatment consisting of nutritional and lifestyle modification. The control group consisted of 20 obese adolescents who received the conventional treatment alone. The duration of follow-up was 11 months (range 5–15 months). Of the 22 participants taking orlistat, 7 withdrew from the study in the first month because of intolerable gastrointestinal side effects. These participants were not included in the results. The mean BMI in the orlistat group decreased 4.09 (SD 2.9) kg/m<sup>2</sup> and increased 0.11 (SD 2.49) kg/m<sup>2</sup> in the control group.

In a third trial of orlistat,<sup>6</sup> which has recently been reported, 539 obese adolescents (aged 12–16 years) participated in a 54-week double-blind RCT. They were randomly assigned, in a ratio of about 2:1, to receive orlistat or placebo ( $n = 357$  and 182, respectively). All participants took part in a behavioural modification program, were counselled to consume a reduced-energy diet and undertake an exercise routine. At the end of the trial, the mean BMI in the orlistat group had decreased by 0.55 kg/m<sup>2</sup> and in the placebo group had in-

creased by 0.31 kg/m<sup>2</sup>. A significantly greater proportion of treated patients decreased their BMI by 5% and 10% compared with those taking placebo. Participants in the orlistat group had a greater decrease in waist-to-hip ratio than did those in the control group. There were no significant differences within or between the groups in terms of lipid profile or fasting glucose or insulin levels 2 hours after glucose loading either at baseline or at the end of the study. Diastolic blood pressure decreased significantly over the course of the study among those taking orlistat but not among those taking placebo. Gastrointestinal side effects were common in the orlistat group, although no major safety concerns were raised.

## Sibutramine

Sibutramine is a selective serotonin and noradrenaline re-uptake inhibitor that is thought to increase satiety. Berkowitz and colleagues<sup>7</sup> carried out a double-blind RCT involving 82 adolescents aged 13–17 years. Both the active (*n* = 43) and placebo (*n* = 39) groups participated in a behaviour modification protocol. Over the 6-month study period, the BMI z-score in the sibutramine-treated group decreased an average of 0.2 (SD 0.2), compared with an average decrease of 0.1 (SD 0.1) in the placebo group. Mean waist circumference decreased 8.2 (SD 6.9) cm in the sibutramine group, compared with 2.8 (SD 5.6) cm in the placebo group. No differences in

**Table 16:** Summary of case series assessing bariatric surgery among adolescents since 1966

Study	No. and age of participants	Initial mean BMI, kg/m <sup>2</sup>	Type of surgery (n)	Results	Complications (n)	Follow-up
Anderson et al <sup>13</sup>	15 girls, 15 boys; age < 20 yr	238% ideal body weight (BMI not available)	Gastric bypass (23) Gastroplasty (7)	187% ideal body weight	Revision of surgery because of failure to lose weight (4) Wound infection or dehiscence (3) Stomal obstruction (2) Atelectasis (3) Pneumonia (2) Subphrenic abscess (1) Death 3 days after surgery (1) Unexplained death 36 months after surgery (1) Incisional hernia repair (3)	5 yr
Rand et al <sup>14</sup>	27 girls, 7 boys; age 11-19 yr	47	RYGB (30) VGB (4)	Mean BMI 32 kg/m <sup>2</sup>	Staple-line failure (1) Inadequate weight loss (3) Hypoglycemia (3) Anemia (3) Gallbladder disease (3)	6 yr
Strauss et al <sup>15</sup>	7 girls, 3 boys; age 15-17 yr	52.4	RYGB	62% weight loss	Gallbladder disease (2) Pregnancy (3) Protein-calorie malnutrition (1)	1 yr
Stanford et al <sup>16</sup>	3 girls, 1 boy; age 17-19 yr	55.1	Laparoscopic RYGB	87% weight loss	Persistent sleep apnea (1)	4-22 mo
Capella and Capella <sup>17</sup>	19 participants; age 13-17 yr	49	VGB-RYGBP	Mean BMI 28 kg/m <sup>2</sup>	Anemia (3) Ulcer (1) Gastrogastric fistulas (2) Gallbladder disease (2)	1-10 yr
Abu-Abeid et al <sup>18</sup>	8 girls, 3 boys; age 11-17 yrs	46.6	LAGB	Mean BMI 33.1 kg/m <sup>2</sup>	Anemia (4)	23 mo
Sugerman et al <sup>19</sup>	19 girls, 14 boys; age 12-18 yr	52		77% weight loss	Pulmonary embolism (1) Wound infection (1) Ulcers (4) Death (2) Small-bowel obstruction (1) Incision hernia (6)	14 yr
Dolan and Fielding <sup>20</sup>	14 girls, 3 boys; age 12-19 yr	44.7	LAGB	Mean BMI 30.2 kg/m <sup>2</sup>	Leaking port (1) Slipped gastric band (1)	12-46 mo
Inge et al <sup>21</sup>	20 participants; age 13-18 yr	> 40	RYGBP with hand-sewn gastro-jejunosomy (10) Gastric bypass using open laparotomy (2) Laparoscopic bypass (8)	33%-53% excess weight loss	Partial obstruction of roux limb (1) Bowel leak (1) Deep-vein thrombosis (1) Dumping syndrome (1) Dehydration (1) Food impaction in the gastrojejunal anastomosis (1)	3-24 mo

Note: BMI = body mass index, LAGB = laparoscopic adjustable gastric banding, RYGBP = roux-en-Y gastric bypass, VGB = vertical banded gastroplasty.  
Source: Adapted from Apovian et al.<sup>22</sup>

serum lipids, glucose or insulin levels or in insulin resistance were observed during the controlled portion of the study. An increase in systolic blood pressure or heart rate necessitated the reduction or discontinuation of sibutramine in 44% of the study participants. Two episodes of arrhythmia in the sibutramine group also raised concern.

A second RCT of sibutramine involving obese adolescents has recently been published.<sup>8</sup> This 6-month trial enrolled 60 adolescents (aged 14–17 years) who had completed their linear growth; 30 received the drug, 30 the placebo. All received dietary counselling and physical activity guidelines at the start of the study. At the end of the trial, the reduction in BMI was significantly greater in the sibutramine group than in the placebo group: 3.6 (SD 2.5) kg/m<sup>2</sup> v. 0.9 (SD 0.9) kg/m<sup>2</sup> respectively ( $p < 0.001$ ). The reduction in waist circumference was also greater in the sibutramine group: 7.2 (SD 5.5) cm v. 1.8 (SD 2.6) cm ( $p < 0.001$ ). Triglyceride and very-low-density lipoprotein levels decreased significantly from the baseline level in the sibutramine group ( $p < 0.05$ ). In this trial, no significant changes were seen in blood pressure or heart rate. No serious adverse events were noted.

## Bariatric surgery in children and adolescents

Data on bariatric surgery in children and adolescents are extremely limited. Published reports are limited to case series and individual clinical experience (Table 16). No RCTs were available for review. Although several reports comment on improvement in obesity-related comorbidities, the numbers are limited. Thus, it is premature to provide meaningful conclusions on the effect of bariatric surgery on comorbidities in adolescents. Series involving jejunio-ileal bypass<sup>9–12</sup> were excluded, because this procedure has now been abandoned in adults owing to high rates of morbidity and mortality.

## Conclusions

Orlistat may aid in weight reduction in adolescents for up to 12 months. No data from a controlled study are available regarding reduction of cardiovascular risk factors. The long-term effects on obesity and obesity-related morbidity have yet to be determined. Gastrointestinal side effects may limit use in adolescents. The dose of orlistat in each of the 3 studies reviewed was 120 mg by mouth 3 times a day. There is insufficient evidence to recommend an optimum dose. Sibutramine may aid in weight loss for up to 6 months in conjunction with a behavioural therapy program. However, long-term effects on obesity and obesity-related morbidity have yet to be determined. Caution is recommended because increases in blood pressure and arrhythmias have been associated with its use.

Given the complete lack of data in the prepubertal child, the use of bariatric surgery in this group should be restricted to controlled trials.

In adolescents, experience with bariatric surgery remains limited and is associated with significant morbidity and mortality in this age group. Long-term follow up studies are lack-

ing in this population. Use of bariatric surgery in adolescents should be limited to exceptional cases and performed by experienced teams.

## Recommendations

1. We suggest that orlistat be considered to aid in weight reduction and weight maintenance when added to a regimen of lifestyle intervention among adolescents [*grade B, level 1*].
2. Because of lack of data for prepubertal children, the use of pharmacologic agents in this group should be considered only within the context of a supervised clinical trial [*grade C, level 4*].
3. We suggest that bariatric surgery in adolescents be limited to exceptional cases and performed only by experienced teams [*grade C, level 4*].

From the Department of Pediatrics and Child Health, University of Manitoba, Winnipeg, Man.

**Competing interests:** None declared.

## REFERENCES

1. Tremblay MS, Willms JD. Secular trends in the body mass index of Canadian children (published erratum *CMAJ* 2001;164:970). *CMAJ* 2000;163:1429–33.
2. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics* 1998;101:518–25.
3. Dietz WH. Childhood weight affects adult morbidity and mortality. *J Nutr* 1998;128 (2 Suppl):411S–4S.
4. McDuffie JR, Calis KA, Uwaifo GI, et al. Efficacy of orlistat as an adjunct to behavioural treatment in overweight African American and Caucasian adolescents with obesity-related co-morbid conditions. *J Pediatr Endocrinol Metab* 2004;17:307–19.
5. Ozkan B, Bereket A, Turan S, et al. Addition of orlistat to conventional treatment in adolescents with severe obesity. *Eur J Pediatr* 2004;163(12):738–41.
6. Chanoine JP, Hampl S, Jensen C, et al. Effect of orlistat on weight and body composition in obese adolescents: a randomized controlled trial. *JAMA* 2005;293(23):2873–83.
7. Berkowitz RI, Wadden TA, Tershakovec AM, et al. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. *JAMA* 2003;289:1805–12.
8. Godoy-Matos A, Carraro L, Vieira A, et al. Treatment of obese adolescents with sibutramine: a randomized, double-blind, controlled study. *J Clin Endocrinol Metab* 2005;90:1460–5.
9. Silber T, Randolph J, Robbins S. Long-term morbidity and mortality in morbidly obese adolescents after jejunioileal bypass. *J Pediatr* 1986;108:318–22.
10. Randolph JG, Weintraub WH, Rigg A. Jejunioileal bypass for morbid obesity in adolescents. *J Pediatr Surg* 1974;9:341–5.
11. Rigg CA. Proceedings: Jejunioileal bypass by morbidly obese adolescent. *Acta Paediatr Scand Suppl* 1975;256:62–4.
12. White JJ, Cheek D, Haller JA Jr. Small bowel bypass is applicable for adolescents with morbid obesity. *Am Surg* 1974;40:704–8.
13. Anderson AE, Soper RT, Scott DH. Gastric bypass for morbid obesity in children and adolescents. *J Pediatr Surg* 1980;15:876–81.
14. Rand CS, Macgregor AM. Adolescents having obesity surgery: a 6-year follow-up. *South Med J* 1994;87:1208–13.
15. Strauss RS, Bradley LJ, Brolin RE. Gastric bypass surgery in adolescents with morbid obesity. *J Pediatr* 2001;138:499–504.
16. Stanford A, Glascock JM, Eid GM, et al. Laparoscopic Roux-en-Y gastric bypass in morbidly obese adolescents. *J Pediatr Surg* 2003;38:430–3.
17. Capella JF, Capella RF. Bariatric surgery in adolescence. is this the best age to operate? *Obes Surg* 2003;13:826–32.
18. Abu-Abaid S, Gavert N, Klausner JM, et al. Bariatric surgery in adolescence. *J Pediatr Surg* 2003;38:1379–82.
19. Sugerman HJ, Sugerman EL, DeMaria EJ, et al. Bariatric surgery for severely obese adolescents. *J Gastrointest Surg* 2003;7:102–7.
20. Dolan K, Fielding G. A comparison of laparoscopic adjustable gastric banding in adolescents and adults. *Surg Endosc* 2004;18:45–7.
21. Inge TH, Garcia V, Daniels S, et al. A multidisciplinary approach to the adolescent bariatric surgical patient. *J Pediatr Surg* 2004;39:442–7.
22. Apovian CM, Baker C, Ludwig DS, et al. Best practice guidelines in pediatric/adolescent weight loss surgery. *Obes Res* 2005;13:274–82.

# 21. Prevention of obesity in adults

Bruce Reeder, Peter T. Katzmarzyk

Excess body weight is associated with significant health risks.<sup>1,2</sup> In recent years, BMI has been adopted in Canada<sup>3</sup> and elsewhere<sup>2</sup> as a useful indicator of excess body weight for clinical and public health purposes. On the basis of this measure, adults are considered overweight if their BMI is 25–29.9 kg/m<sup>2</sup> and obese if it is over 30 kg/m<sup>2</sup>. Several studies have documented an increase in the prevalence of overweight and obesity in Canada in recent decades.<sup>4,5</sup> The most recent data from the 2004 Canadian Community Health Survey indicate that about 36% of Canadian adults are overweight and about 23% are obese.<sup>6</sup> Given the health risks associated with excess body weight, these high prevalences translate into a substantial public health burden. For example, it has recently been estimated that approximately 1 in 10 premature deaths in Canada is directly attributable to overweight and obesity.<sup>7</sup> Further, obesity accounts for approximately \$1.6 billion in direct health care spending annually, in addition to \$2.7 billion in indirect costs, which include the loss of economic output due to illness, injury-related work disability and premature death.<sup>8,9</sup> Given that long-term weight loss is difficult to achieve once an individual becomes obese,<sup>10</sup> the prevention of weight gain and obesity is an important public health priority.<sup>11,12</sup>

One narrative<sup>13</sup> and 2 systematic<sup>14,15</sup> reviews published before 2002 examined interventions to prevent weight gain and obesity. The narrative review considered there to be only modest evidence of the effectiveness of community-wide programs for cardiovascular disease prevention in preventing obesity, and no evidence to support individual-level education on nutrition and exercise in the one study identified (Pound of Prevention study). Fogelholm and Lahti-Koski<sup>15</sup> identified 5 community intervention studies published since 1990 that evaluated the use of physical activity in the prevention of obesity. They concluded that most of the studies showed no effect of the interventions on BMI. Hardeman and colleagues<sup>14</sup> reviewed both observational studies and trials involving children and adults. They concluded that the variability of the study designs, samples and outcome variables made it difficult to identify effective interventions.

The role of physical activity in the prevention of weight gain has been recently examined. In a systematic review of the literature published before 2000, Fogelholm and Kukkonen-Harjula<sup>16</sup> concluded that weight maintenance following weight loss was superior in groups whose program included exercise. Yet, there is growing evidence that the typical exercise prescription for health benefits (30 minutes of moderate-intensity activity on most days of the week) is insufficient for

weight-gain prevention. In a recent consensus statement,<sup>17</sup> the International Association for the Study of Obesity indicated that “there is compelling evidence that prevention of weight gain in formerly obese individuals requires 60–90 minutes of moderate intensity activity or lesser amounts of vigorous intensity activity.”

In this chapter, we review the literature on the prevention of weight gain and obesity in an evidence-based fashion in order to provide an up-to-date assessment of strategies to prevent weight gain and reduce the incidence of obesity.

## Data sources

A full report of the methods and results of this systematic review is available from the Canadian Task Force on Preventive Health Care ([www.ctfphc.org](http://www.ctfphc.org)). In brief, we searched MEDLINE, HealthSTAR and the Cochrane Controlled Trials Register for peer-reviewed literature published from 1966 to March 2004. Nonrandomized trials and RCTs of interventions to prevent weight gain and obesity in adults 18 years of age and older with a mean BMI of less than 30 kg/m<sup>2</sup> were reviewed. We included studies if they provided at least 6 months' follow-up and either BMI or weight as an outcome variable. Trials were systematically reviewed by both of us. Only studies that were considered of good or fair quality were included.<sup>18</sup> Interventions were grouped into the following categories: community, dietary, exercise, and combined diet and exercise interventions.

## Results

Five community intervention studies were identified; they were carried out in the United States between 1972 and 1993. All 5 were considered to be of only fair quality in view of their quasi-experimental design<sup>19</sup> and relatively high nonresponse rates and loss to follow-up. None was designed specifically to prevent weight gain or obesity; rather, all were intended to reduce the prevalence of cardiovascular disease risk factors in the population. The intervention in the Stanford 3 Community Study,<sup>20</sup> conducted early in the period using only a media campaign, appears to have been successful in preventing weight gain, at least in women. The interventions in the Stanford 5 City Project<sup>21</sup> and the Pawtucket Heart Health Program,<sup>22</sup> conducted in the 1980s and early 1990s, were able only to slow the rate of weight gain in the intervention communities compared with the control communities, and then only according to the cross-sectional, not cohort, analyses.

The study with the greatest emphasis on weight, the Minnesota Heart Health Program,<sup>23</sup> failed to show a significant difference between the intervention and control communities with respect to weight gain during this same period.

The most positive results from a community intervention were seen in a 5-year program in 2 South Carolina cities with a high black population.<sup>24</sup> Community-wide campaigns promoting a healthy lifestyle resulted in a difference in the prevalence of overweight in the intervention city (increase of 0.3%) compared with the control city (increase of 3.2%;  $p < 0.0002$ ). During the period when these studies were conducted, the United States experienced a strong secular trend in increasing prevalence of obesity.<sup>25</sup> It appears that this trend generally overwhelmed the impact of the community interventions.

