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Chronic Diseases in Canada (CDIC) is a quarterly scientific journal focussing on current evidence relevant to the control and prevention of chronic (i.e. non-communicable) diseases and injuries in Canada. Since 1980 the journal has published a unique blend of peer-reviewed feature articles by authors from the public and private sectors and which may include research from such fields as epidemiology, public/community health, biostatistics, the behavioural sciences, and health services or economics. Only feature articles are peer reviewed. Authors retain responsibility for the content of their articles; the opinions expressed are not necessarily those of the CDIC editorial committee nor of the Public Health Agency of Canada.

Chronic Diseases in Canada
Public Health Agency of Canada
785 Carling Avenue
Address Locator 6805B
Ottawa, Ontario K1A 0K9

Fax: (613) 941-9502
E-mail: cdic-mcc@phac-aspc.gc.ca

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Editorial

Chronic Diseases in Canada Grows Up

H. Morrison, PhD, Editor-in-Chief, M. Tracy, MA, Managing Editor,
D. Wigle, MD, PhD, former Principal Scientific Editor

This fall, two of us (H. M., M. T.) attended the JAMA/BMJ Peer Review Congress on Peer Review and Biomedical Publication as Editor-in-Chief and Managing Editor of *Chronic Diseases in Canada*. Both being new at the job, we were painfully aware of the relative importance of our niche journal when placed side-by-side with the Big Five (*New England Journal of Medicine*, JAMA, BMJ, *Lancet*, *Annals of Internal Medicine*).

Chronic Diseases in Canada (CDIC) started thirty years ago at Health and Welfare Canada's Laboratory Centre for Disease Control (LCDC) in a dingy little building at Tunney's Pasture in Ottawa. The first issue was four-pages long in courier font and a red 'fifties-style banner. The then Director General of the LCDC, Dr. A. J. Clayton, launched "the New Bulletin" with a short editorial containing the following statement of purpose: "We propose to include material based on research, surveillance and control aspects of non-communicable diseases or conditions such as cancer, heart disease and accidents." The main audience was the estimated 300-400 Canadians involved directly or indirectly in programs related to chronic disease.

LCDC initiated CDIC in 1980 because there was no national publication to address the needs of public health professionals responsible for chronic disease prevention and control. Many people contributed to the early development of CDIC, including Dr. Don Wigle, Walter Litvin, Lori Anderson and Dr. Christina Mills.

Chronic disease science has evolved considerably since the 1980s, when the journal was first founded: we now know a great deal more about risk factors, but we still don't know very much about interventions. As we approached our 30th anniversary, it was apparent that the journal needed to catch up. Starting in December 2007, the journal hired a new Managing Editor, named a new Editor-in-Chief and reconfigured the Editorial Board. Since then, we've seen improvements in our process and output – article submissions are up 40%, our timelines are more competitive, and we're getting ready to implement an online manuscript management system and allow for advance access of articles.

At our first Editorial Board Meeting this past October, a member asked the provocative question: "Why bother?" I would like to posit a reply in stating my vision for the journal.

Chronic Diseases in Canada is, and always will be, a niche journal. Our audience, now considerably larger than the original 300-400 chronic disease experts, is a mixed constituency of researchers, students and policy makers. I would argue that we provide a useful forum for mixed-methods research papers – evaluating interventions using both qualitative and quantitative approaches. In the words of Dr. Sylvie Stachenko, former Principal Scientific Editor of the journal, "CDIC should publish both quantitative and qualitative research results since both types of research make important contributions to chronic disease prevention. For instance, qualitative research documents the context within which prevention programs must operate."

With regard to the future, I believe that CDIC should address currently underserved topics. Although the journal will continue to publish surveillance and original etiologic research findings, the focus will be increasingly on chronic disease prevention research and evaluation of intervention programs, particularly those from a Canadian context. This will include review articles on the relative importance of various prevention programs and policies in reducing the burden of chronic disease in Canada.

And while we may be a niche journal, we are, in many ways, unique. As a government-funded journal, we are part of the Open Access movement, indexed with Open Access Journals as well as PubMed. As CDIC is housed within the Public Health Agency of Canada, its editors have prime access to national intervention research and to partnership opportunities with bodies such as the Canadian Institutes for Health Research, Statistics Canada, the Institut national de la santé publique du Québec, the US Centers for Disease Control and Prevention and provincial health departments. Also, the journal is the only national bilingual journal in its field. We have a long way to go, but we are well on our way.

References

1. Clayton A J. Guest Editorial – Launching of New Bulletin. Chronic Dis Can. 1980 June 1(1):1.

Editorial

Chronic disease or chronic diseases: is the whole different from the sum of the parts?

R. A. Spasoff, MD, Associate Scientific Editor

About 15 years ago I spent a sabbatical year at the University of Amsterdam. When I first arrived, I naturally made the rounds meeting the staff. When I asked one senior professor about her research interests, she replied "Chronic disease." "That's nice," I said, assuming that she meant etiological research on one or more chronic diseases, "Which ones?" She was as mystified by my question as I was by her first response. It turned out that she studied chronic disease as a phenomenon: its impact on individuals, their caregivers and society, its implications for health care, etc. And this was my introduction to chronic disease (singular) as a research topic.

Since then, I have increasingly realized that the phenomenon of chronic disease *in general* is worthy of research, teaching, policy and programs. Gerontologists have had a similar approach for years, being often more interested in the fact that an individual has one or more chronic diseases and in the impact of these on that person's health, than on the individual diseases. (Of course, this is sometimes of necessity, since it can be hard to make a specific diagnosis in very old people.) Similar thinking underlies the International Classification of Functioning, Disability and Health[1] (ICF) and its predecessor, the International Classification of Impairment, Disability and Handicap, which emphasizes the individual's ability to carry out functions and roles rather than the individual diseases that cause these limitations. The title of the US sister publication to *Chronic Diseases in Canada* (CDIC), *Preventing Chronic Disease* (singular), published by the Centers for Disease Control and Prevention (CDC), seems to hint at the same point. But note the use of both singular and plural in the documents mentioned in the next paragraph.

The risk factors for several major chronic diseases have turned out to be remarkably similar, further suggesting that prevention can often focus on chronic disease in general rather than (or as well as) individual chronic diseases. Perhaps too strongly (because it ignores the role of genetic factors), the WHO's *Preventing Chronic Diseases: a vital investment*[2] points out that "common, modifiable risk factors underlie the major chronic diseases. These risk factors explain the vast majority of chronic disease deaths at all ages, in men and women, and in all parts of the world." The Chronic Disease Prevention Alliance of Canada (CDPAC; slogan, "reducing chronic disease in Canada") in its vision document *Primary Prevention of Chronic Diseases in Canada: a Framework for Action* calls for a comprehensive and integrated approach to primary prevention: "A key stimulus for initiating a framework for primary prevention was a shared interest and need among the national disease strategies—cancer, stroke, diabetes, heart health, lung, healthy living and chronic disease—to align their contributions to primary prevention."[3] Again, this suggests that the target will often be chronic disease in general. Of course, we also need to consider the individual diseases, especially for their treatment.

The first in the CDPAC framework's four components is resources, which includes research and innovation and also knowledge exchange. CDIC is an appropriate venue for dissemination of these activities, and is interested in both chronic diseases and chronic disease. Probably most of our articles have dealt with the former—consistent with the plural in our title—but we would welcome more submissions on the latter.

References

1. World Health Organization. International Classification of Functioning, Disability and Health. Geneva: World Health Organization, 2001 [cited on 2009 Sep 4]. Available from: http://apps.who.int/classifications

2. Le Gales-Camus C, Beaglehole R, Epping-Jordan J, Vita-Finzi L, editors. World Health Organization. Preventing chronic diseases: a vital investment. WHO global report [Internet]. Geneva: World Health Organization, 2005 [cited on 2009 Sep 4]. Available from: http://www.who.int/chp/chronic_disease_report/contents/en/index.html

3. Garcia J, Riley B. Primary prevention of chronic diseases in Canada: a framework for action [Internet]. Ottawa (ON): Chronic Disease Prevention Alliance of Canada; July 2008 [cited on 2009 Sep 4]. Available from: http://www.cdpac.ca/media.php?mid=451

Socio-demographic and geographic analysis of overweight and obesity in Canadian adults using the Canadian Community Health Survey (2005)

J. Slater, PhD (1); C. Green, PhD (4); G. Sevenhuysen, PhD (1); J. O'Neil, PhD (2); B. Edginton, PhD (3)

Abstract

Using the 2005 Canadian Community Health Survey, this study examined how overweight and obesity in Canadian adults are distributed across socio-demographic and geographic groupings. Overweight and obesity prevalence were modeled against socio-demographic indicators using Poisson regression and were assessed geographically using choropleth maps. The Gini coefficient was used to assess the distribution of prevalence across risk groups. The potential impacts of high risk versus population-based prevention approaches on the population prevalence of obesity were also examined. Of adults aged 25 to 64 years, 17% were obese and 53% were overweight or obese, with the highest proportions observed in older age groups, among those who were physically inactive, white or non-immigrant, with low educational levels, and living in the prairie and east coast regions. Recalculation of obesity rates under the different prevention scenarios demonstrated that population-based approaches could achieve a four-fold greater decrease in obesity cases than high risk approaches, highlighting the need for broader population strategies for obesity prevention in Canada.

Keywords: *obesity, prevalence, prevention, policy, population-based, geographic trend*

Introduction

Overweight and obesity continue to be major public health issues in Canada.[1,2] Studies conducted over the past several decades show that body mass index (BMI) for both adults and children have increased significantly[3,4] despite efforts to address increasing body weights.[5,6] This upward trend calls for a re-assessment of current approaches to understanding the phenomenon of population overweight and obesity. To date, public health interventions have tended to focus on high risk individuals and the promotion of "healthy lifestyles" to change individual behaviours.[5-7] Given the dramatic growth in population body weights in recent decades, a re-examination of the distribution of overweight and obesity in the population is warranted. Our objective is to examine how overweight and obesity in Canadian adults are distributed across socio-demographic and geographic groupings. The study results will provide important information for public health policy and program planners to use in designing effective strategic interventions to decrease the population prevalence of overweight and obesity.

Methods

Data sources

Our analysis is based on data derived from the Public Use Microdata File (PUMF) of the 2005 Canadian Community Health Survey (CCHS), Cycle 3.1, obtained through the Data Liberation Initiative[8] at the University of Manitoba. The methodology of the survey has been detailed elsewhere.[9] BMI measures were derived from self-reported height and weight. While the 2005 CCHS does provide a subsample of measured height and weight, this sub-sample was too small to allow the predictive modeling and geographic analysis used by the study. Our study was restricted to adults 25 to 64 years of age: the secondary education variable used to model overweight/obesity rates is only meaningful in adults 25 years and older and the BMI measure is valid only in adults younger than 65 years of age.

For geographic mapping, an electronic map file (shape file) was obtained from the Statistics Canada web site.[10] Since the PUMF collapses the number of health regions from 122 to 101 in order to protect data confidentiality, the health region shape file was similarly modified to contain only 101 health region polygons. This was accomplished using ArcGIS 9.1.[11]

Data preparation and analysis

CCHS data was imported into STATA version 9[12] and a program was written to extract records for individuals 25 to 64 years of age and to code survey variables into the categorical variables required for the study. Individuals with a BMI greater than or equal to 30 were categorized as obese, while those with a BMI greater than or equal to 25 were categorized as overweight/obese.

Author References

1 Department of Human Nutritional Sciences, University of Manitoba
2 Faculty of Health Sciences, Simon Fraser University
3 Department of Sociology, University of Winnipeg
4 Department of Community Health Sciences, University of Manitoba
Correspondence: Joyce Slater, Department of Human Nutritional Sciences, University of Manitoba, Winnipeg, Manitoba, R3T 2N2; Tel.: (204) 977-5678; Fax: (204) 789-3905; E-mail: slater@cc.umanitoba.ca

All statistical calculations were undertaken in STATA version 9[12] using the Survey Data Analysis module, which used the survey weight on the CCHS record to compute correct parameter estimates for calculated statistics. Since the CCHS is based upon a complex sampling design, the standard error required for an assessment of statistical significance may not be estimated properly using standard statistical techniques that do not take into account a complex sampling design.[9] In order to accurately estimate standard errors associated with output statistics, survey design details are required, including the strata and primary sampling units. The impact of a complex survey design on variance estimates can be summarized as the survey design effect, which is the ratio of the true variance associated with the survey to a comparable variance estimate from a simple random sample of the population.[13] Statistics Canada suggests using the bootstrap resampling method in order to calculate accurate standard errors for the CCHS which take into account the survey design effect. However, the bootstrap method is not available for use with the PUMF and does not support two of the statistical routines employed in this study (Poisson regression and Gini coefficient).

An approximate method[13] was used to incorporate the survey design effect into the calculation of 95% confidence interval estimates for all statistical routines employed in the study. First, the survey design factor was derived by taking the square root of the average survey design effect for the 2005 CCHS of 2.51.[9] Standardized variables (z-scores) used in the calculation of 95% confidence intervals were then re-scaled by the survey design effect ($z = 1.96 * \sqrt{2.51} = 3.10$). In order to streamline this calculation within STATA, confidence intervals were set to 0.998, which is functionally equivalent to using a z-score of 3.10.

In order to describe the demographic and geographic distribution of overweight and obesity, rates were calculated by age, gender and geography and standardized (where appropriate) by age and/or gender using the 2005 CCHS sample population as the standard population. Geographic patterns were visualized through choropleth maps, with rates of overweight and obesity classified into quintiles using the Jenks natural breaks algorithm in ArcGIS 9.1.[11] To model the population characteristics associated with overweight and obesity, categorical Poisson regression analysis was used to generate comparative rate ratios, which are more easily interpretable than the odds ratio generated by logistic regression.[14,15] Model predictor variables included age group, gender, education, fruit and vegetable consumption, physical activity level, immigration status, visible minority status, household income and food security.

The public health significance of the variability in rates of overweight and obesity by immigrant status, education, income and geography was assessed using the Gini coefficient,[16] a measure of inequality ranging from 0 (absolute equality in rates) to 1 (absolute inequality in rates), which has been used previously to examine the geographic variability of infant mortality,[16] sexually transmitted diseases[17] and *campylobacter*.[18] It is calculated by ordering risk categories from lowest to highest rank by case rate, calculating a Lorenz curve which is a plot of the cumulative proportion of the population (x axis) against the cumulative proportion of cases in each risk category (y axis), and then calculating the area between the axis of equality and the Lorenz curve as a percentage of the total area below the axis of equality (Figures 2a and 2b). The greater the degree to which cases are concentrated in a small number of risk categories (i.e. distributed disproportionately in relationship to the population at risk), the greater the deflection of the Lorenz curve downwards from the axis of equality and the higher the Gini coefficient. A high Gini coefficient indicates that the majority of cases are located in a small proportion of the population, and a public health intervention would only need to focus on factors affecting this high risk population in order to be successful in treating or preventing the majority of cases. Alternatively, a low Gini coefficient indicates that cases are spread out relatively evenly across all population groups (despite there being some small groups with very high rates); a public health intervention that focused on these small high-risk groups would end up treating or preventing only a very small percentage of total cases.

We calculated the Gini coefficients and associated confidence intervals for geography (health regions), immigration, education and income for both obesity and overweight/obesity using the *Ineqerr* program in Stata,[12] with associated Lorenz curves produced in EpiDat version 3.1.[19]

To explore how the success of high risk versus population-based prevention strategies may be constrained or enabled by the statistical distribution (Gini coefficient) of obesity cases in the population, we developed two high risk scenarios and two population-based prevention scenarios and calculated their potential impacts on population obesity prevalence. Prevention scenarios were developed for adult obesity (both genders) across health regions and for adult obesity in women by income quintile. The high risk prevention scenarios assumed that public health interventions would successfully prevent 50% of the cases of obesity in the 10% of the population at highest risk for obesity, with the number of cases prevented ascertained directly from the Lorenz curve plot. The population-based scenarios assumed that the obesity prevalence of all population groups could be reduced to the same level as the best performing/lowest risk groups in the study. In the health region analysis, we applied age-specific obesity rates in the three lowest rate health regions (making up 11% of the study population) to the total study population; in the women by income analysis, we applied age-specific obesity rates in the lowest rate quintile (household income > $80,000; 35% of the population) to the total study population. For all scenarios, we calculated the number of obesity cases prevented and the percentage reduction in obesity prevalence.

Results

In 2005, based on self-reported data, almost 3 million (17.31%) of Canadians aged 25 to 64 years were obese, while close to 9 million (52.74%) were overweight/obese (Table 1). Rates of both obesity and

overweight/obesity were the highest in men and increased significantly with age for both genders.

Poisson regression analysis revealed that rates of both obesity and overweight/obesity were significantly graded by the demographic characteristics of the study population. This relationship was more pronounced for obesity than for overweight/obesity and for women as compared to men. Tables 2a and 2b express the outputs of the Poisson regression analysis in terms of rate ratios. A rate ratio (RR) is the ratio of the prevalence rate in the category of interest compared to the prevalence rate in the reference or comparison category. As illustrated in Table 2a, rates of obesity were 1.52 times higher in women in the oldest age group (55 to 64 years) compared to the youngest age group (25 to 34 years) and 2.01 times higher in the most physically inactive group compared to the most physically active group. Similarly, in comparison to the reference category, significantly higher rates of obesity were observed in women who were white (RR = 1.35), were non-immigrant (RR = 1.98), had less than a secondary grade level of education (RR = 1.69), an annual household income of less than $15,000 (RR = 1.95) and moderate levels of food insecurity (RR = 1.99). For men, rates of obesity varied significantly and in the same direction as for women, but only for age (RR = 1.43), physical activity levels (RR = 1.48), non-immigrant status (RR = 2.09) and white racial status (RR = 1.53). For both sexes combined, significant rate ratios were observed for age, sex, physical activity, non-immigration and white racial status, education, household income and food security. Paradoxically, the relationship between obesity and education did not vary in a linear fashion, with higher obesity rates (RR = 1.32) occurring in those with some post-secondary education than in those who had completed only secondary education. Directionally similar but weaker trends were observed for overweight/obesity (Table 2b).

Rates of obesity (Figure 1a) varied by health region from a low of 8.90% to a high of 32.24%. The highest rates of obesity were observed in Saskatchewan, south central Manitoba and the east coast (Newfoundland, Labrador and New Brunswick) and the lowest in southern British Columbia, central Alberta, southern Ontario and Quebec. The major urban centers of Vancouver and lower mainland British Columbia, Calgary, Ottawa, Montreal and Toronto were included in the lowest rate regions. Similar geographic patterns were observed for rates of overweight/obesity (Figure 1b), which ranged from a low of 36.28% to a high of 71.11%.

