Call for action: preventing and managing the expansive and expensive obesity epidemic

David C.W. Lau, MD, PhD

See related articles pages 483 and 513

The prevalence of obesity is increasing worldwide and is reaching epidemic proportions in industrialized countries.[1,2] According to the large population-based Canadian Heart Health Surveys, close to half of adult Canadians are overweight and 1 in 6 is obese.[3] US statistics suggest an even greater prevalence rate in that country, with a steady upward trend over the last 2 decades.[1,4] What is even more alarming is the increasing prevalence of childhood obesity in many countries, including Canada.[5]

The recognition that obesity represents a rapidly growing health threat worldwide led to the formation of the International Obesity Task Force, whose mandate was to develop a public health policy and program for improving the prevention and management of obesity. The comprehensive report by the task force was released in the fall of 1998[5] and has been endorsed by the World Health Organization (WHO). The WHO and the International Association for the Study of Obesity are working with the national obesity associations in various countries to help implement the report and, more important, to persuade health policy-makers and key health professional organizations to recognize obesity as a disease that requires urgent action.

Developing strategies for the prevention and management of obesity requires a systematic approach that includes the following steps: defining the problem of obesity, establishing associated health risks, determining the benefits and risks associated with weight reduction, estimating the economic cost of obesity and outlining the principles of prevention and management of obesity in at-risk individuals and populations. The growing problem of obesity and the increased health risks attributable to obesity are well established[6–8] and were reaffirmed in the Canadian Heart Health Surveys.[3] In the group surveyed who were 18 to 54 years old, for example, the prevalence rates of dyslipidemia, hypertension and self-reported diabetes were 2- to 10-fold higher among people with a body mass index (BMI) of 30 or greater than among those with the lowest BMI.[9]

In this issue, 2 articles address the other relevant steps toward developing prevention and management strategies. In the first article Dr. C. Laird Birmingham and associates provide an estimate of the direct cost of obesity in Canada (page 483).[10] In the other article Dr. James D. Douketis and colleagues, with the Canadian Task Force on Preventive Health Care, examine the use of the BMI as a measurement of obesity and make recommendations on the prevention and treatment of obesity (page 513).[11] Their review of the evidence is important because the data concerning the health benefits and risks of weight reduction are less robust and require closer scrutiny. Both papers underscore the need to develop sound and rational strategies for implementing a systematic approach to tackle the growing problem of obesity in Canada.

In addition to the disease burden of obesity on affected individuals and their families, the economic costs of obesity are high, ranging from 2%–10% of the total health care budgets of different countries.[12–14] For the first time we have a realistic estimate of the direct cost of obesity in Canada: about $2 billion per annum, or 2.4% of the total health care budget.[10] By using the prevalent-based approach in their cost-of-illness study, Birmingham and associates may have underestimated the age- and sex-specific incidence of disease attributable to a chronic disease like obesity. Moreover, they did not include all of the diseases attributable to obesity in their analysis, which may further underestimate the real cost. Nevertheless, their estimated direct cost of obesity in Canada is similar to figures obtained for New Zealand, Australia and France but somewhat lower than those for Germany, the United States and Sweden.[14] The high cost should provide incentives to all physicians and health care professionals, not just obesity specialists and researchers, to prevent and manage this prevalent disease. It follows from this paper that there is a call for more data on the quality of life and the treatment benefits by means of cost-benefit and cost-utility analyses.

Obesity is characterized by excessive body fat accumulation in adipose tissue to an extent that health may be

adversely affected. Defining obesity without standardized methodology has been a challenge. A standardized measure of body fat and its distribution and the classification of obesity will allow for more accurate estimates of prevalence rates of obesity in the population, the identification of individuals or groups at increased health risks, the identification of priorities for intervention at the individual and community levels and, more important, the evaluation of intervention and treatment strategies.

The BMI is a simple index of weight to height that provides the best anthropometric measure of body adiposity. Canada adopted the use of the BMI for guidelines for healthy weights in 1988 and was one of the first countries to do so.[15] A BMI of 20–25 is considered a good value for most people, whereas a BMI above 25 is associated with an increasing risk of health problems.[15] The WHO consultation report currently defines overweight as a BMI of 25 or greater and obesity as a BMI of 30 or greater. Obesity can be further classified as moderate (class I, BMI 30–34.9), severe (class II, BMI 35–39.9) or very severe (class III, BMI 40 or greater).[5]

Because the BMI does not take into account the distribution of fat and the associated health consequences that vary considerably among individuals, additional markers are needed to complement BMI in assessing health risks. Indeed, recent studies suggest that obesity-related comorbidities are better correlated with the presence of upper-body or abdominal fat distribution and are independent of the degree of excess fat.[6,8,16–19]

Douketis and colleagues conclude from their extensive literature search that there is insufficient evidence to recommend the inclusion or exclusion of BMI measurement for the general population as part of the periodic health examination; instead they recommend that BMI measurement be left to the discretion of individual health care providers. The authors, however, do recommend BMI measurement in people with obesity-related comorbidities. The importance of fat distribution in assessing health risks was acknowledged, but no recommendations were made because of lack of available standardized indices.