Three identified studies employed dietary intervention alone. One good-quality study demonstrated that small-group counselling promoting a low-fat diet produced a sustained weight loss over 2 years in highly motivated middle-aged women.<sup>26</sup> In an evaluation of the effect of a program of individual clinical counselling on diet and exercise, Williams and colleagues<sup>27</sup> randomly allocated sedentary overweight but otherwise healthy men (aged 30–59 years) to 1 of 3 groups: energy-reduced diet, endurance exercise or no treatment. After 1 year of follow-up, the men in the diet and exercise groups had significantly reduced their body weight (BMI decreased by 2.45 kg/m<sup>2</sup> and 1.41 kg/m<sup>2</sup>, respectively) compared with those in the control group, whose BMI increased by 0.18 kg/m<sup>2</sup>. The weight loss in the diet group was significantly greater than that in the exercise group. A third study, of fair quality, illustrated that a less-intense self-help program promoting a healthy diet (offered through a health maintenance organization) failed to have a significant impact on weight after 12 months' follow-up.<sup>28</sup>

Four studies examined the impact of exercise programs alone in the prevention of weight gain. One good-quality study demonstrated that strength-training classes (offered twice weekly over 39 weeks to a group of healthy middle-aged women) produced significant favourable changes in body composition, but no significant change in weight or waist circumference.<sup>29</sup> Three studies of fair quality reported the outcomes associated with endurance exercise programs for middle-aged men and women.<sup>27,30,31</sup> The positive results seen by Williams and colleagues<sup>27</sup> are reported above. One study<sup>31</sup> showed a significant graded relation between the intensity and duration of exercise and the amount of weight loss over 8 months compared with a control group, while another<sup>30</sup> did not. Participants in the former study were heavier than those in the latter study (mean BMI 29.7 v. 27.0 kg/m<sup>2</sup>), a feature that may have contributed to their greater weight loss with the exercise program.

Five studies of good<sup>32,33</sup> or fair<sup>34–36</sup> quality compared a combination of diet and endurance exercise with a control group in the prevention of weight gain. Only in the Da Qing study<sup>36</sup> was a direct comparison made between the individual strategies and the combination of the 2. In this instance, the 3 intervention groups did not significantly differ from each other or the control group in terms of weight change. The other 5 studies all offered programs with a combination of

diet and physical activity. The programs that were effective in weight reduction or prevention of weight gain were those that included small-group counselling.<sup>32,33</sup> Although attractive because of its low cost, intervention by telephone or mail appears ineffective.<sup>34</sup> The use of a financial incentive has not been shown to be of benefit.<sup>34</sup> Furthermore, the limited evidence available suggests that an intervention that focuses on weight may be more effective than one that targets a broad range of preventive health measures.<sup>35</sup>

## Conclusions

Several observations can be made after examining the range of individual-level programs for the prevention of obesity. The impact of intervention programs is greater in the earlier, more intensive phase than in the maintenance phase. Benefits are maintained in programs that use individual<sup>27</sup> and small-group<sup>26,33</sup> counselling, but not in those using telephone support.<sup>37</sup> Counselling is most likely to be effective with intensive intervention in highly motivated participants.

Evidence from the Da Qing study<sup>36</sup> and exercise intervention studies<sup>30,31</sup> suggests that a greater benefit in terms of weight reduction and weight-gain prevention is observed among those who are overweight than among those who are of normal weight. A number of life stages have been identified as high-risk periods for the development of obesity. Two studies in premenopausal women indicate that strength-training exercise<sup>29</sup> or a combination of diet and endurance exercise<sup>33</sup> can either prevent the weight gain commonly associated with menopause<sup>32</sup> or produce favourable changes in body composition.<sup>29</sup> Similarly, studies in middle-aged adults (aged 30–65 years) demonstrate that endurance exercise<sup>27,31</sup> or an energy-reduced diet have a positive influence.

## Recommendations

1. Programs that combine a low-fat or energy-reduced diet and endurance exercise have not been shown to be more effective than programs using either component alone for obesity prevention; both approaches should be considered [*grade B, level 3*].
2. We suggest that individual and small-group counselling for dietary interventions be considered for the prevention of obesity in adults [*grade B, level 2*]. Counselling by telephone, counselling by mail and financial incentives do not appear to be effective, and we do not encourage their use [*grade C, level 3*].
3. There is insufficient evidence to recommend in favour of or against broad community interventions aimed at cardiovascular disease risk reduction for the prevention of obesity [*grade C, level 3*].

From the Department of Community Health and Epidemiology, University of Saskatchewan, Saskatoon, Sask. (Reeder); and the School of Kinesiology and Health Studies, Department of Community Health and Epidemiology, Queen's University, Kingston, Ont. (Katzmarzyk)

**Competing interests:** None declared.

**Acknowledgement:** This review was originally conducted under the auspices of the Canadian Task Force on Preventive Health Care ([www.ctfphc.org](http://www.ctfphc.org)). We gratefully acknowledge the excellent critical evaluation and advice provided by members of the task force (2005): Drs. John Feightner (Chair), Harriet MacMillan (Vice-Chair), Wayne Elford, Denice Feig, Joanne Langley, Valerie Palda, Christopher Patterson, Bruce Reeder and Ruth Walton (technical support).

## REFERENCES

- US National Institutes of Health. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report [published erratum appears in *Obes Res* 1998;6:464]. *Obes Res* 1998;6(Suppl 2):51S-209S.
- World Health Organization. *Obesity: preventing and managing the global epidemic — report of a WHO consultation on obesity, 3–5 June, 1997*. Geneva: World Health Organization; 1998.
- Canadian guidelines for body weight classification in adults*. Ottawa: Health Canada; 2003.
- Torrance GM, Hooper MD, Reeder BA. Trends in overweight and obesity among adults in Canada (1970–1992): evidence from national surveys using measured height and weight. *Int J Obes Relat Metab Disord* 2002;26:797–804.
- Tremblay MS, Katzmarzyk PT, Willms JD. Temporal trends in overweight and obesity in Canada, 1981–1996. *Int J Obes Relat Metab Disord* 2002;26:538–43.
- Tjepkema M. Adult obesity in Canada: measured height and weight. In: *Nutrition: Findings from the Canadian Community Health Survey*. Issue 1. Ottawa: Statistics Canada; 2005. Cat no 82-620-MWE2005001.
- Katzmarzyk PT, Ardern CI. Overweight and obesity mortality trends in Canada, 1985–2000. *Can J Public Health* 2004;95:16–20.
- Katzmarzyk PT, Janssen I. The economic costs associated with physical inactivity and obesity in Canada: an update. *Can J Appl Physiol* 2004;29:90–115.
- Katzmarzyk PT, Janssen I, Ardern CI. Physical inactivity, adiposity and premature mortality. *Obes Rev* 2003;4:257–90.
- Kassirer JP, Angell M. Losing weight—an ill-fated new year's resolution. *N Engl J Med* 1998;338:52–4.
- Raine K. *Overweight and obesity in Canada. A population perspective*. Ottawa: Canadian Institute for Health Information; 2004.
- Manson JE, Skerrett PJ, Greenland P, et al. The escalating pandemics of obesity and sedentary lifestyle. *Arch Intern Med* 2004;164:249–58.
- Muller MJ, Mast M, Asbeck I, et al. Prevention of obesity — Is it possible? *Obes Rev* 2001;2:15–28.
- Hardeman W, Griffin S, Johnston M, et al. Interventions to prevent weight gain: a systematic review of psychological models and behavior change methods. *Int J Obes Relat Metab Disord* 2000;24:131–43.
- Fogelholm M, Lahti-Koski M. Community health-promotion interventions with physical activity: Does this approach prevent obesity? *Scandinavian J Nutr* 2002;46:173–7.
- Fogelholm M, Kukkonen-Harjula K. Does physical activity prevent weight gain: a systematic review. *Obes Rev* 2000;1:95–111.
- Saris WHM, Blair SN, van Baak MA, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock conference and consensus statement. *Obes Rev* 2003;4:101–14.
- Harris RP, Helfand M, Woolf SH, et al. Current methods of the US Preventive Services Task Force: a review of the process. *Am J Prev Med* 2001;20(3 Suppl):21–35.
- Issel LM. *Health program planning and evaluation*. Mississauga (ON): Jones and Bartlett Publishers; 2004.
- Fortmann SP, Williams PT, Hulley SB, et al. Effect of health education on dietary behavior: the Stanford Three Community Study. *Am J Clin Nutr* 1981;34:2030–8.
- Taylor CB, Fortmann SP, Flora J, et al. Effect of long-term community health education on body mass index. The Stanford Five-City Project. *Am J Epidemiol* 1991;134:235–49.
- Carleton RA, Lasater TM, Assaf AR, et al. The Pawtucket Heart Health Program: community changes in cardiovascular risk factors and projected disease risk. *Am J Public Health* 1995;85:777–85.
- Jeffery RW, Gray CW, French SA, et al. Evaluation of weight reduction in a community intervention for cardiovascular disease risk: changes in body mass index in the Minnesota Heart Health Program. *Int J Obes Relat Metab Disord* 1995;19:30–9.
- Goodman RM, Wheeler FC, Lee PR. Evaluation of the Heart To Heart Project: lessons from a community-based chronic disease prevention project. *Am J Health Promot* 1995;9:443–55.
- Flegal KM, Carroll MD, Kuczmarski RJ, et al. Overweight and obesity in the United States: prevalence and trends, 1960–1994. *Int J Obes Relat Metab Disord* 1998;22:39–47.
- Henderson MM, Kushi LH, Thompson DJ, et al. Feasibility of a randomized trial of a low-fat diet for the prevention of breast cancer: dietary compliance in the Women's Health Trial Vanguard Study. *Prev Med* 1990;19:115–33.
- Williams PT, Krauss RM, Vranizan KM, et al. Changes in lipoprotein subfractions during diet-induced and exercise-induced weight loss in moderately overweight men. *Circulation* 1990;81:1293–304.
- Kristal AR, Curry SJ, Shattuck AL, et al. A randomized trial of a tailored, self-help dietary intervention: the Puget Sound Eating Patterns study. *Prev Med* 2000;31:380–9.
- Schmitz KH, Jensen MD, Kugler KC, et al. Strength training for obesity prevention in midlife women. *Int J Obes Relat Metab Disord* 2003;27:326–33.
- King AC, Haskell WL, Taylor CB, et al. Group- vs home-based exercise training in healthy older men and women. A community-based clinical trial. *JAMA* 1991;266:1535–42.
- Slentz CA, Duscha BD, Johnson JL, et al. Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRRIDE — a randomized controlled study. *Arch Intern Med* 2004;164:31–9.
- Kuller LH, Simkin-Silverman LR, Wing RR, et al. Women's Healthy Lifestyle Project: A randomized clinical trial: results at 54 months. *Circulation* 2001;103:32–7.
- Simkin-Silverman LR, Wing RR, Boraz MA, et al. Lifestyle intervention can prevent weight gain during menopause: results from a 5-year randomized clinical trial. *Ann Behav Med* 2003;26:212–20.
- Jeffery RW, French SA. Preventing weight gain in adults: the Pound of Prevention study. *Am J Public Health* 1999;89:747–51.
- Mayer JA, Jermanovich A, Wright BL, et al. Changes in health behaviors of older adults: the San Diego Medicare Preventive Health Project. *Prev Med* 1994;23:127–33.
- Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* 1997;20:537–44.
- Jeffery RW, Sherwood NE, Brelje K, et al. Mail and phone interventions for weight loss in a managed-care setting: Weigh-To-Be one-year outcomes. *Int J Obes Relat Metab Disord* 2003;27:1584–92.

## 22. Individual approaches to the prevention of pediatric obesity using physical activity

Claire M.A. LeBlanc, Alison Irving, Mark S. Tremblay

The prevalence of childhood obesity in Canada has tripled over the past 20 years.<sup>1</sup> Research shows that obese children who remain obese throughout adolescence are predisposed to overweight and obesity in adulthood.<sup>2</sup> Efforts to treat adult obesity often fail, and even the best children's programs are only modestly successful. Primary prevention strategies are key to reversing current obesity trends.

The main focus of initiatives to date has been to attempt to balance the energy equation. Indeed, much of the published prevention literature reports interventions to improve the intake of healthy foods, increase physical activity and reduce sedentary behaviour or to achieve some combination of these. Considerable cross-sectional and longitudinal data suggest that children and adolescents who are physically active at high levels have less adiposity than those who are less active.<sup>3-14</sup> Similar evidence supports a strong association between excessive television watching and childhood obesity.<sup>6,8,10,15</sup>

In this chapter, we focus on prospective intervention research with strong methodologic rigour (RCTs) that demonstrated a reduction in obesity rates. The literature does not always include obesity outcome measures (e.g., BMI, percent body fat); hence, we will examine interventions that also improved healthy food intake, increased physical activity or reduced sedentary behaviour.

### Data sources

A systematic search of MEDLINE, EMBASE, Cochrane Controlled Clinical Trials Register, HealthSTAR, CINAHL, Eric, PsychInfo, BIOSIS and SportsDiscus was conducted. The search was restricted to studies of increased physical activity (with or without modifying diet) or reduced physical inactivity. We also identified and analyzed articles from 3 recent major reviews: the Cochrane Collaboration,<sup>16</sup> an evidence-based physical activity promotional summary by Timperio and others<sup>17</sup> and a detailed report by Thomas and colleagues<sup>18</sup> evaluating RCTs or cohort studies with strong methodological rigour. Because some of these reviews did not examine articles written after July 2003, we subsequently performed a literature search for more recent original papers. Few studies were long term ( $\geq 1$  year); therefore, we included original papers with an intervention duration of at least 3 months.

### Increasing physical activity

Thomas and colleagues<sup>18</sup> critically analyzed 9 RCTs and 12 cohort studies that focused on increasing physical activity.

They found that only 8 were free of serious methodological threats to internal or external validity, and of these, only 5 were of sufficient duration ( $\geq 3$  months). Of these 5 studies, 4 commented on obesity-related outcomes; 2 of these showed benefit from the intervention (Table 17).<sup>19,20</sup>

Specifically, Stephens and Wentz<sup>19</sup> randomly assigned black children in grade 4 to either a control group (45 minutes of physical education a week) or an intervention group (an additional 25 minutes of classroom-based aerobic physical activity 3 times a week) over 15 weeks. They found smaller skinfold measurements and weight gains as well as better fitness and flexibility scores in the intervention group. Tuckman and Hinkle<sup>20</sup> compared a program of running 30 minutes 3 times a week with regular physical education classes over 12 weeks for mostly nonwhite students in grades 4-6. They noted that, after the intervention, boys had less body fat and better endurance fitness, and these characteristics persisted 8 months after completion of the intervention.

With the 2-year SPARK program, Sallis and colleagues<sup>21</sup> found that specialist-led physical education classes increased vigorous physical activity in school and increased endurance in girls more effectively than in classes with trained classroom teachers or non-trained teachers. However, there was no change in BMI or skinfold measurements. Hansen and colleagues<sup>22</sup> randomly assigned 9-11 year olds (with and without hypertension) to 3 extra physical education classes a week for 8 months. Although fitness levels increased significantly, no change in weight, height or triceps skinfold measurements were noted compared with the control group. In their RCT, Ernst and Pangrazi<sup>23</sup> studied a 12-week, teacher-led program entitled Promoting Lifetime Activity for Youth (PLAY) for grade 4-6 students. Teachers increased lifestyle-related physical activity by 15 minutes a day in addition to physical education classes and encouraged regular physical activity outside of school. Physical activity increased significantly in the treatment group, and girls became more attracted to physical activity.

Timperio and others<sup>17</sup> identified 13 RCTs published between 1999 and 2003 of sufficient duration to meet our criteria. Of these, 11 had multifaceted interventions<sup>32-42</sup> and will be discussed later in this chapter. The remaining 2 papers focused on increasing physical activity behaviour (Table 17).<sup>24,25</sup> Ransdell and coworkers<sup>24</sup> discovered that self-selected sedentary (primarily white) mother-and-daughter pairs randomly assigned to a 12-week home- or community-based exercise program all had improved fitness levels. However, no reduction in percent body fat as measured by bioelectrical im-



pedance was seen. In a cluster RCT of 21 elementary schools, Rowland and colleagues<sup>25</sup> noted that intervention schools with a travel coordinator hired to increase active commuting to school significantly increased the development of such travel plans; however, no changes in commuting patterns or parental anxieties about traffic dangers were seen.

In a Cochrane review, Summerbell and others<sup>16</sup> critically reviewed long-term ( $\geq 1$  year) and short-term ( $\geq 3$  months) intervention studies of the effects of physical activity in preventing childhood obesity (Table 17). In the only long-term outcome study,<sup>26</sup> female kindergarten students taking part in an aerobic exercise program over 30 weeks were less likely to have an increase in BMI than those in the control group; however, the opposite was true for boys. Skinfold measurements were lower in the intervention group at 30 weeks, but this was of questionable clinical relevance. Flores' Dance for Health program<sup>27</sup> targeted mostly black and Hispanic students in grades 5–8 in a multifaceted intervention. Existing low-aerobic physical education classes were replaced by health classes twice a week and 50-minute dance classes 3 times a week for 3 months. Significant reductions in BMI and increases in fitness testing scores were noted only in girls compared with those in the control group. The PLAY model was used by Pangrazi and colleagues<sup>28</sup> to evaluate physical activity (pedometer step-counts) and measured BMI of grade 4 students over a 12-week period. Students were either in intervention schools (PLAY with or without physical education classes) or control schools (with or without physical education classes only). Increases in physical activity were seen in

all intervention students (especially girls), but there was no effect on BMI.

