Chronic Diseases in Canada

Volume 31 • Number 1 • December 2010

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Public Health Agency of Canada
Agence de la santé publique du Canada
Canada

Chronic Diseases in Canada
a publication of the Public Health Agency of Canada

Sue Gordon is a printmaker/painter living in Winnipeg, Manitoba. She has two children and a beautiful granddaughter who are a real source of inspiration for her.

Her artwork was chosen to illustrate this special joint issue on Aboriginal health because of its focus on fostering well-being through local foods. She created this monoprint with mixed media for the Manitoba Association of Community Health, Diabetes Prevention Project for Children (1999-2000). At that time, she worked on a project for kids who live mainly in the North. She was illustrating small books and cards to encourage a healthy lifestyle among the families that would receive them. She wished to show the energy and spirit of the kids, as well as some of the nutritious foods that could be part of their harvesting and cooking routines.

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

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Indexed in Index Medicus/MEDLINE, SciSearch® and Journal Citation Reports/ Science Edition

To promote and protect the health of Canadians through leadership, partnership, innovation and action in public health.
— Public Health Agency of Canada

Published by authority of the Minister of Health.

ISSN 0228-8699

This publication is also available online at www.publichealth.gc.ca/cdic
Également disponible en français sous le titre : *Maladies chroniques au Canada*

INTRODUCTION
Chronic Diseases in Canada and *Preventing Chronic Disease:* copublishing on health in Aboriginal populations*

* This article is part of a joint publication initiative between *Chronic Diseases in Canada* and *Preventing Chronic Disease*. *Chronic Diseases in Canada* is the secondary publisher, while *Preventing Chronic Disease* is the primary publisher. The primary publication can be found at Morrison H, Posner SF. Chronic Diseases in Canada and Preventing Chronic Disease: copublishing on health in Aboriginal populations. Prev Chronic Dis. 2011;8(1). http://www.cdc.gov/pcd/issues/2011/jan/10_0238.htm

The January 2011 issue includes 6 papers that are copublished by *Chronic Diseases in Canada (CDIC)* and *Preventing Chronic Disease (PCD)*. In this example of copublishing, each journal is the primary publisher of 3 of the papers and secondary publisher of the other 3. Copublication is uncommon among scientific journals; however, it does offer an opportunity for journals to reach a broader readership with information about areas of common interest. The International Committee of Medical Journal Editors identifies this model of publishing as appropriate when 2 journals with different readerships are publishing information about topics that may appeal to both.

The idea of copublishing a collection of articles was born of discussions between the 2 journals about how to improve quality, reach and impact. Both journals have similar missions of publishing research and best practices in public health in the United States and Canada. The premise of copublishing is based on the understanding that public health research and practice are global, crossing the physical barriers of national borders. Public health professionals in Canada and the United States face many of the same challenges in developing and implementing programs to prevent and manage chronic diseases. Copublishing of scientific papers helps demonstrate how public health is a global issue and allows both journals to reach a broader audience interested in chronic disease prevention and control.

In March 2010, the opportunity to copublish became a reality when we identified a set of papers on chronic disease prevention and control among Canadian Aboriginal people.

Copublishing requires more coordination than when either journal publishes independently. Editing styles, publication schedules and multilingual translations are just a few of the elements that need to be coordinated. We appreciate the efforts of the staff of both journals in collaborating effectively. This collection is translated into 3 common languages spoken in the United States and Canada—French, Spanish, and English—reflecting our effort to make the collection available to a broad range of researchers. The authors are key partners in the copublishing effort; without their agreement, this initiative would not have been possible.

The collection includes 4 original research papers as well as an editorial by Malcolm King, PhD, scientific director of the Canadian Institutes of Health Research's Institute of Aboriginal Peoples' Health and a member of the Mississaugas of the New Credit First Nation (Ontario). Dr King's career spans 30 years; he is responsible for advancing Aboriginal public health research in Canada and has a clear understanding of the role of social determinants of health in achieving overall health. It is an honor to have him pen the editorial that preceded this collection. This special issue is also preceded by our introduction, as editors-in-chief of the 2 journals. Two of the 4 original research papers (Bruce et al. and Riediger et al.) are companion studies that involve a Manitoba First Nation community. Bruce et al. looked at obesity in this population and the related comorbidities—dyslipidemia, hypertension, and diabetes. The other 2 papers (Ng et al. and Tjepkema et al.) are national studies. Ng and colleagues studied arthritis in the Canadian Aboriginal population, focusing on the differences between Canada's northern territories and the 10 provinces to the south. Tjepkema compared mortality patterns and rates among the urban-dwelling Canadian Aboriginal population and other urban residents.

These papers report data from Canadian Aboriginal people, but others have reported similar findings among US American Indian/Alaska Native people. These and other studies show a substantial burden of risk factors and chronic diseases in these populations. The articles document the need for public health interventions to address chronic disease prevention and management. Ng and colleagues note regional differences in arthritis. This finding is an example of the importance of recognizing that Aboriginal people are not a monolithic population and that different groups will require different interventions. Moving forward, development and evaluation of interventions are needed. As with any other high-risk group, documenting the burden is insufficient. Action to address the issues is where public health will improve the well-being of the populations it serves.

This landmark copublishing effort is a first for both *PCD* and *CDIC* and an innovation in the world of scholarly publication. With this effort, we are able to reach a broader range of researchers. Our initiative has the power to facilitate information sharing and discussion among researchers in this field. We hope that the readership of both journals will find joint publication useful and that it will be a model for further publishing efforts. In addition to the scientific discussion, we are interested in feedback on copublishing.

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Chronic diseases and mortality in Canadian Aboriginal peoples: learning from the knowledge*

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* This article is part of a joint publication initiative between *Preventing Chronic Disease* and *Chronic Diseases in Canada*. *Preventing Chronic Disease* is the secondary publisher, while *Chronic Diseases in Canada* is the primary publisher.

It is a sad fact that Canada's Aboriginal people, whether living in rural communities or in urban centers, have a significantly lower life expectancy than non-Aboriginal Canadians.[1] The gap in health status of Canada's Aboriginal peoples is a matter of ongoing concern;[2] recognizing and understanding the social determinants of health is key to understanding the difference in health status and, in my view, key to achieving success in addressing and correcting this problem. However, it is important to realize that there are unique social determinants for Aboriginal peoples associated with their cultures, histories and colonization, and the current social, economic, political and geographic context.[3]

Bruce et al.[4] examined obesity and the related comorbidities—dyslipidemia, hypertension and diabetes—in a Manitoba First Nations community. As in other Aboriginal populations (e.g. in neighbouring Saskatchewan[5]), they found the prevalence of obesity and these comorbidities is higher in women than in men, and that the comorbidities are common even in young adults. Undiagnosed hypertension is also common. In the same Manitoba First Nations community, Riediger et al.[6] found a high cardiovascular risk due to low plasma apolipoprotein A1 levels, particularly among women.

Ng et al.[7] studied arthritis in the national Aboriginal population, focusing on the differences between the northern territories and the 10 provinces in the south. They used data from the 2006 Aboriginal Peoples Survey (APS), a post-census survey conducted by Statistics Canada that includes health, social and economic questions. The APS covers Inuit and Métis, as well as First Nations living outside of Indian reserves; on-reserve First Nations are covered by the 2002/03 Regional Longitudinal Health Survey conducted by the Assembly of First Nations.[8] Because of oversampling of northern residents in the APS, effective comparisons can be made with the more populated south. Self-reported arthritis and rheumatism is the most common medical condition reported by Aboriginal people in Canada. In the south, where more than 90% of the Aboriginal population live, the overall prevalence is 20.1% compared with 25.3% for on-reserve First Nations in the same geographic area.[8] The prevalence of arthritis or rheumatism in the north is considerably less (12.7% overall), and is lower for both First Nations and Inuit in comparison with the south. A higher proportion of individuals with arthritis report at least one other chronic disease, and more people with arthritis consult health professionals, while fewer—perhaps not surprising—are employed, although cause and effect was not established.