With respect to the prevention and treatment of obesity, Douketis and colleagues believe that this should be a priority for health care providers. They recommend weight reduction for obese adults with obesity-related comorbidities such as diabetes mellitus and hypertension, but they could not recommend for or against treatment of obesity in adults without obesity-related comorbidities because of a lack of evidence supporting the long-term effectiveness of weight-reduction methods.

The recommendations by Douketis and colleagues are consistent with those of a much more comprehensive evidence-based report recently published by the National Institutes of Health,[20] with 3 notable exceptions. First, the US expert panel recommended that BMI be measured in adults to classify overweight and obesity and to estimate relative risk of disease compared with normal weight. This makes sense because health care professionals are notoriously poor in estimating overweight and obesity by visual inspection alone. Furthermore, the BMI can easily be deduced using normograms or tables from height and weight measurements, both of which are part of the periodic health examination. Second, the US panel recommended that waist circumference be included to assess abdominal fat content given the compelling data correlating abdominal fat to obesity-related comorbidities.[6,16,17,19] People could be classified as high risk with the use of waist-circumference cutoffs of more than 40 inches (102 cm) and more than 35 inches (88 cm) for men and women respectively. Finally, the US panel recommended treatment for overweight patients if they have 2 or more obesity-related comorbidities or risk factors.[20]

Obesity is a complex chronic disease with diverse multifactorial etiology. Minimalists attribute the rising prevalence of obesity largely to physical inactivity and ready access to highly palatable foods with a high fat content. Current research aims to uncover how and why obesity develops, the comprehensive understanding on which successful treatment rests. The intensive search for the genes responsible for obesity has led to a steady increase in the number of putative genes or loci linked to, or associated with, obesity. The number is currently approaching 200, with loci mapped in all but the Y chromosomes.[21] However, single genetic mutations are rare causes of human obesity. To date, there are only 25 reported cases of obesity resulting from 11 single mutations in 7 genes,[22–29] an increase from 3 cases reported in 1997.[30] Hence, emerging data suggest that multiple genes are more likely involved in human obesity, in contrast to the widely studied rodent models of obesity, where single gene mutations are the rule.[31]

Because genetic heritability accounts for only 25%–30% of the obesity phenotype,[32] prevention and treatment should logically be directed mainly at environmental factors (social, behavioural and cultural) as principal determinants. Modification of behaviours and lifestyle habits remains the cornerstone treatment of obesity. Earlier data suggest that the effectiveness of weight-reduction methods is rather limited. This is true if the primary outcome is body weight rather than improved health status. The overall goal for obesity management is to reduce the health risks, which begin to lessen even with modest weight loss, long before a desirable body weight is achieved. Recent evidence indicates that even a modest loss can alleviate symptoms of obesity-related comorbidities.[33,34]

Obtaining a desirable body weight often proves difficult and elusive. The initial goal is to reduce body weight

by approximately 10% from baseline over 6 to 12 months. In terms of strategies for weight loss and weight maintenance, the most successful treatment to date includes a controlled diet with a deficit of 500–1000 kcal (2100–4200 kJ) per day, with reduced dietary fat intake of less than 30% of total energy intake, regular physical activity programs and reinforcement of behavioural modification techniques. The extensive review by Douketis and colleagues[11] on the effectiveness of obesity treatment ignored the benefits of regular physical activity when incorporated as an integral component of a weight-reduction program. When coupled with a judicious controlled diet, exercise accelerates fat loss while maintaining lean body mass, and it helps to sustain weight loss over the long term.[35,36] Indeed, healthy lifestyle measures incorporating both diet and regular physical activity have been shown to significantly delay the onset or prevent the development of type 2 diabetes in obese and nonobese subjects.[37,38]

Pharmacotherapy can be useful as an adjunct to controlled diet and physical activity for people with a BMI of 30 or greater with no concomitant risk factors and for those with a BMI of 27 or greater with concomitant risk factors or diseases.[39] Two drugs with novel modes of action and known efficacy in long-term studies will soon be available in Canada: sibutramine, an appetite suppressant that specifically inhibits norepinephrine and serotonin reuptake,[40] and orlistat, which is a pancreatic lipase inhibitor that reduces fat absorption.[41] Sibutramine, unlike dexfenfluramine or fenfluramine, is not associated with primary pulmonary hypertension or heart valve abnormalities.[42] The availability of these drugs will be a welcome addition to the armamentarium in long-term obesity management.