Additional articles published since July 2003 are discussed here (Table 17).<sup>29–31</sup> Only 1 reported measures of obesity as an outcome and found no benefit from the intervention, whereas the other 2 demonstrated increases in physical activity levels. A 4-month controlled trial (Project FAB) evaluated a daily 60-minute physical activity class for sedentary adolescent girls.<sup>29</sup> The class offered aerobic dance, sports and martial arts as well as lessons on the health benefits of physical activity and ways to become more active. This intervention prevented the decline in cardiovascular fitness (maximum oxygen consumption) seen in the control group and increased lifestyle physical activity inside and outside physical education class. However, no differences in exercise enjoyment, BMI or percent body fat were seen.<sup>29</sup>

McKenzie and colleagues<sup>30</sup> randomly selected half of 24 schools to receive a 2-year Middle School Physical Activity and Nutrition program (M-SPAN). Classroom teachers were trained to design and implement more active physical education curricula. Students at the intervention schools increased physical activity levels in class, but there was limited overall increase in daily physical activity, which suggested that physical education classes alone did not provide the students with adequate daily physical activity.

Saakslahti and colleagues<sup>31</sup> looked at the physical activity levels of children aged 4–7 years randomly assigned to the intervention (designed to encourage physical activity through an annual intensive session on the health benefits of physical

**Table 17:** Outcomes of programs to enhance physical activity

Study	Study design	Duration of intervention (follow-up)	Grade or age of participants	Results	
				Change in obesity	Other changes
Stephens et al <sup>19</sup>	RCT cluster (school)	15 wk	Grade 4	↓ skinfold measurements	↓ weight gain ↑ fitness/flexibility
Tuckman et al <sup>20</sup>	RCT	12 wk (8 mo)	Grades 4-6	↓ body fat (boys)	↑ fitness (boys)
Sallis et al <sup>21</sup>	RCT cluster (school)	2 yr	Grades 4 and 5	None	↑ physical activity ↑ fitness (girls)
Hanson et al <sup>22</sup>	RCT	8 mo	9-11 yr	None	↑ fitness
Ernst et al <sup>23</sup>	RCT cluster (classroom)	12 wk	Grades 4-6	Not measured	↑ physical activity
Ransdell et al <sup>24</sup>	Randomized (no control)	12 wk	Girls, 14-17 yr	None	↑ fitness
Rowland et al <sup>25</sup>	Cluster RCT (school)	1 yr	Primary school	None	No ↑ active transportation
Mo-suwan et al <sup>26</sup>	RCT	30 wk (1 yr)	Kindergarten	None	↑ physical activity
Flores <sup>27</sup>	RCT cluster (classroom)	12 wk	Grades 5-8	↓ BMI (girls)	↑ fitness (girls)
Pangrazi et al <sup>28</sup>	RCT cluster (school)	12 wk	Grade 4	None	↑ physical activity
Jamner et al <sup>29</sup>	RCT cluster (school)	4 mo	Girls, grade 10 or 11	None	↑ physical activity
McKenzie et al <sup>30</sup>	RCT cluster (school)	2 yr	Middle school	Not measured	↑ physical activity in class
Saakslahti et al <sup>31</sup>	RCT	3 yr	4-7 yr	Not measured	↑ physical activity outside

activity, increases in family-based physical activity and physical activity lessons promoting fundamental motor skills) or no intervention. Children in the intervention group played outside more often and engaged in more high-activity play than those in the control group, according to parent-recorded observations.

## Reducing sedentary behaviour

The review by Thomas and colleagues<sup>18</sup> identified 6 projects that measured the effects of reducing time spent watching television and videos and playing video games (Table 18). The duration of 5 of these was at least 3 months. In 1 RCT,<sup>32</sup> the efficacy of a 2-year Planet Health project on behaviour change in grade 6–7 students was studied. Intervention schools taught children to reduce television viewing to less than 2 hours a day. They also promoted healthy eating and physical activity. A reduction in obesity prevalence (BMI, skinfold measurements) was seen in girls but not boys. Television viewing was reduced in both sexes in the intervention schools, and this behaviour change had the greatest independent effect on reducing obesity.

In Robinson's<sup>33</sup> RCT, grade 3–4 students were evaluated following a 7-month screen-time-reduction intervention. The children were encouraged to watch less television through classroom curricula, home newsletters and an electronic television-time manager system. Significant decreases in television viewing time (not videotapes and video games) and obesity measures (BMI, skinfold measurements, waist-to-hip ratio) were seen, but there were no changes in fatty meal intake, physical activity levels or fitness test results.

In a 12-week pilot RCT (GEMS Stanford), Robinson and others<sup>34</sup> studied the effects of dance classes and a home-based program to reduce television viewing (curriculum and electronic television-time manager system) and obesity in black girls 8–10 years old. No significant differences in BMI,

waist circumference, fat intake or physical activity levels (measured by accelerometry) were noted, but girls in the intervention group watched less television while eating dinner.

The Cochrane Review<sup>16</sup> identified 2 other multidimensional programs that looked specifically at sedentary activity (Table 18).<sup>35,43</sup> The APPLES program was evaluated in a randomized trial that studied differences between elementary students undergoing the intervention (1-year school curriculum promoting healthy eating and physical activity and reducing sedentary activity) or the control treatment (usual health curriculum). The researchers noted greater gains in healthy living knowledge but no significant changes in BMI, physical activity or sedentary activity and only a modest increase in daily vegetable servings in the intervention group.<sup>35</sup> Dennison and colleagues<sup>43</sup> randomly assigned 176 daycare children to intervention and control groups. Children in the intervention group were encouraged to read instead of watching television. Although BMI was unchanged at the end of the study, fewer children in the intervention group watched more than 2 hours of television, and their total number of hours watched was significantly lower than in the control group.

An extensive search of more recent literature revealed 1 additional randomized school-based feasibility trial (Table 18).<sup>44</sup> Salmon and others<sup>44</sup> enrolled 10-year-old Australian children in the Switch-Play intervention, in which 1 group was encouraged to increase physical activity and reduce sedentary time through behaviour modification; another group increased physical activity by developing fundamental motor skills; and a third group engaged in both interventions. The third group played outdoors more often and reduced computer and electronic game use more than the children in the behaviour modification group, according to self-reported evaluation scores.

## Multifaceted interventions

In their systematic review, Thomas and colleagues<sup>18</sup> identi-

**Table 18:** Outcomes of programs to reduce sedentary behaviour

Study	Study design	Duration of intervention	Grade or age of participants	Results	
				Change in obesity	Other changes
Gortmaker et al <sup>32</sup>	RCT cluster (school)	2 yr	Grades 6-7	↓ BMI (girls) ↓ skinfold measurements (girls)	↓ TV viewing
Robinson <sup>33</sup>	RCT cluster (school)	7 mo	Grades 3-4	↓ BMI ↓ skinfold measurements ↓ waist-to-hip ratio	↓ TV viewing
Robinson et al <sup>34</sup>	RCT	12 wk	Black girls 8-10 yr	None	↓ TV viewing at dinner
Sahota et al <sup>35</sup>	Randomized cluster (school)	1 yr	Elementary school	None	↑ vegetable intake
Dennison et al <sup>43</sup>	RCT cluster (daycare)	12 wk	2.6-5.5 yr	None	↓ TV viewing
Salmon et al <sup>44</sup>	RCT cluster (classroom)	8 mo	10 yr	Not measured	↓ computer and electronic game use ↑ outdoor play

fied 27 RCTs evaluating the efficacy of preventive strategies combining nutrition and physical activity. Of these, 20 were of sufficient duration to be included in this report. Four showed improvements in BMI or percent body fat (Table 19).<sup>27,32,36,45</sup> Ten showed modifications in secondary outcome measures, including improvements in fitness or physical activity levels (1), improved nutritional intake (5) or both (4) (Table 19).<sup>35,37,41,46-52</sup> Six articles failed to demonstrate improvements in physical activity, diet or measures of obesity and are not included in Table 19.<sup>34,39,40,42,54,55</sup>

In their study, Sallis and coworkers<sup>36</sup> looked at the effects of dietary modifications (lower-fat lunches from home and school) and increased physical activity (daily physical education classes and school-based physical activity outside of classes) on students in middle-school (M-SPAN) over 2 years.

Twenty-four schools were randomly assigned to control or intervention groups; their ability to amend policy and implement these changes varied. All students in the intervention schools increased their physical activity levels, but the improvement was significant only for boys. Increased physical activity was associated with a significant reduction in BMI. Boys increased their physical activity during and outside physical education classes, whereas girls did so only during classes. There was no significant change in fat intake at school.

Planet Health incorporated a reduction in television viewing time, a reduction in total fat and saturated fat intake, and an increase in physical activity and fruit and vegetable intake over 2 years in grade 6-8 students.<sup>32</sup> A significant reduction in television viewing was noted, but the prevalence of obesity (BMI, skinfold measurements) decreased only in girls. Girls

**Table 19:** Outcomes of multifaceted interventions to reduce obesity

Study	Study design	Duration of intervention (follow-up)	Grade or age of participants	Results	
				Change in obesity	Other changes
Sallis et al <sup>36</sup>	RCT cluster (school)	2 yr	Grades 6-8	↓ BMI (boys)	↑ physical activity (significant) (boys)
Gortmaker et al <sup>32</sup>	RCT cluster (school)	2 yr	Grades 6-8	↓ BMI (girls) ↓ skinfold measurements (girls)	↓ TV viewing ↑ fruit and vegetable intake (girls)
Burke et al <sup>45</sup>	RCT cluster (school)	1 yr	10-12 yr	↓ body fat (fitness + school nutrition group)	↑ fitness
Flores <sup>27</sup>	RCT cluster (classroom)	12 wk	Grades 5-8	↓ BMI (girls)	↑ fitness (girls)
Bush et al <sup>46</sup>	RCT cluster (school)	3 yr	Elementary school	None	↑ fitness
Luepker et al <sup>37</sup>	RCT cluster (school)	2.5 yr	Grades 3-5	None	↑ vigorous physical activity ↓ fat intake
Story et al <sup>41</sup>	RCT	12 wk	Black girls 8-10 yr	None	↑ physical activity ↑ low-fat food intake
Nader et al <sup>48</sup>	RCT cluster (school)	1 yr (4 yr)	Grades 5-6	None	↑ fitness (girls) ↓ fat intake
Fardy et al <sup>47</sup>	RCT	3 mo (2 yr)	Grades 9-10	None	↑ fitness (girls) ↓ high-fat food intake (girls)
Sahota et al <sup>35</sup>	RCT cluster (school)	1 yr	Elementary school	None	↑ vegetable intake
Walter <sup>49</sup>	RCT cluster (school)	5 yr	Grades 4-9 (low v. higher socioeconomic status)	None	↓ fat intake (in high socioeconomic schools)
Caballero et al <sup>50</sup>	RCT cluster (school)	3 yr	Grade 3 First Nations people (US)	None	↓ energy intake
Warren et al <sup>51</sup>	RCT	14-16 mo	5-7 yr	None	↑ fruit intake
Stolley et al <sup>52</sup>	RCT	12 wk	Black girls 7-12 yr	None	↓ fat intake
Harvey-Berino et al <sup>38</sup>	RCT	16 wk	Preschool First Nations people (US)	↓ weight-for-height percentile (not significant)	↓ energy intake
Danielzik et al <sup>53</sup>	RCT cluster (school)	4 yr	German students 5-7 yr	Trend in improved skinfold measurements (girls)	

also reported eating more fruits and vegetables, but this was probably not clinically significant.

Burke and colleagues<sup>45</sup> compared 5 different interventions over 1 year in groups of students aged 10–12 years: aerobic physical education classes; school nutrition lessons; aerobic physical education classes plus school nutrition lessons; school nutrition and home nutrition messaging; and home nutrition teaching only. Although the 2 interventions that included physical education resulted in improved fitness levels in both sexes, only the group receiving physical education plus school nutrition lessons had decreased percentage body fat. There was a modest trend toward better results in physical activity and nutrition behaviour when parents were involved.

In Flores' study of the effects of aerobic-dance-oriented physical education and health education classes (healthy nutrition, substance abuse prevention, stress management) versus regular physical education for middle-school students, a greater reduction in BMI was seen among girls in the intervention group.<sup>27</sup> These girls also had a lower resting heart rate after the intervention compared with those in the control group.

Programs described in 5 articles increased physical activity or fitness levels with or without improved nutrition (Table 19).<sup>37,41,46–48</sup> Bush and colleagues<sup>46</sup> studied the effects of a multifaceted program (Know Your Body II) on elementary students in 9 schools over 3 years. This program included a school curriculum promoting healthy nutrition and endurance physical activity, parent education and screening for cardiovascular disease risk factors. Although there were no differences in ponderosity index (weight divided by height cubed) and triceps skinfold measurements, the intervention group had a better post-exercise recovery rate. Luepker and coworkers<sup>37</sup> studied the impact of the Child and Adolescent Trial for Cardiovascular Health (CATCH) program on grade 3–5 students over 2.5 years. This study included 2 intervention groups: the first involved changes in school food service plus increased physical activity in physical education classes and the CATCH curriculum; the other involved this school program plus family education. Compared with controls, both intervention groups showed higher levels of vigorous physical activity and lower fat intake but no change in BMI or skinfold measurements. Story and others<sup>41</sup> randomly selected 54 girls aged 8–10 years for a 12-week community-based pilot program that focused on healthy eating and increased physical activity. Family involvement was included. Although no change in BMI or waist circumference was noted, intervention students increased their physical activity levels and low-fat food intake. In the 1-year San Diego Family Health Project, Nader and colleagues<sup>48</sup> studied the effects of interactive games focused on increasing physical activity and healthy diets in low-to-middle-income Anglo-American and Mexican-American grade 5–6 students and their families. There was a modest decrease in fat intake among Anglo-American students and improved fitness scores among girls in the intervention group but not among Mexican-Americans. Fardy and others<sup>47</sup> studied 879 grade 9–10 students in the PATH project over 3 months. Students were assigned to an experimental (25 minutes of aerobic exercise and 5 minutes of nutrition and stress management) or control group (traditional physi-

cal education classes). Two years after the study period, girls in the intervention group were consuming fewer high-fat foods and had significantly higher maximum oxygen consumption than those in the control group.

In 5 studies, nutrition improved without an effect on measures of obesity (Table 19).<sup>35,49–52</sup> In an evaluation of the APPLES program over 1 academic year, Sahota and colleagues<sup>35</sup> found that the intervention group ate more servings of vegetables a day (0.3 servings) than the control group. In the Know Your Body I project, Walter<sup>49</sup> used classroom curricula to promote healthy diet and endurance physical activity over 5 years. Students and parents were scored on behaviour risk status for chronic disease. Walter discovered a significant reduction in total fat intake among students of higher socioeconomic status, but no difference among those of lower socioeconomic status. Caballero and colleagues<sup>50</sup> implemented a 3-year program for grade 3 children of Native American descent (Pathways). This program included increased physical activity during and after school, improved school food choices, healthy new food tasting at school and a home program to encourage healthy active living. Intervention schools offered lunches with lower fat content; their students had significantly lower daily energy intake, although there may have been some reporting bias. In the Be Smart Program, Warren and associates<sup>51</sup> divided students aged 5–7 years from high-income families into 1 control and 3 intervention groups (nutrition; physical activity; nutrition and physical activity) with school- and home-based components and followed them over 14–16 months. Although not likely clinically relevant, fruit intake increased significantly among the students in the nutrition groups. In a 12-week RCT, Stolley and Fitzgibbon<sup>52</sup> studied 62 black mother-and-daughter (aged 7–12 years) pairs, in which the intervention focused on culturally appropriate modifications in diet and physical activity. The mean percentage of daily energy from fat was significantly lower in the treatment group than in the control group.

Two articles were published after July 2003 (Table 19).<sup>38,53</sup> Harvey-Berino and Rourke<sup>38</sup> evaluated the effects of home visiting on preschool Native American children. Indigenous peers taught 2 groups of mothers over 16 weeks through home parenting classes. Lessons focused on healthy diet and exercise or provided typical parenting support. Although not significant, there were trends toward decreasing weight-for-height percentile and reduced energy intake in the group receiving lessons on healthy lifestyles. In a long-term intervention study involving children in elementary school (Kiel Obesity Prevention Study) Danielzik and colleagues<sup>53</sup> assessed 780 first-grade students from 18 schools randomly assigned to the intervention (nutrition education plus 15-minute physical activity breaks) or the control group. Four years later, the intervention had produced only a small effect on the prevalence of overweight, with skinfold thickness increasing to a lesser degree in the intervention group than in the control group.

## Conclusions

The number of well-designed, long-term, controlled prevention studies targeting children is relatively low. Some

short-term, school-based interventions achieved modest success in increasing physical activity levels and reducing sedentary activities. However, evidence of a significant impact in terms of obesity prevention is limited. Few studies focused on the preschool age group, and thus an important opportunity for early intervention was missed. Although some studies addressed racial minorities, research on effective physical activity strategies for Canada's ethnic minorities and immigrant population is lacking. The need for quality obesity prevention studies examining a wide range of interventions in all pediatric age groups and cultures remains a priority.

Multifaceted programs implemented in multiple settings and targeting behaviour change rather than isolated knowledge acquisition are encouraged. Ensuring parental and family involvement may be an important component. Such programs should be designed with methodological rigour and evaluated over the long term.

Schools have emerged as pivotal settings for the promotion of healthy active living. School-based interventions to increase daily physical activity should include dedicated physical education class time with a focus on a variety of aerobic activities as well as other opportunities for active recreation before, after and during school. School-based initiatives that reduce sedentary activity also show promise in preventing obesity.

Long-term prospective intervention studies, particularly those focused on early childhood and minority populations, are needed.