Tjepkema et al.[1] looked at mortality of urban Aboriginal adults over an 11-year period (1991–2001), linking mortality registry data with census data and data on tax filers to achieve an urban Aboriginal cohort of some 25 500 among a total urban cohort of 2.6 million. The main variable, remaining life expectancy at age 25 years, is significantly lower for Aboriginal people living in urban centres than for urban non-Aboriginal Canadians. For men, the life expectancy is 4.7 years lower; for women, 6.5 years. These differences are about the same as the overall figures that include on-reserve and non-urban residents recently published by the same researchers;[9] these also show the same gender bias, i.e. the gap in life expectancy is greater for Aboriginal women by about 2 years.[9] The researchers found that the two leading causes of mortality in Aboriginal Canadians are circulatory system diseases and cancers, the former being elevated for both men and women compared with the non-Aboriginal population, the latter only for Aboriginal women.[1] Many specific causes of death are remarkably elevated, particularly alcohol-related causes and external causes, including suicides and accidents; deaths amenable to medical intervention are elevated in both men and women.[1]

These descriptions and understandings of the health gap and risk factors for chronic diseases in Canada's Aboriginal populations are important, but we need to understand all the causes and then move on to interventions that will address, prevent and reverse the problems. The four studies described here all take their fields of interest from the mere descriptive to understanding the physical, socioeconomic and societal risk factors. The next step, and perhaps the crucial one, is how to use the knowledge to correct the disparity. The study by Bruce et al.[4] ultimately offers hope: the community is engaged in projects to address, prevent and perhaps even reverse the otherwise inevitable increasing obesity predicted by the risk factors. Few details are given, but we are told that a project aiming to prevent gestational diabetes through controlling weight gain during pregnancy is underway; that the community operates a fitness center; that the health center offers education on diet, exercise and wellness; and that walking groups have been organized, as well as activity programs in the schools. In all of this, the investigators continue to work with the community. It is unfair to expect the same degree of follow-up with national survey data because action to utilize health information and transform behaviours and

conditions generally requires local community engagement. However, it is important to work with national organizations to transform the health knowledge into action and policy.

The Canadian Institutes of Health Research (CIHR) embraces the need to reduce health inequities in Aboriginal peoples as one of its main strategic directions.[10] The CIHR Institute of Aboriginal Peoples' Health (IAPH) supports community engagement, capacity building and network development as key platforms towards achieving health equity for Aboriginal peoples, and will continue to do so. But we need to move beyond this into the realms of intervention research (what worked, what didn't, and why) and knowledge translation (transformation to different contexts and scaling up) to define the models of good practice that will enable our communities to achieve their goals of health equity. Since health equity can never be achieved without the wholeness that includes the mental, physical, emotional and spiritual parts of our lives, we must go beyond the conventional social determinants of health to look at factors that include promotion of resilience through spirituality, culture, language revitalization, traditional activities and other forms of cultural connectedness.[3] With follow-up by researchers and with local, regional and national community engagement, we hope to eventually eliminate the gap in health status that divides Canada's Aboriginal peoples from the non-Aboriginal mainstream.

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Mortality of urban Aboriginal adults in Canada, 1991–2001*

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* This article is part of a joint publication initiative between *Preventing Chronic Disease* and *Chronic Diseases in Canada*. *Preventing Chronic Disease* is the secondary publisher, while *Chronic Diseases in Canada* is the primary publisher.

Abstract

Objective: To compare mortality patterns for urban Aboriginal adults with those of urban non-Aboriginal adults.

Methods: Using the 1991–2001 Canadian census mortality follow-up study, our study tracked mortality to December 31, 2001, among a 15% sample of adults, including 16 300 Aboriginal and 2 062 700 non-Aboriginal persons residing in urban areas on June 4, 1991. The Aboriginal population was defined by ethnic origin (ancestry), Registered Indian status and/or membership in an Indian band or First Nation, since the 1991 census did not collect information on Aboriginal identity.

Results: Compared to urban non-Aboriginal men and women, remaining life expectancy at age 25 years was 4.7 years and 6.5 years shorter for urban Aboriginal men and women, respectively. Mortality rate ratios for urban Aboriginal men and women were particularly elevated for alcohol-related deaths, motor vehicle accidents and infectious diseases, including HIV/AIDS. For most causes of death, urban Aboriginal adults had higher mortality rates compared to other urban residents. Socio-economic status played an important role in explaining these disparities.

Conclusion: Results from this study help fill a data gap on mortality information of urban Aboriginal people of Canada.

Keywords: *Aboriginal people, First Nations, Métis, Inuit, North American Indians, age-standardized mortality rates, mortality rate, life expectancy*

Introduction

The number of Aboriginal people (First Nations, Métis and Inuit) living in urban Canada has increased dramatically over the last half-century; in 1950, about 7% resided in urban Canada,[1] but by 2006 that figure had risen to 54%.[2] However, the amount of health research on urban Aboriginal people is not proportional to their weight in the total population;[3,4] nor does it reflect their increasing proportion within the total Aboriginal population.

Aboriginal people choose to live in urban areas for various reasons, including family reasons, employment opportunities, education, training and health (for example, to be closer to medical services);[5,6] they face different challenges to their rural counterparts, such as finding adequate housing and locating existing services and support to assist them in the transition.[5,7]

Although it is widely known that, compared to other Canadians, Aboriginal people experience a disproportionate burden of death and disease,[8-12] specific information for those residing in urban areas is less well known.[13] Similarly, while overall life expectancy for First Nations, Métis and Inuit is considerably shorter than that of the general population,[14-18] mortality indicators for Aboriginal people residing in urban Canada are difficult to estimate because Aboriginal identifiers are not reported on death registrations in most provinces. Mortality patterns for Registered Indians living in Manitoba and British Columbia have been analysed and provide results for sub-provincial regions including Winnipeg[19] and Vancouver.[20] However, these studies only show part of the picture, as they exclude First Nations not registered under the *Indian Act*, as well as Métis and Inuit, and they provide no information specific to Aboriginal people living in other urban areas of Canada.

The 1991–2001 Canadian census mortality follow-up study provides an opportunity to examine patterns of mortality for a reasonably large number of Aboriginal people living in urban areas *at the beginning of the follow-up period* in all provinces and territories, regardless of whether they were registered under the *Indian Act*.

The objectives of this study are (1) to determine to what extent urban Aboriginal adults may be at risk of premature mortality; (2) to calculate life expectancy and probability of survival to age 75 years; and (3) to identify the causes of death with the highest risk.

Author References

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Methods

Data sources

The Canadian census mortality follow-up study consists of a 15% sample (n = 2 735 152) of the non-institutionalized population of Canada aged 25 years or older, all of whom were enumerated via the 1991 census long-form questionnaire. This cohort was tracked for mortality from June 4, 1991, to December 31, 2001. Briefly, the creation of the census mortality database required two linkages because the electronic files of census data contained no names, but names were needed to find the corresponding deaths. Using common variables such as date of birth, postal code, plus spousal date of birth (if applicable), the census file was first probabilistically linked to an encrypted name file abstracted from non-financial tax-filer data. Then this census plus encrypted name file was matched to the Canadian Mortality Database using probabilistic record linkage methods[21]—an approach similar to that used for other mortality follow-up studies at Statistics Canada.[22] Complete details of the construction and contents of the linked file are reported elsewhere.[16]

Eligibility

Only people who were enumerated by the 1991 census long-form questionnaire, were 25 years old by census day, and were Canadian residents were eligible to be part of the cohort. Data quality reports estimated that the 1991 census missed 3.4% of Canadian residents of all ages. The missed individuals were more likely to be young, mobile, low income, of Aboriginal ancestry[23,24] or homeless. Only cohort members living in urban areas (defined below) on census day were in the scope of this study. The long-form census questionnaire is usually given to one in five Canadian households, to all residents of Indian Reserves, to all residents of many remote and northern communities and to all residents of non-institutional collective dwellings. In addition, it was necessary to obtain encrypted names from tax filer data (the name file), as only tax filers could be followed for mortality. However, there were no major differences in demographic and socio-economic characteristics between eligible census respondents and those successfully linked to the name file (Appendix Tables A, B and C).

Analytical techniques

For each member of the cohort, we calculated person-days of follow-up from the beginning of the study (June 4, 1991) to the date of death or emigration (ascertained from the name file and known for 1991 only) or the end of study (December 31, 2001). To calculate person-years at risk, we divided person-days of follow-up by 365.25.

Using the total Aboriginal[†] cohort population structure (person-years at risk) as the standard population, we used age- and sex-specific mortality rates by 5-year age groups (at baseline) to calculate age-standardized mortality rates (ASMRs) for subgroups of the population. We calculated corresponding 95% confidence intervals (CIs) for the ASMRs as described by Carrière and Roos,[25] and used a similar method to calculate CIs for the ASMR ratios (RRs).

