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Cause-specific mortality by occupational skill level in Canada: a 16-year follow-up study

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This article has been peer reviewed.

Abstract

Introduction: Mortality data by occupation are not routinely available in Canada, so we analyzed census-linked data to examine cause-specific mortality rates across groups of occupations ranked by skill level.

Methods: A 15% sample of 1991 Canadian Census respondents aged 25 years or older was previously linked to 16 years of mortality data (1991–2006). The current analysis is based on 2.3 million people aged 25 to 64 years at cohort inception, among whom there were 164 332 deaths during the follow-up period. Occupations coded according to the National Occupation Classification were grouped into five skill levels. Age-standardized mortality rates (ASMRs), rate ratios (RRs), rate differences (RDs) and excess mortality were calculated by occupational skill level for various causes of death.

Results: ASMRs were clearly graded by skill level: they were highest among those employed in unskilled jobs (and those without an occupation) and lowest for those in professional occupations. All-cause RRs for men were 1.16, 1.40, 1.63 and 1.83 with decreasing occupational skill level compared with professionals. For women the gradient was less steep: 1.23, 1.24, 1.32 and 1.53. This gradient was present for most causes of death. Rate ratios comparing lowest to highest skill levels were greater than 2 for HIV/AIDS, diabetes mellitus, suicide and cancer of the cervix as well as for causes of death associated with tobacco use and excessive alcohol consumption.

Conclusion: Mortality gradients by occupational skill level were evident for most causes of death. These results provide detailed cause-specific baseline indicators not previously available for Canada.

Keywords: *socio-economic status, differential mortality, occupational skill level, Canada*

Introduction

The relationship between an individual's occupation and mortality is well known. Findings from the Whitehall Study showed an inverse social gradient, where rates of coronary heart disease mortality were highest for British civil servants in occupations that required few or no skills, and lowest for those in occupations that required more specific skills, education or other qualifications.[1] Similar social gradients in mortality have been found in other countries and for other occupations.[2-7]

The association between health and occupation is complex. It has been theorized that occupation affects the health of people through both material and psychosocial pathways as well as by exposure to hazardous conditions or materials at the workplace.[7-12] For example, people in higher skilled occupations, which tend to be more highly paid, may have better access to material resources that support good health, such as good quality housing and food. Occupation may also have a positive or negative influence on health as a result of the particular demands and rewards associated with different types of work, such as social networks, work-based stress and level of autonomy and control over work conditions.[9,10,12-14] Exposures to hazardous materials at the workplace also vary by occupation and contribute to differences in mortality rates.

In Canada, large population-based studies examining mortality by occupation are less common than elsewhere. This is in part because the information about usual occupation that is included on death registrations in most provinces tends not to be captured in machine-readable form or coded. However, several record linkage-based follow-up studies have examined the association between occupation and mortality, with each showing higher mortality rates among occupations with lower skill levels.[15-18] Nevertheless, those results were limited by the scope of the population covered (geographically or by age, sex and/or occupation), small sample size, lack of information about causes of death or a combination of these factors.

Recently, Census data from a 15% sample of Canadian residents aged 25 years and older were linked to almost 16 years of mortality data.[19,20] Results based on the first 11 years of follow-up showed that mortality rates overall and for suicide, unintentional injuries and causes amenable to medical care were lower in each successively higher ranked occupational

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skill level.[19,21-23] However, the findings were not examined across a broad range of detailed causes of death.

The objective of this analysis is to use the full 16 years of linked data to examine mortality rates by occupational skill level among cohort members aged 25 to 64 years at baseline, using the Global Burden of Disease cause of death groupings, and to examine causes of death grouped by three risk factors (smoking, alcohol and drugs) and deaths before age 75 years that were potentially amenable to medical care.

Methods

Data source

This is a secondary analysis of data from the 1991 to 2006 Canadian Census Mortality and Cancer Follow-up Study.[19,20] Individuals were eligible for the cohort (''in-scope'') if they were 25 years or older when enumerated by the 1991 Census long-form questionnaire, which excluded residents of institutions such as hospitals, nursing homes and prisons. To be followed for mortality, in-scope Census respondents first had to be linked to an encrypted name file abstracted from non-financial tax-filer data. About 80% of in-scope Census respondents (n = 2 860 244) were linked to the name file. A random sample (n = 125 409) was then removed so the final cohort (n = 2 734 835) would be a 15% sample of the 1991 Canadian population aged 25 years or older, as stipulated in the record linkage protocol. This cohort was then matched to the Canadian mortality database (4 June 1991 to 31 December 2006) using probabilistic record linkage methods primarily based on names and dates of birth.[24] In the absence of a match to a death registration, follow-up status (alive, dead, emigrated, or lost to follow-up) could usually be determined from tax-filer data.[20] Additional details on the construction and contents of the linked file are reported elsewhere.[19,20] For this study, the analysis was restricted to people aged 25 to 64 years at cohort inception (n = 2 312 400). Almost 2 million people in this age range had a coded occupation, and of those with a coded occupation, 6% died during the follow-up period. About 313 400 cohort members aged 25 to 64 years did not have a coded occupation. Table 1 shows the number of cohort members, person-years at risk and deaths ascertained by occupational skill level, age group and sex.

Definitions

Occupation was coded based on the kind of work an individual was doing the week prior to the 1991 Census enumeration, or if the person did not have a job that week, based on the job of longest duration since 1 January 1990. Respondents were asked to specify the kind of work they were doing and the most important activities or duties of their job.[25] This information was then coded to an occupational category based on the 1990 National Occupational Classification.[26] The skill level of each occupation was then assigned to one of the following categories: professional, managerial, skilled/technical/supervisory, semi-skilled or unskilled. Skill level was broadly defined as the amount and type of education and training required to enter and perform the duties of an occupation. In the National Occupational Classification, managerial occupations are not assigned a skill level because factors other than education and training (such as previous experience) are often more significant determinants of managerial employment. For the purposes of this study, managers were ranked between professional and supervisory occupations. People who had not worked within the reference period were retained as a separate ''no occupation'' category, which included long-term unemployed, mature students, stay-at-home parents, people who were unable to work, retirees and others who had not worked in the reference period.

Analytical techniques

For each cohort member, person-days of follow-up were calculated from the day of the Census (4 June 1991) to the date of death, date of emigration or the last day of the study period (31 December 2006). Person-days of follow-up were divided by 365.25 to obtain person-years at risk. Age at baseline-, sex- and occupational skill level-specific mortality rates by 5-year age groups were used to calculate age-standardized mortality rates (ASMRs), using the cohort population structure (person-years at risk), both sexes together, as the standard population.

Relative inequalities were assessed by rate ratios (RRs) and percent excess mortality. RRs were calculated by dividing the ASMR for a specific occupation level (unskilled, semi-skilled, skilled/technical/supervisory, managerial) by the ASMR for those in professional occupations. RRs greater than one indicate an increased mortality risk. Percent excess mortality was calculated by subtracting the ASMR for those in professional occupations from the ASMR for all cohort members with any occupation, then dividing by the ASMR for all occupationally-active cohort members and multiplying by 100.

Absolute inequalities were assessed by rate differences (RDs) and absolute excess mortality. RDs were calculated by subtracting the ASMR for unskilled, semi-skilled, skilled/technical/supervisory, and managerial occupations, respectively, from the ASMR for those in professional occupations. RDs greater than zero indicate excess mortality. Absolute excess mortality was calculated by subtracting the ASMR of those in professional occupations from the ASMR for all cohort members with an occupation. The difference represents the number of deaths (per 100 000) that could hypothetically have been avoided if all occupationally active cohort members had experienced the mortality rates of those in professional occupations.

For ASMRs, RRs and RDs, 95% confidence intervals (CIs) were calculated according to previously described methods.[27]

Mortality data included underlying cause of death coded based on the World Health Organization's ICD-9 (*International Classification of Diseases, 9th Revision*[28]) for deaths prior to 2000, and on ICD-10 (*International Classification of Diseases, 10th Revision*[29]) for deaths between 2000 and 2006. Deaths were grouped by Global Burden of Disease categories.[30] Using conservative definitions, causes of death

TABLE 1
Cohort members, person-years at risk and deaths ascertained, by age group, sex and occupational skill level at baseline, Canada, 1991–2006

	Men			Women		
	Cohort members, n	PYAR	Deaths ascertained, n	Cohort members, n	PYAR	Deaths ascertained, n
Age 25–64 years (at baseline)						
No occupation	85 000	1 112 820	25 469	228 400	3 319 420	24 048
All occupations	1 073 900	15 872 090	79 176	925 100	13 924 000	35 639
Professional	140 300	2 070 010	6 946	158 100	2 381 480	4 445
Managerial	153 400	2 267 990	10 020	64 400	966 430	2 405
Skilled/technical/supervisory	375 600	5 573 320	27 508	252 300	3 805 080	9 411
Semi-skilled	294 500	4 351 650	23 592	352 500	5 304 770	14 241
Unskilled	110 100	1 609 130	11 110	97 800	1 466 250	5 137
Age 25–44 years (at baseline)						
No occupation	25 000	353 230	2 493	118 600	1 764 980	3 854
All occupations	700 600	10 489 520	20 574	646 500	9 772 560	11 569
Professional	92 800	1 373 680	1 839	113 900	1 718 170	1 653
Managerial	91 100	1 360 990	2 233	44 400	669 300	760
Skilled/technical/supervisory	246 000	3 700 050	7 010	177 700	2 693 240	3 068
Semi-skilled	200 300	3 004 390	6 767	247 300	3 739 050	4 655
Unskilled	70 400	1 050 410	2 725	63 100	952 800	1 433
Age 45–64 years (at baseline)						
No occupation	60 100	759 590	22 976	109 700	1 554 440	20 194
All occupations	373 400	5 382 570	58 602	278 600	4 151 440	24 070
Professional	47 400	696 330	5 107	44 100	663 310	2 792
Managerial	62 300	907 000	7 787	20 000	297 120	1 645
Skilled/technical/supervisory	129 700	1 873 270	20 498	74 600	1 111 840	6 343
Semi-skilled	94 200	1 347 260	16 825	105 200	1 565 720	9 586
Unskilled	39 700	558 720	8 385	34 700	513 450	3 704

Source: 1991–2006 Canadian Census Mortality and Cancer Follow-up Study.[20]

Abbreviation: PYAR, person-years at risk.

were grouped by behavioural health risk factors, namely smoking-related diseases[2] (e.g. cancers of buccal cavity, pharynx, esophagus, larynx, trachea, bronchus, lung, chronic obstructive pulmonary disease), alcohol-related diseases[2] (e.g. alcoholic psychosis, alcoholic cirrhosis of liver and pancreas, accidental poisoning by alcohol) and drug-related diseases[31] (e.g. accidental poisoning by narcotics and other drugs, drug use disorders). We also examined deaths among those aged less than 75 years that were potentially amenable to medical intervention, such as deaths due to cerebrovascular disease, hypertension, breast cancer and pneumonia/influenza.[2,32] The detailed definitions of the cause groupings are available on request.

The Canadian Census Mortality and Cancer Follow-up Study was approved by the Statistics Canada Policy Committee, after consultations with the Statistics Canada Confidentiality and Legislation Committee, the Data Access and Control Services Division, and the Federal Privacy Commissioner.

Results

Of the 2.3 million cohort members aged 25 to 64 years at cohort inception, 7% of men and 20% of women had no occupation coded by the census. Of the 2 million cohort members with a reported occupation, 13% of men and 17% of women were in professional occupations; 14% of men and 7% of women were in managerial positions; 35% of men and 27% of women were in skilled, technical or supervisory occupations; and 27% of men and 38% of women were in semi-skilled occupations. The remaining 10% for men and 11% for women were in unskilled occupations (see Table 1).

As shown in Table 2, for cohort members of both sexes, ASMRs for all causes of death were graded by occupational skill level, with higher mortality rates for those in less skilled occupations. Compared with men in professional occupations, the RRs were 1.16 for men in managerial occupations, 1.40 for men in skilled, technical or supervisory occupations, 1.63 for men in semi-skilled occupations and 1.83 for men in unskilled occupations. For women, the

TABLE 2
Number of deaths, age-standardized mortality rates per 100 000 person-years at risk, rate ratios and rate differences, by occupational skill level and sex, cohort members aged 25 to 64 years at baseline, Canada, 1991–2006

	Deaths	ASMR	95% CI	RR	95% CI	RD	95% CI
Men							
Professional (Reference group)	6 946	372.8	363.9–382.0	1.00	—	0.0	—
Managerial	10 020	433.5	424.8–442.3	1.16*	1.13–1.20	60.7*	48.1–73.3
Skilled/technical/supervisory	27 508	521.6	515.4–527.8	1.40*	1.36–1.44	148.8*	137.8–159.7
Semi-skilled	23 592	606.9	599.1–614.8	1.63*	1.58–1.67	234.1*	222.1–246.0
Unskilled	11 110	680.8	668.2–693.7	1.83*	1.77–1.88	308.0*	292.4–323.7
No occupation	25 469	1 331.4	1 307.9–1 355.3	3.57*	3.47–3.68	958.6*	933.2–984.0
Women							
Professional (Reference group)	4 445	237.7	230.1–245.7	1.00	—	0.0	—
Managerial	2 405	293.5	281.3–306.2	1.23*	1.17–1.30	55.7*	41.0–70.4
Skilled/technical/supervisory	9 411	294.9	288.7–301.1	1.24*	1.19–1.29	57.1*	47.2–67.1
Semi-skilled	14 241	314.6	309.3–320.0	1.32*	1.28–1.37	76.8*	67.4–86.3
Unskilled	5 137	364.1	354.1–374.3	1.53*	1.47–1.60	126.3*	113.6–139.1
No occupation	24 048	522.0	514.6–529.5	2.20*	2.12–2.28	284.3*	273.5–295.0

Source: 1991–2006 Canadian Census Mortality and Cancer Follow-up Study.[20]

Abbreviations: ASMR, age-standardized mortality rate; CI, confidence interval; RD, rate difference; RR, rate ratio.

Notes: Reference population (person-years at risk) for age standardization was taken from internal cohort age distribution (5-year age groups).

— : not applicable.

* Significantly different from Professional ($p < .05$).

corresponding RRs were 1.23, 1.24, 1.32 and 1.53, respectively. For those without an occupation, the RRs were 3.57 for men and 2.20 for women. The RD comparing professional to other occupational skill levels was greatest for those in unskilled occupations (308 per 100 000 for men; 126 per 100 000 for women).

The mortality gradient by occupational skill level differed by cause of death groupings (Tables 3 and 4). For men, RRs comparing unskilled to professional occupations were greater than 2 for deaths due to alcohol use disorders (3.94), chronic obstructive pulmonary disease (2.74), trachea, bronchus and lung cancers (2.69), unintentional injuries (2.56), cirrhosis (2.44), diabetes mellitus (2.24) and suicide (2.11) (Table 3). By contrast, the gradient was reversed for HIV/AIDS deaths (0.68). The RR for dementias was not statistically significant (1.17).

For women, RRs comparing unskilled to professional occupations were greater than 2 for deaths due to cervix uteri cancer (3.19), diabetes mellitus (2.54), alcohol use disorders (2.42), ischemic heart disease (2.29), trachea, bronchus and lung cancers (2.24), chronic obstructive pulmonary disease (2.06) and cirrhosis (2.05) (Table 4). By contrast, the gradient was reversed for breast cancer (0.85). RRs were not statistically significant for stomach cancer (1.35), dementias (1.28), respiratory infections (1.24), colon and rectal cancers (1.13) or ovarian cancer (0.91).

The percentage excess mortality related to occupational skill level is shown in the last column of Tables 3 and 4. If all occupationally active cohort members had experienced the ASMRs of those in professional occupations, then the all-cause ASMR would have been 29% lower for men and 21% lower for women, representing 155 and 64 fewer deaths per 100 000 person-years at risk, respectively. About half of this excess mortality was due to deaths from cardiovascular diseases and cancers of the trachea, bronchus and lung.

Causes of death were also grouped by risk factor (smoking-related diseases, alcohol-related diseases and drug-related diseases). For smoking-related diseases, the RR was 2.61 for men in unskilled occupations compared with those in professional occupations (Table 3). For women, the corresponding RR was 2.15 (Table 4). The RRs for alcohol- and drug-related disease deaths were also elevated (3.41 and 2.68 for men; 2.35 and 2.07 for women). The RRs for deaths prior to age 75 years that were potentially amenable to medical intervention were 1.45 for men and 1.11 for women.

Table 5 presents ASMRs for all causes and for selected cause of death groupings, by occupational skill level, age group at baseline and sex. In terms of RRs, the mortality gradient by occupational skill level was slightly steeper for those aged 25 to 44 years (at baseline) compared with those aged 45 to 64 years. For men, the RR was 2.19 at ages 25 to 44 years compared with 1.72 for those aged 45 to 64 years. For women, the RR was 1.65 at ages 25 to 44 years compared with 1.49 at ages 45 to 64 years. Although RRs across occupational skill levels were higher in the 25- to 44-year age group, absolute differences were greater for those aged 45 to 64 years.

TABLE 3
Age-standardized mortality rates per 100 000 person-years at risk, rate ratios and excess mortality for selected causes of death, by occupational skill level, male cohort members aged 25 to 64 years at baseline, Canada, 1991–2006

Cause	ASMR		Rate ratios (compared with Professional)				Excess[a]	
	All Occupations	Professional[b]	Managerial	Skilled/ Technical/ Supervisory	Semi-skilled	Unskilled	Rate per 100 000	Percent Excess,[c] %
All causes	528.2	372.8	1.16*	1.40*	1.63*	1.83*	155.4	29.4
Communicable diseases	15.6	15.3	0.87	0.88	1.24*	1.24*	0.4	2.4
HIV/AIDS	5.8	8.4	0.64*	0.52*	0.81*	0.68*	−2.6	−44.3
Respiratory infections	4.5	3.1	1.01	1.31	1.89*	1.90*	1.4	30.2
Non-communicable diseases	436.2	306.2	1.19*	1.41*	1.64*	1.81*	130.0	29.8
Malignant neoplasms	207.1	149.6	1.22*	1.40*	1.54*	1.67*	57.5	27.7
Stomach cancer	8.1	5.3	1.33*	1.61*	1.61*	1.85*	2.7	33.8
Colon and rectal cancers	22.4	18.4	1.18*	1.24*	1.29*	1.31*	4.1	18.2
Liver cancer	5.3	4.4	1.13	1.15	1.22	1.64*	0.9	17.0
Pancreatic cancer	11.3	8.8	1.43*	1.25*	1.38*	1.38*	2.5	22.4
Trachea, bronchus, and lung cancers	64.9	33.5	1.44*	1.94*	2.38*	2.69*	31.5	48.5
Prostate cancer	12.6	9.3	1.30*	1.47*	1.37*	1.38*	3.3	25.9
Diabetes mellitus	13.9	9.1	1.17	1.37*	1.88*	2.24*	4.8	34.3
Neuropsychiatric conditions	15.4	13.3	0.83*	1.15	1.26*	1.56*	2.1	13.3
Alcohol use disorders	3.2	1.6	0.95	2.01*	2.33*	3.94*	1.6	49.9
Alzheimer disease and other dementias	3.7	3.5	0.75	1.06	1.14	1.17	0.2	4.1
Cardiovascular diseases	148.6	102.0	1.20*	1.44*	1.70*	1.86*	46.5	31.3
Ischemic heart disease	99.9	67.5	1.22*	1.45*	1.75*	1.91*	32.4	32.5
Cerebrovascular disease	18.1	12.0	1.21*	1.52*	1.76*	1.93*	6.2	34.0
Respiratory diseases	17.1	8.9	1.21	1.82*	2.59*	2.60*	8.2	47.8
Chronic obstructive pulmonary disease	11.1	5.3	1.27	1.98*	2.99*	2.74*	5.8	52.5
Digestive diseases	19.0	12.9	1.01	1.41*	1.75*	2.26*	6.1	32.0
Cirrhosis	10.2	6.6	1.02	1.46*	1.91*	2.44*	3.6	35.5
Injuries	52.2	32.0	1.08	1.66*	1.90*	2.39*	20.2	38.7
Unintentional injuries	28.3	16.4	1.13	1.80*	1.96*	2.56*	11.9	42.0
Road traffic accidents	10.6	6.9	1.06	1.60*	1.83*	1.94*	3.8	35.3
Intentional injuries	22.3	14.8	0.98	1.49*	1.79*	2.16*	7.5	33.6
Suicide	20.8	14.1	0.96	1.48*	1.74*	2.11*	6.8	32.5
Smoking-related diseases[d]	91.0	48.3	1.38*	1.86*	2.34*	2.61*	42.7	46.9
Alcohol-related diseases[d]	10.9	5.7	1.01	1.88*	2.35*	3.41*	5.2	47.7
Drug-related diseases[d]	4.7	3.4	0.80	1.22	1.56*	2.68*	1.3	27.4
Amenable to medical intervention[d] (<75 years[e])	40.1	34.1	0.97	1.14*	1.34*	1.45*	6.1	15.1

Source: 1991–2006 Canadian Census Mortality and Cancer Follow-up Study.[20]

Abbreviation: ASMR, age-standardized mortality rate.

Note: Reference population (person-years at risk) for age-standardization was taken from the internal cohort age distribution (5-year age group).

[a] Excess (All occupations − Professional).

[b] Reference group.

[c] Percent excess [100 × (All occupations − Professional)/All occupations].

[d] Detailed ICD codes are available on request.

[e] Deaths before age 75 years that were potentially amenable to medical intervention, e.g. due to cerebrovascular disease, hypertension, breast cancer and pneumonia/influenza.

* Significantly different from rate for Professional ($p < .05$).

TABLE 4
Age-standardized mortality rates per 100 000 person-years at risk, rate ratios and excess mortality for selected causes of death, by occupational skill level, female cohort members aged 25 to 64 years at baseline, Canada, 1991–2006

Cause	ASMR		Rate ratios (compared with Professional)				Excess[a]	
	All Occupations	Professional[b]	Managerial	Skilled/ Technical/ Supervisory	Semi-skilled	Unskilled	Rate per 100 000	Percent Excess,[c] %
All causes	301.7	237.7	1.23*	1.24*	1.32*	1.53*	64.0	21.2
Communicable diseases	6.2	4.9	1.44	1.09	1.34*	1.59*	1.3	20.4
HIV/AIDS	0.3	0.5	—[d]	0.49	0.94	—[d]	−0.1	−32.5
Respiratory infections	2.4	2.4	1.12	0.74	1.07	1.24	0.0	−1.4
Non-communicable diseases	262.1	205.6	1.23*	1.26*	1.33*	1.52*	56.5	21.6
Malignant neoplasms	162.5	135.9	1.24*	1.22*	1.21*	1.31*	26.6	16.3
Stomach cancer	3.4	3.3	0.92	1.02	1.02	1.35	0.2	4.4
Colon and rectal cancers	13.5	12.5	1.25	1.09	1.07	1.13	1.0	7.6
Liver cancer	2.2	1.4	1.25	1.54	1.68*	1.85*	0.8	34.6
Pancreatic cancer	7.8	6.5	1.62*	1.25	1.16	1.37*	1.4	17.5
Trachea, bronchus, and lung cancers	40.5	22.5	1.74*	1.76*	2.02*	2.24*	18.0	44.4
Female breast cancer	34.2	36.4	1.03	0.97	0.91*	0.85*	−2.2	−6.3
Cervix uteri cancer	3.1	1.6	2.04*	1.78*	2.00*	3.19*	1.5	47.6
Ovarian cancer	9.8	9.8	0.85	1.08	1.00	0.91	0.1	0.5
Diabetes mellitus	6.5	4.2	1.10	1.36	1.61*	2.54*	2.3	35.0
Neuropsychiatric conditions	9.9	8.9	1.10	1.05	1.15	1.35*	1.0	10.4
Alcohol use disorders	0.9	0.4	1.50	2.08*	2.30*	2.42*	0.4	49.5
Alzheimer disease and other dementias	3.5	2.6	1.10	1.54*	1.39	1.28	0.9	25.8
Cardiovascular diseases	52.9	36.1	1.32*	1.34*	1.60*	1.97*	16.8	31.8
Ischemic heart disease	26.4	16.4	1.48*	1.41*	1.77*	2.29*	9.9	37.6
Cerebrovascular disease	12.6	10.9	1.03	1.07	1.21*	1.45*	1.7	13.6
Respiratory diseases	10.1	6.7	1.09	1.55*	1.67*	1.80*	3.4	33.9
Chronic obstructive pulmonary disease	6.5	3.8	1.31	1.67*	1.89*	2.06*	2.6	40.5
Digestive diseases	9.9	6.5	1.13	1.38*	1.72*	2.06*	3.4	34.1
Cirrhosis	4.0	2.8	1.33	1.40	1.48*	2.05*	1.2	30.2
Injuries	16.7	13.6	0.97	1.07	1.26*	1.65*	2.6	15.5
Unintentional injuries	9.8	8.3	1.03	1.08	1.23	1.61*	1.5	15.2
Road traffic accidents	5.1	4.6	1.08	1.07	1.07	1.39*	0.4	8.4
Intentional injuries	6.1	5.3	0.91	0.99	1.25*	1.66*	0.8	13.1
Suicide	5.4	4.7	0.92	0.98	1.25	1.66*	0.7	12.8
Smoking-related diseases[e]	50.9	29.3	1.64*	1.70*	1.93*	2.15*	21.5	42.3
Alcohol-related diseases[e]	3.5	2.1	1.44	1.55*	1.82*	2.35*	1.4	39.2
Drug-related diseases[e]	3.3	2.8	0.71	1.03	1.31	2.07*	0.6	17.5
Amenable to medical intervention[e] (< 75 years[f])	57.6	54.1	1.07	1.06	1.08*	1.11*	3.5	6.0

Source: 1991–2006 Canadian Census Mortality and Cancer Follow-up Study.[20]

Abbreviation: ASMR, age-standardized mortality rate.

Note: Reference population (person-years at risk) for age-standardization was taken from the internal cohort age distribution (5-year age group).

[a] Excess (All occupations − Professional).

[b] Reference group.

[c] Percent excess [100 × (All occupations − Professional)/All occupations].

[d] Suppressed due to Statistics Canada disclosure rules.

[e] Detailed ICD codes are available on request.

[f] Deaths before age 75 years that were potentially amenable to medical intervention, e.g. due to cerebrovascular disease, hypertension, breast cancer and pneumonia/influenza.

* Significantly different from rate for Professional ($p < .05$).

TABLE 5
Age-standardized mortality rates per 100 000 person-years at risk, rate ratios and excess mortality for selected causes of death, by occupational skill level, age group, cohort members aged 25 to 64 years at baseline, Canada, 1991–2006

Sex, age group at baseline and cause	ASMR		Rate ratios (compared with Professional)				Excess[a]	
	All Occupations	Professional[b]	Managerial	Skilled/ Technical/ Supervisory	Semi-skilled	Unskilled	Rate per 100 000	Percent excess,[c] %
Men								
Age 25 to 44								
All causes	194.9	126.5	1.16*	1.49*	1.85*	2.19*	68.4	35.1
Unintentional injuries	25.9	12.5	1.25*	2.14*	2.38*	3.32*	13.4	51.7
Ischemic heart disease	28.4	16.5	1.36*	1.64*	2.20*	2.44*	11.9	42.0
Intentional injuries	24.1	14.9	1.04	1.57*	1.94*	2.39*	9.2	38.1
Trachea, bronchus, and lung cancers	12.4	6.2	1.38*	2.03*	2.62*	2.87*	6.2	50.1
Age 45 to 64								
All causes	1157.8	838.1	1.16*	1.37*	1.56*	1.72*	319.8	27.6
Trachea, bronchus, and lung cancers	164.2	85.0	1.45*	1.93*	2.35*	2.67*	79.1	48.2
Ischemic heart disease	235.1	163.9	1.20*	1.41*	1.66*	1.81*	71.2	30.3
Chronic obstructive pulmonary disease	30.3	14.5	1.30	2.00*	2.95*	2.65*	15.8	52.2
Cerebrovascular	43.8	28.6	1.22	1.55*	1.76*	1.96*	15.2	34.7
Women								
Age 25 to 44								
All causes	118.8	92.6	1.17*	1.23*	1.38*	1.65*	26.2	22.0
Trachea, bronchus, and lung cancers	13.0	6.8	1.82*	1.86*	2.25*	2.63*	6.2	47.7
Unintentional injuries	7.6	5.7	1.24	1.16	1.48*	1.91*	1.9	25.2
Ischemic heart disease	5.1	3.5	0.94	1.14	1.84*	2.35*	1.7	32.2
Intentional injuries	7.0	5.6	0.99	1.05	1.35*	1.91*	1.3	19.3
Age 45 to 64								
All causes	647.3	511.9	1.26*	1.24*	1.30*	1.49*	135.4	20.9
Trachea, bronchus and lung cancers	92.3	52.1	1.72*	1.74*	1.97*	2.14*	40.2	43.6
Ischemic heart disease	66.5	40.9	1.56*	1.45*	1.76*	2.29*	25.5	38.4
Chronic obstructive pulmonary disease	17.8	10.9	1.26	1.60*	1.82*	2.05*	6.9	38.7
Diabetes mellitus	15.8	10.4	1.11	1.33	1.57*	2.52*	5.4	34.4

Source: 1991–2006 Canadian Census Mortality and Cancer Follow-up Study.[20]

Abbreviations: ASMR, age-standardized mortality rate; RR, rate ratio.

Note: Reference population (person-years at risk) for age-standardization was taken from the cohort age distribution (5-year age group).

[a] Excess (All occupations − Professional).

[b] Reference group (RR=1.00 not shown).

[c] Percent excess [100 × (All occupations − Professional)/All occupations].

* Significantly different from rate for Professional ($p < .05$).

The causes of death that contributed the most to excess mortality differed somewhat by sex and age group. For cohort members aged 25 to 44 years, unintentional injuries were the largest contributor for men, while cancers of the trachea, bronchus and lung were the largest contributor for women. For both men and women aged 45 to 64, cancers of the trachea, bronchus and lung were the largest contributor.

Discussion

Substantial mortality gradients by occupational skill level were evident for most causes of death for both men and women. If all cohort members with an occupation had experienced the age-specific mortality rates of those in professional occupations, then the all-cause ASMR would have been 29% lower for men and 21% lower for women. For men, this would be equivalent to eliminating all deaths from cardiovascular diseases, while for women it would be equivalent to eliminating all deaths from both cardiovascular and respiratory diseases.

With few exceptions, mortality rates for the causes of death examined were associated with occupational skill level. However, the gradient and strength or magnitude of the association varied

considerably by cause of death. RRs were highest for causes of death more closely associated with health risk behaviours (such as smoking and excessive alcohol consumption) and lowest for causes not closely associated with those behaviours (such as breast and prostate cancer), and causes where less is known regarding prevention. Studies from Sweden[6] and the United States[33] have demonstrated similar results. Phelan et al.[33] found that socio-economic status was less strongly associated with causes of death that have low preventability. Although the pathways between occupation and health are complex, acting at both individual and ecological levels,[34,35] causes of death that are more preventable tended to demonstrate a closer association with socio-economic status. From an individual perspective, this may be in part because people with greater resources may be better able to adapt their behaviour to take advantage of new knowledge about risk factors or preventive measures.[36]

Reducing socio-economic inequalities in health is an explicit objective of health policies in Canada.[37] A strength of this study is that results are based on a large, broadly representative sample of Canadians aged 25 to 64 years at the time of the 1991 census. The large sample size allowed for analysis of mortality differences by occupational skill level within detailed cause of death groupings and for the detection of small effects. However, a person's occupation was only known at cohort inception (1991) and could have changed during the follow-up period (1991–2006); as such, the listed occupation may not necessarily represent a person's long-term occupation skill level.

This study was not intended to assess the relative importance of direct and indirect effects of occupation on mortality—for example, the extent to which differences in mortality by occupational skill level may be explained by associated differences in education and income. The data also did not include information on risk factors (such as smoking) and thus may have overestimated the direct effect of occupation on mortality. Nevertheless, other research concludes that socio-economic differences in various health outcomes (including mortality) largely persist even after controlling for behavioural risk factors.[38-40]

Conclusion

This is the first time that detailed cause-specific mortality rates by occupational skill level have been examined for Canada across a wide range of causes of death. Results from this study confirm what is known about mortality gradients by occupational skill level in the international literature, and help to quantify the importance of such inequalities in Canada.

We found that most causes of death showed substantial differences in mortality rates by occupational skill level. Causes of death that were more preventable, including those more closely associated with smoking and excessive alcohol consumption, tended to have steeper gradients compared with less preventable causes. With the extension of the 1991–2006 Canadian Census Mortality Study to include linkage to cancer incidence data, future work could examine the nature and extent of inequalities in cancer incidence and survival.

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References

1. Marmott MG, Bosma H, Hemingway H, Brunner E, Stansfeld S. Contribution of job control and other risk factors to social variations in coronary heart disease incidence. Lancet. 1997;350(9073):235-9.

2. Mackenbach JP, Stirbu I, Roskam AJ, et al. Socioeconomic inequalities in health in 22 European countries. N Engl J Med. 2008;358(23):2468-81.

3. Langford A, Johnson B. Trends in social inequalities in male mortality, 2001-08. Intercensal estimates for England and Wales. Health Stat Q. 2010;(47):5-32.

4. Saurel-Cubizolles MJ, Chastang JF, Menvielle G, Leclerc A, Luce D; EDISC group. Social inequalities in mortality by cause among men and women in France. J Epidemiol Community Health. 2009;63(3): 197-202.

5. Weires M, Bermejo JL, Sundquist K, Sundquist J, Hemminki K. Socio-economic status and overall and cause-specific mortality in Sweden. BMC Public Health. 2008;8:340.

6. Erikson R, Torssander J. Social class and cause of death. Eur J Public Health. 2008;18(5):473-8.

7. Commission on Social Determinants of Health. Closing the gap in a generation: health equity through action on the social determinants of health: final report of the Commission on Social Determinants of Health. Geneva (CH): World Health Organization; 2008 [cited 2012 Aug 15]. Available from: http://whqlibdoc.who.int/publications/2008/9789241563703_eng.pdf

8. Stringhini S, Dugravot A, Shipley M, et al. Health behaviours, socioeconomic status, and mortality: further analyses of the British Whitehall II and the French GAZEL prospective cohorts. PLoS Med. 2011;8(2): e1000419.

9. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socio-economic position (part 1). J Epidemiol Community Health. 2006;60(1):7-12.

10. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 2). J Epidemiol Community Health. 2006;60(2):95-101.

11. Lynch J, Kaplan G. Socioeconomic position. In: Berkman LF, Kawachi I, eds. Social Epidemiology. New York: Oxford University Press; 2000. p.13-35.

12. Mikkonen J, Raphael D. Social determinants of health: the Canadian facts. Toronto: York University School of Health Policy and Management; 2010 [cited 2012 Aug 15]. Available from: http://www.thecanadianfacts.org/The_Canadian_Facts.pdf

13. Karasek R, Baker D, Marxer F, Ahlbom A, Theorell T. Job decision latitude, job demands, and cardiovascular disease: a prospective study of Swedish men. Am J Public Health. 1981;71(7):694-705.

14. Shields M. Stress and depression in the employed population. Health Rep. 2006; 17(4):11-29.

15. Aronson KJ, Howe GR, Carpenter M, Fair ME. Surveillance of potential associations between occupations and causes of death in Canada, 1965-91. Occup Environ Med. 1999;56(4):265-9.

16. Chen J, Beavon D, Wilkins R. Mortality of retired public servants in Canada. Proceedings of the Social Statistics Section, Annual Meeting of the American Statistical Association. Chicago (IL): American Statistical Association; 1996. p.86-91.

17. Wigle DT, Semenciw RM, Wilkins K, et al. Mortality study of Canadian male farm operators: non-Hodgkin's lymphoma mortality and agricultural practices in Saskatchewan. J Natl Cancer Inst. 1990;82(7):575-82.

18. Mustard CA, Derksen S, Berthelot JM, Wolfson M, Roos LL. Age-specific education and income gradients in morbidity and mortality in a Canadian province. Soc Sci Med. 1997;45(3):383-97.

19. Wilkins R, Tjepkema M, Mustard C, Choinière R. The Canadian census mortality follow-up study, 1991 through 2001. Health Rep. 2008;19(3):25-43.

20. Peters PA, Tjepkema M. 1991-2011 Canadian Census Mortality and Cancer Follow-up Study. Proceedings of Statistics Canada Symposium 2010. Social Statistics: The Interplay among Censuses, Surveys and Administrative Data. 2011:150-6. Ottawa (ON): Statistics Canada. [Statistics Canada, Catalogue no. 11-522-XCB].

21. Mustard CA, Bielecky A, Etches J, et al. Suicide mortality by occupation in Canada, 1991-2001. Can J Psychiatry. 2010;55(6): 369-76.

22. Mustard CA, Bielecky A, Etches J, et al. Avoidable mortality for causes amenable to medical care, by occupation in Canada, 1991-2001. Can J Public Health. 2010; 101(6):500-6.

23. Burrows S, Auger N, Gamache P, Hamel D. Individual and area socioeconomic inequalities in cause-specific unintentional injury mortality: 11-year follow-up study of 2.7 million Canadians. Accid Anal Prev. 2012;45:99-106.

24. Fair M. Generalized Record Linkage System – Statistics Canada's record linkage software. Austrian J Stat. 2004;33(1&2):37-53.

25. Statistics Canada. 1991 Census dictionary. Ottawa (ON): Supply and Services Canada; 1992. [Statistics Canada, Catalogue No.: 92-301E].

26. Employment and Immigration Canada. National Occupational Classification: occupational descriptions. Ottawa (ON): Canada Communications Group; 1993.

27. Spiegelman M. Introduction to Demography. Revised edition. Cambridge (MA): Harvard University Press; 1968.

28. World Health Organization. Manual of the international statistical classification of diseases, injuries and causes of death. 9th rev. Geneva (CH): World Health Organization; 1977.

29. World Health Organization. International statistical classification of diseases and related health problems, 10th rev. Geneva (CH): World Health Organization; 1992.

30. World Health Organization. The global burden of disease: 2004 update. Geneva (CH): World Health Organization; 2008.

31. Deaths related to drug poisoning: England and Wales, 1999-2003. Health Stat Q. 2005 Spring;(25):52-9.

32. Stirbu I, Kunst AE, Bopp M, et al. Educational inequalities in avoidable mortality in Europe. J Epidemiol Community Health. 2010;64(10):913-20.

33. Phelan JC, Link BG, Diez-Roux A, Kawachi I, Levin B. ''Fundamental causes'' of social inequalities in mortality: a test of the theory. J Health Soc Behav. 2004;45(3): 265-85.

34. Krieger N. Workers are people too: societal aspects of occupational health disparities – an ecosocial perspective. Am J Ind Med. 2010;53(2):104-15.

35. Lipscomb HJ, Loomis D, McDonald MA, Argue RA, Wing S. A conceptual model of work and health disparities in the United States. Int J Health Serv. 2006(1);36:25-50.

36. Phelan JC, Link BG, Tehranifar P. Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. J Health Soc Behav. 2010; 51(Suppl):S28-40.

37. Chief Public Health Officer's report on the state of public health in Canada, 2008: Addressing health inequalities. Ottawa (ON): Minister of Health, 2008. [Health Canada, Catalogue No.: HP2-10/2008].

38. McGrail KM, van Doorslaer E, Ross NA, Sanmartin C. Income-related health inequalities in Canada and the United States: a decomposition analysis. Am J Public Health. 2009;99(10):1856-63.

39. Kim HJ, Ruger JP. Socioeconomic disparities in behavioral risk factors and health outcomes by gender in the Republic of Korea. BMC Public Health. 2010;10:195.

40. Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J. Socioeconomic factors, health behaviors, and mortality: results from a nationally representative prospective study of US adults. JAMA. 1998;279(21):1703-8.

Hospitalizations for unintentional injuries among Canadian adults in areas with a high percentage of Aboriginal-identity residents

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Abstract

Introduction: Injuries are a leading cause of death and morbidity. While individual Aboriginal identifiers are not routinely available on national administrative databases, this study examines unintentional injury hospitalization, by cause, in areas with a high percentage of Aboriginal-identity residents.

Methods: Age-standardized hospitalization rates (ASHRs) and rate ratios were calculated based on 2004/2005-2009/2010 data from the Discharge Abstract Database.

Results: Falls were the most frequent cause of injury. For both sexes, ASHRs were highest in high-percentage First Nations-identity areas; high-percentage Métis-identity areas presented the highest overall ASHR among men aged 20–29 years, and high-percentage Inuit-identity areas presented the lowest ASHRs among men of all age groups. Some causes, such as falls, presented a high ASHR but a rate ratio similar to that for all causes combined; other causes, such as firearm injuries among men in high-percentage First Nations-identity areas, presented a relatively low ASHR but a high rate ratio. Residents of high-percentage Aboriginal-identity areas have a higher ASHR for hospitalization for injuries than residents of low-percentage Aboriginal-identity areas.

Conclusion: Residents of high-percentage Aboriginal-identity areas also live in areas of lower socio-economic conditions, suggesting that the causes for rate differences among areas require further investigation.

Keywords: *First Nations, Métis, Inuit, Aboriginal people, injuries, hospitalization, Census, geographical methods*

Introduction

Aboriginal people in Canada (i.e., First Nations, Métis and Inuit) generally experience poorer health and lower life expectancy than the overall Canadian population;[1-9] they also experience high rates of mortality and morbidity due to injuries.[10-12] Unintentional injuries are important to study because they are considered largely preventable, are a leading cause of death and morbidity, have long-term health effects and are associated with large health care costs.[13]

Individual Aboriginal identifiers are not routinely available on national hospitalization or mortality databases that contain injury information. As a result, existing studies tend to either use provincial databases that do contain this information or a geographical approach. Provincial studies that use hospitalization data containing individual Aboriginal identifiers have been limited to those of the western provinces, where there is information on people registered under the Indian Act. For example, Karmali et al.[12] found that people with Registered Indian status had an unintentional trauma rate about 3 times higher than the general population in Alberta, while a Health Canada study that used hospitalization data for the western provinces found that First Nations had an unintentional injury rate 4 times higher than the general population.[11] We found no injury-specific studies for Métis or Inuit populations using national hospitalization data. However, using census-linked mortality data, Tjepkema et al.[5] found that Registered Indians and Métis were more likely to die due to external causes (i.e. injury) than the non-Aboriginal population.

Several studies have also used area-based approaches to examine injury hospitalization and mortality in regions with a high percentage of Aboriginal-identity residents. Fantus et al.[14] found that those living in First Nations communities in Ontario had an all-cause injury rate 2.5 times higher than northern Ontario communities and 3.0 times higher than southern Ontario communities. National hospitalization data (excluding Quebec) revealed higher rates of all-cause injury in areas with a high percentage of Aboriginal-identity residents.[15] Two studies focusing on children—one national study[16] and one in Newfoundland and Labrador[17]—found that rates of hospitalizations for unintentional injuries among children living in areas with a high

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percentage of Aboriginal residents were higher than those among children living in areas with a low percentage of Aboriginal residents. Furthermore, Peters[18] found that 52.0% of the total gap in life expectancy between residents of Inuit Nunangat and the rest of Canada was as a result of injuries.

In this study, we examined unintentional injury hospitalization, by cause, among adults (aged 20 years or older) living in areas where at least 33% of residents reported an Aboriginal identity. Our two purposes were to: (1) calculate rates of unintentional injury hospitalization, by age group, sex, and cause of injury for geographical areas with a relatively high percentage of residents who self-identify as First Nations, Métis or Inuit, and (2) compare these rates with those for areas with a low percentage of Aboriginal-identity residents. Our study differs from those of others (for example, Garner et al.,[4] Carrière et al.[15] and Oliver et al.[16]) as it focuses on unintentional injuries among adults, examines different causes of injury and compares results for high-percentage First Nations-, Métis- and Inuit-identity areas and low-percentage Aboriginal-identity areas.

