

Utilization of health care resources by obese Canadians



Kostas Trakas,*¶ MSc; Kate Lawrence,‡ MB BS, MSc;
Neil H. Shear,*†§¶ MD

Abstract

Background: The prevalence of obesity in Canada has been increasing in recent years. Using data from the National Population Health Survey (NPHS), the authors determined the prevalence of obesity among Canadians, the associated comorbidities and the patterns of resource utilization by obese people.

Methods: The NPHS, a cross-sectional survey conducted in 1994, was administered to 17 626 Canadians 12 years of age or older who were not long-term residents of hospitals or long-term care facilities and were not residing on First Nations reserves or Canadian Armed Forces bases, or in the Yukon and Northwest Territories. For the authors' analysis, the study population consisted of 12 318 Canadians aged 20–64 years who were not pregnant and for whom the body mass index (BMI) had been calculated. The prevalence of comorbidities, health status index scores, self-esteem, self-rated health, restriction of activity, health care resource utilization (physician visits, disability days, admissions to hospital and medication use) were determined for obese people (BMI of 27 or greater) and nonobese people.

Results: The NPHS data revealed that 35.2% of men and 25.8% of women in Canada were obese in 1994. Obese respondents were more likely than nonobese respondents to suffer from stress (adjusted odds ratio [OR] 1.20, 95% confidence interval [CI] 1.11–1.31), activity restrictions (adjusted OR 1.39, 95% CI 1.26–1.54) and a number of chronic comorbidities. Obese respondents were also more likely to consult with physicians (adjusted OR 1.32, 95% CI 1.22–1.43), be prescribed a number of medications and to require excess disability days (adjusted OR 1.22, 95% CI 1.08–1.36).

Interpretation: Obesity represents a substantial burden on the health of Canadians and on Canada's health care resources.

Over the past 30 years health surveys have shown that between 24% and 50% of Canadians are obese^{1–3} and that the prevalence has grown substantially between the mid-1980s and early 1990s.^{4,5} Increases in the prevalence of obesity have also been observed in other countries over the last 15 to 20 years.^{6–12} In 1997 the Canadian Heart Health Surveys Research Group reported that 35% of men and 27% of women in Canada are obese (body mass index [BMI] of 27 or greater).¹³

A number of comorbidities have been linked to obesity, the most common being diabetes mellitus,^{14,15} hypertension¹⁶ and cardiovascular disease.¹⁶ Associations have also been reported with certain types of cancer (colorectal and prostate in men, and breast, cervical, endometrial, gallbladder and ovarian in women),^{17,18} osteoarthritis^{19,20} and gallbladder disease.²¹ In the United States, obesity is now reported to be second only to smoking as an attributable cause of death.²² Studies have shown that obesity and its comorbidities have been responsible for a substantial increase in health care costs.^{15,23,24}

We conducted this study to assess the impact of obesity in Canada. We did this by examining the prevalence of obesity and associated comorbidities and the utilization of health care resources using data from the 1994 National Population Health Survey (NPHS).

Evidence

Études

From the Departments of
*Pharmacology and
†Medicine, University of
Toronto, Toronto, Ont.;
‡the Medical Department,
Hoffmann–La Roche Limited,
Mississauga, Ont.; and
§the Division of Clinical
Pharmacology and ¶the
HOPE Research Centre,
Sunnybrook & Women's
College Health Sciences
Centre, Toronto, Ont.

This article has been peer reviewed.

CMAJ 1999;160:1457-62



Methods

We performed all of our analyses using data from the NPHS.²⁵ The NPHS was administered in 1994 to 17 626 Canadians in interviews conducted by telephone or in person. The BMI was calculated only for subjects 20–64 years of age who were not pregnant, which reduced our study population to 12 318. For the NPHS, data were collected on health care resource utilization, comorbidities, and economic, social and demographic correlates of health.

The target population of the NPHS included people in all provinces who were not long-term residents of hospitals or long-term care facilities and were not residing on First Nations reserves or Canadian Armed Forces Bases, or in the Yukon and Northwest Territories.