For age-specific analyses, cohort members were categorized by 10-year age groups at baseline from 25-to-34 to 65-to-74, and 75 or older. Most analyses used age at baseline (June 4, 1991), while life table analyses used age at the beginning of each year of follow-up.

Based on Chiang's method,[26] we calculated period life tables for each sex, with corresponding standard errors and 95% CIs. We calculated these after converting from age at baseline to age at the beginning of each year of follow-up, and then calculated deaths and person-years at risk separately for each year (or partial year) of follow-up. We then pooled deaths and person-years at risk by age at the beginning of each year of follow-up, before calculating the life tables.

We calculated Cox proportional mortality hazard ratios by sex, first controlling for age (in years) and then controlling for place of residence (metropolitan areas, smaller urban centres), lone parent (yes, no), education (less than high school diploma, high school diploma, post-secondary diploma, university degree), income quintile (1–5), occupation skill level (professional, managerial, skilled-technical-supervisory, semi-skilled, unskilled, no occupation), work status (employed, unemployed, not in labour force) and place of birth (Canada or elsewhere). Place of birth was included in the models to reduce the "healthy immigrant effect" among non-Aboriginal cohort members. Note that detailed definitions of these variables (all ascertained only at baseline) have been previously described.[16] We interpreted differences in excess mortality between the age-adjusted model and the fully adjusted model as estimates of the effect of the socio-economic variables (place of residence, lone parent, education, etc., as listed above) on the extent of the disparities between urban Aboriginal adults and urban non-Aboriginal adults. The proportion of excess mortality attributed to the socio-economic variables was calculated as follows: the difference between age-adjusted and fully adjusted hazard ratios for Aboriginal person (yes/no), divided by the age-adjusted hazard ratio minus 1.

The underlying cause of death of those who died in the period 1991 through 1999 was coded based on the World Health Organization's *International Classification of Diseases, Ninth Revision* (ICD-9)[27] and those who died in 2000 or 2001 based on the *Tenth Revision* (ICD-10).[28] For analyses by cause of death, deaths were grouped by ICD-9 chapter, categories within chapters, and by risk factors (smoking-related, alcohol-related, drug-related, or amenable to medical intervention).[29,30] This data is presented in Appendix Table D.

Definitions

The 1991 census did not collect information on self-identification with an Aboriginal group (North American Indian, Métis or Inuit). For our analysis, we defined this population on the basis of two questions, offering three distinct dimensions of Aboriginality:

[†] Anyone who indicated North American Indian, Métis or Inuit ancestry, Registered Indian status or membership in a North American Indian band or First Nation in the long-form census questionnaire—see Definitions.

1. Ancestry: Question 15 from the long-form census questionnaire asked respondents to check from a list of 15 the ethnic or cultural group(s) their ancestors belonged to, including North American Indian, Métis and Inuit/Eskimo.[31] Respondents were instructed to specify as many as applicable.

2. Registered Indian status: Question 16 from the long-form census questionnaire asked "Is this person a Registered Indian as defined by the *Indian Act* of Canada?" (Yes, No).

3. Indian band/First Nation membership: Question 16 also asked respondents to write down the name of the Indian band or First Nation to which they belonged.

For our study, a person was considered Aboriginal if they reported a single Aboriginal—and no other—ancestry or two or more Aboriginal ancestries (with or without any non-Aboriginal ancestry), or if they reported that they were a Registered Indian or member of an Indian band or First Nation. Based on an analysis of 1996 census data where ethnic origins were cross-classified by Aboriginal identity,[32] over 94% of 1996 census participants who met these ancestry-based definitions self-identified as Aboriginal. The number of Aboriginal people (especially Métis) would be underestimated in our study since persons reporting one Aboriginal ancestry but at least one non-Aboriginal ancestry were considered as non-Aboriginal (unless they indicated being a Registered Indian or member of an Indian band or First Nation).

"Urban areas" can be defined differently depending on the research question and data availability,[33] and our definition differs from the standard census definition.[31] We defined "urban areas" as any census metropolitan area ("metropolitan areas," with a population of ≥ 100 000) or census agglomeration ("smaller urban centres," with a population of ≥ 10 000), excluding reserves or other Aboriginal settlements within those areas. Other urban areas were out of the scope of this study.

Cohort members, linkage rates, deaths and person-years at risk

Appendix Table A shows that there were 2.6 million eligible census respondents in urban areas of Canada, including 25 500 Aboriginal adults. Linkage rates to the name file (comparing cohort members to long-form census respondents) for urban Aboriginal people (61% for men and 66% for women) were lower than for the urban non-Aboriginal population (80% for men and 76% for women). Despite the lower linkage rate, the demographic and socio-economic characteristics of urban Aboriginal cohort members were generally similar to those of all eligible (in-scope) urban Aboriginal adults in the weighted census population, with the following exceptions: persons who were employed, those with higher household income adequacy, and those who were married were slightly more likely to be successfully linked (a similar finding to that for non-Aboriginal cohort members), suggesting that our sample of urban Aboriginal people was not biased with respect to those characteristics (Appendix Tables B and C).

Based on deaths in 1991, which could be identified independently in the Canadian Mortality Database and/or the name file, we estimated that ascertainment of deaths in the cohort followed for mortality (1991–2001) was about 97% overall and about 95% to 96% among Aboriginal people.

Overall, the cohort followed for mortality included 16 300 urban Aboriginal adults who accounted for 166 570 person-years at risk and 1126 deaths during the 11-year follow-up period (Appendix Table A).

Results

According to the 1991 census, there were an estimated 259 800 Aboriginal persons aged 25 or older, representing 1.5% of the total adult population of Canada. About 45% lived in urban areas (30% in metropolitan areas, 15% in smaller urban centres). In comparison, 78% of non-Aboriginal persons lived in urban areas (62% in metropolitan areas, 16% in smaller urban centres). In all urban areas taken together, 69% of the Aboriginal population were First Nations (40% Registered Indians, 29% non-status Indians), 28% Métis and 3% Inuit.

There were 16 300 Aboriginal cohort members residing in either a metropolitan area or a smaller urban centre at the beginning of the follow-up period (June 4, 1991). Table 1 shows the demographic and socio-economic characteristics for Aboriginal and non-Aboriginal cohort members residing in urban Canada. Almost three-quarters of Aboriginal cohort members were aged 25 to 44 years compared to 54% for non-Aboriginal adults. About 44% of Aboriginal adults had less than a high school diploma (31% for non-Aboriginal adults) and 61% were in the two lowest income adequacy quintiles (36% for non-Aboriginal adults).

Remaining life expectancy at age 25 years and probability of survival from ages 25 to 75 years

For urban Aboriginal adults of both sexes, remaining life expectancy at age 25 years (conditional on surviving to age 25 years) was substantially shorter compared to urban non-Aboriginal adults. Table 2 shows that life expectancy at age 25 years for urban Aboriginal men was 48.1 years (95% CI: 47.1-49.1), compared to 52.8 years (95% CI: 52.8-52.9) for urban non-Aboriginal men, a difference of 4.7 years. Life expectancy at age 25 years for urban Aboriginal women was longer than that for urban Aboriginal men, but the gap between the life expectancy of urban Aboriginal women (52.7 years; 95% CI: 51.7-53.7) and urban non-Aboriginal women (59.2 years; 95% CI: 59.2-59.3) was larger (6.5 years). Life expectancy for Aboriginal adults residing in metropolitan areas was similar to that for Aboriginal adults residing in smaller urban centres.

Table 2 also shows the probability of survival to age 75 years, conditional on survival to age 25 years, for urban cohort members. About 52% (95% CI: 48-56) of urban Aboriginal men were expected to survive to age 75 years compared to 65% (95% CI: 64-65) of urban non-Aboriginal men, a difference of 12 percentage points. For urban Aboriginal women, 63% (95% CI: 59-66) were expected to survive to age 75 years, compared to 80% (95% CI: 79-80) for urban non-Aboriginal women, a difference of 17 percentage points.

Age-specific and age-standardized mortality rates

Table 3 shows age-specific and age-standardized mortality rate ratios (RRs) for urban Aboriginal adults compared to urban non-Aboriginal adults. Overall, rate ratios were significantly higher for urban Aboriginal men (RR = 1.56; 95% CI: 1.43-1.70) and women (RR = 1.94; 95% CI: 1.78-2.11) compared to urban non-Aboriginal men and women. For urban Aboriginal adults of both sexes, rate ratios were highest in the younger age groups and diminished with advancing age.