Methods

Hospitalization data

Hospitalization data for 6 fiscal years, 2004/2005 to 2009/2010, came from the Discharge Abstract Database.[19] This file contains information on all in-patient acute-care hospital separations (due to discharges, deaths, sign-outs and transfers) in the Canadian provinces and territories excluding Quebec. For each separation, information is available on age, sex, residential postal code, the date of admission and discharge and diagnoses codes based on the *International Classification of Diseases 10th Revision, Canadian version* [ICD-10-CA].[20] (Data quality reports indicate that the accuracy of ICD-10-CA on separation records is high.[19]) Using a classification developed by the International Collaborative Effort on Injury Prevention,[21] we examined 12 categories of unintentional injury based on ICD-10-CA codes: cut/pierce, drowning/suffocation, falls, fire/hot substance (i.e. burns), firearms, machinery, motor vehicle traffic, other land transportation, natural/environmental, poisoning, injury due to being struck, and other (which includes categories such as overexertion, water transport accidents, exposure to electric transmission lines, etc). Because this last category contains heterogeneous causes, we did not analyze it specifically, but we do present the results in the tables for comparison. In addition, we excluded adverse effects due to drugs or medical care. Injury codes and examples for each category of unintentional injury are available on request.

Because separation records contain multiple diagnosis codes, more than one type of unintentional injury identified (e.g. fall and burn) could be identified. Also, patients transferred between hospitals would have multiple separation records, which would result in counting a single injury episode many times. To account for this, we counted discharge and admission occurring on the same day as a single injury episode. Thus, data represent injury episodes rather than the number of individuals injured, as it is possible that an individual was hospitalized for the same injury more than once over the six-year period.

Geozones method

Because the Discharge Abstract Database does not contain information on patients' Aboriginal identity, we used a geographical method[22] to determine Dissemination Areas (DAs) with a high percentage of residents identifying as Aboriginal (i.e. First Nations, Métis or Inuit) in the 2006 Census. DAs, which consist of one or more neighbouring dissemination blocks and have a population of 400 to 700, are the smallest geographical unit for which information from the census is available nationally. Following earlier Statistics Canada research into hospitalizations and Aboriginal identity in Canada,[15] DAs where at least 33% of the population reported an Aboriginal identity in the 2006 census are classified as areas with a relatively high percentage of Aboriginal-identity residents. The population is further classified as First Nations, Métis or Inuit based on the predominant Aboriginal-identity group. Excluding Quebec, 1929 DAs were classified as high-percentage First Nations identity, 186 as high-percentage Métis identity and 59 as high-percentage Inuit identity, with the Aboriginal population accounting for 80%, 55% and 81%, respectively, of the population in the DAs. In contrast, the Aboriginal population accounted for 3% of the population in low-percentage Aboriginal-identity areas. It has to be mentioned that, because many Aboriginal people do not live in the areas identified as being high-percentage Aboriginal identity and because these areas also contain individuals who do not report an Aboriginal identity, results of this study represent characteristics of areas of residence and not characteristics of individuals. The 4 types of areas—high-percentage First Nations-identity, Métis-identity or Inuit-identity DAs or low-percentage Aboriginal-identity DAs—differ according to several socio-economic characteristics (see Table 1, which includes only DAs for which those characteristics were available).

The Postal Code Conversion File[23] was used to determine the DA of residence for each hospital separation record based on the patient's residential postal code. Over 99% of hospital records were successfully assigned to a DA.

Results produced

Denominators were derived from the 2006 Census, which corresponds to the midpoint of the hospitalization data, and multiplied by 6 to account for the 6 years of hospitalization data. Because of small populations, global non-response or incompletely enumerated Indian Reserves, a small number of DAs lacked the detailed age and sex data needed to provide a complete denominator. To retain these DAs in the analysis, age and sex were estimated from total population counts or population estimates of incompletely enumerated Indian Reserves.

Rates (per 10 000 person-years) were age-standardized in 5-year age intervals according to the age distribution of the Aboriginal-identity population in the 2006

TABLE 1
Socio-economic characteristics of types of areas defined by Aboriginal identity group[a]

	High-percentage Aboriginal-identity DAs[a,b]			Low-percentage Aboriginal-identity DAs[a]
	High-percentage First Nations-identity DAs	High-percentage Métis-identity DAs	High-percentage Inuit-identity DAs	
Number of DAs, n	1288	178	56	38710
Aboriginal identity, %	79.9	54.7	81.4	2.8
Living in crowded conditions, %	19.7	8.1	27.4	3.2
Living in dwellings in need of major repairs, %	36.7	20.5	23.7	6.9
Population aged 25–64 years without high school diploma, %	42.1	32.6	41.5	14.4
Population aged $\geq$ 15 years who are unemployed, %	20.0	12.3	16.5	6.2
Population aged $\geq$ 15 years in the labour force, %	55.5	63.6	66.3	67.7
DA in CMA/CA, %	21.8	27.4	0.0	78.9
DA in strong/moderate MIZ[c], %	6.8	14.0	0.0	11.8
DA in weak/no MIZ, %	71.3	58.6	100.0	9.3
Mean household income (SD), $	22512 (10541)	32163 (10517)	41252 (14528)	47406 (25792)

Source: 2006 Census.

Abbreviations: CMA/CA, Census Metropolitan Area/ Census Agglomeration; DA, Dissemination Area; MIZ, Metropolitan Influence Zone.

[a] According to the 2006 Census, excluding Quebec.

[b] DAs where at least 33% of the population reported Aboriginal identity are classified as high-percentage Aboriginal identity. Classification as high-percentage First Nations, Métis or Inuit is based on the predominant group.

[c] The MIZ assigns a category to municipalities outside of a CMA/CA based on the percentage of the employed labour force that commute to work in a CMA/CA.

Census. They are presented for high-percentage First Nations-identity areas, high-percentage Métis-identity areas, high-percentage Inuit-identity areas, and low-percentage Aboriginal-identity areas, and are produced by cause of injury, sex and age group (20–29, 30–44, 45+ years). Rate ratios allow for the comparison of rates for high-percentage First Nations-, high-percentage Métis-, and high-percentage Inuit-identity areas relative to low-percentage Aboriginal-identity areas. According to Statistics Canada rules on confidentiality, rates and rate ratios were not shown in any cell in a table if the number of episodes for that cell was less than 10. For rates and rate ratios, 95% confidence intervals (CIs) were produced according to the assumption of log-normality.[24] Data manipulation and computations were done using statistical package SAS version 9.1.3 (SAS Institute Inc., Cary, NC, US).

Results

Slightly more than 730 000 episodes of unintentional injuries requiring hospitalization among adults aged 20 years plus were reported in the Canadian provinces and territories (excluding Quebec) for the 6 years of data (2004/2005-2009/2010), among which more than 26 000 occurred in areas with high percentage of Aboriginal-identity residents (Table 2).

Age-standardized hospitalization rates

Among men, overall age-standardized hospitalization rates (ASHRs) for injury were highest in high-percentage First Nations-identity areas (146/10 000 person-years; 95% CI: 144–148), followed by high-percentage Métis-identity areas (112/10 000 person-years; 95% CI: 108–116), high-percentage Inuit-identity areas (100/10 000 person-years; 95% CI: 95–107) and low-percentage Aboriginal-identity areas (55/10 000 person-years; 95% CI: 54–55) (Table 3). Among women, ASHRs were highest in high-percentage First Nations-identity areas (103/10 000 person-years; 95% CI: 102–105), followed by high-percentage Inuit-identity areas (87/10 000 person-years; 95% CI: 82–92), high-percentage Métis-identity areas (74/10 000 person-years; 95% CI: 71–77), and low-percentage Aboriginal-identity areas (37.2/10 000 person-years; 95% CI: 37.0–37.3). However, the patterns were more complex for specific sex–age combinations: in high-percentage First Nations-identity areas, ASHRs for total causes increase with age, from 133/10 000 (95% CI: 128–138) person-years for men aged 20 to 29 years to 158/10 000 (95% CI: 154–162) person-years for men aged 45 years plus and from 77/10 000 (95% CI: 73–81) person-years for women aged 20 to 29 years to 141/10 000 (95% CI: 138–145) person-years for women aged 45 years plus. In contrast, ASHRs in high-percentage Métis-identity areas decreased with age among men and presented a U-shape pattern among women. In high-percentage Inuit-identity areas, such a U-shape was observed for men and an increasing trend was observed for women. For all areas and both sexes, the highest rates were observed for the oldest age group, with the exception of men living in high-percentage Métis-identity areas for which the highest rates were observed among the youngest group aged 20 to 29 years.

Rates of hospitalizations for falls were high in all areas for both sexes and all age groups: for men, they accounted for about one-third of all hospitalizations, at 55/10 000 (95% CI: 54–56) in high-

TABLE 2
Number and percentage distribution of unintentional injury-hospitalizations by age group, sex, and by Aboriginal identity group[a], DAs, population aged ≥ 20 years, Canada excluding Quebec, 2004/2005–2009/2010

	Population ≥ 20 years							
	Total		20–29 years		30–44 years		≥ 45 years	
	n	%	n	%	n	%	n	%
Men	**349 426**		**49 991**		**71 817**		**227 618**	
Areas with high percentage of Aboriginal residents[b]								
First Nations	12 224	3.5	2458	4.9	3784	5.3	5982	2.6
Métis	709	0.2	209	0.4	191	0.3	309	0.1
Inuit	1867	0.5	397	0.8	507	0.7	963	0.4
Areas with low percentage of Aboriginal residents	334 626	95.8	46 927	93.9	67 335	93.8	220 364	96.8
Women	**380 960**		**19 879**		**35 083**		**325 998**	
Areas with high percentage of Aboriginal residents[b]								
First Nations	9736	2.6	1473	7.4	2164	6.2	6099	1.9
Métis	531	0.1	100	0.5	152	0.4	279	0.1
Inuit	1613	0.4	179	0.9	257	0.7	1177	0.4
Areas with low percentage of Aboriginal residents	369 080	96.9	18 127	91.2	32 510	92.7	318 443	97.7

Source: Discharge Abstract Database, 2004/2005–2009/2010.

Abbreviation: DA, Dissemination Area.

[a] The percentage of Aboriginal identity is provided by the 2006 Census.

[b] Dissemination areas where at least 33% of the population reported Aboriginal identity are classified as high-percentage Aboriginal identity. Classification as high-percentage First Nations, Métis or Inuit is based on the predominant group.

percentage First Nations-identity areas, 37/10 000 (95% CI: 35–40) in high-percentage Métis-identity areas, 35/10 000 (95% CI: 32–38) high-percentage Inuit-identity areas, and 21.3/10 000 (95% CI: 21.2–21.4) in low-percentage Aboriginal-identity areas; for women, they accounted for more than half, at 55/10 000 (95% CI: 54–56) in high-percentage First Nations-identity areas, 39/10 000 (95% CI: 37–41) in high-percentage Métis-identity areas, 49/10 000 (95% CI: 46–53) in high-percentage Inuit-identity areas and 22/10 000 (95% CI: 22–23) in low-percentage Aboriginal-identity areas. The proportion of hospitalizations due to falls increased with age: for men aged 45 years plus, this reason accounted for about half of all unintentional injuries; for women of the same age, it accounted for about two-thirds of all unintentional injuries, which is in line with results observed in the general population.[25]

Rates of hospitalization for motor vehicle, traffic and other land transportation injuries together accounted for about one-quarter of all hospitalizations among men and one-sixth of all hospitalizations among women. Variations of their combined rate were observed between age groups (i.e. they were much higher among individuals aged 20–29 years than among other age groups) and sex (i.e. they were higher among men). Also, the main contributor to their combined rate varied according to the predominant Aboriginal identity group: whereas in high-percentage Inuit-identity areas, hospitalizations for other land transport were more frequent than for motor vehicle traffic, this pattern was reversed in the other areas.

Among men, unintentional injuries due to poisoning and being struck had similar ASHRs for all ages combined within every area. Among women, injuries due to being struck were less frequent than poisoning. Other noteworthy causes of injuries include, among men, cut/pierce and environmental/natural for high-percentage First Nations-identity areas, high-percentage Métis-identity areas, and high-percentage Inuit-identity areas, as well as being burned by fire or a hot substance and injured by machinery for high-percentage First Nations-identity areas and high-percentage Métis-identity areas; and, among women, being cut/pierced, sustaining environmental/natural injuries and being burned by fire or a hot substance for high-percentage First Nations-identity areas, high-percentage Métis-identity areas, and high-percentage Inuit-identity areas.

Rate ratios

Rate ratios comparing areas with a high percentage of Aboriginal-identity residents with those with a low percentage of Aboriginal-identity residents vary according to the predominant Aboriginal-identity group, cause of injury, sex and age group (Table 4). The CIs for most rate ratios contain lower and higher bounds greater than 1.00, which means that the ASHRs observed in areas with a high percentage of Aboriginal-identity residents are significantly higher than those observed in areas with a low percentage of Aboriginal-identity residents. Among men, rate ratios for all causes combined are highest in high-percentage First Nations-identity areas (2.7; 95% CI: 2.6–2.7) followed by high-percentage Métis-identity areas (2.0; 95% CI: 2.0–2.1) and high-percentage Inuit-identity areas (1.8; 95% CI: 1.7–1.9). Among women, rate ratios are highest in high-percentage First Nations-identity

TABLE 3
Age-standardized hospitalization rates (per 10 000 person-years) for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	Total		20–29 years		30–44 years		≥ 45 years	
	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI
Men								
Total								
High % First Nations	145.94	144.13–147.77	132.93	127.77–138.29	142.13	137.67–146.74	157.54	153.50–161.68
High % Métis	111.76	107.71–115.97	137.57	124.68–151.79	106.05	97.20–115.70	100.46	93.89–107.49
High % Inuit	100.47	95.14–106.09	108.88	95.04–124.72	71.15	61.72–82.03	121.00	108.18–135.33
Low % Aboriginal	54.53	54.36–54.70	52.27	51.80–52.74	44.76	44.43–45.10	64.58	64.29–64.88
Cut/Pierce								
High % First Nations	6.08	5.73–6.47	9.03	7.76–10.51	6.58	5.67–7.63	3.78	3.18–4.50
High % Métis	4.41	3.65–5.33	8.32	5.58–12.41	3.16	1.90–5.25	3.03	2.00–4.61
High % Inuit	5.04	4.09–6.21	8.77	5.45–14.13	4.57	2.59–8.05	x	x
Low % Aboriginal	1.75	1.72–1.78	2.39	2.29–2.50	1.77	1.71–1.84	1.32	1.27–1.36
Drowning, Suffocation								
High % First Nations	1.09	0.92–1.28	x	x	0.79	0.52–1.21	1.80	1.42–2.28
High % Métis	0.74	0.45–1.19	x	x	x	x	1.21	0.68–2.15
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	0.40	0.39–0.42	0.19	0.16–0.22	0.18	0.16–0.21	0.73	0.70–0.76
Fall								
High % First Nations	54.56	53.50–55.64	29.18	26.82–31.75	45.93	43.42–48.57	78.24	75.48–81.10
High % Métis	37.21	34.97–39.59	27.29	21.89–34.02	31.63	26.98–37.09	48.41	44.08–53.17
High % Inuit	34.96	32.01–38.18	22.28	16.51–30.06	19.19	14.61–25.20	56.90	48.31–67.02
Low % Aboriginal	21.32	21.21–21.42	12.52	12.29–12.75	13.02	12.84–13.20	34.20	34.00–34.41
Fire/Hot substance								
High % First Nations	3.45	3.15–3.77	2.59	1.95–3.44	3.53	2.89–4.32	3.91	3.31–4.63
High % Métis	2.70	2.07–3.50	x	x	2.91	1.73–4.92	2.24	1.40–3.60
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	0.81	0.79–0.84	0.83	0.77–0.89	0.73	0.68–0.77	0.88	0.84–0.91
Firearm								
High % First Nations	1.01	0.86–1.20	1.79	1.27–2.52	1.08	0.75–1.56	0.46	0.27–0.76
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	0.19	0.18–0.21	0.45	0.41–0.50	0.16	0.14–0.18	0.07	0.06–0.08
Machinery								
High % First Nations	2.27	2.07–2.48	1.73	1.23–2.45	2.25	1.75–2.90	2.62	2.13–3.22
High % Métis	3.25	2.73–3.88	x	x	5.20	3.51–7.69	2.28	1.41–3.69
High % Inuit	1.62	1.11–2.35	x	x	x	x	x	x
Low % Aboriginal	1.31	1.29–1.34	1.30	1.23–1.38	1.34	1.28–1.40	1.31	1.26–1.35
Motor vehicle traffic								
High % First Nations	19.64	18.98–20.32	28.92	26.56–31.48	18.99	17.41–20.72	14.34	13.13–15.66
High % Métis	18.92	17.39–20.59	31.88	25.99–39.11	18.88	15.35–23.21	10.76	8.67–13.35
High % Inuit	6.08	4.78–7.73	10.69	6.96–16.41	x	x	5.08	2.94–8.76
Low % Aboriginal	7.52	7.46–7.58	10.53	10.32–10.74	6.72	6.59–6.86	6.31	6.21–6.41

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TABLE 3 (continued)
Age-standardized hospitalization rates (per 10 000 person-years) for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	Total		20–29 years		30–44 years		≥ 45 years	
	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI
Other land transportation								
High % First Nations	13.82	13.22–14.44	18.45	16.59–20.51	15.06	13.65–16.60	9.79	8.79–10.90
High % Métis	12.10	10.65–13.76	19.74	15.23–25.59	12.73	9.90–16.36	6.72	5.10–8.86
High % Inuit	18.09	15.83–20.67	33.25	26.01–42.50	11.72	8.23–16.68	14.11	10.22–19.48
Low % Aboriginal	5.39	5.33–5.45	7.37	7.19–7.55	5.45	5.34–5.57	4.08	4.00–4.16
Environmental/Natural								
High % First Nations	5.21	4.88–5.57	3.84	3.05–4.85	6.10	5.23–7.12	5.30	4.59–6.11
High % Métis	4.30	3.63–5.11	4.89	2.90–8.26	3.95	2.52–6.20	4.24	3.05–5.88
High % Inuit	5.39	4.25–6.83	x	x	5.80	3.55–9.48	7.09	4.46–11.27
Low % Aboriginal	0.97	0.94–0.99	0.77	0.71–0.83	0.80	0.75–0.84	1.24	1.20–1.29
Poisoning								
High % First Nations	9.69	9.23–10.18	8.16	6.96–9.57	11.22	10.02–12.57	9.32	8.36–10.38
High % Métis	6.28	5.39–7.33	9.37	6.43–13.67	5.70	3.91–8.31	4.84	3.56–6.58
High % Inuit	5.87	4.79–7.19	x	x	6.24	3.88–10.05	5.77	3.48–9.58
Low % Aboriginal	2.39	2.36–2.43	2.17	2.07–2.26	2.15	2.08–2.22	2.76	2.69–2.82
Struck								
High % First Nations	9.90	9.39–10.44	14.00	12.40–15.82	10.30	9.15–11.60	6.96	6.13–7.90
High % Métis	7.37	6.24–8.69	11.46	8.15–16.12	8.50	6.23–11.59	3.77	2.63–5.41
High % Inuit	6.28	4.89–8.07	11.18	7.29–17.17	4.75	2.75–8.18	4.53	2.50–8.20
Low % Aboriginal	3.84	3.78–3.90	5.72	5.57–5.88	3.92	3.82–4.02	2.58	2.52–2.65
Others[d]								
High % First Nations	19.22	18.59–19.88	14.86	13.20–16.72	20.30	18.66–22.09	21.04	19.57–22.61
High % Métis	13.63	12.32–15.08	15.94	11.94–21.28	12.75	9.92–16.39	12.95	10.68–15.71
High % Inuit	13.44	11.62–15.54	10.90	7.10–16.73	8.80	5.89–13.14	19.13	14.40–25.41
Low % Aboriginal	8.64	8.57–8.70	8.04	7.86–8.23	8.52	8.38–8.67	9.12	9.00–9.23
Women								
Total								
High % First Nations	103.47	101.95–105.02	77.32	73.47–81.37	79.37	76.09–82.78	141.24	137.54–145.04
High % Métis	73.63	70.51–76.87	58.98	50.94–68.28	52.14	46.14–58.92	101.93	95.50–108.78
High % Inuit	86.87	81.77–92.28	51.13	42.01–62.22	59.55	50.77–69.85	133.45	118.43–150.39
Low % Aboriginal	37.17	37.04–37.29	19.90	19.62–20.19	20.53	20.30–20.75	62.75	62.49–63.01
Cut/Pierce								
High % First Nations	1.53	1.34–1.76	2.26	1.67–3.04	1.79	1.35–2.37	0.86	0.59–1.24
High % Métis	1.68	1.27–2.22	3.62	2.01–6.54	x	x	x	x
High % Inuit	2.85	2.11–3.85	x	x	x	x	x	x
Low % Aboriginal	0.42	0.40–0.43	0.55	0.51–0.60	0.44	0.41–0.48	0.31	0.28–0.33
Drowning/Suffocation								
High % First Nations	0.55	0.43–0.70	x	x	x	x	0.99	0.72–1.36
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	0.25	0.23–0.26	0.09	0.07–0.11	0.11	0.10–0.13	0.47	0.44–0.49

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TABLE 3 (continued)
Age-standardized hospitalization rates (per 10 000 person-years) for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	Total		20–29 years		30–44 years		≥ 45 years	
	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI
Fall								
High % First Nations	54.74	53.71–55.78	24.99	22.84–27.34	30.61	28.60–32.76	94.75	91.81–97.78
High % Métis	39.14	37.12–41.27	20.78	16.23–26.60	20.92	17.25–25.38	66.82	61.90–72.13
High % Inuit	49.33	45.59–53.37	16.03	11.27–22.80	24.33	18.91–31.30	92.32	79.81–106.80
Low % Aboriginal	22.49	22.41–22.58	6.11	5.96–6.28	8.31	8.17–8.46	45.32	45.11–45.54
Fire/hot substance								
High % First Nations	1.46	1.25–1.70	1.78	1.28–2.50	1.39	1.01–1.91	1.32	0.99–1.75
High % Métis	1.93	1.45–2.58	x	x	2.45	1.39–4.32	1.65	0.98–2.76
High % Inuit	2.13	1.40–3.22	x	x	x	x	x	x
Low % Aboriginal	0.35	0.33–0.36	0.27	0.24–0.30	0.29	0.27–0.32	0.44	0.42–0.47
Firearm								
High % First Nations	x	x	x	x	x	x	x	x
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	0.01	0.01–0.02	0.03	0.02–0.04	0.01	0.01–0.02	0.01	0.00–0.01
Machinery								
High % First Nations	0.19	0.14–0.27	x	x	x	x	x	x
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	0.10	0.10–0.11	0.10	0.08–0.12	0.09	0.08–0.11	0.12	0.11–0.13
Motor vehicle traffic								
High % First Nations	14.73	14.14–15.34	19.52	17.63–21.61	14.21	12.87–15.70	12.22	11.10–13.46
High % Métis	9.53	8.37–10.84	13.25	9.72–18.06	9.10	6.80–12.19	7.60	5.82–9.92
High % Inuit	4.42	3.28–5.95	x	x	4.88	2.83–8.41	x	x
Low % Aboriginal	4.03	3.99–4.08	4.87	4.73–5.02	3.24	3.15–3.33	4.23	4.15–4.30
Other land transportation								
High % First Nations	4.91	4.54–5.32	6.56	5.50–7.82	5.15	4.37–6.08	3.68	3.08–4.40
High % Métis	3.95	3.07–5.09	4.28	2.48–7.37	3.24	1.98–5.29	4.39	3.08–6.26
High % Inuit	9.40	7.78–11.35	9.54	6.08–14.96	8.19	5.33–12.58	10.39	6.80–15.89
Low % Aboriginal	1.72	1.69–1.76	1.81	1.72–1.90	1.65	1.59–1.72	1.74	1.69–1.79
Environmental/Natural								
High % First Nations	2.30	2.06–2.56	2.15	1.59–2.93	1.98	1.52–2.59	2.66	2.18–3.26
High % Métis	1.53	1.08–2.16	x	x	x	x	2.17	1.33–3.53
High % Inuit	1.76	1.12–2.79	x	x	x	x	x	x
Low % Aboriginal	0.62	0.60–0.64	0.45	0.40–0.49	0.49	0.45–0.52	0.84	0.81–0.87
Poisoning								
High % First Nations	10.25	9.75–10.77	8.72	7.49–10.15	11.50	10.29–12.85	10.08	9.07–11.20
High % Métis	5.90	5.04–6.91	4.88	2.94–8.10	4.88	3.27–7.29	7.45	5.72–9.69
High % Inuit	5.05	3.97–6.41	7.15	4.23–12.08	4.93	2.80–8.70	x	x
Low % Aboriginal	2.28	2.25–2.32	1.79	1.71–1.88	1.91	1.85–1.98	2.92	2.86–2.98

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TABLE 3 (continued)
Age-standardized hospitalization rates (per 10 000 person-years) for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	Total		20–29 years		30–44 years		≥ 45 years	
	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI	ASHR	95% CI
Struck								
High % First Nations	3.06	2.77–3.37	3.77	3.00–4.76	3.04	2.45–3.77	2.62	2.14–3.22
High % Métis	2.51	1.89–3.32	3.96	2.25–6.97	x	x	2.22	1.39–3.53
High % Inuit	2.07	1.43–3.00	x	x	x	x	x	x
Low % Aboriginal	0.85	0.82–0.87	1.00	0.94–1.07	0.76	0.72–0.80	0.83	0.80–0.86
Others[d]								
High % First Nations	9.65	9.17–10.15	7.14	6.04–8.45	8.99	7.93–10.19	11.78	10.72–12.95
High % Métis	6.74	5.82–7.80	5.58	3.47–8.97	6.48	4.58–9.16	7.69	5.99–9.88
High % Inuit	9.39	7.76–11.36	6.08	3.45–10.71	7.24	4.62–11.37	13.36	9.30–19.19
Low % Aboriginal	4.04	4.00–4.09	2.83	2.73–2.95	3.21	3.12–3.30	5.54	5.45–5.62

Source: Discharge Abstract Database, 2004/2005–2009/2010.

Abbreviations: ASHR, age-standardized hospitalization rate; CI, confidence interval.

Note: "x" indicates that the data was suppressed to meet the confidentiality requirements of the Statistics Act.

[a] The percentage of Aboriginal identity is provided by the 2006 Census.

[b] Dissemination areas where at least 33% of the population reported Aboriginal identity are classified as high-percentage Aboriginal identity. Classification as high-percentage First Nations, Métis or Inuit is based on the predominant group.

[c] Categories of unintentional injury based on ICD-10-CA codes. More information available on request.

[d] Includes categories such as overexertion, water transport accidents, exposure to electric transmission lines, etc.

areas (2.8; 95% CI: 2.7–2.8) followed by high-percentage Inuit-identity areas (2.3; 95% CI: 2.2–2.5) and high-percentage Métis-identity areas (2.0; 95% CI: 1.9–2.1).

Several unintentional injury causes present a significant rate ratio for all sex–age combinations. In high-percentage First Nations-identity areas, consistent disparities with low-percentage Aboriginal-identity areas are observed for 8 causes of injuries (cuts, falls, fire/hot substance, motor vehicle traffic, other land transport, environmental/natural causes, poisoning and being struck). Consistent disparities across all sex–age combinations are observed for 4 causes of injuries (falls, motor vehicle traffic, other land transport, and poisoning) in high-percentage Métis-identity areas and for 2 causes of injuries (falls and other land transport) in high-percentage Inuit-identity areas.

Rates of unintentional injury hospitalizations due to being burned by a fire or a hot substance, environmental/natural causes and poisoning in high-percentage First Nations-identity areas are more than 3 times those in low-percentage Aboriginal-identity areas, and this is observed for all sex–age combinations. For high-percentage Inuit-identity areas, other land transportation accidents present a rate ratio higher than 3.0 among all sex–age combinations, with the exception of men aged 30 to 44 years, where the rate ratio was closer to 2.0 (2.1; 95% CI: 1.5–3.1). For those people living in high-percentage Métis-identity areas, no cause presents a rate ratio consistently higher than this threshold among the 6 sex–age combinations.

Firearm injuries, which represent a low rate of injury (Table 3), have a high rate ratio for men living in high-percentage First Nations-identity areas (Table 4): the rate ratio increases from 4.0 (95% CI: 2.8–5.7) for 20- to 29-year-olds to 7.0 (95% CI: 3.9–12.4) for those aged 45 years plus. Likewise, drowning/suffocation injuries, although relatively rare among men aged 30 to 44 years living in high-percentage First Nations-identity areas, present a high rate ratio of 4.3 (95% CI: 2.8–6.7) in this age group. In contrast, falls, the most frequent cause of injury, do not present the highest rate ratios observed, but are still significantly greater than 1.0. With the exception of men aged 30 to 44 years living in high-percentage First Nations-identity areas, rate ratios for falls do not exceed the rate ratios for all causes combined.

Discussion

This study examined unintentional injury hospitalizations, by cause, among adults living in high-percentage First Nations-identity, Métis-identity and Inuit-identity areas and low-percentage Aboriginal-identity areas. Falls account for approximately one-third to two-thirds of all injury hospitalizations. In general, for all high-percentage Aboriginal-identity areas and for both sexes, the highest injury rates are observed among the oldest age group, the only exception being for men living in high-percentage Métis-identity areas among whom the highest rates were observed for the 20- to 29-year age group.

The rate ratios are consistently higher in areas with high proportions of First Nations-, Métis- and Inuit-identity residents: for all causes and all ages combined, rate ratios lie between 1.8 and 2.7 for men and 2.0 and 2.8 for women.

TABLE 4
Age-standardized rate ratios per 10 000 person-years for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	TOTAL ≥ 20 years		20–29 years		30–44 years		≥ 45 years	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Men								
Total								
High % First Nations	2.68	2.64–2.71	2.54	2.44–2.65	3.18	3.07–3.28	2.44	2.38–2.50
High % Métis	2.05	1.97–2.13	2.63	2.38–2.91	2.37	2.17–2.59	1.56	1.47–1.65
High % Inuit	1.84	1.74–1.95	2.08	1.81–2.39	1.59	1.38–1.83	1.87	1.67–2.10
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Cut								
High % First Nations	3.48	3.27–3.71	3.77	3.22–4.41	3.71	3.19–4.33	2.87	2.39–3.46
High % Métis	2.52	2.08–3.05	3.48	2.32–5.20	1.78	1.07–2.97	2.30	1.44–3.67
High % Inuit	2.88	2.34–3.55	3.66	2.27–5.92	2.58	1.45–4.59	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Drowning/Suffocation								
High % First Nations	2.71	2.28–3.22	x	x	4.31	2.76–6.72	2.46	1.99–3.05
High % Métis	1.83	1.13–2.98	x	x	x	x	1.66	1.05–2.61
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Fall								
High % First Nations	2.56	2.51–2.61	2.33	2.14–2.54	3.53	3.33–3.74	2.29	2.22–2.36
High % Métis	1.75	1.64–1.86	2.18	1.75–2.72	2.43	2.07–2.85	1.42	1.31–1.53
High % Inuit	1.64	1.50–1.79	1.78	1.32–2.41	1.47	1.13–1.93	1.66	1.41–1.97
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Fire/Hot substance								
High % First Nations	4.25	3.86–4.67	3.13	2.34–4.18	4.87	3.95–6.02	4.47	3.76–5.30
High % Métis	3.32	2.55–4.33	x	x	4.02	2.38–6.79	2.56	1.60–4.11
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Firearm								
High % First Nations	5.19	4.36–6.19	3.97	2.78–5.66	6.89	4.69–10.12	6.97	3.92–12.38
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Machinery								
High % First Nations	1.72	1.57–1.89	1.33	0.94–1.90	1.68	1.30–2.18	2.00	1.62–2.48
High % Métis	2.48	2.07–2.96	x	x	3.89	2.63–5.75	1.75	1.01–3.02
High % Inuit	1.23	0.85–1.79	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Motor vehicle								
High % First Nations	2.61	2.52–2.71	2.75	2.52–3.00	2.82	2.58–3.09	2.27	2.08–2.49
High % Métis	2.52	2.31–2.74	3.03	2.47–3.72	2.81	2.28–3.46	1.71	1.37–2.12
High % Inuit	0.81	0.64–1.03	1.01	0.66–1.55	x	x	0.80	0.46–1.40
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a

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TABLE 4 (continued)
Age-standardized rate ratios per 10 000 person-years for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	TOTAL ≥ 20 years		20–29 years		30–44 years		≥ 45 years	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Other land transport								
High % First Nations	2.56	2.45–2.68	2.50	2.25–2.79	2.76	2.50–3.05	2.40	2.14–2.68
High % Métis	2.25	1.97–2.55	2.68	2.06–3.48	2.33	1.82–3.00	1.65	1.23–2.20
High % Inuit	3.36	2.94–3.84	4.51	3.52–5.79	2.15	1.50–3.07	3.46	2.51–4.76
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Environmental/Natural								
High % First Nations	5.39	5.02–5.78	5.01	3.92–6.40	7.65	6.49–9.01	4.26	3.69–4.92
High % Métis	4.45	3.74–5.29	6.37	3.74–10.87	4.96	3.16–7.78	3.41	2.54–4.58
High % Inuit	5.57	4.39–7.07	x	x	7.28	4.51–11.73	5.71	3.55–9.17
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Poisoning								
High % First Nations	4.05	3.85–4.26	3.77	3.19–4.45	5.22	4.64–5.88	3.38	3.03–3.77
High % Métis	2.62	2.25–3.06	4.33	2.96–6.33	2.65	1.81–3.89	1.76	1.34–2.31
High % Inuit	2.45	2.00–3.00	x	x	2.91	1.82–4.64	2.09	1.26–3.47
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Struck								
High % First Nations	2.58	2.44–2.72	2.45	2.16–2.77	2.63	2.33–2.97	2.70	2.37–3.07
High % Métis	1.92	1.62–2.27	2.00	1.42–2.82	2.17	1.58–2.97	1.46	1.03–2.07
High % Inuit	1.64	1.27–2.10	1.95	1.25–3.05	1.21	0.71–2.06	1.76	0.93–3.31
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Others[d]								
High % First Nations	2.23	2.15–2.30	1.85	1.64–2.08	2.38	2.18–2.60	2.31	2.15–2.48
High % Métis	1.58	1.43–1.75	1.98	1.48–2.65	1.50	1.16–1.92	1.42	1.18–1.71
High % Inuit	1.56	1.34–1.80	1.36	0.88–2.09	1.03	0.70–1.53	2.10	1.56–2.81
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Women								
Total								
High % First Nations	2.78	2.74–2.83	3.88	3.68–4.10	3.87	3.70–4.04	2.25	2.20–2.30
High % Métis	1.98	1.90–2.07	2.96	2.56–3.43	2.54	2.25–2.87	1.62	1.55–1.70
High % Inuit	2.34	2.20–2.48	2.57	2.11–3.13	2.90	2.47–3.40	2.13	1.87–2.42
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Cut								
High % First Nations	3.68	3.19–4.25	4.07	2.98–5.56	4.02	3.02–5.37	2.81	1.89–4.16
High % Métis	4.03	3.04–5.34	6.54	3.59–11.89	x	x	x	x
High % Inuit	6.84	5.05–9.27	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Drowning/Suffocation								
High % First Nations	2.22	1.72–2.86	x	x	x	x	2.12	1.62–2.79
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a

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TABLE 4 (continued)
Age-standardized rate ratios per 10 000 person-years for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	TOTAL ≥ 20 years		20–29 years		30–44 years		≥ 45 years	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Fall								
High % First Nations	2.43	2.39–2.48	4.09	3.72–4.49	3.68	3.43–3.95	2.09	2.04–2.14
High % Métis	1.74	1.65–1.84	3.40	2.65–4.36	2.52	2.07–3.06	1.47	1.40–1.55
High % Inuit	2.19	2.03–2.37	2.62	1.83–3.76	2.93	2.26–3.78	2.04	1.73–2.40
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Fire/Hot substance								
High % First Nations	4.21	3.58–4.94	6.68	4.67–9.57	4.71	3.39–6.55	2.98	2.31–3.83
High % Métis	5.58	4.16–7.48	x	x	8.33	4.67–14.85	3.72	2.43–5.70
High % Inuit	6.13	4.04–9.31	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Firearm								
High % First Nations	x	x	x	x	x	x	x	x
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Machinery								
High % First Nations	1.82	1.29–2.56	x	x	x	x	x	x
High % Métis	x	x	x	x	x	x	x	x
High % Inuit	x	x	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Motor vehicle								
High % First Nations	3.65	3.50–3.81	4.01	3.60–4.45	4.39	3.96–4.87	2.89	2.62–3.19
High % Métis	2.36	2.07–2.69	2.72	1.99–3.72	2.81	2.10–3.77	1.80	1.33–2.43
High % Inuit	1.10	0.81–1.48	x	x	1.51	0.89–2.54	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Other land transport								
High % First Nations	2.85	2.63–3.10	3.63	3.03–4.36	3.12	2.64–3.70	2.12	1.76–2.55
High % Métis	2.29	1.78–2.96	2.37	1.37–4.09	1.96	1.20–3.21	2.53	1.67–3.82
High % Inuit	5.45	4.51–6.60	5.28	3.37–8.27	4.96	3.23–7.61	5.99	3.78–9.48
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Environmental/Natural								
High % First Nations	3.72	3.32–4.17	4.83	3.50–6.66	4.07	3.09–5.37	3.17	2.62–3.84
High % Métis	2.47	1.74–3.51	x	x	x	x	2.58	1.52–4.38
High % Inuit	2.86	1.81–4.53	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Poisoning								
High % First Nations	4.49	4.26–4.73	4.87	4.15–5.71	6.01	5.35–6.75	3.45	3.11–3.83
High % Métis	2.59	2.21–3.03	2.73	1.65–4.51	2.55	1.71–3.82	2.55	1.95–3.34
High % Inuit	2.21	1.74–2.81	3.99	2.35–6.79	2.58	1.42–4.68	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a

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TABLE 4 (continued)
Age-standardized rate ratios per 10 000 person-years for unintentional injuries by sex, age group, cause of injury, and by Aboriginal identity group[a], dissemination areas[b], population ≥ 20 years, Canada (excluding Quebec), 2004/2005–2009/2010

Cause of injury[c]	TOTAL ≥ 20 years		20–29 years		30–44 years		≥ 45 years	
	RR	95% CI	RR	95% CI	RR	95% CI	RR	95% CI
Struck								
High % First Nations	3.61	3.26–4.00	3.77	2.96–4.79	4.01	3.21–5.01	3.17	2.60–3.85
High % Métis	2.96	2.23–3.93	3.95	2.23–7.00	x	x	2.68	1.80–3.98
High % Inuit	2.45	1.69–3.55	x	x	x	x	x	x
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a
Others[d]								
High % First Nations	2.39	2.27–2.51	2.52	2.12–3.00	2.80	2.46–3.18	2.13	1.96–2.32
High % Métis	1.67	1.44–1.93	1.97	1.22–3.17	2.02	1.43–2.86	1.39	1.12–1.72
High % Inuit	2.32	1.92–2.81	2.15	1.22–3.79	2.26	1.46–3.50	2.41	1.67–3.49
Low % Aboriginal	1.00	n/a	1.00	n/a	1.00	n/a	1.00	n/a

Source: Discharge Abstract Database, 2004/2005–2009/2010.

Abbreviations: CI, confidence interval; n/a, Not applicable; RR, rate ratio.

Note: "x" indicates that the data was suppressed to meet the confidentiality requirements of the Statistics Act.

[a] The percentage of Aboriginal identity is provided by the 2006 Census.

[b] Dissemination areas where at least 33% of the population reported Aboriginal identity are classified as high-percentage Aboriginal identity. Classification as high-percentage First Nations, Métis, or Inuit is based on the predominant group.

[c] Categories of unintentional injury based on ICD-10-CA codes. More information available on request.

[d] Includes categories such as overexertion, water transport accidents, exposure to electric transmission lines, etc.

However, rate ratios present high variability as some causes of unintentional injuries produce a rate ratio as large as 7.0 (firearms for men aged 45 years plus living in high-percentage First Nations-identity areas) and others have a rate ratio less than 1.0, suggesting smaller disparities compared to low-percentage Aboriginal-identity areas.

Our findings show that ASHRs and rate ratios are two measures of injury hospitalization that are complementary but not overlapping. Indeed, causes of unintentional injuries that present both a "high" ASHR and a "high" rate ratio are relatively rare. Only 13 instances have both an injury rate higher than 10/10 000 and rate ratio higher than 3.0,* which means that the injury being considered is both much more frequent than other injuries and much more frequent in the high-percentage Aboriginal-identity area than in low-percentage Aboriginal-identity areas: (1) for falls, among men aged 30 to 44 years living in high-percentage First Nations-identity areas, among women aged 20 to 29 years or 30 to 44 years living in high-percentage First Nations-identity areas and among women aged 20 to 29 years living in high-percentage Métis-identity areas; (2) for motor vehicle traffic accidents, among men aged 20 to 29 years living in high-percentage Métis-identity areas and among women aged 20 to 29 years or 30 to 44 years living in high-percentage First Nations-identity areas; (3) for other land transport accidents, among men aged 20 to 29 years or 45 years or more living in high-percentage Inuit-identity areas and among women aged 45 years or more living in high-percentage Inuit-identity areas; and (4) for poisoning, among men aged 30 to 44 years living in high-percentage First Nations-identity areas and among women aged 30 to 44 or 45 years or more living in high-percentage First Nations-identity areas.

In summary, areas with high percentage of Aboriginal-identity residents can be characterized as follows:

- High-percentage First Nations-identity areas present the highest total ASHRs among the 4 types of areas for each sex–age combination, a high ASHR of 29 per 10 000 for motor vehicle traffic among men aged 20 to 29 years, a relatively high ASHR for poisoning for all sex–age combinations, a relatively high ASHR for being struck for all age groups among men, and relatively high rate ratios for drowning/suffocation, fire/hot substance and firearm injuries (for all sex–age combinations for which results were available), even though the ASHR for these causes is low;
- High-percentage Métis-identity areas present a total ASHR among men aged 20 to 29 years that is higher than in other age groups, the lowest total ASHRs among women living in high-percentage Aboriginal-identity areas and a relatively high ASHR for machinery among men aged 30 to 44;
- High-percentage Inuit-identity areas present the lowest total ASHRs among men of all age groups living in high-percentage Aboriginal-identity areas, the highest ASHR for other land transportation for most sex–age combinations and a high rate ratio for environmental/natural causes among

* Thresholds of 10/10 000 person-years for ASHR and 3.0 for rate ratios were chosen arbitrarily in this section.

men, for all age groups for which results were available.

Although we used a different methodology, our results are in line with those of Fantus et al.[14] concerning falls and motor vehicle traffic accidents: these authors found an age- and sex-adjusted rate of 57 and 14 per 10 000 person-years respectively for these 2 causes, whereas we found ASHRs of 55 per 10 000 for falls for both sexes and of 20 and 15 per 10 000 respectively for men and women for motor vehicle accidents. Also, even though we examined hospitalizations rather than deaths, used geozones instead of a record-linkage approach and did not use the same age groups, our results for rate ratios on falls for high-percentage First Nations and high-percentage Métis identity areas are similar to those found by Tjepkema et al.[5]

Limitations

This analysis only included injuries resulting in hospitalizations, and not those that caused death.[13] Also, individuals presenting to emergency departments, physicians' offices or clinics were not captured by these data.

As with any study based on an ecological approach, bias can occur because the results are based on geographical areas and not on individuals.[22,26] Our results relate to people living in areas with high proportions of Aboriginal-identity residents—according to a previously defined threshold—and include those who do not necessarily self-identify as Aboriginal; therefore, the results are not representative of First Nations, Métis or Inuit individuals in Canada. As well, any difference observed between high-percentage Aboriginal-identity areas and low-percentage Aboriginal-identity areas may be explained by other factors such as socio-economic characteristics (not related to Aboriginal identity), some of which are described in Table 1. In particular, residing in rural or urban areas could be a confounding factor. This variable, represented in Table 1 by Metropolitan Influence Zones (MIZs), was not used in this study. A limitation related to not using MIZs is the fact that, because Aboriginal identity is defined from the 2006 Census whereas the Discharge Abstract Database is used for 6 fiscal years (2004/2005 to 2009/2010), there may be a discrepancy in how the regions are defined in these two databases.