Other obesity treatment options include bariatric surgery, which is usually reserved for people with class 3 obesity (BMI of 40 or greater). The large prospective randomized Swedish Obesity Study, still under way, has provided encouraging interim data to suggest that weight reduction following bariatric surgery reduces the 2-year incidence of diabetes, hypertension and other health risks by 3- to 32-fold.[43]

Because of the limited success of current obesity treatments, the challenges as we approach the new millennium will be to understand how obesity develops and to design strategies targeting the undesirable societal, cultural and other environmental influences. Different levels of preventive action can be undertaken: universal or public health prevention directed at the entire population, selective prevention directed at groups at risk, and targeted prevention directed at individuals at high risk and those who are overweight but not obese.

Enhancing the knowledge and attitudes of health care professionals about obesity is equally important. The acceptance of obesity as a medical disease with increased morbidity and mortality should supplant the previous prevailing perception of obesity as a cosmetic concern. Promoting healthy eating habits and increased physical activity are clearly priorities that should be established with active participation from governments, the private business sector, the media and, above all, consumers. This will provide an excellent opportunity for synergism of government public health policies directed at improving health. There is a call for action to form an obesity network in Canada to develop and implement effective programs and policies in the prevention and treatment of the growing epidemic of obesity in this country.

Dr. Lau is Professor of Medicine and of Biochemistry, Microbiology and Immunology and is Head of the Division of Endocrinology and Metabolism, University of Ottawa and The Ottawa Hospital.

Competing interests: None declared.