Causes of death

Table 4 shows ASMRs by major causes of death for urban Aboriginal cohort members while Table 5 shows ASMRs by major causes of death for urban non-Aboriginal cohort members. Among urban Aboriginal men, the most common causes of death were circulatory system diseases (accounting for 33% of the total ASMR), followed by all cancers (23%) and external causes (16%)—a similar ranking to that for urban non-Aboriginal men. For urban Aboriginal women, the most common causes of death were circulatory system diseases (29% of the total ASMR), followed by all cancers (26%), external causes (10%) and digestive system diseases (9%); for urban non-Aboriginal women, cancer was the most common cause of death (42%), followed by circulatory system diseases (29%), respiratory system diseases (6%) and external causes (6%).

Table 5 shows age-standardized rate ratios (RRs) by major causes of death. (The corresponding number of deaths and ASMRs are shown in Table 4 and Appendix Table D.) Rate ratios for urban Aboriginal men were substantially elevated for deaths due to circulatory system diseases (RR = 1.50; 95% CI: 1.29-1.74) such as ischemic heart disease (RR = 1.52; 95% CI: 1.26-1.83), but not for all cancers combined (RR = 1.09; 95% CI: 0.92-1.30); however, the rate ratio was elevated for deaths due to trachea, bronchus and lung cancer (RR = 1.42; 95% CI: 1.08-1.88) especially for Aboriginal men living in metropolitan areas at the beginning of the follow-up period. Rate ratios for urban Aboriginal men were particularly elevated for digestive system diseases (RR = 3.00; 95% CI: 2.09-4.30), all external causes of death (RR = 2.80; 95% CI: 2.29-3.43)—notably motor vehicle accidents (RR = 3.51; 95% CI: 2.32-5.32) and, to a lesser extent, suicides (RR = 1.57; 95% CI: 1.04-2.38)—as well as for deaths due to infectious diseases (RR = 2.04; 95% CI: 1.33-3.11) including HIV/AIDS (RR = 2.03, CI: 1.22-3.39). With some exceptions (such as endocrine system diseases and suicide), rate ratios for Aboriginal men residing in metropolitan areas were similar to rate ratios for Aboriginal men residing in smaller urban centres.

Rate ratios for urban Aboriginal women were elevated for almost all major causes of death except breast cancer. For example, rate ratios were elevated for circulatory system diseases (RR = 1.93; 95% CI: 1.64-2.28) and all cancers combined (RR = 1.21; 95% CI: 1.03-1.42), the two most common causes of death. Rate ratios were particularly elevated for deaths due to infectious diseases (RR = 5.76; 95% CI: 3.68-9.01) including HIV/AIDS (RR = 10.65; 95% CI: 4.56-24.88), digestive system diseases (RR = 4.82; 95% CI: 3.67-6.34), external causes (RR = 3.37; 95% CI: 2.59-4.37)—notably motor vehicle accidents (RR = 4.13; 95% CI: 2.46-6.93)—and endocrine system diseases such as diabetes mellitus (RR = 2.61; 95% CI: 1.73-3.94). With some exceptions, rate ratios for Aboriginal women residing in metropolitan areas were higher than for Aboriginal women residing in smaller urban centres.

In Table 5, deaths are also categorized as smoking-related, alcohol-related, drug-related or amenable to medical care.[28,29] Compared to urban non-Aboriginal men and women, rates for smoking-related causes (accounting for 15% and 7% of the total ASMR among urban Aboriginal men and women respectively) were elevated for urban Aboriginal men (RR = 1.46; 95% CI: 1.17-1.82) and women (RR = 1.36; 95% CI: 1.04-1.78). Rates for alcohol-related causes were considerably higher for urban Aboriginal men (RR = 4.55; 95% CI: 3.14-6.61) and women (RR = 11.44; 95% CI: 8.02-16.34), and rates for drug-related deaths were also significantly higher for Aboriginal men (RR = 3.71; 95% CI: 2.22-6.22) and women (RR = 6.43; 95% CI: 4.26-9.73). Rates of premature death (before the age of 75 years) due to causes considered amenable to medical intervention (for example, those due to breast and cervical cancer, infectious diseases, cerebrovascular disease, pneumonia or influenza) were also significantly higher for urban Aboriginal adults of both sexes.

Within the urban Aboriginal adult population, men were more likely than women to die from smoking-related causes (ASMR = 130 per 100 000 person-years at risk versus 58), but less likely to die from causes amenable to medical care (69 versus 92), a similar pattern to that of the non-Aboriginal population. The risks of dying from alcohol-related causes were slightly elevated for urban Aboriginal men compared to urban Aboriginal women (42 versus 34), a different pattern than for the non-Aboriginal population, where men had a much higher risk than did women (9 versus 3) (Table 4, Appendix Table D).

Table 6 shows unadjusted and adjusted all-cause mortality hazard ratios that compare urban Aboriginal adults to their non-Aboriginal counterparts. Urban Aboriginal men and women both had elevated hazard ratios (1.60 and 2.00, respectively); after controlling for community size, lone parenthood, educational attainment, income adequacy, occupation skill level, work status, and immigration, the hazard ratios were reduced to 1.22 and 1.68, respectively, suggesting that 63% (for men) and 32% (for women) of the differences in hazard ratios could be explained by those socio-economic variables.

Discussion

This is the first in-depth study to examine mortality patterns for a large number of Aboriginal adults living in urban Canada. It is important to stress that place of residence and all demographic and socio-economic characteristics were measured only at baseline (June 4, 1991) and do not necessarily reflect the situation later in the follow-up period. Research shows that the Aboriginal population tends to move more frequently than the non-Aboriginal population.[6] For example, about 70% of the Aboriginal population (all ages) residing in metropolitan areas changed residences

between 1991 and 1996, with 45% moving within the same community.[6]

In this cohort, urban Aboriginal adults had higher mortality rates, shorter life expectancy and lower probability of survival to age 75 years compared to urban non-Aboriginal adults. This pattern of higher mortality is consistent with that previously observed for Registered Indians residing in Winnipeg,[19] Vancouver[20] and Canada as a whole.[14]

The higher mortality rates for Aboriginal people are thought to be the product of a wide range of social determinants, experienced from early childhood to old age, that influence health in complex and dynamic ways.[34,35] Our study demonstrates that socio-economic variables were an important contributor to the elevated mortality rates of urban Aboriginal adults, especially for urban Aboriginal men.

Results by major causes of death revealed different patterns of risks. Compared to urban non-Aboriginal cohort members, rate ratios for urban Aboriginal adults were particularly elevated for some causes of death such as digestive system diseases, motor vehicle collisions, alcohol- and drug-related diseases and HIV/AIDS, while rate ratios for other causes, such as all cancers combined, were either similar or only slightly elevated. In such cases, rate ratios were generally similar between Aboriginal adults living in metropolitan areas and those living in smaller urban centres.

Circulatory system diseases were the most common cause of death among urban Aboriginal adults aged 25 years or older, accounting for 32% and 29% of all deaths for urban Aboriginal men and women, respectively. The majority of these deaths were due to ischemic heart disease. The relative risk of deaths due to circulatory system diseases was elevated for urban Aboriginal adults, as was found for Registered Indians in British Columbia.[20] A study of Ontario First Nations found that the rate of hospital admission for ischemic heart disease rose dramatically from 1981 to 1997;[36] some participants in that study may have moved to cities to obtain the use of specialized health care services that were not available in rural or remote settings.