Other limitations related to geographical data should be mentioned. First, the province of Quebec as well as one hospital from the territories did not provide administrative data and thus were not included. Second, the geographical location where the injury occurred was not available and the residential postal code was used as a proxy. Third, it should be noted that, for some rural areas, postal codes are not an accurate representation of residential location because of the use of P.O. Box numbers, which may be located in a different area than the place of residence; also, rural postal codes may map on to more than one DA, thus reducing the ability to determine the specific place of residence.[19]

Conclusion

We presented hospitalization data for unintentional injuries in Canada, allowing for comparisons between areas with a high percentage of Aboriginal (First Nations, Métis or Inuit)-identity residents and areas with a low percentage of Aboriginal-identity residents. Health disparities in the Aboriginal population need to be considered within their broader social context: Aboriginal people in Canada
generally live in areas characterized by lower socio-economic conditions than the general Canadian population, including lower income, higher rates of unemployment, crowded living conditions and houses in need of repairs.[7] The results presented in Table 1, showing that high-percentage Aboriginal-identity areas are made up of a majority of Aboriginal-identity individuals and are characterized by lower socio-economic conditions than low-percentage Aboriginal-identity areas, lend support to this. In addition, our data also show a higher rate of hospitalizations due to unintentional injuries in areas with a high proportion of Aboriginal-identity residents, which may indicate people living in lower socio-economic conditions who are at risk of problems related to health.

Acknowledgements

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References

1. Tjepkema M, Wilkins R. Remaining life expectancy at age 25 and probability of survival to age 75, by socio-economic status and Aboriginal ancestry. Health Rep. 2011;22(4):31-6.

2. Tjepkema M, Wilkins R, Pennock J, Goedhuis N. Potential years of life lost at ages 25 to 74 among Status Indians, 1991 to 2001. Health Rep. 2011;22(1):25-36.

3. Tjepkema M, Wilkins R, Senécal S, Guimond E, Penney C. Potential years of life lost at ages 25 to 74 among Métis and non-Status Indians, 1991 to 2001. Health Rep. 2011;22(1):37-46.

4. Garner R, Carrière G, Sanmartin C; Longitudinal Health and Administrative Data Research Team. The health of First Nations living off-reserve, Inuit, and Métis adults in Canada: the impact of socio-economic status on inequalities in health. Health Research Working Paper Series. Ottawa (ON): Statistics Canada; 2010 Jun. [Statistics Canada, Catalogue No.: 82-622-X-No. 004].

5. Tjepkema M, Wilkins R, Senécal S, Guimond E, Penney C. Mortality of Métis and Registered Indian adults in Canada: an 11-year follow-up study. Health Rep. 2009;20(4): 31-51.

6. Wilkins R, Uppal S, Finès P, Senécal S, Guimond E, Dion R. Life expectancy in the Inuit-inhabited areas of Canada, 1989 to 2003. Health Rep. 2008;19(1):7-19.

7. Adelson N. The embodiment of inequity – health disparities in Aboriginal Canada. Can J Public Health. 2005;96 Suppl 2:S45-61.

8. Allard YE, Wilkins R, Berthelot JM. Premature mortality in health regions with high Aboriginal populations. Health Rep. 2004;15(1):51-60.

9. Unintentional and intentional injury profile for Aboriginal people in Canada – 1990-1999. Ottawa (ON): Health Canada; 2001. [Catalogue No.: H35-4/8-1999].

10. Tjepkema M. Non-fatal injuries among Aboriginal Canadians. Health Rep. 2005:16(2);9-22.

11. A statistical profile on the health of First Nations in Canada. Health services utilization in western Canada, 2000. Ottawa (ON): Health Canada; 2009. [Catalogue No.: H34-193/4-2008].

12. Karmali S, Laupland K, Harrop AR, et al. Epidemiology of severe trauma among status Aboriginal Canadians: a population-based study. CMAJ. 2005;172 (8):1007-11.

13. Haas B, Poon V, Waller B, Sidhom P, Fortin CM. National Trauma Registry 2011 report: hospitalizations for major injury in Canada, 2008-2009 data. Toronto (ON): Canadian Institute for Health Information; 2011.

14. Fantus D, Shah BR, Qiu F, Hux P, Rochon P. Injury in First Nations communities in Ontario. Can J Public Health. 2009;100(4): 258-62.

15. Carrière G, Garner R, Sanmartin C; LHAD Research Team. Acute-care hospitalizations and Aboriginal identity in Canada, 2001/2002. Health Research Working Paper. Ottawa (ON): Statistics Canada; 2010. [Catalogue No.: 82-622-X-No. 005].

16. Oliver LN, Kohen DE. Unintentional injury hospitalization among children living in areas with a high percentage of Aboriginal identity residents: 2001/2002 to 2005/2006. Health Rep. 2012;23(3):7-15.

17. Alaghehbandan R, Sikdar KC, MacDonald D, Collins KD, Rossignol AM. Unintentional injuries among children and adolescents in Aboriginal and non-Aboriginal communities, Newfoundland and Labrador, Canada. Int J Circumpolar Health. 2010; 69:1:61-71.

18. Peters PA. Shifting transitions: health inequalities of Inuit Nunangat in perspective. J Rural Community Dev. 2012;7(1): 36-58.

19. Executive summary: data quality documentation, Discharge Abstract Database, 2006–2007. Ottawa (ON): CIHI; 2007 Oct.

20. ICD-10-CA: International statistical classification of diseases and related health problems, 10th rev. Vol. 1, tabular list. Ottawa (ON): CIHI; 2012.

21. Injury surveillance on-line: ICD10 - ICD9 transition matrix for external cause of injury group: based on recommended frameworks for presenting injury mortality data [Internet]. Ottawa (ON): Public Health Agency of Canada; 2005 Feb [cited 2012 Jul 20]. Available from: http://dsol-smed.phac-aspc.gc.ca/dsol-smed/is-ib/chirpp/ICD10-ICD9Transition-MatrixISOL.pdf

22. Peters PA, Oliver LN, Carrière GM. Geozones: an area-based method for analysis of health outcomes. Health Rep. 2012 Mar;23(1):55-64.

23. Wilkins R, Peters PA. PCCF+ Version 5K User's Guide: Automated geocoding based on the Statistics Canada Postal Code Conversion Files. Ottawa (ON): Statistics Canada; 2012. [Catalogue No.: 82F0086-XDB].

24. Kegler SR. Applying the compound Poisson process model to the reporting of injury-related mortality rates. Epidemiol Perspect Innov. 2007;4:1.

25. Wilkins K. Health care consequences of falls for seniors. Health Rep. 1999;10(4).

26. Diez Roux AV. Investigating neighborhood and area effects on health. Am J Public Health. 2001 Nov;91(11):1783-9.

Chronic bronchitis in Aboriginal people—prevalence and associated factors

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Abstract

Introduction: Knowledge about chronic bronchitis (CB) among Aboriginal people in Canada is limited. The aim of this study was to determine the prevalence of CB and its associated factors among Aboriginal people aged 15 years plus.

Methods: Logistic regression analysis was used on data from the cross-sectional 2006 Aboriginal Peoples Survey to determine risk factors associated with CB.

Results: CB prevalence was 6.6% among First Nations, 6.2% among Métis and 2.4% among Inuit. Prevalence was higher among females than males (7.2% versus 5.0%). Individuals with CB were more likely to be older, living at a lower income, with a lower educational attainment and residing in rural areas. Smoking status and body mass index were also significantly associated with CB, but their effect differed by sex. Obesity was particularly significantly associated with CB among females compared with males, and current smoking and non-smoking status was significantly associated with CB among females but not males.

Conclusion: These findings identify factors associated with CB among Aboriginal people. As such, they may represent potentially preventable risk factors that can inform health promotion and disease prevention practices.

Keywords: *chronic bronchitis, Aboriginal people, Aboriginal Peoples Survey*

Introduction

The health of Aboriginal people—First Nations, Metis and Inuit—is notably poorer than that of the general Canadian population,[1] a trend also observed in their respiratory health.[2] Approximately 15% of Aboriginal people have been diagnosed with at least one of four respiratory diseases (asthma, chronic bronchitis [CB], emphysema and chronic obstructive pulmonary disorder [COPD]) compared to 10% for non-Aboriginal people in Canada, according to the 2005 Canadian Community Health Survey (CCHS).[3] Age-standardized hospital separation rates in western Canada for Aboriginal people for all respiratory diseases in 2000 were 3040 per 100 000 population compared with 920 per 100 000 population in their non-Aboriginal counterparts.[4]

CB is one such respiratory disease defined as ''cough productive of sputum for at least three months of the year for at least two years.''[5] CB is a significant cause of morbidity and an underlying condition for the development of COPD.[6]

Our knowledge of CB and its associated factors in Canadian Aboriginal people is limited. The 2002/03 First Nations Regional Longitudinal Health Survey found age-standardized prevalence of self-reported physician-diagnosed CB to be 3.7% in First Nations living on-reserve;[7] the prevalence in Aboriginal people living off-reserve is 4.9%, according to the 2005 CCHS.[3] Both of these rates are higher than the prevalence of 2.4% found in the non-Aboriginal Canadian population, according to the 2005 CCHS.[3]

The prevalence of CB in Aboriginal people may be high due to the high prevalence of various risk factors. Smoking, low family income, poor schooling and inadequate housing, which have been significantly associated with the prevalence and incidence of CB,[8-10] are more prevalent among Aboriginal people. According to the 2002/03 First Nations Regional Longitudinal Health Survey, roughly 59% of First Nations self-reported currently smoking, with smoking rates for on-reserve First Nations slightly higher than for those living off-reserve.[7] Smoking rates among Inuit have been reported to be as high as 70%.[11]

In 2005, Aboriginal people aged 25 to 54 years had a much lower median total individual income ($22 000) compared with their non-Aboriginal counterparts ($33 000).[12] Of those aged 25 to 64 years, 44% of Aboriginal people compared with 60% of the general population had completed some post-secondary schooling.[13] Lower education is often associated with lower socio-economic status, which may correlate with lower income and worse housing conditions. In 2006, Aboriginal people were almost four times as likely to live in crowded homes, and three times as likely to live in a dwelling in need of major repairs than non-Aboriginal people.[14] Poor

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housing conditions are often associated with damp and mould, which may lead to adverse respiratory outcomes.[2]

We carried out a descriptive study to assess the relationship between demographic, environmental and population characteristics and CB. To date, the determinants of CB among Aboriginal people in Canada have not been well established. Thus, the objective of this study was to confirm the prevalence (crude and adjusted) of CB and determine its associated factors in off-reserve Canadian Aboriginal people aged 15 years and older.

Methods

Study population and data source

The Aboriginal Peoples Survey (APS) 2006 is a national cross-sectional survey conducted from October 2006 through March 2007 by Statistics Canada in partnership with Aboriginal organizations.[15] This is the third time that Statistics Canada has administered the APS, the first being in 1991 and the second in 2001. The target population of this survey was off-reserve First Nations, Métis and Inuit people living in urban, rural and northern locations throughout Canada. A multi-stage sampling design was used to select and collect data from all the provinces. Details of this sampling design can be found elsewhere.[15] Briefly, a target sample was created based on responses to four screening questions in the 2006 Census long form that indicated that the respondents had Aboriginal ancestors and/or identified as North American Indian and/or Métis and/or Inuit and/or had treaty or registered Indian status and/or had Indian Band membership. The sample was then divided according to domains of estimation, based on Aboriginal identity, age groups and geographical regions. A random sample was then selected within each domain of estimation. The APS included information on Aboriginal identity and ancestry, education, language, labour activity, income, health, communication technology, mobility, housing and family background. There were a total of 48 921 participants, with a response rate of 80.1%. Data were collected via self-

TABLE 1
Characteristics of Aboriginal people[a] (≥ 15 years) stratified by self-reported chronic bronchitis, 2006, Canada (N = 48 921)

	Chronic Bronchitis, %		OR (95% CI)
	Yes	No	
Demographic characteristics			
Ethnicity			
North American Indian	6.57	93.43	1.00
Métis	6.19	93.81	0.93 (0.79–1.11)
Inuit	2.38	97.62	0.35 (0.25–0.47)
Sex			
Male	5.00	95.00	1.00
Female	7.20	92.80	1.47 (1.23–1.76)
Age, years			
15–19	2.67	97.33	1.00
20–24	3.12	96.88	1.17 (0.73–1.86)
25–34	3.70	96.30	1.40 (0.95–2.06)
35–44	6.12	93.88	2.38 (1.67–3.38)
45–54	9.09	90.91	3.64 (2.57–5.17)
≥ 55	10.06	89.94	4.07 (2.83–5.86)
Marital status			
Legally married	6.85	93.15	1.00
Never married	4.28	95.72	0.61 (0.50–0.74)
Divorced or widowed	10.59	89.41	1.61 (1.30–2.00)
Environmental characteristics			
Number of persons per household			
≥ 5	4.08	95.92	1.00
3–4	5.32	94.68	1.34 (1.01–1.71)
≤ 2	8.22	91.78	2.11 (1.63– 2.72)
Location of residence[b]			
Urban	6.61	93.39	1.00
Rural	5.19	94.81	0.77 (0.66–0.91)
Geographical area			
Territories[c]	1.85	98.15	1.00
British Columbia	4.95	95.05	2.78 (1.94–3.98)
Prairies[d]	4.96	95.04	2.78 (2.05–3.78)
Ontario	9.05	90.95	5.31 (3.82–7.37)
Quebec	6.89	93.11	3.95 (2.75–5.66)
Atlantic[e]	7.44	92.56	4.29 (3.08–5.97)
Socio-economic status			
Educational attainment			
University completed	3.93	96.07	1.00
Some university	6.82	93.18	1.78 (1.31–2.43)
High school completed	5.29	94.71	1.36 (0.94–1.98)
Less than high school	6.95	93.05	1.82 (1.33–2.49)
Yearly income, $			
≥ 100 000	2.69	97.31	1.00
80 000–99 999	3.88	96.12	1.46 (1.01–2.09)
60 000–79 999	5.71	94.29	2.19 (1.58–3.02)

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TABLE 1 (continued)
Characteristics of Aboriginal people[a] (≥ 15 years) stratified by self-reported chronic bronchitis, 2006, Canada (N = 48 921)

	Chronic Bronchitis, %		OR (95% CI)
	Yes	No	
40 000–59 999	6.46	93.54	2.49 (1.83–3.40)
20 000–39 999	7.08	92.92	2.75 (2.05–3.69)
< 20 000	11.45	88.55	4.66 (3.44–6.33)
Lifestyle characteristics			
Smoking status			
Never smoked	3.25	96.75	1.00
Ex-smoker	6.27	93.73	1.99 (1.54–2.56)
Current smoker	8.32	91.68	2.70 (2.14–3.40)
Health-related characteristics			
General health status			
Excellent	2.21	97.79	1.00
Very good	3.43	96.57	1.57 (1.13–2.16)
Good	6.20	93.80	2.92 (2.16–3.94)
Fair	14.36	85.64	7.41 (5.39–10.17)
Poor	21.94	78.06	12.41 (8.88–17.35)
Diabetes			
No	8.10	91.90	1.00
Yes	13.16	86.84	1.72 (1.01–2.96)
BMI (kg/m^2)			
< 24.9	6.00	94.00	1.00
25.0–29.9	5.51	94.49	0.91 (0.73–1.13)
> 29.9	7.34	92.66	1.26 (1.02–1.55)

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio.

[a] Based on participants in the APS self-identifying as North American Indian and/or Métis and/or Inuit and/or having treaty or registered Indian status and/or Indian Band membership and/or Aboriginal ancestors.

[b] Based on Statistics Canada determinations.[15]

[c] Yukon, Northwest Territories, Nunavut.

[d] Alberta, Saskatchewan, Manitoba.

[e] New Brunswick, Prince Edward Island, Nova Scotia, Newfoundland.

administered questionnaires or personal interviews over the phone or in person.

The target populations of this survey were Aboriginal children and youth (6–14 years) and Aboriginal adults (≥ 15 years). Since our study focused on the adult population, we excluded APS participants aged less than 15 years.

The University of Saskatchewan Research Ethics Board approved this research. We obtained permission to access the data from Statistics Canada and conducted all analyses within the Statistics Canada Research Data Centre at the University of Saskatchewan.

Measures

The APS included a set of questions designed to investigate survey participants' chronic conditions. The variables used for the analysis are defined below.

Outcome

In this report, the outcome variable of interest for adults was based on the following question: ''Have you been told by a doctor, nurse or other health professional that you have: chronic bronchitis?''[15]

Factors

Of interest were demographic, environmental, and health and lifestyle variables (see Table 1). Demographic variables consisted of age, sex, ethnicity and marital status; environmental variables consisted of location of residence, number of persons per household and geographical area. Location of residence, rural or urban, was based on Statistics Canada determinations (minimum population concentrations and population density per square kilometer). Geographical areas were broken down into Territories (Yukon, Northwest Territories, Nunavut), British Columbia, Prairies (Alberta, Saskatchewan, Manitoba), Ontario, Quebec, and Atlantic (New Brunswick, Prince Edward Island, Nova Scotia, Newfoundland and Labrador). Health-related variables consisted of self-perceived general health status, smoking status and body mass index (BMI). BMI was introduced as a continuous variable in the multivariate model, and was afterwards categorized for a schematic depiction (Figure 2). Socio-economic status variables consisted of education and income.

Statistical analysis

We calculated the percentage of participants reporting CB and associated factors. Weight variables computed by Statistics Canada methodologists used in all analyses ensured that the final estimates were representative of the surveyed population. We used weighted multiple logistic regression modelling based on a maximum likelihood to test the association of CB risk factors. Balanced repeated replication resampling technique was used to estimate the standard errors of regression coefficients in order to account for clustering inherited in the study design of the cross-sectional complex survey. Statistically significant two-way interactions were examined. The results of the models are presented as odds ratios (OR) along with the 95% confidence intervals (CIs). Statistical packages SAS version 9.2 (SAS Institute Inc., Cary, NC, US) and

STATA version 11.0 were used to conduct all analyses.

Results

Of the adult APS respondents, 50.0% were First Nations, 45.2% were Metis and the remaining 4.8% were Inuit. Due to the small number of Inuit in the dataset, they were excluded from all multivariate analyses.

Crude prevalence of chronic bronchitis

Table 1 summarizes both the prevalence and odds ratio for CB. The crude prevalence of CB was 6.6%, 6.2% and 2.4% among First Nations, Metis and Inuit, respectively (Table 1). Overall prevalence was 6.0% for off-reserve Aboriginal people. Prevalence was 8.3% among smokers and 3.3% among non-smokers. CB was more prevalent among females than males (5.0% vs. 7.2%) and increased with age, from 2.7% for those aged 15 to 19 years to 10.1% for those aged 55 years and older. The prevalence was highest in Ontario, at 9.1%, and the Atlantic region, at 7.4%. Prevalence was also higher in those living at a lower income and with a lower educational attainment.

Those with diabetes had a prevalence of 13.2%, while those without had a prevalence of 8.1%.

Adjusted prevalence of chronic bronchitis

Table 2 summarizes all the variables that were found to be significant predictors of CB in the multivariate model.

In the multivariate model, the prevalence of CB among Métis did not significantly differ from that among First Nations (OR = 1.05; 95% CI = 1.00–1.10). As expected, older respondents were more likely to report CB compared to those in the youngest age group (≥ 55 years: OR = 3.06; 95% CI = 2.73–3.43). Those who had never married or else were divorced or widowed were less likely to report CB (never married: OR = 0.72; 95% CI = 0.68–0.78; divorced/widowed: OR = 0.90; 95% CI = 0.84–0.96). Income and educational attainment were inversely associated with CB; participants who had not completed high school had 1.4 (95% CI = 1.30–1.57) times greater odds of having CB than those with a university degree, and those with an income of less $20 000 had 3.4 (95% CI = 3.1–3.6) times greater odds of having CB than those with an income of $80 000 or more. Urban residence was also positively associated with CB (OR = 1.31; 95% CI = 1.25–1.38). BMI was found to be a significant predictor as a quadratic term, representing a U-shaped relationship (BMI = 25.0–29.9 kg/m^2: OR = 0.91, CI = 0.73–1.13; BMI > 29.9 kg/m^2: OR = 1.26, CI = 1.02–1.55).

TABLE 2
Results of logistic regression of the prevalence of chronic bronchitis in Aboriginal peoples[a] (≥ 15 years), 2006, Canada (N = 48 921)

	Regression estimates ($\hat{\beta}$) $\hat{\beta}$ (s.e.($\hat{\beta}$))	OR_{adj} (95% CI)
Demographic characteristics		
Ethnicity		
First Nation (ref)	—	1.00
Métis	0.05 (0.02)	1.05 (1.00–1.10)
Sex		
Male (ref)	—	1.00
Female	0.53 (0.13)	1.71 (1.32–2.21)
Age, years		
15–19 (ref)	—	1.00
20–24	0.08 (0.06)	1.08 (0.95–1.23)
25–34	0.08 (0.06)	1.08 (0.96–1.21)
35–44	0.65 (0.06)	1.92 (1.72–2.14)
45–54	1.08 (0.06)	2.94 (2.63–3.29)
≥ 55	1.12 (0.06)	3.06 (2.73–3.43)
Marital Status		
Legally married (ref)	—	1.00
Never married	−0.32 (0.03)	0.72 (0.68–0.78)
Divorced/widowed	−0.11 (0.04)	0.90 (0.84–0.96)
Location of residence[b]		
Rural (ref)	—	1.00
Urban	0.25 (0.02)	1.31 (1.25–1.38)
Educational attainment		
University	—	1.00
Some university	0.29 (0.04)	1.33 (1.22–1.45)
High school completed	0.09 (0.05)	1.09 (0.99–1.21)
Less than high school	0.36 (0.05)	1.43 (1.30–1.57)
Income, $		
≥ 80 000 (ref)	—	1.00
60 000–79 999	0.66 (0.04)	1.94 (1.79–2.10)
40 000–59 999	0.66 (0.04)	1.93 (1.78–2.08)
20 000–39 999	0.76 (0.04)	2.14 (1.98–2.31)
< 20 000	1.21 (0.04)	3.36 (3.11–3.63)
BMI (kg/m^2)	−0.07 (0.01)	0.93 (0.91–0.95)
BMI^2	0.00 (0.00)	1.00 (1.00–1.00)
Smoking status		
Never smoked (ref)	—	1.00
Ex-smoker	0.78 (0.07)	2.19 (1.91–2.50)
Current smoker	1.18 (0.06)	3.24 (2.86–3.67)

Continued on the following page

TABLE 2 (continued)
Results of logistic regression of the prevalence of chronic bronchitis in Aboriginal peoples[a] (≥ 15 years), 2006, Canada (N = 48 921)

	Regression estimates ($\hat{\beta}$) $\hat{\beta}$ (s.e.($\hat{\beta}$))	OR_{adj} (95% CI)
Interactions		
(Sex plus smoking status)		
Female plus ex-smoker	−1.01 (0.08)	0.36 (0.31–0.43)
Female plus current smoker	−0.60 (0.07)	0.55 (0.48–0.63)
(Sex plus BMI)		
Female plus BMI	0.01 (0.00)	1.01 (1.00–1.02)

Abbreviations: BMI, body mass index; CI, confidence interval; OR_{adj}, adjusted odds ratio; s.e., standard error.

[a] Based on participants in the APS self-identifying as North American Indian and/or Métis and/or Inuit and/or having treaty or registered Indian status and/or Indian Band membership and/or Aboriginal ancestors.

[b] Based on Statistics Canada's determinations.[15]

There were also two significant interactions between sex and smoking status and sex and BMI. Among non-smokers and current smokers, females have a higher probability of CB than do men, whereas among ex-smokers, the probability of CB was slightly lower for females than males (Figure 1). In all the three categories of BMI (healthy and underweight, overweight, and obese), the probability of CB was significantly higher in females than males. However, this difference was notably greater in obese people.

Discussion

By using a cross-sectional cohort, this study determined the prevalence of CB and examined the associated factors in Aboriginal adults. We found the prevalence of CB to be 6.0% overall, 6.6% for First Nations, 6.2% for Metis, and 2.4% for Inuit. The multivariate analysis showed older age, smoking, obesity, lower educational attainment, lower income, and urban residence to be significantly associated with self-reported physician-diagnosed CB. Two-way interactions between sex and smoking and between sex and BMI were also observed.

Our analysis found the prevalence of CB to be slightly higher than the 4.9% found by the 2005 CCHS among off-reserve Aboriginal people and the 2.4% found among non-Aboriginal people. The CCHS measures self-reported health-provider-diagnosed CB in a way similar to the APS.

FIGURE 1
Error-bar graph showing probability of chronic bronchitis in Aboriginal people (≥ 15 years) by sex and smoking status, 2006, Canada

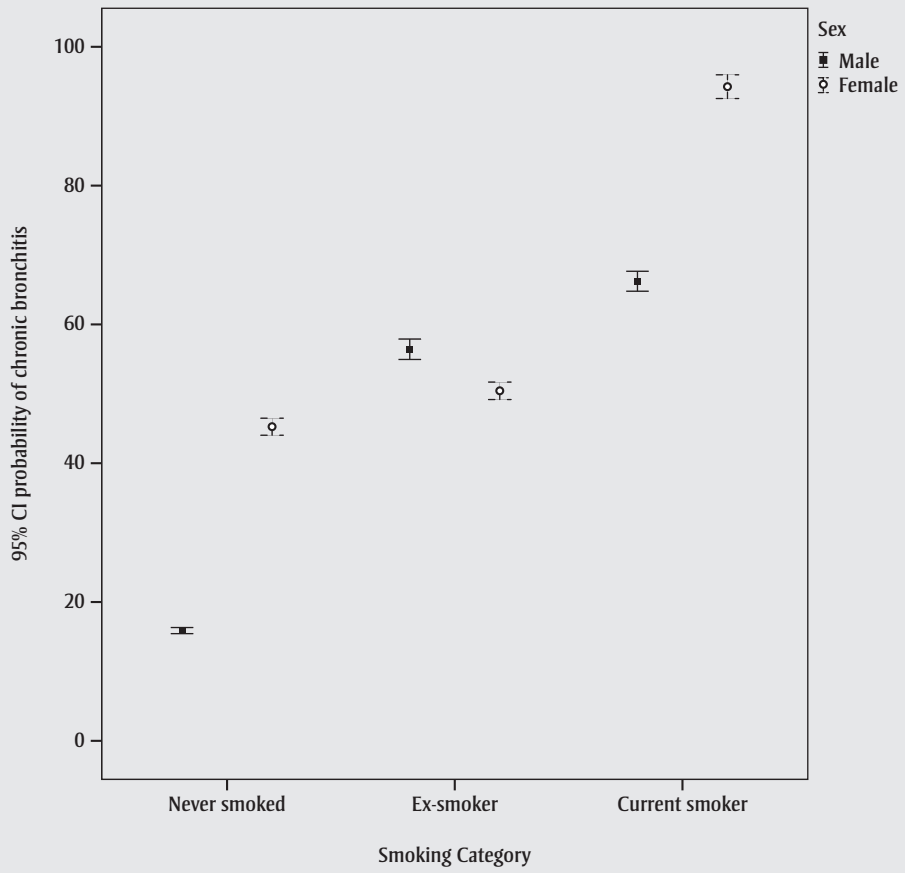
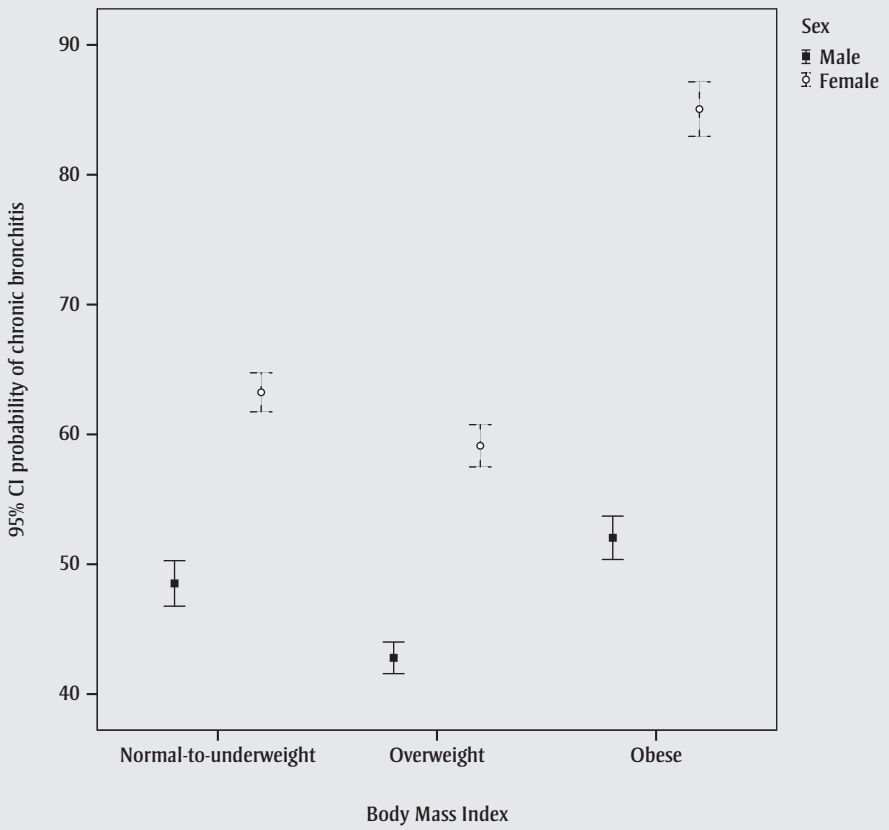


FIGURE 2
Error-bar graph showing probability of chronic bronchitis in Aboriginal people (≥ 15 years) by sex and body mass index, 2006, Canada



The prevalence of CB was particularly low among Inuit compared with First Nations and Métis. Since the rates of smoking were highest in this group,[16] the low prevalence of CB may be attributed to geographical barriers in access to care and thus decreased opportunities for a diagnosis. This rationale could also be used to at least partly explain the difference observed between locations of residence, in which urban residents were more likely to self-report physician-diagnosed CB compared with rural residents.

Supporting our findings of differences by sex in the prevalence of CB, a study from a small Saskatchewan town that focused on a grain-farming population found the prevalence of CB to be 9.6% among women and 4.2% among men.[17]

Numerous other studies also found smoking, income and poor schooling to be independently associated with CB.[5,18,19] Smoking is an established and major risk factor for CB.[19] Income and education, indicators of socio-economic status, suggest that other variables may be mediating this association.[18] Low income, for example, limits individual options in healthy living environments and foods, which may, in turn, contribute to obesity.[20]

The link between obesity and chronic respiratory diseases has also become increasingly recognized. In a longitudinal cohort, Guerra et al.[21] found that patients with CB were more likely to be obese. In our study, we observed a possible U-shaped risk trend (shown in Figure 1), meaning that both low and high BMI correlated with the disease. Guerra et al.[21] also observed a similar, albeit non-significant, trend. In addition, they observed a temporal relationship; a BMI of 28 kg/m^2 or more increased the risk of receiving a physician-confirmed diagnosis of CB (OR = 1.80; 95% CI = 1.32–2.46) two years later.[21] While their study suggests a causal relationship, more research is needed to elucidate this relationship. Nevertheless, obesity increases the risk of respiratory dysfunction, as indicated by a review of obesity.[22]

Limitations

There were several limitations to our study. In surveys such as the APS, the measurement of CB lacks clinical accuracy, which could introduce misclassification.[23] The APS asks a single question about CB, whether respondents have been told by a health care professional that they have CB. Diagnosis of chronic diseases may also be influenced by availability and use of health care services, possibly causing systemic bias. In addition, all answers in this survey are self-reported: self-reporting may under-

estimate the prevalence of some risk factors, such as weight, smoking status and income. Finally, this survey only collected data on off-reserve First Nations. Based on the 2006 Census, about 40% of First Nations people live on reserve.[14] Various statistics do show significant differences between on-reserve and off-reserve First Nations, and thus these results may not necessarily be generalizable to all First Nations. In addition, Inuit were removed from the multivariate analysis, further limiting the generalizability of these findings to this population.

Conclusion

To our knowledge this is the first report that has specifically examined factors associated with CB among the Aboriginal population. Our research provides a snapshot of CB and its determinants; nevertheless, further analyses are needed to explore these associations, particularly how low socio-economic status and obesity may be affecting CB. Our study highlights the importance of smoking cessation and reduction in BMI in this population, particularly among females.

In conclusion, this study showed that potentially preventable risk factors (low socio-economic status, obesity and smoking) were significantly associated with CB after adjusting for possible confounders. Such information may be useful for designing and promoting preventive campaigns specifically for the Aboriginal population.

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Conflict of interest: none.

References

1. MacMillan HL, MacMillan AB, Offord DR, Dingle JL. Aboriginal Health. CMAJ. 1996;155:1569-78.

2. Sin DD, Wells H, Svenson LW, Man SF. Asthma and COPD among Aboriginals in Alberta, Canada. Chest. 2002;121:1841-6.

3. Canadian Community Health Survey (CCHS): detailed information for 2005 (cycle 3.1) [Internet]. Ottawa (ON): Statistics Canada; [modified 2007 Oct 10; cited 2010 Jun 1]. Available from: http://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&SurvId=3226&SurvVer=0&InstaId=15282&InstaVer=3&SDDS=3226&lang=en&db=imdb&adm=8&dis=2

4. Health Canada. A statistical profile on the health of First Nations in Canada: health service utilization in Western Canada, 2000 [Internet]. Ottawa (ON): Health Canada; 2009 [cited 2010 Jun 1]. Available from: http://www.hc-sc.gc.ca/fniah-spnia/pubs/aborig-autoch/2009-stats-profil-vol2/index-eng.php

5. American Thoracic Society. Definitions and classifications of chronic bronchitis, asthma and pulmonary emphysema. Am Rev Respir Dis. 1962;85:762-8.

6. Pelkonen M. Smoking: relationship to chronic bronchitis, chronic obstructive pulmonary disease and mortality. Curr Opin Pulm Med. 2008;14:105-9.

7. First Nations Information Governance Committee. First Nations Regional Longitudinal Health Survey (RHS) 2002/03: results for adults, youth and children living in First Nations communities. Ottawa (ON): First Nations Centre; Nov 2005.

8. Minore B, Hill ME, Park J, et al. Understanding respiratory conditions among Ontario's Aboriginal population. Thunder Bay (ON): Centre for Rural and Northern Health Research; 2010.

9. Melia RJ, Chinn S, Rona RJ. Respiratory illness and home environment of ethnic groups. Br Med J. 1988;296:1438-41.

10. Cooreman J, Redon S, Levallois M, Liard R, Perdrizet S. Respiratory history during infancy and childhood, and respiratory conditions in adulthood. Int J Epidemiol. 1990;19:621-7.

11. Wong S. Use and misuse of tobacco among Aboriginal peoples. Paediatr Child Health. 2006;11(10):681-5.

12. Aboriginal statistics at a glance. Income [Internet]. Ottawa (ON): Statistics Canada; 2010 [cited 2010 Jul 22]. Available from: http://www.statcan.gc.ca/pub/89-645-x/2010001/income-revenu-eng.htm

13. Educational portrait of Canada, 2006 Census [Internet]. Ottawa (ON): Statistics Canada; 2008 [cited 2010 Jul 22]. Available from: http://www12.statcan.gc.ca/english/census06/analysis/education/pdf/97-560-XIE2006001.pdf

14. Aboriginal Peoples in Canada in 2006: Inuit, Metis and First Nations, 2006 Census [Internet]. Ottawa (ON): Statistics Canada; 2008 [cited 2010 Jul 20]. Available at: http://www12.statcan.ca/census-recensement/2006/as-sa/97-558/pdf/97-558-XIE2006001.pdf

15. Aboriginal Peoples Survey (APS) [Internet]. Ottawa (ON): Statistics Canada; 2009 [cited 2010 July 22]. Available at: http://www.statcan.gc.ca/imdb-bmdi/3250-eng.htm

16. Hare J. Aboriginal women and healthcare. Friends of women and children in B.C. Report Card, (2004). 3(12).

17. Chen Y, Horne SL, McDuffie HH, Dosman JA. Combined effect of grain farming and smoking on lung function and the prevalence of chronic bronchitis. Int J Epidemiol. 1991;20(2):416-23.

18. Menezes AM, Victora CG, Rigatto M. Prevalence and risk factors for chronic bronchitis in Pelotas, RS, Brazil: a population based study. Thorax. 1994;49:1217-21.

19. Sethi JM, Roschester CL. Smoking and chronic obstructive pulmonary disease. Clin Chest Med. 2000;21:67-86.

20. Obesity in Canada. Determinants and contributing factors [Internet]. Ottawa (ON): Statistics Canada; 2010 [cited 2010 Jul 20]. Available at: http://www.phac-aspc.gc.ca/hp-ps/hl-mvs/oic-oac/determ-eng.php

21. Guerra S, Sherrill DL, Bobadilla A, Martinez FD, Barbee RA. The relation of body mass index to asthma, chronic bronchitis, and emphysema. Chest. 2002;122:1256-63.

22. Poulain M, Doucet M, Major GC, et al. The effect of obesity on chronic respiratory diseases: pathophysiology and therapeutic strategies. CMAJ. 2006;174(9):1293-9.

23. Bobadilla A, Guerra S, Sherrill D, Barbee R. How accurate is the self-reported diagnosis of chronic bronchitis? Chest. 2002;122: 1234-9.

Changes in fall-related mortality in older adults in Quebec, 1981–2009

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Abstract

Introduction: Our purpose was to evaluate changes in fall-related mortality in adults aged 65 years and over in Quebec and to propose a case definition based on all the causes entered on Return of Death forms.

Methods: The analysis covers deaths between 1981 and 2009 recorded in the Quebec vital statistics data.

Results: While the number of fall-related deaths increased between 1981 and 2009, the adjusted falls-related mortality rate remained relatively stable. Since the early 2000s, this stability has masked opposing trends. The mortality rate associated with certified falls (W00–W19) has increased while the rate for presumed falls (exposure to an unspecified factor causing a fracture) has decreased.

Conclusion: For fall surveillance, analyses using indicators from the vital statistics data should include both certified falls and presumed falls. In addition, a possible shift in the coding of fall-related deaths toward secondary causes should be taken into account.

Keywords: *trends, mortality, falls, seniors, older adults, fractures, injuries, reporting, Quebec*

Introduction

Fall-related injuries among older adults are a major public health problem. Because of the severity of the outcome, fall-related mortality is one of the basic indicators of fall surveillance.[1]

While there are little recent Canadian data,[2] a substantial increase in fall-related mortality was recently reported in the population aged 65 years and over in the United States.[3-5] In the absence of significant changes in fall-related morbidity in the same period, Hu and Baker[6] recently suggested that this increase in fall-related mortality was due to improved recording of falls as the cause of death. However, their hypothesis depends on a debatable methodology. First, in contrast to similar studies,[7] their analyses do not include fractures from unspecified causes.[6] Inclusion of such fractures affects the scope of the problem considerably.[8-10] Since fractures from unspecified causes are usually hip fractures, and can thus be primarily attributed to falls,[11,12] these cases could be included in the analyses. Second, because most deaths do not result from a single cause but from a series of health problems,[13] the design of mortality indicators based solely on the initial cause of death has been criticized.[14-16] The importance of comorbidities in fall-related deaths,[17,18] and the greater likelihood of the injury being entered as a secondary cause of death in older women,[19] also suggests that all conditions entered on the Return of Death form could be analyzed to produce a more accurate picture of the trends. Thus, while causes of death are systematically recorded for administrative purposes, their use for public health surveillance is sometimes limited by a lack of accuracy. However, it appears possible to bypass this obstacle by refining the measures normally used.

The primary goal of our study was to describe the trends in mortality over time for fall-related deaths in adults aged 65 years and over in Quebec from 1981 to 2009 by identifying two major categories of fall-related deaths and determining whether these trends vary by sex and age. A secondary objective was to estimate the impact of a broader case definition based on the secondary causes of death and take into account a possible shift in the coding of fall-related deaths toward secondary causes.

Methodology

This study is a descriptive trend analysis of fall-related mortality in the Quebec population aged 65 years and over between 1981 and 2009.

Data sources

The data used in our study are from the Quebec Ministry of Health and Social Services (Santé et Services sociaux Québec; MSSS) vital statistics data. The database contains demographic and medical information on deaths in the Quebec population collected through the ''Return of Death,'' a document on which the causes and circumstances of death are

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entered as accurately as possible. The causes and circumstances have been recorded in this database using *International Classification of Diseases, 10th Revision* (ICD-10) codes since 2000, while *International Classification of Diseases, 9th Revision* (ICD-9) codes were used between 1981 and 1999. Since 1 January 2000, an underlying cause of death and up to 10 secondary causes can be recorded in the Quebec vital statistics data. Before 1 January 2000, only one secondary cause could be added to the underlying cause of death, specifically in cases of deaths attributed to an external cause.

Particular difficulties related to case definition

The use of ICD-10 rather than ICD-9 codes to record deaths in Canada has led to a major under-identification (by about 50%) of fall-related deaths.[10] The category for falls (E880–E888) in ICD-9 included E887, ''Fracture, cause unspecified.'' ICD-10 does not contain an equivalent code in the falls category (W00–W19). In Quebec, this situation is especially important because code E887 was used disproportionately compared to other Canadian provinces.[20] However, these deaths cannot simply be excluded from the analyses, because they generally result from a fall that the Return of Death form does not explicitly mention.[9,21]

Using a methodology proposed by Kreisfeld and Harrison,[21] we first identified deaths specifically associated with a fall as the underlying cause of death, defined here as the injury that initiated the train of morbid events leading directly to death.[22] These deaths are categorized as ''certified falls'' (Table 1). We also created another category, ''presumed falls,'' to satisfactorily estimate the extent of fall-related deaths and identify a seamless trend in spite of the changes in ICD classification. For the years when deaths were coded using ICD-9, 1981 to 1999, the presumed falls category was made up of ''fractures of unspecified causes'' (code E887). Since 2000, the ''presumed falls'' category has been made up of deaths due to ''exposure to unspecified factors'' (code X59) with at least one fracture recorded among the secondary causes. (The World Health Organization recently introduced code X59.0, ''Exposure to unspecified factor causing fracture,'' to compensate for the difficulties caused by the discontinuation of code E887.[22]) We also examined all the secondary causes of death entered on the Return of Death forms to identify ''additional falls,'' including those cases where a fall or exposure to an unspecified factor was not specified as the underlying cause of death (see Table 1). We selected both the specific codes for falls and those for exposure to an unspecified factor combined with a fracture code. Because it is based on the secondary causes of death, this identification strategy is only possible for the years since 2000. This complementary category makes it possible to take into account a possible shift of fall-related deaths toward secondary causes.

Statistical analysis

We calculated the number of fall-related deaths and annual rates using population estimates for the years 1981 to 2005 and population projections for the years 2006 to 2009.[23] The rates are shown per 100 000 population and express the number of deaths in a year in relation to the number of individuals at risk for the same period (estimated from population numbers as of July 1 of each corresponding year). The rates shown for the population aged 65 years and over were standardized using the direct method to limit the confounding effects created by differences related to the population age structure and also to permit comparisons over time. The 2001 Quebec population was chosen as the reference population. We also calculated specific rates by sex and age group.

We used negative binomial modelling to determine whether the time trends for fall-related mortality rates were statistically significant. This strategy is especially suited to modelling a count of events in a given period in which a parameter related to overdispersion must be controlled for.[24] The model includes the intercept (α), the parameters associated with the variables included in the model (β_i) and an overdispersion term ($\sigma\varepsilon$), and takes the following form:

$$\ln(\text{number of deaths}) = \alpha + \beta_{\text{year}} + \beta_{\text{age}} + \beta_{\text{sex}} + \ln(\text{population}) + \sigma\varepsilon$$

To model the trends of the annual rates of fall-related mortality, two periods were chosen to mitigate the transition from ICD-9 to ICD-10 codes and evaluate the impact of a case definition based on the secondary causes of death available only since 2000. The first period includes the years 1981 to 1999, whereas the second is from 2000 to 2009, thus covering the last 10 years of the period under study. For each of the two periods, the parameter associated with the year (β_{year}) was used to estimate the annual average percentage change (AAPC) in fall-related mortality rates. The AAPC used to describe the trend was calculated as follows:

$$\text{AAPC} = (e^{\beta_{\text{year}}} - 1) \times 100$$

TABLE 1
List of codes for fall-related deaths by ICD version

Terminology used	Ninth revision of the International Classification of Diseases (ICD-9)	Tenth revision of the International Classification of Diseases (ICD-10)
Certified falls	E880–E886 or E888 as primary cause of death	W00–W19 as underlying cause of death (e.g. fall on stairs or from bed)
Presumed falls	E887 as primary cause of death	X59 as underlying cause of death and at least one fracture code recorded among the secondary causes (e.g. hip fracture)
Additional falls	—	Fall codes, certified or presumed, recorded among the secondary causes, irrespective of the primary cause (e.g. hip fracture and code X59 among the secondary causes, the primary cause of which corresponds to Alzheimer's disease)

We calculated 95% confidence intervals (CI) for the AAPCs using the Wald method. These estimates demonstrate whether the rate trend is, generally speaking, increasing or decreasing over a given period. The modelling strategy was also used to illustrate the time trends established based on the number of deaths predicted by the model and population estimates. All statistical analyses were performed using SAS statistical software version 9.2 (SAS Institute Inc., Cary, NC, US).