For our analysis obesity was defined as a BMI of 27 or greater, a cutoff that is consistent with that used in previous Canadian surveys.^{1,2,5} Other cutoffs have been used in the medical literature, and controversy still exists regarding the appropriate BMI for defining obesity. There are a number of commonly used measures of obesity. Although abdominal obesity has been shown to be most closely associated with health risk factors,²⁶ the BMI has been shown to correlate with other measures of obesity such as skinfold and body density measures.²⁷

The prevalence of obesity was determined for Canada as a whole, by region (Atlantic: Newfoundland, Prince Edward Island, Nova Scotia and New Brunswick; central: Quebec, Ontario and Manitoba; and western: Saskatchewan, Alberta and British Columbia) and by community size (rural v. urban). The prevalence and associations with age, sex, smoking status (daily, occasional, nonsmoker), level of physical activity (active, moderately active, inactive), education level (none, elementary, some secondary, some postsecondary, postsecondary) and household income level (low, middle, high) and were also determined.

We explored the potential relation between obesity and several comorbid and health-related conditions. These included chronic conditions, level of stress, level of activity restriction and health status index scores. In the NPHS each respondent's health status had been quantified using McMaster University's Health Utilities Index–Mark III (HUI–Mark III),²⁸ which consists of the 8 health attributes: vision, hearing, speech, mobility, dexterity, cognition, emotion and pain/discomfort. The HUI–Mark III is a preference-based, interval-scaled instrument that assigns death a utility score of 0.0 and perfect health a utility score of 1.0. The NPHS respondents had been asked to rate their health as excellent, very good, good, fair or poor. Major depression (measured using the Composite International Diagnostic Interview [CIDI]²⁹) had also been assessed in the obese and nonobese populations. For our analysis, respondents with a CIDI score of 5 or greater and those who were taking an antidepressant were categorized as having major depression.

We compared health care resource utilization between obese and nonobese respondents. Factors studied in the NPHS included the number of annual general practitioner (GP), mental health and total physician visits; the number of annual hospital admissions; the number of disability days (days that respondents had to stay in bed for most of the day or had to restrict activities for all or most of the day because of illness or injury) during the 2 weeks before the survey; and the use of common medications.

The number of GP visits made by each respondent was categorized as high (greater than the population median of 2 visits per year) or low (less than or equal to the population median). The total number of physician visits was determined by the NPHS for

both GPs and specialists (excluding ophthalmologists). Again, this number was categorized as high (greater than the population median of 2 visits per year) and low (less than or equal to the population median). The median number of disability days was found to be 0. Respondents were categorized as either requiring disability days during the 2 weeks before the survey (disability days > 0) or not requiring disability days during this period (disability days = 0).

Student's *t*-test was used for all comparisons of 2 independent, interval and normally distributed samples. The Mann–Whitney *U* test was used to compare 2 sample independent nonparametric ordinal data. Adjusted odds ratios (ORs) expressing the relation between obesity and various comorbidities and between obesity and medication use were derived using the comorbidity or drug as the dependent variable and age, sex, smoking status, level of physical activity, education level and household income level as covariates. The last 4 covariates were used only in the regression analysis if they were found to be significant ($p < 0.05$). Marital status and level of support (measured using 4-point scale, with 0 representing no perceived support and 4 greatest level of perceived support) were included only in the regression analysis between obesity and depression because these 2 factors are strong predic-

Table 1: Prevalence of obesity and mean body mass index (BMI) in Canada in 1994, by selected characteristics*

Characteristic	No. of people	Prevalence of obesity† (and 95% CI), %	Mean BMI (and 95% CI)
Age, yr			
20–34	4 545	23.2 (20.8–25.6)	24.3 (24.5–24.9)
35–54	5 997	33.3 (30.9–35.7)	25.6 (25.5–25.7)
55–64	1 776	40.1 (37.9–42.3)	26.3 (26.1–26.5)
Sex			
Male	6 245	35.2 (34.0–36.3)	25.9 (25.8–26.0)
Female	6 073	25.8 (24.7–26.9)	24.6 (24.5–24.7)
Region			
Atlantic	998	38.1 (35.0–41.1)	26.0 (25.8–26.3)
Central	7 814	29.3 (28.3–30.3)	25.2 (25.1–25.2)
Western	3 506	31.1 (29.6–32.7)	25.2 (25.1–25.3)
Smoking status			
Daily	3 550	29.0 (27.5–30.5)	24.9 (24.8–25.1)
Occasional	625	29.8 (26.2–33.4)	25.1 (24.8–25.4)
Nonsmoker	8 144	31.3 (30.3–32.3)	25.4 (25.3–25.5)
Level of physical activity			
Active	2 010	26.1 (24.2–28.0)	24.9 (24.7–25.0)
Moderately active	2 659	30.3 (28.6–32.1)	25.2 (25.1–25.4)
Inactive	7 183	32.3 (31.2–33.4)	25.4 (25.3–25.5)
Education level			
None	606	42.8 (38.9–46.8)	26.6 (26.2–26.9)
Elementary	1 905	37.2 (35.0–39.3)	25.9 (25.7–26.0)
Some secondary	2 042	32.7 (30.7–34.8)	25.3 (25.1–25.5)
Some postsecondary	5 722	29.0 (27.9–30.2)	25.1 (25.0–25.2)
Postsecondary	2 033	22.7 (20.9–24.5)	24.6 (24.5–24.8)
Household income level			
Low	1 907	30.3 (26.8–33.8)	25.2 (25.0–25.4)
Middle	7 830	31.1 (28.5–33.7)	25.3 (25.2–25.4)
High	2 130	29.7 (28.1–31.3)	25.2 (25.0–25.3)
All	12 318	30.6 (29.7–31.4)	25.3 (25.2–25.4)