References

1. VanItallie TB. Prevalence of obesity. *Endocrinol Metab Clin North Am* 1996;25:887-905.
2. Keil U, Kuulasmaa K. WHO MONICA Project: risk factors. *Int J Epidemiol* 1989;18(Suppl 1):S46-S55. [Erratum appears in *Int J Epidemiol* 1990;19 (3):775.]
3. Macdonald SM, Reeder BA, Chen Y, Després JP, and the Canadian Heart Health Surveys Research Group. Obesity in Canada: a descriptive analysis. *CMAJ* 1997;157(1 Suppl):S3-S9.
4. Flagel KM, Carroll M, Kuczmarski RJ, Johnson C. Overweight and obesity in the United States: prevalence and trends, 1960-1994. *Int J Obes* 1998;22:39-47.
5. *Obesity. Preventing and managing the global epidemic. Report of a WHO consultation on obesity*. Geneva: World Health Organization; 1998. p. 1-276.
6. Lapidus L, Bengtsson C, Larsson B, et al. Distribution of adipose tissue and risk of cardiovascular disease and health: a 12-year follow-up of participants in the population study of women in Gothenburg, Sweden. *BMJ* 1984;288:1257-61.
7. Lee I, Paffenbarger RS Jr. Change in body weight and longevity. *JAMA* 1992;268:2045-9.
8. Lee IM, Manson JE, Hennekens CH, Paffenbarger RS Jr. Body weight and mortality: a 27-year follow-up of middle-aged men. *JAMA* 1993;270:2823-8.
9. Rabkin SW, Chen Y, Leiter L, Liu L, Reeder BA, and the Canadian Heart Health Surveys Research Group. Risk factor correlates of body mass index. *CMAJ* 1997;157(1 Suppl):S26-S31.
10. Birmingham CL, Muller JL, Palepu A, Spinelli JJ, Anis AH. The cost of obesity in Canada. *CMAJ* 1999;160:483-8.
11. Douketis JD, Feightner JW, Attia J, Feldman WF, with the Canadian Task Force on Preventive Health Care. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. *CMAJ* 1999;160:513-25.
12. Colditz GA. Economic costs of obesity. *Am J Clin Nutr* 1992;55:503S-507S.
13. Seidell JC. The impact of obesity on health status: some implications for health care costs. *Int J Obes* 1995;19(Suppl):S13-S16.
14. Kurscheid T, Lauterbach K. The cost implications of obesity for health care and society. *Int J Obes* 1998;22(Suppl 1):S3-S5.
15. *Promoting healthy weights: a discussion paper*. Ottawa: Health Services and Promotion Branch, Department of National Health and Welfare; 1988. Cat no H39-131/1988E.
16. Ohlson LO, Larsson B, Svardsudd 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.
17. Després JP, Moorjani S, Lupien PJ, et al. Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis* 1990;10:497-511.
18. Després JP. Abdominal obesity as an important component of insulin-resistance syndrome. *Nutrition* 1993;9:452-9.
19. Chan JM, Rimm ER, Colditz GA, et al. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. *Diabetes Care* 1994;17:961-9.
20. 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.
21. Pérusse L, Chagnon YC, Weisnagel J, Bouchard C. The human obesity gene map: the 1998 update. *Obes Res*. In press.
22. 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.
23. Strobel A, Issad T, Camoin L, et al. A leptin missense mutation associated with hypogonadism and morbid obesity. *Nature Genet* 1998;18:213-5.
24. Clément K, Vaisse C, Lahlou N, et al. A mutation in the human leptin receptor gene causes obesity and pituitary dysfunction. *Nature* 1998;392:398-401.
25. Behr M, Ramsden DB, Loos U. Deoxyribonucleic acid binding and transcriptional silencing by a truncated c-erbAB1 thyroid hormone receptor identified in a severely retarded patient with resistance to thyroid hormone. *J Clin Endocrinol Metab* 1997;82:1081-7.
26. Jackson RS, Creemers JWM, Ohagi S, et al. Obesity and impaired prohormone processing associated with mutations in the human prohormone convertaase 1 gene. *Nature Genet* 1997;16:303-6.
27. Krude H, Biebermann H, Luck W, et al. Severe early-onset obesity, adrenal insufficiency and red hair pigmentation caused by POMC mutations in humans. *Nature Genet* 1998;19:155-7.
28. Yeo GSH, Farooqi IS, Aminian S, et al. A frameshift mutation in MC4R associated with dominantly inherited human obesity. *Nature Genet* 1998;20:111-2.
29. Ristow M, Muller-Wieland D, Pfeiffer A, et al. Obesity associated with a mutation in a genetic regulator of adipocyte differentiation. *N Engl J Med* 1998;339:953-9.
30. Chagnon YC, Pérusse L, Bouchard C. The human obesity gene map: the 1997 update. *Obes Res* 1998;6:76-92.
31. West DB. Genetics of obesity in humans and animal models [review]. *Endocrinol Metab Clin North Am* 1996;25:801-13.
32. Bouchard C. Genetics of body fat content. In: Angel A, Anderson H, Bouchard C, Lau D, Leiter L, Mendelson R, editors. *Progress in obesity research:* 7. London (UK): John Libbey; 1996. p. 33-41.
33. Wood PD, Stefanick ML, Dreon DM, et al. Changes in plasma lipids and lipoproteins in overweight men during weight loss through dieting as compared with exercise. *N Engl J Med* 1988;319:1173-9.
34. Goldstein DJ. Beneficial health effects of modest weight loss. *Int J Obes* 1991;16:397-415.
35. Pavlou K, Krey S, Steffee W. Exercise as an adjunct to weight loss and maintenance in moderately obese subjects. *Am J Clin Nutr* 1989;49: 1115-20.
36. Tremblay A, Després JP, Maheux J, et al. Normalization of the metabolic profile in obese women by exercise and a low fat diet. *Med Sci Sports Exerc* 1991;23:1326-31.
37. Manson JE, Nathan DM, Krolewski AS, Stampfer MJ, Willett WC, Hennekens CH. A prospective study of exercise and incidence of diabetes among US male physicians. *JAMA* 1992;268:63-7.
38. Pan X, Li G, Hu YH, et al. Effect 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.
39. Guidelines for the approval and use of drugs to treat obesity. A position paper of the North American Association for the Study of Obesity. *Obes Res* 1995;3:473-8.
40. Stock MJ. Sibutramine — a review of the pharmacology of a novel anti-obesity drug. *Int J Obes* 1997;21(Suppl 1):S25-S29.
41. Sjstrom L, Rissanen A, Andersen T, et al. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. *Lancet* 1998;352:167-72.
42. Heal DJ, Aspley S, Prow MR, et al. Sibutramine: a novel anti-obesity drug. A review of the pharmacological evidence to differentiate it from amphetamine and d-fenfluramine. *Int J Obes* 1998;22(Suppl 1):S30-S35.
43. Sjstrom L. What does SOS teach us in 1998? *Int J Obes* 1998;22(Suppl 3):S93.

Reprint requests to: *Dr. David C.W. Lau, Division of Endocrinology and Metabolism, Ottawa Hospital — Civic Campus, 1053 Carling Ave., Ottawa ON K1Y 4E9; fax 613 761-5358; lau@civich.ottawa.on.ca*