Results

In Quebec, the number of deaths directly associated with a certified or presumed fall rose from 255 in 1981 to 819 in 2009 in the population aged 65 years and over. During this period, the adjusted fall-related mortality rate varied from 48.8 to 71.1 deaths per 100 000 population (Table 2). The annual numbers of fall-related deaths were higher in women than in men. On the other hand, the adjusted mortality rates were higher in men (Table 2 and Figure 1). Since the early 2000s, adjusted fall-related mortality rates have shown no significant variation in women, but have shown a downward trend in men, especially those aged 85 and over (Table 3). In addition, the increase in fall-related mortality rates (certified or presumed) observed in the 1980s and 1990s in women aged 85 and over seems to have stopped in the early 2000s (Table 3 and Figure 2).

Since the early 2000s, the rate of certified falls rose by an average of 3.0% per year in men and 6.3% in women. On the other hand, the rate of presumed falls fell by an average of 4.5% per year in men and 3.5% in women (Table 4 and Figure 3).

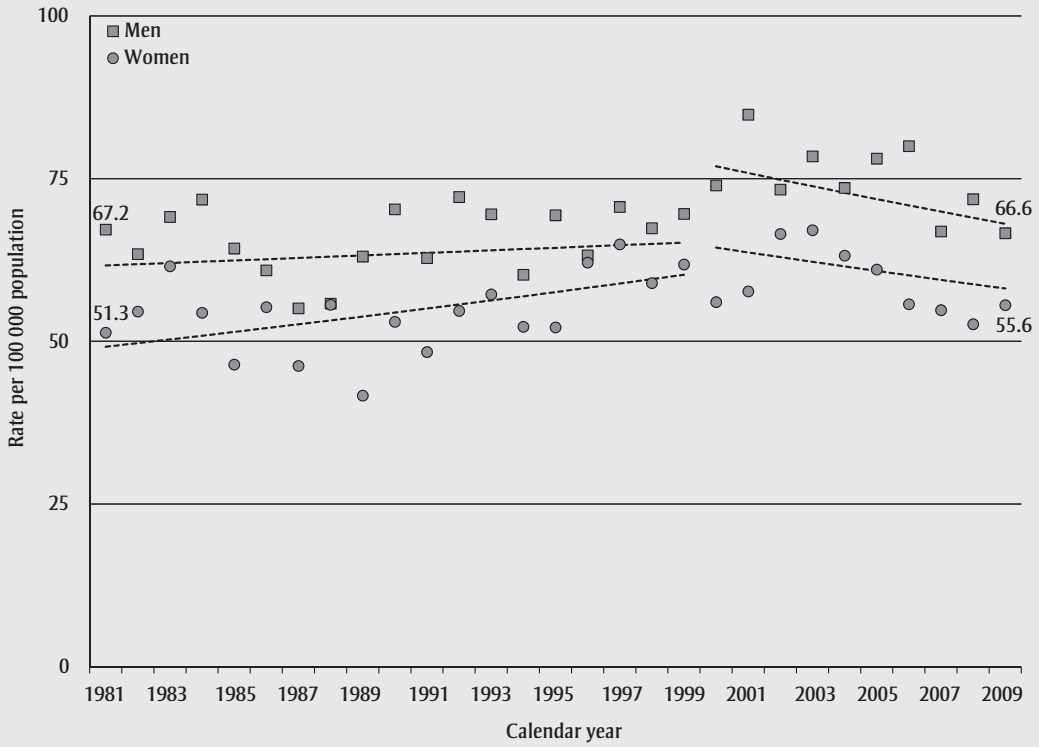
When the analyses include only secondary causes (additional falls), no significant variation appears in either men or women (Table 4 and Figure 3). However, this seems to be largely due to the low rates observed for the years 2000 and 2001 for

TABLE 2
Number and adjusted rate of deaths related to certified or presumed falls per 100 000 population, ≥ 65 years, by sex, Quebec, 1981–2009

Year	Men		Women		Sexes combined		
	Number	Rate	Number	Rate	Number	Rate	*p** value
1981	112	67.2	143	51.3	255	57.5	.039
1982	107	63.4	161	54.6	268	58.1	.244
1983	118	69.1	189	61.5	307	64.6	.335
1984	126	71.8	176	54.4	302	60.7	.020
1985	119	64.3	159	46.4	278	52.9	.009
1986	113	60.9	197	55.3	310	57.6	.422
1987	109	55.1	176	46.2	285	49.5	.162
1988	115	55.8	222	55.6	337	56.1	.979
1989	132	63.0	175	41.7	307	48.8	.001
1990	161	70.3	233	53.0	394	59.7	.007
1991	143	62.8	223	48.4	366	53.3	.017
1992	163	72.2	264	54.7	427	59.8	.006
1993	177	69.5	289	57.2	466	62.7	.045
1994	150	60.2	273	52.2	423	55.3	.171
1995	172	69.4	281	52.1	453	57.7	.004
1996	167	63.2	345	62.1	512	63.6	.854
1997	189	70.7	373	64.9	562	67.2	.353
1998	188	67.4	352	59.0	540	62.6	.149
1999	197	69.6	381	61.8	578	64.9	.187
2000	223	74.0	362	56.0	585	63.0	.001
2001	258	84.8	387	57.7	645	66.9	< .001
2002	234	73.3	461	66.5	695	69.4	.234
2003	257	78.4	485	67.1	742	71.1	.047
2004	263	73.6	474	63.2	737	68.3	.052
2005	289	78.1	475	61.0	764	67.9	.001
2006	314	80.0	453	55.7	767	64.8	< .001
2007	277	66.9	456	54.8	733	59.8	.010
2008	310	71.8	462	52.6	772	59.7	< .001
2009	305	66.6	514	55.6	819	60.8	.013

* *p* value associated with the difference between the adjusted rates for men and women for a given year. A value of less than .05 indicates that the difference is statistically significant.

FIGURE 1
Adjusted mortality rate for certified or presumed falls, population ≥ 65 years, by sex, Quebec, 1981–2009



this type of death. Excluding these two years from the analyses, the trend is similar to the one for presumed falls (AAPC of −4% and −6.3% for men and women, respectively) (Table 4).

Discussion

Owing to the aging population, the number of fall-related deaths in Quebec increased between 2000 and 2009. In contrast, the adjusted mortality rate remained fairly stable in women and even decreased slightly in men. However, this relative statistical stability has masked opposing trends. The mortality rate for falls specifically recorded as the underlying cause of death (certified falls) increased, whereas the mortality rate associated with fractures of unspecified cause (presumed falls) decreased in both men and women. Between 2002 and 2009, the decline in the mortality rate associated with falls mentioned among the secondary causes (additional falls) corresponds to the reduction in the mortality rate associated with presumed falls, which suggests that the deaths removed from the presumed falls are not among the secondary causes. For the final analysis, the calcula-

TABLE 3
Annual average percentage change (AAPC) in the mortality rate for certified falls or presumed falls, population ≥ 65 years, by sex and age group, Quebec, 1981–1999 and 2000–2009

Age range, years	Time period	Men		Women	
		AAPC	95% CI	AAPC	95% CI
65–74	1981–1999	−0.2	(−1.7 to 1.3)	0.8	(−1.3 to 2.9)
	2000–2009	−0.8	(−5.1 to 3.9)	−3.1	(−6.4 to 0.3)
75–84	1981–1999	−0.0	(−1.1 to 1.1)	0.6	(−0.2 to 1.4)
	2000–2009	−1.2	(−2.9 to 0.5)	0.3	(−0.7 to 1.3)
≥ 85	1981–1999	0.9	(−0.1 to 1.9)	1.6[a]	(0.7 to 2.6)
	2000–2009	−1.7[a]	(−3.2 to −0.1)	−1.7	(−3.5 to 0.2)
Total ≥ 65	1981–1999	0.3	(−0.4 to 1.0)	1.1[a]	(0.5 to 1.8)
	2000–2009	−1.3[a]	(−2.5 to −0.1)	−1.1	(−2.4 to 0.1)

Abbreviations: AAPC, annual average percentage change; CI, confidence interval.

[a] Significant AAPC.

FIGURE 2
Mortality rate for certified or presumed falls, population ≥ 65 years, by age group and sex, Quebec, 1981–2009

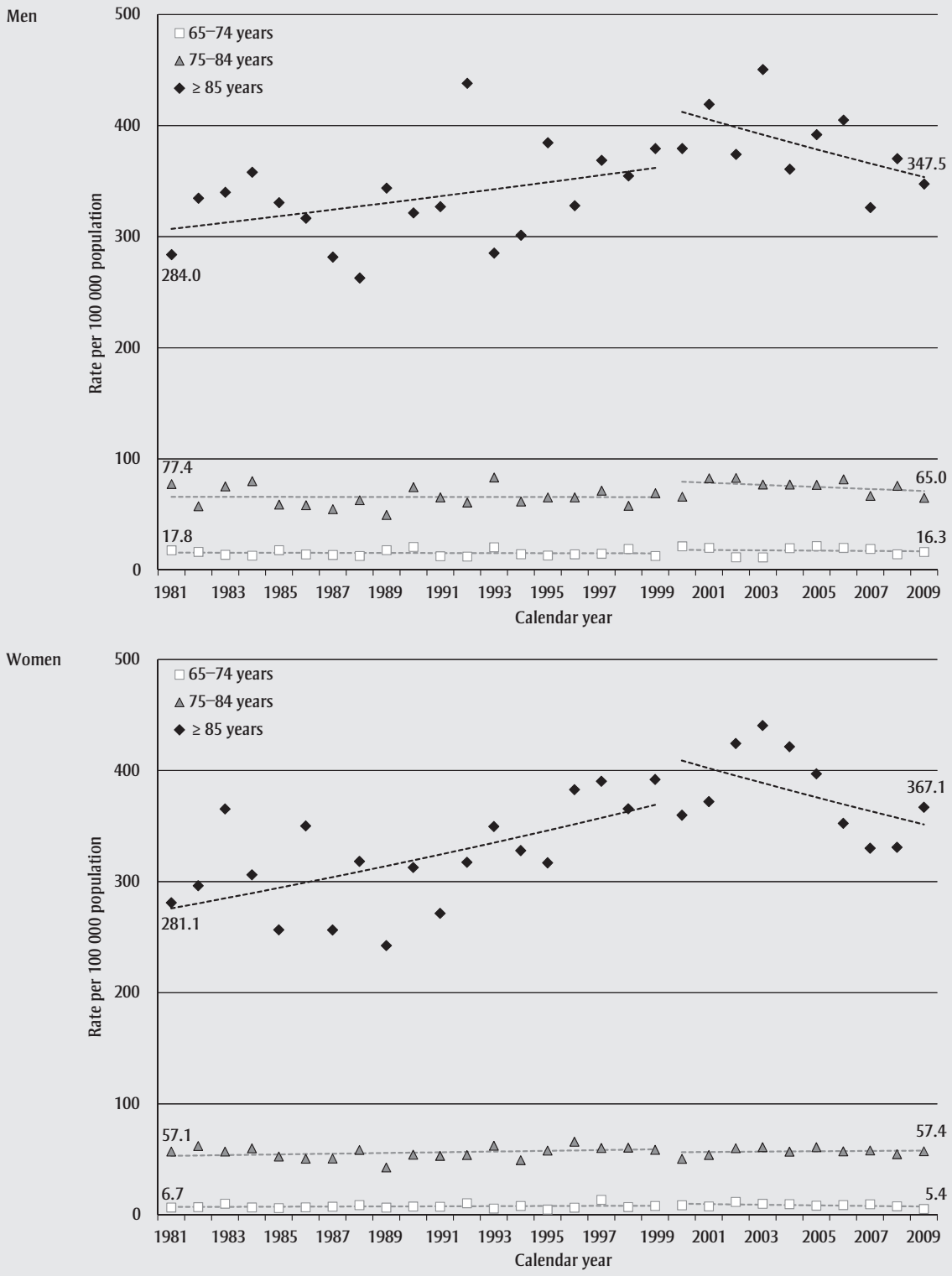


TABLE 4
Annual average percentage change (AAPC) in the fall-related mortality rate, population ≥ 65 years, by fall category and sex, 1985–1999 and 2000–2009

		Men		Women	
	Segment	AAPC	95% CI	AAPC	95% CI
Certified fall	1985–1999	1.9	(0.1 to 3.9)	2.7[a]	(0.4 to 5.1)
	2000–2009	3.0[a]	(0.8 to 5.3)	6.3[a]	(4.6 to 8.0)
	2002–2009	3.9[a]	(0.8 to 7.0)	5.9[a]	(3.6 to 8.1)
Presumed fall	1985–1999	0.5	(−0.7 to 1.7)	2.1[a]	(1.4 to 2.9)
	2000–2009	−4.5[a]	(−5.6 to −3.2)	−3.5[a]	(−5.0 to −1.9)
	2002–2009	−5.5[a]	(−6.9 to −4.2)	−6.1[a]	(−7.5 to −4.6)
Total	1985–1999	0.9	(0.0 to 1.8)	2.2[a]	(1.4 to 3.0)
	2000–2009	−1.3[a]	(−2.5 to −0.1)	−1.1	(−2.4 to 0.1)
	2002–2009	−1.6	(−3.2 to 0.0)	−3.7[a]	(−4.6 to −1.9)
Additional fall	1985–1999	—	—	—	—
	2000–2009	1.0	(−1.9 to 3.9)	−0.5	(−3.7 to 2.7)
	2002–2009	−4.0[a]	(−6.1 to −1.9)	−6.3[a]	(−7.9 to −4.6)

Abbreviations: AAPC, annual average percentage change; CI, confidence interval.

Note: The years 1981–1984, which precede a directive issued by Statistics Canada on the coding of deaths, were excluded from the analyses.

[a] Significant annual AAPC.

tions for the years 2000 and 2001 were excluded because of the low rates observed, probably due to this being the time of transition to the new ICD.

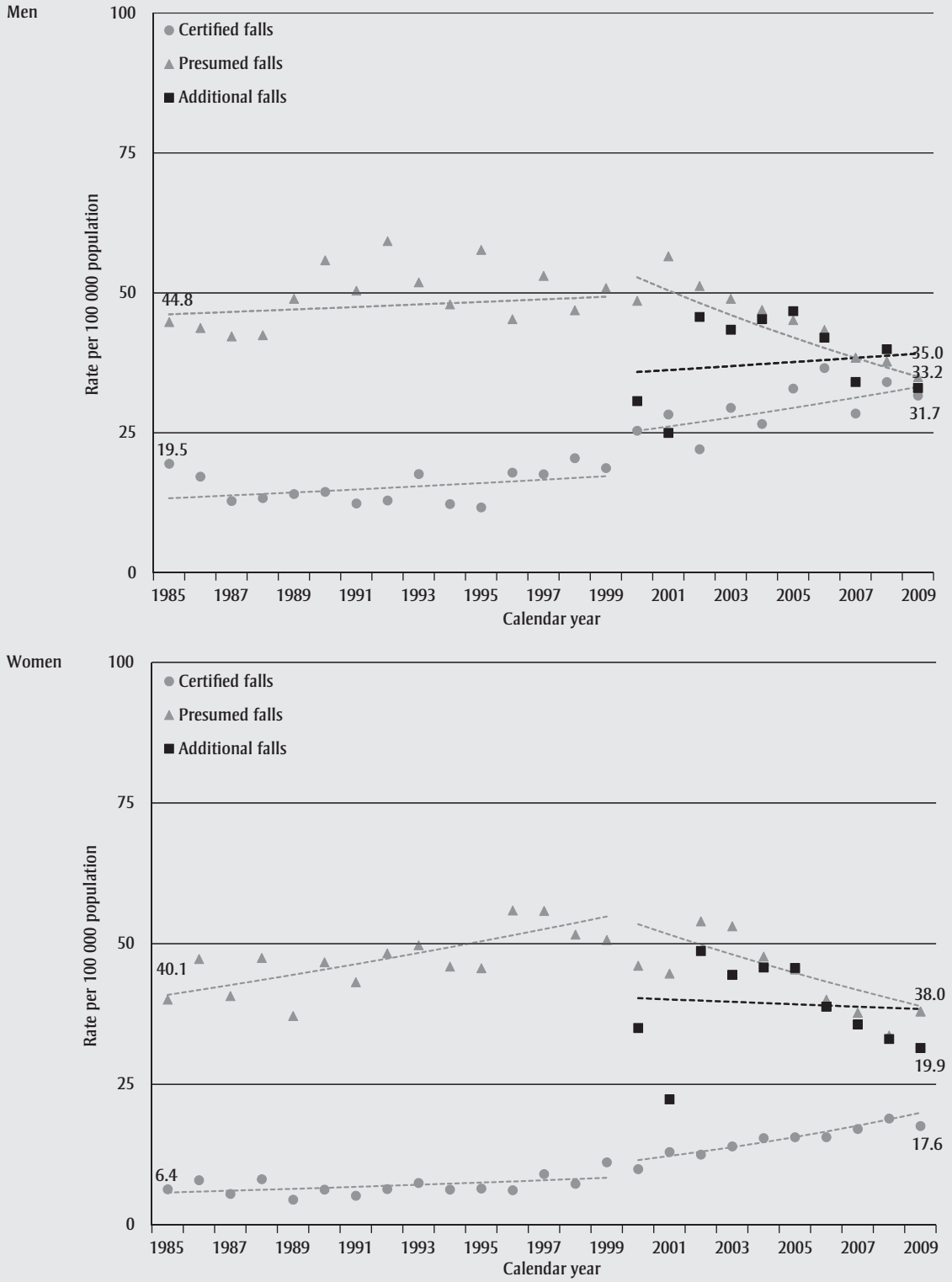
In Canada as a whole, the mortality rate for certified falls in the adult population aged 65 years and over rose significantly between 1997/1999 and 2000/2002, especially in women.[2] A similar upward trend occurred in the United States, where the mortality rate for certified falls in this age group rose by 42% between 2000 and 2006.[4] In the Netherlands, a smaller increase has been observed in men since 1997, despite that the presumed falls category was also included in the analyses.[7] In Finland, the mortality rate due to certified falls has fallen in women since the early 2000s.[25]

The small increases in the rates of fall-related emergency department visits or hospital admissions in the United States is at odds with the large increase in fall-related mortality rate (42% between 2000 and 2006) in older adults.[4] This apparent discrepancy has led to the suggestion that this difference is as a result of more falls being selected as the initial cause of death.[4,6] Our results seem to confirm this hypothesis, since the decrease in the mortality rate for presumed falls seems to be partially compensated for by an increase in deaths related to certified falls. This finding also holds when the mortality rate takes into account all secondary causes.

Is the trend in the adjusted fall-related mortality rate associated with improved recording of cause of death?

Most deaths in older adults result from a combination of morbidities, the chronological sequence of which can be difficult to establish.[26-27] The number of deaths as a direct result of falls may be underreported.[28] In the case of older women who die after a fall,[29] who present with multiple medical conditions[30] and who die following a long period of hospitalization[29] (as is generally the case with hip fractures[31]), the cause of death is less likely to be attributed to the correct underlying cause. Reporting on the causes of death could be more accurate,[32] and it is possible that the trends observed in Quebec are the result of improved identification of fall-related deaths as certified falls. On the other hand, as has been reported elsewhere,[9,16] the presumed falls and the additional falls categories are essentially made up of hip fractures of unspecified external cause (see Appendix A). That said, the incidence of hip fractures seems to be declining in several countries[33-36] including Canada.[37] Similarly, despite the persistent excess mortality associated with hip fractures,[38] the fatality rate seems to have declined in recent years.[39,40] Because the mortality rate results from the combination of incidence and fatality related to a health problem, it seems plausible that the decline in the adjusted mortality rate associated with presumed falls reflects a change related to hip fractures. The increase in the mortality rate associated with certified falls may also be due in part to the increase in the incidence of traumatic brain injury-related deaths in older adults[41] because the circumstances surrounding these deaths are more likely to be accurately recorded.[31]

FIGURE 3
Fall-related mortality rate per 100 000 population, ≥ 65 years, by fall category and sex, Quebec, 1985–2009



Risk factors and fall prevention

While we do not attempt to identify the determinants of the observed trends in this article, it is worth mentioning that many factors may have influenced the changes in fall-related mortality over the period of this study.

Falls among older adults generally result from a complex interaction of risk factors associated with the growing vulnerability of this population due to aging and illness.[42] Impaired balance can increase risk of falls, as can chronic health problems such as hypotension, cardiovascular disease[43] and the use of certain prescription drugs.[2,43]

Some interventions, including improving individuals' physical capacity, have proven effective in reducing the likelihood of falls.[44] Since the mid-2000s, MSSS has taken various measures to prevent falls among older adults in Quebec, particularly those with balance issues.[45] These measures include having health providers monitor risk factors among older patients.[45] While the interventions are generally considered effective,[44] their benefits have only been demonstrated with respect to the risk of falls and not with respect to mortality. In addition, the interventions had been only partially implemented in Quebec by 2008[46] despite that fall prevention has been a concern for a number of years.

Strengths and limitations

This study has several limitations. First, we did not examine the validity and accuracy of the causes of death recorded on Return of Death forms in Quebec. The quality of vital statistics information has been criticized in various countries, particularly with respect to identifying underlying causes of death[15,16,31] and the accuracy of the recorded external causes.[19,47] The use of a broader case definition appears to have mitigated the effects of replacing specific codes for external causes with unspecific codes. This strategy has also limited the under-identification of fall-related deaths due to the transition from ICD-9 to ICD-10. Second, our study does not encompass the many known risk factors for falls that might have influenced the reported time trends. The inclusion of these factors could explain a portion of the fluctuations observed here. Finally, most falls do not result in death. This overview portrays only the tip of the iceberg. Further analyses could build on efforts to refine the surveillance indicators for fall-related morbidity[48] and look at whether the trends reported here reflect the changes in the incidence and fatality of fall-related injuries.

Conclusion

Because of the aging of the population, the number of fall-related deaths rose between 2000 and 2009 in Quebec. However, the adjusted fall-related mortality rate in people aged 65 years and over remained fairly stable in women and even fell slightly in men. This information is significant because—to the extent that incidence and fatality associated with these injuries does not change—the frequency of fall-related injuries will likely rise in the coming years as the population continues to age.

So far, no standard definition has been suggested to analyze and describe the extent of fall-related deaths in older Canadians. The definition used in our study merits attention. Using it has practical implications for measuring the problem because it resolves the under-identification and apparent decrease in fall-related deaths created by the transition to ICD-10. Studies designed to estimate the extent and time trends of fall-related mortality should include certified falls (W00–W19) and the presumed falls coded as being due to exposure to an unspecified factor (X59) causing a fracture. The possible shift in coding from fall-related deaths to secondary causes should also be taken into consideration so as to identify additional cases of fall-related deaths.

APPENDIX A
Characteristics of fall-related deaths, population ≥ 65 years, by fall category, Quebec, 2000–2009

	Certified fall		Presumed fall		Additional fall	
	N[a]	%	N[a]	%	N[a]	%
Sex						
Men	117	51.1	156	31.4	142	33.1
Women	112	48.9	341	68.6	387	66.9
Age group, years						
65–74	46	20.0	28	5.6	41	9.6
75–84	82	36.0	146	29.3	148	34.5
≥ 85	101	44.0	324	65.2	240	56.0
Hip fracture						
Yes	20	8.8	390	78.3	290	67.7
No	209	91.2	108	21.7	138	32.3
Traumatic brain injury						
Yes	133	58.4	3	0.6	13	2.9
No	95	41.6	495	99.4	416	97.1
Total	229	100.0	497	100	429	100
Age, years						
Mean (SD)	82.4	(8.4)	86.8	(7.2)	85.1	(7.5)
Median	83		87		86	
Secondary causes of death	N[b]		N[b]		N[b]	
Mean number (SD)	4.7	(2.1)	5.0	(1.8)	5.8	(1.8)
Median number	4		5		5	

Abbreviation: SD, standard deviation.

[a] Average annual number, average or median value.

[b] Average annual number or median number of medical conditions among vital statistics data.

References

1. Dowling AM, Finch CF. Baseline indicators for measuring progress in preventing falls injury in older people. Aust N Z J Public Health. 2009 Oct;33(5):413-7.

2. Public Health Agency of Canada. Report on seniors' falls in Canada. Ottawa (ON): Public Health Agency of Canada, Division of Aging and Seniors; 2005. Catalogue No.: HP25-1/20005E.

3. Dessypris N, Dikalioti SK, Skalkidis I, Sergentanis TN, Terzidis A, Petridou ET. Combating unintentional injury in the United States: lessons learned from the ICD-10 classification period. J Trauma. 2009 Feb;66(2):519-25.

4. Hu G, Baker SP. Recent increases in fatal and non-fatal injury among people aged 65 years and over in the USA. Inj Prev. 2010 Feb;16(1):26-30.

5. Paulozzi LJ, Ballesteros MF, Stevens JA. Recent trends in mortality from unintentional injury in the United States. J Safety Res. 2006;37(3):277-83.

6. Hu G, Baker SP. An explanation for the recent increase in the fall death rate among older Americans: a subgroup analysis. Public Health Rep. 2012 May;127(3): 275-81.

7. Hartholt KA, Polinder S, van Beeck EF, et al. End of the spectacular decrease in fall-related mortality rate: men are catching up. Am J Public Health. 2012 Mar 8;Suppl 2:S207-11.

8. Gjertsen F, Bruzzone S, Vollrath ME, Pace M, Ekeberg O. Comparing ICD-9 and ICD-10: the impact on intentional and unintentional injury mortality statistics in Italy and Norway. Injury. 2013 Jan;44(1):132-8.

9. Griffiths C, Rooney C. The effect of the introduction of ICD-10 on trends in mortality from injury and poisoning in England and Wales. Health Stat Q. 2003;19:10-21.

10. Statistics Canada. Comparability of ICD-10 and ICD-9 for mortality statistics in Canada. Ottawa (ON): Statistics Canada; 2005. Catalogue No.: 84-548-XIE.

11. Nyberg L, Gustafson Y, Berggren D, Brannstrom B, Bucht G. Falls leading to femoral neck fractures in lucid older people. J Am Geriatr Soc. 1996 Feb;44(2):156-60.

12. Parkkari J, Kannus P, Palvanen M, et al. Majority of hip fractures occur as a result of a fall and impact on the greater trochanter of the femur: a prospective controlled hip fracture study with 206 consecutive patients. Calcif Tissue Int. 1999 Sep;65(3):183-7.

13. Rothman KJ, Greenland S. Causation and causal inference in epidemiology. Am J Public Health. 2005;95 Suppl 1:S144-50.

14. Cryer C, Gulliver P, Langley J, Davie G, Samaranayaka A, Fowler C. A proposed theoretical definition to address the under-counting of injury deaths. Inj Prev. 2011 Aug;17(4):219-21.

15. Jansson B. Coding errors and underestimation of fall injury mortality. Am J Public Health. 2005 Aug;95(8):1305-6.

16. Kreisfeld R, Harrison JE. Use of multiple causes of death data for identifying and reporting injury mortality. Canberra (AU): AIHW; 2007. Injury Technical Paper Series n°9. AIHW Catalogue No.: INJCAT 98.

17. Deprey SM. Descriptive analysis of fatal falls of older adults in a Midwestern county in the year 2005. J Geriatr Phys Ther. 2009;32(2):67-72.

18. Wilkins K, Wysocki M, Morin C, Wood P. Multiple causes of death. Health Rep. 1997;9(2):21-32.

19. McKenzie K, Chen L, Walker SM. Correlates of undefined cause of injury coded mortality data in Australia. HIM J. 2009;38(1):8-14.

20. Gagné M, Robitaille Y, Hamel D. Note technique concernant les regroupements pour l'analyse des décès par traumatisme au Québec. Québec (QC): INSPQ; 2009.

21. Kreisfeld R, Harrison JE. Injury deaths, Australia, 1999: with a focus on the transition from ICD-9 to ICD-10. Adelaide (AU): AIHW; 2005. Report No.: AIHW Catalogue No.: INJCAT 67.

22. World Health Organization, Update and Revision Committee. Cumulative official updates to ICD-10. WHO Collaborating Centres for the Family of International Classifications; 2011.

23. Ministère de la Santé et des Services sociaux. La population du Québec par territoire des centres locaux de services communautaires, par territoire des réseaux locaux de services et par région socio-sanitaire, de 1981 à 2031. Québec (QC) : Ministère de la Santé et des Services sociaux, Service du développement de l'information/Publications du Québec; 2010.

24. Bouche G, Lepage B, Migeot V, Ingrand P. Intérêt de la détection et de la prise en compte d'une surdispersion dans un modèle de Poisson : illustration à partir d'un exemple. Rev Epidemiol Sante Publique. 2009 Aug;57(4):285-96.

25. Korhonen N, Niemi S, Parkkari J, Palvanen M, Kannus P. Unintentional injury deaths among adult Finns in 1971-2008. Injury. 2011 Sep;42(9):885-8.

26. Ravakhah K. Nobody dies of old age any more? J Palliat Med. 2011 Apr;14(4):386.

27. Shannon RP, Green AR. Dying of natural and specific causes in old age, not of old age. J Palliat Med. 2011 Sep;14(9):984-5.

28. Koehler SA, Weiss HB, Shakir A, et al. Accurately assessing elderly fall deaths using hospital discharge and vital statistics data. Am J Forensic Med Pathol. 2006 Mar;27(1):30-5.

29. Dijkhuis H, Zwerling C, Parrish G, Bennett T, Kemper HC. Medical examiner data in injury surveillance: a comparison with death certificates. Am J Epidemiol. 1994 Mar 15;139(6):637-43.

30. Charles A, Ranson D, Bohensky M, Ibrahim JE. Under-reporting of deaths to the coroner by doctors: a retrospective review of deaths in two hospitals in Melbourne, Australia. Int J Qual Health Care. 2007 Aug;19(4): 232-6.

31. Cryer C, Gulliver P, Samaranayaka A, Davie G, Langley J, Fowler C. New Zealand Injury Prevention Strategy indicators of injury death: are we counting all the cases? Dunedin (NZ): University of Otago; 2010 Aug. IPRU Occasional Report OR085.

32. Aung E, Rao C, Walker S. Teaching cause-of-death certification: lessons from international experience. Postgrad Med J. 2010 Mar;86(1013):143-52.

33. Cassell E, Clapperton A. A decreasing trend in fall-related hip fracture incidence in Victoria, Australia. Osteoporos Int. 2013 Jan;24(1):99-109.

34. Chevalley T, Guilley E, Herrmann FR, Hoffmeyer P, Rapin CH, Rizzoli R. Incidence of hip fracture over a 10-year period (1991-2000): reversal of a secular trend. Bone. 2007 May;40(5):1284-9.

35. Kannus P, Niemi S, Parkkari J, Palvanen M, Vuori I, Jarvinen M. Nationwide decline in incidence of hip fracture. J Bone Miner Res. 2006 Dec;21(12):1836-8.

36. Stevens JA, Anne RR. Declining hip fracture rates in the United States. Age Ageing. 2010 Jul;39(4):500-3.

37. Leslie WD, O'Donnell S, Jean S, et al. Trends in hip fracture rates in Canada. JAMA. 2009 Aug 26;302(8):883-9.

38. Haentjens P, Magaziner J, Colon-Emeric CS, et al. Meta-analysis: excess mortality after hip fracture among older women and men. Ann Intern Med. 2010 Mar 16;152(6):380-90.

39. Brauer CA, Coca-Perraillon M, Cutler DM, Rosen AB. Incidence and mortality of hip fractures in the United States. JAMA. 2009 Oct 14;302(14):1573-9.

40. Maravic M, Taupin P, Landais P, Roux C. Decrease of inpatient mortality for hip fracture in France. Joint Bone Spine. 2011 Oct;78(5):506-9.

41. Coronado VG, Xu L, Basavaraju SV, et al. Surveillance for traumatic brain injury-related deaths--United States, 1997-2007. MMWR Surveill Summ. 2011 May 6;60(5):1-32.

42. Rubenstein LZ. Falls in older people: epidemiology, risk factors and strategies for prevention. Age Ageing. 2006 Sep;35 Suppl 2:ii37-41.

43. Tinetti ME, Kumar C. The patient who falls: ''It's always a trade-off''. JAMA. 2010 Jan 20;303(3):258-66.

44. Gillespie LD, Robertson MC, Gillespie WJ, et al. Interventions for preventing falls in older people living in the community. Cochrane Database Syst Rev.2009;(2): CD007146.

45. Ministère de la Santé et des Services sociaux. La prévention des chutes dans un continuum de services pour les aînés vivant à domicile : Cadre de référence. Québec (QC) : Ministère de la Santé et des Services sociaux, Direction générale de la santé publique; 2004.

46. Champagne F, Gagnon I, Baldé T. Évaluation de l'implantation du continuum de services en prévention des chutes chez les aînés vivant à domicile : Rapport final. Montréal (QC): Université de Montréal, Groupe de recherche interdisciplinaire en santé; 2009.

47. Lu TH, Walker S, Anderson RN, McKenzie K, Bjorkenstam C, Hou WH. Proportion of injury deaths with unspecified external cause codes: a comparison of Australia, Sweden, Taiwan and the US. Inj Prev. 2007 Aug;13(4):276-81.

48. Robitaille Y, Gratton J. Les chutes chez les adultes âgés : vers une surveillance plus fine des données d'hospitalisation. Quebec (QC) : Institut national de santé publique du Québec; 2005.

Improved estimation of the health and economic burden of chronic disease risk factors in Manitoba

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Abstract

Introduction: There are analytic challenges involved with estimating the aggregate burden of multiple risk factors (RFs) in a population. We describe a methodology to account for overlapping RFs in some sub-populations, a phenomenon that leads to ''double-counting'' the diseases and economic burden generated by those factors.

Methods: Our method uses an efficient approach to accurately analyze the aggregate economic burden of chronic disease across a multifactorial system. In addition, it involves considering the effect of body weight as a continuous or polytomous exposure that ranges from no excess weight through overweight to obesity. We then apply this method to smoking, physical inactivity and overweight/obesity in Manitoba, a province of Canada.

Results: The annual aggregate economic burden of the RFs in Manitoba in 2008 is about $1.6 billion ($557 million for smoking, $299 million for physical inactivity and $747 million for overweight/obesity). The total burden represents a 12.6% downward adjustment to account for the effect of multiple RFs in some individuals in the population.

Conclusion: An improved estimate of the aggregate economic burden of multiple RFs in a given population can assist in prioritizing and gaining support for primary prevention initiatives.

Keywords: *population attributable fraction, risk factors, obesity, physical inactivity, tobacco smoking, chronic disease*

Introduction

Health care planners have long been concerned with the ''epidemiologic transition,'' the process whereby chronic illnesses displace pandemics of infection as the primary source of morbidity and mortality in the world.[1] The latest phase of this transition is marked by increased prevalence of overweight/obesity and physical inactivity in many countries.[2] Excess body weight and/or physical inactivity have been implicated in chronic diseases such as cardiovascular disease, stroke, type 2 diabetes, chronic kidney disease, osteoarthritis and certain cancers.[3-12] Consequently, these risk factors (RFs) have joined tobacco smoking[13] as key prevention targets.

Estimations of the economic burden generated by such RFs have been undertaken in many jurisdictions in the world,[14] including Canada as a whole[15-19] and a few Canadian provinces.[20,21] In addition to understanding the costs related to a single RF such as tobacco smoking, estimating the aggregate economic burden generated by two or more RFs in a population is often of interest. This information can inform prevention strategies aimed at more than one RF, for example, public health programs that address both physical inactivity and overweight/obesity. There are, however, analytical challenges involved with the estimation of the aggregate burden of multiple RFs in a population.[22] Certain costs (such as those generated by incident disease or by death) are by definition accrued only once. Thus, it is important to account for the confounding effect of multiple RFs in the same individual, and specifically to adjust for any increase in the calculated economic burden due to double-counting cases and costs.

Population attributable fraction (PAF) offers a powerful way to interpret causation in the practical terms of prevention. In short, PAF is that *proportion of disease incidence (or costs) that will be removed if exposure to the causative RF is removed*. The approach, however, becomes more complicated when the aim is to assess the combined effect of multiple RFs.

A number of innovative approaches have been developed to quantify the effects of multiple RFs in specific cohorts.[23] The World Cancer Research Fund, for example, used a process that could be described as ''sequential prevention,'' explained as follows:[24,p149]

> Because no individual case of cancer can be prevented more than once, this calculation was done in a way that avoided the possibility of ''double

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counting.'' The PAF for the first exposure was subtracted from 100 per cent and the PAF for the second exposure was applied to the remainder. This process was performed sequentially for all relevant exposures, resulting in an estimated PAF for all exposures combined.

While it makes sense as a goal, the work of disentangling the impact of overlapping RFs is often omitted from estimations of the related economic burden. A case in point is the series of papers published from 2005 to 2009 by a British Heart Foundation group on the burden of ill health in the United Kingdom due to physical inactivity,[25] overweight/obesity,[26] tobacco smoking[27] and other RFs.[28,29] In a summary paper, the authors acknowledge that ''the possible overlap between risk factors (such as overweight and obesity) was not addressed here but should be considered when calculating the total economic burden of these risk factors.''[30,p534]

To address this challenge, we describe a methodology to account for overlapping RFs when estimating the aggregate economic burden of associated chronic illnesses. The approach involves four steps: (1) consideration of the function of body weight as a continuous or ''polytomous'' exposure ranging from no excess weight through overweight to obesity; (2) estimating an aggregate burden of chronic disease across a multifactorial system in a manner that adjusts for the effect of more than one RF; (3) estimating the aggregate economic burden adjusted for multiple RFs occurring in some individuals; and (4) disaggregating the total burden to provide an estimate of the economic cost notionally attached to each RF.

To our knowledge, this is the first published attempt to address the issue of double-counting costs due to overlapping RFs in some individuals when addressing the economic burden of multiple RFs.

As a demonstration of the utility of this approach, the economic burden of diseases attributable to tobacco smoking, physical inactivity, and overweight/obesity are estimated for the Canadian province of Manitoba. Manitoba has a population of about 1.2 million.[31] Although the province is marked by a strong agriculture and resource-based economy, some 60% of Manitobans reside in Winnipeg, the provincial capital. There is also a large First Nations presence in Manitoba (about 11% of the provincial population).[32]

Methods

We used an approach based on PAF to estimate the economic burden associated with the various RFs. At its simplest, the PAF statistic refers to the proportion of disease incidence generated in a population by a particular RF.[33] The results we report in this paper required calculating a Manitoba-specific PAF for each of the diseases related to the RFs of interest and then combining that information with the estimates of Manitoba-specific costs associated with both disease treatment and the indirect impacts of mortality/morbidity.

PAF is a statistic that combines two facets of an RF and its impact on disease: relative risk (RR) of the RF in reference to a particular disease, and the prevalence of exposure to the RF in the population of interest.

Relative risk

The source for the RRs associated with physical inactivity is the meta-analyses by Katzmarzyk and Janssen.[16] The majority of the studies incorporated in the Katzmarzyk and Janssen[16] review include an index of obesity in the analysis so that the effects of physical activity on disease risk can be considered to be independent of obesity. The source for the RRs associated with overweight and obesity is the meta-analyses by Guh et al.[34] The authors did not include physical inactivity as a potentially confounding RF as ''physical inactivity is often poorly reported and requiring its inclusion would have reduced the number of included studies.''[34,p15]

We consulted two sources to assemble RRs for diseases attributable to tobacco smoking. A 2008 paper by Gandini et al.[35] offers a detailed meta-analysis specific to smoking-related cancers, including RRs adjusted for known confounding factors (esophageal and upper digestive tract cancers for alcohol consumption, stomach cancer for diet, liver cancer for infection with hepatitis B or C, cervical cancer for infection with the human papillomavirus and kidney cancer for body mass index).[35] Note that tobacco smoking is no longer a significant RF for liver or cervical cancers after these adjustments. The RR of cardiovascular and respiratory diseases were taken from a publication by Thun et al.[36] Thun et al.[36] adjusted all RRs for age, race, education, marital status, employment, consumption of vegetables and fruits, aspirin use, alcohol consumption, body mass index (BMI), physical activity and consumption of fatty foods. In addition, the RR for pneumonia, influenza, bronchitis and emphysema were adjusted for occupational asbestos exposure.

Most sources, with the exception of those dealing with physical inactivity, offered RR data by sex. An additional review of research for sex variations associated with physical inactivity supported the assumption that there is no significant difference in RR between males and females for this RF.[37,38,39]

The point estimates of the RRs are used for calculations in the base model with the upper and lower bounds of the 95% confidence intervals (CIs) assessed in a sensitivity analysis.

Risk factor exposure

The other half of a PAF calculation depends on high-quality RF prevalence data.[40] The analysis of Manitoba's population exposure to tobacco smoking, physical inactivity and overweight/obesity began with information drawn from the 2008 Canadian Community Health Survey (CCHS). Tobacco smoking included all ''current smokers'' (daily and occasional smokers); overweight and obesity included individuals with a calculated BMI of between 25 kg/m^2 and 30 kg/m^2 for overweight and of 30 kg/m^2 and greater for obesity (based on self-reported height and weight); and physical inactivity included individuals categorized in the CCHS as ''inactive.''

We made several adjustments to the base CCHS data to address acknowledged

weaknesses. First, we used data from the Manitoba Youth Health Survey (MYHS) to adjust for youth smoking and physical inactivity.[41] Data from the CCHS suggested that about 10% of Manitoba youth aged 12 to 19 years were current smokers in 2008 versus 21.2% of youth in Grades 6 to 12 in the MYHS. On the other hand, the prevalence of physically inactive youth was reduced from 32% (in CCHS) to 19.3% (in MYHS).

Second, we estimated rates of physical inactivity for children aged under 12 years based on rates in the MYHS (16.4% for males and 22.1% in females). Rates of overweight and obesity for children and youth aged under 18 years were estimated based on Manitoba-specific CCHS rates for ages 20 to 34 years (34.5%/36.6% overweight in males/females and 15.6%/14.7% obesity in males/females).[42] While CCHS provides an estimate of overweight and obesity combined for ages 12 to 19 years, the results have a high coefficient of variation and are to be used with caution.[42] Furthermore, obesity-related behaviours including physical (in)activity and diet tend to track from childhood into adulthood.[43]

Third, the CCHS does not include individuals living on First Nation reserves, which represents about 55 000 Manitobans.[44] We used results from the 2002/03 *Manitoba First Nations Regional Health Survey* to identify and then adjust for the high prevalence of smoking (62%) and overweight/obesity (75%) among adults aged 18 years and over in the on-reserve population.[45]

A final adjustment was guided by the work of Anis et al.,[18] who used the prevalence of waist circumference rather than BMI for specific disease categories including ischemic heart disease, hypertension, type 2 diabetes and gallbladder disease.

Multiple exposure levels

The most basic version of a PAF calculation, derived from the prevalence of a single RF and the RR of a related disease, uses the formula (E(RR−1)) / (E(RR−1)+ 1), where E is the proportion of the population exposed to the factor of interest and RR is the relative risk of disease developing in the group exposed to the factor.

However, more sophisticated approaches are required to calculate PAF when a polytomous RF is involved, that is, one that is made up of many parts. This is the case for overweight and obesity. These two biological categories lie on a continuum. As such, it is *not* algebraically accurate to calculate basic PAFs for each of overweight and obesity, and then simply sum the two figures to derive an overall PAF for exposure to excess weight. Instead, overweight and obesity should be conceived as a trichotomous exposure to excess body weight; that is, three categories of exposure are involved: (1) no excess weight, (2) intermediate excess, or overweight (prevalence E_{OW}), (3) more extreme excess, or obesity (prevalence E_{OB}). The PAF calculation is as follows:[46]

$$E_{OW}(RR_{OW}-1)+E_{OB}(RR_{OB}-1)/$$
$$E_{OW}(RR_{OW}-1)+E_{OB}(RR_{OB}-1)+1$$

Multiple risk factors

When complete information is known about both the exposure to multiple RFs (i.e. smoking and overweight/obesity in the same individual) and about the RR related to each set of causes, then it is straightforward to calculate the PAF for a combined system. However, when information on the RF overlap is lacking, as is often the case, it is once again important to avoid simply adding the basic PAFs for each RF in order to obtain a combined PAF for the multifactorial system. A more accurate approximation of PAF of the system is obtained using the equation[47]

$$1-[(1-PAF_1)(1-PAF_2)(1-PAF_3)]$$

where the notation PAF_1 stands for the PAF related to the first RF, and so on.