All cancer deaths represented about one in four deaths among urban Aboriginal adults. Compared to urban non-Aboriginal adults, rate ratios for all-cancer mortality were not elevated for urban Aboriginal men and only slightly elevated for urban Aboriginal women, a similar finding to that for Registered Indians in British Columbia.[20] However, grouping of all cancers together might mask important differences, as previous research has shown that Aboriginal people are at increased risk for certain cancers but not for others.[37-41] Limited sample size prevented a detailed analysis of all types of cancer in this study, but our results show that the risk of cancer of the trachea, bronchus and lung was higher among urban Aboriginal adults, specifically those residing in metropolitan areas. Smoking prevalence, a risk factor for lung and other cancers, was more than twice as high among urban Aboriginal people aged 15 years or older compared to that of urban non-Aboriginal persons (43% versus 21%).[42]

Other studies have shown that the HIV/AIDS epidemic is particularly acute among Aboriginal people, especially among the young.[20,43] Results from our study agree: rate ratios for HIV/AIDS mortality were more than twice for Aboriginal men and more than 10 times for Aboriginal women. Among Registered Indians in British Columbia, the rate of deaths due to HIV disease more than doubled from 1993 to 2006.[20]

The risk of dying from external injuries such as motor vehicle accidents and suicide was higher among urban Aboriginal adults than among urban non-Aboriginal adults. Other studies have also shown that Registered Indians and Aboriginal people in general are more likely to die from these causes than are other Canadians.[20,44,45] A detailed breakdown of the different types of external causes of deaths was not possible due to the relatively small number of urban Aboriginal adults in the cohort, but the risk of dying from an external cause appeared greater for urban Aboriginal adults living in metropolitan areas than for those living in smaller urban centres. External causes of death accounted for a smaller proportion of all-cause mortality in this study compared to other studies, in part because our cohort followed people aged 25 years or older, whereas external injury deaths are most common among younger people.[44,45] Because our study excluded the population under the age of 25 years, suicide rates reported in this study also failed to demonstrate the full extent of this problem as the mean age of deaths due to suicide was 27 years among Aboriginal people in Manitoba compared to 45 years for other Manitobans.[46]

The risk of dying from smoking-related diseases was higher among urban Aboriginal cohort members but the relative risk was not as high as for some other causes of death. In comparison, the relative risk of dying from alcohol-related diseases was considerably higher among urban Aboriginal adults (especially women) compared to urban non-Aboriginal adults. Other studies have shown that Registered Indians have a higher relative risk of dying of alcohol-related diseases compared to non-Aboriginal people.[17,20,47] Despite this increased relative risk, deaths among urban Aboriginal adults due to alcohol-related diseases accounted for a smaller proportion of all deaths than did deaths due to smoking-related diseases.

Deaths prior to age 75 years that were amenable to medical care were elevated for urban Aboriginal adults compared to urban non-Aboriginal adults. Although the cause for this increased risk is not known, a 2004 study found that the proportion of persons who reported having a regular doctor did not vary between Aboriginal and non-Aboriginal persons living in urban Canada, but that urban Aboriginal people were more likely to report unmet health care needs than their non-Aboriginal counterparts.[42]

Strengths and limitations

The large size of the Canadian census mortality follow-up study provides an opportunity to examine mortality patterns for urban Aboriginal adults. However, to be eligible for the study, and to be successfully linked, a person must have been enumerated by the 1991 long-form census and must have been a tax filer for the year

TABLE 1
Characteristics of urban Aboriginal and urban non-Aboriginal cohort members by place of residence and sex, non-institutional population aged 25 years or older at baseline, Canada, 1991

	Aboriginal			Non-Aboriginal		
	All urban areas	Metropolitan areas[a]	Smaller urban centres[b]	All urban areas	Metropolitan areas[a]	Smaller urban centres[b]
Both sexes						
Number	16 300	10 400	5 900	2 062 700	1 633 600	429 100
Age 25–44 (%)	73	73	73	54	55	53
Age 65+ (%)	5	5	5	15	15	17
Married or common law (%)	62	60	67	73	72	76
Lone parent (%)	14	14	14	5	5	5
Less than high school diploma (%)	44	42	46	31	30	36
University degree (%)	5	6	3	16	17	10
Employed (%)	56	57	55	67	67	64
Two lowest income quintiles (%)	61	61	62	36	37	36
Activity limitation (%)	15	15	15	10	10	12
Men						
Number	6 900	4 400	2 500	1 013 300	799 800	213 400
Age 25–44 (%)	71	72	71	54	54	52
Age 65+ (%)	5	4	5	14	14	16
Married or common law (%)	67	64	73	79	78	82
Lone parent (%)	3	3	4	2	2	2
Less than high school diploma (%)	45	43	47	31	29	36
University degree (%)	5	7	2	18	19	12
Employed (%)	65	66	64	74	75	71
Two lowest income quintiles (%)	57	56	58	33	34	33
Activity limitation (%)	16	16	16	10	10	12
Women						
Number	9 400	6 000	3 400	1 049 400	833 700	215 700
Age 25–44 (%)	74	74	74	55	55	55
Age 65+ (%)	5	5	6	17	16	18
Married or common law (%)	59	56	63	68	67	70
Lone parent (%)	22	23	22	8	8	8
Less than high school diploma (%)	43	42	45	32	31	36
University degree (%)	5	6	3	13	15	9
Employed (%)	50	51	49	60	61	57
Two lowest income quintiles (%)	65	64	65	39	39	39
Activity limitation (%)	14	14	14	10	10	11

Source: 1991–2001 Canadian census mortality follow-up study.

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE 2

Remaining life expectancy at age 25 years and probability of survival to age 75 years (conditional on surviving to age 25 years) for urban Aboriginal adults and urban non-Aboriginal adults by place of residence and sex, non-institutional population aged 25 years or older at baseline, Canada, 1991–2001

	Total	95% CI	Men	95% CI	Women	95% CI
Life expectancy at age 25 years (years)						
Aboriginal						
All urban areas	50.4	(49.7-51.1)	48.1	(47.1-49.1)	52.7	(51.7-53.7)
Metropolitan areas[a]	50.3	(49.4-51.2)	48.2	(46.9-49.5)	52.4	(51.1-53.7)
Smaller urban centres[b]	50.9	(49.8-52.0)	48.2	(46.6-49.8)	53.6	(52.1-55.0)
Non-Aboriginal						
All urban areas	56.0	(56.0-56.1)	52.8	(52.8-52.9)	59.2	(59.2-59.3)
Metropolitan areas[a]	56.2	(56.1-56.2)	53.0	(52.9-53.1)	59.3	(59.3-59.4)
Smaller urban centres[b]	55.5	(55.4-55.6)	52.2	(52.1-52.4)	58.8	(58.7-59.0)
Probability of survival to age 75 years (%)						
Aboriginal						
All urban areas	57.5	(54.8-60.1)	52.2	(48.2-56.3)	62.7	(59.2-66.2)
Metropolitan areas[a]	55.4	(52.0-58.7)	52.5	(47.5-57.6)	58.2	(53.6-62.7)
Smaller urban centres[b]	61.3	(57.1-65.6)	51.8	(44.9-58.6)	70.9	(65.5-76.3)
Non-Aboriginal						
All urban areas	72.0	(71.8-72.2)	64.5	(64.3-64.8)	79.5	(79.3-79.7)
Metropolitan areas[a]	72.4	(72.2-72.5)	65.0	(64.7-65.3)	79.7	(79.5-79.9)
Smaller urban centres[b]	70.7	(70.3-71.0)	62.9	(62.4-63.4)	78.5	(78.0-78.9)

Source: 1991–2001 Canadian census mortality follow-up study.

Abbreviations: CI, confidence interval.

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE 3

Deaths and mortality rate ratios, by age group at baseline, sex and place of residence, for urban Aboriginal adults compared to urban non-Aboriginal adults, non-institutional population aged 25 years or older at baseline, Canada, 1991–2001

	All urban areas			Metropolitan areas[a]			Smaller urban centres[b]		
Sex and age group at baseline	Deaths	RR	95% CI	Deaths	RR	95% CI	Deaths	RR	95% CI
Men									
Total, 25 + years	563	1.56	(1.43-1.70)	354	1.59	(1.43-1.77)	209	1.45	(1.26-1.66)
25 to 34	67	2.17	(1.71-2.77)	42	2.14	(1.58-2.91)	25	2.16	(1.45-3.22)
35 to 44	78	1.77	(1.41-2.21)	55	1.96	(1.50-2.56)	23	1.41	(0.93-2.13)
45 to 54	123	1.94	(1.62-2.32)	81	2.04	(1.64-2.54)	42	1.68	(1.24-2.28)
55 to 64	122	1.43	(1.20-1.71)	78	1.49	(1.19-1.86)	44	1.29	(0.96-1.74)
65 to 74	103	1.31	(1.08-1.58)	63	1.23	(0.96-1.58)	40	1.41	(1.03-1.93)
75+	70	1.27	(1.01-1.61)	35	1.27	(0.91-1.77)	35	1.23	(0.89-1.72)
Women									
Total, 25 + years	563	1.94	(1.78-2.11)	377	2.10	(1.89-2.32)	186	1.68	(1.44-1.97)
25 to 34	72	3.19	(2.52-4.04)	52	3.71	(2.81-4.89)	20	2.25	(1.43-3.53)
35 to 44	100	2.55	(2.09-3.11)	70	2.87	(2.27-3.64)	30	1.96	(1.36-2.83)
45 to 54	112	2.39	(1.98-2.88)	80	2.72	(2.18-3.39)	32	1.78	(1.25-2.53)
55 to 64	93	1.71	(1.39-2.10)	65	1.87	(1.46-2.39)	28	1.38	(0.95-2.00)
65 to 74	115	1.61	(1.34-1.94)	76	1.80	(1.44-2.26)	39	1.31	(0.95-1.79)
75+	71	1.13	(0.90-1.43)	34	0.92	(0.66-1.29)	37	1.41	(1.02-1.95)

Source: 1991–2001 Canadian census mortality follow-up study.