Although obesity and overweight/obesity varied significantly by immigration status, education, income and geography, the very low Gini coefficient values observed (Table 3) suggest that, for the population as a whole, the cumulative number of cases of obesity and overweight are distributed relatively equally in relation to the cumulative population in each risk category. The highest Gini coefficients observed in the study were for obesity by geography (0.153 to 0.169) and for female obesity by income (0.129), however these values are much closer to 0 (absolute equality) than to 1 (absolute inequality). The Lorenz curves (Figures 2a and 2b) show that in the case of geography (both genders), with a Gini coefficient of

TABLE 1
Prevalence of obese* and overweight/obese† adults (25–64 years), Canadian Community Health Survey, 2005

OBESE						
	Women		Men		Both sexes	
Age Group	Cases	Cases/ 100 (95% CI)	Cases	Cases/100 (95% CI)	Cases	Cases/100 (95% CI)
25–34	242 903	12.81 (11.45–14.18)	312 594	15.42 (13.81–17.02)	555 497	14.16 (13.10–15.22)
35–44	329 047	13.83 (12.32–15.34)	453 461	17.76 (16.11–19.41)	782 508	15.86 (14.74–16.99)
45–54	410 160	17.55 (15.64–19.46)	474 471	20.36 (18.20–22.52)	884 630	18.95 (17.51–20.40)
55–64	333 358	19.42 (17.74–21.10)	388 590	21.98 (19.88–24.08)	721 948	20.72 (19.37–22.07)
All Ages	1 315 467	15.79 (14.96–16.63)	1 629 116	18.77 (17.82–19.72)	2 944 583	17.31 (16.68–17.95)

OVERWEIGHT AND OBESE						
	Women		Men		Both sexes	
Age Group	Cases	Cases/ 100 (95% CI)	Cases	Cases/100 (95% CI)	Cases	Cases/100 (95% CI)
25–34	637 572	33.63 (31.57–35.68)	1 090 256	53.78 (51.51–56.05)	1 727 828	44.05 (42.47–45.62)
35–44	922 299	38.76 (36.53–40.98)	1 585 951	62.11 (58.95–64.27)	2 508 250	50.85 (49.26–52.44)
45–54	1 080 994	46.25 (43.61–48.89)	1 511 101	64.83 (62.21–67.45)	2 592 095	55.53 (53.63–57.44)
55–64	955 247	55.65 (53.37–57.93)	1 187 170	67.10 (64.68–69.63)	2 142 417	61.50 (59.80–63.19)
All Ages	3 596 112	43.17 (41.98–44.36)	5 374 478	61.92 (60.72–63.13)	8 970 590	52.74 (51.88–53.61)

* BMI ≥ 30

† BMI ≥ 25

Abbreviations: BMI, body mass index; CI, confidence interval

TABLE 2a
Poisson regression analysis, obese* adults (25-64 years), Canadian Community Health Survey, 2005

	Women		Men		Both sexes	
Predictor	**RR (95% CI)**	**Cases**	**RR (95% CI)**	**Cases**	**RR (95% CI)**	**Cases**
Age group†						
25–34‡	1.00	242 903	1.00	312 549	1.00	555 497
35–44	1.08 (0.93–1.26)	329 047	1.15 (1.10–1.32)§	453 461	1.12 (1.04–1.24)§	782 508
45–54	1.37 (1.17–1.60)§	410 160	1.32 (1.14–1.53)§	474 471	1.34 (1.20–1.49)§	884 630
55–64	1.52 (1.32–1.74)§	333 358	1.43 (1.24–1.64)§	388 590	1.46 (1.32–1.62)§	721 948
Sex‖						
Male‡	–	–	–	–	1.00	1 629 116
Female	–	–	–	–	0.84 (0.78–0.91)§	1 315 467
Fruit & veg‖,#						
< 5 times/day	1.15 (0.78–1.69)	423 385	1.22 (0.78–1.91)	667 688	1.19 (0.88–1.59)	1 091 073
5–10 times/day	1.02 (0.69–1.52)	317 615	0.98 (0.62–1.55)	246 134	1.00 (0.74–1.35)	563 749
> 10 times/day‡	1.00	29 008	1.00	20 571	1.00	49 579
Physical activity‖,#						
Active‡	1.00	181 269	1.00	303 724	1.00	484 992
Moderately active	1.39 (1.16–1.66)§	300 577	1.23 (1.05–1.45)§	383 320	1.29 (1.14–1.45)§	683 896
Inactive	2.01 (1.72–2.36)§	823 720	1.48 (1.29–1.70)§	916 999	1.68 (1.52–1.87)§	1 740 719
Immigration‖,#						
≤ 9 years‡	1.00	39 324	1.00	48 298	1.00	
≥ 10 years	1.47 (0.90–2.41)	173 104	1.29 (0.75–2.21)	179 361	1.37 (0.95–1.99)	87 622
Non-immigrant	1.98 (1.24–3.15)§	1 103 039	2.09 (1.26–3.47)§	1 401 458	2.04 (1.44–2.89)§	352 465
Culture/race‖,#						
Visible minority‡	1.00	160 140	1.00	180 998	1.00	341 138
White	1.35 (1.12–1.64)§	1 138 427	1.53 (1.25–3.47)§	1 410 287	1.45 (1.26–1.66)§	2 548 713
Education‖,#						
Less than secondary school graduation	1.69 (1.44–1.99)§	108 941	1.15 (0.96–1.38)	85 064	1.40 (1.24–1.58)§	194 005
Secondary school graduation	1.42 (1.21–1.66)§	154 345	1.18 (1.02–1.37)§	166 622	1.29 (1.15–1.43)§	320 967
Some post-secondary school	1.53 (1.25–1.87)§	90 428	1.15 (0.93–1.43)	88 776	1.32 (1.14–1.52)§	179 204
Post-secondary graduation‡	1.00	883 344	1.00	1 132 957	1.00	2 016 301
Household income‖,#						
< $15,000	1.95 (1.59–2.39)§	89 606	0.92 (0.73–1.15)	51 960	1.33 (1.15–1.54)§	141 566
$15-29,999	1.72 (1.44–2.06)§	158 826	0.98 (0.82–1.17)	106 427	1.27 (1.13–1.44)§	265 253
$30-49,999	1.69 (1.42–2.00)§	288 184	0.97 (0.84–1.12)	248 683	1.23 (1.11–1.37)§	536 866
$50-79,999	1.39 (1.18–1.64)§	337 446	1.10 (0.97–1.25)	457 773	1.20 (1.08–1.32)§	795 219
≥ $80,000‡	1.00	301 465	1.00	597 414	1.00	898 879
Food security‖,#						
Food secure‡	1.00	972 905	1.00	1 286 838	1.00	2 259 743
Insecure-no hunger	1.76 (1.40–2.22)§	67 940	0.98 (0.70–1.38)	33 550	1.39 (1.15–1.69)§	101 490
Insecure-moderate	1.99 (1.52–2.60)§	40 183	1.00 (0.68–1.48)	15 296	1.56 (1.24–1.95) §	55 479
Insecure-severe	1.73 (0.90–3.33)	7 858	1.11 (0.57–2.15)	6 312	1.39 (0.88–2.19)	14 170

* BMI ≥ 30
† adjusted for sex for "Both sexes" category only
‡ reference group
‖ adjusted for age
adjusted for age and sex for "Both sexes" category only
§ significant at $p < 0.002$

Abbreviations: BMI, body mass index; CI, confidence interval; RR, rate ratio

TABLE 2b
Poisson regression analysis, overweight/obese* adults (25-64 years), Canadian Community Health Survey, 2005

	Women		Men		Both sexes	
Predictor	**RR (95% CI)**	**Cases**	**RR (95% CI)**	**Cases**	**RR (95% CI)**	**Cases**
Age group†						
25–34‡	1.00	637 572	1.00	1 090 256	1.00	1 727 828
35–44	1.15 (1.06–1.25)§	922 299	1.15 (1.09–1.22)§	1 585 951	1.15 (1.10–1.21)§	2 508 250
45–54	1.38 (1.27–1.50)§	1 080 994	1.21 (1.14–1.28)§	1 511 101	1.26 (1.20–1.32)§	2 592 095
55–64	1.65 (1.54–1.78)§	955 247	1.25 (1.18–1.32)§	1 187 170	1.39 (1.33–1.46)§	2 142 417
Sex‖						
Male‡	–	–	–	–	1.00	5 374 478
Female	–	–	–	–	0.70 (0.67–0.72)§	3 596 112
Fruit & veg‖,#						
< 5 times/day	1.02 (0.84–1.25)	1 139 785	1.09 (0.91–1.29)	2 154 975	1.05 (0.92–1.20)	3 294 758
5–10 times/day	0.97 (0.80–1.19)	913 057	1.04 (0.87–1.24)	940 696	1.00 (0.87–1.14)	1 853 753
> 10 times/day‡	1.00	87 329	1.00	74 918	1.00	162 246
Physical activity‖,#						
Active‡	1.00	647 576	1.00	1 268 731	1.00	1 916 308
Moderately active	1.18 (1.08–1.28)§	910 129	1.04 (0.99–1.10)	1 351 167	1.09 (1.04–1.14)§	2 261 296
Inactive	1.37 (1.27–1.48)§	2 009 411	1.04 (0.99–1.09)	2 679 689	1.15 (1.11–1.20)§	4 689 100
Immigration‖,#						
≤ 9 years‡	1.00	145 252	1.00	249 879	1.00	395 130
≥ 10 years	1.21 (0.97–1.50)	533 770	1.13 (0.97–1.31)	776 447	1.16 (1.02–1.31)§	1 310 217
Non-immigrant	1.40 (1.15–1.71)§	2 917 090	1.29 (1.13–1.48)§	4 348 153	1.33 (1.19–1.49)§	7 265 242
Culture/race‖,#						
Visible minority‡	1.00	475 260	1.00	710 468	1.00	1 185 728
White	1.21 (1.10–1.34)§	3 049 991	1.27 (1.18–1.37)§	4 534 480	1.25 (1.17–1.33)§	7 584 470
Education‖,#						
Less than secondary school graduation	1.36 (1.25–1.47)§	255 474	0.98 (0.91–1.05)	241 266	1.15 (1.09–1.21)§	496 740
Secondary school graduation	1.21 (1.12–1.31)§	379 298	1.03 (0.97–1.10)	495 401	1.11 (1.05–1.16)§	874 699
Some post-secondary school	1.23 (1.10–1.37)§	207 534	0.99 (0.90–1.08)	260 348	1.08 (1.01–1.16)§	467 882
Post-secondary graduation‡	1.00	2 524 264	1.00	3 874 291	1.00	6 398 556
Household income‖,#						
< $15,000	1.27 (1.14–1.43)§	195 229	0.76 (0.68–0.86)§	154 021	0.97 (0.89–1.05)	349 250
$15-29,999	1.21 (1.10–1.33)§	371 437	0.79 (0.73–0.87)§	309 627	0.96 (0.90–1.02)	681 064
$30-49,999	1.25 (1.15–1.36)§	706 962	0.86 (0.81–0.92)§	792 320	1.00 (0.95–1.05)	1 499 283
$50-79,999	1.15 (1.06–1.24)§	911 915	0.97 (0.92–1.01)	1 444 676	1.02 (0.98–1.07)	2 356 590
≥ $80,000‡	1.00	985 790	1.00	2 140 754	1.00	3 126 544
Food security‖,#						
Food secure‡	1.00	2 782 467	1.00	4 341 777	1.00	7 124 244
Insecure-no hunger	1.31 (1.15–1.49)§	145 053	0.94 (0.81–1.08)	110 069	1.12 (1.01–1.23)§	255 122
Insecure-moderate	1.45 (1.25–1.68)§	83 672	0.90 (0.75–1.07)	46 576	1.18 (1.05–1.33)§	130 248
Insecure-severe	1.39 (1.01–1.93)§	17 943	0.85 (0.63–1.13)	16 371	1.07 (0.84–1.35)	34 314

* BMI ≥ 25
† adjusted for sex for "Both sexes" category only
‡ reference group
‖ adjusted for age
adjusted for age and sex for "Both sexes" category only
§ significant at $p < 0.002$

Abbreviations: BMI, body mass index; CI, confidence interval; RR, rate ratio

FIGURE 1a
Adult obesity (BMI ≥ 30) prevalence by health region, age and sex standardized to the 2005 Canadian population (25-64 years), Canadian Community Health Survey, 2005

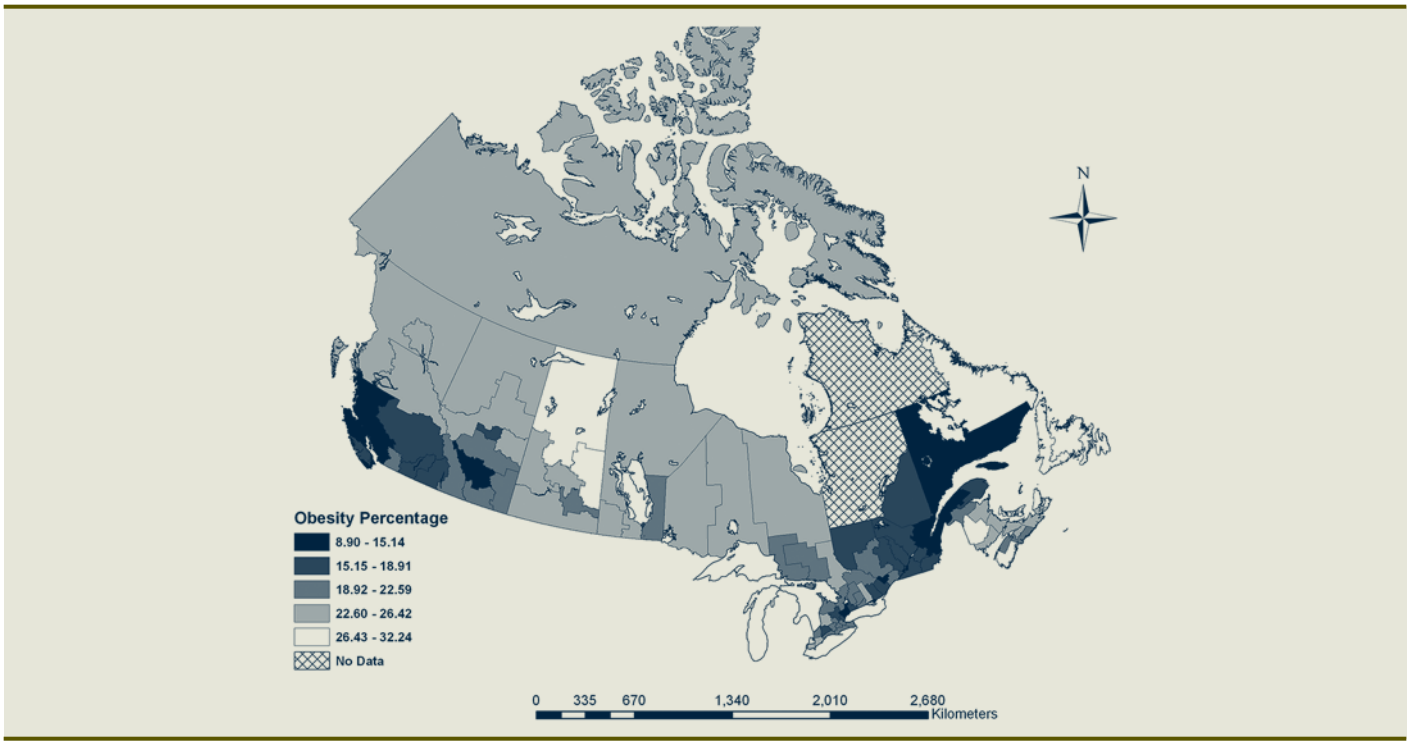


FIGURE 1b
Adult overweight/obesity (BMI ≥ 25) prevalence by health region, age and sex standardized to the 2005 Canadian population (25-64 years), Canadian Community Health Survey, 2005

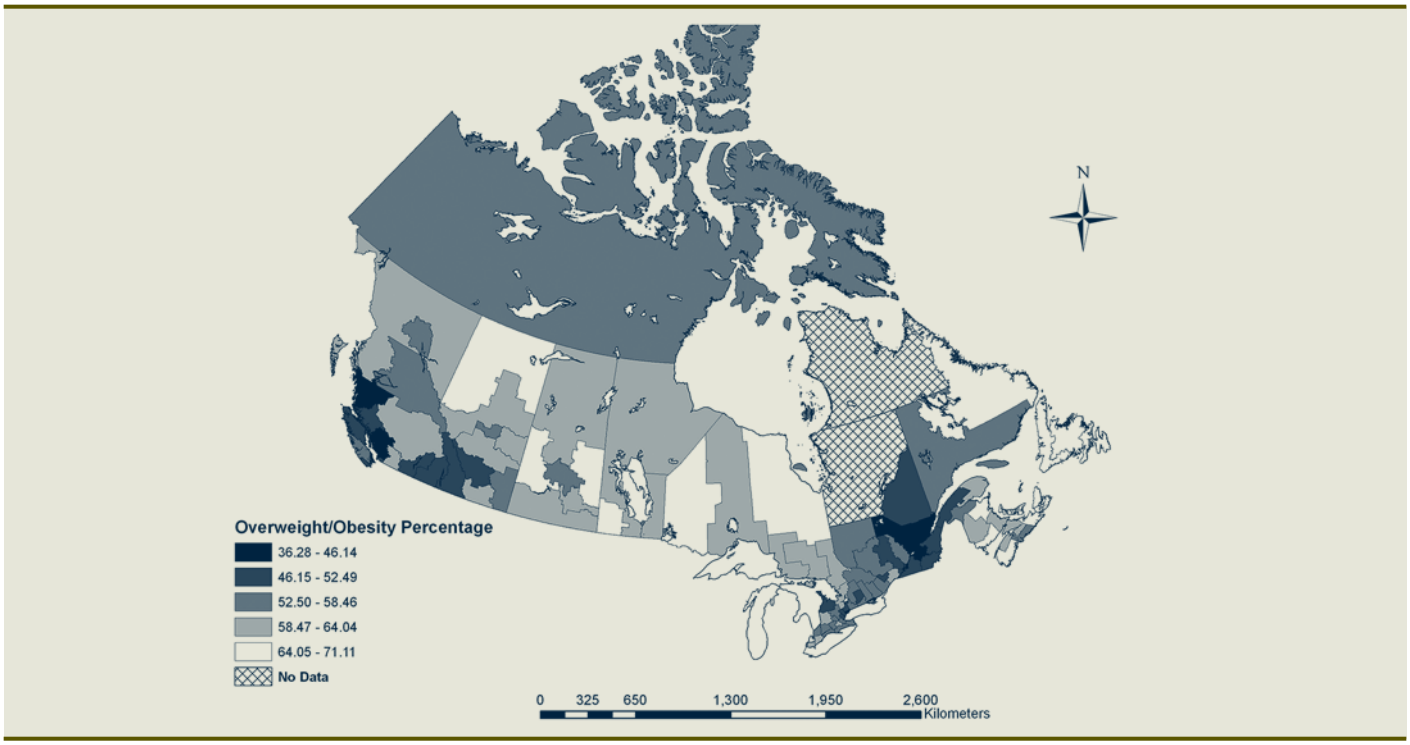


TABLE 3
Gini coefficient analysis for adult (25–64 years) obese* and overweight/obese† prevalence, Canadian Community Health Survey, 2005