This equation is most accurate when two conditions apply: (1) the RFs involved are statistically independent (i.e. experiencing one makes an individual no more or less likely to experience the other, or the clustering of RFs is limited), and (2) their joint effects are multiplicative (i.e. synergistic). These two conditions can be shown to apply very well to a system involving obesity and smoking,[48,49] and reasonably well to obesity and physical inactivity.[50,51] Equivalent investigations of smoking combined with inactivity are scarce.

This adjustment equation can be extended to additional RFs. It can also be applied to aspects of disease development beyond basic incidence, including rates of mortality, disability, etc. In this analysis, we used the adjustment equation to generate a more accurate PAF of the direct costs of disease.

Direct costs

We estimated the economic burden (direct and indirect costs) associated with the RFs in Manitoba using a prevalence-based cost-of-illness approach[52] and reported this in 2008 Canadian dollars.

We began calculating direct costs using the approach adopted by Anis et al.[18] In short, direct costs including hospital care, physician services, other health care professionals (but excluding dental services), drugs, health research, and ''other'' health care expenditures were extracted from the National Health Expenditure Database for Manitoba.[53] All costs, with the exception of hospital care, were allocated to each of the comorbidity categories based on weights published in the *Economic Burden of Illness in Canada (EBIC)* for 1998.[54] Hospital costs were allocated to each comorbidity based on the proportion of total patient bed-days (based on data from the Canadian Institute for Health Information Hospital Morbidity Database 2000/2001[55]) used in treating patients in Manitoba with that comorbidity. Estimated total direct costs were distributed between males and females based on the proportion of hospital bed-days in 2000/2001 utilized by males and females for each of the comorbidities. Finally, the Manitoba sex-specific costs by comorbidity were multiplied by the calculated sex- and comorbidity-specific PAF.

Adjusting direct costs in a multifactorial system

We then applied the formula introduced earlier for calculating the combined PAF in

a multifactorial system to the calculated crude direct costs attributable to each of tobacco smoking, overweight/obesity and physical inactivity. Crude direct costs for each RF were inserted into the adjustment formula (i.e. PAF_1 = crude PAF of cost for tobacco smoking, etc.) in order to generate an adjusted PAF of direct costs for the multifactorial system. This approach reduced combined direct costs by 12.6% (from $560.8 to $490.3 million per year).

Having determined as accurately as possible the combined population impact of multiple RFs, it is still useful for the purposes of high-level prevention prioritization, public educational messages, etc., to have a sense of the approximate impact of a particular RF. Thus we applied a disaggregation step at the end of the direct costing process to notionally assign an economic burden to each RF. We did this by returning to the crude costs for each RF, dividing each of these figures by their sum (i.e. the crude total cost for the combined system) and thereby generating a ratio. This ratio was then applied to the adjusted total direct costs, yielding a disaggregated, adjusted economic burden by disease that is notionally attributable to each RF.

Indirect costs

We calculated indirect costs (premature mortality, short- and long-term disability) following the method used in *EBIC, 1998* (a modified human-capital approach).[54]

Specifically, the steps involved in estimating indirect costs were as follows:

1. Six diagnostic categories within *EBIC, 1998* were identified that cover the comorbidities/diseases of interest; the direct and indirect costs for these six categories were extracted.
2. This information was used to determine a ratio between direct and indirect costs for each of the diagnostic categories, stratified by the specific category of indirect cost. For example, the indirect costs associated with cancer are 4.6 times (459%) higher than direct costs, largely driven by premature mortality. On the other hand, indirect costs associated with musculoskeletal diseases are 5.2 times (519%) higher than direct costs; in this instance, however, the majority of the higher costs are associated with long-term disability rather than premature death (see Table 1).
3. The pertinent ratios (by diagnostic category and specific indirect cost category) were then applied to the previously identified direct costs attributable to each RF and adjusted for a multifactorial system in order to generate the equivalent indirect cost data.

A detailed description of the steps taken in this analysis, with examples, is available on request.

Results

Table 2 shows the fully adjusted prevalence of RF exposure, the statistically significant RR data by sex and the calculated PAF of disease incidence related to each RF. The PAF for all comorbidities, with the obvious exception of gynecological and breast cancers, vary by sex. For example, 38.8% of type 2 diabetes in Manitoba is attributable to obesity in males versus 48.2% in females. This is despite the higher prevalence of obesity in Manitoba males (19.8%) than in females (18.7%). The higher overall PAF in females is due to a much higher RR (12.41) than in males (6.74) for type 2 diabetes. This type of detailed analysis has important implications in determining direct and indirect costs.

Table 3 includes a summary of the adjusted estimates of the prevalence of the chronic disease RFs, the absolute numbers of Manitobans with each RF, and the fully adjusted results from the economic burden analysis. The total direct costs in Manitoba in 2008 attributable to the health effects of smoking, physical inactivity and excess weight are estimated at $490.3 million, while the indirect costs are estimated at $1113.8 million, yielding a total annual economic burden of $1604.2 million.

This aggregate RF burden is somewhat higher for females ($824.9 million) than males ($779.3 million). The costs associated with smoking are higher in males than females ($319.5 million versus $237.9 million); whereas the economic burden associated with excess weight ($417.7 million versus $329.5 million in males) and physical inactivity ($169.3 million versus $130.2 million in males) is higher in females.

Figure 1 represents the RF-specific burden graphically, with additional information on the components that constitute the indirect costs of disease. The indirect burden related to premature mortality dominates as an outcome of tobacco smoking ($241.8 million, or 64.4% of $375.4 million in total indirect costs for that RF), and it is also marginally higher than disability in the case of physical inactivity. The reverse is true for overweight/obesity, where the economic burden of short- and long-disability related to disease ($311.5 million) outstrips the costs of premature mortality ($218.6 million).

This analysis indicates that the notionally disaggregated economic burden for excess weight is larger than the economic burden related to smoking. Thus, the economic burden for the combination of overweight and obesity in Manitoba was $283.7 plus

TABLE 1
Economic burden of illness in Canada by diagnostic category

Diagnostic category	Indirect costs as percentage of direct costs, Canada, 1998			
	Mortality, %	Long-term disability, %	Short-term disability, %	Total indirect cost, %
Cancer	415	38	7	459
Cardiovascular diseases	121	46	4	171
Respiratory diseases	48	28	70	146
Endocrine and related diseases	64	51	3	119
Digestive diseases	32	14	20	65
Musculoskeletal diseases	5	476	38	519

TABLE 2
Relative risk, prevalence of risk factors, and population attributable fraction in Manitoba, 2008

		Smoking				*Physical inactivity*				*Overweight*				*Obesity*			
		Male		**Female**		**Male**		**Female**		**Male**		**Female**		**Male**		**Female**	
Prevalence of risk factor in Manitoba in 2008		***25.1%***		***20.6%***		***38.8%***		***42.3%***		***39.3%***		***30.2%***		***19.8%***		***18.7%***	
		All ages				***All ages***				***All ages***				***All ages***			
	ICD-9 code	**RR**	**PAF, %**	**RR**	**PAF, %**	**RR**	**PAF, %**	**RR**	**PAF, %**	**RR**	**PAF, %**	**RR**	**PAF, %**	**RR**	**PAF, %**	**RR**	**PAF, %**
Neoplasms																	
Trachea, bronchus, lung	162	8.96	66.7	8.96	62.1												
Larynx	161	6.98	60.0	6.98	55.2												
Lip, oral cavity, pharynx	140–149	4.03	43.2	4.03	38.4												
Esophagus	150	3.00	33.4	3.00	29.1					1.13	4.9						
Urinary bladder	188	2.77	30.8	2.77	26.7												
Kidney, other urinary	189	1.69	14.8	1.69	12.4					1.40	11.9	1.82	16.4	1.82	12.3	2.64	19.3
Pancreas	157	1.70	15.0	1.70	12.6									2.29	20.3	1.60	10.1
Stomach	151	2.22	23.5	2.22	20.1												
Endometrial cancer	179, 181, 182											1.53	11.7			3.22	24.8
Ovarian cancer	183											1.18	4.9			1.28	4.7
Breast cancer	174, 175							1.31	11.6								
Postmenopausal breast cancer	174, 175											1.08	2.3			1.13	2.3
Colorectal cancer	153, 154					1.41	13.7	1.41	14.8	1.51	14.4	1.45	10.7	1.95	13.6	1.66	9.9
Cardiovascular diseases																	
Pulmonary embolism	415.1									1.91	20.4	1.91	17.2	3.51	25.7	3.51	25.5
Congestive heart failure	428													1.79	13.5	1.78	12.7
Ischemic heart disease	410–414					1.45	14.9	1.45	16.0	1.29	5.2	1.80	10.8	1.72	10.4	3.10	24.1
Aged 35–64 y		2.60	28.7	3.20	31.1												
Aged ≥ 65 y		1.50	11.2	1.60	12.6												

Continued on the following page

TABLE 2 (continued)
Relative risk, prevalence of risk factors, and population attributable fraction in Manitoba, 2008

Prevalence of risk factor in Manitoba in 2008		*Smoking*				*Physical inactivity*				*Overweight*				*Obesity*			
		Male		Female		Male		Female		Male		Female		Male		Female	
		25.1%		*20.6%*		*38.8%*		*42.3%*		*39.3%*		*30.2%*		*19.8%*		*18.7%*	
		All ages				*All ages*				*All ages*				*All ages*			
	ICD-9 code	RR	PAF, %	RR	PAF, %	RR	PAF, %	RR	PAF, %	RR	PAF, %	RR	PAF, %	RR	PAF, %	RR	PAF, %
Other heart disease	390–398, 415–417	1.80	16.7	1.50	12.6												
Stroke / cerebrovascular disease	430–438					1.60	18.9	1.60	20.3	1.23	7.6	1.15	4.1	1.51	8.4	1.49	7.9
Aged 35–64 y		2.40	26.0	3.80	36.5												
Aged ≥ 65 y		1.50	11.2	1.60	11.0												
Atherosclerosis	440	3.90	42.1	3.80	36.5												
Aortic aneurysm	441	3.90	42.1	3.80	36.5												
Other arterial disease	442–448	3.90	42.1	3.80	36.5												
Hypertension	401–405					1.30	10.4	1.30	11.3	1.28	5.1	1.65	9.3	1.84	11.8	2.42	18.5
Respiratory diseases																	
Asthma	493									1.20	6.8	1.25	6.5	1.43	7.3	1.78	11.7
Bronchitis, emphysema	490–492	10.80	71.1	9.90	69.9												
Chronic airway obstruction	496	10.80	71.1	9.90	69.9												
Pneumonia, influenza	480–487	1.90	18.4	1.70	19.8												
Other																	
Type 2 diabetes	250.x0, 250.x2					1.50	16.2	1.50	17.5	2.40	17.4	3.92	24.5	6.74	38.8	12.41	48.2
Gallbladder disease	574, 575											1.44	6.7	1.43	7.0	2.32	17.9
Osteoarthritis	715					1.59	18.6	1.59	20.0	2.76	29.2	1.80	16.6	4.20	27.7	1.96	13.0
Chronic back pain	720–724									1.59	15.5	1.59	12.8	2.81	21.7	2.81	21.3

Abbreviations: ICD, *International Classification of Disease*; PAF, population attributable fraction; RR, relative risk; y, years.

Note: Blank cells indicate that there is no significant relationship between the risk factor and the disease.

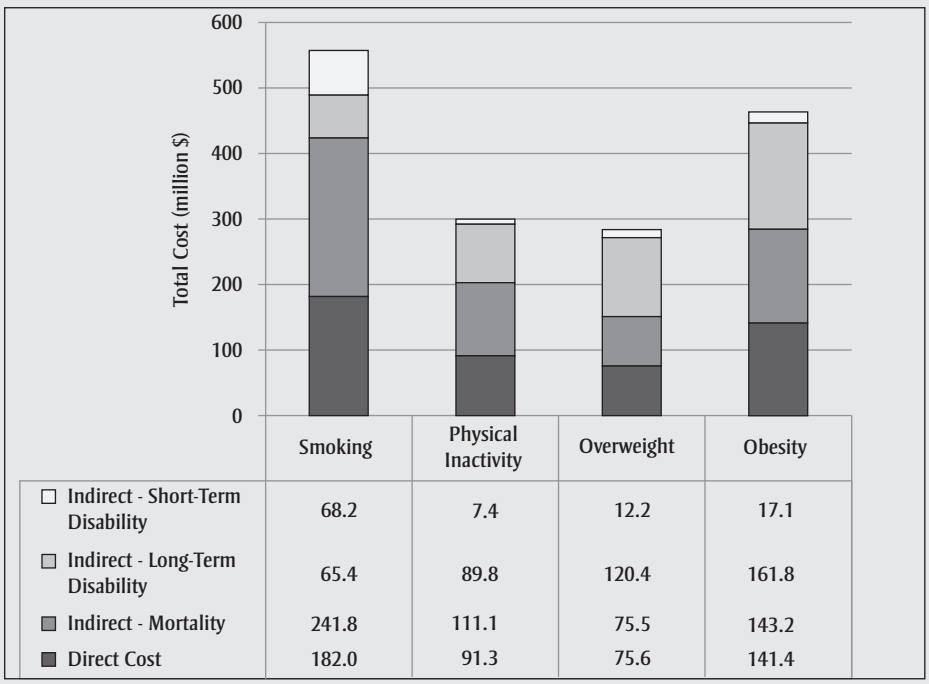
TABLE 3
Estimated prevalence of risk factors, total economic burden for multifactorial system and disaggregated costs by risk factor, Manitoba, 2008, by sex[a]

	Percentage of population with RF, %	Number of individuals with RF	Direct cost per individual with RF, $	Indirect cost per individual with RF, $	Total cost per individual with RF, $	Total direct cost of RF, million $	Total indirect cost of RF, million $	Total cost of RF, million $
Males								
Smokers	25.1	148 460	690.3	1461.9	2152.2	102.5	217.0	319.5
Inactive	38.8	229 124	180.2	388.2	568.4	41.3	88.9	130.2
Overweight	39.3	232 251	141.6	418.1	559.7	32.9	97.1	130.0
Obesity	19.8	116 970	498.6	1207.1	1705.8	58.3	141.2	199.5
Subtotal						**235.0**	**544.3**	**779.3**
Females								
Smokers	20.6	125 013	636.1	1266.5	1902.7	79.5	158.3	237.9
Inactive	42.3	257 429	194.2	463.6	657.7	50.0	119.3	169.3
Overweight	30.2	183 858	232.4	603.8	836.2	42.7	111.0	153.7
Obesity	18.7	113 786	730.2	1589.5	2319.8	83.1	180.9	264.0
Subtotal						**255.3**	**569.6**	**824.9**
Both sexes								
Smokers	22.8	273 473	665.5	1372.6	2038.1	182.0	375.4	557.4
Inactive	40.6	486 553	187.6	428.1	615.7	91.3	208.3	299.6
Overweight	34.7	416 109	181.7	500.2	681.9	75.6	208.1	283.7
Obesity	19.2	230 757	612.8	1395.7	2008.5	141.4	322.1	463.5
Total						**490.3**	**1113.8**	**1604.2**

Abbreviations: CCHS, Canadian Community Health Survey; RF, risk factor.

[a] Adjusted for selected CCHS data limitations and multiple risk factors in one individual.

FIGURE 1
Estimated Direct and Indirect Economic Burden of Smoking, Physical Inactivity and Overweight/Obesity, Manitoba, 2008[a]



[a]Adjusted for selected CCHS data limitations and multiple risk factors in one individual.

$463.5 million (or $747.2 million) in 2008, exceeding the economic burden associated with tobacco smoking (at $557.4 million) by 34%.

Sensitivity analysis

The point estimates for RR are used in the base model results presented above. Some degree of uncertainty is attached to these point estimates as reflected by the 95% CI. To assess the effect of this uncertainty on the results, we used the lower and upper bounds of the 95% CI for the RR associated with each RF and disease in a sensitivity analysis. Using the lower bounds resulted in a decrease in the total economic burden from $1,604.2 million to $1,251.5 million (or −22.0%) while applying the upper bounds increased the total economic burden to $1,927.7 million (or +20.2%) (see Table 4).

Discussion

The analytic approach outlined in this document begins to address the issue of double-counting costs when estimating the aggregate economic burden of chronic illnesses associated with multiple RFs in one individual. Applied to the province of Manitoba, the approach suggests a reduction of 12.6% in the aggregate economic burden over the total that would be generated by crude summation of costs generated by each of the key RFs.

This analysis used an extension of the basic PAF formula to produce a more accurate result, including addressing both complications in assessing PAF when a polytomous RF is involved (i.e. overweight and obesity) and accounting for the possibility of multiple RFs in any given individual.

The analysis of the economic burden related to the RF system and (notionally) the individual RFs of smoking, physical inactivity and overweight/obesity is the first phase of any attempt to project the potential economic impact of applying known primary prevention initiatives.

Using the methods outlined in this paper, we estimated the total annual economic burden of the RFs in Manitoba in 2008 to be $1.6 billion ($490 million in direct costs and $1,114 million in indirect costs).

Another important result, generated by having access to sex-specific RF prevalence and RR data, was the difference between males and females in contributing to the total economic burden. The costs associated with tobacco smoking are higher in males, which is partly a reflection of the continuing higher prevalence of tobacco smoking among men. On the other hand, the economic burden associated with excess weight is higher in females, a result that appears to be anomalous since the prevalence of obesity and (especially) overweight is in fact higher in males. In addition to the burden in women that is specific to gynecological cancers, an explanation for the anomaly leans on the fact that the RR related to excess weight is higher in females for several costly conditions, including renal cancer, ischemic heart disease, hypertension and type 2 diabetes (see Table 2). The resulting overall sex-specific distribution for the burden of key modifiable RFs has important implications for prevention planning and public health messaging.

The current analysis also confirmed the emergence of overweight/obesity as a public health concern, a phenomenon that has also been noted in other jurisdictions.[56,57] In fact, the estimated 2008 economic burden associated with excess weight in Manitoba ($747.2 million) is greater than that associated with tobacco use ($557.4 million). Even though the economic burden associated with smoking still exceeds that of obesity strictly defined, once the health effects of overweight are included, the area as a whole moves into the forefront. The United Kingdom project introduced earlier in this paper found similar results with direct costs due to overweight/obesity exceeding the total related to tobacco smoking (UK £5 billion vs. UK £3.3 billion) by a differential similar in proportion to that found in the current analysis for Manitoba.[26] However, the point at which overweight is associated with a significant increase in health effects is likely higher than a BMI of 25 kg/m^2 in the North

TABLE 4
Estimated total economic burden for multifactorial system and disaggregated costs by risk factor, Manitoba, 2008, by sex: sensitivity analysis

	Sensitivity analysis				
	Best estimate of RR	Low estimate of RR	Variance	High estimate of RR	Variance
Males					
Smokers	319.5	266.3	−16.7	363.0	13.6
Inactive	130.2	102.4	−21.4	157.2	20.7
Overweight	130.0	95.2	−26.8	159.0	22.3
Obesity	199.5	147.5	−26.1	248.3	24.5
Subtotal	**779.3**	**611.4**	**−21.5**	**927.5**	**19.0**
Females					
Smokers	237.9	203.3	−14.5	272.3	14.5
Inactive	169.3	129.7	−23.4	206.3	21.9
Overweight	153.7	110.8	−27.9	192.3	25.1
Obesity	264.0	196.3	−25.6	329.3	24.7
Subtotal	**824.9**	**640.1**	**−22.4**	**1000.2**	**21.3**
Both sexes					
Smokers	557.4	469.6	−15.8	635.3	14.0
Inactive	299.6	232.1	−22.5	363.5	21.4
Overweight	283.7	206.0	−27.4	351.3	23.8
Obesity	463.5	343.8	−25.8	577.6	24.6
Total	**1604.2**	**1251.5**	**−22.0**	**1927.7**	**20.2**

Abbreviations: RF, risk factor; RR, relative risk.

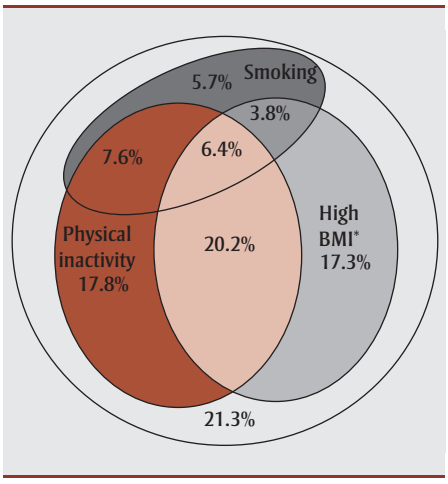
American population though it may also be lower in certain ethnic groups.[58]

The quality of the results derived from a PAF analysis is inevitably limited by the quality of the inputs, specifically RR and prevalence data. The effect of any potential inaccuracies in this project was first mitigated by correcting known gaps in the RF exposure information obtained through routine Canadian population surveys. Variation in regional PAF estimates often reflects uncertainty in the degree of exposure to the RF being analyzed.[33] Thus, it is vital to refine prevalence information as much as possible.

A consistent dependence on meta-analyses, which were adjusted for known confounding factors whenever possible, was used in estimating RRs. A sensitivity analysis using the 95% CI associated with each RR indicates the importance of using robust and accurate RR estimates.

Does a 12.6% adjustment (reduction) for overlapping RFs in certain individuals have face validity? Figure 2 summarizes the degree of potentially confounding RF overlaps in Canadians, based on CCHS data from 2000.[59] Summing across the pertinent sub-categories, 10.2% of the population is exposed both to smoking and overweight/obesity, 26.6% to overweight/obesity and physical inactivity and 14.0% to physical inactivity and smoking. While the overlap related to elevated BMI and physical inactivity is relatively high, the required correction (to avoid double-counting disease incidence) was, in fact, used here for RR data for physical inactivity adjusted for overweight/obesity.[16] When compared to the proportions of the population with multiple RF exposures, the 12.6% adjustment to the Manitoba economic burden appears to have face validity.

FIGURE 2
Overlap of risk factor exposure in Canada, Canadian Community Health Survey, Cycle 1.1 (2000)



Source: Klein-Geltink et al., *Chronic Diseases in Canada*, 2006.[59]

Abbreviation: BMI, body mass index.

*BMI $\geq$ 25 kg/m^2.

Despite the attempts to optimize the accuracy of the estimated economic burden, some limitations remain, partly related to the assumptions required to creatively integrate several data sources compiled at different points of time. For instance, a key assumption of using older CIHI and EBIC data was acknowledged by Anis et al.,[18] namely that ''the distribution of costs for each cost category did not change significantly from 1998 to 2006.''[18,p34] Similarly, the method of scaling up from direct costs to indirect costs depends on the assumption that the ratios of costs between different comorbidities are the same for direct and indirect costs. Furthermore, the RRs for tobacco smoking are based on a comparison of current versus never-smokers and do not take into account smoking intensity. Potential changes (reductions) over time in smoking intensity would modify the RRs.

Health care planners in many jurisdictions in the world share an interest in having a reasonable estimation of the economic burden of disease generated by modifiable RFs. Such information is vital to prioritizing and gaining support for primary prevention programs. Indeed, understanding the baseline economic burden associated with specific RFs is a prerequisite for developing a persuasive business case for prevention. The current findings, for example, have been a catalyst for action in Manitoba, supporting development of a Primary Prevention Syndicate, a risk factor reduction challenge to provincial politicians, and creation of Heart and Stroke Foundation Challenge Grants and a Research Chair in primary prevention.

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References

1. Omran AR. The epidemiologic transition. A theory of the epidemiology of population change. Milbank Mem Fund Q. 1971;49(4): 509-38.

2. Gaziano JM. Fifth phase of the epidemiologic transition: the age of obesity and inactivity. JAMA. 2010;303(3):275-6.

3. Klein S, Burke LE, Bray GA, Garbagnati F, Cappuccio FP, Scalfi L. Clinical implications of obesity with specific focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. Circulation. 2004;110(18):2952-67.

4. Strazzullo P, D'Elia L, Cairella G, Garbagnati F, Cappuccio FP, Scalfi L. Excess body weight and incidence of stroke: meta-analysis of prospective studies with 2 million participants. Stroke. 2010;41(5):e418-26.

5. Abdullah A, Peeters A, de Courten M, Stoelwinder J. The magnitude of association between overweight and obesity and the risk of diabetes: a meta-analysis of prospective cohort studies. Diabetes Res Clin Pract. 2010;89(3):309-19.

6. Wang Y, Chen X, Song Y, Caballero B, Cheskin LJ. Association between obesity and kidney disease: a systematic review and meta-analysis. Kidney Int. 2008;73(1):19-33.

7. Jiang L, Rong J, Wang Y, et al. The relationship between body mass index and hip osteoarthritis: a systematic review and meta-analysis. Joint Bone Spine. 2011;78(2): 150-5.

8. Yang P, Zhou Y, Chen B, et al. Overweight, obesity and gastric cancer risk: results from a meta-analysis of cohort studies. Eur J Cancer. 2009;45(16):2867-73.

9. Larsson SC, Wolk A. Overweight, obesity and risk of liver cancer: a meta-analysis of cohort studies. Br J Cancer. 2007;97(7):1005-8.

10. Moghaddam AA, Woodward M, Huxley R. Obesity and risk of colorectal cancer: a meta-analysis of 31 studies with 70,000 events. Cancer Epidemiol Biomarkers Prev. 2007;16(12):2533-47.

11. Warburton DE, Charlesworth S, Ivey A, Nettlefold L, Bredin SS. A systematic review of the evidence for Canada's Physical Activity Guidelines for Adults. Int J Behav Nutr Phys Act. 2010;7:39.

12. Friedenreich CM, Neilson HK, Lynch BM. State of the epidemiological evidence on physical activity and cancer prevention. Eur J Cancer. 2010;46(14):2593-604.

13. Glantz S, Gonzalez M. Effective tobacco control is key to rapid progress in reduction of non-communicable diseases. Lancet. 2012;379(9822):1269-71.

14. Withrow D, Alter DA. The economic burden of obesity worldwide: a systematic review of the direct costs of obesity. Obes Rev. 2011;12(2):131-41.

15. Katzmarzyk PT, Gledhill N, Shephard RJ. The economic burden of physical inactivity in Canada. CMAJ. 2000;163(11):1435-40.

16. Katzmarzyk PT, Janssen I. The economic costs associated with physical inactivity and obesity in Canada: an update. Can J Appl Physiol. 2004;29(1):90-115.

17. Birmingham CL, Muller JL, Palepu A, Spinelli JJ, Anis AH. The cost of obesity in Canada. CMAJ. 1999;160(4):483-8.

18. Anis AH, Zhang W, Bansback N, Guh DP, Amarsi Z, Birmingham CL. Obesity and overweight in Canada: an updated cost-of-illness study. Obes Rev. 2010;11(1):31-40.

19. Kaiserman MJ. The cost of smoking in Canada, 1991. Chronic Dis Can. 1997; 18(1):13-9.

20. Moffatt E, Shack LG, Petz GJ, Sauvé JK, Hayward K, Colman R. The cost of obesity and overweight in 2005: a case study of Alberta, Canada. Can J Public Health. 2011;102(2):144-8.

21. Janssen I, Lam M, Katzmarzyk PT. Influence of overweight and obesity on physician costs in adolescents and adults in Ontario, Canada. Obes Rev. 2009;10(1):51-7.

22. Fine LJ, Philogene GS, Gramling R, Coups EJ, Sinha S. Prevalence of multiple chronic disease risk factors. 2001 National Health Interview Survey. Am J Prev Med. 2004;27(2 Suppl):18-24.

23. Ezzati M, Hoorn SV, Rodgers A, et al. Estimates of global and regional potential health gains from reducing multiple major risk factors. Lancet. 2003;362(9380):271-80.

24. World Cancer Research Fund. Policy and action for cancer prevention - Food, nutrition, and physical activity: a global perspective - appendices [Internet]. Washington (DC): 2009 [cited 2012 May 15]. Available from: http://www.dietandcancerreport.org/cancer_resource_center/downloads/chapters/pr/Appendix%20A%20and%20B.pdf

25. Allender S, Foster C, Scarborough P, Rayner M. The burden of physical activity-related ill health in the UK. J Epidemiol Community Health. 2007;61(4):344-8.

26. Allender S, Rayner M. The burden of overweight and obesity-related ill health in the UK. Obes Rev. 2007;8(5):467-73.

27. Allender S, Balakrishnan R, Scarborough P, Webster P, Rayner M. The burden of smoking-related ill health in the UK. Tob Control. 2009;18(4):262-7.

28. Rayner M, Scarborough P. The burden of food related ill health in the UK. J Epidemiol Community Health. 2005;59(12): 1054-7.

29. Balakrishnan R, Allender S, Scarborough P, Webster P, Rayner M. The burden of alcohol-related ill health in the United Kingdom. J Public Health (Oxf). 2009; 31(3):366-73.

30. Scarborough P, Bhatnagar P, Wickramasinghe KK, Allender S, Foster C, Rayner M. The economic burden of ill health due to diet, physical inactivity, smoking, alcohol and obesity in the UK: an update to 2006-07 NHS costs. J Public Health (Oxf). 2011; 33(4):527-35.

31. Statistics Canada. Focus on geography series, 2011 Census: Province of Manitoba [Internet]. Ottawa (ON): Statistics Canada; 2012 [cited 2012 May 15]. Available from: http://www12.statcan.gc.ca/census-recensement/2011/as-sa/fogs-spg/Facts-pr-eng.cfm?Lang=Eng&GK=PR&GC=46

32. Aboriginal Affairs and Northern Development Canada. First Nations in Manitoba [Internet]. Ottawa (ON): Aboriginal Affairs and Northern Development Canada; 2012 [cited 2012 May 15]. Available from: http://www.aadnc-aandc.gc.ca/eng/1100100020400

33. Walter SD. Calculation of attributable risks from epidemiological data. Int J Epidemiol. 1978;7(2):175-82.

34. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health. 2009;9:88.

35. Gandini S, Botteri E, Iodice S, et al. Tobacco smoking and cancer: a meta-analysis. Int J Cancer. 2008;122(1):155-64.

36. Thun MJ, Apicella LF, Henley SJ. Smoking vs other risk factors as the cause of smoking-attributable deaths: confounding in the courtroom. JAMA. 2000;284(6):706-12.

37. Wendel-Vos GC, Schuit AJ, Feskens EJ, et al. Physical activity and stroke. A meta-analysis of observational data. Int J Epidemiol. 2004;33(4):787-98.

38. Wolin KY, Yan Y, Colditz GA, Lee IM. Physical activity and colon cancer prevention: a meta-analysis. Br J Cancer. 2009; 100(4):611-6.

39. Harriss DJ, Atkinson G, George K, et al. Lifestyle factors and colorectal cancer risk (1): systematic review and meta-analysis of associations with body mass index. Colorectal Dis. 2009;11(6):547-63.

40. Walter SD. Local estimates of population attributable risk. J Clin Epidemiol. 2010; 63(1):85-93.

41. Health in common. Youth health survey report 2009: students in Manitoba (grades 9-12) [Internet]. Winnipeg (MB): Partners in Planning for Healthy Living; 2009 [cited 2012 May 15]. Available from: http://www.healthincommon.ca/wp-content/uploads/Youth-Health-Survey-Report-2009.pdf

42. Statistics Canada. CANSIM Table 105-0501: Health indicator profile, annual estimates, by age group and sex, Canada, provinces, territories, health regions (2011 boundaries) and peer groups [Internet]. Ottawa (ON): Statistics Canada; [cited 2012 May 15]. Available from: http://www5.statcan.gc.ca/cansim/a05?lang = eng&id = 1050501

43. Craigie AM, Lake AA, Kelly SA, Adamson AJ, Mathers JC. Tracking of obesity-related behaviours from childhood to adulthood: a systematic review. Maturitas. 2011;70(3): 266-84.

44. Statistics Canada. Aboriginal peoples in Canada in 2006: Inuit, Métis and First Nations, 2006 Census: findings [Internet]. Ottawa (ON): Statistics Canada; 2006 [cited 2012 May 15]. Available from: http://www12.statcan.ca/census-recensement/2006/as-sa/97-558/index-eng.cfm

45. Regional health survey, 2002/03 [Internet]. Winnipeg (MB): Manitoba First Nations; 2005 [cited 2012 May 15]. Available from: http://www.manitobachiefs.com

46. Hanley JA. A heuristic approach to the formulas for population attributable fraction. J Epidemiol Community Health. 2001; 55(7):508-14.

47. Steenland K, Armstrong B. An overview of methods for calculating the burden of disease due to specific risk factors. Epidemiology. 2006;17(5):512-9.

48. Healton CG, Vallone D, McCausland KL, Xiao H, Green MP. Smoking, obesity, and their co-occurrence in the United States: cross sectional analysis. Br Med J. 2006; 333(7557):25-6

49. Freedman DM, Sigurdson AJ, Rajaraman P, Doody MM, Linet MS, Ron E. The mortality risk of smoking and obesity combined. Am J Prev Med. 2006;31(5):355-62.

50. Katzmarzyk PT, Janssen I, Ardern CI. Physical inactivity, excess adiposity and premature mortality. Obes Rev. 2003; 4(4): 257-90.

51. Lee DC, Sui X, Blair SN. Does physical activity ameliorate the health hazards of obesity? Br J Sports Med. 2009; 43(1): 49-51.

52. Tarricone R. Cost-of-illness analysis: what room in health economics? Health Policy. 2006;77:51-63.

53. National health expenditure trends, 1975 to 2011 [Internet]. Ottawa (ON): CIHI; 2011 Nov [cited 2012 Jun 1]. Available from: http://secure.cihi.ca/ free_products/nhex_trends_report_2011_en.pdf

54. Policy Research Division, Strategic Policy Directorate, Population and Public Health Branch, Health Canada. Economic burden of illness in Canada (EBIC), 1998 [Internet]. Ottawa (ON): Health Canada; 2002 [cited 2012 May 15]. Available from: http://www.phac-aspc.gc.ca/publicat/ebic-femc98/pdf/ebic1998.pdf

55. Hospital morbidity database, 2000/01 Tabular Results [Internet]. Ottawa (ON): CIHI; 2002 [cited 2012 Jun 1]. Available from: https://secure.cihi.ca/free_products/HospitalMorbidityTabularReports2000-2001.pdf

56. Jia H, Lubetkin EI. Trends in quality-adjusted life-years lost contributed by smoking and obesity. Am J Prev Med. 2010;38(2):138-44.

57. Hoad V, Somerford P, Katzenellenbogen J. High body mass index overtakes tobacco as the leading independent risk factor contributing to disease burden in Western Australia. Aust NZ J of Publ Heal. 2010;34(2):214-5.

58. Krueger H, Williams D, Kaminsky B, McLean D. The health impact of smoking and obesity and what to do about it. Toronto (ON): University of Toronto Press; 2007.

59. Klein-Geltink JE, Choi BC, Fry RN. Multiple exposures to smoking, alcohol, physical inactivity and overweight: prevalences according to the Canadian Community Health Survey Cycle 1.1. Chronic Dis Can. 2006;27(1):25-33

Estimating cancer risk in relation to tritium exposure from routine operation of a nuclear-generating station in Pickering, Ontario

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Abstract

Introduction: Evidence suggests that current levels of tritium emissions from CANDU reactors in Canada are not related to adverse health effects. However, these studies lack tritium-specific dose data and have small numbers of cases. The purpose of our study was to determine whether tritium emitted from a nuclear-generating station during routine operation is associated with risk of cancer in Pickering, Ontario.

Methods: A retrospective cohort was formed through linkage of Pickering and north Oshawa residents (1985) to incident cancer cases (1985–2005). We examined all sites combined, leukemia, lung, thyroid and childhood cancers (6–19 years) for males and females as well as female breast cancer. Tritium estimates were based on an atmospheric dispersion model, incorporating characteristics of annual tritium emissions and meteorology. Tritium concentration estimates were assigned to each cohort member based on exact location of residence. Person-years analysis was used to determine whether observed cancer cases were higher than expected. Cox proportional hazards regression was used to determine whether tritium was associated with radiation-sensitive cancers in Pickering.

Results: Person-years analysis showed female childhood cancer cases to be significantly higher than expected (standardized incidence ratio [SIR] = 1.99, 95% confidence interval [CI]: 1.08–3.38). The issue of multiple comparisons is the most likely explanation for this finding. Cox models revealed that female lung cancer was significantly higher in Pickering versus north Oshawa (HR = 2.34, 95% CI: 1.23–4.46) and that tritium was not associated with increased risk. The improved methodology used in this study adds to our understanding of cancer risks associated with low-dose tritium exposure.

Conclusion: Tritium estimates were not associated with increased risk of radiation-sensitive cancers in Pickering.

Keywords: *cancer, tritium, nuclear power plant, historical cohort study*

Introduction

According to a survey conducted in 2012 for the Canadian Nuclear Association, 55% of the Canadians surveyed think that ''dangerous'' describes nuclear energy extremely well or very well.[1] This perception may stem from studies that found elevated risks of adult cancers resulting from high levels of exposure to radiation[2] experienced by survivors of the nuclear bombs dropped on Japan in WWII or from events such as the Chernobyl nuclear disaster. On the other hand, reviews examining risk at low levels of exposure, conditions consistent with working in the Canadian nuclear industry, suggest increased risks are possible but undetectable.[3-6]

The developing fetus is particularly sensitive to radiation effects. As such, all childhood cancers and leukemia are a concern even at low levels of exposure. Several studies have been conducted on childhood leukemia near nuclear power plants (NPPs).[7-9] Most reported no increased risk. Recent case-control studies in Germany[10,11] found that the risk of childhood leukemia (age < 5 years) doubled within 5 km of German NPPs. The reasons for this increase remain unclear.[12] Studies in France,[13,14] Britain[15] and Finland[16] did not find increased risks.

The uncertainty around health effects from low-dose exposures is related to the small numbers of cases and the lack of tritium-specific dose data in these studies. This uncertainty contributes to the continued public concern in communities near NPPs.

The Pickering Nuclear Generating Station (PNGS), along with most of the city's population, is in the southern part of Pickering, Ontario, a municipality east of the city of Toronto with a population of 87 838.[17] PNGS began operating in 1971 and decommissioning is planned for 2020.

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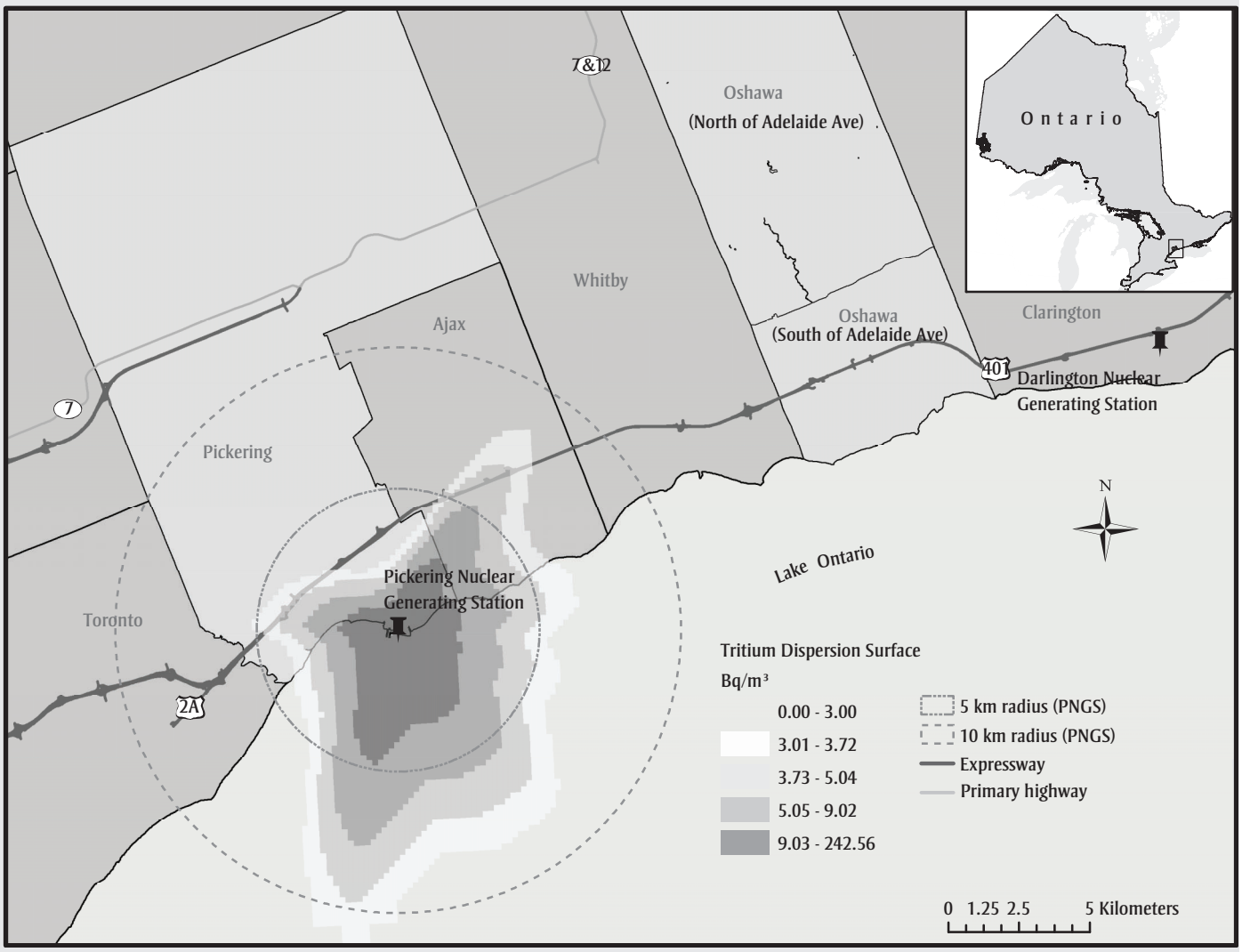
PNGS consists of two distinct stations, A and B, each with four Canadian Deuterium Uranium (CANDU) reactor units, two of which were shut down in 1997. CANDU and other heavy water reactors (HWRs) comprise a small proportion of nuclear reactors worldwide, operating in Canada and several other countries.[18] HWRs emit one or two orders of magnitude more tritium (per gigawatt of energy produced) than any other type of nuclear reactor.[19] Tritium is a by-product of routine operation, emitted mostly as tritiated water vapour (HTO), and its decay results in emission of beta radiation.[20] Tritium constitutes 99% of all radioactive emissions from PNGS.[21] PNGS provides a unique opportunity to examine cancer risks in a large urban population that may arise from low-dose radiation exposure from tritium emissions.

HTO can be inhaled, absorbed through the skin or ingested and can be incorporated into organic molecules in the body as organically bound tritium (OBT).[3] Dose estimates referred to or calculated in this study include contributions from both HTO and OBT. Estimates assume that 97.8% of tritium entering the body as HTO remains as HTO (half-life of 9.7 days) and 2.2% is converted to OBT (half-life of 48.5 days).[3] Human cells that reproduce quickly are especially sensitive to ionizing radiation.

In 2011, the total radiological dose resulting from the operation of PNGS was estimated to be 0.9 µSv for an urban resident in the Pickering and Ajax area[22] (see Figure 1). This is well below the public dose regulatory limit of 1000 µSv/year. It also represents 0.1% of the 1400 µSv naturally occurring annual radiation dose near PNGS, or 8% of the 12 µSv dose from two hours of airplane travel.[22]

The purpose of our study was to determine whether tritium emissions from routine operations at PNGS were associated with higher risk of radiation-sensitive cancers in Pickering, Ontario. Our three objectives

FIGURE 1
Study areas, PNGS tritium dispersion surface and location of nuclear power plants, Pickering, Ontario, and Oshawa, Ontario



Abbreviation: PNGS, Pickering Nuclear Generating Station.

were to: (1) evaluate the health of the cohort of Pickering residents by comparing the observed cases of cancer to the expected number of cases given cancer rates in all Ontario; (2) determine whether tritium estimates explain cancer risk among Pickering residents compared with residents of north Oshawa; and (3) determine whether tritium estimates are associated with cancer risk among Pickering residents exposed to stable tritium (''non-movers,'' resident at the same address for the previous 6 years). Our study minimized the limitations of previous studies by using tritium estimates based on actual emissions data as well as a population-based retrospective cohort with sufficient follow-up and a large sample size.