Note: The rate ratio for all ages combined has been age standardized.

Reference population (person-years at risk) for age standardization was taken from the Aboriginal age distribution (5-year age groups).

Abbreviations: CI, confidence interval; RR, rate ratio.

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE 4

Deaths and age-standardized mortality rates per 100 000 person-years at risk for urban Aboriginal adults by sex and place of residence, non-institutional population aged 25 years or older at baseline, Canada, 1991–2001

	All urban areas			Metropolitan areas[a]			Smaller urban centres[b]		
	Deaths	ASMR	95% CI	Deaths	ASMR	95% CI	Deaths	ASMR	95% CI
Men									
All causes	563	875.4	(804.5-952.4)	354	880.9	(791.0-981.1)	209	860.0	(749.9-986.3)
Infectious diseases	22	31.8	(20.9-48.5)	17	39.3	(24.2-63.8)	5	19.3	(8.0-46.3)
HIV/AIDS	15	21.3	(12.8-35.4)	—	—		—	—	
Other infectious diseases	7	10.5	(4.9-22.4)	—	—		—	—	
Cancer	132	203.8	(171.5-242.3)	79	197.9	(157.7-248.4)	53	215.6	(164.3-282.9)
Trachea/bronchus/lung cancers	51	79.8	(60.4-105.4)	32	83.5	(58.2-119.8)	19	76.7	(48.7-120.9)
Other cancers	81	124.1	(99.6-154.6)	47	114.4	(85.5-153.1)	34	138.9	(99.0-194.9)
Endocrine diseases	16	24.2	(14.7-39.7)	7	16.5	(7.8-34.9)	9	36.1	(18.7-69.9)
Circulatory system	178	285.1	(245.6-331.0)	107	279.5	(229.6-340.3)	71	299.3	(236.5-378.9)
Ischemic heart disease	116	185.0	(153.8-222.5)	71	189.1	(148.6-240.8)	45	193.0	(143.6-259.4)
Other circulatory diseases	62	100.1	(77.7-128.9)	36	90.4	(64.5-126.7)	26	106.3	(72.0-157.0)
Respiratory diseases	39	67.5	(49.1-92.7)	22	63.4	(41.0-98.0)	17	71.0	(43.8-115.1)
Digestive system diseases	37	60.2	(42.0-86.2)	28	73.5	(49.5-109.1)	9	33.8	(17.5-65.3)
External causes	97	138.3	(113.3-168.8)	64	142.4	(111.4-182.0)	33	132.9	(94.4-187.1)
Suicide	23	32.3	(21.4-48.6)	17	37.0	(23.0-59.6)	6	23.7	(10.6-52.8)
Motor vehicle	23	33.2	(22.0-50.0)	14	31.7	(18.8-53.6)	9	37.0	(19.2-71.4)
Other external causes	51	72.9	(55.4-95.9)	33	73.7	(52.4-103.7)	18	72.1	(45.4-114.6)
All other causes	42	64.5	(47.5-87.6)	30	68.5	(47.8-98.2)	12	51.9	(29.3-91.9)
Smoking-related diseases	81	130.3	(104.4-162.5)	51	137.6	(103.3-183.1)	30	122.4	(85.3-175.8)
Alcohol-related diseases	29	41.7	(28.8-60.3)	20	44.8	(28.7-70.0)	9	36.0	(18.7-69.5)
Drug-related diseases	15	20.6	(12.4-34.2)	—	—		—	—	
Amenable to medical intervention (< 75 years)	48	68.6	(51.7-91.1)	30	66.3	(46.4-94.9)	18	72.7	(45.7-115.7)
Women									
All causes	563	615.9	(566.2-670.0)	377	657.3	(593.0-728.7)	186	559.4	(478.9-653.5)
Infectious diseases	20	20.9	(13.5-32.4)	12	20.0	(11.4-35.3)	8	23.6	(11.7-47.2)
HIV/AIDS	6	6.1	(2.8-13.6)	—	—		—	—	
Other infectious diseases	14	14.8	(8.7-25.0)	—	—		—	—	
Cancer	153	162.9	(139.0-190.9)	98	166.9	(136.8-203.6)	55	158.5	(121.5-206.8)
Trachea/bronchus/lung cancers	36	38.6	(27.9-53.6)	26	45.9	(31.2-67.5)	10	27.3	(14.6-50.9)
Breast cancers	26	27.1	(18.4-39.8)	14	23.1	(13.7-39.1)	12	34.7	(19.7-61.2)
Other cancers	91	97.2	(79.1-119.4)	58	97.9	(75.6-126.7)	33	96.5	(68.4-136.1)
Endocrine	23	25.2	(16.7-37.9)	17	28.6	(17.7-46.0)	6	18.6	(8.4-41.6)
Circulatory system	154	178.4	(151.4-210.3)	108	197.3	(162.3-240.0)	46	158.2	(111.3-224.9)
Ischemic heart disease	72	83.5	(65.7-106.2)	50	89.0	(67.2-117.8)	22	78.9	(47.7-130.7)
Other circulatory diseases	82	94.9	(75.8-119.0)	58	108.3	(82.6-142.2)	24	79.3	(48.6-129.4)
Respiratory diseases	34	37.9	(26.8-53.5)	23	41.5	(27.3-62.9)	11	30.0	(16.6-54.3)
Digestive system diseases	53	55.9	(42.7-73.2)	38	62.6	(45.5-86.1)	15	43.5	(26.2-72.5)
External causes	58	59.9	(46.3-77.5)	43	70.5	(52.2-95.1)	15	42.6	(25.7-70.8)
Suicide	14	14.3	(8.5-24.1)	—	—		—	—	
Motor vehicle	15	15.6	(9.4-26.0)	11	18.1	(10.0-32.6)	4	11.4	(4.3-30.4)
Other external (excluding suicide)	29	30.0	(20.8-43.1)	—	—		—	—	
Other external (including suicide)	43	44.2	(32.8-59.7)	32	52.4	(37.0-74.1)	11	31.2	(17.3-56.5)
All other causes	68	74.9	(58.8-95.3)	38	70.0	(50.1-97.8)	30	84.3	(58.9-120.8)
Smoking-related diseases	54	57.8	(44.3-75.6)	36	62.5	(45.0-86.8)	18	49.1	(30.8-78.0)
Alcohol-related diseases	33	34.2	(24.3-48.1)	24	39.3	(26.4-58.7)	9	25.4	(13.2-48.9)
Drug-related diseases	24	24.5	(16.4-36.6)	17	27.5	(17.1-44.3)	7	19.6	(9.3-41.1)
Amenable to medical intervention (< 75 years)	86	92.1	(74.5-113.8)	60	101.5	(78.8-130.8)	26	77.0	(52.4-113.4)

Source: 1991–2001 Canadian census mortality follow-up study.

Abbreviations: —, suppressed due to disclosure rules or not applicable; AIDS, acquired immune deficiency syndrome; ASMR, age-standardized mortality rates; CI, confidence interval; HIV, human immunodeficiency virus.

Reference population (person-years at risk) for age standardization was taken from the Aboriginal age distribution (5-year age groups).