	OBESE			OVERWEIGHT/OBESE		
	Women (95% CI)	Men (95% CI)	Both sexes (95% CI)	Women (95% CI)	Men (95% CI)	Both sexes (95% CI)
Immigration	0.060 (0.000–0.160)	0.078 (0.000–0.170)	0.070 (0.000–0.167)	0.032 (0.000–0.107)	0.026 (0.000–0.065)	0.029 (0.000–0.082)
Education	0.086 (0.003–0.169)	0.030 (0.000–0.064)	0.055 (0.000–0.113)	0.049 (0.000–0.102)	0.004 (0.000–0.016)	0.022 (0.001–0.043)
Income	0.129 (0.011–0.247)	0.026 (0.000–0.057)	0.052 (0.000–0.104)	0.056 (0.006–0.106)	0.040 (0.003–0.077)	0.011 (0.000–0.024)
Geography	0.169 (0.134–0.204)	0.156 (0.105–0.207)	0.153 (0.120–0.186)	0.094 (0.069–0.119)	0.066 (0.043–0.089)	0.076 (0.053–0.099)

* BMI ≥ 30
† BMI ≥ 25

Abbreviations: BMI, body mass index; CI, confidence interval

TABLE 4
Potential impact of obesity* prevention scenarios: high risk vs. population-based

	Geographic (health region) analysis	Female income analysis
No prevention (from CCHS)		
Observed cases	2 944 583	1 315 467
Observed rate	17.3/100	15.8/100
Prevention scenarios		
High risk†		
Cases prevented	265 012	85 505
Cases remaining	2 679 571	1 229 961
Rate	15.8/100	14.8/100
% decrease in rate	9%	6.5%
Population-based‡		
Cases prevented	1 064 341	333 040
Cases remaining	1 880 242	982 426
Rate	11.1/100	11.8/100
% decrease in rate	36.1%	25.3%

* BMI ≥ 30
† Preventing 50% of obesity cases in 10% of population at highest risk
‡ Reducing obesity prevalence of all population groups to the level of lowest risk group

0.153, only 18% of the cases of obesity are contained in the 10% of the geographically defined population having the highest risk of obesity; in the case of income, with a Gini coefficient of 0.129, only 13% of the cases of obesity are contained in the 10% of the income classified population at highest risk of obesity.

Recalculating obesity rates under the different prevention scenarios showed that the population-based scenarios could achieve a four-fold greater decrease in obesity cases than the high risk scenario. As illustrated in Table 4, the population prevention approach led to a decrease of 1 064 341 and 330 040 cases in the geographic and female income scenarios respectively, compared to 265 012 and 85 505 cases in the high risk prevention scenarios. These translated into only modest decreases in prevalence for the high risk scenarios (geographic: 17.3% to 15.8%; female income: 15.8% to 14.8%), with more substantial decreases observed for the population prevention scenarios (health region: 17.3% to 11.1%; female income: 15.8% to 11.8%).

Discussion

In this study we demonstrated that there is significant variability in rates of overweight and obesity across geographic and socio-demographic groupings. Age, physical inactivity, income, education, non-immigrant status, white racial status and moderate food insecurity predicted varying degrees of overweight and obesity in both men and women. The lowest rates of overweight and obesity were observed in major urban centers.

For both men and women increasing age was a strong predictor of higher rates of both overweight and obesity. This is not surprising given that metabolism slows with advancing age, increasing risk of weight gain.[20,21] What is notable, however, are the dramatically higher rates of obesity and overweight in younger age groups than previously reported.[3,22] Katzmarzyk has also demonstrated that there are now more men and women moving into the highest classes of obesity, i.e. class II (BMI = 35–39.99) or class III (BMI ≥ 40) than before,[23] suggesting that Canadians are experiencing an accelerated weight gain in younger ages, a conclusion supported by the increasingly high rates of childhood obesity.[2]

Although household income was a strong predictor of overweight and obesity for women, with the highest rates of obesity and overweight among those in the lowest income quartile, low income for men was not associated with high rates of overweight and obesity and appeared to be protective. The reason for this is not clear and requires further study.

Food insecurity was also predictive for overweight and obesity, but only for women. Food insecurity is directly related to low income,[24] and the situation for poor women is frequently exacerbated by being

FIGURE 2a
Gini coefficient, adult (males and females, 25-64 years) obesity (BMI ≥ 30) by health region

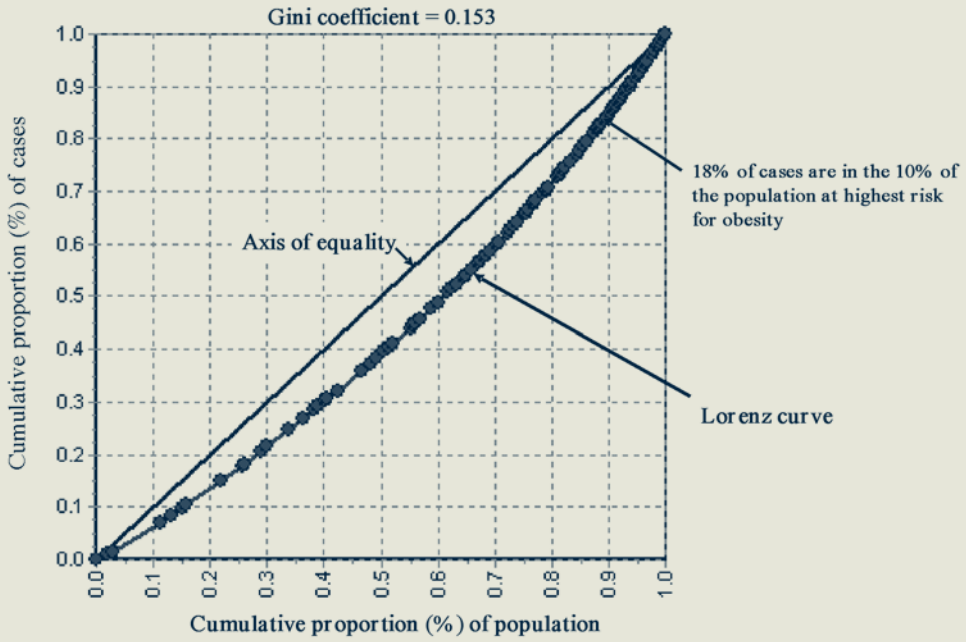
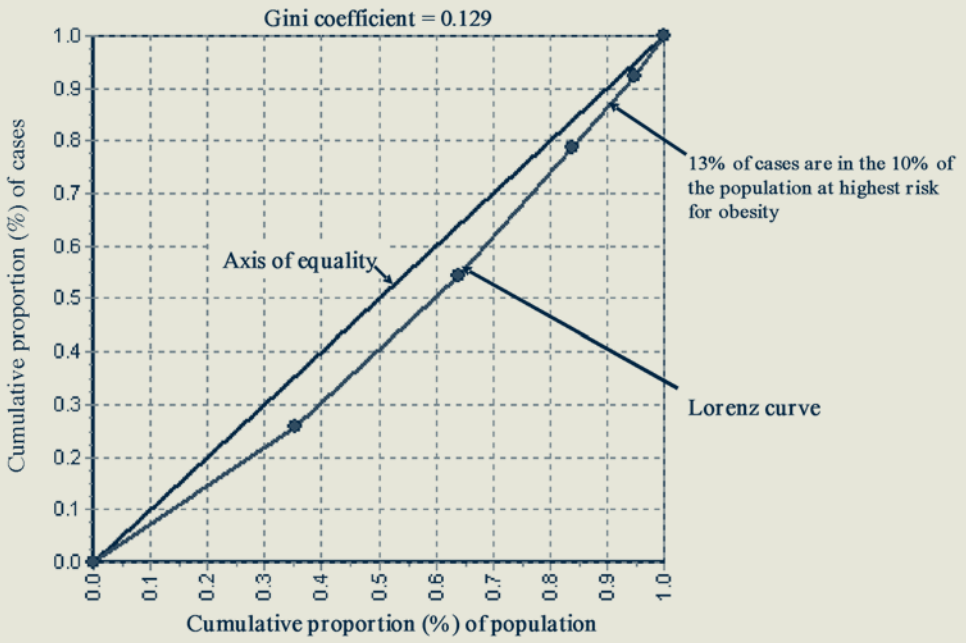


FIGURE 2b
Gini coefficient, adult (females 25-64 years) obesity (BMI ≥ 30), by income



a single parent. Paradoxically, this has been shown to increase BMI. Low income and food insecurity may lead to a reliance on food assistance, such as food banks, where food procured is often energy-dense and of low nutritional quality, which contributes to overweight, obesity and other poor health outcomes.[25]

Low education was predictive of overweight and obesity only for women. This may be linked with low income, as women with low education levels earn significantly less money than men with similar education levels.[26]

Recent immigrant status appeared to be protective against both overweight and obesity. This may be because many new arrivals come from countries with low levels of overweight and obesity. For example, 57.7% % of new arrivals to Canada in 2001 were from Asian countries just entering the "nutrition transition,"[27] a phenomenon where countries rapidly adopt a Western-style diet with high levels of energy-dense animal-source and processed foods.[28] These transitional countries are currently experiencing rapid increases in population BMI, but have not reached the current Canadian levels. This immigration pattern may explain the low rates of overweight and obesity observed in the major urban centres of Vancouver and the lower mainland of British Columbia, Calgary, Toronto, Ottawa and Montreal, where the majority of new arrivals to Canada (80% in 2001) settle.[27]

Visible minority status was also a significant predictor of low BMI. This contradicts findings which show that black and Hispanic populations in the United States are at higher risk of overweight and obesity.[29] This may be because recent non-white immigrants to Canada have lower BMIs and foodways that pre-date the nutrition transition in their countries of origin. Black and Hispanic populations have resided in the U.S. for much longer and tend to experience higher rates of poverty.

Not unexpectedly, low rates of physical activity were also predictive of overweight and obesity for both genders, since sedentary behaviour is associated with weight gain.[30,31] However, low consumption of fruits and vegetables was not associated with higher BMI. Further studies are required to determine the role of fruits and vegetables in maintaining body weight.

Although this study has demonstrated variations across socio-demographic groupings and geography, the Gini analysis suggests these differences are less significant than they may initially appear. While there may be pockets of more susceptible groups with very high rates of obesity and overweight, the high rates across all socio-demographic and geographic groupings suggest that the "causes" of overweight and obesity are also widely dispersed, affecting all population groups. This observation is consistent with Rose's population approach.[32] Rose argued it is imperative to know how much of the burden of ill-health (i.e. absolute number of cases) is compressed within an identifiable group where increased exposure carries increased personal risk. If the burden is not highly concentrated in identifiable high risk sub-populations which are small in size (resulting in a low Gini coefficient), then a high risk targeted prevention approach will do little to affect the population prevalence of the health issue since most cases are outside the high risk group. Our study results empirically confirmed that this is the case currently in Canada. Prevention scenarios focusing on high-risk populations, even if successful (i.e. 50% of cases prevented), would decrease the population prevalence of obesity much less than broad population-based approaches. The lack of clear and often paradoxical patterns of obesity observed (e.g. high rates of overweight/obesity in high income men) also supports the notion that prevention programs focusing on identifiable high risk groups (e.g. low income) will likely fail to achieve significant decreases in the population prevalence of obesity.

Looking at trends over time may be a useful adjunct to the study of cross-sectional variability (as was undertaken in this study) for understanding the dynamics of the obesity epidemic and identifying opportunities for intervention. Rates of overweight and obesity were high in all sub-populations and significantly higher than previously observed. These significant upward temporal trends in obesity prevalence suggest that there are strong forces driving rates upward in almost all population groups. This is not surprising, considering the obesogenic environment that has emerged in Canada over the past several decades which encourages overeating and minimizes opportunities for physical activity.[33-35] It has become very easy to consume excess kilocalories through convenience, snack and fast foods, including soft drinks. These food products tend to be low in fibre and high in sodium, sugar and fat,[36,37] and the resultant excess energy intake, coupled with decreased activity, provides the right circumstances to promote overweight and obesity.[38]

Although the results of this study are consistent with others' which have shown moderately graded relationships between socio-demographic predictors and obesity/overweight,[39-42] when interpreted from this wider perspective, they cast doubt on the importance that has been placed on high risk sub-populations and poor lifestyle choices as major explanations for overweight, obesity and related chronic diseases.[43-46] These analyses highlight why it is important to move beyond statistically significant risk differences in population-based health surveys in order to generate policy and program relevant insights into prevention approaches. As the results of this study have shown, examining the degree to which cases are distributed across risk groups using tools such as the Gini coefficient and prevention scenarios can facilitate an analysis of whether proposed prevention efforts could realistically achieve their goals over time.

These study results bring into question the emphasis on intervention strategies targeted to high risk individuals and groups. They suggest alternatively that prevention efforts should focus on the emerging obesogenic environment which affects all population groups. This perspective does not invalidate or deny that some identifiable social groups (i.e. low income women) are at elevated risk of obesity; however, it argues that the primary cause of the obesity epidemic is the obesogenic environment to which all population groups are exposed, with some populations being

more vulnerable for various reasons. This reframing of the disparities argument, supported empirically by the results of this study, strongly suggests that a population-wide approach to prevention of overweight and obesity is warranted. Further research is required to explore effective program and policy mechanisms.

This study has a number of limitations. First, the research was conducted using cross-sectional data and does not model factors over time. Cross-sectional studies may be blind to significant etiological factors which may have developed over time. The Gini analysis, however, highlighted the limitations of exclusively cross-sectional predictors and explanations for the obesity epidemic.

Second, the CCHS data set has a limited number of socio-demographic variables available for analysis. There may be other important predictors of elevated BMI such as family structure, neighbourhood characteristics or work status which were not covered by the survey.

Third, self-reported height and weight were used with the result that the reported rates of obesity and overweight were most likely underestimated.[47,48] For the 2005 CCHS, self-reported versus measured height and weight underestimated BMI by 1.3 kg/m^2;[49] however, as indicated, the measured height and weight sample was too small to allow the predictive modeling and geographic analysis used by the study.

A fourth limitation was the size of the geographic areas used in the analyses. This may have masked variability in overweight and obesity rates within geographic areas. However, this problem was unavoidable as the samples in the 2005 CCHS were too small to allow defensible small-area parameter estimates. Other studies have used even larger areas, conducting geographic analysis of BMI at the provincial level.[41,50]

A fifth limitation is that the 2005 CCHS does not contain information for the on-reserve Aboriginal population, a group shown to be at high risk for obesity.[51] This omission may have biased the geographic patterns observed, especially in northern areas of Canada where there are a large number of reserve communities.

Finally, the assumptions underlying the high risk and population-based scenarios created in this study were likely overly optimistic. It is unlikely that a prevention strategy focusing only on high risk populations would be able to achieve a 50% reduction in obesity prevalence given the obesogenic environment in which high risk groups (and most of the Canadian population) live. Two key influences on body weight, the food system and the built environment, are difficult to modify at the level of the individual or local community, and initiatives which have attempted to do so have had limited success in decreasing BMI.[52] Even in the population-based scenario, which assumed that all population groups could obtain the prevalence rates already being achieved in the lowest rate geographic and demographic groups, a "scaling up" of preventive processes for the entire Canadian population would be necessary, which would require significant political will to address broader determinants of elevated BMI. The implication is that within the short term the effectiveness of the high risk and the population-based prevention scenarios would likely be less than reported in this study. However, our scenario assumptions may become more realistic over the long term if governments begin to make a serious and concerted effort to prevent obesity, although we do not know what outcomes would actually be possible. No country to date has ever implemented a serious and successful obesity prevention strategy, with most efforts restricted to social marketing campaigns, labelling regulations and voluntary adoption of dietary guidelines by schools and other institutions.[5,6] We believe that these scenarios can provide strategic guidance to policy processes by quantifying the constraints impacting the potential success of alternative prevention initiatives. In addition, these scenarios provide a basis from which to critique studies which seek to highlight the importance of sub-populations at statistically higher risk of obesity, without also examining the population distribution of obesity and the implication this may have for successful prevention efforts.

A potential criticism of the scenarios in this paper is that they assume an overly modest prevention effect for the high risk scenarios, with the effect of stacking the analysis in favour of the population-based prevention approaches. However, as illustrated in Table 4, the high risk scenarios assume a much greater prevention success rate (50% decrease in cases) than do either of the population-based approaches (36.1% and 25.3%) respectively. If we recalculated the analysis so that the high risk scenarios had the same prevention success as the population based prevention approaches, the high risk approaches would perform even more poorly in comparison to the population based prevention approaches.

Despite the limitations, this research has highlighted the need to examine the implications of different prevention approaches through methods that go beyond merely establishing statistical significance between risk groups. The Gini analysis and the prevention scenarios used in this study have been shown to be a useful heuristic for exploring the potential impacts of different prevention approaches and for empirically demonstrating which approaches could make a difference over the long-term, and which would be less likely to do so.

The study highlights that further research is required to understand the dimensions of this obesogenic environment and how these have evolved over time to impact the body weight of Canadians. This will require committed, comprehensive surveillance of the Canadian diet and physical activity levels, and on-going surveys of population weight status using measured height and weight. This research will be important in building the empirical argument for population-based preventions efforts in order to turn the page on this insalubrious phase in Canadian public health.