Methods

A 20-year retrospective cohort including residents of Pickering (n = 36 805) and north Oshawa (n = 43 035, comparison population) in 1985 followed forward for cancer incidence and mortality until the end of 2005. These data were analyzed in two ways: person-years analysis (objective 1) and Cox proportional hazards regression (objectives 2 and 3).

Data sources

Pickering and north Oshawa property assessment files (PAFs)

The Durham Region Planning Department provided 1979 and 1985 property assessment files (PAFs) for the cities of Pickering and Oshawa (n = 162 986). These files contained the surname, given name(s), birth year, birth month, full address and postal code of each person living in the region. These files were securely transferred to the study investigators and were stored on a secure server at Cancer Care Ontario. Analysis of the cohort excluded those residents aged 5 years or less and 85 years or more since these age groups were underrepresented in the PAF.

We tried to increase the sample size and distribution of exposures by including a large comparison population with no tritium exposure. We chose north Oshawa because we were limited to municipalities for which we had the PAF (Durham Region) and we needed a population similar to Pickering but far enough away from both PNGS and the Darlington Nuclear Generating Station (see Figure 1) to minimize tritium exposure.

Members of the 1985 Pickering cohort living in the same residence for the previous 6 years (non-movers) were identified through deterministic linkage to the 1979 PAF. We assumed the stability of non-movers' residence and therefore assumed more stable tritium exposure in comparison to the rest of the cohort. Non-movers were analyzed separately.

Additional information on data quality and data preparation, including linkage methodology, is available from the authors on request.

Ontario Cancer Registry

We obtained incident cancer cases for this study from the Ontario Cancer Registry (OCR). The OCR captures all new cases of invasive neoplasia, except for non-melanoma skin cancers, in the province of Ontario.[23]

The 1985 Pickering and Oshawa PAFs were probabilistically linked[24] to the OCR to determine incident cases of cancer diagnosed from 1 July 1985, to 31 December 2005. Cohort members diagnosed with cancer contributed person-time until their diagnosis date.

Cancers were chosen a priori based on evidence from moderate-to-high dose studies that achieved reasonable statistical power and precise estimates.[2] Elevated risks were substantial for leukemia and especially pronounced for those exposed at a young age. Female breast, thyroid and lung cancers were also elevated. A review supported the linear extrapolation of these results to low-dose scenarios.[25] All cancers combined were examined for comparison. The relevant *International Classification of Diseases, 9th revision* (ICD-9) diagnosis codes were 140 to 239 (all cancers), 162 (lung), 174 (breast), 193 (thyroid) and 204 to 208 (leukemia).

Vital Statistics - Mortality Data[26]

These data were used to remove cohort members who had not been diagnosed with cancer but who died from any cause within the follow-up period (1985–2005). These subjects contributed person-years until their date of death. The Pickering and Oshawa PAFs were probabilistically linked to these data.

PNGS modelled tritium estimates

To characterize the spatial distribution of tritium originating from PNGS, we implemented the AERMOD Gaussian atmospheric dispersion model.[27] Average regional meteorological data observed at Toronto Pearson International Airport (1996–2000) and facility characteristics that included average annual tritium emissions reported by Ontario Power Generation (1994–1998) were incorporated into the model, as were the velocity and temperature of the emissions. Atmospheric tritium radiation levels were estimated in becquerels (one unit of radioactive decay per second) per cubic meter (Bq/m^3) for each unit in a spatial grid 50 km by 50 km that covered the study area. Tritium estimates were assigned to each cohort member based on the value calculated for the grid cell that overlapped the exact residential address as indicated in the 1985 PAF (see Figure 1 for tritium dispersion surface).

Average annual household income

We used average household income as a proxy for smoking[28] and adjusted for this in the analyses. Average household income was assigned as a continuous variable to each cohort member using the average household income in 1990 as recorded by the 1991 Census at the enumeration area[29] level. The 1991 Census was the earliest time for which average household income information was released at this fine spatial level. Individual income information was not available.

Analytical methods

Person-years analysis

For objective 1, we undertook a standard person-years analysis[30] of the Pickering and north Oshawa cohort to estimate standardized incidence ratios (SIRs) by five-year periods (1986–1990, 1991–1995,

1996–2000, 2001–2005) and assess differences over time as well as over the whole time period (1986–2005). We conducted this analysis to assess the overall health of the cohort in comparison to a standard population.

We used the LEXIS SAS macro[31] to calculate person-years for the specified time periods for Pickering residents, Pickering non-movers and north Oshawa residents, by major cancer site (all sites combined, female breast, leukemia, lung, thyroid and childhood cancers combined for 6–19 years), sex and 5-year age group. The childhood cancers combined category was limited to 6 to 19 years due to PAF exclusions (see ''Data Sources'' section). We obtained cancer rates by sex and 5-year age group for Ontario from SEER*Stat[32] (data available from 1986 onwards) for the time periods specified. Site-specific expected counts were calculated by multiplying sex- and age-stratified person-years for each cancer site by Ontario age-specific cancer rates.[33] Expected (E) and observed (O) counts were summed across age groups and overall SIRs (O/E) and mid-p exact confidence intervals (CIs) were calculated[34] for Pickering residents, Pickering non-movers and north Oshawa residents.

Cox models

We conducted Cox proportional hazards regression[35] with R version 2.13.2 (R Foundation, Vienna, Austria) to address objectives 2 and 3. Cox models are preferred for time-to-event analysis over other statistical methods in the epidemiological literature for several reasons, the most often cited being that specifying a probability distribution for follow-up times is not required.[36] Models focused on male and female lung cancer and female breast cancer. We could not analyze thyroid cancer and leukemia in the cohort due to small sample sizes.[37]

Two exposure scenarios were tested: one where Pickering (higher tritium concentrations) was compared with north Oshawa (low tritium concentrations) with risk estimates adjusted for tritium concentration; the other where risk of cancer associated with increasing tritium concentration was examined in a model limited to Pickering non-movers. Given a sample size of about 18 000 exposed (Pickering) and about 22 000 unexposed (north Oshawa), we have 80% power to detect: (1) a doubling of breast cancer risk; (2) a 2.5 times increase in female lung cancer risk; and (3) a 2.4 times increase in male lung cancer risk. Considering the much smaller sample size in the Pickering non-mover analysis, these analyses are underpowered. We note that obtaining adequate sample sizes is a common problem in this area of research; however, we stress the unique character of this study in examining cancer risks from tritium exposure in a sizeable population-based cohort.

In all Cox models, age was used as the time scale[38,39] rather than follow-up time to (1) more efficiently adjust for the non-parametric effect of age, taking into account the risk of cancer increasing non-linearly with age[40] and (2) put subjects with similar risks, related to age, in a risk set together rather than forming the risk set based on subjects with similar follow-up time.[41] The hazard ratio (HR) in these models is interpreted as an age-specific risk rather than a time-specific risk.[39]

We assumed that average annual household income would confound the relationship between tritium exposure and cancer, and therefore we did not formally build models.[42] Non-linearity of tritium exposure and average household income were accommodated by creating a change-point* at the average values of 2.9 Bq/m^3 and $64 725, respectively. HRs and associated 95% CIs for tritium were associated with a unit increase in tritium exposure. Non-normality of average household income was corrected by square root transformation of standardized values. HRs and associated 95% CIs for average income were associated with a $10 000 increase in income. Interactions between income and tritium exposure were also tested and retained only if significant ($p \leq .05$). Models were also adjusted for frailty, taking into account potential clustering of cancer risk in adjacent census tracts.[43,44]

The study received ethics approval from the Ontario Cancer Research Ethics Board. Access to OCR and Vital Statistics Mortality data was approved by the Data Access Committee at CCO. The Durham Region Planning Department provided approval for use of the PAF.

Results

Description of study cohort

Characteristics of the Pickering (n = 36 805), north Oshawa (n = 43 035) and Pickering non-mover cohorts (n = 10 084) are summarized in Table 1. Of note, the average annual household income in 1990 was significantly lower (~$10 000; $p < .0001$) and the average age at the beginning of follow-up for both sexes was significantly older (~3 to 4 years; $p < .0001$) in north Oshawa compared to Pickering. Compared with all Pickering residents, the average age of Pickering non-movers at the beginning of follow-up for both sexes was significantly older. In addition, average annual household income was significantly lower (~$1500; $p < .0001$) among Pickering non-movers compared with all Pickering residents.

More than half of Pickering and all of north Oshawa residents experienced average tritium concentration levels below 2.9 Bq/m^3 (range: 0–14.74 Bq/m^3). This value is estimated to be an average effective dose of 0.47 µSv/year (range 0–2.36 µSv/year) for an average adult[45] (assuming a radiological biological effectiveness of 1 and the dose coefficient recommended by the Canadian Nuclear Safety Commission, 2.0×10^{-11} Sv/Bq), consistent with Ontario Power Generation dose estimates[22] and not registering on the low-dose range (1–100 mSv, where 1 mSv = 1000 µSv).[46] If the provisional radiological biological effectiveness value for tritium of 2 was used,[6] dose estimates would be double that indicated but would still be far below the regulatory limit.

Person-years analysis

We observed little difference in SIRs across the four time periods for any of

* Point along the distribution of values for the independent variables where the nature of the relationship with the dependent variable is thought to change.

TABLE 1
Characteristics of Pickering, north Oshawa and Pickering non-mover[a] cohorts, 1985

	Population (n)					
	All Pickering		North Oshawa		Pickering Non-movers[a]	
	Females (n = 18 200)	Males (n = 18 605)	Females (n = 21 731)	Males (n = 21 304)	Females (n = 4845)	Males (n = 5239)
Starting age, mean (SD) years	31.84 (16.50)	31.58 (16.25)	35.73* (19.03)	34.55* (18.55)	35.14* (17.74)	34.41* (17.60)
Follow-up time in years, n (%)						
< 1	43 (<1)	53 (<1)	86 (<1)	92 (<1)	16 (<1)	19 (<1)
1 to < 10	502 (3)	599 (3)	985 (5)	1217 (6)	183 (4)	243 (5)
10 to < 20	815 (4)	1012 (5)	1503 (7)	1652 (8)	293 (6)	435 (8)
20	16 840 (93)	16 941 (91)	19 157 (88)	18 343 (86)	4353 (90)	4561 (87)
1990 Average EA income[b], $ (SD)	67 000 (13 395)	67 050 (13 279)	56 732* (15 525)	57 507* (15 403)	65 488* (12 524)	65 238* (12 876)
1990 Average EA income[b], n (%)						
$0–$64 725[c]	8241 (45)	8391 (45)	17 196 (79)*	16 557 (78)*	2424 (50)*	2666 (51)*
$64 726–$115 015[d]	9959 (55)	10 214 (55)	4535 (21)*	4747 (22)*	2421 (50)*	2573 (49)*
Tritium dispersion in Bq/m^3, n (%)						
≥ 2.9[d]	7127 (39)	7268 (39)	0 (0)*	0 (0)*	2645 (55)*	2851 (55)*
< 2.9[c]	11 073 (61)	11 337 (61)	21 731 (100)*	21 304 (100)*	2200 (45)*	2388 (46)*

Abbreviations: EA, enumeration area; SD, standard deviation.

[a] Resident at the same address in 1979.

[b] Source: Census of Canada, 1991.[28]

[c] Below average.

[d] Above average.

* $p < .05$ compared with All Pickering and same sex mean or proportion; significance tests not conducted for follow-up time.

the cancer sites for Pickering, Pickering non-mover or north Oshawa residents. As a result, we reported only results across the whole time period (1986–2005) (see Table 2).

In Pickering the observed number of cases for the majority of cancer sites examined was significantly lower than expected across the entire time period. However, the observed number of female childhood cancers was significantly higher than expected (SIR = 1.99, 95% CI: 1.08–3.38).

None of the SIRs among all Pickering non-movers and north Oshawa residents were significantly elevated across the entire time period.

Cox models

The models comparing Pickering to north Oshawa (Table 3) reveal a significantly higher risk of female lung cancer in the Pickering cohort compared with the north Oshawa cohort (HR = 2.34; 95% CI: 1.23–4.46) after adjusting for modelled tritium dispersion, average household income and frailty. Of note, there was no evidence that tritium exposure was significantly associated with the risk of female lung cancer (< 2.9 Bq/m^3: HR = 0.56, 95% CI: 0.21–1.48; ≥ 2.9 Bq/m^3: HR = 1.00, CI: 0.39–2.55). An increase of $10 000 in average household income was associated with a significant 33% reduction in female lung cancer risk among those with below average household income (HR = 0.67, 95% CI: 0.55–0.82).

There was no significant difference in the risk of male lung cancer (HR = 0.93, 95% CI: 0.53–1.66) or female breast cancer (HR = 1.20, 95% CI: 0.82–1.77) between Pickering and north Oshawa residents. There was a significant 20% reduction in male lung cancer risk for every $10 000 increase in household income, irrespective of average neighbourhood household incomes. Frailty in these models indicated non-significant clustering of cancer risk at the census tract level. No significant interactions were found.

In the Cox models limited to Pickering non-movers, tritium had no significant effect on male and female lung cancer risk and female breast cancer risk (results available from the authors on request). Average household income, frailty and interactions were non-significant in all models.

Discussion

Person-years analysis of this retrospective cohort does not provide sufficient evidence for significantly elevated risks of cancer in Pickering, Ontario. For all Pickering residents, Pickering non-movers and north Oshawa residents, 19 of 33 categories of observed cancer cases were, in fact, significantly lower than expected. The one exception was female childhood cancers (all types combined, for 6–19 years) where the observed number of cases in Pickering was significantly higher than expected. However, this should be interpreted with caution for several reasons. First, radiation-induced cancer risks do not differ for boys and girls, yet there was no increased risk among boys. Second, the small expected value of 6 suggests this finding could be

TABLE 2
Age- and sex-standardized incidence ratios for Pickering, Pickering non-mover[a] and north Oshawa cohorts, 1986–2005 (using Ontario reference rates)[b]

	Pickering				North Oshawa				Pickering Non-movers[a]			
	Females		Males		Females		Males		Females		Males	
Population, n	18 169		18 584		24 016		23 756		4889		5276	
PY	350 131		356 033		407 819		395 197		92 017		98 579	
Cancer	O	SIR (95% CI)	O	SIR (95% CI)	O	SIR (95% CI)	O	SIR (95% CI)	O	SIR (95% CI)	O	SIR (95% CI)
All sites	1019	0.75 (0.70–0.79)	1150	0.75 (0.71–0.79)	1593	0.79 (0.75–0.83)	1896	0.84 (0.80–0.88)	367	0.82 (0.74–0.91)	471	0.82 (0.75–0.90)
Breast	351	0.82 (0.74–0.91)	n/a	n/a	444	0.76 (0.69–0.83)	n/a	n/a	128	0.94 (0.79–1.11)	n/a	n/a
Leukemia	14	0.70 (0.40–1.15)	13	0.44 (0.25–0.74)	22	0.73 (0.47–1.08)	25	0.61 (0.41–0.89)	— [c]	0.79 (0.29–1.75)	— [c]	0.40 (0.13–0.96)
Lung	114	0.78 (0.64–0.93)	165	0.66 (0.57–0.77)	158	0.67 (0.58–0.79)	264	0.71 (0.63–0.80)	45	0.85 (0.63–1.13)	68	0.70 (0.55–0.88)
Thyroid	37	0.69 (0.50–0.95)	14	0.92 (0.52–1.50)	42	0.69 (0.50–0.92)	20	1.14 (0.71–1.73)	6	0.43 (0.17–0.90)	— [c]	0.45 (0.05–1.61)
All childhood (6–19 years)	12	1.99 (1.08–3.38)	6	0.88 (0.36–1.83)	— [c]	0.84 (0.31–1.86)	— [c]	0.74 (0.27–1.64)	— [c]	2.06 (0.52–5.60)	— [c]	0.78 (0.25–1.87)

Abbreviations: CI, confidence interval; O, number of observed cases; PY, person-years; SIR, standardized incidence ratio.

[a] Resident at the same address for the previous 6 years.

[b] Cancer rates from Cancer Care Ontario (Ontario Cancer Registry).[32]

[c] Suppressed due to counts ≤ 5.

due to chance. Third, in this analysis we simultaneously conducted 33 hypothesis tests and under these conditions there is a large statistical probability that one test will be significantly higher than expected by chance alone. We believe this issue of multiple comparisons is the most likely explanation of the increased risk in female childhood cancers. We also examined the observed number of cases for individual cancer sites in this age group and found none were higher than expected. In addition, the cancer site with the largest observed count has no association with ionizing radiation. We also note that the studies conducted in Germany[10,11] found elevated risk of childhood leukemia in the under-five age group, which is younger than the age group in this study.

The Cox models did not provide evidence of a statistically significant association between tritium emissions originating from PNGS and cancer risk.

The Cox models did show that risk of female lung cancer is over twice as high among Pickering residents compared with north Oshawa residents; however, tritium estimates do not significantly contribute to this risk. It is estimated that more than 85% of lung cancers in Canada are related to smoking[47]—32% of Canadian women were reported to be daily smokers in 1981[48]—and we did not have information on individual or small area level smoking estimates to adjust for this in our analyses. We did adjust for smoking in Cox models using average household income as a proxy; however, this may have been insufficient. It is possible that there was substantial disparity in smoking prevalence as well as other confounders and period or cohort effects between Pickering and north Oshawa residents in the 1970s and 1980s that we were unable to estimate and adjust for and that could have contributed to the difference in female lung cancer risk seen here.

Using Pickering non-movers in a separate Cox model was the best method available to control for potential migration of cohort members and the effect of this on tritium exposure estimates. However, these analyses were adequately powered to detect only very large differences in risk, which

TABLE 3
Cox models for Pickering versus north Oshawa residents for female and male lung cancer, and female breast cancer

Variable	Hazard Ratio (95% CI)		
	Female Lung Cancer (n = 39 521)	Male Lung Cancer (n = 39 562)	Female Breast Cancer (n = 39 521)
Pickering (vs. north Oshawa)	2.34 (1.23–4.46)	0.93 (0.53–1.66)	1.20 (0.82–1.77)
Tritium, Bq/m^3			
< 2.9[a]	0.56 (0.21–1.48)	1.60 (0.69–3.71)	0.71 (0.40–1.26)
≥ 2.9[a]	1.00 (0.39–2.55)	0.84 (0.40–1.75)	1.52 (0.92–2.50)
Income, $			
$< 64\ 725$[b]	0.67 (0.55–0.82)	0.81 (0.68–0.95)	1.15 (0.99–1.34)
$\geq 64\ 725$[b]	0.95 (0.80–1.14)	0.82 (0.71–0.95)	1.01 (0.92–1.12)
Frailty (Census tract)	n.s.	n.s.	n.s.

Abbreviations: CI, confidence interval; n.s., non-significant

[a] Change-point at the average tritium concentration. Interpret as per unit increase in tritium.

[b] A square root transformation was applied, income was standardized and change-point made at the average income for Pickering. Interpret per $10 000 increase in average income.

would not be expected from low levels of tritium exposure.

The number of research studies examining cancer risks in relation to CANDU reactors and other HWRS are limited. McLaughlin et al.[49] and Clarke et al.[50,51] examined risk of childhood leukemia around PNGS and a nuclear-generating station in Bruce County (also in Ontario) in a cross-sectional study. They found elevated but non-significant risks among children born within 25 km and among children whose mothers lived within 25 km of either plant.[49,50,51] In 2007, Durham Region Health Department released a surveillance report that examined cancer incidence in Ajax-Pickering (Ajax is a municipality adjacent to Pickering) compared with that of two nearby regions with no nuclear facilities, over two time periods.[52] This report found that female breast, lung, thyroid, leukemia and childhood cancer risks were not consistently higher in Ajax-Pickering compared with reference areas.[52] The results of our cohort study are consistent with these findings.

In terms of occupational studies related to CANDU nuclear reactors, Zablotska et al.[53] found significant excess relative risks (but with wide-ranging CIs) for leukemia and all solid tumours combined. However, the authors indicated that it was possible that these results were due to chance. Concerns about the data prompted a re-analysis[54] and no increased cancer risk was found. McLaughlin et al.[55] found that childhood leukemia was not associated with paternal occupational radiation exposure. Potentially important confounders were unavailable to use for adjustment in all studies.

Strengths

The cohort design we used in our study permitted explicit consideration of the long latency period of cancer by enabling follow-up of cohort members for a period of time (about 20 years) sufficient for most cancers to develop.

We were able to adjust for income in our Cox models whereas the studies mentioned[49-55] above did not. We were also able to identify non-moving Pickering residents to further isolate a sub-population of the cohort that likely had more stable tritium exposure.

Ours appears to be the only population-based epidemiological study examining risks from any type of nuclear power plant that used formal estimates of tritium concentrations in the environment—an important strength. All previous studies around CANDU reactors assumed tritium exposure by proximity alone.

Better aligned data not being available, there is some misalignment of dates for data sources used in tritium estimation. The impact of this on the validity of these tritium estimates is, however, minimal. Long-term meteorological data are relatively constant over many years, and thus the estimated exposure gradient would be similar over many years both before and after the period of the data source (1996–2000). In terms of the tritium emissions and facility characteristics used in this study (1994–1998), historical data show that the quantity of annual tritium emissions has been relatively consistent since the mid-1970s.[3,56]

There are marked differences between on-site meteorology at PNGS and meteorology observed at Toronto Pearson International Airport. However, when predicted model estimates using either meteorology are compared with observed tritium concentrations for a number of on-site monitors, predicted model estimates were quite similar to each other and higher than concentrations observed by on-site monitors.[57]

Limitations

We are reasonably confident that our tritium estimates are appropriate given that modelled estimates closely align with on-site monitors. However, we are less confident that these ecological estimates represent true dose for cohort members because we could not reconstruct personal activity patterns or consider other sources of radiation exposure. We could have made assumptions to reconstruct the dose; however, this would add little value to these analyses because assumptions would be uniformly applied across the

cohort and would not change the distribution of exposure among cohort members.

This inability to assign individual exposure accurately can lead to measurement error.[58] Considering the wide CIs around these tritium risk estimates and the large sample sizes in the Pickering versus north Oshawa analyses, potential misclassification of tritium would not likely change the interpretation of its contribution to cancer risk.

Loss-to-follow-up is a potential bias that may affect the results. Potential loss-to-follow-up due to name changes was minimized because alternative names were available in the OCR. There was 88% agreement between two record linkage analysts working independently to review uncertain matches. It is also possible that loss-to-follow-up occurred through emigration from Ontario. As long as cohort members remained in Ontario, there is reasonable certainty that cancer and mortality information were captured by the probabilistic linkages. Unfortunately, no estimate of emigration from the study area is available. The bias caused by migration is not well understood.[59]

Future studies

Future studies would benefit from using a larger retrospective cohort to examine rare cancers. In addition, reconstruction of personal dose estimates using knowledge of other sources of radiation exposure, residential history and activity patterns would be useful.

Conclusion

We did not find increased risk of cancer associated with tritium exposure from PNGS. Improving the validity of individual tritium exposure estimates is crucial to allay public concern. The use of a retrospective cohort with sufficient follow-up time, a large sample size and tritium estimation in this study are substantial methodological improvements. This study increases our understanding of cancer risks and low level tritium exposure.

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References

1. Innovative Research Group, Inc. 2012 public opinion research - national nuclear attitude survey [Internet]. Ottawa (ON): Canadian Nuclear Association; 2012 [cited 2012 Jul 19]. Available from: http://www.cna.ca/wp-content/uploads/2012NuclearAttitudeReport.pdf

2. Wakeford R. The cancer epidemiology of radiation. Oncogene. 2004;23(38):6404-28.

3. Canadian Nuclear Safety Commission. Health effects, dosimetry and radiological protection of tritium: part of the Tritium Studies Project [Internet]. Ottawa (ON): CNSC; 2010 [cited 2011 Oct 26]. Available from: http://www.nuclearsafety.gc.ca/pubs_catalogue/uploads/CNSC_Health_Effects_Eng-web.pdf

4. Report of the Committee Examining Radiation Risks of Internal Emitters (CERRIE) [Internet]. London (UK): CERRIE; 2004 [cited 2013 Jan 30]. Available from: http://www.rachel.org/lib/cerrie_report.041015.pdf

5. Little MP, Wakeford R. Systematic review of epidemiological studies of exposure to tritium. J Radiol Prot. 2008;28:9.

6. Health Protection Agency. Review of risks from tritium: report of the independent Advisory Group on Ionising Radiation [Internet]. London (UK): HPA Radiation Protection Division; 2007 [cited 2013 Jan 23]. Available from: http://www.hpa.org.uk/webc/HPAwebFile/HPAweb_C/1197382221858

7. Laurier D, Bard D. Epidemiologic studies of leukemia among persons under 25 years of age living near nuclear sites. Epidemiol Rev. 1999;21(2):188-206.

8. Laurier D, Grosche B, Hall P. Risk of childhood leukaemia in the vicinity of nuclear installations--findings and recent controversies. Acta Oncol. 2002;41(1):14-24.

9. Laurier D, Jacob S, Bernier M, et al. Epidemiological studies of leukaemia in children and young adults around nuclear facilities: a critical review. Radiat Prot Dosimetry. 2008;132(2):182-90.

10. Kaatsch P, Spix C, Schulze-Rath R, Schmiedel S, Blettner M. Leukaemia in young children living in the vicinity of German nuclear power plants. Intl J Cancer. 2008 Feb 15;122(4):721-6.

11. Spix C, Schmiedel S, Kaatsch P, Schulze-Rath R, Blettner M. Case-control study on childhood cancer in the vicinity of nuclear power plants in Germany 1980-2003. Eur J Cancer. 2008 Jan;44(2):275-84.

12. Commission on Radiological Protection (SSK). Assessment of the epidemiological study on childhood cancer in the vicinity of nuclear power plants (KiKK Study) [Internet]. Bonn (DE): Strahlenschutzkommission; 2008 Sep [cited 2013 Jan 28]. Available from: http://www.ssk.de/SharedDocs/Beratungsergebnisse_PDF/2008/Kikk_Studie_e.pdf?__blob=publicationFile

13. Laurier D, Hémon D, Clavel J. Childhood leukaemia incidence below the age of 5 years near French nuclear power plants. J Radiol Prot. 2008 Sep;28(3):401-3.

14. Sermage-Faure C, Laurier D, Goujon-Bellec S, et al. Childhood leukemia around French nuclear power plants--the Geocap study, 2002-2007. Int J Cancer. 2012 Sep 1;131(5):769-80.

15. Bithell JF, Keegan TJ, Kroll ME, Murphy MF, Vincent TJ. Childhood leukaemia near British nuclear installations: methodological issues and recent results. Radiat Prot Dosimetry. 2008;132(2):191-7.

16. Heinavaara S, Toikkanen S, Pasanen K, Verkasalo PK, Kurttio P, Auvinen A. Cancer incidence in the vicinity of Finnish nuclear power plants: an emphasis on childhood leukemia. Cancer Causes Control. 2010;21(4):587-95.

17. Government of Canada SC. Statistics Canada: 2006 Community Profiles [Internet]. 2007 [cited 2013 Jan 28]. Available from: http://www12.statcan.gc.ca/census-recensement/2006/dp-pd/prof/92-591/details/Page.cfm?Lang=E&Geo1=CSD&Code1=3518001&Geo2=PR&Code2=35&Data=Count&SearchText=pickering&SearchType=Begins&SearchPR=01&B1=All&Custom=

18. International Atomic Energy Agency. Heavy water reactors: status and projected development [Internet]. Vienna (AT): International Atomic Energy Agency; 2002 Apr [cited 2013 Jan 28]. Technical reports series no. 407. Available from: http://www-pub.iaea.org/MTCD/publications/PDF/TRS407_scr/D407_scr1.pdf

19. United Nations Scientific Committee on the Effects of Atomic Radiation. Sources and effects of ionizing radiation. UNSCEAR 2008 report to the General Assembly with Scientific Annexes. Volume 1 [Internet]. New York (NY): United Nations; 2010 [cited 2013 Jan 28]. Available from: http://www.unscear.org/docs/reports/2008/09-86753_Report_2008_Annex_B.pdf

20. Canadian Nuclear Safety Commission. Investigation of the environmental fate of tritium in the atmosphere: Part of the Tritium Studies Project [Internet]. Ottawa (ON): CNSC; 2010 [cited 2012 July 19]. Available from: http://nuclearsafety.gc.ca/pubs_catalogue/uploads/Investigation_of_Environmental_Fate_of_Tritium_in_the_Atmosphere_INFO-0792_e.pdf

21. Ontario Power Generation. 2006 results of radiological environmental monitoring programs [Internet]. Toronto (ON): Ontario Power Generation: 2006 [cited 2013 Jan 28]. Available from: http://www.opg.com/pdf/Nuclear%20Reports%20and%20Publications/2006%20Radiological%20Environmental%20Monitoring%20Program%20%28REMP%29%20Report.pdf

22. Ontario Power Generation. 2011 results of radiological environmental monitoring programs [Internet]. Toronto (ON): Ontario Power Generation; 2012 [cited 2013 Jan 28]. Available from: http://www.opg.com/pdf/Nuclear%20Reports%20and%20Publications/2011%20Radiological%20Environmental%20Monitoring%20Program%20%28REMP%29%20Report.pdf

23. Holowaty EJ, Chong N. The Ontario cancer registry: a registry with almost complete automated data collection. In: Black RJ, Simonato L, Storm H, editors. Automated data collection in cancer registry, IARC technical reports, No. 32. Lyon (FR): IARC Press; 1998:18(32).

24. Jaro M. Probabilistic linkage of large public health data files. Stat Med. 1995;14:491-8.

25. Brenner DJ, Doll R, Goodhead DT, et al. Cancer risks attributable to low doses of ionizing radiation: Assessing what we really know. Proc Natl Acad Sci U.S.A. 2003;100(24):13761-6.

26. Health Analytics Branch. Health analyst's toolkit [Internet]. Toronto (ON): Ontario Ministry of Health and Long-Term Care; 2012 [cited 2013 Jan 10]. Available from: http://www.health.gov.on.ca/english/providers/pub/healthanalytics/health_toolkit/health_toolkit.pdf

27. AERMOD Implementation Workgroup. AERMOD implementation guide [Internet]. Atlanta (GA): U.S. Environmental Protection Agency. 2009 Mar [cited 2013 Jan 28]. Available from: http://www.epa.gov/scram001/7thconf/aermod/aermod_implmtn_guide_19March2009.pdf

28. Schaap MM, Kunst AE. Monitoring of socio-economic inequalities in smoking: learning from the experiences of recent scientific studies. Public Health. 2009;123(2):103-9.

29. Statistics Canada. Census of Canada, 1991: profile of enumeration area - part B (B9105) [Internet]. Ottawa (ON): Statistics Canada; [cited 2011 October 11]. Available from: http://prod.library.utoronto.ca:8090/datalib/codebooks/c/cc91/profilea/b9105.reclay

30. Szklo M, Nieto J. Epidemiology: beyond the basics. 2nd ed. Burlington (MA): Jones and Bartlett Publishers; 2006.

31. Carstensen B. Lexis: a SAS-macro for splitting follow-up time [Internet]. Copenhagen (DK): University of Copenhagen; 1999 [cited 2011 Oct 18]. Available from: http://bendixcarstensen.com/Lexis/Lexis.sas

32. Cancer Care Ontario (Ontario Cancer Registry). SEER*Stat Release - OCRIS.

33. Breslow NE, Day NE, editors. Statistical methods in cancer research: volume II: the design and analysis of cohort studies. IARC Scientific Publications No. 82. Lyon (FR): IARC; 1994.

34. Fleiss JL, Levin B, Paik MC, Fleiss J. Statistical methods for rates and proportions. 3rd ed. Hoboken (NJ): Wiley-Interscience; 2003.

35. Cox D. Regression models and life-tables. J R Stat Soc Ser B Stat Methodol. 1972; 34(2):187-220.

36. Allison PD. Survival analysis using SAS: a practical guide. Cary (NC): SAS Publishing; 1995.

37. Brown P, Jiang H. Simulation-based power calculations for large cohort studies. Biom J. 2010;52(5):604-15.

38. Thiebaut AC, Benichou J. Choice of time-scale in Cox's model analysis of epidemiologic cohort data: a simulation study. Stat Med. 2004;23:3803-20.

39. Commenges D, Letenneur L, Joly P, Alioum A, Dartigues JF. Modelling age-specific risk: application to dementia. Stat Med. 1998;17(17):1973-88.

40. Kom EL, Graubard BI, Midthune D. Time-to-event analysis of longitudinal follow-up of a survey: choice of the time-scale. Am J Epidemiol. 1997;145(1):72-80.

41. Canchola AJ, Stewart SL, Bernstein L, et al. Cox regression using different time-scales [Internet]. Western Users of SAS Software, 2008 Conference; [cited 2011 Oct 31]. Available from: http://www.lexjansen.com/wuss/2003/DataAnalysis/i-cox_time_scales.pdf

42. Vittinghoff E, Glidden DV, Shiboski SC, McCulloch CE. Regression methods in biostatistics: linear, logistic, survival, and repeated measures models. New York (NY): Springer; 2005.

43. Hosmer DW Jr, Lemeshow S, May S. Applied survival analysis: regression modeling of time to event data. 2nd ed. Hoboken (NJ): Wiley-Interscience; 2008.

44. Banerjee S, Wall MM, Carlin BP. Frailty modeling for spatially correlated survival data, with application to infant mortality in Minnesota. Biostatistics. 2003 Jan;4(1):123-42.

45. Age-dependent dose to members of the public from intake of radionuclides: Part 5. Compilation of ingestion and inhalation dose coefficients. Ann ICRP. 1996 Jan;26(1):1-91.

46. Gilbert ES. Ionising radiation and cancer risks: what have we learned from epidemiology? Int J Radiat Biol. 2009;85(6):467-82.

47. Canadian Cancer Society. Smoking and cancer [Internet]. Toronto (ON): Canadian Cancer Society; 2013 [cited 2013 May 24]. Available from: http://www.cancer.ca/en/prevention-and-screening/live-well/smoking-and-tobacco/?region=on

48. Stephens T. A critical review of Canadian survey data on tobacco use, attitudes and knowledge [Internet]. Ottawa (ON): Tobacco Programs Unit, Health Promotion Directorate, Health and Welfare Canada; 1988 Apr [cited 2012 Jul 19]. Available from: http://tobaccodocuments.org/nysa_ti_s2/TI14132323.html

49. McLaughlin JR, Clarke EA, Nishri ED, Anderson TW. Childhood leukemia in the vicinity of Canadian nuclear facilities. Cancer Causes Control. 1993;4(1):51-8.

50. Clarke EA, McLaughlin J, Anderson TW. Childhood leukemia around Canadian nuclear facilities - phase I: final report [Internet]. Ottawa (ON): Atomic Energy Control Board; 1989 May [cited 2012 Jul 19]. Available from: http://www.nuclearsafety.gc.ca/eng/about/past/timeline-dev/resources/documents/infohistorical/info-0300-1.pdf

51. Clarke EA, McLaughlin J, Anderson TW. Childhood leukemia around Canadian nuclear facilities - phase II: final report [Internet]. Ottawa (ON): Atomic Energy Control Board; 1991 Jun [cited 2012 Jul 19]. Available from: http://www.nuclearsafety.gc.ca/eng/about/past/timeline-dev/resources/documents/infohistorical/info-0300-2.pdf

52. Durham Region Health Department. Radiation and health in Durham region [Internet]. Whitby (ON): Durham Region Health Department; 2007 [cited 2012 June 27]. Available from: http://www.durham.ca/departments/health/health_statistics/radiationHealthReport2007.pdf

53. Zablotska LB, Ashmore JP, Howe GR. Analysis of mortality among Canadian nuclear power industry workers after chronic low-dose exposure to ionizing radiation. Radiat Res. 2004;161(6):633-41.

54. Canadian Nuclear Safety Commission. Verifying Canadian nuclear energy worker radiation risk: a reanalysis of cancer mortality in Canadian nuclear energy workers (1957-1994) Summary report INFO-0811 [Internet]. Ottawa (ON): CNSC; 2011 Jun [cited 2013 Jan 30]. Available from: http://www.nuclearsafety.gc.ca/pubs_catalogue/uploads/INFO-0811-Verifying-Canadian-Nuclear-Energy-Worker-Radiation-Risk-A-Reanalysis-of-Cancer-Mortality-in-Canadian-Nuclear-Energy-Workers-1957-1994_e.pdf

55. McLaughlin JR, King WD, Anderson TW, Clarke EA, Ashmore JP. Paternal radiation exposure and leukaemia in offspring: the Ontario case-control study. BMJ. 1993;307(6910):959-66.

56. United Nations Scientific Committee on the Effects of Atomic Radiation. UNSCEAR 1988 Report Sources, Effects and Risks of Ionizing Radiation -Annex B: Exposures from nuclear power production [Internet]. New York: United Nations;1988 [cited 2012 July 19]. Available from: http://www.unscear.org/docs/reports/1988/1988e-f_unscear.pdf

57. SENES Consultants Limited. Air dispersion modeling in support of the Ontario Health and Environment Integrated Surveillance (OHEIS) project. Richmond Hill (ON): SENES Consulting; 2009.

58. Nuckols JR, Ward MH, Jarup L. Using geographic information systems for exposure assessment in environmental epidemiology studies. Environ Health Perspect. 2004;112:1007-15.

59. Hatch M, Thomas D. Measurement issues in environmental epidemiology. Environ Health Perpect. 1993;101(Suppl 4):49-57.

Knowledge exchange systems for youth health and chronic disease prevention: a tri-provincial case study

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Abstract

Introduction: The research teams undertook a case study design using a common analytical framework to investigate three provincial (Prince Edward Island, New Brunswick and Manitoba) knowledge exchange systems. These three knowledge exchange systems seek to generate and enhance the use of evidence in policy development, program planning and evaluation to improve youth health and chronic disease prevention.

Methods: We applied a case study design to explore the lessons learned, that is, key conditions or processes contributing to the development of knowledge exchange capacity, using a multi-data collection method to gain an in-depth understanding. Data management, synthesis and analysis activities were concurrent, iterative and ongoing. The lessons learned were organized into seven ''clusters.''

Results: Key findings demonstrated that knowledge exchange is a complex process requiring champions, collaborative partnerships, regional readiness and the adaptation of knowledge exchange to diverse stakeholders.

Discussion: Overall, knowledge exchange systems can increase the capacity to exchange and use evidence by moving beyond collecting and reporting data. Areas of influence included development of new partnerships, expanded knowledge-sharing activities, and refinement of policy and practice approaches related to youth health and chronic disease prevention.

Keywords: *knowledge exchange, youth health, chronic disease prevention, knowledge use, evidence to action, surveillance, partnerships*

Introduction

The burden of chronic disease is increasing worldwide, and chronic disease accounts for 89% of deaths in Canada.[1] Canadian youth are at risk of developing chronic diseases due to their high rates of modifiable harmful health behaviours such as physical inactivity,[2,3] unhealthy eating[4] and tobacco use[5] and may have shorter life expectancies than their parents as a result.[4] The greatest leverage of risk reduction might be achieved through timely intervention early in life.[6]

With these increasing rates of chronic disease, we need to urgently generate and use relevant evidence to inform and guide effective youth health policies and programs. Evidence-based planning enhances prevention programs[7,8] by targeting and evaluating programs and policies and setting priorities.[9] As a result, locally relevant and contextual data on modifiable risk factors are in demand.

Various terms, including ''knowledge exchange,'' ''knowledge translation'' and ''knowledge development'' refer to the process of undertaking research with the intention of effectively applying the resultant data. According to the Canadian Health Services Research Foundation, knowledge exchange (KE) emphasizes the two-way interaction between groups with separate and distinct cultures to ensure that the knowledge created is both useful and relevant to all stakeholders.[10,11] This definition fits with the philosophical approach and the proposed interventions of this study.

Several existing KE frameworks identify the key processes, people and contextual conditions necessary to develop knowledge and act on it. Jacobson et al.[12] provided a practical guide to KE to assist researchers in gathering relevant information about the critical target groups for KE. The Canadian Institutes of Health Research conceptualizes knowledge translation as a dynamic and iterative process that includes synthesis, dissemination, exchange and ethically sound application of knowledge as well as evaluation and monitoring of knowledge translation activities.[13] A third framework is the knowledge-to-action research framework, which is composed of two fluid, complex

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and dynamic cycles: knowledge creation and action.[11]

Although KE has long been recognized as a key to translating knowledge into action, the research to inform and support such efforts is still being developed. Within Canada, stakeholders from policy, practice and research sectors of provincial and national health promotion and chronic disease organizations agree on the importance of better understanding KE processes and examples of evidence-informed practice in local, regional and provincial contexts. They also recognize the need for systems thinking in public health as an emerging method to address complex public health issues.[14]

Building on existing frameworks, three provinces have independently created their own provincial youth health KE systems: Prince Edward Island's School Health Action, Planning, and Evaluation System - Prince Edward Island (SHAPES-PEI; http://www.upei.ca/cshr/shapes); New Brunswick's Student Wellness Survey and Knowledge Exchange Initiative (SWS/KE; http://www.unbf.ca/education/herg/wellness/index.php); and Manitoba's Risk Factor Surveillance System (MRFSS; http://partners.healthincommon.ca). Each of the three provinces established a knowledge-to-action process that recognizes the value of providing evidence-to-inform actions and learning from action-to-refine evidence (see Figures 1, 2 and 3). Four core components of youth health KE were identified in the three provincial KE frameworks:

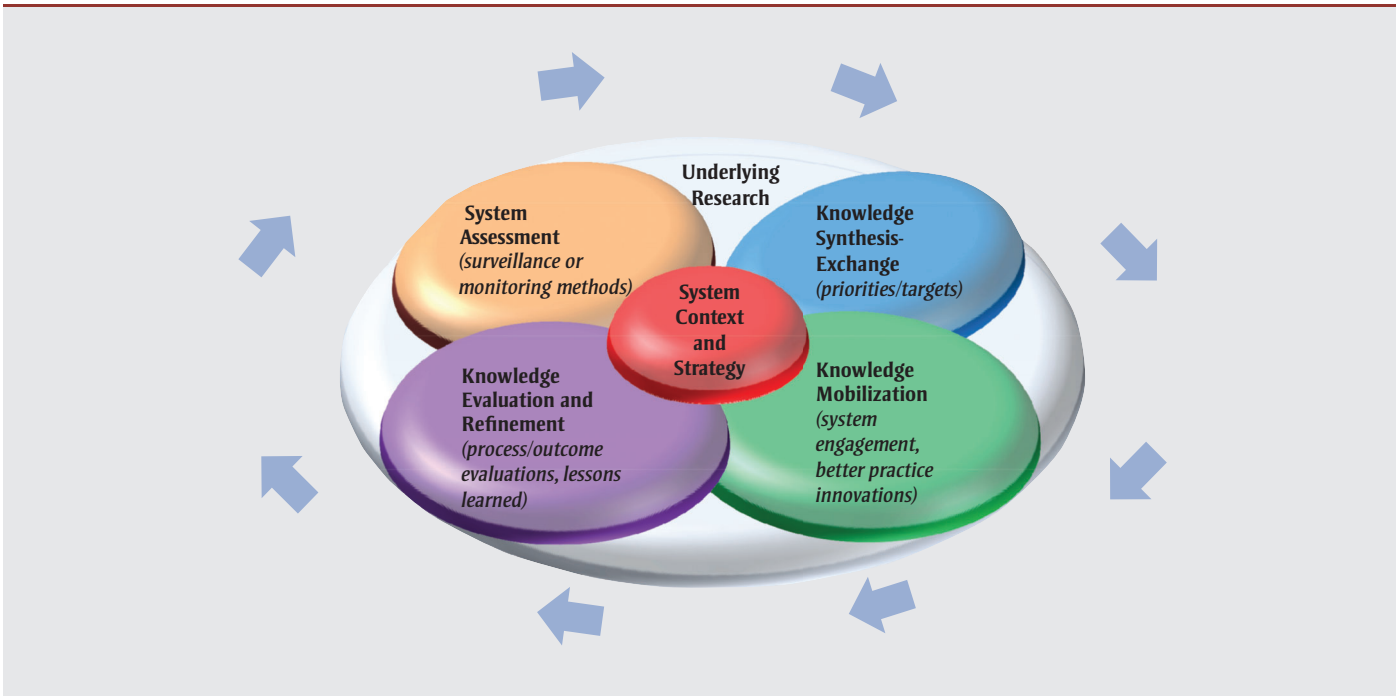
(1) Surveillance systems to support planning and evaluating of policies and programs for children and youth (i.e. collecting local data including risk factor data);
(2) The ability to synthesize relevant evidence with respect to the kinds of interventions that prove to be effective (i.e. interpretation of data informed by literature, program evaluations and the local context);
(3) The capacity to move evidence into action (i.e. using the knowledge derived from interpreting data to implement better practices); and
(4) The means of generating evidence from action (i.e. learning from and sharing better practices, programs, policies, interventions, experiences and evaluations).