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE 5

Age-adjusted rate ratios by major causes of death and by sex, for urban Aboriginal adults compared to urban non-Aboriginal adults in same urban classification, non-institutional population aged 25 years or older at baseline, Canada, 1991–2001

	All urban areas		Metropolitan areas[a]		Smaller urban centres[b]	
	RR	95% CI	RR	95% CI	RR	95% CI
Men						
All causes	1.56	(1.43-1.70)	1.59	(1.43-1.77)	1.45	(1.26-1.66)
Infectious diseases	2.04	(1.33-3.11)	2.19	(1.34-3.56)	2.89	(1.19-7.03)
HIV/AIDS	2.03	(1.22-3.39)	—		—	
Other infectious diseases	2.04	(0.96-4.37)	—		—	
Cancer	1.09	(0.92-1.30)	1.07	(0.85-1.34)	1.11	(0.85-1.46)
Trachea/bronchus/lung cancers	1.42	(1.08-1.88)	1.53	(1.06-2.19)	1.26	(0.80-1.99)
Other cancers	0.95	(0.76-1.18)	0.88	(0.66-1.18)	1.04	(0.74-1.46)
Endocrine diseases	1.42	(0.86-2.33)	0.98	(0.46-2.08)	2.00	(1.03-3.89)
Circulatory system	1.50	(1.29-1.74)	1.51	(1.24-1.84)	1.45	(1.14-1.83)
Ischemic heart disease	1.52	(1.26-1.83)	1.59	(1.25-2.03)	1.46	(1.08-1.96)
Other circulatory diseases	1.47	(1.14-1.89)	1.36	(0.97-1.91)	1.43	(0.96-2.11)
Respiratory diseases	1.72	(1.25-2.37)	1.68	(1.08-2.59)	1.62	(1.00-2.64)
Digestive system diseases	3.00	(2.09-4.30)	3.67	(2.47-5.45)	1.68	(0.86-3.25)
External causes	2.80	(2.29-3.43)	3.04	(2.37-3.89)	2.26	(1.60-3.20)
Suicide	1.57	(1.04-2.38)	1.91	(1.18-3.08)	0.96	(0.43-2.16)
Motor vehicle	3.51	(2.32-5.32)	3.67	(2.16-6.23)	2.96	(1.51-5.78)
Other external causes	3.76	(2.84-4.96)	3.92	(2.78-5.54)	3.32	(2.07-5.33)
All other causes	1.47	(1.08-2.00)	1.57	(1.09-2.25)	1.18	(0.66-2.09)
Smoking-related diseases	1.46	(1.17-1.82)	1.59	(1.19-2.11)	1.24	(0.86-1.78)
Alcohol-related diseases	4.55	(3.14-6.61)	4.81	(3.06-7.55)	4.22	(2.16-8.24)
Drug-related diseases	3.71	(2.22-6.22)	—		—	
Amenable to medical intervention (< 75 years)	1.80	(1.35-2.39)	1.65	(1.15-2.37)	2.39	(1.49-3.82)
Women						
All causes	1.94	(1.78-2.11)	2.10	(1.89-2.32)	1.68	(1.44-1.97)
Infectious diseases	5.76	(3.68-9.01)	5.25	(2.95-9.32)	8.08	(3.91-16.69)
HIV/AIDS	10.65	(4.56-24.88)	—		—	
Other infectious diseases	4.84	(2.84-8.24)	—		—	
Cancer	1.21	(1.03-1.42)	1.25	(1.02-1.52)	1.16	(0.88-1.51)
Trachea/bronchus/lung cancers	1.33	(0.96-1.85)	1.61	(1.10-2.38)	0.86	(0.46-1.61)
Breast cancers	0.91	(0.62-1.34)	0.77	(0.46-1.31)	1.21	(0.68-2.14)
Other cancers	1.29	(1.05-1.58)	1.30	(1.00-1.69)	1.26	(0.89-1.78)
Endocrine	2.61	(1.73-3.94)	3.00	(1.86-4.85)	1.83	(0.82-4.12)
Circulatory system	1.93	(1.64-2.28)	2.19	(1.80-2.67)	1.56	(1.10-2.22)
Ischemic heart disease	1.73	(1.36-2.21)	1.89	(1.43-2.50)	1.52	(0.91-2.52)
Other circulatory diseases	2.15	(1.71-2.69)	2.53	(1.93-3.32)	1.61	(0.98-2.63)
Respiratory diseases	1.91	(1.35-2.71)	2.15	(1.41-3.26)	1.39	(0.77-2.52)
Digestive system diseases	4.82	(3.67-6.34)	5.41	(3.92-7.46)	3.73	(2.23-6.27)
External causes	3.37	(2.59-4.37)	4.12	(3.04-5.58)	2.09	(1.25-3.50)
Suicide	2.46	(1.45-4.19)	—		—	
Motor vehicle	4.13	(2.46-6.93)	5.24	(2.87-9.59)	2.22	(0.82-6.06)
Other external (excluding suicide)	3.65	(2.53-5.28)	—		—	
Other external (including suicide)	3.16	(2.33-4.28)	3.84	(2.70-5.46)	2.04	(1.12-3.73)
All other causes	2.63	(2.06-3.36)	2.43	(1.74-3.40)	3.10	(2.15-4.46)
Smoking-related diseases	1.36	(1.04-1.78)	1.50	(1.08-2.08)	1.07	(0.67-1.71)
Alcohol-related diseases	11.44	(8.02-16.34)	12.87	(8.49-19.50)	9.38	(4.70-18.73)
Drug-related diseases	6.43	(4.26-9.73)	7.40	(4.53-12.08)	4.65	(2.14-10.10)
Amenable to medical intervention (< 75 years)	1.99	(1.61-2.47)	2.20	(1.71-2.85)	1.65	(1.11-2.43)

Source: 1991–2001 Canadian census mortality follow-up study.

Abbreviations: —, suppressed due to disclosure rules or not applicable; AIDS, acquired immune deficiency syndrome; ASMR, age-standardized mortality rates; CI, confidence interval; HIV, human immunodeficiency virus.

Reference population (person-years at risk) for age standardization was taken from the Aboriginal age distribution (5-year age groups).

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE 6

Adjusted and unadjusted all-cause mortality hazard ratios for Aboriginal and non-Aboriginal adults residing in all urban areas, by sex, non-institutional population aged 25 years or older at baseline, Canada, 1991–2001

Characteristic at baseline	Men				Women			
	Unadjusted		Adjusted		Unadjusted		Adjusted	
	Hazard ratio	95% CI	Hazard ratio	95% CI	Hazard ratio	95% CI	Hazard ratio	95% CI
Aboriginal								
Yes	1.60	(1.47-1.73)	1.22	(1.13-1.33)	2.00	(1.84-2.17)	1.68	(1.55-1.83)
No (ref)	1.00	...	1.00	...	1.00	...	1.00	...
Age (years)	1.10	(1.10-1.10)	1.09	(1.09-1.09)	1.10	(1.10-1.10)	1.09	(1.09-1.09)
Lone parent								
Yes	...	...	1.04	(0.99-1.08)	...	...	1.10	(1.07-1.13)
No (ref)	...	...	1.00	...	...	...	1.00	...
Place of residence								
Metropolitan areas[a]	...	...	1.01	(1.00-1.03)	...	...	0.99	(0.98-1.01)
Smaller urban centres[b]	...	...	1.00	...	...	...	1.00	...
Highest educational attainment								
Less than high school diploma	...	...	1.35	(1.32-1.39)	...	...	1.24	(1.19-1.28)
High school diploma	...	...	1.22	(1.19-1.25)	...	...	1.15	(1.11-1.19)
Post-secondary diploma	...	...	1.09	(1.06-1.13)	...	...	1.07	(1.03-1.11)
University degree (ref)	...	...	1.00	...	...	...	1.00	...
Income adequacy quintile								
Quintile 1 – lowest	...	...	1.41	(1.38-1.44)	...	...	1.30	(1.27-1.33)
Quintile 2	...	...	1.18	(1.16-1.20)	...	...	1.13	(1.10-1.15)
Quintile 3	...	...	1.10	(1.07-1.12)	...	...	1.08	(1.05-1.11)
Quintile 4	...	...	1.04	(1.01-1.06)	...	...	1.04	(1.01-1.07)
Quintile 5 – highest (ref)	...	...	1.00	...	...	...	1.00	...
Occupation – skill-based categories								
Professional (ref)	...	...	1.00	...	...	...	1.00	...
Managerial	...	...	0.99	(0.95-1.03)	...	...	1.07	(0.99-1.15)
Skilled/Technical/Supervisory	...	...	1.09	(1.05-1.13)	...	...	1.10	(1.04-1.16)
Semi-skilled	...	...	1.19	(1.14-1.23)	...	...	1.11	(1.05-1.17)
Unskilled	...	...	1.27	(1.22-1.33)	...	...	1.18	(1.11-1.26)
No occupation	...	...	1.29	(1.24-1.34)	...	...	1.29	(1.22-1.36)
Work status								
Unemployed (ref)	...	...	1.00	...	...	...	1.00	...
Employed	...	...	0.82	(0.79-0.85)	...	...	0.89	(0.84-0.94)
Not in labour force	...	...	1.16	(1.11-1.20)	...	...	1.09	(1.03-1.16)
Place of birth								
Canada (ref)	...	...	1.00	...	...	...	1.00	...
Overseas	...	...	0.74	(0.73-0.75)	...	...	0.84	(0.83-0.86)

Source: 1991-2001 Canadian census mortality follow-up study.