Note: CI = confidence interval.

*All estimates were weighted to account for the sampling strategy used in the National Population Health Survey.

†Defined as BMI of 27 or greater.



tors of depression. Variables were modelled as continuous (age, smoking status, level of physical activity and education level) or as indicator variables (sex and income level) after assessing and confirming the appropriateness of such modelling. To explore the possibility of a changing relation between the comorbidities and obesity with age, an age-obesity interaction variable was tested in the regression model. The level of significance was set at 0.05 a priori for all tests.

Because the NPHS was based on a stratified, multiple-stage selection design with unequal probabilities of selecting respondents, we rescaled all sample weights to allow for statistical analysis. We did this by using an analysis weight equal to the original weight and dividing it by the mean of the original weights for the sampled units (respondents) contributing to the estimate in question.²⁵

Results

All analyses were based on data from the 12 318 respondents for whom a BMI measure was calculated (50.7% men, 49.3% women). Men were found to have a significantly higher mean BMI than women: 25.9 (95% confidence interval [CI] 25.8–26.0) v. 24.6 (95% CI 24.5–24.7) ($p < 0.001$). Overall, 35.2% of the men and 25.8% of the women were found to be obese (Table 1). The Atlantic region had the highest prevalence of obesity. Logistic regression analysis controlling for age, sex, level of physical activity and education level showed that Canadians in urban locations were less likely than those in rural areas to be obese (OR 0.77, 95% CI 0.68–0.87).

The odds of being obese significantly increased with increasing age (OR for a 5-year increase in age 1.51, 95% CI 1.43–1.60) and with decreasing level of physical activity (OR for active v. moderately active v. inactive 1.14, 95% CI 1.06–1.23). Nonsmokers were more likely to be obese (OR 1.08, 95% CI 1.01–1.15) than occasional or regular smokers; people with a higher education level (OR for no school v. elementary school v. some secondary v. some postsecondary v. postsecondary 0.83, 95% CI 0.78–0.87) and women (OR 0.80, 95% CI 0.77–0.83) were both associated with a decreased likelihood of obesity. Overall, obesity was not associated with household income level.

Fig. 1 displays the adjusted odds ratios associated with comorbid conditions in obese people versus nonobese people. The obese respondents were significantly more likely to have heart

disease, hypertension, diabetes mellitus, effects of a stroke, asthma, arthritis or rheumatism, back problems, emphysema or bronchitis, epilepsy, gastrointestinal ulcers and urinary incontinence. A statistical test of significance showed no change in the positive association between obesity and other comorbidities at different ages on this sample size.

Compared with the nonobese respondents, obese respondents were more likely to have a stress score above the median value (3 on a scale of 0–16, with higher values indicating higher stress) (adjusted OR 1.20, 95% CI 1.11–1.31), to report that they were limited in the amount of activities they could do at home, school, work or elsewhere because of a chronic physical or mental condition or health problem (adjusted OR 1.39, 95% CI 1.26–1.54), and to report feeling mild to severe pain ($p < 0.001$).