The purpose of this paper is to present the lessons learned from this tri-provincial case study of KE systems for youth health and chronic disease prevention.

Methods

We used the Yin[15] case study design to explore the phenomena of youth health KE across three diverse provinces: Manitoba, New Brunswick and Prince Edward Island. Case study design is useful for answering how and why questions whereas multiple case design can be used to explore differences between and within cases and to predict similar results or to predict contrasting results, but for foreseeable reasons.[15] For this study, we used a multi-data collection method to gain an

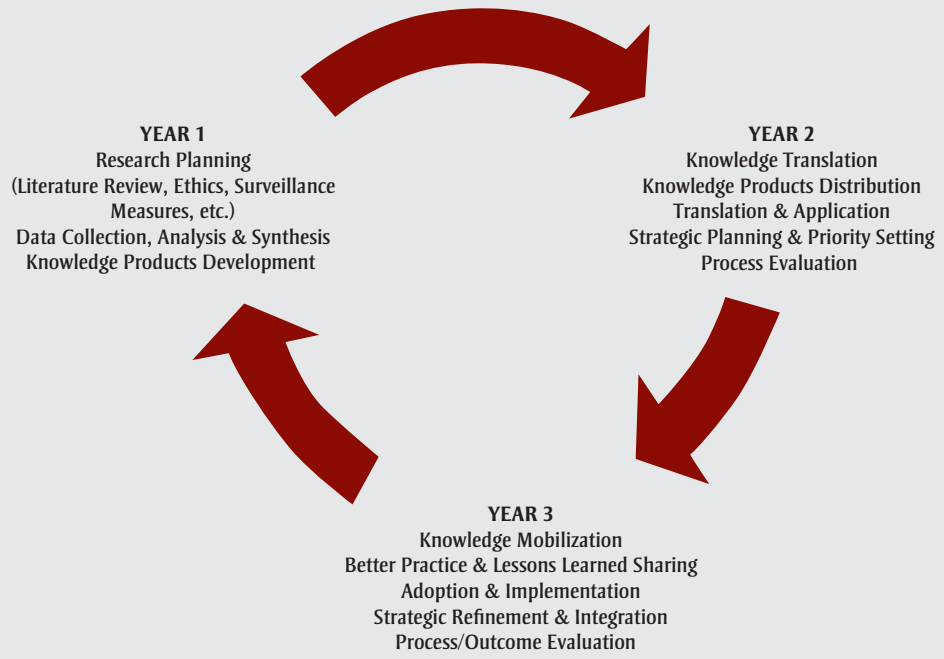
FIGURE 1
SHAPES-PEI Knowledge Development and Exchange Model



Abbreviation: SHAPES-PEI, School Health Action, Planning and Evaluation System - Prince Edward Island.

Note: Figure developed by partners from across Canada including Propel Centre for Population Health Impact (University of Waterloo, Waterloo, Ontario, Canada) and the Health and Education Research Group (University of New Brunswick, Fredericton, New Brunswick, Canada).

FIGURE 2
New Brunswick Student Wellness Survey and Knowledge Exchange Model



Note: Developed by partners from across Canada, including the Health and Education Research Group (University of New Brunswick, Fredericton, New Brunswick, Canada) and Propel Centre for Population Health Impact (University of Waterloo, Waterloo, Ontario, Canada).

in-depth understanding of KE within the real life context of youth health.[15]

Procedures

Each provincial case study developed a research team and advisory committees for this initiative. In addition, the three provinces formed a multi-site research team that consisted of the principle investigators and research staff from each province. While study protocols provided focus and direction, each provincial research team had the autonomy to explore their cases using methods best suited to their context. The teams collaborated to refine processes and instruments for data collection. The many sources of evidence (document analyses, interviews, focus groups and an online survey in Prince Edward Island) enhanced the reliability and validity of case study results (see Table 1).[15]

Collaborating with provincial and national stakeholders, the research teams developed semi-structured interview guides (available on request). Interviews and focus groups were tape-recorded and the recordings transcribed; field notes were also constructed immediately following each interview.[16] Interviews lasted about 45 to 60 minutes. A structured online survey in Prince Edward Island, used to understand the viewpoints of a larger spectrum of partners, end-users and stakeholders, took about 10 to 15 minutes to complete. The documents reviewed included planning and resource documents, meeting minutes, grant applications, communications and press clippings. Data were collected until saturation, (when identified themes became repetitive) was achieved within each provincial case.

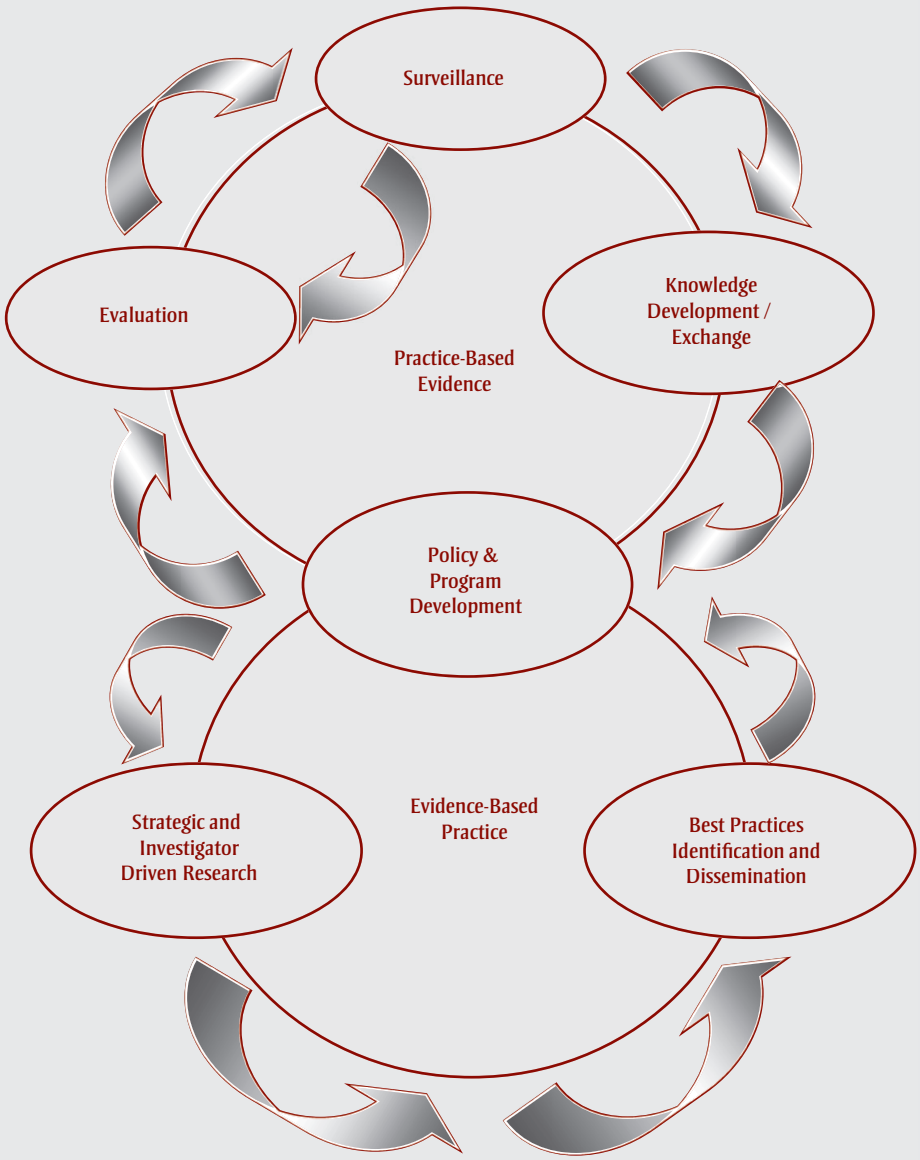
We took steps to prevent interviewers leading or influencing participants by sharing opinions, etc.[17] We used member-checking to reach saturation, to make sure that we thoroughly understood emerging themes and that our findings reflected participants' contributions, and to clarify and explore details of participants' initial interviews. About six months after the initial interviews and focus groups, and after preliminary analyses were completed and themes identified, we shared the initial findings with participants; however, only half were able to participate in follow-up interviews.

The appropriate research ethics board(s) in each province gave ethical approval for the research.

Participants

We used purposeful sampling to identify participants in existing KE networks. This was followed by snowball sampling to reach key stakeholders. All participants were told about the project by email and/or in person and provided informed consent prior to participation. Participants included representatives of provincial health/wellness and education government departments; non-governmental organizations; regional health authorities; schools and school districts; universities; and other key stakeholders who were involved directly in the KE system in their

FIGURE 3
Manitoba Risk Factor Surveillance System



Source: Riley and Harvey, 2006.[18]

province as either partners and/or end-users (see Table 2). Fewer than ten participants from any one province declined participation in the study.

Data analysis

Data management, synthesis and analysis activities were concurrent, iterative and ongoing. We used NVivo 8/9 software (QSR International (Americas) Inc., Burlington, MA, US) to manage and analyze data. Analysis focused on thematic surveys and conceptual/thematic description.[17] Each provincial team used thematic analysis to examine, categorize and tabulate data from multiple sources. Themes were used to label and order portions of the data, and interpretative analysis was used to understand the meaning of the themes.[19] Findings were cross-checked between provincial final reports, participants, document reviews and cross-case discussions.

The provincial teams agreed to use a modified multiple case study analysis procedure as described by Stake.[20] An initial framework was built upon a priori themes identified in the literature and emergent themes resulting from each

TABLE 1
Summary of data collection activities

	Documents, n	Interviews,[a] n	Survey Respondents, n	Focus Groups, n (Participants, n)
MB	137	32	0	6 (35)
NB	78	32	0	2 (48)
PEI	119	26	69	7 (50)

Abbreviations: MB, Manitoba; NB, New Brunswick; PEI, Prince Edward Island.

[a] Total number of interviews conducted (some individuals may have participated in multiple data collection activities).

provincial case and cross-province discussion. Findings were sorted into the framework's identified ''clusters.'' Next, an intensive iterative process across the provincial case study teams resulted in identifying patterns from which emerged a final framework (see Table 3). Common strategies, partners and activities that lead to increased KE uptake could be examined within the framework.

The results of this cross-case study focus on similarities between KE systems, but we also looked for counter-evidence to avoid holistic bias and to make sure that we did not assume greater meaning in the patterns than actually existed.[17] Examining counter-evidence along with supporting evidence resulted in modifications to and/or support for the emerging framework. Focusing on similarities allowed for the emergence of key elements, processes and lessons learned in implementing a KE system. This mutually inductive and deductive process served to deepen critical reflection and to identify the potential range of impact of emerging lessons from each provincial case.

Results

The diverse context (social, political, physical) of each provincial KE system has led to different partnership, funding and structure models. Nevertheless, our cross-case comparison identified similarities between the three provincial KE systems that we expressed as lessons learned within seven ''clusters.'' Lessons learned are defined as key conditions or processes contributing to the development of KE capacity across at least two provincial contexts. Select quotes from research participants are included to demonstrate support for our lessons learned. We purposefully did not identify the provinces where a particular interview took place to preserve the anonymity of all research participants.

1. Guiding knowledge exchange models

All three provinces used existing system frameworks with key processes, people and contextual conditions as a foundation for their surveillance initiatives to plan and execute activities and to guide and communicate the ongoing work. Although these models were different in each province, using KE models helped to communicate and understand different stakeholders' roles in developing, sharing or applying knowledge. Two interviewees explained:

> I think for [the student survey] to be really successful, the participants, whether they are the principals or the parents or the kids … need to get a sense of what is next and understand that this is going to inform the next step and this is the timeline to the next step so that everybody knows that this is the start of a process versus the end of a process. (Province 1)

> It is critical to have a road map and to prioritize as part of the way we do our business. (Province 2)

2. State of readiness

All provinces acknowledged a need for health-related data to inform policy or practice development, and health/wellness and education stakeholders expressed an interest in establishing youth health KE activities.

> Some schools are ready to rock-and-roll with this sort of stuff; other schools are just [on] the cusp of getting involved. (Province 1)

All three provinces lacked comprehensive local level data related to youth health behaviours. Existing networks, coalitions and working relationships were critical to providing an initial foundation for promoting the value of youth health surveillance and KE to inform policy development and practices. Champions who promoted and facilitated the development of surveillance and KE processes came from a variety of stakeholder groups.

> We have a very diverse region. We have affluent, healthy … population[s and] areas [with] high rates of chronic disease. [A] regional average puts it somewhere in the middle. So having the school data would really help

TABLE 2
Interview and focus group participant descriptives

Interviews	PEI (n = 23)	NB (n = 32)	MB (n = 32)
Roles, %			
Research	26	16	0
Policy	26	19	16
Practice	39	65	84
Other	9	0	0
Focus Groups	**PEI (n = 50)**	**NB (n = 48)**	**MB (n = 35)**
Roles, %			
Research	0	8	0
Policy	0	15	9
Practice	0	77	91
Student	100	0	0

Abbreviations: MB, Manitoba; NB, New Brunswick; PEI, Prince Edward Island.

TABLE 3
Cross-case comparison analytical framework

	Cluster Name	Cluster Description
1.	Guiding knowledge exchange models	Existing system frameworks that identified key processes, people and contextual conditions
2.	State of readiness	An acknowledged need for health-related data to inform policy or practice development at either local, provincial or national levels and expressed interest from health/wellness and education stakeholders
3.	Knowledge exchange products	Communication resources, such as reports, facts sheets, websites, etc., intended to engage and inform multiple audiences
4.	Knowledge exchange activities	Events, forums, meetings, presentations or planning sessions designed to engage stakeholders
5.	Strategic partnerships in knowledge exchange	Specific relationships or collaborations identified as playing a key leadership or influential role
6.	Systems and structures	Established or emerging knowledge exchange networks or decision-making systems
7.	Knowledge exchange impacts	Concrete ways in which surveillance outcomes or knowledge exchange activities have contributed to embedding or linking knowledge-to-action processes within existing or emerging planning and decision-making systems

Abbreviation: KE, knowledge exchange.

determine what programs need to go in what communities. (Province 3)

3. Knowledge exchange products

KE products, for example, communications resources such as reports, facts sheets, websites, newsletters, project summaries, conference proceedings, and media communications used to engage and inform multiple audiences provided a common entry point for all three provinces to initiate dialogues with existing and new stakeholders. They were used to present comprehensive findings on youth health behaviours that affect chronic disease such as healthy eating, physical activity, tobacco use and mental fitness. A variety of KE products, written in familiar and simple language, were designed for specific audiences and stakeholder groups (see Table 4). Concise summaries or fact sheets highlighting key youth health outcomes were identified as appealing and interesting to senior policy makers and leaders. Websites were used to make youth health data and resources for KE accessible to a wider range of stakeholders.

> I found [the profile report] easy to go through, easy to read, from my perspective. I mean, I know some parents may be challenged to go through it, but I liked the format … here is the data; this is what it means; this is the action that you could take. (Province 1)

> The website is fantastic. For isolated communities it's the most beneficial. My team goes there for resources quite a bit. (Province 3)

4. Knowledge exchange activities

Focusing on exchanging information with stakeholders at all levels was important in each province; creating engaging KE activities—events, forums, meetings, presentations or planning sessions—was considered essential. KE activities were planned and implemented based on strategic processes within each respective provincial KE model. Regional and provincial KE champions were often identified as co-ordinators, hosts and/or presenters at KE activities. KE activities were identified as beneficial for bringing together stakeholders and facilitating the development of partnerships.

> It is these sharing and exchanging opportunities that provide us with new networks, ideas and successes … this keeps us motivated. (Province 2)

> We presented the information from the reports and had discussions around what does this mean to you [*sic*]. It gave them an opportunity to ask questions and for us to clarify. (Province 3)

5. Strategic partnerships in knowledge exchange

Leadership and established collaborations between stakeholders with expertise in youth health/wellness, education and research were identified as critical for supporting and maintaining surveillance initiatives. Developing partnerships within the education sector was necessary for obtaining and sustaining the participation of schools and districts.

> Truthfully, we had spent a lot of years really ensuring we had built those relationships, that we had made the calls. We had meetings with them on a regular basis. We asked, ''What are we doing right? What are we doing wrong? How can we make this better?'' So we did work hard at that. (Province 1)

TABLE 4
Knowledge exchange products

Product	Intended Audience
School reports / summary reports	School administrators, teachers, students, parents, school and community committees
District/division reports / summary reports	School district/division staff, school boards, communities, health practitioners
Regional reports	Health practitioners, municipal leaders
Provincial reports / summary reports	Provincial government departments, health alliances, non-governmental organizations, general public

> In our small province, it is the practitioners that enable us to accomplish so much with limited resources … Partnerships are key to the strength of the initiative. (Province 2)

6. Systems and structures

Established or emerging KE networks or decision-making systems were recognized as playing a key role in the development and expansion of KE capacity. Pre-existing national networks provided the initial network structure from which to initiate and foster relationships among research, policy and practice stakeholders. Health coalitions, groups, networks and initiatives made use of youth health surveillance data for program planning and health promotion. Surveillance and KE activities were also identified as supporting the development of youth health/wellness planning committees and structures.

> What [the school health network's] role would be to formalize those discussions that we have informally and that probably should be created so when the players change … those conversations continue in a formalized way. (Province 1)

> Members benefit from the unique contributions of all of our partners based on their experiences, resources and expertise. (Province 3)

7. Knowledge exchange outputs

Stakeholders were helped in interpreting and using results so that they could effectively move evidence to action. KE outputs included applying surveillance results, assessing priorities, engaging partners and leveraging funding. Grant programs linked with school health surveillance were associated with increased uptake of KE reports and the use of evidence. Success stories were identified as important sources of motivation and learning. Repetition of the surveillance and KE activities provided an important foundation for building and sustaining school health partnerships. The use of youth health data by departmental stakeholders and/or external groups to set regional and provincial health/wellness plans and priorities as well as to establish program benchmarks was recognized as contributing to widespread support for sustaining school level surveillance and KE activities.

> Some of our schools have embedded the information from the [survey] into ongoing school improvement plans. This works in districts too. (Province 2)

> I remember getting the results and because there was the healthy living grant we shared it with the student council and asked them what they wanted to use the grants for and asked them to apply. (Province 3)

Discussion

In this paper, we describe the lessons learned about the development and implementation of KE systems in three different Canadian provinces. Our findings demonstrate that the three provincial KE systems are similar and that KE is a complex process that requires champions, collaborative partnerships, readiness and the tailoring of KE to diverse stakeholders. All of these components serve to build capacity and sustain KE systems that lead to the creation of real outcomes promoting healthy living.

Our cross-case study findings contribute to the limited empirical research on KE models. Similar themes emerged across the provinces, including the necessity of utilizing a guiding model of KE when implementing such systems. While each of the three provinces had context-specific approaches, they implemented comparable KE systems as demonstrated through the common analytical framework that emerged.

Several existing frameworks such as the Knowledge-to-Action Process Framework,[11] Understanding-User Context Framework,[12] and Model of Knowledge Translation[13] illustrate specific KE processes designed to bridge the gap between researcher and end-user. Similar to these models, the three provincial KE models focus on including stakeholders in the KE processes and recognize the role of context in developing, interpreting and applying knowledge. The provincial models reflect many years of effort when knowledge was acted upon in a timely manner by communities mobilized to use evidence in decision making. Repetition allowed for evidence-informed policies and practices to be evaluated and refined. When practices proved ineffective, the systems adapted and incorporated new knowledge gleaned from those systems that applied models in such a way as to effectively use resources and build capacity. As the model was repeated, communication between and collaboration among partners was also extended, elaborated and enhanced.

The analytical KE framework from this study is based on empirical evidence from three different ''real life'' Canadian jurisdictional experiences, leading to further understanding of KE.

Champions at all levels (local, regional, provincial and national) were essential for eliciting widespread support and advocacy for implementing and continuing surveillance and KE activities. Engaging such networks and champions necessitated promoting the value of evidence-based decision making and the need for collecting and understanding local data. Consistent with the findings of Walter et al.,[21] when these champions endorsed and used youth health data to develop local, regional and provincial health/wellness plans and to establish program benchmarks, the value of local surveillance and KE activities was enhanced among all stakeholders.[21] Champions act as catalysts by introducing new ideas and practices, endorsing these,[21] and mentoring others to take action.

Research, policy and practice often have different priorities, use different language, operate on different time scales and are subject to different reward systems.[22,23] The Centers for Disease Control and Prevention, for example, responded to the need for a common language and conceptualization to expand their understanding of the knowledge-to-action process they were undertaking.[24] In developing collaborative partnerships, opportunities to increase awareness of work functions and partnership expectations help to create a process of mutual

understanding that, in turn, leads to mutual respect and collaborative partnerships and actions. KE models and frameworks can serve as important tools in engaging a variety of partners in a systems approach to preventing chronic disease. The use of a KE model helped stakeholders understand, become involved with and sustain their participation in knowledge-to-action activities related to youth health.

Further, positive working partnerships within the education sector were critical for obtaining and sustaining the participation of schools and districts. By maintaining positive relationships through a clearly articulated mutual and respectful process, all partners were welcome to contribute and felt valued. Gagnon[25] identified four factors for successfully integrating KE within networks and practice communities: the development of shared understanding about the health problem; explicit descriptions of roles/responsibilities; team members with competencies and experience in building and maintaining effective collaborations; and a strategy for ensuring that relationships are maintained.[25]

Key collaborative actions undertaken by the provinces included joint planning of surveillance approaches and their timing, as well as how data will be used and shared across local, regional and provincial jurisdictions. Co-creation of knowledge was found to influence the uptake and use of research by allowing for greater consideration and ability to address contextual factors, thus creating credible and valid information that was both trusted by and useful to stakeholders.[26] Knowledge that addresses areas of concern and priority for stakeholders increases the likelihood that it will be used or applied.[27,28] Consistent with our findings, Williams et al.[29] stressed the importance of involving end-users in all key activities that reflect the knowledge development process. However, examples of sustained, collaborative partnerships and ongoing communication among knowledge producers and end-users are rare and unusual.[30] Our research demonstrated that repetition of surveillance and KE activities helped sustain the partnerships involved in youth health KE. Partnerships evolved and expanded as partners worked together on common surveillance and KE activities.

The engagement of leaders from various stakeholder groups built the capacity to initiate preliminary actions related to province-wide surveillance and KE activities. Successes with health-related surveillance activities and evidence-to-action planning generated further commitment and support for youth health surveillance from both individuals and organizations, as did deriving evidence from action. Ward et al.[31] also found that personal, interpersonal, organizational and professional characteristics and context influenced the KE processes, supporting the importance of building upon existing assets such as expertise, partnerships and infrastructure when implementing a KE system.[31]

Collaborative exchanges are facilitated when relevant KE products are accessed and used. Our findings highlighted the importance of tailoring KE products to diverse stakeholder groups. Appealing features of such products included the use of familiar language and locally relevant information, inclusion of examples of better practices, incorporation of practice-based evidence or success stories, and availability of reports, summaries or fact sheets in multiple formats and locations. KE products such as fact sheets, websites, newsletters, reports, project summaries, conference proceedings, and media communications have promoted collaboration between researchers and research users.[24,32] KE products should include suggested actions tailored to further the uptake and use of evidence.[33,34]

A variety of KE activities are essential to reach and interest diverse stakeholders. These included individual consultations with stakeholders on youth health/wellness outcomes and better practices; group presentations of school/district/provincial outcomes; events based on local and regional surveillance findings; and formal conference presentations and papers. Face-to-face meetings, both formal and informal, of researchers, policy makers and practitioners consistently emerge as the most efficient way to overcome disconnections between partners.[25] In addition, these KE activities take place within a larger system in which interactions occur among many partners with dynamic priorities, processes, contexts, expectations and incentives to change. Therefore, the use of numerous KE strategies that give end-users sufficient choice in content, format and delivery has been found to be important to uptake and use of evidence.[27]

With the increasing rates of chronic disease, it is urgent that Canada generates and uses relevant evidence to inform and guide effective interventions and healthy living policies and programs geared to youth. Research has shown that evidence-based planning enhances chronic disease prevention programs[7,8] when it is used to target and evaluate programs and policies and set priorities.[9] Generating data at a given time is not sufficient to evaluate chronic disease programs/policies and monitor changes in youth health. Utilizing systems thinking can bridge the gap between generating, disseminating and utilizing data.[14] Systems thinking is a key tool for integrating knowledge production and use that is relevant for local action.[14]

Limitations

Our findings can be applied to other jurisdictions that share characteristics similar to those of Manitoba, New Brunswick and Prince Edward Island. Further research should examine the application of our findings from these three prominently rural provinces to larger, more urban jurisdictions and within complex situations. Intervention studies should explore various KE products and activities to test for their effectiveness. Also needed are more refined partnership tools and models that facilitate and support youth health KE processes. Although we have described the similarities across the three KE systems in this paper, we found differences across the three systems that we did not discuss in detail but only acknowledged in the analysis to avoid holistic bias.

Conclusion

Our findings support a KE systems approach that increases the capacity to exchange and use evidence by moving beyond simply collecting data and producing reports. Such

systems can contribute to expanded partnership development and knowledge-sharing activities, as well as the creation of comprehensive policy and practice initiatives designed to promote youth health and chronic disease prevention.

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References

1. World Health Organization. Preventing chronic disease: a vital investment [Internet]. Geneva (CH): World Health Organization; 2005 [cited 2012 Feb 24]. Available from: http://www.who.int/chp/chronic_disease_report/en/

2. Active Healthy Kids Canada. Don't let this be the most physical activity our kids get after school. The active healthy kids Canada 2011 report card on physical activity for children and youth [Internet]. Toronto (ON): Active Healthy Kids Canada; 2011 [cited 2012 Feb 24]. Available from: http://www.activehealthykids.ca/ReportCard/2011ReportCardOverview.aspx

3. Freeman JG, King M, Pickett W, et al. The health of Canada's young people: a mental health focus. Ottawa (ON): Public Health Agency of Canada; 2011. [Public Health Agency of Canada, Catalogue No.: 978-1-100-19335-9].

4. Marshall H, Boyd R. The Chief Public Health Officer's report: report on the state of public health in Canada [Internet]. Ottawa (ON): Public Health Agency of Canada; 2008 [cited 2012 Feb 24]. [Public Health of Canada, Catalogue No.: HP2-10/2011E-PDF]. Available from: http://www.phac-aspc.gc.ca/cphorsphc-respcacsp/2008/fr-rc/pdf/CPHO-Report-e.pdf

5. Health Canada. Health concerns: summary of results of the 2008-09 Youth Smoking Survey [Internet]. Ottawa (ON); [modified 2010 Aug 20; cited 2012 Feb 27]. Available from: http://www.hc-sc.gc.ca/hc-ps/tobac-tabac/research-recherche/stat/_survey-sondage_2008-2009/result-eng.php

6. Hanson M, Gluckman P. Developmental origins of noncommunicable disease: population and public health implications. Am J Clin Nutr. 2011;94(6 supplemental):1754S-8S.

7. Brownson RC, Smith CA, Jorge NE, Deprima LT, Dean CG, Cates RW. The role of data-driven planning and coalition development in preventing cardiovascular disease. Public Health Rep. 1992 Jan-Feb;107(1):32-7.

8. Alciati MH, Glanz K. Using data to plan public health programs: experience from state cancer prevention and control programs. Public Health Rep. 1996;111(2):165-72.

9. Mokdad AH, Remington PL. Measuring health behaviors in populations. Prev Chronic Dis. 2010;7(4):A75.

10. Canadian Health Services Research Foundation. Glossary of knowledge exchange terms used by CHSRF [Internet]. Ottawa (ON): CHSRF; [cited 2012 Sept 18]. Available from: http://74.81.206.232/PublicationsAndResources/ResourcesForResearchers/KEYS/GlossaryOfKnowledgeExchangeTerms.aspx

11. Graham ID, Logan J, Harrison MB, et al. Lost in knowledge translation: time for a map? J Contin Educ Health Prof. 2006; 26:13-24.

12. Jacobson N, Butterill D, Goering P. Development of a framework for knowledge translation: understanding user context. J Health Serv Res Policy. 2003;8(2):94-9.

13. Canadian Institutes of Health Research. More about knowledge translation at CIHR: knowledge transfer – definition [Internet]. Ottawa (ON): CIHR; [cited 2012 Sept 19]. Available from: http://www.cihr-irsc.gc.ca/e/39033.html

14. Best A, Moor G, Holmes B, et al. Health promotion dissemination and systems thinking: towards integrative model. Am J Health Behav. 2003;27(suppl 3):S206-16.

15. Yin RK. Case study research: design and methods. 4th ed. Thousand Oaks (CA): Sage Publications; 2009.

16. Halcomb EJ, Davidson PM. Is verbatim transcription of interview data always necessary? Appl Nurs Res. 2006;19(1):38-42.

17. Miles MB, Huberman AM. Qualitative data analysis: a sourcebook of new methods. Thousand Oaks (CA): Sage Publications; 1984.

18. Riley B, Harvey D. A Manitoba Integrated Knowledge System: for the Primary Prevention of Chronic Disease [Internet]. Manitoba (MB): Partners in Planning for Healthy Living; [cited 2013 Apr 5]. Available from: http://www.healthincommon.ca/wp-content/uploads/Manitoba-Integrated-Knowledge-System-Feb-2006.pdf

19. Sandelowski M, Barroso J. Classifying the findings in qualitative studies. Qual Health Res. 2003 Sept;13(7):905-23.

20. Stake RE. Multiple case study analysis. New York: The Guilford Press; 2006.

21. Walter I, Nutley S, Davies H. What works to promote evidence-based practice? A cross-sector review. Evid Policy. 2005;1(3): 335-64.

22. Rynes SL, Bartunek JM, Daft RL. Across the great divide: knowledge creation and transfer between practitioners and academics. Acad Manage J. 2001;44(2):340-55.

23. Jansen MW, van Oers HA, Kok G, de Vries NK. Public health: disconnections between policy, practice and research [Internet]. Health Res Policy Syst. 2010 [cited 2012 Mar 22];8(37). Available from: http://www.health-policy-systems.com/content/8/1/37

24. Wilson KM, Brady TJ, Lesesne C, on behalf of the NCCDPHP Work Group on Translation. An organizing framework for translation in public health: the knowledge to action framework. Prev Chronic Dis [Internet]. 2011 [cited 2012 Mar 22];8(2). Available from: http://www.cdc.gov/pcd/issues/2011/mar/10_0012.htm

25. Gagnon ML. Moving knowledge to action through dissemination and exchange. J Clin Epidemiol. 2011 Jan;64(1):25-31.

26. Lomas J. Decision support: a new approach to making the best healthcare management and policy choices. Healthc Q. 2007; 10(3):16-8.

27. Dobbins M, DeCorby K, Twiddy T. A knowledge transfer strategy for public health decision makers. Worldviews Evid Based Nurs. 2004;1(2):120-8.

28. Rosenbaum P. From research to clinical practice: considerations in moving research into people's hands. Personal reflections that may be useful to others. Pediatr Rehabil. 2005;8(3):165-71.

29. Williams A, Holden B, Krebs P, et al. Knowledge translation strategies in a community-university partnership: examining local Quality of Life (QoL). Soc Indic Res. 2008;85(1):111-25.

30. Broner N, Franczak M, Dye C, McAllister W. Knowledge transfer, policymaking and community empowerment: a consensus model approach for providing public mental health and substance abuse services. Psychiatr Q. 2001;72(1):79-102.

31. Ward V, Smith S, House A, Hamer S. Exploring knowledge exchange: a useful framework for practice and policy. Soc Sci Med. 2012 Feb;74(3):297-304.

32. Kiefer L, Frank J, Di Ruggiero E, et al. Fostering evidence-based decision-making in Canada: examining the need for a Canadian population and public health evidence centre and research network. Can J Public Health. 2005; 96(3):I1-40.

33. Dobbins M, Hanna SE, Ciliska D, et al. A randomized controlled trial evaluating the impact of knowledge translation and exchange strategies. Implement Sci. 2009;4:61.

34. Lavis J, Robertson D, Woodside J, et al. How can research organizations more effectively transfer research knowledge to decision makers? Milbank Q. 2003; 81(2):221-48.

Methodology of the 2009 Survey on Living with Chronic Diseases in Canada—hypertension component

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Abstract

Introduction: The Survey on Living with Chronic Diseases in Canada—hypertension component (SLCDC-H) is a 20-minute cross-sectional telephone survey on hypertension diagnosis and management. Sampled from the 2008 Canadian Community Health Survey (CCHS), the SLCDC-H includes Canadians (aged ≥ 20 years) with self-reported hypertension from the ten provinces.

Methods: The questionnaire was developed by Delphi technique, externally reviewed and qualitatively tested. Statistics Canada performed sampling strategies, recruitment, data collection and processing. Proportions were weighted to represent the Canadian population, and 95% confidence intervals (CIs) were derived by bootstrap method.

Results: Compared with the CCHS population reporting hypertension, the SLCDC-H sample (n = 6142) is slightly younger (SLCDC-H mean age: 61.2 years, 95% CI: 60.8–61.6; CCHS mean age: 62.2 years, 95% CI: 61.8–62.5), has more post-secondary school graduates (SLCDC-H: 52.0%, 95% CI: 49.7%–54.2%; CCHS: 47.5%, 95% CI: 46.1%–48.9%) and has fewer respondents on hypertension medication (SLCDC-H: 82.5%, 95% CI: 80.9%–84.1%; CCHS: 88.6%, 95% CI: 87.7%-89.6%).

Conclusion: Overall, the 2009 SLCDC-H represents its source population and provides novel, comprehensive data on the diagnosis and management of hypertension. The survey has been adapted to other chronic conditions—diabetes, asthma/chronic obstructive pulmonary disease and neurological conditions. The questionnaire is available on the Statistics Canada website; descriptive results have been disseminated by the Public Health Agency of Canada.

Keywords: *epidemiological survey, hypertension, chronic disease, data collection, health surveys, questionnaires, Canadian Community Health Survey*

Introduction

More than one in five Canadians aged over 20 years have been diagnosed with hypertension,[1,2] and a further 17% of the adult population may be unaware that they have the condition.[3] Elevated blood pressure is a major etiological factor for cardiovascular diseases, but it can be effectively controlled with lifestyle changes in physical activity, diet, sodium intake, alcohol use, weight management and tobacco use, or through pharmacotherapy, when required.[4] Despite this, about 33% of Canadians diagnosed with hypertension have blood pressure levels that are not well-controlled.[3] Improving the understanding of the knowledge, attitudes and behaviours of Canadians diagnosed with hypertension would support the development and the enhancement of programs for blood pressure control.

In 2009, the Public Health Agency of Canada (PHAC) conducted the Survey on Living with Chronic Diseases in Canada—hypertension component (SLCDC-H) to determine how Canadians live with and manage their hypertension. The 20-minute survey was the first survey administered to a nationally representative sample of Canadians diagnosed with a specific chronic condition, providing new variables that could be used to monitor and report on health-related indicators. This paper describes the objectives and methodology of the 2009 SLCDC-H and examines the representativeness of the final sample.

Methods

Survey objectives

PHAC initiated the SLCDC in 2006 to: (1) assess the impact of chronic conditions on quality of life of individuals and their families; (2) collect information on how people manage their chronic conditions; (3) identify the use of interventions for chronic condition management among people living in the community; (4) identify health behaviours that influence disease outcomes; and (5) examine barriers to self-management of chronic conditions. PHAC selected hypertension

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and arthritis for the first iteration of the SLCDC after taking into consideration the importance to public health, the existence of complementary national surveillance work, and the prevalence and sample size of several chronic conditions. After consulting with Statistics Canada, it was determined that ethics approval was not required for this survey because physical measures were not being taken. No privacy or confidentiality risks, as governed by the Privacy Impact Assessment policy, were identified, and the Chief Statistician of Statistics Canada allowed the survey to proceed.

Survey content development

In 2007, PHAC collaborated with the Canadian Hypertension Education Program (CHEP) to create a Working Group with expertise in hypertension or survey development and validation. The Working Group developed the telephone-administered questionnaire used in the cross-sectional survey. Questions were derived from publicly available population surveys including the core, theme and optional contents of the various cycles of the Canadian Community Health Survey (CCHS);[5] Cycle 4 of the Canadian National Population Health Survey (NPHS);[6] the blood pressure and cardiovascular disease questionnaires of the American National Health and Nutrition Examination Survey (NHANES) 2005–06;[7] the American Harris Interactive Survey—Hypertension Education (2007)[8] as well as surveys on specific content areas such as physical activity or diet. Peer-reviewed literature was consulted for other instruments and well-known scales, such as general self-efficacy scales or the Morisky medical adherence scale.[9-11] Certain questions on blood pressure management and monitoring were adapted based on consultations with experts and from existing national guidelines, including those by CHEP[12,13], the National Institutes of Health,[14] and the National Cholesterol Education Program in the United States.[15]

A preliminary review determined if questions were age- and population-appropriate, amenable to telephone administration and within the scope of the SLCDC while general enough to be reproducible to other chronic conditions and in future iterations. Using the CCHS as a guide, the retained questions were organized by theme and reformatted with a focus on sequencing and skip patterns, standardization of questions, categories and ranges, and consistent use of language and narrative point of view. Response bias was considered when removing leading or repetitive questioning. The time constraints of a telephone interview and respondent fatigue also dictated the length of the survey.

A Delphi panel approach was used to reach consensus on content. In general, those questions to which answers would be difficult to analyze or interpret were deleted. Similarly, those which would be difficult to translate into actionable recommendations were also deleted. This included concepts that (1) were already targeted on the main CCHS and thus obtainable through linkage (e.g. nutrition or physical activity); (2) were too lengthy to be adequately addressed (e.g. health utility, stages of change); (3) required detailed explanation (e.g. expectations of self-efficacy); or (4) would yield response categories too small to analyze. Final content of the English survey was translated into French to allow for implementation in Canada's two official languages, and translated content was verified for accuracy.

External review

Using a working draft of the questionnaire, 15 CHEP members (30% response rate) reviewed the survey and supplied detailed feedback, which was used to confirm key content areas and addressed potential gaps. Some of their recommendations were outside the scope of the survey, for example, 24-hour food recall, use of speciality clinics, ambulatory blood pressure monitoring, exploration of other macrovascular conditions, and global cardiovascular risk. However, other areas were added or expanded, including usefulness and availability of written educational material on hypertension, knowledge of key issues, and barriers to adherence to lifestyle changes.

Qualitative testing

Statistics Canada conducted qualitative testing of both English and French surveys for clarity, face validity, question flow, and ease of administration and response using a sub-sample of respondents with a current or past diagnosis of hypertension (regardless of pharmacotherapy for hypertension), randomly drawn from about 10 000 CCHS 2007 respondents. Every effort was made to obtain as diverse a sample as possible in terms of age, sex, level of education and income, and place of residence (city core versus greater metropolitan area). Verbal consent was obtained during screening, and the participants were informed that the interview would be recorded and staff would be observing them.

One hour was allotted for individual face-to-face interviews. Of the 16 interviews scheduled, 13 were successfully completed (eight in English, five in French). During the interview, staff made general observations on participants' reactions to the content and their willingness and ability to provide responses. The interviewers probed participants on their blood pressure measurements and adherence to medication, and also asked for their overall feedback on the content of the survey. Due to the small sample, results were used for their qualitative input and were not considered statistically representative. The time taken to administer the questionnaire averaged between 30 and 40 minutes, suggesting the need to reduce the content by an additional 15 minutes (dictated by the longer French version). Also, question order was revised to improve the flow, sensitive questions and reference periods were modified, language was simplified, terminology and translations were clarified and answer keys and skips were edited to better reflect actual responses.

Final questionnaire

The final 20-minute questionnaire included eight hypertension-specific modules (Table 1) as well as entry and exit components (totalling five minutes) and a general health module. The full questionnaire is available on the Statistics Canada website (www.statcan.gc.ca/imdb-bmdi/instrument/5160_Q4_V1-eng.htm). The final survey was implemented with a computer-assisted telephone interview

TABLE 1
Modules of the 2009 SLCDC-H questionnaire

SLCDC-H module[a]		Content focus	Number of questions[b]	Brief description
1	Survey introduction	Administrative	0	Provides the background and purpose of the survey to the respondent
2	General health	General	5	Eases the respondent into hypertension-specific questions by asking general questions about their current health status
3	Confirmation of high blood pressure diagnosis	Hypertension-specific	5	Authenticates that the respondent belongs to the target population and asks for the age at diagnosis
4	Blood pressure measurement	Hypertension-specific	9	Obtains information related to the respondent's most recent blood pressure measurement, including diastolic and systolic values, target readings, and whether the respondent has a plan for blood pressure control
5	Medication use	Hypertension-specific	9 (10)[c]	Focuses on overall pharmacotherapy, pharmacotherapy specific to hypertension and explores adherence patterns
6	Health care utilization	Hypertension-specific	7	Asks about the respondent's interactions with various health care professionals in the 12 months prior to survey administration
7	Clinical recommendations	Hypertension-specific	8	Documents the specific recommendations suggested by a health care professional to help control the respondent's high blood pressure
8	Self-management	Hypertension-specific	14 (22)[d]	Asks about the recommendations that were attempted, the status of self-management at the time of interview, and any barriers that the respondent experienced
9	Self-monitoring of blood pressure	Hypertension-specific	6	Focuses on blood pressure monitoring practices outside of the health care professional's office and what this information means to the respondent
10	Information and training	Hypertension-specific	8	Asks about hypertension-related information: who provides information, what sort of material/resources have been made available, and what material/resources the respondent would prefer to receive
11	Administration	Administrative	4	Wraps up the survey by obtaining permission for linkages and sharing

Abbreviations: CCHS, Canadian Community Health Survey; SLCDC-H, Survey on Living with Chronic Diseases in Canada – Hypertension Component.

[a] The 11 modules associated with the SLCDC-H are linked to the 2008 CCHS, resulting in a total of 87 modules available for analysis.

[b] The number of questions delivered to each respondent depends on skip patterns and the eligibility of the respondent for particular questions.

[c] Although 9 questions make up this module, one is split into two parts, resulting in a total of 10 questions.

[d] Although 14 questions make up this module, several are split into parts, resulting in a total of 22 questions.

(CATI) application, which facilitated consistent survey administration. The CATI application controlled the logical flow of questions, specified ranges for valid answers, identified minimum and maximum values for quantitative responses and provided standardized procedures for non-response.[16] End-to-end testing on the application was done in a simulated collection environment.

Target population

The target population for the SLCDC-H was the Canadian adult (≥ 20 years) population diagnosed with hypertension, with the CCHS used as the sampling frame. The CCHS is a cross-sectional national survey that has provided self-reported data on health status, health care utilization and health determinants in the Canadian population since 2000.[17-19] The SLCDC-H obtained detailed information on the population with hypertension, while permitting linkage back to the main CCHS for additional socio-demographic and risk factor data.

The eligible population for the 2009 SLCDC-H included Canadians living in privately occupied dwellings in the ten provinces. Residents of the three northern territories were not surveyed due to insufficient sample sizes, which lead to the inability to properly weight findings to represent all residents. Also excluded from the CCHS, and subsequently from the 2009 SLCDC-H, were full-time members of the Canadian Forces, people living on Indian reserves or Crown lands, and residents of institutions or of certain remote regions (together representing less than 2% of the target population).[16,18]

To identify the population for the SLCDC-H, a standard module in the CCHS that asks about chronic conditions diagnosed by a health care professional and lasting six months or more was used. Respondents who were 20 years of age or older who answered ''yes'' to the questions ''Do you have high blood pressure?'' or ''In the past month, have you taken any medicine for high blood pressure?'' (total of n = 17 437) were eligible.[16] Women with pregnancy-induced hypertension were excluded. Only the CCHS respondent, not the whole household, was eligible for selection. Proxy interviews were not permitted.