References

1. Tjepkema M. Canadian Community Health Survey. Measured obesity: Adult obesity in Canada. Ottawa (ON): Statistics Canada; 2005. Available from: http://www.statcan.gc.ca/pub/82-620-m/2005001/pdf/4224906-eng.pdf

2. Shields M. Measured obesity: overweight Canadian children and adolescents. Ottawa (ON): Statistics Canada; 2005. (Canadian Community Health Survey; issue no. 1). Report No.: 82-620-MWE2005001.

3. Tremblay MS, Katzmarzyk PT, Willms JD. Temporal trends in overweight and obesity in Canada, 1981-1996. Int J Obes Relat Metab Disord. 2002 Apr;26(4):538-43.

4. Katzmarzyk PT, Ardern CI. Overweight and obesity mortality trends in Canada, 1985-2000. Can J Public Health. 2004 Jan-Feb;95(1):16-20.

5. Health Canada. Obesity: it's your health [Internet]. Ottawa (ON): Health Canada; 2006 [cited 2008 Aug 1]. Available from: URL: http://www.hc-sc.gc.ca/hl-vs/iyh-vsv/life-vie/obes-eng.php#ro

6. Public Health Agency of Canada. Childhood obesity: Government of Canada's role [Internet]. Ottawa (ON): Public Health Agency of Canada; c2007 [modified 2007 Aug 21; cited 2008 Aug 1]. Available from: URL: http://www.phac-aspc.gc.ca/ch-se/obesity/obesitybck-eng.php

7. Doak C. Large-scale interventions and programmes addressing nutrition-related chronic diseases and obesity: examples from 14 countries. Public Health Nutr. 2002 Feb;5(1A):275-7.

8. Statistics Canada. Data liberation initiative (DLI) [Internet]. Ottawa (ON): Statistics Canada;2007 [modified 2009 Jun 1; cited 2008 Aug 1]. Available from: http://www.statcan.ca/english/Dli/dli.htm

9. Statistics Canada. Canadian community health survey (CCHS) cycle 3.1 (2005): Public use microdata file (PUMF) user guide. Ottawa (ON): Statistics Canada;2006.

10. Statistics Canada. Health region boundary files [Internet]. Ottawa (ON): Statistics Canada; c2006. [modified 2006 Apr 13; cited 2008 Aug 1]. Available from: http://www.statcan.ca/english/freepub/82-402-XIE/2006001/region.htm

11. ArcGIS [computer program]. Version 9.1. Redlands (CA): ESRI; 2003.

12. Stata statistical software [computer program]. Release 9. College Station (TX): StataCorp LP; 2005.

13. Napier University (UK); National Centre for Social Research (UK). Practical exemplars and survey analysis [Internet]. Economic and Social Research Council (UK); 2006 [cited 2007 Jan 7]. Available from: http://www2.napier.ac.uk/depts/fhls/peas/about.asp

14. Zocchetti C, Consonni D, Bertazzi PA. Relationship between prevalence rate ratios and odds ratios in cross-sectional studies. Int J Epidemiol. 1997 Feb;26(1):220-3.

15. Barros AJ, Hirakata VN. Alternatives for logistic regression in cross-sectional studies: an empirical comparison of models that directly estimate the prevalence ratio. BMC Med Res Methodol. 2003 Oct 20;3:21.

16. Castillo-Salgado C, Schneider M, Loyola E, Mujica O, Roca A, Yerg T. Measuring health inequalities: Gini coefficient and concentration index. Epidemiol Bull. 2001;22:3-4.

17. Elliott LJ, Blanchard JF, Beaudoin CM, Green CG, Nowicki DL, Matusko P, Moses S. Geographical variations in the epidemiology of bacterial sexually transmitted infections in Manitoba, Canada. Sex Transm Infect. 2002 Apr;78 Suppl 1:i139-i144.

18. Green CG, Krause DO, Wylie JL. Spatial analysis of campylobacter infection in the Canadian province of Manitoba. Int J Health Geogr. 2006 Jan;5:2.

19. EpiDat [computer program]. Version 3. Washington (DC): Pan American Health Organization; 2000.

20. Williams PT. Evidence for the incompatibility of age-neutral overweight and age-neutral physical activity standards from runners. Am J Clin Nutr. 1997 May;65(5):1391-6.

21. Lobo RA. Metabolic syndrome after menopause and the role of hormones. Maturitas. 2008 May 20;60(1):10-8.

22. Katzmarzyk PT. The Canadian obesity epidemic: an historical perspective. Obesity Research 2002;10(7):666-74.

23. Katzmarzyk PT, Mason C. Prevalence of class I, II and III obesity in Canada. CMAJ. 2006 Jan 17;174(2):156-7.

24. Che J, Chen J. Food insecurity in Canadian households. Health Rep. 2001 Aug;12(4):11-22.

25. Vozoris NT, Tarasuk VS. Household food insufficiency is associated with poorer health. J Nutr. 2003 Jan;133(1):120-6.

26. O'Donnell V, Almey M, Lindsay C, Fournier-Savard P, Mihorean K, Charmant M, Taylor-Butts A, Johnson S, Pottie-Bunge V, Aston C. Women in Canada: a gender-based statistical report. Ottawa (ON); Statistics Canada; 2006 Mar 7. Catalogue no.: 89-503-XPE. English.

27. Schellenberg G. Immigrants in Canada's census metropolitan areas. Ottawa (ON): Statistics Canada; 2004. Catalogue no.: 89-613-MIE, No. 003.

28. Popkin BM, Gordon-Larsen P. The nutrition transition: worldwide obesity dynamics and their determinants. Int J Obes Relat Metab Disord. 2004 Nov;28 Suppl 3:S2-S9.

29. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. JAMA. 2004 Jun 16;291(23):2847-50.

30. Anderssen SA, Engeland A, Søgaard AJ, Nystad W, Graff-Iversen S, Holme I. Changes in physical activity behavior and the development of body mass index during the last 30 years in Norway. Scand J Med Sci Sports. 2008 Jun;18(3):309-17.

31. Shields M, Tremblay MS. Sedentary behaviour and obesity. Health Rep. 2008 Jun;19(2):19-30.

32. Rose G. The strategy of preventive medicine. Oxford: Oxford University Press; 1994.

33. Frank LD, Engelke PO, Schmid TL. Health and community design the impact of the built environment on physical activity. Washington, DC: Island Press; 2003.

34. Nestle M. Food politics: how the food industry influences nutrition and health. Berkeley: University of California Press, Ltd.; 2002.

35. Winson A. Bringing political economy into the debate on the obesity epidemic. Agric Human Values. 2004;21:299-312.

36. Garriguet D. Canadians' eating habits. Health Rep. 2007 May;18(2):17-32.

37. Paeratakul S, Ferdinand DP, Champagne CM, Ryan DH, Bray GA. Fast-food consumption among US adults and children: dietary and nutrient intake profile. J Am Diet Assoc. 2003 Oct;103(10):1332-8.

38. Slater J, Green C, Sevenhuysen G, O'Neil J, Edginton B, Heasman M. The growing Canadian energy gap: more the can than the couch? Public Health Nutrition. 2009. 12(11):2216-2224.

39. McLaren L. Socioeconomic status and obesity. Epidemiol Rev. 2007;29:29-48.

40. Ward H, Tarasuk V, Mendelson R. Socioeconomic patterns of obesity in Canada: modeling the role of health behaviour. Appl Physiol Nutr Metab. 2007 Apr;32(2):206-16.

41. Willms JD, Tremblay MS, Katzmarzyk PT. Geographic and demographic variation in the prevalence of overweight Canadian children. Obes Res. 2003 May;11(5):668-73.

42. Oliver LN, Hayes MV. Neighbourhood socio-economic status and the prevalence of overweight Canadian children and youth. Can J Public Health. 2005 Nov-Dec;96(6):415-20.

43. Nader PR, O'Brien M, Houts R, Bradley R, Belsky J, Crosnoe R, Friedman S, Mei Z, Susman EJ; National Institute of Child Health and Human Development Early Child Care Research Network. Identifying risk for obesity in early childhood. Pediatrics. 2006 Sep;118(3):e594-e601.

44. Dubois L, Girard M. Early determinants of overweight at 4.5 years in a population-based longitudinal study. Int J Obes (Lond). 2006 Apr;30(4):610-7.

45. Oliver LN, Hayes MV. Effects of neighbourhood income on reported body mass index: an eight year longitudinal study of Canadian children. BMC Public Health. 2008;8:16.

46. Butler-Jones D. The Chief Public Health Officer's report on the state of public health in Canada, 2008: addressing health inequalities = Rapport de L'administrateur en chef de la santé publique sur l'état de la santé publique au Canada. Ottawa (ON): Public Health Agency of Canada; 2008.

47. MacLellan DL, Taylor RD, Van Til L, Sweet L. Measured weights in PEI adults reveal higher than expected obesity rates. Can J Public Health. 2004 May-Jun;95(3):174-8.

48. Tremblay M. The need for directly measured health data in Canada. Can J Public Health. 2004 May-Jun;95(3):165-8.

49. Shields M, Gorber SC, Tremblay MS. Effects of measurement on obesity and morbidity. Health Rep. 2008;19(2):77-84.

50. Katzmarzyk PT. The Canadian obesity epidemic, 1985-1998. CMAJ. 2002 Apr 16; 166(8):1039-40.

51. Hanley AJ, Harris SB, Gittelsohn J, Wolever TM, Saksvig B, Zinman B. Overweight among children and adolescents in a Native Canadian community: prevalence and associated factors. Am J Clin Nutr. 2000 Mar 71:693-700.

52. Susser M. The tribulations of trials – Intervention in communities. Am J Public Health. 1995 Feb 85(2):156-8.

Using cancer registry data: agreement in cause-of-death data between the Ontario Cancer Registry and a longitudinal study of breast cancer patients

D. R. Brenner, MSc (1,2); M. C. Tammemägi, PhD (3); S. B. Bull, PhD (1,2); D. Pinnaduwaje, PhD (1); I. L. Andrulis, PhD (1,4)

Abstract

Data from the Ontario Cancer Registry (OCR) were compared with data from a multi-centred prospective cohort of 1655 node-negative breast cancer patients with intensive clinical follow-up. Agreement in cause of death was evaluated using kappa statistics. The accuracy of OCR classification was evaluated against the Mount Sinai Hospital (MSH) study oncologist's interpretation of intensely followed, cohort-collected data as the reference standard. The two sources showed a high level of agreement (kappa statistic [κ] = 0.88; 95% confidence interval [CI]: 0.86, 0.90) in vital status and cause of death. Among those cases where both sources reported a death, the OCR had a sensitivity of 95% (95% CI: 90.5, 98.8) and a specificity of 88% (95% CI: 79.6, 92.4). The OCR is a valuable tool for epidemiologic studies of breast cancer to acquire adequate and easily attainable cause-of-death information.

Keywords: *epidemiology methods, data collection, data registries, vital statistics, breast neoplasms, cause of death, Ontario Cancer Registry*

Introduction

The use of cancer registry mortality and follow-up data in epidemiologic studies is common.[1] However, it is unclear to what extent bias may be introduced because of incomplete or inaccurate cause-of-death data in the registries.[2,3]

The Ontario Cancer Registry (OCR), maintained and operated by Cancer Care Ontario since 1964, collects vital information on all new cases of cancer in the province except for non-melanoma skin cancers. Validation studies have shown the registry to be effective in ascertaining cancer cases in the province (98% sensitivity).[4] The registry collects data from pathology reports, patient records, hospital discharge records and death certificates from the Registrar General of Ontario. Probabilistic linkage is then used to reconcile the data sources into a central database.[5] The registry performs regular internal data quality evaluations; however, registry data are rarely compared to actual detailed medical records and data collected from additional external sources. One comparative study of head-and-neck cancer outcomes reported that the OCR had excellent agreement in index tumour site assignment, vital status and date of death; however, there was a 31% error rate in cause of death (cancer vs. noncancer).[6] To our knowledge, no studies have examined agreement of cause-of-death data among breast cancer patients in the OCR with cause of death determined in an independent study with rigorous follow-up.

A multi-centred prospective cohort study based at Mount Sinai Hospital (MSH) in Toronto commenced patient enrollment in 1987. The MSH study collected incident cases of pathologically confirmed node-negative breast cancer from eight participating sites in the greater Toronto area. The study aim was to evaluate the associations between genetic and molecular tumour alterations and recurrence of disease and death due to breast cancer.[7] Study managers systematically collected data from hospital and medical records, patient interviews, pathology reports, patient charts, coroner reports and death certificates. The study oncologist, a specialist in breast cancer, made the final determination as to the classification of cause of death after examining the collected information.

The aim of our study was to evaluate the agreement between cause-of-death data from the Ontario Cancer Registry and the MSH study, which has regular and systematic patient monitoring and follow-up, and specialist-determined outcome based, for the most part, on relatively complete and accurate data. Other studies have found that specialist classification of cancer outcomes is more accurate than registry classification, possibly due to more extensive data availability or experience or both.[8] For these reasons, our study also evaluates the accuracy of OCR cause-of-death data using the MSH study data as the reference standard.

Author References

1 Samuel Lunenfeld Research Institute, Mount Sinai Hospital, Toronto, ON
2 Dalla Lana School of Public Health, University of Toronto, Toronto, ON
3 Department of Community Health Sciences Brock University, St. Catharines, ON
4 Department of Molecular Genetics, University of Toronto, Toronto, ON
Correspondence: Darren Brenner, The Prosserman Centre for Health Research, Samuel Lunenfeld Research Institute, 60 Murray St., Toronto, ON M5T 1X5, Tel.: 416-586-4800 ext. 8110, Fax: 416-586-5800, Email: brenner@lunenfeld.ca

Methods

OCR data were linked to MSH study patients according to OCR standard procedures based on probabilistic linkage using personal identifiers in the MSH study database. ICD-9* and ICD-10† codes described causes of death, and these were then classified as 1) due to breast cancer or 2) due to other/competing causes. Those individuals without cause-of-death information in both the MSH study and the OCR data were considered to be alive. The MSH study followed patients from the time of diagnosis and enrolment, from 1987 until the spring of 2005. Data from the OCR contained events that occurred up to 2006; however, data quality was only verified until the end of 2004. This created some discrepancy in the duration of follow-up. Any discrepant deaths were examined for date of death in order to address the discrepancy.

Kappa statistics were calculated to determine the agreement in cause-of-death classification between the two sources.[9] We calculated a weighted kappa with the rationale that a missed cancer-related death is of great importance to the MSH study and the OCR. Statistics were calculated using SAS® (SAS V9.1; SAS Institute Inc., Cary, NC) and all 95% confidence intervals (CI) were two-sided. Weighted kappa was determined using the default weighting scheme in SAS, based on the classification order displayed in Table 1. OCR classification accuracy was evaluated by estimating sensitivity and specificity using the MSH classification as the reference standard. The study was approved by the Mount Sinai Hospital Research Ethics Board.

Results

The study population consisted of the 1655 patients in the hospital-based study with no axillary nodal involvement at diagnosis (stage I [72%] and II [28%]). Of these patients, one did not have a record linkage match in the OCR (i.e. there was no information in the OCR), and six were marked as deceased in the OCR with no cause of death provided. These patients were excluded from analysis as the data provided no potential for comparison. Sensitivity analyses showed that misclassification of these deaths had little impact on the results: kappa statistics changed from 0.87 to 0.86 when all these patients were treated as still alive, and the percent agreement changed from 90.0% to 87.6% when all seven deaths were considered as errors.

The two sources showed a high level of agreement (kappa = 0.88; 95% CI: 0.86, 0.90) and a weighted kappa of 0.90 (95% CI: 0.80, 0.92). Of the 87 patients lost to follow-up by the MSH study, 11 had died and were located via the OCR. An additional 13 deaths not originally recorded by the MSH study were identified by the OCR, while 12 deaths recorded by the MSH Study were not identified by the OCR. These 12 discrepant deaths were checked for date of death; in three of the cases the deaths occurred in early 2005, but in the remaining nine cases the deaths occurred before 2000 and were yet to be picked up by the OCR. Table 1 shows the distribution of the causes of death provided by the study and the OCR.

Comparing the cases where both the OCR and the MSH study reported a death, the percent agreement on classification of death was 90.0% (Table 2, [(124 + 129)/281]). Using the MSH study data as the reference standard, the OCR had a sensitivity of 95% (95% CI: 90.5, 98.8) and a specificity of 88% (95% CI: 79.6, 92.4).

Discussion

These findings present several important points. First, the cause of death from the OCR abstraction and collection system strongly agreed with those from an intensively followed cohort study where cause

TABLE 1
Agreement between cause of death and vital status from cohort study and the Ontario Cancer Registry

		MSH study			
		No death info. or LTFU*	Competing cause of death	Breast cancer	Total
Ontario Cancer Registry	No death info.	1331	6	6	1343
	Competing cause of death	19	129	7	155
	Breast cancer death	5	21	124	150
	Total	**1355**	**156**	**137**	**1648**

Kappa
Simple: 0.87 (0.85, 0.90)
Weighted: 0.90 (0.88, 0.92)

Additional deaths provided by OCR

* LTFU – Lost to follow-up

TABLE 2
Comparison of cause of death between a cohort study with intensive follow-up and the Ontario Cancer Registry (MSH study cause of death assumed reference standard)

		MSH study (assumed reference standard)		
		Breast cancer death	Competing cause	Total
Ontario Cancer Registry	Breast cancer death	124	21	145
	Competing cause	7	129	136
Total		**131**	**150**	**281**

Sensitivity 95% (90.5-98.8)
Specificity 86% (79.6-92.4)

* International Statistical Classification of Diseases and Related Health Problems, 9th Revision.

† International Statistical Classification of Diseases and Related Health Problems, 10th Revision.

of death was determined by a medical oncologist. This indicates that OCR data may be useful in studies where patient follow-up is incomplete or not available; it also highlights the utility of the OCR for epidemiologic studies that are unable to acquire adequate clinician expertise for interpretation of cause of death. In these instances, particularly for studies of breast cancer, the OCR may be used as a relatively accurate and easily attainable source of cause of death. Second, there were several deaths that were missed by the MSH study, as patients were lost to follow-up. In these instances, the OCR collected deaths that, due to the province-wide coverage, enhanced the follow-up data of the MSH study. Third, our study found a high level of accuracy in the abstraction techniques of the OCR: we observed high sensitivity and specificity when the results were compared with those of an experienced medical oncologist making informed decisions from extensive cohort data.