## Recommendations

1. We suggest limiting "screen time" (i.e., television watching, playing video or computer games) to no more than 2 hours a day to encourage more activity and less food consumption, and to limit exposure to food advertising [*grade B, level 3*].
2. The role of schools as pivotal settings for the promotion of healthy active living and school-based prevention programs to reduce the risk of childhood obesity is encouraged, as are interventions to increase daily physical activity through physical education class time and opportunities for active recreation [*grade C, level 4*].
3. The development of programs in multiple settings targeting behaviour change with parental and family involvement is encouraged [*grade C, level 4*].

**Competing interests:** None declared.

From the University of Ottawa, Children's Hospital of Eastern Ontario, Ottawa, Ont. (LeBlanc); QEI Research Services, Ottawa, Ont. (Irving); College of Kinesiology, University of Saskatchewan, and Statistics Canada, Ottawa, Ont. (Tremblay)

## REFERENCES

1. Tremblay MS, Willms JD. Secular trends in the body mass index of Canadian children published erratum in *CMAJ* 2001;164:970]. *CMAJ* 2000;163:1429-33.
2. Whitaker RC, Wright JA, Pepe MS, et al. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med* 1997;337:869-73.
3. Moore LL, Gao D, Bradlee ML, et al. Does early physical activity predict body fat change throughout childhood? *Prev Med* 2003;37:10-7.

4. Berkey CS, Rockett HR, Field AE, et al. Activity, dietary intake, and weight changes in a longitudinal study of preadolescent and adolescent boys and girls. *Pediatrics* 2000;105:E56.
5. Ekelund U, Aman J, Yngve A, et al. Physical activity but not energy expenditure is reduced in obese adolescents: a case-control study. *Am J Clin Nutr* 2002;76:935-41.
6. Eisenmann JC, Bartee RT, Wang MQ. Physical activity, TV viewing, and weight in U.S. youth: 1999 Youth Risk Behavior Survey. *Obes Res* 2002;10:379-85.
7. Kawabe H, Murata K, Shibata H, et al. Participation in school sports clubs and related effects on cardiovascular risk factors in young males. *Hypertens Res* 2000;23:127-32.
8. Lazzer S, Boirie Y, Bitar A, et al. Assessment of energy expenditure associated with physical activities in free-living obese and nonobese adolescents. *Am J Clin Nutr* 2003;78:471-9.
9. Macek M, Bell D, Rutenfranz J, et al. A comparison of coronary risk factors in groups of trained and untrained adolescents. *Eur J Appl Physiol Occup Physiol* 1989;58:577-82.
10. Proctor MH, Moore LL, Gao D, et al. Television viewing and change in body fat from preschool to early adolescence: the Framingham Children's Study. *Int J Obes Relat Metab Disord* 2003;27:827-33.
11. Raitakari OT, Taimela S, Porkka KV, et al. Associations between physical activity and risk factors for coronary heart disease: the Cardiovascular Risk in Young Finns Study. *Med Sci Sports Exerc* 1997;29:1055-61.
12. Rowlands AV, Eston RG, Ingledew DK. Relationship between activity levels, aerobic fitness, and body fat in 8- to 10-year-old children. *J Appl Physiol* 1999;86:1428-35.
13. Schmitz KH, Jacobs DR Jr, Hong CP, et al. Association of physical activity with insulin sensitivity in children. *Int J Obes Relat Metab Disord* 2002;26:1310-6.
14. Suter E, Hawes MR. Relationship of physical activity, body fat, diet, and blood lipid profile in youths 10-15 yr. *Med Sci Sports Exerc* 1993;25:748-54.
15. Tremblay MS, Willms JD. Is the Canadian childhood obesity epidemic related to physical inactivity? *Int J Obes Relat Metab Disord* 2003;27:1100-5.
16. Summerbell CD, Ashton V, Campbell KJ, et al. Interventions for treating obesity in children. *Cochrane Database Syst Rev* 2003;(3):CD001872.
17. Timperio A, Salmon J, Ball K. Evidence-based strategies to promote physical activity among children, adolescents and young adults: review and update. *J Sci Med Sport* 2004;7(1 Suppl):20-9.
18. Thomas H, Ciliska D, Wilson-Abra J, et al. *Effectiveness of physical activity enhancement and obesity prevention programs in children and youth*. Ottawa: Health Canada; 2004. Project no. 6795-15-2002/4470007. Available: www.hc-sc.gc.ca/sr-sr/alt\_formats/iacob-dgiac/pdf/finance/hprp-prpms/final/2004-obesit-activit\_e.pdf (accessed 2007 Feb 26).
19. Stephens MB, Wentz SW. Supplemental fitness activities and fitness in urban elementary school classrooms. *Fam Med* 1998;30:220-3.
20. Tuckman BW, Hinkle JS. An experimental study of the physical and psychological effects of aerobic exercise on schoolchildren. *Health Psychol* 1986;5:197-207.
21. Sallis JF, McKenzie TL, Alcaraz JE, et al. Project SPARK. Effects of physical education on adiposity in children. *Ann NY Acad Sci* 1993;699:127-36.
22. Hansen HS, Froberg K, Hyldebrandt N, et al. A controlled study of eight months of physical training and reduction of blood pressure in children: the Odense school-child study. *BMJ* 1991;303:682-5.
23. Ernst MP, Pangrazi RP. Effects of a physical activity program on children's activity levels and attraction to physical activity. *Ped Exerc Sci* 1999;11:393-405.
24. Ransdell LB, Taylor A, Oakland D, et al. Daughters and mothers exercising together: effects of home- and community-based programs. *Med Sci Sports Exerc* 2003;35:286-96.
25. Rowland D, DiGuseppi C, Gross M, et al. Randomised controlled trial of site specific advice on school travel patterns. *Arch Dis Child* 2003;88:8-11.
26. Mo-suwan L, Junjana C, Puetpaiboon A. Increasing obesity in school children in a transitional society and the effect of the weight control program. *Southeast Asian J Trop Med Public Health* 1993;24:590-4.
27. Flores R. Dance for health: improving fitness in African American and Hispanic adolescents. *Public Health Rep* 1995;110:189-93.
28. Pangrazi RP, Beighle A, Vehige T, et al. Impact of Promoting Lifestyle Activity for Youth (PLAY) on children's physical activity. *J Sch Health* 2003;73:317-21.
29. Jamner MS, Spruijt-Metz D, Bassin S, et al. A controlled evaluation of a school-based intervention to promote physical activity among sedentary adolescent females: project FAB. *J Adolesc Health* 2004;34:279-89.
30. McKenzie TL, Sallis JF, Prochaska JJ, et al. Evaluation of a two-year middle-school physical education intervention: M-SPAN. *Med Sci Sports Exerc* 2004;36:1382-8.
31. Saakslahhti A, Numminem P, Salo P, et al. Effects of a three-year intervention on children's physical activity from age 4 to 7. *Ped Exerc Sci* 2004;16:167-80.
32. Gortmaker SL, Peterson K, Wiecha J, et al. Reducing obesity via a school-based interdisciplinary intervention among youth: Planet Health. *Arch Pediatr Adolesc Med* 1999;153:409-18.
33. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA* 1999;282:1561-7.
34. Robinson TN, Killen JD, Kraemer HC, et al. Dance and reducing television viewing to prevent weight gain in African-American girls: the Stanford GEMS pilot study. *Ethn Dis* 2003;13(Suppl 1):S65-77.
35. Sahota P, Rudolf MC, Dixey R, et al. Randomised controlled trial of primary school based intervention to reduce risk factors for obesity. *BMJ* 2001;323:1029-32.
36. Sallis JF, McKenzie TL, Conway TL, et al. Environmental interventions for eating and physical activity: a randomized controlled trial in middle schools. *Am J Prev Med* 2003;24:209-17.
37. Luepker RV, Perry CL, McKinlay SM, et al. Outcomes of a field trial to improve children's dietary patterns and physical activity. The Child and Adolescent Trial for Cardiovascular Health. CATCH collaborative group. *JAMA* 1996;275:768-76.

38. Harvey-Berino J, Rourke J. Obesity prevention in preschool native-American children: a pilot study using home visiting. *Obes Res* 2003;11:606-11.
39. Baranowski T, Baranowski JC, Cullen KW, et al. The Fun, Food, and Fitness Project (FFFP): the Baylor GEMS pilot study. *Ethn Dis* 2003;13(Suppl 1):S30-9.
40. Beech BM, Klesges RC, Kumanyika SK, et al. Child- and parent-targeted interventions: the Memphis GEMS pilot study. *Ethn Dis* 2003;13(Suppl 1):S40-53.
41. Story M, Sherwood NE, Himes JH, et al. An after-school obesity prevention program for African-American girls: the Minnesota GEMS pilot study. *Ethn Dis* 2003; 13(Suppl 1):S54-64.
42. Neumark-Sztainer D, Story M, Hannan PJ, et al. New Moves: a school-based obesity prevention program for adolescent girls. *Prev Med* 2003;37:41-51.
43. Dennison BA, Russo TJ, Burdick PA, et al. An intervention to reduce television viewing by preschool children. *Arch Pediatr Adolesc Med* 2004;158(2):170-6.
44. Salmon J, Ball K, Crawford D, et al. Reducing sedentary behaviour and increasing physical activity among 10-year-old children: overview and process evaluation of the 'Switch-Play' intervention. *Health Promot Int* 2005;20:7-17.
45. Burke V, Thompson C, Taggart AC, et al. Differences in response to nutrition and fitness education programmes in relation to baseline levels of cardiovascular risk in 10 to 12-year-old children. *J Hum Hypertens* 1996;10(Suppl 3):S99-106.
46. Bush PJ, Zuckerman AE, Theiss PK, et al. Cardiovascular risk factor prevention in black schoolchildren: two-year results of the "Know Your Body" program. *Am J Epidemiol* 1989;129:466-82.
47. Fardy PS, Azzollini A, Magel JR, et al. Effects of school-based health promotion on obesity: the Path program. *Med Sci Sports Exerc* 2002;34(5 supp):S68.
48. Nader PR, Sallis JF, Abramson IS, et al. Family-based cardiovascular risk reduction education among Mexican-and-Anglo-Americans and Europeans. *Fam Commun Health* 1992;15:57-74.
49. Walter HJ. Primary prevention of chronic disease among children: the school-based "Know Your Body" intervention trials. *Health Educ Q* 1989;16:201-14.
50. Caballero B, Clay T, Davis SM, et al. Pathways: a school-based, randomized controlled trial for the prevention of obesity in American Indian schoolchildren. *Am J Clin Nutr* 2003;78:1030-8.
51. Warren JM, Henry CJ, Lightowler HJ, et al. Evaluation of a pilot school programme aimed at the prevention of obesity in children. *Health Promot Int* 2003;18:287-96.
52. Stolley MR, Fitzgibbon ML. Effects of an obesity prevention program on the eating behavior of African American mothers and daughters. *Health Educ Behav* 1997;24: 152-64.
53. Danielzik S, Pust S, Landsberg B, et al. First lessons from the Kiel Obesity Prevention Study (KOPS). *Int J Obes (Lond)* 2005;29(Suppl 2):S78-83.
54. Everhart B, Harshaw C, Everhart B, et al. Multimedia software's effects on high school physical education students' fitness patterns. *Physical Educator* 2002;59: 151-7.
55. Petchers MK, Hirsch EZ, Bloch BA. A longitudinal study of the impact of a school heart health curriculum. *J Community Health* 1988;13:85-94.

# 23. Prevention of childhood obesity through nutrition: review of effectiveness

Glenn Berall, Virginia Desantadina

Canadian childhood obesity rates are increasing at an alarming pace. They have tripled since the 1980s.<sup>1</sup> Between 1981 and 1996, the proportion of Canadian children who were obese (BMI  $\geq$  95th percentile) increased from 5% to 16.6% among boys and from 5% to 14.6% among girls.<sup>1</sup> Treating such a high percentage of children is extremely challenging. With mounting evidence of persistent elevated medical risks in children with obesity, even following successful treatment, prevention must be the primary focus. There are numerous opportunities to emphasize prevention of obesity throughout life. In this chapter, we review nutritional interventions for the prevention of childhood obesity.

Recent reviews indicate that evidence regarding the prevention of childhood obesity is limited.<sup>2-4</sup> In this section, we focus on more recent literature; however, there is currently a paucity of high-quality evidence-based studies in this area. For the purposes of this review, we accepted evidence of prevention of childhood obesity in a study if the outcome measures included a change in prevalence of obesity in the studied population.

## Data sources

Studies were selected based on a systematic review of RCTs showing changes in prevalence of obesity or behaviour. The following electronic databases were searched: MEDLINE, EMBASE and the Cochrane Controlled Clinical Trials Register. The focus of selection was prospective clinical trials aimed at obesity prevention in children through nutritional interventions and approaches.

Some studies other than RCTs were included to highlight specific concepts or in the absence of relevant RCTs.

## Nutrition in utero

Evolving evidence suggests that prevention of childhood obesity may begin during pregnancy. It is important to address the expectant mother's weight and metabolic health, since a healthy pregnancy improves the baby's future nutritional health. Babies born either small- or large-for-gestational-age are at risk of later obesity.<sup>5,6</sup> Ravelli and colleagues<sup>7</sup> demonstrated an association between famine in utero and later adult obesity. The relation with mother's state of health (through maternal malnutrition or gestational diabetes) indicates that maternal health may have an influence on obesity risk in the offspring.<sup>7</sup> However, these data are preliminary and limited, no trials have shown how prescribed change in maternal nu-

trition influences obesity in the offspring, and RCT studies of in utero nutrition in humans are not likely to be performed for ethical reasons. However, although there is not enough evidence on which to base a conclusion, the above-noted studies suggest that prevention of childhood obesity commences with proper in utero nutrition.

## Nutrition in infancy

A number of variables in early infancy have been studied to determine their relation to later obesity. Evidence suggests that there are critical periods for the development of obesity, including gestation and early infancy, the period of adiposity rebound that occurs between 5 and 7 years of age, and adolescence.<sup>8</sup> Low and high birth weights are associated with an increased risk of later obesity.<sup>9</sup> Low birth weight, even if in the lower range of normal, is associated with low muscularity in early childhood and central fat deposition in childhood.<sup>10-12</sup>

Exclusive breast-feeding is considered the best way to ensure optimum nutrition for babies until 6 months, but does breast-feeding contribute to the prevention of obesity in childhood? Data from the US Centers for Disease Control and Prevention's Pediatric Nutrition Surveillance System suggest that breast-feeding is protective against pediatric overweight.<sup>13</sup> Dewey<sup>14</sup> found a strong protective effect in children aged 9-18 years. Birch and Fisher<sup>15</sup> showed that children who are breast-fed may learn to self-regulate energy intake better than non-breast-fed infants. Breast-fed and formula-fed infants have different responses to feeding; formula-fed infants demonstrate a greater insulin response (possibly resulting in earlier fat deposition) and higher protein intake.<sup>16-18</sup> Breast-fed infants adapt more readily to new foods, such as vegetables; thus, the subsequent energy density, food preferences and nutrient variety of their diet are influenced by their being breast-fed in infancy. The association between breast-feeding and pediatric weight may also be influenced by behavioural factors.<sup>13</sup> In addition, mothers who breast-fed their infants for longer periods report less restrictive feeding behaviour at 1 year.<sup>19</sup>

The American Academy of Pediatrics<sup>20</sup> recommends the encouragement, support and protection of breast-feeding as a strategy to prevent pediatric overweight and obesity. In 2004 Health Canada<sup>3</sup> stated, "Exclusive breastfeeding is recommended for the first six months of life for healthy term infants, as breast milk is the best food for optimal growth. Infants should be introduced to nutrient-rich, solid foods with particular attention to iron at six months with continued breastfeeding for up to two years and beyond."

In a recent meta-analysis of 29 studies,<sup>21</sup> the authors concluded that initial breast-feeding protects against obesity in later life. They recommended further studies to examine systematically the effects of duration of breast-feeding on obesity in adulthood and the effects of confounding factors on the relation between breast-feeding and obesity.

Formula-feeding is associated with more rapid weight gain in early infancy and an increased risk of obesity in childhood and adolescence.<sup>22-25</sup> Furthermore, a rapid increase in weight in the first weeks of life may increase the risk of obesity.<sup>7</sup>

## Nutrition in childhood

Access to energy-dense foods has been shown to have an impact throughout childhood. The pattern of dietary intake of fats and energy among children resembles that of their parents.<sup>26</sup> Although monogenic conditions contributing to obesity have been identified, they contribute only a minor component to childhood obesity;<sup>27</sup> the major parental contribution appears to be in terms of environmental influence.

According to Tershakovec and others,<sup>28</sup> the early treatment and management of obese people and secondary prevention of obesity depend in part on the relation between resting energy expenditure and age. Because resting energy expenditure decreases as a child develops, the sooner preventative measures are established, the less likely the child will become overweight.

Primary schools are particularly suitable for obesity prevention programs, since children in this age group are responsive to health messages, and behavioural changes may be maintained into adolescence and adulthood. Programs targeting obese children and adolescents have reported positive results, particularly those aimed at children in primary school and those combining diet and exercise with parental involvement. However, most of these studies targeted obese children (often volunteered by parents).

Evidence shows that Canadian children tend not to eat breakfast.<sup>3</sup> Skipping breakfast may lead to overeating later in the day and has been associated with increased BMIs.

Several factors inherent to fast foods may increase energy intake, thus promoting a positive energy balance and increasing risk of obesity. There is a direct association between fast-food consumption and age, with consumption greatest in adolescence. Those who eat more fast food also consume more total fat, more sugar, less fibre and fewer fruits, vegetables and dairy products.<sup>29,30</sup> They also tend to skip breakfast and consume more food at supper.<sup>31</sup> A school-based intervention by James and others<sup>32</sup> demonstrated a moderate decrease in sugar consumption and consequent reduction in overweight and obesity. Bowman and colleagues<sup>33</sup> studied nationally representative household data to determine the habitual diets (self-reported) of children in the United States. They concluded that, on a typical day in which fast food is eaten, children consume substantially more total energy and have worse dietary quality than on a typical day without fast food.