Sampling strategy

Sampling analyses were performed on several cycles of the CCHS during the development of the SLCDC. The multistage cluster sampling strategy applied in all these instances was similar. To begin, the raw unweighted data of all available respondents with hypertension were allocated to various domains (sex; age group: 20–44, 45–64, 65–74, ≥75 years; province; region: Atlantic, Quebec, Ontario, Prairies, British Columbia), and combinations thereof, to ensure that final numbers would be sufficient by these key domains. Administration of the survey to this full population was not feasible. In addition, some of these respondents were required for the arthritis component of the SLCDC; because both surveys were to be delivered concurrently, respondents allocated to one questionnaire became ineligible for the other, regardless of whether they had both conditions. As such, the raw data were filtered within each domain to create a raw sample of respondents with hypertension available for the SLCDC-H. During this process, sample allocation was based on relative proportions of arthritis and hypertension in the main survey to ensure that cell sizes for both surveys were large enough to analyze. In some domains, the full raw counts were retained to ensure a sufficient sample.

Subsequently, the raw sample was again adjusted, this time taking into account probable sample loss. The response rate was estimated to be 70%, allotting about 10% of loss to failure in recruitment or from the denial of permission for sharing/tracing of data and the other 20% due to non-response. Based on this, each domain was adjusted by a factor of 0.70. This produced the number of respondents expected to be available for survey administration and was the basis for further sampling analyses below.

At the onset of SLCDC development, the 2005 CCHS file was used to determine the feasibility of obtaining sufficient samples for both arthritis and hypertension surveys concurrently. Analyses focused on estimating the minimum sample size required to produce reliable estimates by domain. For hypertension, the minimum sample size was determined to be 1324, assuming a fixed design effect of 2.8 for age group and sex, where the sample variance was about 2.8 times larger than it would have been if the survey was based on random selection. For province and region, the fixed design effect was set to 3. Results from this sampling analysis confirmed that sufficient populations were available for independent surveys on arthritis and hypertension.

Closer to survey administration, the 2007 CCHS file was used to estimate the reportability of findings. The goal was to determine the minimum prevalence required, by domain, to achieve a pre-set coefficient of variation (CV) of 16.5%. Although the maximum CV is typically set at 33.3%, beyond which data would be considered unreportable, the CV was targeted to a more conservative 16.5% or less so as to provide reliable estimates. Based on this analysis, estimates would be reliable for most age groups and by sex, but only national or regional estimates would be reportable.

Finally, to identify the eligible 2009 SLCDC-H population, respondents were pulled from verified data in the 2008 CCHS file. Numbers of selected eligible respondents were inflated where possible (from n ≈ 6000 respondents to n = 9055) to lessen the effect of non-response and out-of-scope cases. Additional details, including a distribution of the eligible sample by domain, can be found at http://www.statcan.gc.ca/imdb-bmdi/document/5160_D5_T1_V1-eng.htm.

Recruitment, data collection and processing

Recruitment for the 2009 SLCDC-H began in mid-January 2009 with the mailing of introductory letters to selected respondents, followed by telephone interviews. Measures taken to maximize response rates included mailing supportive letters, offering convenient interview times, tracing respondents who moved or had invalid phone numbers, and providing the interview in either French or English, depending on the respondent's preference.[16] The interviewers were required to disclose the survey title, purpose and authority, that the survey was voluntary and that respondent confidentiality was protected. Respondents provided informed verbal consent to participate.

Data collection began in February 2009 and lasted three months. Between April and December 2009, data underwent processing, estimation and documentation. For respondents who agreed to link and share the surveys, the 2009 SLCDC-H was linked to the 2008 CCHS. To preserve respondent confidentiality, all personal identifiers were removed from the share-link file. Data were ready for use in December 2009 and were made available to PHAC, Health Canada and provincial health ministries. Researchers and third parties are able to access master files through university-based Research Data Centres run by Statistics Canada (http://www.statcan.gc.ca/rdc-cdr/process-eng.htm).

Data analysis

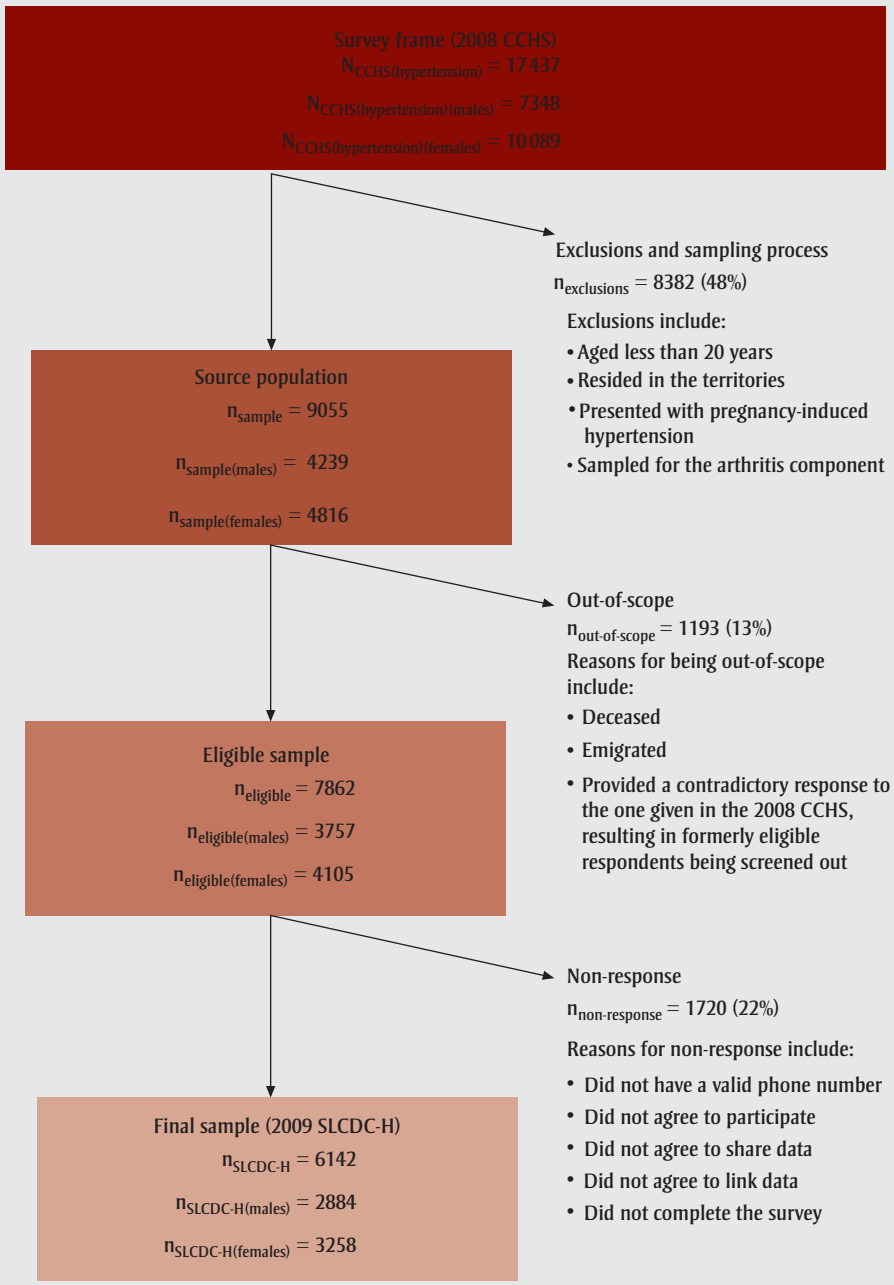
For estimates to be representative of the target population, survey weights were derived. Based on the final SLCDC-H sample, weight values corresponded to the number of people in the Canadian population represented by each respondent. Survey weights and bootstrap replicates were further adjusted to account for out-of-scope cases, non-responses and cases in which the respondent did not agree to share their data.[16,20] To compare the characteristics of respondents with hypertension between the two surveys, the 2008 CCHS population was limited to adults aged 20 years or older, and excluded the territories and pregnant women. Estimates were weighted using appropriate weights for each survey, and the bootstrap resampling method was applied to derive confidence intervals (CIs) using SAS Enterprise Guide version 4 (SAS Institute Inc., Cary, NC, US). Data reporting was subject to reliability guidelines stipulated by Statistics Canada regarding rounding and sampling error.[16]

Results

Final sample population

Figure 1 illustrates the flow of respondent participation in the 2009 SLCDC-H. A total of 17 437 respondents who reported being diagnosed with high blood pressure in the

FIGURE 1
Respondent participation in the 2009 SLCDC-H[a]



Abbreviations: CCHS, Canadian Community Health Survey; SLCDC-H, Survey on Living with Chronic Diseases in Canada – Hypertension Component.

[a]Numbers are unweighted.

2008 CCHS formed the survey frame. Anticipated loss between the survey frame and the final 2009 SLCDC-H sample included loss based on pre-set exclusion criteria (aged < 20 years; resided in the territories; presented with pregnancy-induced hypertension only) and from contacted cases who were found to be out-of-scope (deceased; emigrated; false positive; false negative). In this case, the proportion of out-of-scope cases in the SLCDC-H (13%) exceeded anticipated estimates (10%), largely due to a misclassification of respondents. False positives occurred if respondents pooled for the SLCDC-H later claimed not to have high blood pressure; among other reasons, this could have been due to an emphasis on a diagnosis by a health care professional, eliminating those who self-diagnosed their condition or misinterpreted the original question. False negatives may have resulted in a loss of respondents who actually had hypertension, but who

responded ''no'' to screening questions; among other reasons, this could have been intentionally done to avoid participation in the SLCDC-H.

During the development, administration or processing of the survey, eligible respondents may have also been lost if they (1) were pooled into the sample for the arthritis component; (2) were unwilling to be contacted again after responding to the 2008 CCHS; (3) were repeatedly absent for the interview; (4) refused to respond to the survey; or (5) refused linkages or use of their data. The hit rate, or the eligible sample that was contacted for interview (n = 7862) as a proportion of the source population (n = 9055), varied from 75.2% in men aged 20 to 44 years to 93.1% in those aged 65 to 74 years.[16] Similarly, the hit rate in women was lowest in the youngest age group (51.1%) and highest in the 65- to 74-year age group (94.7%).[16] The response rate, or the final sample who completed the survey (n = 6142) as a proportion of the eligible sample (n = 7862), varied from a low in the 20- to 44-year age group (men: 65.6%; women: 71.7%) to a high in the 65- to 74-year age group (men: 79.7%; women: 82.1%).[16] The final achieved sample available for analysis was 6142, representing an overall response rate of 78.1%.

Population characteristics

Table 2 shows selected socio-demographic and health characteristics of respondents aged 20 years or older reporting hypertension in the 2008 CCHS share file compared to the population of the 2009 SLCDC-H. The 2009 SLCDC-H was a representative sample of the CCHS population for ethnicity, body mass index, smoking status, self-reported diabetes, availability of a regular medical doctor, and number of medical consultations in the past year. A few indicators were significantly different (i.e. p value < 0.05; CIs did not overlap). The SLCDC-H population had a mean age of 61.2 years (95% CI: 60.8–61.6) compared with 62.2 years (95% CI: 61.8–62.5) in the 2008 CCHS, a higher proportion of respondents with post-secondary graduation (SLCDC-H: 52.0%, 95% CI: 49.7%–54.2%; CCHS: 47.5%, 95% CI: 46.1%–48.9%), and a smaller proportion of respondents reporting pharmacotherapy for hypertension (SLCDC-H: 82.5%, 95% CI: 80.9%–84.1%; CCHS: 88.6%, 95% CI: 87.7%–89.6%). Significant differences based on a p value of less than .05 were seen for some categories within other variables, including male sex, poor/fair self-rated health, ''active'' physical activity level, income, and self-reported heart disease and stroke. However, in these instances, CIs overlapped and the ratio of proportions was close to 1 (ranging from 0.87 to 1.27).

Survey response characteristics

An unweighted frequency analysis found that most questions had less than 1% missing data (not shown). Although ''don't know'' (DK) and ''refusal'' (R) options were allowed on most questions, these response categories were not read aloud. Questions with a higher prevalence of DK, R, or ''not stated'' answers were clustered around themes. For instance, respondents were asked to report their systolic and diastolic blood pressure levels. Poor recollection was expected, and 18.0% and 22.3% of respondents did not state a valid answer for systolic and diastolic blood pressure levels, respectively. Nevertheless, these questions were intentionally administered to provide baseline information on awareness of and knowledge about hypertension at the population level.

Most response ranges and distribution by category were reasonable. However, the most prevalent response for some general health questions with a five-category response scale (''excellent''; ''very good''; ''good''; ''fair''; ''poor'') was ''good,'' that is, a central tendency. This suggests that the format of some scales could have contributed to neutral answers.

Discussion

The presented sample survey covers a wide range of issues affecting Canadians with hypertension, such as awareness of blood pressure levels, self-monitoring practices, clinical recommendations, pharmacotherapy, and strategies for and barriers to self-management. The SLCDC-H has generated several findings to date, and has quantified a robust profile of Canadians with hypertension.

Specific findings included a high level of antihypertensive pharmacotherapy in Canada (82.5% of adults with hypertension), with an additional 10% of the population controlling their hypertension by changes in lifestyle alone.[21,22] For those controlling their hypertension with medication, neither an increasing number of medications nor the frequency of dosing were associated with non-adherence.[21] Strategies based on lifestyle change were reported by an impressive number of respondents—the majority—but less than half performed these actions consistently, and a disconcerting proportion reported not receiving advice from their health care professional about lifestyle change strategies.[22-24] Further, Gee et al.[24] noted that barriers to ceasing negative health behaviours differed from barriers to initiating positive behaviours.

Profiles of higher risk sub-groups were generated, including a description of those at risk of not engaging in lifestyle behaviour changes or those less likely to monitor their blood pressure outside of a health care professional's office.[24,25] Various negative impacts were associated with a respondent's sense of poor control over their hypertension and when a health care professional does not offer advice or education on lifestyle management.[23,26] Findings such as these provide direction for targeted interventions.

Overall, the 2009 SLCDC-H represents its source population, though respondents to the SLCDC-H are somewhat younger, better educated, and less likely to be pharmacologically treated for their hypertension. The effects of this potential selection bias may be that data represent a newly diagnosed, potentially healthier group, living with hypertension for a shorter period of time. Based on p values alone, other statistically significant differences exist, but CIs overlap and the relative magnitude of one proportion compared to the other is close to 1. In short, despite significant p values, meaningful differences may not exist and users

TABLE 2
Comparison of characteristics between source population with hypertension (2008 CCHS) and respondents to the 2009 SLCDC-H

	Population with hypertension, ≥ 20 years				*p* value[d]	Ratio CCHS:SLCDC-H
	2008 CCHS (N = 13 896[a])		2009 SLCDC-H (N = 6142)			
	n[b]	%[c] (95% CI)	n[b]	%[c] (95% CI)		
Sex						
Male	5961	48.2 (47.1–49.4)	2884	46.7 (45.1–48.4)	.03[e]	1.03
Age, years						
20–44	982	10.1 (9.2–11.1)	629	11.2 (10.1–12.2)	.04[e]	0.90
45–64	5411	45.4 (44.1–46.7)	2025	48.0 (46.2–49.8)	.0009[e]	0.95
≥ 65	7503	44.5 (43.4–45.6)	3484	40.8 (39.2–42.4)	< .0001[f]	1.09
Mean		62.2 (61.8–62.5)		61.2 (60.8–61.6)	< .0001[f]	1.02
Ethnicity						
White	12 535	85.3 (83.8–86.8)	5676	86.8 (84.6–89.0)	.13	0.98
Aboriginal off-reserve	419	2.4 (2.0–2.7)	174	2.1 (1.6–2.6)	.35	1.14
Other	629	12.4 (10.9–13.8)	261	11.0 (8.9–13.2)	.20	1.13
Education level						
< Secondary school graduation	4419	25.9 (24.7–27.2)	1798	23.3 (21.5–25.1)	.001[e]	1.11
Secondary school graduation	2170	16.8 (15.7–17.9)	961	17.6 (15.7–19.4)	.37	0.95
Some post-secondary	772	6.2 (5.4–6.8)	358	7.2 (5.9–8.4)	.06	0.86
Post-secondary graduation	6177	47.5 (46.1–48.9)	2988	52.0 (49.7–54.2)	< .0001[f]	0.91
Total household income, $						
< 15,000	1247	7.1 (6.4–7.8)	473	6.1 (5.0–7.2)	.04[e]	1.16
15,000–29,999	3106	19.4 (18.3–20.5)	1410	19.5 (17.7–21.4)	.85	0.99
30,000–49,999	2846	21.9 (20.7–23.1)	1351	20.0 (18.2–21.7)	.02[e]	1.10
50,000–79,999	2526	24.8 (23.2–26.3)	1255	23.7 (21.6–25.8)	.29	1.05
≥ 80,000	2100	26.8 (25.3–28.4)	1058	30.7 (28.0–33.4)	.0007[e]	0.87
Self-rated health						
Poor/fair	3861	27.1 (25.8–28.4)	1431	25.1 (22.8–27.4)	.04[e]	1.08
Good	5271	38.4 (37.0–39.8)	2370	39.7 (37.2–42.2)	.25	0.97
Very good/excellent	4728	34.5 (33.1–36.0)	2335	35.2 (32.9–37.6)	.49	0.98
BMI, kg/m^2						
< 25 (under/normal weight)	3873	29.8 (28.4–31.2)	1792	28.5 (26.5–30.6)	.17	1.05
25–29 (overweight)	5103	39.3 (37.8–40.8)	2415	38.4 (36.1–40.8)	.42	1.02
≥ 30 (obese)	4098	30.9 (29.6–32.3)	1805	33.0 (30.6–35.4)	.05	0.94
Physical activity level						
Active	2286	16.8 (15.8–17.8)	1177	18.5 (16.9–20.1)	.02[e]	0.91
Moderately active	3157	22.8 (21.6–23.9)	1490	23.2 (21.3–25.0)	.61	0.98
Inactive	8022	56.8 (55.4–58.2)	3472	58.4 (56.2–60.5)	.11	0.97
Smoking status						
Current daily	1984	14.1 (13.2–15.0)	842	14.0 (12.5–15.5)	.90	1.01
Current occasional	311	2.3 (1.8–2.8)	149	3.1 (2.1–4.2)	.08	0.74
Non-smoker	11 564	83.2 (82.2–84.3)	5149	82.9 (81.1–84.7)	.64	1.00
Co-morbidities						
Diabetes	2830	20.3 (19.1–21.5)	1172	19.2 (17.0–21.3)	.21	1.06
Heart disease	2590	16.3 (15.4–17.3)	1077	14.7 (13.0–16.4)	.03[e]	1.11
Effects of stroke	627	3.8 (3.3–4.2)	223	3.0 (2.4–3.6)	.006[e]	1.27

Continued on the following page

TABLE 2 (continued)
Comparison of characteristics between source population with hypertension (2008 CCHS) and respondents to the 2009 SLCDC-H

	Population with hypertension, ≥ 20 years				*p* value[d]	Ratio CCHS:SLCDC-H
	2008 CCHS (N = 13 896[a])		2009 SLCDC-H (N = 6142)			
	n[b]	%[c] (95% CI)	n[b]	%[c] (95% CI)		
Medical care						
Has a regular medical doctor	13 179	94.9 (94.3–95.6)	5825	95.1 (94.2–96.0)	.72	1.00
Takes medication for high blood pressure	12 717	88.6 (87.7–89.6)	5171	82.5 (80.9–84.1)	<.0001[f]	1.07
Mean number of medical consultations in the past year		5.6 (5.4–5.8)		5.7 (5.3–6.0)	.74	0.98

Abbreviations: BMI, body mass index; CCHS, Canadian Community Health Survey; SLCDC-H, Survey on Living with Chronic Diseases in Canada – Hypertension Component; CI, confidence interval.

[a] CCHS data are based on the share file. The sample of n = 13 896 in this table does not match the sample of n = 17 437 for the survey frame (Figure 1) because exclusions were applied in this case (age < 20 years; residents of territories; people with pregnancy-induced hypertension). Further, in this case, individuals with arthritis are retained, whereas in Figure 1 some respondents may have later been removed for the arthritis component.

[b] Numbers are unweighted.

[c] Proportions are based on weighted numbers to reflect the Canadian population living in the ten provinces.

[d] *p* values are based on *z* tests to determine significant differences between the two ratios.

[e] Statistically significant differences based on $p < .05$. However, it should be noted that CIs overlap and the difference between populations is small.

[f] Statistically significant difference based on $p < .05$; CIs do not overlap.

should decide whether this may impact their analyses.

Strengths and limitations

On a broader scope, the 2009 SLCDC-H was developed to be nationally representative. However, the representativeness of the data to the Canadian population may be limited due to the exclusion of the territories and other populations. Administrative data have shown that the age-standardized incidence rate of hypertension in the Yukon is far above the Canadian average (37.7 per 1000 population versus 25.8), but that the age-standardized prevalence rate is lower (17.9% versus 19.6%).[1,2] It would be interesting to explore hypertension diagnosis and management in the Yukon. Moreover, other potentially excluded populations (e.g. specific ethnic groups) would have likely presented with different characteristics.[27] Since the SLCDC-H was only administered in two languages, it may have excluded some of the 493 (1.7%; unweighted) participants who originally responded to the 2008 CCHS in a language other than English and French. Oversampling of vulnerable and/or ethnic populations is encouraged for future surveys.

A well-known limitation of self-reported surveys is that they are subject to various sampling and non-sampling errors, such as response bias, recall bias and non-differential misclassification. Since the objective of the survey was to understand hypertension management in those aware of their condition, the target population was based on individuals who self-reported a diagnosis of hypertension, excluding those with undiagnosed hypertension. Although the majority of Canadians with hypertension (83%) are aware of their condition,[3] the accuracy of self-reported hypertension status remains unclear. Individuals without actual diagnosis may report having the condition (false positive) while individuals who have their hypertension controlled may not report themselves as having hypertension (false negative). However, the rate of misclassification is likely lower in the SLCDC-H given that many of these cases were identified during the screening process.

Attempts were made to identify whether lifestyle changes were attributable to a diagnosis of hypertension. Nevertheless, lifestyle changes can be influenced by a number of factors outside of such a diagnosis. Another limitation of this survey is that, while linkage to the CCHS for additional variables improved efficiency, participant characteristics may have changed in the time between the surveys (averaging 8.5 months),[26] leading to potential misclassification. Statistics Canada has taken measures to reduce survey errors, such as using the CATI system and extensive training of interviewers to minimize non-response. Specific to the SLCDC-H, the Lawson Health Research Institute has initiated a validation study to perform test-retest comparisons of the questionnaire in two populations with hypertension.

Conclusion

The 2009 SLCDC-H provides novel, comprehensive data on the diagnosis of hypertension and management mechanisms used by Canadians with self-reported high blood pressure. Based on the success of the first iteration of the SLCDC, the methodology and content have since been adapted to two subsequent cycles of the survey (diabetes and asthma/chronic obstructive pulmonary disease), with data released in 2011.[28] The methodology was also adapted for the Survey on Living with Neurological Conditions in Canada, with data released in late 2012.[29] It is anticipated that these data will create opportunities for new research, influence policy development and guide strategies to improve chronic disease prevention and control in Canada.

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References

1. Public Health Agency of Canada. Report from the Canadian Chronic Disease Surveillance System: hypertension in Canada, 2010 [Internet]. Ottawa (ON): Chronic Disease Surveillance Division; 2011 [cited 2011 Mar 3]. Available from: http://www.phac-aspc.gc.ca/cd-mc/cvd-mcv/ccdss-snsmc-2010/pdf/CCDSS_HTN_Report_FINAL_EN_20100513.pdf

2. Robitaille C, Dai S, Waters C, et al. Diagnosed hypertension in Canada: incidence, prevalence, and associated mortality. CMAJ. 2012 Jan;184(1):E49-56.

3. Wilkins K, Campbell NR, Joffres MR, et al. Blood pressure in Canadian adults. Health Rep. 2010 Mar;21(1):37-46.

4. Rabi DM, Daskalopoulou SS, Padwal RS, et al. The 2011 Canadian Hypertension Education Program recommendations for the management of hypertension: blood pressure measurement, diagnosis, assessment of risk, and therapy. Can J Cardiol. 2011 Jul-Aug;27(4):415-33.e1-2.

5. Other reference periods – Canadian Community Health Survey – annual component [Internet]. Ottawa (ON): Statistics Canada; 2011 [cited 2011 Mar 25]. Available from: http://www.statcan.gc.ca/cgi-bin/imdb/p2SV.pl?Function=getInstanceList&SurvId=3226&SurvVer=1&InstaId=15282&SDDS=3226&lang=en&db=imdb&adm=8&dis=2

6. National Population Health Survey – household component – longitudinal (NPHS) [Internet]. Ottawa (ON): Statistics Canada; 2011 [updated 2011 Jul 20; cited 2011 Mar 23]. Available from: http://www.statcan.gc.ca/cgi-bin/imdb/p2SV.pl?Function=getSurvey&SDDS=3225&lang=en&db=imdb&adm=8&dis=2

7. National Health and Nutrition Examination Survey – NHANES 2005-2006 [Internet]. Atlanta (GA): Centers for Disease Control and Prevention; 2011 [updated 2011 Apr 29; cited 2011 Mar 23]. Available from: http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/nhanes05_06.htm

8. Moser M, Franklin SS. Hypertension management: results of a new national survey for the hypertension education foundation: Harris interactive. J Clin Hypertens. 2007 May;9(5):316-23.

9. Reis JP, Dubose KD, Ainsworth BE, Macera CA, Yore MM. Reliability and validity of the occupational physical activity questionnaire. Med Sci Sports Exerc. 2005 Dec;37(12):2075-83.

10. Morisky DE, Green LW, Levine DM. Concurrent and predictive validity of a self-reported measure of medication adherence. Med Care. 1986 Jan;24(1):67-74.

11. Grace SL, Barry-Bianchi S, Stewart DE, Rukholm E, Nolan RP. Physical activity behavior, motivational readiness and self-efficacy among Ontarians with cardiovascular disease and diabetes. J Behav Med. 2007 Feb;30(1):21-9. Epub 2006 Nov 16.

12. Quinn RR, Hemmelgarn BR, Padwal RS, et al. The 2010 Canadian Hypertension Education Program recommendations for the management of hypertension: part I - blood pressure measurement, diagnosis, and assessment of risk. Can J Cardiol. 2010 May;26(5):241-8.

13. Hackam DG, Khan NA, Hemmelgarn BR, et al. The 2010 Canadian Hypertension Education Program recommendations for the management of hypertension: part 2 – therapy. Can J Cardiol. 2010 May;26(5):249-58.

14. National Heart, Lung, and Blood Institute. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure (JNC7) [Internet]. Bethesda (MD): U.S. Department of Health and Human Services; 2003 [cited 2011 Mar 23]. Available from: http://www.nhlbi.nih.gov/guidelines/hypertension/

15. National Heart, Lung, and Blood Institute. Third report of the Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III) [Internet]. Bethesda (MD): U.S. Department of Health and Human Services; 2003 [cited 2011 Mar 23]. Available from: http://www.nhlbi.nih.gov/guidelines/cholesterol/

16. Statistics Canada. Documentation - Survey on Living with Chronic Diseases in Canada - user guide – 2009 [Internet]. 2010 [updated 2011 Nov 29; cited 2011 Apr 12]. Available from: http://www23.statcan.gc.ca/imdb-bmdi/pub/document/5160_D5_T1_V1-eng.htm

17. Canadian Community Health Survey – annual component (CCHS) [Internet]. Ottawa (ON): Statistics Canada; 2010 [updated 2011 Jun 9; cited 2011 Mar 3]. Available from: http://www.statcan.gc.ca/cgi-bin/imdb/p2SV.pl?Function=getSurvey&SDDS=3226&lang=en&db=imdb&adm=8&dis=2

18. Béland Y. Canadian Community Health Survey – methodological overview. Health Rep. 2002 Mar;13(3):9-14.

19. Desmeules M. Appendix A: overview of National Population Health and Canadian Community Health Surveys. BMC Women's Health. 2004 Aug 24;4(Suppl I):S35.

20. Rao JN. Bootstrap methods for analyzing complex sample survey data. Proceedings of Statistics Canada International Symposium Series. Symposium 2006: Methodological Issues in Measuring Population Health; 2006; catalogue no. 11-522-XIE.

21. Gee ME, Campbell NR, Gwadry-Sridhar F, et al.; Outcomes Research Task Force of the Canadian Hypertension Education Program. Antihypertensive medication use, adherence, stops and starts in Canadians with hypertension. Can J Cardiol. 2012 May; 28(3):383-9.

22. Hypertension [Internet]. Ottawa (ON): Public Health Agency of Canada; [updated 2009 Dec 10; cited 2011 Mar 29]. Available from: http://www.phac-aspc.gc.ca/cd-mc/slcdcfs-epamccfi/hypertension-eng.php

23. Walker RL, Gee ME, Bancej C, et al. Health behaviour advice from health professionals to Canadian adults with hypertension: results from a national survey. Can J Cardiol. 2011 Jul-Aug;27(4):446-54.

24. Gee ME, Bienek A, Campbell NRC, et al. Prevalence of, and barriers to, preventive lifestyle behaviours in hypertension (from a national survey of Canadians with hypertension). Am J Cardiol. 2012 Feb;109(4): 570-5.

25. Bancej CM, Campbell N, McKay DW, Nichol M, Walker R, Kaczorowski J. Home blood pressure monitoring among Canadian adults with hypertension: results from the 2009 Survey on Living with Chronic Diseases in Canada. Can J Cardiol. 2010 May;26(5): e152-7.

26. Gee ME, Campbell NR, Bancej CM, et al. Perception of uncontrolled blood pressure and behaviours to improve blood pressure: findings from the 2009 Survey on Living with Chronic Diseases in Canada. J Hum Hypertens. 2011 Mar; 26(3):188-95; doi:10.1038/jhh.2011.5.

27. Chiu M, Austin PC, Manuel DG, Tu JV. Comparison of cardiovascular risk profiles among ethnic groups using population health surveys between 1996 and 2007. CMAJ. 2010 May;182(8):E301-10.

28. Other reference periods - Survey on Living with Chronic Diseases in Canada (SLCDC) [Internet]. Ottawa (ON): Statistics Canada; 2011 [cited 2011 Mar 22]. Available from: http://www.statcan.gc.ca/cgi-bin/imdb/p2SV.pl?Function = getInstanceList&SurvId = 38899&SurvVer = 2&InstaId = 38900&SDDS = 5160&lang = en&db = imdb&adm = 8&dis = 2

29. Survey on Living with Neurological Conditions in Canada [Internet]. Ottawa (ON): Statistics Canada; 2012 [cited 2012 Apr 13]. Available from: http://www.statcan.gc.ca/survey-enquete/household-menages/5182-eng.htm

How we identify and count Aboriginal people—does it make a difference in estimating their disease burden?

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Abstract

Introduction: We examined the concordance between the Canadian Community Health Survey (CCHS) ''identity'' and ''ancestry'' questions used to estimate the size of the Aboriginal population in Canada and whether the different definitions affect the prevalence of selected chronic diseases.

Methods: Based on responses to the ''identity'' and ''ancestry'' questions in the CCHS combined 2009–2010 microdata file, Aboriginal participants were divided into 4 groups: (A) identity only; (B) ancestry only; (C) either ancestry or identity; and (D) both ancestry and identity. Prevalence of diabetes, arthritis and hypertension was estimated based on participants reporting that a health professional had told them that they have the condition(s).

Results: Of participants who identified themselves as Aboriginal, only 63% reported having an Aboriginal ancestor; of those who claimed Aboriginal ancestry, only 57% identified themselves as Aboriginal. The lack of concordance also differs according to whether the individual was First Nation, Métis or Inuit. The different method of estimating the Aboriginal population, however, does not significantly affect the prevalence of the three selected chronic diseases.

Conclusion: The lack of concordance requires further investigation by combining more cycles of CCHS to compare discrepancy across regions, genders and socio-economic status. Its impact on a broader list of health conditions should be examined.

Introduction

The great disparities in health outcomes between Aboriginal people in Canada and other Canadians are well documented in research studies and in governmental agency and Aboriginal organization reports.[1-3] A major problem in assessing the health of Aboriginal people in Canada is identifying the population denominator, a fundamental requirement in any epidemiological study.

The Constitution of Canada recognizes Aboriginal people as First Nations, Inuit and Métis. Among First Nations, the Indian Act further defines whether the person is ''status'' or ''non-status,'' and residing ''on-reserve'' or ''off-reserve.'' Over the decades, Statistics Canada has changed the approach it uses in the Census and in various other surveys.[4] In brief, it has used two concepts, that of ''identity'' (i.e. does the individual consider himself or herself to be an Aboriginal person) and ''ancestry'' or ''origin'' (i.e. does the individual have an ancestor who was an Aboriginal person). This dual approach has been a source of some confusion in estimating the size and composition of the Aboriginal population.

The objective of our study was to determine if the dual definition of who is an Aboriginal person affects the estimates of disease burden. We analyzed the Canadian Community Health Survey (CCHS), an important source of information on the health of Canadians and of Canadian communities and regions that is regularly conducted by Statistics Canada.[5,6] The CCHS excludes reserves in its sampling but does include the northern territories; as a result, for the First Nations population the CCHS is generalizable only to the off-reserve population.

Methods

We used the CCHS 2009–2010 combined file available at the Research Data Centre of Statistics Canada at the University of Toronto. CCHS identifies Aboriginal people using two questions:

- SDC_Q4: ''To which ethnic or cultural groups did your ancestors belong? (For example: French, Scottish, Chinese, East Indian).'' Interviewers were instructed to mark all the answers that apply. Among the choices available were ''North American Indian,'' ''Métis'' and ''Inuit,'' but no single ''Aboriginal'' category. In this paper, we refer to this as the ''ancestry question.''
- SDC_Q4_1: ''Are you an Aboriginal person, that is, North American Indian, Métis or Inuit?'' This is fol-

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lowed by SDC_Q4_2: ''Are you North American Indian?'', ''Are you Métis?'' and ''Are you Inuit?'' In this paper, we refer to this as the ''identity question.''

In this study, we defined various groups based on the responses to these two questions as follows:

- Group A: Those who answered only the identity question in the affirmative (ancestry = no and identity = yes)
- Group B: Those who answered only the ancestry question in the affirmative (ancestry = yes and identity = no)
- Group C: Those who answered either the ancestry question or the identity question in the affirmative (ancestry = yes or identity = yes)
- Group D: Those who answered to both questions in the affirmative (ancestry = yes and identity = yes).

Those who answered ''don't know,'' ''refused'' and ''not stated'' were considered as not having either Aboriginal ancestry or identity.

We compared the prevalence of chronic diseases among the different Aboriginal groups defined by the ''ancestry'' question versus those defined by the ''identity'' question. We selected diabetes, arthritis and hypertension for analysis. Individuals were classified as having a chronic disease if they answered ''yes'' to the CCHS questions on diagnoses made by a health professional.

All analyses were carried out using SAS version 9.3 (SAS Institute Inc., Cary, NC, US). Because the CCHS has a complex sampling design, estimates and standard errors were obtained using the weighted bootstrap method as per Statistics Canada guidelines.[7] To obtain counts and prevalences of chronic diseases for each Aboriginal ancestry and/or identity group, the sample weights and the 500 bootstrap weights supplied by Statistics Canada were used in the SAS procedure PROC SURVEYFREQ.

Results

Cross-tabulations of the counts of Aboriginal people in Canada based on the identity question and the ancestry question show that the two populations do not completely overlap (see Table 1).

TABLE 1
Size of Aboriginal population in Canada based on the ancestry[a] and identity[b] questions in CCHS 2009–2010

		Ancestry[a]		
		Yes	No	Total
Identity[b]	Yes	582 789	336 377	919 166
	No	433 891	27 384 067	
	Total	1 016 680		28 737 123

Abbreviation: CCHS, Canadian Community Health Survey.

Note: Shaded cells refer to individuals who reported EITHER Aboriginal ancestry OR Aboriginal identity.

[a] Those CCHS participants who responded ''North American Indian,'' ''Métis'' or ''Inuit'' to the ancestry question, ''To which ethnic or cultural groups did your ancestors belong? (For example: French, Scottish, Chinese, East Indian).''

[b] Those CCHS participants who responded in the affirmative to the identity question, ''Are you an Aboriginal person, that is, North American Indian, Métis or Inuit?'' followed by one of the following: ''Are you North American Indian?'', ''Are you Métis?'' or ''Are you Inuit?''

Based on responses to the ancestry question, there were 1 016 679 Aboriginal people in Canada (3.5% of the Canadian population), whereas using the identity question there were 919 166 Aboriginal people (3.2% of the Canadian population). Of the 919 166 individuals who identified themselves as Aboriginal, only 582 789 (63.4%) reported an Aboriginal ancestor. Of the 1 016 680 individuals who claimed Aboriginal ancestry, only 582 789 (57.3%) actually identified themselves as Aboriginal. Individuals who claimed Aboriginal ancestry AND identified themselves as Aboriginal (n = 582 789) made up 43.1% of those who EITHER claimed Aboriginal ancestry OR identified themselves as Aboriginal (1 353 056, the sum of the shaded cells in Table 1).

TABLE 2
Size of First Nations, Métis and Inuit populations in Canada based on the ancestry and identity questions in CCHS 2009–2010

	First Nations	Métis	Inuit
Population, n			
(A) Identity only[a]	446 701	414 697	35 288
(B) Ancestry only[b]	727 627	264 510	38 825
(C) Either[c]	870 934	483 185	48 124
(D) Both[d]	303 394	196 022	25 989
Proportion, %			
(A)/(C)	51.3	85.8	73.3
(B)/(C)	83.5	54.7	80.7
(D)/(C)	34.8	40.6	54.0
(D)/(A)	67.9	47.3	73.6
(D)/(B)	41.7	74.1	66.9

Abbreviation: CCHS, Canadian Community Health Survey.

[a] Those CCHS participants who responded in the affirmative only to the identity question, ''Are you an Aboriginal person, that is, North American Indian, Métis or Inuit?'' followed by one of the following: ''Are you North American Indian?'', ''Are you Métis?'' or ''Are you Inuit?'' (ancestry = no and identity = yes).

[b] Those CCHS participants who responded ''North American Indian,'' ''Métis'' or ''Inuit'' only to the ancestry question, ''To which ethnic or cultural groups did your ancestors belong? (For example: French, Scottish, Chinese, East Indian)''(ancestry = yes and identity = no).

[c] Those CCHS participants who answered to either the ancestry question or the identity question in the affirmative (ancestry = yes or identity = yes).

[d] Those CCHS participants who answered to both in the affirmative (ancestry = yes and identity = yes).

The lack of concordance between the two methods of counting Aboriginal people also differed according to whether the individual was First Nation, Métis or Inuit (see Table 2).

Table 3 shows the crude prevalence estimates (and 95% confidence interval) for diabetes, arthritis and hypertension between the non-Aboriginal and Aboriginal population as variously defined. The major differences are between the Aboriginal population, however defined, and the non-Aboriginal population. The different methods of defining the Aboriginal population have little impact on the magnitude of the chronic disease estimates.

Discussion

Redressing health disparities between Aboriginal and non-Aboriginal people in Canada is an important policy objective of governmental agencies, Aboriginal organizations and health care providers. Accurate assessment of both the population denominator and disease burden is a prerequisite in defining the scope of the problem. However, there is a lack of concordance in responses to the identity question and the ancestry question in the Census (personal communication, Paul Peters, Statistics Canada, 31 October, 2011), the reasons for which are poorly understood. In that aspect, we demonstrated differences between the First Nations, Métis and Inuit populations. There could well also be differences between regions, genders and socio-economic status. We wish to alert users of Statistics Canada health surveys to the discrepancy. Further investigation is warranted, which will require merging even more cycles of CCHS than we had done, or using Census data.

Conclusion

It is reassuring that the prevalence estimates of three chronic diseases (self-reported diabetes, arthritis and hypertension) do not differ significantly between those based on the identity question and those based on the ancestry

TABLE 3
Crude prevalence of selected chronic diseases based on self-report in CCHS 2009–2010

	Population, n	Cases, n	Prevalence, %	95% CI
Diabetes				
Non-Aboriginal	27 371 441	1 679 098	6.1	5.9–6.4
Aboriginal				
Identity only[a]	918 849	67 799	7.4	6.3–8.4
Ancestry only[b]	1 015 718	71 371	7.0	6.1–8.0
Either identity or ancestry[c]	1 352 095	94 321	7.0	6.1–7.9
Both identity and ancestry[d]	582 472	44 848	7.7	6.5–8.9
Arthritis				
Non-Aboriginal	26 618 055	4 103 368	15.4	15.2–15.8
Aboriginal				
Identity only[a]	873 695	161 251	18.5	16.7–20.2
Ancestry only[b]	978 118	165 383	16.9	15.3–18.5
Either identity or ancestry[c]	1 296 515	228 474	17.6	16.2–19.1
Both identity and ancestry[d]	555 299	98 161	17.7	15.6–19.8
Hypertension				
Non-Aboriginal	27 320 981	4 703 035	17.2	16.9–17.5
Aboriginal				
Identity only[a]	911 895	114 689	12.6	11.3–13.9
Ancestry only[b]	1 009 344	130 005	12.9	11.6–14.2
Either identity or ancestry[c]	1 344 813	169 462	12.6	11.5–13.7
Both identity and ancestry[d]	576 426	75 232	13.1	11.5–14.6

Abbreviations: CCHS, Canadian Community Health Survey; CI, confidence interval.

[a] Those CCHS participants who responded in the affirmative to only the identity question, ''Are you an Aboriginal person, that is, North American Indian, Métis or Inuit?'' (ancestry = no and identity = yes).

[b] Those CCHS participants who responded in the affirmative to the ancestry question, ''To which ethnic or cultural groups did your ancestors belong? (For example: French, Scottish, Chinese, East Indian)'' (ancestry = yes and identity = no).

[c] Those CCHS participants who answered either the ancestry question or the identity question in the affirmative (ancestry = yes or identity = yes).

[d] Those CCHS participants who responded in the affirmative to both the identity question and the ancestry question (ancestry = yes and identity = yes).

question. All show the same relationship relative to non-Aboriginal people, confirming studies done using the CCHS[5,6] and other surveys such as the Aboriginal Peoples Survey.[8] Whether other chronic diseases vary according to the method of ascertaining the Aboriginal population denominator remains to be investigated.

References

1. Waldram JB, Herring DA, Young TK. Aboriginal health in Canada: historical, cultural, and epidemiological perspectives. 2nd edition. Toronto (ON): University of Toronto Press; 2006.

2. Health Canada. Statistical profile on the health of First Nations in Canada. Ottawa (ON): First Nations and Inuit Health Branch, Health Canada; 2011 [cited 2012 Jan 9]. Available from: http://www.hc-sc.gc.ca/fniah-spnia/intro-eng.php

3. Health indicators of Inuit Nunangat within the Canadian context: 1994-1998 and 1999-2003. Ottawa (ON): Inuit Tapiriit Kanatami; 2010 [cited 2012 Jan 9]. Available from: http://www.itk.ca/sites/default/files/20100706Health-Indicators-Inuit-Nunangat-EN.pdf

4. Statistics Canada. How Statistics Canada identifies Aboriginal people. Ottawa (ON): Statistics Canada; 2007 [cited 2012 Jan 9] [Statistics Canada, Catalogue No.: 12-592-XIE]. Available from: www.statcan.gc.ca/pub/12-592-x/12-592-x2007001-eng.pdf

5. Lix LM, Bruce S, Sarkar J, Young TK. Risk factors and chronic conditions among Aboriginal and non-Aboriginal populations. Health Rep. 2009;20(4):21-9.

6. Sarkar J, Lix LM, Bruce S, Young TK. Ethnic and regional differences in prevalence and correlates of chronic diseases and risk factors in northern Canada. Prev Chronic Dis. 2010;7(1):A13.

7. Canadian Community Health Survey (CCHS) annual component: user guide 2010 and 2009-2010 Microdata files. Ottawa (ON): Statistics Canada; 2011 Jun [cited 2012 Jan 9]. Available from: www23.statcan.gc.ca/imdb-bmdi/document/3226_D7_T9_V8-eng.pdf

8. Ng C, Chatwood S, Young TK. Arthritis in the Canadian Aboriginal population: north-south differences in prevalence and correlates. Chronic Dis Can. 2010;31:22-6.

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