Health status index scores were found to be significantly lower among the obese subjects than among the nonobese subjects ($p < 0.001$). Obese respondents were marginally more likely than nonobese respondents to have a score below the Canadian mean of 0.947 (adjusted OR 1.17, 95% CI 1.07–1.27). Both obese men and women rated their

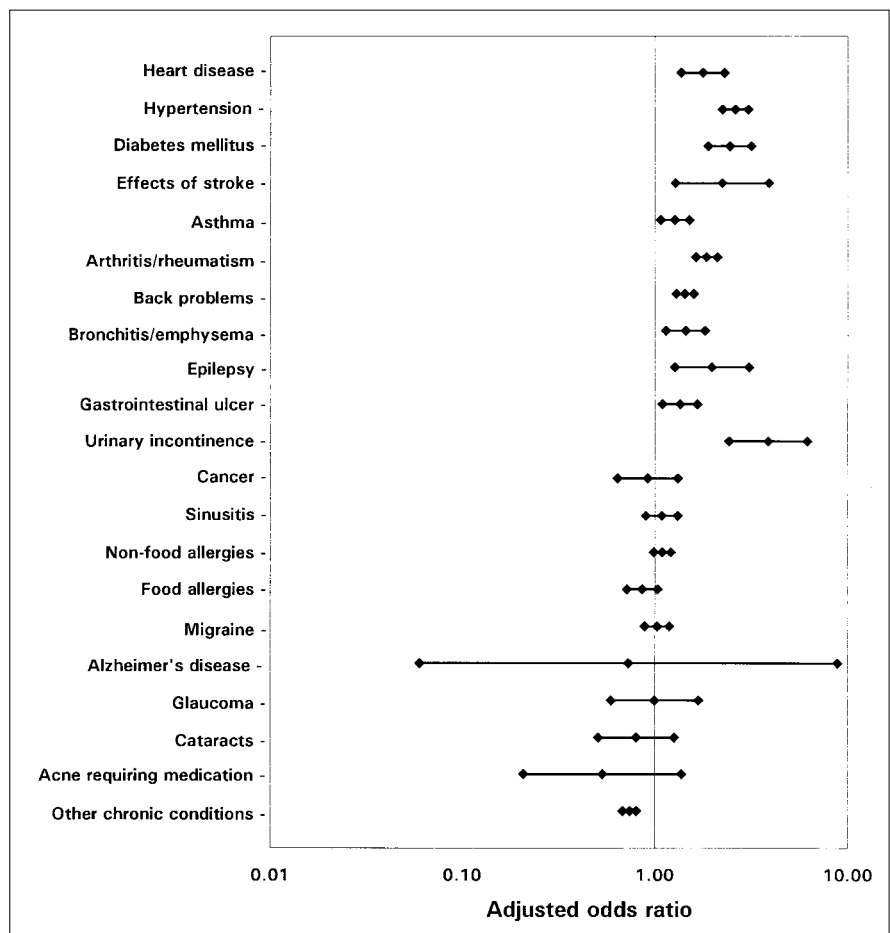


Fig. 1: Adjusted odds ratios (and 95% confidence intervals) of chronic comorbidities among obese people (body mass index ≥ 27) versus nonobese people in the 1994 National Population Health Survey, after controlling for age and sex. Smoking status, level of physical activity, education level and household income level were also controlled for if significant ($p < 0.05$).

health less favourably than their nonobese counterparts ($p < 0.001$) (Fig. 2).

There was no difference in the rate of depression between the obese and nonobese groups after controlling for age, sex, marital status, level of support, household income and smoking status³⁰ (adjusted OR 1.13, 95% CI 0.97–1.32).

A significantly higher rate of GP visits was observed in the obese group than in the nonobese group (adjusted OR for more than 2 GP visits per year 1.40, 95% CI 1.29–1.52). Redefining high GP utilization as more than 3 visits per year yielded similar results (adjusted OR 1.55, 95% CI 1.41–1.69). The obese respondents were found to consult mental health professionals more often than the nonobese respondents (adjusted OR 1.32, 95% CI 1.15–1.52). Overall, the obese respondents reported seeking medical care more often than the nonobese subjects (adjusted OR for more than 2 physician visits per year 1.32, 95% CI 1.22–1.43); redefining high physician utilization as more than 3 visits per year did not change the result (adjusted OR 1.49, 95% CI 1.37–1.62).

Although the obese respondents consulted physicians more often than the nonobese respondents, they had a

lower rate of hospital admission (adjusted OR 0.83, 95% CI 0.72–0.96). Obese individuals were more likely to require at least one disability day during the 2 weeks before the survey (adjusted OR 1.22, 95% CI 1.08–1.36).