The American Academy of Pediatrics policy statement<sup>20</sup> on the prevention of pediatric overweight states: "Families should be educated and empowered through anticipatory

guidance to recognize the impact they have on their child's development of lifelong habits of physical activity and nutritious eating."

Higher weight has been associated with a greater amount of television viewing in North America and Europe.<sup>34-36</sup> However, the positive correlation with obesity is only partly explained by the limited activity. Childhood exposure to food advertising also contributes to consumption of the higher-energy foods being promoted.<sup>37</sup>

Parents as role models are key to promoting preventive change in children, since they have a significant influence, particularly on younger children.<sup>38</sup> Parents appear to have some influence on fruit and vegetable consumption among middle-school students.<sup>39</sup> However, interventions in this realm need to be behaviour-based, because education programs only increase knowledge and do not change behaviour.

## Nutrition in school

School staff have access to large numbers of children in an environment that has the potential to support healthy behaviour and is favourable for the delivery of health promotion programs.<sup>40,41</sup> According to Hayman and coworkers<sup>42</sup> in an Atherosclerosis, Hypertension, and Obesity in Youth statement for health and education professionals and child health advocates, "School health programs initiated in preschool and extending through high school have the potential to influence the cardiovascular health of the majority of US children and youth."

In the American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood, Kavey and others<sup>43</sup> indicate that "an increasing body of research now documents the safety and success of intervention to reduce risk factors in childhood.

Programs that include recommendations for school-based healthy eating appear to be successful: schools with such programs have been found to promote lower rates of overweight and obesity, healthier diets and more physical activities than schools without nutrition programs or interventions.<sup>32,44-47</sup> For example, in a study reported by James and others,<sup>32</sup> education on reduced "fizzy drink" consumption resulted in behaviour change and weight reduction in intervention schools compared with controls, although the impact was moderate.

School interventions also seem to be effective in bringing about increases in children's consumption of fruit and vegetables,<sup>48</sup> and school-based low-fat diets have been found to be effective.<sup>49</sup> Frenn and colleagues<sup>50</sup> found that the fat content in a student's diet varied predictably by the student's stage of behaviour change. They concluded that, when implementing interventions, less classroom time is needed when the focus of the intervention is appropriate to the students' stage of change and when those at more advanced stages act as peer role models. Veugelers and Fitzgerald<sup>51</sup> demonstrated an association between bringing lunches to school and lower risk of overweight.

Although the above studies point in specific directions, they are weak by virtue of the small numbers of participants and design limitations. Nevertheless, we recommend the en-



couragement of school-based prevention programs to reduce the risk of childhood obesity.

## Conclusions

The number of well-designed, long-term, controlled studies of obesity prevention in children is relatively low. Some studies have shown a modest protective effect of breast-feeding against later childhood obesity. However, the connection with adult obesity is not yet clear. Prevention may well best be started while the child is still in utero. Increasing evidence suggests that applying lifestyle changes to the entire child life cycle is justified. In particular, there is a significant opportunity to have an impact by targeting children at school, although identification of successful programs has been challenging.

## Recommendations

1. Discussion of the prevention of childhood obesity with the pregnant mother is encouraged [grade C, level 4].
2. Exclusive breast-feeding of infants is encouraged until at least 6 months of age to prevent later obesity [grade C, level 4].
3. Discussion of limiting consumption of energy-dense snack foods high in sugar and fat during childhood and adolescence is encouraged [grade C, level 4].

From the Department of Pediatrics, University of Toronto (Berall), and the North York General Hospital, Toronto, Ont. (Berall, Desantadina)

**Competing interests:** None declared for Virginia Desantadina. Glenn Berall received funding for research related to Prader–Willi syndrome, a genetic condition including obesity, from the Ontario Prader–Willi Syndrome Association.

## REFERENCES

1. Tremblay MS, Willms JD. Secular trends in the body mass index of Canadian children [published erratum in *CMAJ* 164:970]. *CMAJ* 2000;163:1429-33.
2. Canadian Association of Paediatric Health Centres (CAPHC), the Paediatric Chairs of Canada (PCC), and the Canadian Institutes of Health Research, Institute of Nutrition, Metabolism and Diabetes (CIHR, INMD). *Addressing childhood obesity: the evidence for action*. Burnaby (BC): Institute of Nutrition, Metabolism and Diabetes; 2004. Available: [www.cihr-irsc.gc.ca/e/23293.html](http://www.cihr-irsc.gc.ca/e/23293.html) (accessed 2007 Jan 8).
3. *Nursing best practice guidelines: shaping the future of nursing. Primary prevention of childhood obesity*. Toronto: Registered Nurses' Association of Ontario; 2005. Available: [www.rnao.org/bestpractices/PDF/BPG\\_childhood\\_obesity.pdf](http://www.rnao.org/bestpractices/PDF/BPG_childhood_obesity.pdf) (accessed 2007 Jan 8).
4. Summerbell CD, Ashton V, Campbell KJ, et al. Interventions for treating obesity in children. *Cochrane Database Syst Rev* 2003;(3):CD001872.
5. Hediger ML, Overpeck MD, McGlynn A, et al. Growth and fatness at three to six years of age of children born small- or large-for-gestational age. *Pediatrics* 1999;104:e33.
6. Kokkoris P, Pi-Sunyer FX. Obesity and endocrine disease. *Endocrinol Metab Clin North Am* 2003;32:895-914.
7. Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. *N Engl J Med* 1976;295:349-53.
8. Dietz WH. Critical periods in childhood for the development of obesity. *Am J Clin Nutr* 1994;59:955-9.
9. Charles MA. Early nutrition and weight evolution in children. *Ann Endocrinol (Paris)* 2005;66(2 pt 3):2511-8.
10. Hediger ML, Overpeck MD, Kuczmarski RJ, et al. Muscularity and fatness of infants and young children born small- or large-for-gestational-age. *Pediatrics* 1998;102:E60.
11. Malina RM, Katzmarzyk PT, Beunen G. Birth weight and its relationship to size attained and relative fat distribution at 7 to 12 years of age. *Obes Res* 1996;4:385-90.
12. Barker M, Robinson S, Osmond C, et al. Birth weight and body fat distribution in adolescent girls. *Arch Dis Child* 1997;77:381-3.
13. Grummer-Strawn LM, Mei Z. Does breastfeeding protect against pediatric overweight? Analysis of longitudinal data from the Centers for Disease Control and

- Prevention Pediatric Nutrition Surveillance System. *Pediatrics* 2004;113:e81-6.
14. Dewey KG. Is breastfeeding protective against child obesity? *J Hum Lact* 2003;19:9-18.
15. Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. *Pediatrics* 1998;101:539-49.
16. Lucas A, Sarson DL, Blackburn AM, et al. Breast vs. bottle: endocrine responses are different with formula feeding. *Lancet* 1980;1:1267-9.
17. Lucas A, Boyes S, Bloom SR, et al. Metabolic and endocrine responses to a milk feed in six-day-old term infants: differences between breast and cow's milk formula feeding. *Acta Paediatr Scand* 1981;70:195-200.
18. Burns SP, Desai M, Cohen RD, et al. Gluconeogenesis, glucose handling, and structural changes in livers of the adult offspring of rats partially deprived of protein during pregnancy and lactation. *J Clin Invest* 1997;100:1768-74.
19. Taveras EM, Scanlon KS, Birch L, et al. Association of breastfeeding with maternal control of infant feeding at age 1 year. *Pediatrics* 2004;114:e577-83.
20. Krebs NF, Jacobson MS. Prevention of pediatric overweight and obesity. *Pediatrics* 2003;112:424-30.
21. Owen CG, Martin RM, Whincup PH, et al. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. *Pediatrics* 2005;115:1367-77.
22. Dewey KG. Nutrition, growth, and complementary feeding of the breastfed infant. *Pediatr Clin North Am* 2001;48:87-104.
23. Baker JL, Michaelsen KF, Rasmussen KM, et al. Maternal prepregnant body mass index, duration of breastfeeding, and timing of complementary food introduction are associated with infant weight gain. *Am J Clin Nutr* 2004;80:1579-88.
24. Von Kries R, Koletzko B, Sauerwald T, et al. Breast feeding and obesity: cross sectional study. *BMJ* 1999;319:147-50.
25. Gillman MW, Rifas-Shiman SL, Camargo CA Jr, et al. Risk of overweight among adolescents who were breastfed as infants. *JAMA* 2001;285:2461-7.
26. Oliveria SA, Ellison RC, Moore LL, et al. Parent-child relationships in nutrient intake: the Framingham Children's Study. *Am J Clin Nutr* 1992;56:593-8.
27. O'Rahilly S, Farooqi IS, Yeo GS, et al. Minireview: human obesity-lessons from monogenic disorders. *Endocrinology* 2003;144:3757-64.
28. Tershakovec AM, Kuppler KM, Zemel B, et al. Age, sex, ethnicity, body composition, and resting energy expenditure of obese African American and white children and adolescents. *Am J Clin Nutr* 2002;75:867-71.
29. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet* 2001;357:505-8.
30. French SA, Harnack L, Jeffery RW. Fast food restaurant use among women in the POUND of Prevention study: dietary, behavioral and demographic correlates. *Int J Obes Relat Metab Disord* 2000;24:1353-9.
31. Bowman SA, Vinyard BT. Fast food consumption of U.S. adults: impact on energy and nutrient intakes and overweight status. *J Am Coll Nutr* 2004;23:163-8.
32. James J, Thomas P, Cavan D, et al. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial. *BMJ* 2004;328:1237.
33. Bowman SA, Gortmaker SL, Ebbeling CB, et al. Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. *Pediatrics* 2004;113:112-8.
34. Janssen I, Katzmarzyk PT, Boyce WF, et al. Comparison of overweight and obesity prevalence in school-aged youth from 34 countries and their relationships with physical activity and dietary patterns. *Obes Res* 2005;13:123-32.
35. Dietz WH Jr, Gortmaker SL. Do we fatten our children at the television set? Obesity and television viewing in children and adolescents. *Pediatrics* 1985;75:807-12.
36. Robinson TN. Reducing children's television viewing to prevent obesity: a randomized controlled trial. *JAMA* 1999;282:1561-7.
37. Halford JC, Gillespie J, Brown V, et al. Effect of television advertisements for foods on food consumption in children. *Appetite* 2004;42:221-5.
38. Baranowski T, Cullen KW, Nicklas T, et al. School-based obesity prevention: a blueprint for taming the epidemic. *Am J Health Behav* 2002;26:486-93.
39. Young EM, Fors SW, Hayes DM. Associations between perceived parent behaviors and middle school student fruit and vegetable consumption. *J Nutr Educ Behav* 2004;36:2-8.
40. Wang LY, Yang Q, Lowry R, et al. Economic analysis of a school-based obesity prevention program. *Obes Res* 2003;11:1313-24.
41. Valteau L, Almeida S, Deane ME, et al. Ontario Society of Nutrition Professionals in Public Health School Nutrition Workgroup Steering Committee. *Call to action: creating a healthy school nutrition environment*. Toronto: Ontario Society of Nutrition Professionals in Public Health; 2004. Available: [www.osnpph.on.ca/pdfs/call\\_to\\_action.pdf](http://www.osnpph.on.ca/pdfs/call_to_action.pdf) (accessed 2007 Jan 8).
42. Hayman LL, Williams CL, Daniels SR, et al. Cardiovascular health promotion in the schools: a statement for health and education professionals and child health advocates from the Committee on Atherosclerosis, Hypertension, and Obesity in Youth (AHOY) of the Council on Cardiovascular Disease in the Young, American Heart Association. *Circulation* 2004;110:2266-75.
43. Kavey RE, Daniels SR, Lauer RM, et al. American Heart Association guidelines for primary prevention of atherosclerotic cardiovascular disease beginning in childhood. *Circulation* 2003;107:1562-6.
44. Veugelers PJ, Fitzgerald AL. Effectiveness of school programs in preventing childhood obesity: a multilevel comparison. *Am J Public Health* 2005;95:432-5.
45. Stevens J, Story M, Ring K, et al. The impact of the Pathways intervention on psychosocial variables related to diet and physical activity in American Indian schoolchildren. *Prev Med* 2003;37:S70-9.
46. Caballero B, Clay T, Davis SM, et al. Pathways: a school-based, randomized con-

- trolled trial for the prevention of obesity in American Indian schoolchildren. *Am J Clin Nutr* 2003;78:1030-8.
47. Warren JM, Henry CJ, Lightowler HJ, et al. Evaluation of a pilot school programme aimed at the prevention of obesity in children. *Health Promot Int* 2003;18:287-96.
  48. Horne PJ, Tapper K, Lowe CF, et al. Increasing children's fruit and vegetable consumption: a peer-modelling and rewards-based intervention. *Eur J Clin Nutr* 2004; 58:1649-60.
  49. Contento IR, Kell DG, Keiley MK, et al. A formative evaluation of the American Cancer Society Changing the Course nutrition education curriculum. *J Sch Health* 1992;62:411-6.
  50. Frenn M, Malin S, Bansal NK. Stage-based interventions for low-fat diet with middle school students. *J Pediatr Nurs* 2003;18:36-45.
  51. Veugelers PJ, Fitzgerald AL. Prevalence of and risk factors for childhood overweight and obesity. *CMAJ* 2005;173:607-13.

## 24. Obesity prevention in the Canadian population: policy recommendations for environmental change

Kim Raine, Elinor Wilson

According to the World Health Organization, “the fundamental causes of the obesity epidemic are societal, resulting from an environment that promotes sedentary lifestyles and the consumption of high-fat, energy-dense diets.”<sup>1</sup> In light of the increase in the prevalence of overweight and obesity among adults and children in Canada, the impact of obesity on noncommunicable and chronic diseases, and the substantial costs to the health care system (over \$1.8 billion per year in 1997)<sup>2</sup> and Canadian society as a whole, policies to support environmental change for obesity prevention and control are urgently required.

A number of authors have advocated an ecological approach to addressing the complex interaction of factors influencing population weight status. Ecological approaches suggest multilevel public health strategies to promote healthy lifestyles and reduce obesity.<sup>3–8</sup> Ecological approaches are consistent with health promotion in that they can help to organize strategies that work both to support healthy lifestyles among individuals and to influence policy to create opportunities for social and cultural change.<sup>9</sup> The emphasis here will be on environmental and population-level strategies — not in opposition to strategies at the individual level, but in support of them. There are implications for policy at both levels.

### Informing strategies to address the obesity epidemic

Inadequate surveillance of obesity, its determinants (food intake, physical activity and the social environment that influences these behaviours) and associated noncommunicable diseases continues to limit understanding of the problem and appropriate targeting of public health interventions. Without adequate surveillance data, policy and planning measures cannot be evidence based. A comprehensive and integrated surveillance system must address a wide spectrum of planning, implementation and evaluation needs to guide researchers, policy-makers, health care planners and community organizers in the planning, development, implementation and evaluation of effective obesity prevention interventions and policies.

### Developing an evidence base for environmental and population-level policy interventions

Evidence-based practice is the systematic use of scientifically rigorous evidence to inform practice. Unlike clinical decision-making, where the evidence base is dominated by RCTs, envi-

ronmental and population-level policy interventions for obesity prevention draw on a broad range of types of evidence from a variety of study designs, epidemiologic observations, experience with other public health issues (e.g., parallel evidence from tobacco reduction), theory and informed opinion to provide contextual relevance.<sup>10</sup> There is a need to balance practice-based evidence (community or culturally based practices that are successful within diverse populations) with evidence-based practice.

The increasing prevalence of obesity and its impact on many chronic diseases confirms the urgency with which the obesity problem in Canada needs to be addressed. However, evaluation of long-term RCTs could take years. There is a need to look at practice-based evidence and community interventions to find examples of successful obesity prevention initiatives. Such thinking is consistent with a framework that has been proposed for translating obesity-related evidence into action.<sup>10</sup> This framework suggests that, as the potential population impact increases, the promise of impact is such that intervention is warranted even in light of lower certainty of evidence. However, funding research that includes evaluation of public health interventions and “natural experiments” is essential to increase the evidence base. Research is needed to determine the most effective interventions to deliver, appropriate delivery vehicles and the circumstances required for success. Continual monitoring of programs, evaluation and fine-tuning are required to create effective policy recommendations.

### Promising policy options for confronting obesity

Public health targets the community as a whole in terms of prevention, regardless of the current level of disease risk.<sup>11</sup> The main objective of public health policy is to provide the population with the best chance to enjoy as many years of healthy and active life as possible. Effective implementation of a public health approach to obesity will require a shift away from the traditional focus on clinical management and individual behaviour change toward strategies that change the environment in which these behaviours occur.<sup>12</sup> A public health approach requires the integration of various educational, environmental, economic, technical and legislative measures to influence change.<sup>12</sup> The level of social change that was required for other health and social issues, such as tobacco reduction, seatbelt use, breast-feeding and recycling, will be needed for the current obesity epidemic. These successful models have predominantly targeted environment and population-level policy.<sup>13</sup>

## Environmental strategies to promote healthy living

Environmental strategies involve institution- and community-based interventions to promote healthy living. Their focus is not necessarily on weight reduction or obesity prevention per se, but on encouraging healthy eating, increased physical activity and decreased sedentary living. Implementation of environment-based strategies needed to encourage and support behaviour change requires the involvement of people from relevant sectors outside the obesity field.<sup>14</sup> Schools, work sites, and local communities and municipalities are likely settings for change.