Our study showed a much lower error rate than in previous analyses of cause of death stored in the OCR.[6] This difference may, however, be due to the different tumor sites being compared. Thus, our results may not be generalizable to all cancer sites. We reason, however, that misclassification of cause of death is greatest in those cancers, such as breast or prostate cancers, that have favourable prognoses, because the probabilities of deaths due to cancer and to competing causes approach one another much more than in highly aggressive cancers, such as lung or pancreatic cancers. Also, with aggressive cancers the course of illness is usually dramatic and clinically more clear-cut and thus classification of death should be more accurate. If our reasoning is correct, the results of our study might be reassuring to researchers investigating other cancers or advanced breast cancer.

Our results may not be generalizable to all cancer registries. Due to the centralized nature of the Ontario health care system, the OCR is able to obtain all the pertinent medical documentation in order to provide the epidemiologic data for this type of study. Other registries may not have the infrastructure or ability to be as complete and inclusive as the OCR. However, where registries are population-inclusive and verified, our results may be applicable.

There are a few methodological issues in this study that need to be addressed. Seven patients were removed from the analysis as they were recorded in the OCR as deceased with no cause of death. Sensitivity analyses showed, however, that misclassification of these deaths in either direction would have minimal effect on our conclusions.

Intensive follow-up for the MSH study ended in spring 2005 when funding for the clinical follow-up component of the study came to an end. The request for data from the OCR was made in August of 2006, at which time the quality of the registry was only assured until the end of 2004. Therefore, there was a slight discrepancy in the end of follow-up; however, this did not appear to affect the results. Our analyses used the decisions made by the MSH study medical oncologist from all collected data as the reference standard. It is possible that a small percentage of diagnoses were misclassified by the MSH study medical oncologist, potentially decreasing the agreement between the data sources. However, the high kappa statistics reflect good agreement in the absence of a gold standard.

We used kappa statistics to evaluate the agreement between two sources of categorical cause-of-death data as there was no clear cut gold standard (e.g. OCR found cancer deaths missed by the MSH study). In doing so we were able to provide classification accuracy with sensitivity and specificity, as well as reliability with kappa. These provide complementary pieces of information and strengthen the conclusions made about the utility of the OCR.

In conclusion, the results of our study show that there is strong agreement between the cause-of-death data collected from a longitudinal cohort study of breast cancer patients using a medical oncologist's interpretation based on rigorous prospective data collection and the passive data collection system of the OCR. This information is important to the conclusions drawn from studies conducted using registry data, as it may strengthen their validity. It may also encourage researchers to use cancer registry data when study-specific cancer follow-up data is incomplete, absent or of poor quality. Also, our results suggest that researchers may want to routinely employ registry data to verify follow-up information in ongoing studies.

References

1. Marrett LD, Clarke EA, Hatcher J, Weir HK. Epidemiologic research using the Ontario Cancer Registry. Can J Public Health. 1986; 77 Suppl 1:79-85.

2. Hilsenbeck SG. Quality control practices in centralized tumor registries in North America. J Clin Epidemiol. 1990;43(11):1201-12.

3. Robles SC, Marrett LD, Clarke EA, Risch HA. An application of capture-recapture methods to the estimation of completeness of cancer registration. J Clin Epidemiol. 1988; 41(5):495-501.

4. McLaughlin JR, Kreiger N, Marrett LD, Holowaty EJ. Cancer incidence registration and trends in Ontario. Eur J Cancer. 1991;27(11):1520-4.

5. Holowaty E. Summarization of information from multiple data sources. In: Black R, Simonato, L, Storm, H, ed. Automated data collection in cancer registries. IARC Technical Report 32. Lyon: IART, 1998.

6. Hall S, Schulze K, Groome P, Mackillop W, Holowaty E. Using cancer registry data for survival studies: the example of the Ontario Cancer Registry. J Clin Epidemiol. 2006;59(1):67-76.

7. Andrulis IL, Bull SB, Blackstein ME, Sutherland D, Mak C, Sidlofsky S, Pritzker KP, Hartwick RW, Hanna W, Lickley L, Wilkinson R, Qizilbash A, Ambus U, Lipa M, Weizel H, Katz A, Baida M, Mariz S, Stoik G, Dacamara P, Strongitharm D, Geddie W, McCready D. neu/erbB-2 amplification identifies a poor-prognosis group of women with node-negative breast cancer. Toronto Breast Cancer Study Group. J Clin Oncol. 1998;16(4):1340-9.

8. Schouten LJ, Jager JJ, van den Brandt PA. Quality of cancer registry data: a comparison of data provided by clinicians with those of registration personnel. Br J Cancer. 1993;68(5):974-7.

9. Landis JR, Koch GG. The measurement of observer agreement for categorical data. Biometrics. 1977;33(1):159-74.

Using administrative data to understand the geography of case ascertainment

N. Yiannakoulias (1) PhD; D. P. Schopflocher (2) PhD; L. W. Svenson (3) BSc

Abstract

We examined the geographic variability of information generated from different case definitions of childhood asthma derived from administrative health data used in Alberta, Canada. Our objective was to determine if analyses based on different case ascertainment algorithms identify geographic clusters in the same region of the study area. Our study group was based on a closed cohort of asthmatic children born in 1988. We used a spatial scan statistic to identify variations in the approximate location of geographic clusters of asthma based on different case definitions. Our results indicate that the geographic patterns are not greatly affected by the case ascertainment algorithm or the source of data. For example, asthmatics identified from medical claims data showed similar clustering to asthmatics defined through hospitalization and emergency department data. However, estimates of prevalence and incidence require careful consideration and validation against other data sources.

Keywords: *public health surveillance, spatial analysis, administrative health data, asthma*

Introduction

The growing availability of electronic health data and capacities for computer hardware to warehouse and analyse such data presents new opportunities for research on a variety of health outcomes. There is a growing body of evidence suggesting that administrative health data can be particularly important resources for research and surveillance of chronic diseases.[1-4] One of the most important contributions of administrative health data has been to provide information that facilitates the analysis of entire populations covering large geographic regions. In Canada, this often involves provincial-scale analysis (that is, the comparisons of regions within a province) that can be particularly useful for linking health information systems to decision making in public health. Geographic variations between regions within a province may indicate differences in epidemiology, population attributes, availability of services, exposure to environmental hazards, diagnostic practice and a variety of other factors.

One of the challenges to using administrative health data in research and surveillance is that different methods of case ascertainment may confound group differences especially if how these data were collected and/or generated varies. For example, there may be rural/urban differences in the effectiveness of certain data to identify incident stroke events.[5-7] These differences could be related to delivery of care (particularly in rural areas in which acute care centres often function in primary care roles), availability of diagnostic resources, geographic variations in physician specialty and many other factors. The most commonly discussed solution to these data problems is the development of case ascertainment algorithms that combine multiple administrative health data sources and/or multiple records within a single source.[3,8] With this paradigm, rather than using a single record from a single data source to identify a case, multiple records are combined with data from multiple sources. Integrating multiple sources of data may improve case ascertainment by traditional measures (such as sensitivity and specificity) but also improve the geographic uniformity in the case selection process by ensuring that geographic comparisons are not overly influenced by local or regional anomalies associated with a single data source.

Most case definition algorithms using administrative data have been validated against clinical chart reviews[9,10] or survey responses.[4,11] Although the former is an important benchmark for evaluating a case definition algorithm, it is less able to characterize the ways in which an algorithm's properties might vary between regions. This is particularly true if the validation work is based on a specific site of study, rather than a comprehensive sampling of sites over a large geographic area. While comparing administrative data to survey data can be informative, this process deals with two fallible data sources and no "gold standard."

In this study, we examined the geographic variability of information generated from different case definitions of childhood

Author References

1 School of Geography and Earth Sciences, McMaster University, Hamilton, ON
2 Faculty of Nursing, University of Alberta, Edmonton, AB
3 Surveillance and Environmental Health, Alberta Health & Wellness, Edmonton, AB
Correspondence: Nikolaos Yiannakoulias, School of Geography and Earth Sciences, McMaster University, 1280 Main Street West, Hamilton, ON L8S 4K1, Tel.: 905-525-9140, ext. 20117, Fax: 905-546-0463, Email: yiannan@mcmaster.ca

asthma derived from an administrative health data system. Our objective was to observe how the use of different case ascertainment algorithms can affect the appearance of patterns and variability on a map. Rather than comparing case definitions to a gold standard or a survey of respondents, we compared how analytical information varies across a variety of case definition algorithms. Our specific approach was to search for geographic clusters of children identified as asthma cases. If analyses based on a variety of different case definition algorithms identify clusters in the same region of the study area, the analytical information about geographic patterns is reliable even if the case ascertainment criteria differ. On the other hand, large variations in the resulting analyses could suggest that case definition algorithms should be more geographically specific, and should be designed to take into account local or regional differences in the use of services, conformity to data standards, or other factors.

Information from this study will inform the development of case definitions for childhood asthma based on administrative data by identifying the degree to which algorithms should be geographically specific. More generally, however, we present a simple framework for evaluating the geographical robustness of administrative health data for a variety of chronic conditions.

Methods

Data

Our study area, the province of Alberta, Canada, is well suited to this analysis because of both the availability of multiple sources of administrative health data and the existence of a population registry that can be used to identify the location of residence over time. Like other Canadian provinces, Alberta maintains a publicly funded single payer health care insurance system that covers most health services. Residents of the province who do not opt out of the provincially insured health care system are required to register with the Alberta Health Care Insurance Plan and are then provided a unique lifetime identifier that enables the linkage of health data sources. Over 99% of the province's population is registered under this system.

Alberta Health and Wellness has designed a system for the creation of longitudinal health data profiles in support of a variety of public health surveillance activities. These profiles are based on a linkage of four databases: the fee-for-service medical claims (claims), the Ambulatory Care Classification System containing emergency department admissions (emergency), an in-patient hospital services system (in-patient) and the Alberta Health Care Insurance registry system. Data from these systems are linked based on the unique numeric identifier and then tabulated into annual counts of services associated with a particular condition (defined by ICD-9* or ICD-10† codes) over time. When linked to the population registry system (which contains information about sex, age and place of residence) this system provides a simple method for observing changes in population estimates of incidence and/or prevalence based on different case definition algorithms.

We used a data profile for asthma-related services (identified using ICD-9 code 493 or ICD-10 J45 in the primary diagnostic field) as the primary source of data in this study. Our study period was between the 1998 and 2004 calendar years. In order to control for the variations in asthma management practice that have occurred in recent years, our study is restricted to persons born in the same year. Although the claims system has records back to the early eighties, the in-patient system has electronic records dating back to 1993 and the emergency data system has electronic records that only go back to 1998. In 1998, there were 44 651 children born in 1988 residing in the province and registered with the health care insurance plan. We restricted our study population to children born in 1988 who resided in the province continuously between 1998 and 2004; thus the subjects were old enough for reasonable asthma diagnoses to be made, but still less than 18 years old for the study period. This gave a closed study cohort of 38 905 children. Of these, 8965 had at least one medical service recorded within one of the three health databases in which asthma was the primary diagnosis associated with the service. We assumed that the minimum threshold to identify a child as asthmatic is two or more asthma-related services (not on the same day) over the study period. We referred to this as the "baseline" asthmatic group. Subject to this definition, 5110 children in the cohort were considered baseline asthmatics as of 2004, giving an asthma prevalence of roughly 13% within this cohort.

We used the data profile system to define cases based on several different case definition algorithms (Table 1), referring to these as "test case-definitions." Definitions vary based on the number and type of services within each of the data systems. These definitions were chosen to exaggerate differences in the case identification algorithms; most algorithms would require fewer contacts to qualify persons as a case than the definitions presented here. Therefore, the interpretation of our results should be viewed as an extreme example of how different case ascertainment algorithms might present different information on the geographic distribution of disease. We avoided over-counting contacts for particular episodes by counting only one of any of these contacts in a single day. When there were multiple contacts between the data systems on a particular day, we preferentially retained in-patient records over emergency records and preferentially retained emergency records over claims records.

Using 2004 residential postal codes linked to the data profile, all data were aggregated to the level of municipality. Municipalities, consisting of cities, towns and villages, were restricted to those with at least 10 members of the cohort, making a total of 294. Smaller municipalities were joined to larger municipalities to ensure no members of the cohort were dropped from the analysis. Children living outside of municipal areas (e.g. on farms) were assigned

* International Statistical Classification of Diseases and Related Health Problems, 9th Revision

† International Statistical Classification of Diseases and Related Health Problems, 10th Revision

to the municipality within which their residential postal code was included (typically, where they pick up their mail). As a result, some rural-dwelling children may have been assigned to a municipality that is neither the closest to their residence nor where they receive the majority of their medical services. However, any errors in geo-referencing are common to both the numerator and denominator and should not bias our results.

Analysis

For visualization purposes, we mapped the prevalence of baseline asthmatics in Alberta. Rather than mapping crude prevalence rates, which would be greatly affected by small numbers, we used a modelled approach to estimate relative risk in a way that manages stochastic variation in the data. We used a Poisson model to predict a function of the mean number of baseline asthmatics and included population of children as an offset to control for variations in the geographic distribution of the cohort. This model also includes a random intercept effect for each municipality and estimates spatial parameters to smooth out local variations in prevalence. In simple terms, this process averages geographically neighbouring observations with each other in a way conceptually similar to a one-dimensional moving window average. This modelling process is referred to as a generalized linear mixed model (or GLMM) approach.[12] We used the SAS procedure PROC GLIMMIX to solve this model.[13] We mapped the predicted baseline asthma morbidity ratio in the cohort at the municipality level. The map produced is of polygons shaded according to relative risk, with each polygon representing the area surrounding a municipality (Figure 1).

Our primary analysis involved explicit hypothesis tests for geographic clusters based on the different test case-definition algorithms. We used the spatial scan statistic[14] to identify these geographic clusters. The spatial scan approach uses a moving and variably sized window (a circular one in this application) to search a large number of potential clusters. The method then identifies the cluster in the set that is most likely to cause the rejection of a null hypothesis of constant risk. This cluster is referred to as the "most-likely" cluster of disease. The statistical significance of a most-likely cluster was evaluated through Monte Carlo simulation. By testing the significance of only the disease cluster most likely to cause the rejection of a null hypothesis of constant risk, the method avoids problems of multiple testing common to some other methods of local cluster detection.[14]

In our analysis, we investigated two general hypotheses for each of the six test case-definitions of asthma described in Table 1. The first hypothesis was that the spatial distribution of asthmatics of all definitions (including the baseline asthmatics) differed from the spatial distribution of the study cohort population. This corresponds to a null hypothesis of constant risk; that is, that there is no geographically clustered subset of municipalities that have an excess risk of asthma. We refer to this as the *constant risk null hypothesis*. Our test statistic was the Poisson model likelihood ratio,[14]

$$\left(\frac{c_i}{e(c_i)}\right)^{c_i}\left(\frac{C - c_i}{C - e(c_i)}\right)^{C - c_i}$$

where C is the total number of cases who are defined as asthmatic according to a particular definition, c_i is the number of these cases in municipality i and $e(c_i)$ is the expected number of these cases in municipality i. Here we calculated $e(c_i)$ as

$$e(c_i) = m_i g.$$

For all case definitions, m_i is the number of children in the study cohort residing in municipality i and g is the overall rate of asthmatics for a particular definition in the study cohort.

Results from the analysis above indicated whether or not to reject a null hypothesis of constant risk and approximately where there are clusters of asthmatics in the study cohort for the different test case-definitions. To determine the geographic variation in test case-definitions more explicitly, we also determined whether the geographic distribution of asthmatics according to each of the test case-definitions differed from the distribution of baseline asthmatics. As before, we used the Poisson model likelihood-ratio test, but in this case,

$$e(c_i) = a_i h,$$

where a_i is the number of children who are asthmatic by a specified case definition residing in municipality i and h is the proportion of asthmatics according to this definition among the baseline asthmatic population. Here, a rejection of the null hypothesis for a particular test case-definition indicates that the geographic distribution of asthmatics identified by this case definition algorithm is no different from the geographic distribution of the baseline asthmatics. This corresponds to a test of a null hypothesis of constant case definition, and we refer to this as the *constant case definition null hypothesis*.

We used SaTScan v. 6.1 to search for clusters.[15] All clusters were bound to a size no larger than 50% of the population of Alberta, and all clusters searched were constrained to a circular shape. In all cases, a significance level of 0.05 was used to assess whether there is a most-likely cluster against the null hypotheses of constant risk and constant case definition.

Results

Test case-definition "A" provided a prevalence estimate of 4.4%, which was less than half of the baseline group (Table 2). At the other extreme, test case-definition "F" provided prevalence estimates of less than 0.2%. For each of the test case-definitions, we also tabulated the average number of services for each child by the type of services within the medical system. For all test case-definitions, all children appeared to have frequent asthma-related contacts with the emergency system when compared to the cohort as a whole. Children in all the test case-definitions appeared to experience more contacts with medical system (for any reason) than baseline asthmatics and non-asthmatics in the cohort.

Figure 1 illustrates the model-predicted geographic distribution of baseline asthmatics in the study cohort. There is variation in the rate of asthma among the

TABLE 1
Test case-definitions

Definition label	Test case-definition
A	6 or more services of any type, 1998-2004
B	6 or more services including a minimum of 2 or more emergency or in-patient admissions, 1998-2004
C	12 or more services of any type, 1998-2004
D	12 or more services including a minimum of 2 or more emergency or in-patient admissions, 1998-2004
E	6 or more emergency or in-patient admissions, 1998-2004
F	12 or more emergency or in-patient admissions, 1998-2004

baseline asthmatics, with the relative risk highest in the city of Calgary where it was 22% higher than the provincial average. The Edmonton area had relative risk very close to the provincial average (0.3% higher than the provincial average). Rural areas of central and northern Alberta had the lowest prevalence of asthma in the cohort.

Statistically significant clusters under the two null hypotheses are mapped on Figures 2 and 3. Based on our null hypothesis of constant-risk, all test case-definitions of asthma with the exception of "B" were associated with a statistically significant most-likely cluster. Cluster "B", though not statistically significant, was located in a similar region to cluster "D". The relative risk associated with each cluster is relatively small; in most cases, the study population located inside the cluster had a 25% higher risk of asthma than the study population located outside the cluster. The one exception corresponded to case definition "F", for which the relative risk of asthma inside the cluster is more than double the risk outside the cluster. All mapped clusters represent regions where the likelihood of rejecting the null hypothesis of constant risk was highest for each of the case definitions.