As for medication use, the obese respondents were more likely than the nonobese respondents to be taking heart medication (unspecified), antihypertensive medication (unspecified), diuretics, oral diabetes medication, antidepressants, diet pills, antibiotics, stomach remedies (unspecified), pain relievers (for arthritis and back pain) and “other” medications (Fig. 3).

Interpretation

The findings in this population were consistent with those from previous Canadian surveys.^{13,31} The associations we observed between obesity and heart disease, hypertension, diabetes, effects of stroke, asthma, arthritis and rheumatism, back problems, chronic bronchitis and emphysema, and urinary incontinence have been described previously.^{14–16,32–39} The association between epilepsy and obesity has also been reported but is regarded as a reverse causation. Evidence points to the use of valproic acid for seizure control as the catalyst for weight gain in people with epilepsy.^{40,41} Cancer was not found to be associated with obesity in our analysis. Because cancer was not subclassified, it is possible that any association was diluted by inclusion of cancers not associated with obesity.

That the obese respondents had lower health status index scores and rated themselves as being in poorer health than the nonobese respondents is concordant with findings from a survey in Sweden.⁴²

Evidence in the literature is currently conflicting as to whether depression and obesity are associated.^{43–45} Our study did not demonstrate this association in Canada. However, obese respondents were found to use antidepressants and to visit mental health professionals more often than nonobese respondents. This contradiction may reflect limitations in the properties of the instrument used in the NPHS to measure depression.²⁹ Future research on this contentious issue may help in improving the quality of life of obese people.

Our finding that the obese respondents visited health care practitioners (GPs, mental health professionals and specialists) more often than the nonobese respondents is consistent with results from a previous study.⁴⁶ However, the obese respondents had fewer hospital admissions than the nonobese group in the year before the survey. Although the community-based nature of the NPHS could have introduced some bias into the accurate assessment of hospital admission rates, the survey indicates that the costs of obesity and associated comorbidities in primary care are substantial.

The drugs found to be most often associated with obesity in the NPHS were heart medications, diuretics, oral diabetes medications, antidepressants, diet pills, pain relievers, antibiotics and stomach remedies. The use of some of

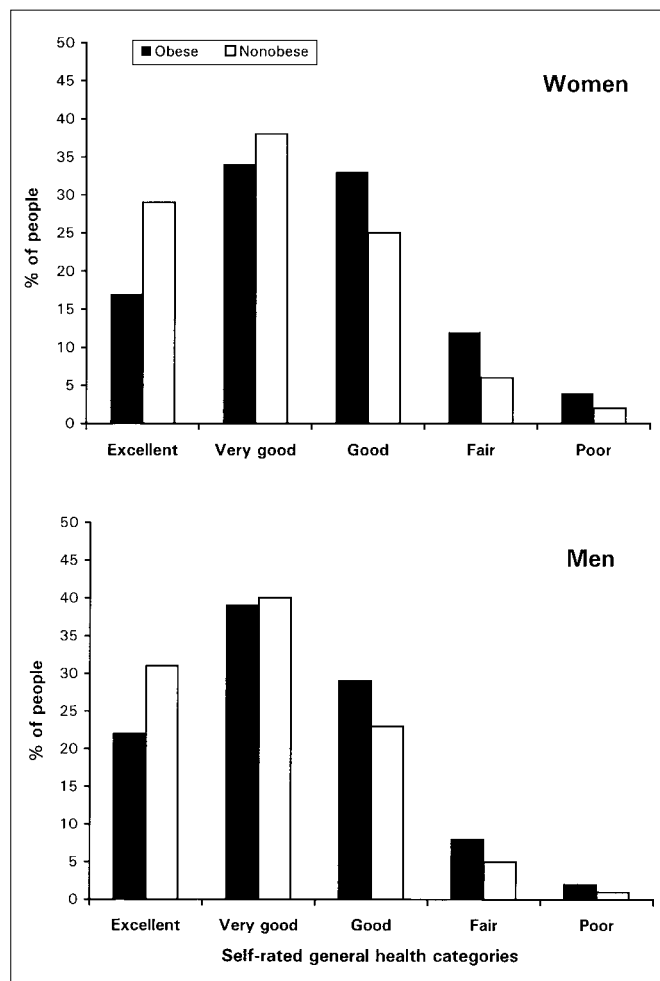


Fig. 2: Self-rated health of obese and nonobese men and women.



these medications might be reduced if the underlying problem of obesity were managed effectively.