### A comprehensive school health program

A model for a comprehensive school health program has been proposed to address obesity. This approach includes a broad spectrum of activities and services that take place in schools and their surrounding communities to enable children and adolescents to improve their health, to develop to their fullest potential and to establish productive and satisfying relations in their present and future lives. Evidence suggests that such a program would include 8 interacting components: health instruction, health services, school environment, food service, school-site health promotion for faculty and staff, social support services, physical education classes, and integrated and linked family and community health-promotion efforts.<sup>15</sup> Recent Canadian evidence has shown the dramatic effect of comprehensive school health programs on childhood obesity rates, whereas individual components of the model (nutrition programs) showed no effect.<sup>16</sup> At this point, we do not know the impact on obesity rates among children and adolescents of recent provincial policies to address time spent in school physical activity or to implement food policies such as bans on soft drinks sold in school vending machines.<sup>17</sup>

### Work-site obesity programs

Most published reports of work-site obesity programs have emphasized a clinical perspective through occupational health and safety departments that provide employees with counselling, referral or resources for weight management.<sup>18</sup> Comprehensive programs that include physical activity and, in some cases, smoking cessation, weight reduction and substance abuse control have been shown to provide a cost-benefit ratio to the corporation as high as 1:5; that is, an investment of \$100 per employee in health promotion programs can save up to \$500 per employee through increased productivity and decreased absenteeism and turnover, medical costs, occupational injuries and premature deaths.<sup>19</sup> A recent systematic review concluded that work-site interventions to promote healthy eating and active living can result in healthier weights among employees.<sup>20</sup> A variety of moderately feasible environmental interventions with estimated moderate impact at work sites include the introduction of fitness facilities, bicycle racks, shower facilities, flexible hours to accommodate time for exercise, kitchen facilities, point-of-purchase nutri-

tion information in cafeterias and on vending machines, as well as availability of healthy food choices at a reasonable price through subsidization.<sup>21,22</sup> A unique and relatively low-cost intervention that has been evaluated for effectiveness is making stairwells more easily accessible using prominent signage, combined with artwork and music to increase the attractiveness of use.<sup>23</sup> Although tested at a work site, stairwell interventions have broader community applications.

### Community initiatives

Community initiatives to promote healthy eating provide ready access to a variety of nutritious, affordable foods and disincentives for less-healthy alternatives. Targeted settings for intervention include food retailers, food service operations and community food producers. Point-of-choice nutrition education in food retail and service operations has been used extensively with variable success in motivating consumer choices.<sup>24</sup> Pricing strategies have also been used to promote healthy food choices. For example, reducing the relative price of low-fat snacks has been effective in promoting their purchase from vending machines in both adult and adolescent populations.<sup>25</sup> Studies suggest that price decreases may be more powerful than health messages in increasing consumption of healthy foods.<sup>26</sup> Interventions that take the needs of low-income people into consideration may include community programs, such as collective kitchens with a focus on healthy food choices and access to public green space for community gardens to support production of low-cost nutritious food as well as active living. Little direct evidence exists on the effectiveness of such programs; they are best considered within the context of broader policy change to address economically vulnerable groups, as described later (in the section “Strategies for population-wide structural change”).

Strategies for community-wide active living combine media, newsletters, special events, promotional materials, information lines, health fairs and partnerships with work sites and schools. Based on the demonstrated effectiveness of community interventions in increasing physical activity and improving fitness, the Task Force on Community Preventive Services<sup>27</sup> strongly recommends such campaigns. Canadian examples include Saskatoon’s In Motion program ([www.saskatoonhealthregion.ca/your\\_health/in\\_motion.htm](http://www.saskatoonhealthregion.ca/your_health/in_motion.htm)), Active Healthy Kids Canada ([www.activehealthykids.ca](http://www.activehealthykids.ca)) and Nova Scotia’s Active Kids, Healthy Kids strategy.<sup>28</sup> Community-based campaigns to increase neighbourhood safety, provide adequate lighting and advocate for park space are also plausible means of making communities safer places to be physically active.<sup>29</sup> In rural communities, construction of walking trails has been found effective in promoting physical activity, particularly among people of lower socioeconomic status.<sup>30</sup> Such strategies require buy-in from multiple sectors; thus, they are frequently an entry point to broader, structural change.

### Strategies for population-wide structural change

Most effective interventions to change diet and physical ac-

tivity patterns at the population level adopt an integrated, multidisciplinary, comprehensive approach; involve a complementary range of actions; and work at individual, community, environmental and policy levels.<sup>31</sup> A review of strategies for population-based change reveals that their success requires political support to ensure that programs are well resourced and integrated into existing programs and structures.<sup>22</sup> In addition, intersectoral collaboration and community participation are essential to ensure that programs are sustainable, tailored to meet local needs, able to reach more than just the “motivated healthy” and prepared to capture local opportunities.<sup>22</sup>

### **Dietary and physical activity guidance**

Well-established policy approaches to the promotion of healthy eating and active living are dietary and physical activity guidance. Such guidance builds on the population health model and sets out strategic directions to encourage policy and program development that is coordinated and intersectoral, supports new and existing partnerships, promotes the efficient use of limited resources and strengthens research to improve health. These standards and recommendations could all be used as a foundation for consumer education about portion sizes and the labelling of nutritional content of foods. In January 2003, Health Canada announced new mandatory nutrition labelling to help Canadians make informed choices about healthy eating.<sup>32</sup> A policy option is to consider legislation to regulate “reasonable”-sized portions and enforce disclosure of nutritional content of snack and fast foods at point-of-purchase and on product labels.<sup>33</sup>

### **Land development and transportation planning**

As land uses become separate, distances between them increase, roads are more available than cycling paths or sidewalks, and vehicle travel becomes preferable.<sup>34,35</sup> The proportion of people who are overweight or obese increases as the number of daily miles travelled on foot decreases.<sup>36</sup> According to a study in 2000, a person’s immediate environment or neighbourhood is the most important determinant of physical activity.<sup>37</sup> Applying a public health lens to urban planning would promote mixed land use and planning for active transportation. Streets that incorporate pedestrian (connecting pathways, sidewalks, crosswalks) and bicycle facilities (lanes or paths) and that are “calmed” (discouraging high-speed vehicle traffic through the use of speed bumps and obstacles) facilitate active transportation.<sup>35,38,39</sup> Eighty-two percent of Canadians believe that governments should support spending on bike lanes.<sup>40</sup>

### **Potential legislative action and taxation policies**

Potential legislative actions include taxation and media regulation. Evidence from the tobacco control issue in Canada suggests that taxation policies may be useful in addressing obesity. In Canada, the goods and services tax and harmonized sales tax (GST/HST) function as a “sin tax” for food:

basic groceries are exempt, whereas foods prepared by eating establishments, those for catering and vending machines and specific foodstuffs such as alcoholic beverages, soft drinks and snack foods are taxed.<sup>41</sup> Although initially the GST/HST may have acted as a disincentive to purchasing foods promoting obesity, over the long run there does not seem to be an impact on purchasing. However, revenues from GST/HST on foodstuffs could be earmarked for health promotion and obesity prevention, as is the case in some states in the United States.<sup>42</sup>

Viable policy options for Canada would be:

- Development of a strategy to complement GST/HST by subsidizing the cost of low-energy nutritious foods with taxes of sufficient magnitude to affect sales of high-energy, low-nutrient foods.<sup>43</sup> The effect would be a changed price structure in favour of more nutritious choices.<sup>44</sup>
- Taxation policies that could promote physical activity include the removal of sales taxes on exercise equipment and the offering of tax incentives to employers who provide employees with fitness facilities.<sup>43</sup>
- Taxes to discourage urban sprawl, such as congestion or traffic taxes, rush-hour tolls, subdivision fees and gasoline taxes, may also promote physical activity by encouraging densification and active commuting.<sup>33</sup>

Another policy area that is proving to be successful in tobacco control is regulating exposure to advertising and marketing. The limiting of tobacco advertising through television, billboards, magazines and other media outlets has contributed to the decline in tobacco use in Canada. Children may be particularly vulnerable to advertising of energy-dense foods and marketing of fast foods.<sup>45</sup> In a recent analysis of the results of 123 peer-reviewed studies of food marketing, children’s food consumption and weight status, the Institute of Medicine found adequate evidence to link obesity rates and marketing targeted at children.<sup>46</sup> The following policy options are directed at children’s media and supported by the Institute of Medicine report: restricting advertising of “junk” foods during peak television-viewing times for children;<sup>43</sup> regulating advertising time to ensure equal time for the promotion of healthy foods;<sup>43</sup> and using health warnings to identify energy-dense, nutrient-poor food choices, similar to the health warnings found on cigarette packaging.<sup>43</sup>

Policies to decrease social vulnerabilities also deserve consideration. Canadian data demonstrate inconsistent findings with respect to the risk of obesity in groups of low socioeconomic status. For adults, low education has been shown to be a strong determinant of obesity;<sup>47</sup> for children, living in a low-income household has been shown to play a major role.<sup>48</sup> More recent data from the 2004 Canadian Community Health Survey: Nutrition show that children and adolescents living in middle-income households have the highest rates of obesity, and children and adolescents living in households with the lowest education levels are most likely to be obese.<sup>49</sup> For adults, the association between lower education and higher obesity continues to hold, and higher income status and greater obesity are correlated, especially among men.<sup>50</sup> Despite inconsistencies in data, it is important that policies are implemented to protect socially vulnerable people from po-

tential systemic barriers to achieving healthy lifestyles. Health behaviours are embedded within environmental and social contexts that may be beyond individual control. For example, food insecurity may compromise the quality and quantity of food consumed and indirectly affect weight status. The cost of a low-fat basket of food was found to be higher than a nutritionally adequate but higher-fat basket of food.<sup>51</sup> Among individual foods, those that are the most energy dense are often those that are the least costly.<sup>52</sup> Food banks, on which the most disadvantaged families are dependent, typically offer foods of suboptimal nutritional quality.<sup>53</sup>

Approaches that target and assist individual people, the family and communities to engage in healthy lifestyle patterns through all stages of life are needed. Policies that support adequate income to improve health and encourage healthier choices should be considered.<sup>33</sup>

## Integration for a comprehensive approach to obesity prevention

Obesity is a multifactorial disease, and solutions require a multifactorial approach. Addressing obesity is not simply a problem of public health. There is a need for various sectors to work together to identify and introduce solutions. Policy interventions in the area of tobacco control may provide insights and shed light on potentially effective interventions in the area of obesity prevention. Strategies must be population-based as well as targeting the individual. Cooperation among the food industry, educators, health care providers, families and consumers is required. Governments have a central role in developing strategies, ensuring that actions are implemented and monitoring their impact over the long-term. Ministries must be brought together to develop, design and implement effective policy. Governments must work with the private sector, health professional bodies, consumer groups, academics, the research community and other nongovernmental bodies if sustained progress is to occur.

According to the Public Health Approaches to the Prevention of Obesity Working Group of the International Obesity Task Force,<sup>22</sup> a comprehensive approach to obesity prevention should:

- address both dietary habits and physical activity patterns of the population;
- address both societal and individual level factors;
- address both immediate and distant causes;
- have multiple focal points and levels of intervention (i.e., at national, regional, community and individual levels);
- include both policies and programs; and
- build links between sectors that may otherwise be viewed as independent.

To enhance success, healthy behaviours must be supported by public health measures, such as supportive environments, and effective policy changes that promote healthy weights and prevent obesity. The impact of obesity points to the importance of prevention through healthy behaviours, including increased physical activity and a healthy diet beginning early in life, and continuing through all stages of life.

## Recommendations

1. The use of surveillance systems and measurement tools is encouraged to determine the effectiveness and efficacy of obesity prevention programs and interventions. The development of a comprehensive, coordinated and rigorous surveillance plan with strong links among program developers, advocates, policy-makers and other stakeholders is encouraged as a key component in obesity prevention [grade C, level 4].
2. Obesity prevention should take a multisector approach — similar to that used for tobacco control in Canada. Prevention efforts should invest in and target all age groups and span life from infancy to old age. Innovative ways to provide access and programs to less economically viable citizens should be developed [grade C, level 4].
3. Funding of all types of research at all levels to address knowledge gaps and answer outstanding questions in the area of obesity is a high priority. Research is needed to develop, test and refine effective policies and interventions (best practices) in obesity prevention to enhance the evidence base for future public health interventions. Specific emphasis must be placed on translating research into policy, programs and practice [grade C, level 4].

From the Centre for Health Promotion Studies, University of Alberta (Raine); Canadian Public Health Association (Wilson)

**Competing interests:** Kim Raine declares no competing interests. Elinor Wilson has received travel assistance from Obesity Canada.

## REFERENCES

1. Obesity: Preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* 2000;894:1-253.
2. Birmingham CL, Muller JL, Palepu A, et al. The cost of obesity in Canada. *CMAJ* 1999;160:483-8.
3. Davison KK, Birch LL. Childhood overweight: a contextual model and recommendations for future research. *Obes Rev* 2001;2:159-71.
4. Blocker DE, Freudenberg N. Developing comprehensive approaches to prevention and control of obesity among low-income, urban, African-American women. *J Am Med Womens Assoc* 2001;56:59-64.
5. Breslow L. Social ecological strategies for promoting healthy lifestyles. *Am J Health Promot* 1996;10:253-7.
6. Egger G, Swinburn B. An "ecological" approach to the obesity pandemic. *BMJ* 1997;315:477-80.
7. Goetz DR, Caron W. A biopsychosocial model for youth obesity: consideration of an ecosystemic collaboration. *Int J Obes Relat Metab Disord* 1999;23(Suppl 2):S58-64.
8. Sallis JF, Bauman A, Pratt M. Environmental and policy interventions to promote physical activity. *Am J Prev Med* 1998;15:379-97.
9. McLeroy KR, Bibeau D, Steckler A, et al. An ecological perspective on health promotion programs. *Health Educ Q* 1988;15:351-77.
10. Swinburn B, Gill T, Kumanyika S. Obesity prevention: a proposed framework for translating evidence into action. *Obes Rev* 2005;6:23-33.
11. Bevilacqua N. *A public health approach to promoting healthy weights and preventing obesity: a review of the literature on effectiveness of prevention strategies*. Toronto: Toronto Public Health; 2 June 2000.
12. Gill TP. Key issues in the prevention of obesity. *Br Med Bull* 1997;53:359-88.
13. Economos CD, Brownson RC, DeAngelis MA, et al. What lessons have been learned from other attempts to guide social change? [discussion S57-65]. *Nutr Rev* 2001;59:S40-56.
14. Antipatis VJ, Kumanyika S, Jeffery RW, et al. Confidence of health professionals in public health approaches to obesity prevention. *Int J Obes Relat Metab Disord* 1999;23:1004-6.
15. Story M. School-based approaches for preventing and treating obesity. *Int J Obes Relat Metab Disord* 1999;23(Suppl 2):S43-51.
16. Veugelers PJ, Fitzgerald AL. Effectiveness of school programs in preventing childhood obesity: a multilevel comparison. *Am J Public Health* 2005;95:432-5.
17. *Canadian population initiative. Improving the health of Canadians: promoting healthy weights*. Ottawa: Canadian Institute for Health Information; 2006.