Based on our null hypothesis of constant case definition, only definition "F" reached a level of statistical significance. Definition "F" is the strictest of all test case-definitions, and the total number of cases in this cluster was very small; for this definition, only 52 of the total number of cases were found inside this cluster. Children within this cluster had an 87% higher chance of being cases (according to definition "F") than children outside the cluster.

Discussion

Under the null hypothesis of constant risk, there were few apparent differences in the location of clusters across the case definitions. All but one of the clusters occurred in the southwest area of the province, though the clusters do vary considerably in geographic size; for example, clusters "A" and "C" were smaller than the other clusters. This apparent similarity was based on a qualitative assessment of a map covering a large area, and it is unclear from this map alone if these observations represent a systematically different geography in the test case-definitions when compared to each other or to the baseline asthma group. The search for clusters under the null hypothesis of constant case definition provided a more explicit test of whether or not the location of asthma clusters varied by particular case ascertainment algorithm. All but one of these searches failed to reject the null hypothesis of constant case definition. This suggests that there were relatively small differences in the geographic pattern of asthma across the different test case-definitions and that the detection of clusters was fairly robust to the precise definition selected.

For the null hypothesis of constant case definition, the only statistically significant cluster was based on definition "F". This cluster was located in southwest Alberta, in the same region as the clusters found under the null hypothesis of constant risk. It is possible that the cluster represented a geographically concentrated region of high-service use or serious asthmatics within the asthmatic population of the cohort. The cluster could also be related to changes in the population or health service utilization practices in the region, where there has been noteworthy population growth in recent years. Finally, it is important to be mindful of the fact that this definition corresponds to a prevalence estimate of less than 1%, and is considerably stricter than any case definition likely to be used in epidemiological or surveillance applications. Therefore, though there was a statistically significant difference in relative risk between the areas inside and outside the cluster, this amounts to a very small difference in absolute risk.

Based on these observations, it appears that comparative geographical analysis of asthma risk is not greatly affected by the case ascertainment algorithm used. More generally, our findings indicate that geographic information about relative differences in prevalence (or relative risk) may be invariant to the specific choice of case definition even if prevalence (absolute risk) varies across these case definitions. The implication of our findings, if they can be generalized to other settings or other chronic conditions, are important in applications concerned with geographic variations in illness. When using data sources that have not been validated against a gold standard, it may be more appropriate to report geographic measures of relative risk than absolute risk. This is suitable for applications in which the relationship between risk and risk factors is of primary interest. For example, an ecological correlation study of the relationship between asthma and social and environmental risk factors is likely to produce similar model coefficients across different case ascertainment algorithms. In surveillance applications, where variations and changes in absolute risk are often of interest, precise measures of prevalence and incidence remain important. Our case ascertainment algorithms produced very different estimates in prevalence of asthma in the cohort. Precise prevalence estimates are necessary to understand the actual population burden of disease, and therefore, require data that have been validated against a medically and socially acceptable case definition standard.

TABLE 2
Tabulation of asthma-related and total service utilization by all case definitions (1998-2004)

	Baseline definition	Test case-definitions of asthma						Non-asthmatics
		A	B	C	D	E	F	
		n = 1710	n = 685	n = 638	n = 390	n = 244	n = 71	n = 33 795
Percentage of asthmatics in cohort	100.00	30.81	12.34	11.49	7.03	4.40	1.28	N/A
Percentage of the total cohort population	13.13	4.40	1.76	1.64	1.00	0.63	0.18	100.00
Asthma-related services (1998-2004)								
Mean claims	5.04	9.67	10.47	15.52	14.87	12.09	16.12	N/A
Median claims	3	8	8	14	13	9	13	N/A
Mean emergency	1	2.47	5.75	4.76	7.59	10.95	19.26	N/A
Median emergency	0	1	4	2	5.5	8.5	16	N/A
Mean in-patient	0.05	0.16	0.37	0.322	0.523	0.63	1.12	N/A
Median in-patient	0	0	0	0	0	0	0	N/A
All services (1998-2004)								
Mean claims	43.74	49.7	49.18	56.95	55.03	50.35	60.51	29.44
Median claims	36	42	41	47	46	42	46	23
Mean emergency	10.64	12.2	17.76	15.02	19.24	24.95	32.59	7.08
Median emergency	6	7	13	10	14	20	27	3
Mean in-patient	0.33	0.44	0.75	0.66	0.93	1.11	1.55	0.21
Median in-patient	0	0	0	0	0	1	1	0

Although not an explicit objective of our study, our results do reveal interesting geographic patterns of paediatric asthma in Alberta. Firstly, the distribution of relative asthma risk based on Figure 1 suggests lowest risk in rural central and northern Alberta and highest risk in southern Alberta, particularly Calgary. The clusters of asthmatics based on the test case-definitions also identify the Calgary area as the region of highest risk. Together, these observations reflect high prevalence of asthma, as well as a high incidence of emergency and in-patient hospital admissions. The use of these services in particular reflects a high burden of treatment in a subset of children in the Calgary region. It may also be an indicator that asthma is more severe (and in turn, requires more emergency and hospital care) for patients in this part of the province. As noted above, this pattern may be related to the absence of primary care for children in an area of rapid population growth, it could also reflect fundamental differences in asthma epidemiology in this region. This explanation is supported by the apparently distinct pattern of higher prevalence in rural and urban southern Alberta, which could reflect the role of environmental or meteorological conditions in the region.[16] Further research on the explanation for this geographic pattern, and identifying whether or not it is common to the paediatric or general population, is warranted.

We note two potential limitations to our study. First, we excluded a large number of children immigrating to Alberta between 1998 and 2004. It is possible that children moving into the province have health utilization profiles that are considerably different from those we included in our study group. Immigration into Alberta tends to be into urban areas, and immigration is well known to affect the use of health services. Recent arrivals to the province may be more inclined to use acute-care centres for primary care. This could have resulted in systematic differences in the effectiveness of case definitions, for example, by increasing the sensitivity of definitions "E" and "F" in urban areas. Second, the ability to detect a statistically noteworthy asthma cluster is partly dependent on prevalence, and more specifically, the number of cases. All else being equal, case definitions with a higher overall prevalence are more likely to produce detectable patterns of clustering. It is possible that clusters were not detected for some definitions and detected for others simply based on the different total number of cases identified. Though this may be a limitation of our study design, we note that most case definitions did result in identifiable clusters of asthma in roughly the same part of the province. Furthermore, the one significant cluster found in the test of constant case definition was the least numerous of all case ascertainment algorithms.

Conclusion

Administrative data represent an important resource for public health surveillance and research. Validation studies that compare case definitions based on administrative data to clinical assessments and surveys are important for understanding the strengths and weaknesses of these data, as well as determining good estimates of prevalence and incidence. Assessments of relative risk, across geography, time, age, sex, social class and other measures, are also important for a complete understanding of disease epidemiology. Our results suggest that

FIGURE 1
Model estimated relative risk for baseline definition asthmatics

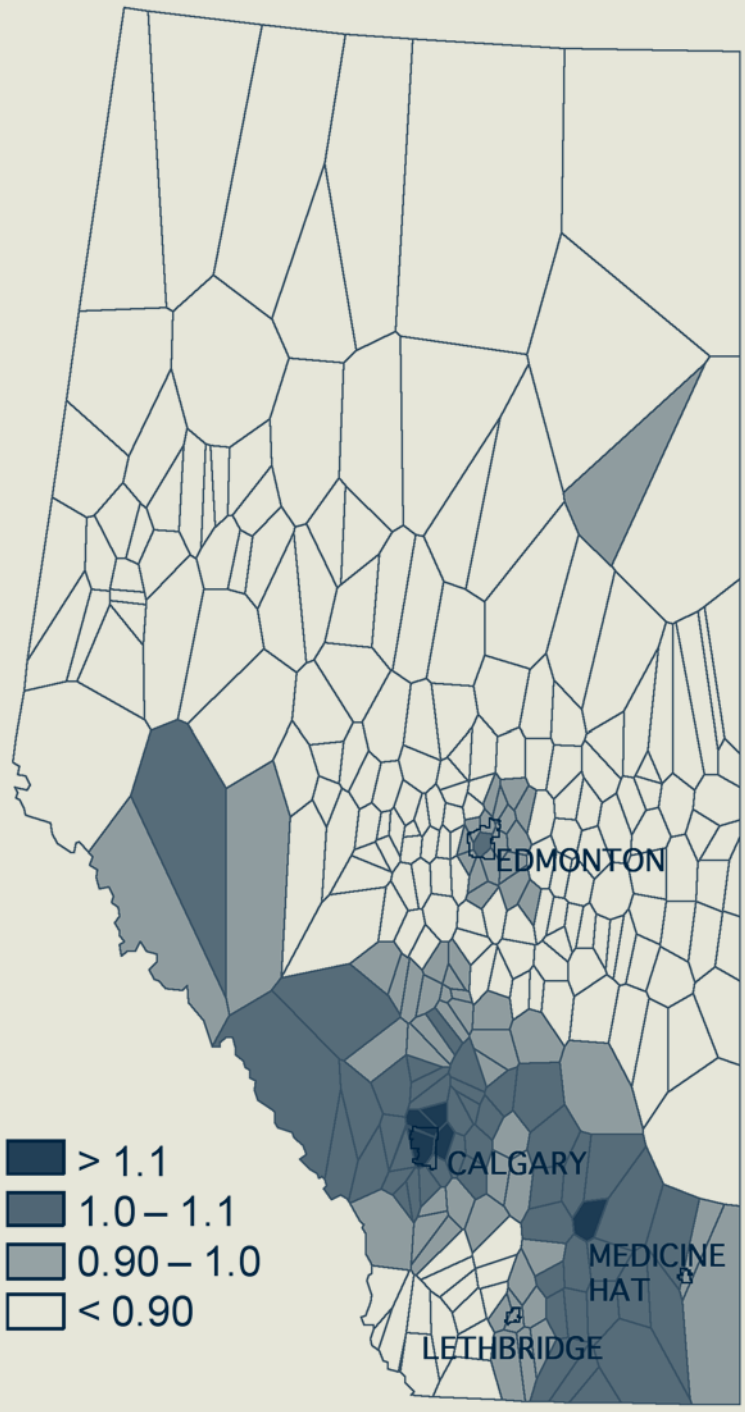


FIGURE 2
Significant clusters under a null hypothesis of constant-risk

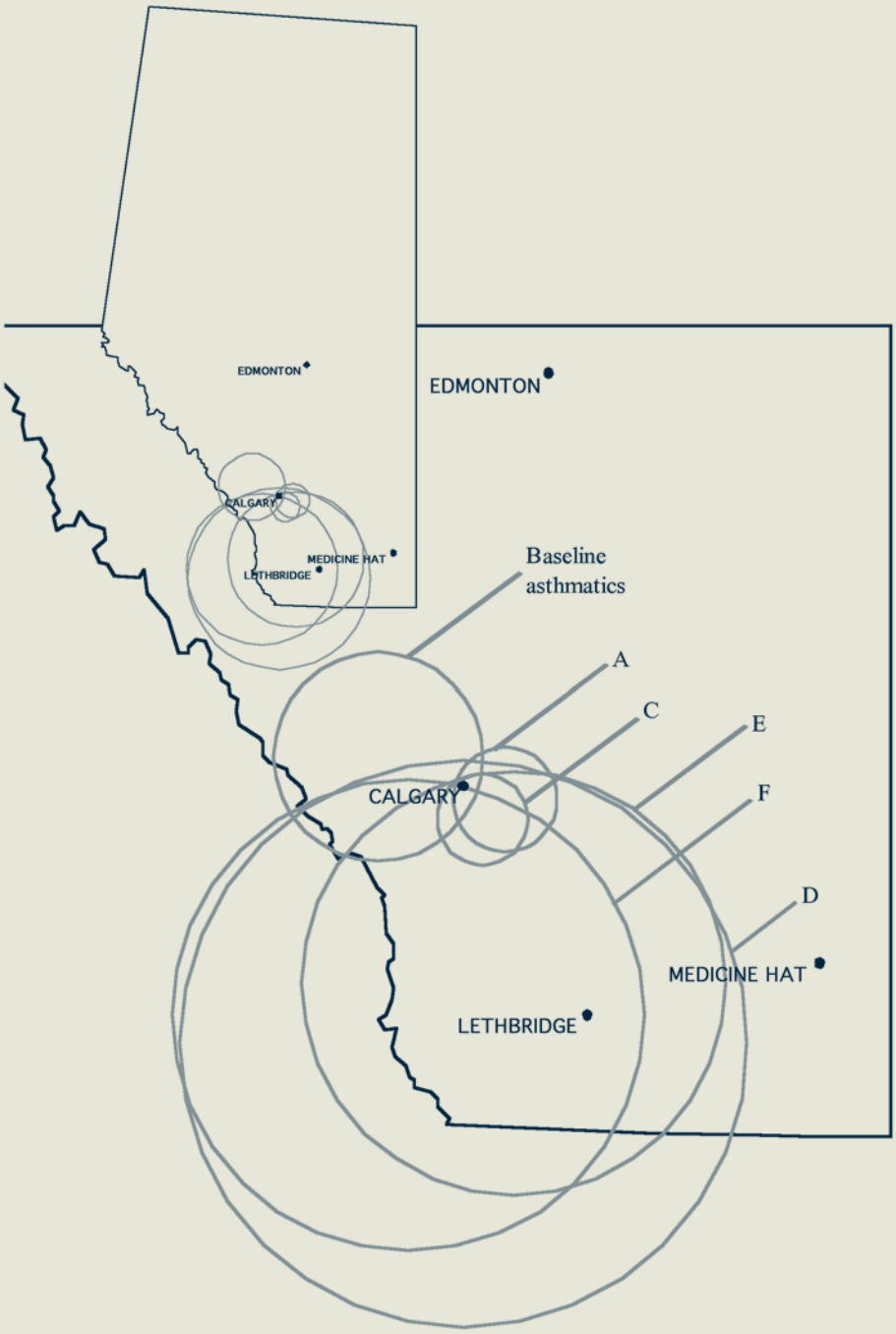
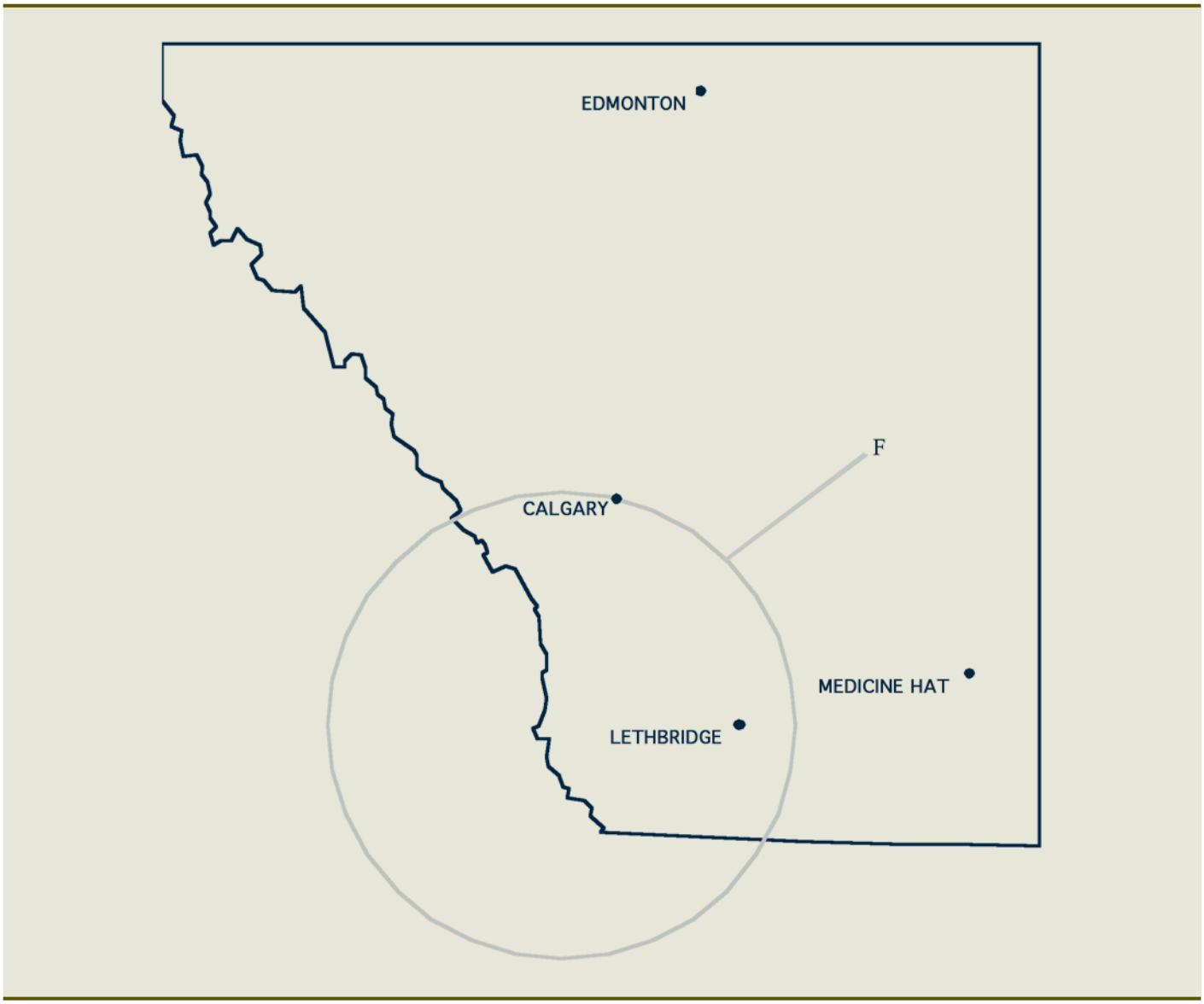


FIGURE 3
Significant clusters under a null hypothesis of constant case definition



relative geographic comparisons of disease based on case definitions from administrative data are not greatly affected by the specifics of the case ascertainment algorithm, even when the case definitions are derived from data from different sources. However, there are considerable variations in prevalence and incidence based across the different definitions, and therefore, routine surveillance requires careful consideration of the precise algorithm used.

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References

1. Tu K, Campbell NR, Chen XL, Cauch-Dudek KJ, McAlister FA. Accuracy of administrative databases in identifying patients with hypertension. Open Med. 2007;1:E3-5.
2. Wilchesky M, Tamblyn RM, Huang A. Validation of diagnostic codes within medical services claims. J Clin Epidemiol. 2004;57:131-41.
3. Hux JE, Ivis F, Flintoft V, Bica A. Diabetes in Ontario: determination of prevalence and incidence using a validated administrative data algorithm. Diabetes Care. 2002; 25:512-6.
4. Robinson JR, Young TK, Roos LL, Gelskey DE. Estimating the burden of disease: comparing administrative data and self-reports. Med Care. 1997;35:932-47.