There are some limitations in the information derived from the NPHS, given its cross-sectional nature. First, although a causal link cannot be conclusively established between the observed comorbidities and obesity in a study of this nature, the validity of this analysis is strengthened by its consistency with previously published surveys. Second, the dependence on patient recall in the NPHS may have resulted in some cases of misclassification. However, the overall effect of this is likely to result in a conservative estimate of the associations observed.

The use of odds ratios as a measure of risk is sometimes criticized. Although odds ratios may overstate the degree of risk arising from a calculation of the relative risk, they rarely lead to qualitatively differing judgements, and the size of any discrepancy will be low at the levels of risk observed in this analysis.⁴⁷

Conclusion

Our analysis demonstrates a significant impact of obesity

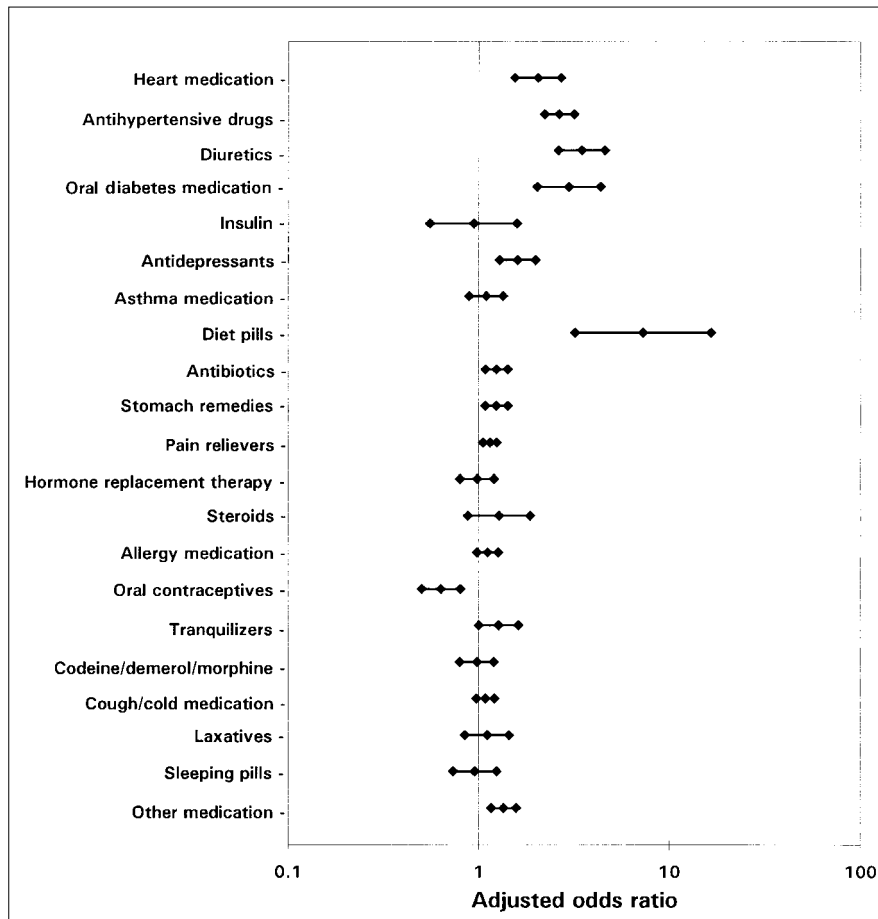


Fig. 3: Adjusted odds ratios (and 95% confidence intervals) for utilization of medications among obese people versus nonobese people, after controlling for age and sex. Smoking status, level of physical activity, education level and household income level were also controlled for if significant ($p < 0.05$).

in the Canadian population. Obesity represents a substantial burden on the health of Canadians and on health care resources. The burden is likely to grow unless the current obesity epidemic is addressed.

Competing interests: Mr. Trakas and Dr. Shear received an educational grant from Hoffmann-La Roche Limited for the purposes of this study. Dr. Lawrence is currently an employee of Hoffman-La Roche Limited.