18. Schmitz MK, Jeffery RW. Public health interventions for the prevention and treatment of obesity. *Med Clin North Am* 2000;84:491-512, viii.
19. Spence JC. *Compilation of evidence of effective active living interventions: a case study approach*. Edmonton: Canadian Consortium of Health Promotion Research; 15 Apr 2001.
20. Worksite programs combining nutrition and physical activity are recommended to control overweight or obesity. Guide to Community Preventive Services Web site. Atlanta: US Centers for Disease Control and Prevention. Available: [www.thecommunityguide.org/obese/obese-int-worksite.pdf](http://www.thecommunityguide.org/obese/obese-int-worksite.pdf) (last updated 2005 Sept 7; accessed 2007 Feb 22).
21. Booth SL, Sallis JF, Ritenbaugh C, et al. Environmental and societal factors affect food choice and physical activity: rationale, influences, and leverage points [discussion S57-65]. *Nutr Rev* 2001;59:S21-39.
22. Kumanyika S, Jeffery RW, Morabia A, et al. Public Health Approaches to the Prevention of Obesity (PHAPO) Working Group of the International Obesity Task Force (IOTF). Obesity prevention: the case for action. *Int J Obes Relat Metab Disord* 2002;26:425-36.
23. Boutelle KN, Jeffery RW, Murray DM, et al. Using signs, artwork, and music to promote stair use in a public building. *Am J Public Health* 2001;91:2004-6.
24. Schmitz KH. Prevention of Obesity. In: Wadden TA, Stunkard AJ, editors. *Handbook of obesity treatment*. New York: Guildford Press; 2002:556-93.
25. French SA, Jeffery RW, Story M, et al. Pricing and promotion effects on low-fat vending snack purchases: the CHIPS Study. *Am J Public Health* 2001;91:112-7.
26. Horgen KB, Brownell KD. Comparison of price change and health message interventions in promoting healthy food choices. *Health Psychol* 2002;21:505-12.
27. Task Force on Community Preventive Services. Recommendations to increase physical activity in communities. *Am J Prev Med* 2002;22(4 Suppl):67-72.
28. Working Group on Physical Activity for Children and Youth. *Active Kids, Healthy Kids: a Nova Scotia physical activity strategy for children and youth*. Halifax: Nova Scotia Sport and Recreation Commission; 2002. Available: [www.gov.ns.ca/hpp/physicalactivity/publications/ACTIVEKIDS\\_Jano3.pdf](http://www.gov.ns.ca/hpp/physicalactivity/publications/ACTIVEKIDS_Jano3.pdf) (accessed 2007 Mar 29).
29. Booth M, Bauman A, Oldenburg B, et al. Effects of a national mass-media campaign on physical activity participation. *Health Promot Int* 1992;7:241-7.
30. Brownson RC, Housemann RA, Brown DR, et al. Promoting physical activity in rural communities: walking trail access, use, and effects. *Am J Prev Med* 2000;18:235-41.
31. Stockley L. Toward public health nutrition strategies in the European Union to implement food based dietary guidelines and to enhance healthier lifestyles. *Public Health Nutr* 2001;4(2A):307-24.
32. Nutrition labelling. Ottawa: Health Canada. Available: [www.hc-sc.gc.ca/fn-an/label-etiquet/nutrition/index\\_e.html](http://www.hc-sc.gc.ca/fn-an/label-etiquet/nutrition/index_e.html) (accessed 2007 Feb 22).
33. Raine KD. *Obesity and overweight in Canada: a population health perspective*. Ottawa: Canadian Institute for Health Information, Canadian Population Health Initiative; August 2004.
34. Frumkin H. Urban sprawl and public health. *Public Health Rep* 2002;117:201-17.
35. Jackson RJ, Kochitzky C. *Creating a healthy environment: the impact of the built environment on public health*. Atlanta: US Centers for Disease Control and Prevention; 2003.
36. Vanderslice E. *Changing policies to promote physical activity and prevent obesity*. Slide presentation at the Interface of Urban Design, Public Health and Physical Activity in Preventing Obesity Conference; Dec 6, 2001. Available: [depts.washington.edu/obesity/confdec2001/PromotingActivity.ppt](http://depts.washington.edu/obesity/confdec2001/PromotingActivity.ppt) (accessed 2007 Feb 2).
37. King AC, Castro C, Wilcox S, et al. Personal and environmental factors associated with physical inactivity among different racial-ethnic groups of U.S. middle-aged and older-aged women. *Health Psychol* 2000;19:354-64.
38. Frank LD, Engelke P. *How land use and transportation systems impact public health: a literature review on the relationship between physical activity and built form*. Atlanta: US Centers for Disease Control and Prevention; 2000. ACEs working paper 1.
39. Frank LD, Engelke PO. The built environment and human activity patterns: exploring the impacts of urban form on public health. *J Planning Literature* 2001;16:202-18.
40. *1998 National survey on active transportation: summary report*. Ottawa: Go For Green, Environics; 1998.
41. *GST/HST memoranda series*. Ottawa: Revenue Canada; May 1998. Cat no ME-04-03-9701-E.
42. Jacobson MF, Brownell KD. Small taxes on soft drinks and snack foods to promote health. *Am J Public Health* 2000;90:854-7.
43. Nestle M, Jacobson MF. Halting the obesity epidemic: a public health policy approach. *Public Health Rep* 2000;115:12-24.
44. Jeffery RW. Public health strategies for obesity treatment and prevention. *Am J Health Behav* 2001;25:252-9.
45. WHO/FAO release independent Expert Report on diet and chronic disease. [Web site of the World Health Organization]. Available: [www.who.int/mediacentre/news/releases/2003/pr20/en/](http://www.who.int/mediacentre/news/releases/2003/pr20/en/) (accessed 2007 Feb 22).
46. McGinnis JM, Gootman JA, Kraak VI, eds. *Food marketing to children and youth: threat or opportunity?* Washington, DC: National Academies Press; 2006.
47. Macdonald SM, Reeder BA, Chen Y, et al. Obesity in Canada: a descriptive analysis. Canadian Heart Health Surveys Research Group. *CMAJ* 1997;157(Suppl 1):S3-9.
48. *National longitudinal survey of children and youth: childhood obesity*. Ottawa: Statistics Canada; 18 Oct 2002.
49. Shields M. Measured obesity: overweight Canadian children and adolescents. In: *Nutrition: findings from the Canadian Community Health Survey*. Issue 1. Ottawa: Statistics Canada; 2005. Available: [www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf](http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/cobesity.pdf) (accessed 2007 Feb 22).
50. Tjepkema M. Measured obesity: adult obesity in Canada: measured height and weight. In: *Nutrition: findings from the Canadian Community Health Survey*. Issue 1. Ottawa: Statistics Canada; 2005. Available: [www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf](http://www.statcan.ca/english/research/82-620-MIE/2005001/pdf/aobesity.pdf) (accessed 2007 Feb 22).
51. Travers KD, Cogdon A, McDonald W, et al. Availability and cost of heart healthy dietary changes in Nova Scotia. *J Can Diet Assoc* 1997;58:176-83.
52. Drewnowski A, Specter SE. Poverty and obesity: the role of energy density and energy costs. *Am J Clin Nutr* 2004;79:6-16.
53. Travers KD. The social organization of nutritional inequities. *Soc Sci Med* 1996;43:543-53.

## 25. The agenda for obesity research in Canada

Diane T. Finegood

This important effort to establish Canadian clinical practice guidelines for the management and prevention of obesity highlights the many gaps in our knowledge in this area. Considerable new knowledge is required to optimize management and to prevent the growing prevalence of overweight and obesity in Canada.

The Obesity Canada Clinical Practice Guidelines Expert Panel and Steering Committee identified major gaps in knowledge and articulated them in the form of 14 recommendations for research (see executive summary, recommendations on research and policy [page S9]). They range from the need for enhanced surveillance and population-based data to new research on the biological, social, cultural and environmental determinants of obesity. They call for funding of all types of research, including research on effective treatment strategies, policies and interventions.

Although the research recommendations are far fewer in number than the recommendations for practice, they constitute a significant agenda. They articulate only the most obvious gaps in knowledge that prevented the experts from developing recommendations in specific areas. Examination of the practice guidelines suggests that our gaps in knowledge are really much greater. Among the individual practice recommendations, only a small percentage could be assigned the highest grade and be supported by the highest level of evidence. Many are based on much less evidence and are consensus recommendations where it is not known whether the benefits outweigh the risks. Significant new knowledge is required to fill these gaps.

### Obesity research priorities

In support of the research recommendations that arose out of the development of these clinical practice guidelines are other efforts, both in Canada and elsewhere, to identify priorities for obesity research. The results of these efforts have much in common, but their focus varies considerably from a detailed consideration of basic science questions to examination of the structural and procedural changes needed to bring about a more integrated system.

In August 2004, the National Institutes of Health (NIH) in the United States published its strategic plan for obesity research.<sup>1</sup> This plan groups the research needs under 4 scientific themes: preventing and treating obesity through behavioural and environmental approaches to modify lifestyle; preventing and treating obesity through pharmacologic, surgical or other medical approaches; breaking the link between

obesity and its associated health conditions; and cross-cutting topics, including health disparities, research resources, multidisciplinary teams and translational research. Specific research topics and short-, medium- and long-term goals under each of these themes address many of the gaps that surfaced during the process of developing these clinical practice guidelines. For example, the need for enhanced surveillance and to identify optimal intervention strategies at the individual and population levels is clearly recognized in both efforts.

In contrast, however, the NIH plan places significant emphasis on goals targeting basic mechanisms and determinants of overweight, obesity and weight loss. For example, specific goals include the use of model organisms (e.g., mice) to identify novel pathways or molecules involved in regulation of body composition, food consumption and physical activity levels; the identification of genes associated with increased or decreased risk of obesity in metabolically well-characterized populations of men and women from diverse racial and ethnic groups; and the identification of components of the central and peripheral nervous systems that are influenced by nutrition, physical activity, and social and environmental factors to affect weight and body composition at critical periods throughout life (e.g., the fetal period, neonatal period, adolescence, pregnancy, menopause and older adulthood).

It is not surprising that an NIH strategic plan would emphasize basic research given that supporting such research is a large part of its mandate. The recommendations arising out of our effort to develop clinical practice guidelines are less focused on knowledge gaps in the laboratory and more focused on gaps at the bedside. In addition, the breadth of the topics covered by these guidelines has led to the appearance of recommendations for new research in populations and at the level of policy.

In Canada, research recommendations relevant to the federal government's Healthy Living Strategy were developed by the Research and Surveillance Working Group of the Intersectoral Healthy Living Network Coordinating Committee (IHLN CC). This working group was struck in September 2004 to support "the development of an integrated research and surveillance agenda for the IHLN CC by building on existing efforts."<sup>2</sup> Priority areas for action were identified through a document review followed by a consensus conference. The review generated a matrix of recommendations<sup>3</sup> based on many efforts to articulate priorities, including the NIH plan, the work of Health Canada's Advisory Committee on Population Health and Health Security, the document *Overweight*



and Obesity in Canada: a Population Health Perspective<sup>4</sup> and many other published and unpublished efforts to identify priorities for action and research. This effort also identified the need to fill research gaps on the determinants of healthy eating and active living and on evaluation of interventions at the individual and population levels. The discussion extended to consideration of infrastructure gaps where federal leadership could play an important role. This working group identified the need for increased capacity to implement an integrated research, surveillance, policy and practice agenda; development of a system to identify, review and disseminate best practices; and development of common evaluation systems.<sup>2</sup>

Further work was done by the Chronic Disease Prevention Alliance of Canada, in collaboration with the Canadian Institutes of Health Research (CIHR) and others to identify the system changes needed to develop a fully integrated research and evaluation effort. The vision of the participants was a research system that would not only address gaps in basic knowledge, but also move knowledge into action and, ultimately, have a real impact on the health of the population. Through synthesis of previous work, a series of key informant interviews and a workshop discussion, this initiative identified several system-level priorities, including the need to fill gaps in basic knowledge about prevention, to understand the complex system within which prevention is realized, to find new approaches and methods and to shift from the paradigm of basic medical sciences, such as anatomy, physiology and molecular biology, to the “basic sciences” of marketing, political science and economics (Asselbergs M, Birdsell J: unpublished report, 2004). This work also identified the need for new funding structures and processes, a systems orientation, a long-term horizon and new capacity in the form of data infrastructure, qualified personnel and systems for knowledge transfer and utilization.

These various efforts to identify research priorities came out of different processes, and each was motivated by different objectives: developing a strategic plan for a research funding agency, identifying the gaps that make construction of practice guidelines difficult, considering the goals of a federal government healthy living strategy. The work reflects input from a wide range of stakeholders, including researchers, health practitioners, policy-makers at multiple levels of government and representatives of nongovernmental organizations. All of these exercises pointed to a need for more population intervention research to enhance the pool of “practice-based” evidence. But to achieve this goal, many other gaps will also need to be filled. Tools to support evaluation, enhanced surveillance, including policy, and integration of systems of knowledge creation, transfer and exchange are some of what is needed to support the creation of new knowledge about what works, for whom, when and under what circumstances. Basic science was also seen as essential to understanding “why” and to fuel innovation to find new solutions.

The combined research recommendations from these various efforts span a wide range of knowledge gaps from genetics to systems to policy. The specific recommendations are too numerous to list, but simply put, they suggest the need for:

- increased support for all types of obesity research, from basic science in order to enhance our understanding of the de-

terminants of obesity, weight loss and weight maintenance to research on the effectiveness of policy interventions; and

- increased capacity development, in the form of people, surveillance systems, data infrastructure and other systems for knowledge creation, transfer and exchange.

## Recent progress in Canada

In June 2000, the federal government established the CIHR as the main federal agency responsible for funding health research in Canada. CIHR consists of 13 “virtual” institutes, each headed by a scientific director and assisted by an institute advisory board and a small staff. The mandate of the Institute of Nutrition, Metabolism and Diabetes (INMD) is to support research to enhance health in relation to diet, digestion, excretion and metabolism; and to address causes, prevention, screening, diagnosis, treatment, support systems and palliation for a wide range of conditions and problems associated with hormone, digestive system, kidney and liver function. In 2001, the INMD undertook an extensive scan of researchers and stakeholders, including policy-makers and nongovernmental organizations, to identify priorities within its mandate.<sup>5</sup> In this scan, obesity emerged as a top priority, both among those who worked in the area and among those who did not. As a result of the scan and other factors considered by the institute’s advisory board, the INMD released its first strategic plan wholly focused on obesity in May 2002.<sup>6</sup> The plan identified 3 broad goals:

- Increase understanding of the measures, causes, prevention, treatment and consequences of obesity and maintenance of healthy body weights through the application of innovative approaches.
- Increase understanding of obesity and healthy body weights in Canada through surveillance of prevalence, risk factors (including environment and policy), comorbidities, program and service utilization and efficacy, and direct and indirect costs.
- Identify and promote utilization of health service and environment policies for preventing obesity and promoting healthy eating, active living and other factors affecting healthy body weights.

Initiatives aimed at achieving these goals have been developed by the INMD in collaboration with many partners, including the Heart and Stroke Foundation of Canada and the Canadian Diabetes Association. Repeated requests for applications have been issued for new obesity research. Specific opportunities have generally been open to any type of obesity research across the spectrum of themes (basic science to population health research). Two competitions were specifically focused on childhood obesity. Researchers have been invited to apply for grants to establish teams, training programs, full-scale projects, pilot projects, and planning and development programs.

During the 1990s, the Medical Research Council of Canada (MRC) was spending less than \$1 million a year on obesity research. By 2000/01, CIHR’s first year of operation, funding had increased to \$3.4 million. With the INMD’s strategic focus on obesity, total CIHR funding grew to nearly \$20 million a year in 2005/06, a 6-fold increase (Fig. 9). From 2000 to

2006, 442 grants and awards related to obesity received a total of \$63 million in support from CIHR. Of these, 70% were projects based in Ontario and Quebec (Fig. 10), but for many projects, collaborating researchers come from multiple institutions and locations. In this period, research specifically addressing childhood obesity received \$10 million, with the bulk of this investment made over the past couple of years.

Sokar-Todd and Sharma<sup>7</sup> found that basic science was the dominant form of obesity research between 1970 and 2003 in Canada. Basic science involving animal (29%) or human experiments (16%) accounted for nearly half of all Canadian obesity papers published during that timeframe. The group originally led by Claude Bouchard at Laval University made significant contributions to Canada's strengths in basic and clinical research. Laval University ranked 11th among institutions in total obesity-related citations from 1991 to 2000<sup>8</sup> and contributed to Canada ranking 4th among nations.<sup>9</sup> Consistent with these indicators, nearly 60% of CIHR-funded obesity researchers who identified a research theme for their project identified it as a basic science project; the remaining investigators picked clinical research, and only 1% picked population and public health. These data must be considered in light of the fact that, in 2000/01, many principle investigators did not identify a theme for their projects; this classification system was introduced during the transition from the MRC to CIHR. In 2000/01, 42% of obesity-related research funds were allocated to projects that had not been categorized; by 2005/06, this had dropped to 3% of allocated dollars.

Although basic science was the predominant form of obesity research in the early days of CIHR, support through obesity-focused competitions has helped to increase research in population health and has created some interest among the small community of health services and policy researchers. The initiative is also attracting the attention of isolated researchers in some of the suggested new "basic sciences" of

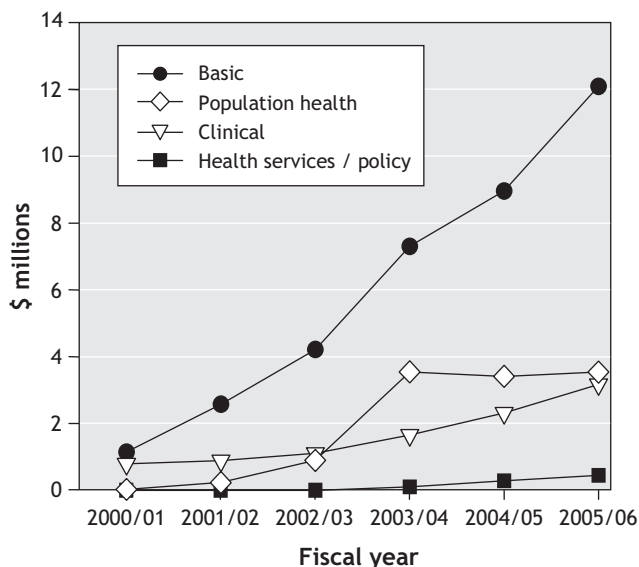


Fig. 9: Funding from the Canadian Institutes of Health Research for obesity-related research, by research theme.

business, economics, urban planning and the social sciences, but more needs to be done to build capacity and break down barriers to the engagement of researchers with a wide range of expertise from transportation to policy to clinical treatment to community-based intervention.

In addition to supporting individual and team-based research projects, the INMD and the other CIHR institutes provide support and originate projects and initiatives aimed at development of the research community. Although some of INMD's development funds go to other areas in INMD's mandate, support has been given to many meetings, projects and initiatives relevant to the focus on obesity. Projects such as Canada on the Move<sup>10</sup> have contributed to innovation in the collection of evidence based on "natural experiments," and a recent team-based research meeting facilitated networking and sharing of best practices among the leaders of obesity and chronic disease research teams.

By creating a strategic focus on obesity research in Canada, the INMD has helped to increase the number of researchers interested in tackling obesity-related questions. The number of people supported through CIHR grants and awards grew from 60 in 2000/01 to more than 400 in 2005/06, a 7-fold increase in the total number of researchers, collaborators and salary awarded trainees. Another indicator of the increased interest in obesity research is the rapidly expanding membership in the Canadian Obesity Network (CON, www.obesitynetwork.ca). Funding for CON, a Network Centre of Excellence, was announced in March 2006, and by August it had 1000 registered members. CON will help to enhance networking between researchers in different disciplines and between researchers, practitioners and policy-makers.