5. Liu L, Reeder B, Shuaib A, Mazagri R. Validity of stroke diagnosis on hospital discharge records in Saskatchewan, Canada: implications for stroke surveillance. Cerebrovasc Dis. 1999;9:224-30.

6. Yiannakoulias N, Svenson LW, Hill MD, Schopflocher DP, James RC, Wielgosz AT, Noseworthy TW. Regional comparisons of in-patient and outpatient patterns of cerebrovascular disease diagnosis in the province of Alberta. Chron Dis Can. 2003; 24:9-16.

7. Yiannakoulias N, Svenson LW, Hill MD, Schopflocher DP, Rowe BH, James RC, Wielgosz AT, Noseworthy TW. Incident cerebrovascular disease in rural and urban Alberta. Cerebrovasc Dis. 2004;17:72-8.

8. Tirschwell DL, Longstreth WT Jr. Validating administrative data in stroke research. Stroke. 2002;33:2465-70.

9. Donahue JG, Weiss ST, Goetsch MA, Livingston JM, Greineder DK, Platt R. Assessment of asthma using automated and full-text medical records. J Asthma. 1997;34:273-81.

10. Kokotailo RA, Hill MD. Coding of stroke and stroke risk factors using international classification of diseases, revisions 9 and 10. Stroke. 2005;36:1776-81.

11. Huzel L, Roos LL, Anthonisen NR, Manfreda J. Diagnosing asthma: the fit between survey and administrative database. Can Respir J. 2002;9:407-12.

12. Breslow NE, Clayton DG. Approximate inference in generalized linear mixed models. J Am Stat Assoc. 1993;88:9-25.

13. SAS [computer program]. Version 9.1.3. Cary, NC: SAS Institute; 2006.

14. Kulldorff M. A spatial scan statistic. Commun Stat Theory. 1997;26:1481-96.

15. SaTScan [computer program]. Version 6.1. Bethesda, MD. Kulldorff M & Information Management Services, Inc.; 2006.

16. Verhoef MJ, Rose MS, Ramcharan S. The relationship between chinook conditions and women's physical and mental well-being. Int J Biometeorol. 1995;38:148-51.

Association of obesity with mood and anxiety disorders in the adult general population

T. M. Gadalla, PhD (1)

Abstract

Obesity is a major health concern. It has been implicated as a risk factor for several physical illnesses, functional limitations and poor quality of life. However, while the physical consequences of obesity are well established, the relationship between obesity and mental health is still unclear. This study used data collected in the Canadian Community Health Survey, cycle 3.1 (2005) to examine this relationship in adults 20 to 64 years old. Obesity was significantly associated with mood disorders, but not with anxiety disorders. When adjusting for sex, place of birth, smoking, and functional limitations, all of which were significantly associated with obesity, the odds of obesity remained significantly higher in persons with mood disorders (with or without anxiety disorders). It is still unclear whether the relationship between obesity and depression is causal, and if so, whether obesity causes depression or depression causes obesity. Implications for health care providers and suggestions for future research are discussed.

Key words: *obesity, mood disorders, anxiety disorders, Canada, adult*

Introduction

Obesity is a growing public health concern in modern societies. Recent studies indicate that between 10% and 23% of adults in Europe and between 22% and 35% of adults in the US are classified as obese, i.e. have a body mass index (BMI) of 30 kg/m^2 or higher.[1] In the US, the prevalence of adult obesity has doubled since 1980[2] and is presently the second major cause of preventable death after smoking.[3] In Canada, the adult obesity rate has also doubled in the last three decades to reach 15.2% in 2003[4] with an estimated economic burden of $4.3 billion.[5]

Physical inactivity and unhealthy diet have been identified as major risk factors for obesity.[6] Prevalence of obesity in women was also found to increase with advanced age, low income and lower levels of education.[7] The decline in the prevalence of smoking among adults may also have contributed to the observed increase in the prevalence of obesity.[8]

Ample research has highlighted the role of obesity as a risk factor for a large number of chronic health complications, such as cardiovascular disease, hypertension, type 2 diabetes, stroke, sleep apnea and certain types of cancer, as well as complications in pregnancy and surgery.[9] Obesity has also been implicated as a risk factor for functional limitations and poor health-related quality of life.[10] However, while the physical consequences of obesity are well established, the relationship between obesity and mental health is still unclear and reported findings have been mixed. Some researchers examined prevalence of obesity in individuals with mental disorders[11,12] and others examined the prevalence of mental disorders in obese individuals.[13] However, most of these studies examined simple associations between depression/depressive symptoms and body fat without accounting for possible mediators and/or moderators of their relationship.[14]

In a review of four longitudinal studies and 20 cross-sectional studies of the effects of obesity on depression, Atlantis and Baker[15] found that longitudinal studies provided consistent evidence that obesity may increase the odds of developing depression or depressive symptoms. They also found most cross-sectional studies from the US supported the above association for women but not men. In contrast most cross-sectional studies from populations other than the US consistently failed to find such associations. But Bruffaerts and colleagues[1] analysed data from six European countries and found that obese individuals were more likely to have mood disorder (odds ratio [OR] = 1.3) and more than one mental disorder (OR = 1.4) compared with individuals of adequate weight. On the other hand, Blaine[16] conducted a meta-analysis of 16 longitudinal studies of the effect of depression on obesity. In five of the studies, initial depression led to weight loss but this association was statistically significant in only one study.[16] After controlling for baseline BMI and background variables, depressed adults were at significantly higher risk for developing later obesity (OR = 1.08) and the risk was particularly high for adolescent females (OR = 2.57) compared to non-depressed people.[16] Yet, some studies found the relationship between obesity and depression non-significant[17] or negative.[18]

Author References

1 Factor-Inwentash Faculty of Social Work, University of Toronto

Correspondence: Dr. Tahany M. Gadalla, Factor-Inwentash Faculty of Social Work, University of Toronto, 246 Bloor Street West, Toronto, Ontario M5S 1A1, Tel.: (416) 946-0623, Fax.: (416) 978-7072, Email: tahany.gadalla@utoronto.ca

Associations between obesity and other mental disorders have also been reported. Petry and colleagues[19] found obese individuals to have significantly elevated odds (ORs ranging from 1.21 to 2.08) of any mood, anxiety and alcohol use disorders as well as any personality disorder.[19] Scott and colleagues[13] found obesity to be significantly associated with any mood disorder (OR = 1.23), major depressive disorder (OR = 1.27) and any anxiety disorder (OR = 1.46). However, when they adjusted for the comorbidity between anxiety and mood disorders, the association between obesity and anxiety disorders remained significant (OR = 1.36) but the association between obesity and mood disorders became statistically insignificant (OR = 1.05).[13] Similarly, Simon and colleagues[20] found obesity to be associated with an approximately 25% increase in odds of mood and anxiety disorders.

Prior research also indicated that individuals' socio-demographic characteristics moderated the relationship of obesity with mental disorders, and especially that with mood disorders.[13] McLaren and colleagues[21] found the association between obesity and mental disorders to vary by type and severity of mental illness, and by gender and age. For example, substance use disorders were elevated among obese men at younger compared to older ages and mood disorders were elevated among obese women compared to women of adequate BMI, while subclinical anxiety/depression was reduced among obese men compared to adequate weight men and to adequate weight women.[21] On the other hand, Simon and colleagues[20] found no gender differences in the association between obesity and mood and anxiety disorders, but noted differences across racial groups and education levels. Based on a review of the literature on the obesity-mood disorders relationship, McElroy and colleagues[22] concluded that obesity is associated with depression in women while abdominal obesity may be associated with depression in both men and women. Gender difference in the relationship between relative body weight and depression was also reported by Carpenter and colleagues[23] who examined data on over 40 000 US adults and found a significant positive association between BMI and depression in women and a significant negative association in men. In the latter study, the authors compared prevalence of depression in obese persons to that in persons of normal weight or overweight. Yet, other researchers did not find such gender differences.[20,24,25] These findings highlight the importance of examining the relationship between obesity and each type of mental disorder separately while taking individual's socio-demographic characteristics into account.

Examination of factors associated with obesity and identification of subgroups of men and women who are at high risk of being obese provide the knowledge required for planning public health policies aimed at curbing the spread of this unhealthy and costly epidemic. The main objective of the present study was to examine the associations between obesity and mood and anxiety disorders while controlling for potential confounders. The potential confounders examined in this study were those that had been identified in the literature as risk factors for mental health and obesity. For example, positive associations between obesity and poor physical health and increased functional limitations,[9,10] and between physical decline and mental ill-health[26,27] have been reported. Thus, number of chronic physical conditions and limitations in daily activities were examined as potential confounders together with socio-demographic and economic characteristics. In addition, the study examined the association between being overweight and mood and anxiety disorders while controlling for the above variables. Further, the above analyses were conducted in the total sample as well as in men and women samples separately.

Methods

Sample

This research was based on data collected by Statistics Canada in the Canadian Community Health Survey-cycle 3.1.[28] The survey was conducted in 2005 and used a multistage stratified cluster probability sampling in which a sample of dwellings was randomly selected from lists of telephone numbers. One person aged 15 years or older was randomly selected from each household to participate in the survey. The survey sample was stratified by province and urban versus rural regions. Introductory letters were mailed to selected participants assuring them of the confidentiality laws governing the release and/or publication of collected data. Participation was voluntary and the response rate was 85%. The sample represented approximately 98% of the Canadian population aged 12 years or older who resided in private dwellings in the ten provinces and the three territories. Fifty percent of the respondents were randomly selected to be interviewed face-to-face using the computer-assisted personal interviewing method, and 50% were interviewed by telephone using the computer-assisted telephone interviewing method.[28] The sample used in this study included 73 110 survey participants 20 to 64 years old for whom complete records of variables used in the analysis were available.

Measures

Body mass index. Each participant's BMI was calculated based on the participant's self-reported height and weight. This variable was not calculated for female participants who were pregnant or did not answer the pregnancy question. BMI was used to assign adults aged 20 years and over (except pregnant women) to one of the following categories: underweight (BMI < 18.5), adequate weight (18.5 ≤ BMI < 25), overweight (25 ≤ BMI < 30) or obese (BMI ≥ 30). These BMI categories are adopted from the widely used body weight classification system recommended by Health Canada and the World Health Organization (WHO).

Mood and anxiety disorders. Participants were asked whether they had been diagnosed by a health professional as having a mood disorder (such as depression, bipolar disorder, mania or dysthymia), or anxiety disorders (such as any type of phobia, obsessive-compulsive disorder, generalized anxiety disorder, post-traumatic stress disorder or panic disorder) in the past year. A categorical variable with four mutually exclusive groups was created and used in the present study: participants with neither disorder, participants with mood disorders

only, participants with anxiety disorders only and participants with both disorders.

Physical Activity Index. This variable categorizes respondents as being "active," "moderately active," or "inactive" based on their average daily energy expenditure values (kcal/kg/day) during their leisure time activities in the past three months. Participants were asked about their leisure physical activities and the length of time they spent on each during the three months prior to the interview. The energy expenditure (EE) of participants' leisure activities was calculated using the frequency and time per session of the physical activity, as well as its metabolic equivalent of task (MET). Metabolic equivalent of task was expressed as a multiple of the resting metabolic rate. Thus, an activity of 4 METs requires four times the amount of energy as compared to when the body is at rest. Survey participants were not asked to specify the intensity level of their activities; therefore, the MET values calculated here correspond to the low intensity value of each activity. This approach was adopted because individuals tend to overestimate the intensity, frequency and duration of their activities.[29] Participants were then classified as active ($EE \geq 3.0$), moderately active ($1.5 \leq EE < 3.0$) and inactive ($EE < 1.5$).

Chronic conditions. Participants were asked about certain chronic health conditions that were expected to last or had already lasted six months or more and that had been diagnosed by a health professional. This variable was a count of the number of chronic physical illnesses participants were diagnosed for in the 12 months prior to the interview.

Functional limitations. Survey participants were asked a series of questions about whether they needed help with instrumental activities of daily living such as preparing meals, shopping for groceries and other necessities, getting to appointments, doing everyday housework, personal care or moving about their home because of a long-term health condition. A long-term health condition was defined as a condition that is expected to last or has lasted six months or more. This variable indicated whether participants required help with their usual daily activities.

Smoking. This variable indicates the type of smoker the respondent was, based on his/her answer to the question: "At the present time, do you smoke cigarettes daily, occasionally or not at all?

Socio-demographic characteristics and health indicators used in this research included sex, age group (20 to 34 years, 35 to 49 years, 50 to 64 years, 65 years or older), marital status (married/common law versus divorced/separated/widower/never married), length of time in Canada (9 years or less, 10 years or more, Canadian born), educational level (less than secondary school degree, secondary school graduate, some post secondary education, post secondary graduate) and income level (low, 30%; middle, 40%; and upper, 30%) of the income distribution in their province of residence.[28] The income level variable was derived by Statistics Canada to measure the rank of adjusted household income as a ratio of the provincial low-income cut-off. The low-income cut-off is defined as the income below which a family is likely to spend a significant portion of its income to purchase necessities such as food, lodging and clothing. First, the ratio of the participant's total household income to the low-income cut-off corresponding to their household size and community size was calculated. Second, these ratios were rescaled to range between zero and 100 within each province. For example, if the highest ratio was 21, i.e. adjusted personal income was 21 times the low-income cut-off, this would be the 100. Similarly, the lowest ratio would be the zero.

Data analysis

Chi-square tests were used to assess bivariate relationships between BMI classification and prevalence of mood and/or anxiety disorders, smoking, limitations in daily living activities, number of chronic physical conditions and socio-demographic and economic characteristics. All variables that were found in the bivariate analysis to be significantly associated with obesity (at the $p = .05$ significance level) were included in the multivariate logistic analysis. A block logistic regression analysis was used to test the association between obesity and a diagnosis of mood and/or anxiety disorders while controlling for potential confounders. Block regression is similar to sequential analysis in which the second block of variables is entered in the model after having accounted for the effects of the variables in the first block. The outcome variable was obese versus adequate weight. The first block of variables included sex, age, living arrangement, length of time in Canada, income level, education level, functional limitations, number of chronic physical conditions, smoking frequency and level of physical activity; the second block included having a diagnosis of mood and/or anxiety disorders. All independent variables were treated as categorical variables to allow for possible curvilinear relationships with the outcome variable. The block logistic regression analysis was conducted on the total sample as well as on men and women samples separately. The above analyses were repeated with the outcome variable being overweight versus adequate weight. Sampling weights were rescaled and used in all analyses. Rescaling the weights takes into account the unequal probabilities of selection of survey participants without inflating the sample size in hypothesis testing.[28]

Results

Of the 73 110 participants in this sample, 1769 (2.4%) were classified as underweight, 34 087 (46.6%) were classified as adequate weight, and 25 145 (34.4%) were classified as overweight and 12 109 (16.6%) were classified as obese (Table 1).

As seen in Table 1, 6.4% of obese individuals had been diagnosed with a mood disorder, compared with 3.6% of those with adequate weight. The prevalence of anxiety disorders in obese individuals is 3.0%, compared with 2.6% in individuals with adequate weight. Additionally, 3.4% of individuals classified as obese had been diagnosed with both mood and anxiety disorders, compared with only 1.9% among those classified as having adequate weight. A chi-square test indicated a significant association between obesity and mood/anxiety disorders (chi-square = 363.83, $df = 9$, $p < .0005$).

Table 2 includes numbers and percentages of obese and adequate weight individuals

TABLE 1
Canadians diagnosed with mood and/or anxiety disorders by BMI classification, 2005

BMI classification	Sample size	Neither mood nor anxiety	Mood disorders	Anxiety disorders	Mood and anxiety disorders
	(*n*)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Underweight	1 769	1 565	74	57	73
(BMI < 18.5)		(88.5)	(4.2)	(3.2)	(4.1)
Adequate weight	34 087	31 326	1 227	886	648
(18.5 ≤ BMI < 25)		(91.9)	(3.6)	(2.6)	(1.9)
Overweight	25 170	23 083	1 031	629	427
(25 ≤ BMI < 30)		(91.8)	(4.1)	(2.5)	(1.7)
Obese	12 109	10 559	775	363	412
(BMI ≥ 30)		(87.2)	(6.4)	(3.0)	(3.4)
Total	73 135	66 533	3 107	1 935	1 560
		(91.0)	(4.3)	(2.6)	(2.1)

Chi-square = 363.83, df = 9, $p < .0005$

Abbreviations: BMI, body mass index; df, degrees of freedom; *n*, sample size; *p*, significance level

by levels of all variables used in the analysis. The table also includes results of the multiple logistic regression analysis. The bivariate chi-square tests revealed a significant association ($p < .0005$) between obesity and sex, age, living arrangement, length of time in Canada, income level, education level, number of chronic physical conditions, limitations in daily living activities, smoking frequency and level of physical activity. The prevalence of obesity increased steadily with advancing age and number of chronic physical conditions and decreased steadily with higher education and physical activity levels. It is also worth noting that the prevalence of obesity among individuals born in Canada was more than double that of recent immigrants.

The likelihood of obesity for men was twice that of women (OR = 2.00, 95% CI = 1.92, 2.10), increased with advancing age (OR = 1.48, 95% CI = 1.40, 1.57 for the 35 to 49 age group; OR = 1.68, 95% CI = 1.58, 1.79 for the 50 to 64 age group) and was higher in individuals living with a partner compared with those living without (OR = 1.22, 95% CI = 1.16, 1.28). Further, the odds of obesity was 2.63 times higher (95% CI = 2.33, 2.96) for those born in Canada compared with recent immigrants. Persons in the low and middle income groups had higher odds of being obese compared with persons in the highest income level (OR = 1.12, 95% CI = 1.05, 1.19 for the low income group; OR = 1.12, 95% CI = 1.06, 1.18 for the middle income group). Those with less than high school education had significantly higher odds of being obese compared with post-secondary graduates (OR = 1.60, 95% CI = 1.49, 1.72). Although the results did not indicate a gradient of risk of obesity with income level (OR = 1.12 for both low and middle income groups), there was an apparent gradient of the odds of obesity with lower levels of education. Odds ratios presented in Table 2 also show a gradient of risk of obesity with level of physical activity with inactive individuals having double the odds of being obese compared with active individuals (OR = 2.01, 95% CI = 1.89, 2.13). Lastly, regular and occasional smokers were less likely to be obese compared with non-smokers (OR = 0.58, 95% CI = 0.55, 0.63 for regular smokers; OR = 0.63, 95% CI = 0.56, 0.69 for occasional smokers).