References

1. *The health of Canadians: report of the Canadian Health Survey*. Ottawa: Department of National Health and Welfare and Statistics Canada; 1981.
2. *Fitness and life-style in Canada: a report by Canada Fitness Survey*. Ottawa: Fitness and Amateur Sport Canada; 1983.
3. Stephens T, Craig C. *The well-being of Canadians: highlights of the 1988 Campbell's Survey*. Ottawa: Canadian Fitness and Lifestyle Research Institute; 1990.
4. Stephens T. *Canada's Health Promotion Survey 1990: technical report*. Ottawa: Supply and Services Canada; 1993.
5. Millar WJ, Stephens T. Social status and health risks in Canadian adults: 1985-1991. *Health Rep* 1993;5(2):143-56.
6. Seidell JC. Obesity in Europe — scaling an epidemic. *Int J Obes Relat Metab Disord* 1995;19(Suppl 3):S1-4.
7. Prentice AM, Jebb SA. Obesity in Britain: Gluttony or sloth? *BMJ* 1995;311:437-9.
8. Blokstra A, Kromhout D. Trends in obesity in young adults in The Netherlands from 1974 to 1986. *Int J Obes* 1991;15:513-21.
9. Skodova Z, Pisa R, Emrova J, Vorlicek J, Vojtisek P, Berka L, et al. Cardiovascular risk factors in the Czech population. *Cor Vasa* 1991;33(2):114-22.
10. Korkeila M, Kaprio J, Koskenvuo M. Weight changes among 14 605 adult Finns over six years. *Int J Obes* 1991;15:1024.
11. Kuskowska-Wolk A, Rossner S. Trends in body mass index in the Swedish population 1981-1989 [abstract]. *Int J Obes* 1991;15:1.
12. Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults. The National Health and Nutrition Examination Survey. *JAMA* 1994;272(3):205-11.
13. 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):S3-9.
14. Felber JP, Acheson KJ, Tapp L. *From obesity to diabetes*. West Sussex (UK): John Wiley & Sons; 1993. p. 1-45.
15. Pi-Sunyer F. Medical hazards of obesity. *Ann Intern Med* 1993;119:655-60.
16. Manson JE, Colditz GA, Stampfer MJ, Willett WC, Rosner B, Monson RR, et al. A prospective study of obesity and risk of coronary heart disease in women. *N Engl J Med* 1990;322(15):882-9.
17. Kissebah AH, Freedman DS, Peiris AN. Health risks of obesity. *Med Clin North Am* 1989;73(1):111-9.
18. Garfinkel L. Overweight and cancer. *Ann Intern Med* 1985;103(6 pt 2):1034-6.
19. Hart DJ, Spector TD. The relationship of obesity, fat distribution, and osteoarthritis in women in the general population: the Chingford Study. *J Rheumatol* 1993;20(2):331-5.
20. Hartz AJ, Fischer ME, Bril G, Kelber S, Rupley D Jr, Oken B, et al. The association of obesity with joint pain and osteoarthritis in the Hanes data. *J Chronic Dis* 1986;39(4):311-9.
21. Maclure KM, Hayes KC, Colditz GA, Stampfer MJ, Speizer FE, Willett WC. Weight, diet, and the risk of symptomatic gallstones in middle-aged women. *N Engl J Med* 1989;321(9):563-9.
22. McGinnis JM, Foege WH. Actual causes of death in the United States. *JAMA* 1993;270(18):2207-12.
23. Colditz GA. Economic costs of obesity. *Am J Clin Nutr* 1992;55(2 Suppl):503S-507S.
24. Sjöström L, Narbro K, Sjöström D. Costs and

benefits when treating obesity. *Int J Obes* 1995;19(Suppl 6):S9-12.