## The future of obesity research in Canada

Although the creation of a focus for obesity research in Canada has helped to increase the cadre of researchers interested in tackling the many relevant questions, it seems clear that these researchers can manage to address only the tip of the proverbial iceberg. Canadian researchers are providing important new information in many areas such as indices of

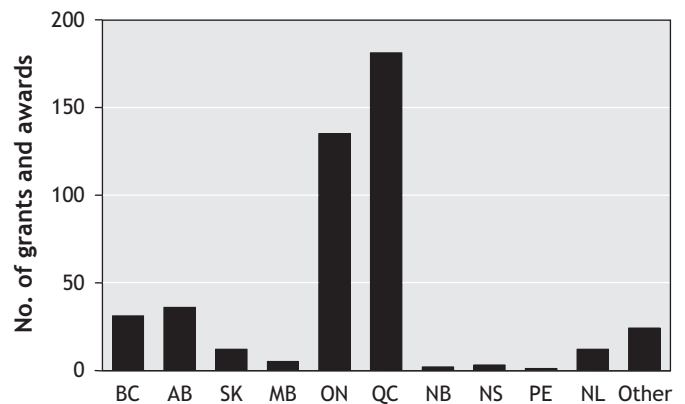


Fig. 10: Number of grants and awards from the Canadian Institutes of Health Research, by province of the principal investigator.

cardiovascular risk,<sup>11</sup> the independent roles of exercise and weight loss in reducing risk<sup>12</sup> and the potential for school-based nutrition programs to prevent childhood obesity.<sup>13</sup> In the future, recently funded work will provide new knowledge on the genetic determinants of obesity, the best design for an integrated, interdisciplinary obesity care management system, and the treatment preferences of overweight children and their parents. Although many other projects are currently underway, many more issues remain to be addressed.

Given that so many gaps remain, the institute advisory board has advised the INMD to remain focused on obesity through 2008. With added support from other agencies, including the Heart and Stroke Foundation of Canada, the Network Centres of Excellence, the Public Health Agency of Canada and the Canadian Population Health Initiative, there is room for optimism that, in a future revision of these guidelines on the management and prevention of obesity, greater certainty will be accorded many of the recommendations.

From the Institute of Nutrition, Metabolism and Diabetes, Canadian Institutes of Health Research, and Simon Fraser University, Burnaby, BC

**Competing interests:** None declared.

## REFERENCES

1. NIH Obesity Research Task Force. *Strategic plan for NIH obesity research.*

- Bethesda (MD): National Institutes of Health; 2004. Available: obesityresearch.nih.gov/About/Obesity\_EntireDocument.pdf (accessed 2007 Feb 18).
2. Connelly CR. *Priorities for research, surveillance, and best practices for healthy eating and physical activity and their relationship to healthy body weight.* Ottawa: Intersectoral Healthy Living Network Coordinating Committee, Public Health Agency of Canada; January 2005.
3. Connelly CR. *A matrix of gaps and recommendations associated with research and evaluation, surveillance and monitoring, best practices, capacity and organizational structures.* Ottawa: Intersectoral Healthy Living Network Coordinating Committee, Public Health Agency of Canada; February 2005.
4. Raine KD. *Obesity and overweight in Canada: a population health perspective.* Ottawa: Canadian Institute for Health Information, Canadian Population Health Initiative; August 2004.
5. Institute of Nutrition, Metabolism and Diabetes (INMD). *Charting our course: a national consultation on strategic directions* [summary report]. Burnaby (BC): INMD; Nov 2001. Available: www.cihr-irsc.gc.ca/e/pdf\_24011.htm (accessed 2007 Feb 18).
6. Institute of Nutrition, Metabolism and Diabetes (INMD). *Strategic plan.* Burnaby (BC): INMD; May 2002. Available: www.cihr-irsc.gc.ca/e/pdf\_24009.htm (accessed 2007 Feb 18).
7. Sokar-Todd HB, Sharma AM. Obesity research in Canada: literature overview of the last 3 decades. *Obes Res* 2004;12:1547-53.
8. Essential Science Indicators Special Topics. Obesity: institutions: top 25 overall; 2001. Available: www.esi-topics.com/obesity/inst/cra.html (accessed 2007 Feb 18).
9. Essential Science Indicators Special Topics. Obesity: top nations: top 25 overall; 2001. Available: www.esi-topics.com/obesity/nations/dra.html (accessed 2007 Feb 18).
10. Dietz WH. Canada on the move: a novel effort to increase physical activity among Canadians. *Can J Public Health* 2006;97(Suppl 1):S3-4.
11. Yusuf S, Hawken S, Ounpuu S, et al. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case-control study. *Lancet* 2005;366:1640-9.
12. Lee S, Kuk JL, Davidson LE, et al. Exercise without weight loss is an effective strategy for obesity reduction in obese individuals with and without type 2 diabetes. *J Appl Physiol* 2005;99:1220-5.
13. Veugelers PJ, Fitzgerald AL. Effectiveness of school programs in preventing childhood obesity: a multilevel comparison. *Am J Public Health* 2005;95:432-5.

## 26. Dissemination and implementation of the guidelines

Denis Drouin

**T**ranslating evidence from clinical trials into clinical practice is an important issue, and the literature is sparse in this area. We must rely on reports describing ongoing initiatives, such as that of the Canadian Hypertension Education Program.<sup>1</sup> Grimshaw and Russell<sup>2</sup> noted that 55 of 59 published assessments of the implementation of clinical practice guidelines reported statistically significant improvements in the process of care and changes in health professional performance. However, there are examples of studies in which the results were varied. Most authors concluded that there is a need for more research on the topic.<sup>3</sup> In Canada, medical and health organizations are aware of their responsibilities in this regard; for example, the Canadian Institutes of Health Research (CIHR) is putting more emphasis on the need to link scientific research to social and health outcomes.

Implementation may be defined as the passage from science into action or the practical result of a transfer of knowledge into clinical practice. Implementation happens when there is adoption and application of scientific recommendations related to a specific outcome.

Implementation is part of a continuum, which includes dissemination through publications and the development of clear messages, educational materials and practical tools. All of these facilitate decision-making in clinical practice. Implementation is successful when the target audience will benefit from the application of evidence-based recommendations. Dissemination can be orchestrated by a central organization, but implementation must be carried out locally by individuals or organizations.

### The transfer of information into clinical practice

Physician, patient and community acceptance of the scientific evidence is a key component of the knowledge-transfer process. For example, in the case of these clinical practice guidelines on the prevention and treatment of obesity, the transfer of information into clinical practice should focus on establishing weight reduction and maintenance as an important secondary prevention strategy for diabetes and cardiovascular disease. Such a message could get attention and respect from clinicians and motivate them to be involved in the adoption and implementation of the guidelines.

Passive dissemination has not had an effect on clinical practice in the short term.<sup>4</sup> Moreover, the publication of guidelines as a stand-alone exercise produces little change in

clinical practice and no change in health outcomes.<sup>5</sup> However, the literature does suggest that a multifaceted set of interventions may affect practice and help in the implementation of evidence-based recommendations. More research is warranted to understand the mechanisms of implementation.<sup>3</sup>

Canadian family physicians say that they prefer to receive new clinical information through discussions with colleagues or consultants<sup>6</sup> and by reading review articles in medical journals and textbooks. They find several formats useful, including short pamphlets summarizing guidelines, pocket cards, flow charts and clinical algorithms. Factors that encourage the adoption of clinical practice guidelines are endorsement by respected colleagues and physicians' own specialty organizations, the user-friendliness of the format in which guidelines are presented and consistency with predominant local practices. Although it has not been demonstrated that physicians actually use guidelines presented to them according to these preferences, it is logical to provide the information in a format that we know will be well received and accepted.

A study to determine whether guidelines increased the use of recommended drugs, such as  $\beta$ -blockers, for the secondary prevention of coronary artery disease showed that the development of a network of local key opinion leaders is an important component of a successful dissemination and implementation strategy.<sup>7</sup>

### Barriers

Barriers to implementation are multiple and can be encountered at different levels. For instance, lack of time, prescription costs and patient nonadherence have been observed as key barriers.<sup>8</sup> Methods for improving implementation should be centred on more education for physicians and patients; the promotion, publication and increased availability of guidelines; simplification of guidelines; and improved clarity.

Most primary care physicians do not treat obesity.<sup>9</sup> They cite lack of time, resources and insurance reimbursement, and lack of knowledge of effective interventions as significant barriers.

In addition, lack of reimbursement programs for patients wishing to seek professional services from a trained dietitian is a major barrier limiting access to an important clinical intervention that has proved to be effective.<sup>10</sup>

Other barriers may be encountered in the patient's environment, and these should be addressed on an individual basis. For example, in an intervention to reduce obesity in schools,<sup>11</sup> the school administration and lack of family partic-

ipation were perceived as barriers at some schools. The problem was not dissemination of information, but rather local barriers that should be addressed individually (e.g., meeting with the director or parents' organization, or a contract with providers of food or drinks in vending machines).

## Outcomes-based approach

Implementation is an ambitious task; however, it can be lim-

ited to specific goals. Table 20 lists various goals of an implementation program and suggests some ways to achieve them.

## Global implementation plan

A plan for the global dissemination and implementation of guidelines on obesity should be multifaceted. The plan should involve a sequence of events, such as publication in peer-reviewed and non-peer-reviewed journals.<sup>12</sup> An abridged version should also be developed and tailored to target audiences (most likely physicians, registered dietitians and nurses). Simple, consistent messages should be developed and repeated over and over in every publication. Slide sets for medical educators and the general public, as well as 1-page summaries, should be made available and should be freely downloadable from a Web site. Sister scientific organizations should be involved in dissemination and implementation. They should be asked to inform their members about the new guidelines on obesity management.

Because education is part of the medical tradition, small-group, problem-based workshops should be developed and held around the country. The support of universities, the College of Family Physicians of Canada and various pharmaceutical sponsors should be sought. These groups could also be asked to facilitate faculty development or "train-the-trainers" sessions. To ensure continual quality improvement, a committee could be created to measure outcomes and monitor the effectiveness of the implementation program.

Key elements in an integrated approach to implementation are a task-oriented management program (task, who, when) and a timeline. A simple checklist for developing an implementation plan<sup>13</sup> is provided in Box 7 as a guide to initiate an implementation process. Coordination is important. This should be done by a steering committee and various working groups.

## Systems-based implementation

There is compelling evidence that, when a guideline can be implemented as a protocol in an organization (e.g., a hospital), it may influence and opti-

**Table 20:** An outcomes-based approach to the implementation of clinical practice guidelines

Area of change	Outcome	Interventions to achieve outcome
Attitude	Changes in awareness toward new understanding of the science	<ul style="list-style-type: none"> <li>• Publication in peer-reviewed journal</li> <li>• Endorsement by respected scientific societies and key opinion leaders</li> <li>• Lectures</li> </ul>
Knowledge	Exposure to new information, new concepts	<ul style="list-style-type: none"> <li>• Publication in peer-reviewed journal and in many scientific journals</li> <li>• Endorsement by respected scientific societies and key opinion leaders</li> <li>• Lectures</li> <li>• Information available on Internet, cited in textbooks</li> </ul>
Skills, behaviour	Development of skills in using a new tool (e.g., a new classification, drug prescription)	<ul style="list-style-type: none"> <li>• Production of clear messages</li> <li>• Provision of simplified summaries</li> <li>• Development of tools, charts and treatment algorithms, as well as personal digital assistant (PDA) applications</li> <li>• Provision of education through interactive sessions and all of the preceding</li> </ul>
Practice, bedside behaviour	Use of the recommendations at the clinical level	<ul style="list-style-type: none"> <li>• All of the preceding</li> <li>• Development of computer-aided decision support systems, prompts and the use of reminders, tracking systems and goal-oriented management</li> </ul>
Organization and system	Adoption and development of a protocol to be used in an organization (e.g., hospital)	<ul style="list-style-type: none"> <li>• Adoption of a protocol</li> <li>• Inclusion and integration of protocol into management software and tracking systems</li> <li>• Encouragement of audits and soliciting of feedback based on group performance</li> <li>• Development of information technology</li> </ul>
Patient	Patient adoption and adherence to the recommendations	<ul style="list-style-type: none"> <li>• Adoption and adherence to the recommendations and treatment</li> <li>• Involvement of family and caregivers</li> <li>• Collaboration of resources at the workplace</li> <li>• Use of tracking systems, prompts</li> <li>• Maintenance of a communication link</li> <li>• Use of computerized reminders</li> <li>• Interdisciplinary intervention involving communication links among professionals</li> </ul>

#### Box 7: Developing an implementation plan

- When should we start implementation?
- How should we start implementation?
- Who will our initial target population be? (Start small!)
- With what services and materials should we start? Which ones should we add later?
- How will we know when we are ready to expand our services?

mize case management. For instance, the Cardiac Hospitalization Atherosclerosis Management Program (CHAMP) study<sup>14</sup> evaluated the implementation of a post-myocardial infarction (MI) management protocol. The authors found that, when the post-MI protocol was used, the rate of death or recurrent MI after 1 year was reduced from 14.6% to 6.4%. Implementation would be facilitated if a clinical protocol on the management of obesity could be developed and adopted by hospitals, specialty clinics, family practice groups and existing obesity clinics.

## Conclusions

Obesity remains a leading condition that is linked to morbidity and mortality worldwide. Goals for weight levels are seldom achieved. It is therefore highly desirable to improve treatment and prevention of this condition. This will require the participation of all professionals involved in health care, from government levels to the individual physician, allied health care professionals, patients and their families.

## Recommendations

1. Dissemination of the guidelines can be orchestrated by a central organization, but implementation should be carried out locally by individuals or local organizations [*grade C, level 4*].
2. The transfer of information into clinical practice should focus on establishing weight reduction and weight control as an important secondary prevention strategy for diabetes and cardiovascular disease [*grade C, level 4*].
3. More research is needed to improve understanding of the mechanisms of clinical practice guidelines implementation [*grade C, level 4*].
4. The guidelines should be disseminated in a simple, clear format that will be well received and accepted [*grade C, level 4*].

5. A network of local key opinion leaders should be developed as an important component of a successful dissemination and implementation strategy [*grade C, level 4*].
6. A multifaceted global dissemination and implementation plan should involve a sequence of events, including publication in peer-reviewed and non-peer-reviewed journals [*grade C, level 4*].
7. To ensure continual quality improvement, a committee should be created to measure outcomes, then monitor the effectiveness of the implementation program [*grade C, level 4*].

From the Faculty of Medicine, Laval University, Québec, Que.

**Competing interests:** Denis Drouin is a consultant to Public Health Quebec City, the Laval University CPD Office, the Heart and Stroke Foundation of Quebec, the Heart and Stroke Foundation of Ontario, the Canadian Medical Association and the College of Physicians of Quebec. He is a paid lecturer, member of advisory board or consultant in CME to Altana Pharma, AstraZeneca Canada Inc., Biovail Pharmaceuticals Canada, Bristol-Myers Squibb Canada, Boehringer Ingelheim, GlaxoSmithKline Inc., Merck Frosst Canada Inc., Novartis, Pfizer Canada Inc., sanofi-aventis Canada Inc., Servier Canada Inc., Solvay Pharma Inc. and Unilever Canada Inc. He has received travel assistance from Bristol-Myers Squibb Canada, Novartis, Pfizer Canada Inc. and Biovail Pharmaceuticals Canada.

## REFERENCES

1. Drouin D, Campbell NR, Kaczorowski J. Implementation of recommendations on hypertension: the Canadian Hypertension Education Program. *Can J Cardiol* 2006; 22:595-8.
2. Grimshaw JM, Russell IT. Effect of clinical guidelines on medical practice: a systematic review of rigorous evaluations. *Lancet* 1993;342:1317-22.
3. Gross PA, Greenfield S, Cretin S, et al. Optimal methods for guideline implementation: conclusions from Leeds Castle meeting. *Med Care* 2001;39(Suppl 2):I185-92.
4. Freemantle N, Harvey EL, Wolf F, et al. Printed educational materials: effects on professional practice and health care outcomes. *Cochrane Database Syst Rev* 2000;(2):CD000172.
5. Chalmers J. Implementation of guidelines for management of hypertension. *Clin Exp Hypertens* 1999;21:647-57.
6. Hayward RSA, Guyatt GH, Moore KA, et al. Canadian physicians' attitudes about and preferences regarding clinical practice guidelines. *CMAJ* 1997;156:1715-23.
7. Sarasin FP, Maschiangelo ML, Schaller MD, et al. Successful implementation of guidelines for encouraging the use of beta blockers in patients after acute myocardial infarction. *Am J Med* 1999;106:499-505.
8. Hobbs FD, Erhardt L. Acceptance of guideline recommendations and perceived implementation of coronary heart disease prevention among primary care physicians in five European countries: the Reassessing European Attitudes about Cardiovascular Treatment (REACT) survey. *Fam Pract* 2002;19:596-604.
9. Bowerman S, Bellman M, Saltsman P, et al. Implementation of a primary care physician network obesity management program. *Obes Res* 2001;9:321-5S.
10. Connor H, Annan F, Bunn E, et al. The implementation of nutritional advice for people with diabetes. *Diabet Med* 2003;20:786-807.
11. Gittelsohn J, Merkle S, Story M, et al. School climate and implementation of the Pathways study. *Prev Med* 2003;37:S97-106.
12. Campbell NR, Nagpal S, Drouin D. Implementing hypertension recommendations. *Can J Cardiol* 2001;17:851-6.
13. *Put prevention into practice. A step-by-step guide to delivering clinical preventative services: a systems approach*. Rockville (MD): Agency for Healthcare Research and Quality; 2001. Publ no AAPP101-0001.
14. Fonarow GC, Gawlinski A, Moughrabi S, et al. Improved treatment of coronary heart disease by implementation of a Cardiac Hospitalization Atherosclerosis Management Program (CHAMP). *Am J Cardiol* 2001;87:819-22.