Results of the logistic regression analysis further indicated that after controlling for the above variables, the odds ratios of obesity were 1.50 (95% CI = 1.36, 1.66) among those with mood disorders and 1.48 (95% CI = 1.29, 1.69) among persons with both mood and anxiety disorders, compared with the odds of obesity in those with neither disorder (Table 2). There was no significant association between the odds of obesity and having been diagnosed with anxiety disorders (OR = 1.02, 95% CI = 0.89, 1.17).

Results of the logistic regression analysis conducted on men and women separately revealed slight differences with regards to sex in the association between obesity and mood and anxiety disorders. Adjusting for socio-demographic and economic characteristics, chronic physical conditions, activity limitations and smoking frequency, the odds ratios of obesity in men and women with anxiety disorders remained insignificant (OR = 0.88 for men; 1.10 for women). The odds ratio of obesity in women with mood disorders was 1.48 (95% CI = 1.30, 1.68, $p < .005$) and in men with mood disorders was 1.50 (95% CI = 1.27, 1.79, $p < .0005$). The odds ratio of obesity among women with both disorders increased to 1.45 (95% CI = 1.22, 1.72, $p < .0005$) and to 1.32 (95% CI = 1.04, 1.67, $p = .02$) among men with both disorders, compared with the odds of obesity in women and men with neither disorders.

Similar logistic regression analysis was conducted with overweight versus adequate weight as the dependent variable. Results of this analysis indicated that after controlling for socio-economic and health characteristics, the odds of being overweight for persons with mood disorders was 1.23 times that for persons with neither mood nor anxiety disorder (95% CI = 1.12, 1.34, $p < .0005$). There was no significant difference between the odds of being overweight for persons with anxiety disorders (OR = 1.04, 95% CI = 0.93, 1.16) nor for

TABLE 2
Association between obesity (BMI ≥ 30) and socio-demographic and economic characteristics and mental health indicators in Canadians 20 to 64 years old (*n* = 46 196 obese or adequate weight)

	Total	Obese *n* (%)	Odds Ratio (95% CI)
Block 1:			
Sex			
Women	24 578	5 391 (21.9)	1 (ref)
Men	21 618	6 718 (31.1)	2.00 (1.92, 2.10)**
Age in years			
20-34	15 641	2 843 (18.2)	1 (ref)
35-49	18 107	4 918 (27.2)	1.48 (1.40, 1.57)**
50-64	12 448	4 348 (34.9)	1.68 (1.58, 1.79)**
Living arrangement			
With a partner	30 869	8 697 (28.2)	1.22 (1.16, 1.28)**
Without a partner	15 327	3 412 (22.3)	1 (ref)
Length of time in Canada			
0-9 years	2 859	356 (12.5)	1 (ref)
10 years or more	6 710	1 391 (20.7)	1.42 (1.25, 1.62)**
Canadian-born	36 626	10 361 (28.3)	2.63 (2.33, 2.96)**
Income level			
Lowest 30%	12 195	3 207 (26.3)	1.12 (1.05, 1.19) *
Middle 40%	18 740	5 081 (27.1)	1.12 (1.06, 1.18) **
Highest 30%	15 261	3 821 (25.0)	1 (ref)
Education level			
Less than high school	4 856	1 867 (38.4)	1.60 (1.49, 1.72)**
High school graduate	7 253	2 032 (28.0)	1.22 (1.15, 1.30)**
Some post-secondary	4 156	1 066 (25.6)	1.14 (1.06, 1.24)*
Post secondary graduate	29 930	7 144 (23.9)	1 (ref)
Number of chronic conditions			
None	16 643	3 125 (18.8)	1 (ref)
One	13 510	3 311 (24.5)	1.35 (1.27, 1.43)**
Two	7 807	2 256 (28.9)	1.61 (1.50, 1.72)**
Three	4 197	1 484 (35.4)	2.09 (1.93, 2.26)**
Four or more	4 039	1 933 (47.9)	3.27 (3.00, 3.56)**
Functional limitations			
None	41 583	10 246 (24.6)	1 (ref)
Any	4 586	1 860 (40.6)	1.28 (1.19, 1.38)**
Physical activity index			
Active	11 597	2 086 (18.0)	1 (ref)
Moderately active	12 002	2 897 (24.1)	1.40 (1.31, 1.49)**
Inactive	22 596	7 125 (31.5)	2.01 (1.89, 2.13)**
Type of smoker			
Daily	9 480	2 238 (23.6)	0.58 (0.55, 0.61)**
Occasionally	2 715	495 (18.2)	0.63 (0.56, 0.69)**
Not at all	34 001	9 376 (27.6)	1 (ref)

Abbreviations: BMI, body mass index; CI, confidence interval; *p*, significance level; ref, reference.

* $p < .005$, ** $p < .0005$

TABLE 2 (*continued*)
Association between obesity (BMI ≥ 30) and socio-demographic and economic characteristics and mental health indicators in Canadians 20 to 64 years old (*n* = 46 196 obese or adequate weight)

	Total	Obese *n* (%)	Odds Ratio (95% CI)
Block 2:			
Mood and/or anxiety disorder			
Neither	41 858	10 549 (25.2)	1 (ref)
Mood only	1 994	768 (38.5)	1.50 (1.36, 1.66)**
Anxiety only	1 233	359 (29.1)	1.02 (0.89, 1.17)
Mood and anxiety	1 059	417 (39.4)	1.48 (1.29, 1.69)**

Abbreviations: BMI, body mass index; CI, confidence interval; *p*, significance level; ref, reference.

* $p < .005$, ** $p < .0005$

persons with both disorders (OR = 0.94, 95% CI = 0.83, 1.08) compared to those with neither disorder.

Discussion

This research examined a range of socio-demographic, economic, physical health and behavioural correlates of obesity using a nationally representative sample of Canadians. It then examined the associations between obesity and mood and anxiety disorders while controlling for the above factors. The above associations were examined in the total sample as well as in men and women samples separately.

Findings of this research revealed significantly elevated levels of obesity in men compared with women, Canadian born compared with recent immigrants, persons living with a partner compared to those not living with a partner, non-smokers compared with smokers, and persons with functional limitations compared to those with no such limitations. Findings also revealed an educational gradient in prevalence of obesity. Further, prevalence of obesity was positively associated with advancing age and number of chronic physical conditions. When adjusting for the above variables, the odds of obesity remained significantly higher in persons with mood disorders (with or without anxiety) compared to those with neither mood nor anxiety disorders.

Based on the results of this study, about 16.6% of the Canadian population 20 to 64 years old were classified as obese (BMI ≥ 30). This is slightly higher than rates reported for European countries[1] and much lower than the 31% observed in the US population.[36] However, the fact that the Canadian obesity rate was based on self-reported data while the US rate was based on measured data could explain some of the difference between the two rates. The association between obesity and physical inactivity, less smoking, advancing age, low income, and lower levels of education are consistent with findings of previous studies (e.g. Bryan and Walsh,[7] Perez,[6] Simon et al.,[20] Torrance et al.[8]). The strong association between obesity and functional limitations and chronic physical conditions are also in line with findings of previous studies (e.g. Larsson et al.,[10] Li et al.[9]).

Results of this research indicated a moderate association between obesity and mood disorders. This result is in line with previously reported reports (e.g. Blaine,[16] Bruffaerts et al.,[1] Jorm et al.[18]). Simon and colleagues[20] reported similar association between obesity and lifetime diagnosis of mood disorders. In addition, the lack of apparent sex difference in the obesity-mood disorders relationship is consistent with results of other studies (e.g. Carr et al.,[24] Dong et al.,[25] Simon et al.[20]).

However, data used in this study revealed a non-significant association between obesity and anxiety disorders. This result contrasts with that reported by Simon and colleagues.[20] Differences in measurement and methodology may account for the different findings. In their study, Simon and colleagues[20] compared the prevalence of mental disorders among obese versus adequate/overweight individuals. They also used the criteria of the Diagnostic and Statistical Manual Disorders (DSM-IV) to identify people with these disorders.

With regard to the association between being overweight and having mood and/or anxiety disorders, findings of this study revealed a significant association of overweight with mood disorders and insignificant association of overweight with anxiety disorders. These findings are different from those reported by Bruffaerts et al.[1] and McLaren et al.[21] who did not find an association of overweight with having mood disorders.

Although the methodology of the present study does not allow for the examination of specific conceptual models that could account for the observed associations between obesity and poor psychological health, a brief discussion of possible explanations that have been put forward regarding them is warranted. One explanation highlights the role of poor physical health, increased functional limitations and interpersonal stressors, such as social stigma[30] experienced by obese persons as mediators in the obesity-poor mental health relationship. For example, Carr and colleagues[24] reported that once these physical and interpersonal stressors were controlled for, obese persons had better psychological health, compared with persons with adequate weight. A theoretical model, which stipulates a bidirectional causal pathway between obesity and depression and defines potential behavioural, cognitive, physiological and social mediators, has also been suggested.[31] Additionally, a genetic susceptibility to both obesity and depression has been proposed, whereby both conditions share some common

genes.[32,33] Stankard and colleagues[33] also emphasized the role of adverse childhood experiences in promoting both conditions. In their review of the literature on obesity and mood disorders, McIntyre and colleagues[11] concluded that both conditions share aspects of phenomenology, comorbidity, family history and biology. More research examining this potential explanation is warranted. If indeed, the relationship between obesity and mood and/or anxiety disorders is causal, more research is needed to establish the direction of this causal relationship.

A number of limitations should be considered when interpreting the findings of this research. First, BMI calculated from self-reported height and weight is known to be lower than that calculated from measured height and weight because of people's tendency to overestimate their height and underestimate their weight.[34] When Shields and colleagues[35] compared self-reported against measured heights and weights in a sample of the Canadian Community Health Survey participants, they concluded that the prevalence estimates of obesity calculated from self-reported data were approximately 9% and 6% lower than estimates based on measured data for men and women, respectively. Similar estimates were reported by Flegal and colleagues[36] who examined the difference between self-reported and measured obesity measures in the US surveys. Second, although the BMI classification system is a useful indicator for comparing body weight patterns and related health risks within and between populations, it does not take into account individual differences in body leanness and/or muscularity. Hence, the health risk associated with each BMI category varies considerably between individuals.

Third, the identification of individuals as having mood disorders and/or anxiety disorders was not done by clinicians. It was based on respondents' answers to the question of whether they had been diagnosed with the disorder. Given the frequently reported under-diagnosis of mental disorders, the data could possibly underestimate the prevalence of mood and anxiety disorders. Additionally, the survey did not include individuals living in nursing homes, mental institutions or chronic care hospitals; thus, the data could further underestimate the prevalence of both disorders. Fourth, participants were not asked about each mood disorder separately. Instead, depression, bipolar disorder, mania or dysthymia were combined in one question. Consequently, the association between obesity prevalence and impact of each of these disorders on quality of life cannot be inferred. Additionally, the cross-sectional nature of the data precluded an examination of the temporal sequence of onset of obesity and mood and anxiety disorders. Thus, a causal relationship between obesity and mood and/or anxiety disorders cannot be inferred.

Given these limitations, the present study determined the prevalence and correlates of obesity using the most up-to-date data available on a representative sample of Canadians. In addition, the study examined the association between obesity and mood and/or anxiety disorders while adjusting for a range of socio-demographic, economic and behavioural characteristics, physical health and smoking frequency. To the authors' knowledge, this is the first study to examine the associations between obesity and mood and anxiety disorders in this population.

Detrimental ramifications of the obesity epidemic are enormous, both to the individuals and society. Health care providers are encouraged to consider an integrated treatment modality to investigate depression in obese patients whereby psychologists and mental health care professionals participate in the assessment and treatment plan of obese patients.[37,31] There is an urgent need for behavioural interventions aimed at targeting unhealthy eating and physical inactivity, especially among the high risk populations identified in this study. There is also a need for more longitudinal research to clarify the temporal relationship between obesity and mood disorders as well as the biological, psychological and socio-demographic moderators and mediators of this important relationship. Specifically, as results of this study revealed a large difference in the prevalence of obesity between new immigrants and Canadian born, more research on the role of ethnicity as a determinant of obesity and possibly as a moderator in the obesity-mental health relationship needs further scrutiny.

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References

1. Bruffaerts R, Demyttenaere K, Vilagut G, Martinez M, Bonnewyn A, De Graaf R, Haro JM, Bernert S, Angermeyer MC, Brugha T, Roick C, Alonso J. The relation between body mass index, mental health, and functional disability: a European population perspective. Can J Psychiatry. 2008;53(10):679-88.

2. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999-2002. JAMA. 2004;291:2847-50.

3. Stein CJ, Colditz GA. The epidemic of obesity. J Clin Endocrinol Metab. 2004; 89:2522-5.

4. Vanasse A, Demers M, Hemiari A, Courteau J. Obesity in Canada: where and how many? Int J Obes. 2006;30:677-83.

5. 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.

6. Pérez CE. Fruit and vegetable consumption. Health Rep. 2002;13:23-31.

7. Bryan S, Walsh P. Physical activity and obesity in Canadian women. BMC Women's Health. 2004;4: Suppl 1:S6.

8. 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.

9. Li Z, Bowerman S, Heber D. Health ramifications of the obesity epidemic. Surg Clin North Am. 2005;85:681-701, v.

10. Larsson U, Karlsson J, Sullivan M. Impact of overweight and obesity on health-related quality of life – a Swedish population study. Int J Obes Relat Metab Disord. 2002; 26:417-24.

11. McIntyre RS, Konarski JZ, Wilkins K, Soczynska JK, Kennedy SH. Obesity in bipolar disorder and major depressive disorder: results from a national community health survey on mental health and well-being. Can J Psychiatry. 2006;51:274-80.

12. Taylor V, Macdonald K, McKinnon MC, Joffe RT, MacQueen GM. Increased rates of obesity in first-presentation adults with mood disorders over the course of four-year follow-up. J Affect Disord. 2008;109 (1-2):127-31.

13. Scott KM, McGee MA, Wells JE, Oakley Browne MA. Obesity and mental disorders in the adult general population. J Psychosom Res. 2008;64:97-105.

14. Faith MS, Matz PE, Jorge MA. Obesity-depression associations in the population. J Psychosom Res. 2002;53:935-42.

15. Atlantis E, Baker M. Obesity effects on depression: systematic review of epidemiological studies. Int J Obes. 2008;32(6):881-91.

16. Blaine B. Does depression cause obesity? A meta-analysis of longitudinal studies of depression and weight control. J Health Psychol. 2008;13(8):1190-7.

17. Faith MS, Flint J, Fairburn CG, Goodwin GM, Allison DB. Gender differences in the relationship between personality dimensions and relative body weight: Results from a British population-based sample. Obes Res. 2001;9:647-650.

18. Jorm AF, Korten AE, Christensen H, Jacomb PA, Rodgers B, Parslow RA. Association of obesity with anxiety, depression, and emotional well-being: a community survey. Aust N Z J Public Health. 2003;27:434-440.

19. Petry NM, Barry D, Pietrzak RH, Wagner JA. Overweight and obesity are associated with psychiatric disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Psychosom Med. 2008;70(3):288-97.

20. Simon GE, Von Korff M, Saunders K, Miglioretti DL, Crane PK, van Belle G, Kessler RC. Association between obesity and psychiatric disorders in the US adult population. Arch Gen Psychiatry. 2006;63(7):824-30.

21. McLaren L, Beck CA, Patten SB, Fick GH, Adair CE. The relationship between body mass index and mental health. A population-based study of the effects of the definition of mental health. Soc Psychiatry Psychiatr Epidemiol. 2008;43:63-71.

22. McElroy SL, Kotwal R, Malhorta S, Nelson EB, Keck PE, Nemeroff CB. Are mood disorders and obesity related? A review for the mental health professional. J Clin Psychiatry. 2004;65:634-651.

23. Carpenter KM, Hasin DS, Allison DB, Faith MS. Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicidal attempts: results from a general population survey. Am J Public Health. 2000;90(2):251-7.

24. Carr D, Friedman MA, Jaffe K. Understanding the relationship between obesity and positive and negative affect: the role of psychosocial mechanisms. Body Image. 2007;4(2):165-77.

25. Dong C, Sanchez LE, Price RA. Relationship of obesity to depression: a family-based study. Int J Obes Metab Disord. 2004; 28:780-95.

26. Gadalla T. Association of comorbid mood disorders and chronic illness with disability and quality of life in Ontario, Canada. Chronic Dis Can. 2008;28(4):148-154.

27. Gadalla TM. Disability associated with comorbid anxiety disorders in women with chronic physical illness in Ontario, Canada. Women Health. 2008;48(1):1-20.

28. Canadian community health survey, cycle 3.1, 2005 [Public Use Microdata Documentation]. Ottawa (ON): Statistics Canada. 2006.

29. Canadian community health survey, cycle 1.2 – Mental health and well-being [Public Use Microdata Documentation]. Ottawa (ON): Statistics Canada. 2003.

30. Schwartz MB, Chambliss HO, Brownell KD, Blair SN, Billington C. Weight bias among health professionals specializing in obesity. Obes Res. 2003;11:1033-9.

31. Markowitz S, Friedman MA, Arent SM. Understanding the relation between obesity and depression: causal mechanisms and implications for treatment. Clin Psychol-Sci Pr. 2008;15(1):1-20.

32. Kendler KS, Walters EE, Neale MC, Kessler RC, Heath AC, Eaves LJ. The structure of the genetic and environmental risk factors for six major psychiatric disorders in women. Phobia, generalized anxiety disorder, panic disorder, bulimia, major depression and alcoholism. Arch Gen Psychiatry. 1995;52:374-83.

33. Stunkard AJ, Faith MS, Allison KC. Depression and obesity. Biol Psychiatry. 2003;54:330-7.

34. Villanueva EV. The validity of self-reported weight in US adults: a population based cross-sectional study. BMC Public Health. 2001;1:11.

35. Shields M, Gorber SC, Tremblay MS. Estimates of obesity based on self-report versus direct measures. Health Reports. 2008;19(2):1-16.

36. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999-2000. JAMA. 2002;288:1723-7.

37. Bean MK, Stewart K, Olbrisch ME. Obesity in America: implications for clinical and health psychologists. J Clin Psychol Med Settings. 2008;15:214-24.

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