25. *National Population Health Survey: public use microdata files*. Ottawa: Statistics Canada; 1994-95. Report no 82F0001XDB.
26. Reeder BA, Senthilselvan A, Després JP, Angel A, Liu L, Wang H, et al, and the Canadian Heart Health Surveys Research Group. The association of cardiovascular disease risk factors with abdominal obesity in Canada. *CMAJ* 1997;157(1 Suppl):S39-45.
27. Micozzi MS, Albanes D, Jones V, Chumlea WC. Correlation of body mass indices with weight, stature, and body composition in men and women in NHANES I and NHANES II. *Am J Clin Nutr* 1986;44(6):725-31.
28. Boyle M, Furlong W, Feeny D, Torrance GW, Hatcher J. Reliability of the Health Utilities Index-Mark III used in the 1991 cycle 6 Canadian General Survey Health Questionnaire. *Qual Life* 1995;4:249-57.
29. World Health Organization. *The composite international diagnostic interview*. Version 1.1, Researcher's manual. Geneva: WHO; 1994.
30. Bromberger JT, Costello EJ. Epidemiology of depression for clinicians. *Soc Work* 1992;37(2):120-5.
31. Reeder BA, Chen Y, Macdonald SM, Angel A, Sweet L, and the Canadian Heart Health Surveys Research Group. Regional and rural-urban differences in obesity in Canada. *CMAJ* 1997;157(1 Suppl):S10-6.
32. Bailey WC, Richards JM Jr, Manzella BA, Brooks CM, Windsor RA, Soong SJ. Characteristics and correlates of asthma in a university clinic population. *Chest* 1990;98(4):821-8.
33. Cherniack RM. Evaluation of respiratory function in health and disease. *Dis Mon* 1992;38(7):505-76.
34. Symmons DP, Bankhead CR, Harrison BJ, Brennan P, Barrett EM, Scott DG, et al. Blood transfusion, smoking, and obesity as risk factors for the development of rheumatoid arthritis: results from a primary care-based incident case-control study in Norfolk, England. *Arthritis Rheum* 1997;40(11):1955-61.
35. Felson DT, Anderson JJ, Naimark A, Walker AM, Meenan RF. Obesity and knee osteoarthritis. The Framingham Study. *Ann Intern Med* 1988;109(1):18-24.
36. Croft PR, Rigby AS. Socioeconomic influences on back problems in the community in Britain. *J Epidemiol Community Health* 1994;48(2):166-70.
37. Kilburn KH, Asmundsson T. Factors influencing the course of COPD. *Postgrad Med* 1973;54(3):135-41.
38. Mommsen S, Foldspang A. Body mass index and adult female urinary incontinence. *World J Urol* 1994;12(6):319-22.
39. Kolbl H, Riss P. Obesity and stress urinary incontinence: significance of indices of relative weight. *Urol Int* 1988;43(1):7-10.
40. Corman CL, Leung NM, Guberman AH. Weight gain in epileptic patients during treatment with valproic acid: a retrospective study. *Can J Neurol Sci* 1997;24(3):240-4.
41. Dinesen H, Gram L, Andersen T, Dam M. Weight gain during treatment with valproate. *Acta Neurol Scand* 1984;70(2):65-9.
42. Sullivan M, Karlsson J, Sjöström L, Backman L, Bengtsson C, Bouchard C, et al. Swedish obese subjects (SOS) — an intervention study of obesity. Baseline evaluation of health and psychosocial functioning in the first 1743 subjects examined. *Int J Obes* 1993;17(9):503-12.
43. Karlsson J, Sjöström L, Sullivan M. Swedish obese subjects (SOS) — an intervention study of obesity. Measuring psychosocial factors and health by means of short-form questionnaires. Results from a method study. *J Clin Epidemiol* 1995;48(6):817-23.
44. Friedman MA, Brownell KD. Psychological correlates of obesity: moving to the next research generation. *Psychol Bull* 1995;117(1):3-20.
45. Ross CE. Overweight and depression. *J Health Soc Behav* 1994;35(1):63-79.
46. Sjöström L, Narbro K, Sjöström D. Costs and benefits when treating obesity. *Int J Obes Relat Metab Disord* 1995;19(Suppl 6):S9-12.
47. Davies HTO, Crombie IK, Tavakoli M. When can odds ratios mislead? *BMJ* 1998;316:989-91.

Reprint requests to: Dr. Neil H. Shear, Division of Clinical Pharmacology, Rm. E240, Sunnybrook & Women's College Health Sciences Centre, 2075 Bayview Ave., Toronto ON M4N 3M5; fax 416 480-6025

CALL FOR PAPERS

CMAJ's Osler issue

On July 12, we will celebrate the 150th anniversary of the birth of William Osler. *CMAJ's* contribution to the festivities will be a special Osler issue, to be published in October. Original articles on Osler's life and works received by June 30 will have a greater chance of acceptance. For details see the editorial in the February 9 issue (*CMAJ* 1999;160:346) or read it online (www.cma.ca/cmaj/vol-160/issue-3/0346.htm).

Can you guess which of these men is Osler?



Answer: The one wearing the tall hat. The other is Osler's McGill cronie, Frank Shepherd.

What's your sign?

We invite you to send us your brief descriptions (250 to 300 words) of physical signs that have been named after Osler or whose discovery is attributed to him. Documentation of the original attribution to Osler and a high-quality photograph or illustration should be provided.