Abbreviations: ..., not applicable; CI, confidence interval; ref, reference.

[a] Population ≥ 100 000

[b] Population ≥ 10 000

1990 or 1991. Thus any individual who did not file a tax return (under Section 87 of the *Indian Act*, Registered Indians are entitled to a tax exemption for income earned or considered to be earned on a reserve[48]) or who was in a long-term care facility, senior's residence or prison could not be included in the cohort. Despite this limitation, we found no major differences in demographic and socio-economic characteristics between eligible census respondents and those successfully linked to the name file.

Compared to life tables for all Canada (for 1995–1997), at age 25 years the entire cohort had remaining life expectancy 1 year longer for men, and 2 years longer for women.

Ascertainment of deaths was estimated to be slightly lower among Aboriginal persons (95% to 96%) compared to the cohort as a whole (97%). This would result in a slight downward bias in calculated mortality rates for the urban Aboriginal population, so the true extent of the disparities compared to the non-Aboriginal cohort could be slightly larger than indicated in this study.

Since a question on Aboriginal self-identity was not part of the 1991 census, this study defined the urban Aboriginal population on the basis of Aboriginal ancestry, Registered Indian status and/or membership in an Indian band or First Nation. This definition undoubtedly excluded many persons who would have self-identified as Aboriginal. According to the 1996 census results concerning self-identification with an Aboriginal group, about 8% of the self-identifying Aboriginal population did not report any Aboriginal ancestry,[32] although some of the latter may have been Registered Indians or members of an Indian band or First Nation.

Studies have shown differences in health indicators for First Nations, Inuit and Métis.[10] Since this study grouped First Nations, Inuit and Métis together, intra-group differences were obscured. Moreover, the results may not be reflective of Inuit living in urban areas since Inuit made up only 3% of the Aboriginal cohort.

Conclusion

Until this study, only limited information on the mortality of the urban Aboriginal people of Canada was available. We found that mortality rates were higher for urban Aboriginal adults compared to urban non-Aboriginal adults. Circulatory system disease deaths and cancer deaths were the most common cause of death for urban Aboriginal and non-Aboriginal adults. However, relative risks were particularly elevated for some causes of death such as digestive system diseases, motor vehicle collisions, alcohol- and drug-related diseases and HIV/AIDS. In agreement with other research, our results also demonstrated that socio-economic status played an important role in explaining these disparities.

Acknowledgements

Major funding for this study was provided by the Canadian Population Health Initiative, part of the Canadian Institute for Health Information. We would also like to acknowledge the key importance of Canada's provincial and territorial registrars of vital statistics, who provide the death data for the Canadian Mortality Data Base.

The views expressed in this article are those of the authors and do not necessarily reflect the views of the above-named organizations or of the institutions with which they are affiliated.

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Appendix

TABLE A

Long-form census respondents, cohort members, linkage rate to name file, deaths ascertained, and person-years at risk, non-institutional population aged 25 years or older at baseline, 1991

	Census respondents (n)	Study cohort (n)	Linkage rate to name file (%)	Deaths ascertained (n)	Person years
Aboriginal population					
All urban areas					
Total	25 500	16 300	64	1 126	166 570
Men	11 300	6 900	61	563	69 580
Women	14 200	9 400	66	563	96 990
Metropolitan areas[a]					
Total	15 800	10 400	66	731	106 030
Men	7 000	4 400	63	354	44 610
Women	8 800	6 000	68	377	61 410
Smaller urban centres[b]					
Total	9 700	5 900	61	395	60 540
Men	4 300	2 500	58	209	24 970
Women	5 400	3 400	64	186	35 570
Non-Aboriginal					
All urban areas					
Total	2 644 400	2 062 700	78	192 932	20 844 280
Men	1 270 400	1 013 300	80	111 126	10 145 220
Women	1 373 900	1 049 400	76	81 806	10 699 060
Metropolitan areas[a]					
Total	2 098 600	1 633 600	78	148 482	16 528 930
Men	1 007 700	799 800	79	84 836	8 022 930
Women	1 090 900	833 700	76	63 646	8 506 000
Smaller urban centres[b]					
Total	545 800	429 100	79	44 450	4 315 350
Men	262 700	213 400	81	26 290	2 122 290
Women	283 000	215 700	76	18 160	2 193 060

Source: 1991–2001 Canadian census mortality follow-up study.

Note: Census population counts rounded to nearest 100, person years rounded to the nearest 10.

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE B

Demographic and socio-economic characteristics at baseline of the in-scope (eligible) urban Aboriginal census respondents compared to urban Aboriginal cohort members, by sex and place of residence, non-institutional population aged 25 years or older at baseline, 1991

	All urban areas			Metropolitan areas[a]			Smaller urban centres[b]		
	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio
	%	%		%	%		%	%	
Men									
Number	49 100	6 900		33 100	4 400		16 000	2 500	
Age group (years)									
25 to 34	43	42	0.97	43	42	0.97	42	41	0.98
35 to 44	28	30	1.04	28	30	1.04	28	29	1.05
45 to 54	16	16	1.04	16	16	1.03	15	16	1.07
55 to 64	8	8	0.95	8	8	0.97	9	8	0.91
65 to 74	3	3	1.01	3	3	1.05	3	3	0.93
75 +	2	1	0.79	1	1	0.78	2	2	0.78
Marital status									
Single (never married)	26	23	0.88	28	25	0.90	21	18	0.84
Common-law	19	17	0.93	18	16	0.90	21	20	0.96
Married	42	50	1.17	41	48	1.17	45	53	1.17
Previously married	13	10	0.79	13	11	0.82	13	10	0.74
Lone parent									
Yes	96	97	1.01	96	97	1.01	96	96	1.01
No	4	3	0.83	4	3	0.79	4	4	0.87
Educational attainment									
Less than high school diploma	47	45	0.94	45	43	0.96	51	47	0.91
High school diploma	38	40	1.05	38	39	1.03	38	41	1.09
Post-secondary diploma	9	10	1.08	10	11	1.06	8	9	1.15
University degree	5	5	1.00	6	7	1.03	2	2	1.01
Labour force status									
Employed	61	65	1.06	62	66	1.05	59	64	1.09
Unemployed	17	16	0.94	15	14	0.92	19	18	0.95
Not in labour force	22	20	0.88	22	20	0.91	22	18	0.81
Income quintile									
Quintile 1 – lowest	39	34	0.88	39	34	0.89	38	33	0.86
Quintile 2	21	23	1.06	21	21	1.01	22	25	1.16
Quintile 3	17	19	1.12	17	19	1.12	16	18	1.11
Quintile 4	14	16	1.07	14	16	1.10	14	15	1.02
Quintile 5 – highest	9	9	1.03	9	9	1.07	9	9	0.97
Activity limitation									
Not stated	2	1	0.59	2	1	0.72	2	1	0.39
No	82	83	1.02	82	83	1.01	82	84	1.02
Yes	17	16	0.96	17	16	0.96	16	16	0.95

TABLE B (Continued)

Demographic and socio-economic characteristics at baseline of the in-scope (eligible) urban Aboriginal census respondents compared to urban Aboriginal cohort members, by sex and place of residence, non-institutional population aged 25 years or older at baseline, 1991

	All urban areas			Metropolitan areas[a]			Smaller urban centres[b]		
	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio
	%	%		%	%		%	%	
Women									
Number	65 500	9 400		43 700	6 000		21 800	3 400	
Age group (years)									
25 to 34	42	44	1.04	43	44	1.03	41	44	1.05
35 to 44	29	30	1.03	29	30	1.01	28	30	1.07
45 to 54	15	15	0.99	15	15	1.00	15	14	0.97
55 to 64	8	7	0.85	8	7	0.91	8	6	0.75
65 to 74	4	4	0.85	4	4	0.86	5	4	0.83
75 +	2	1	0.75	1	1	0.82	2	2	0.65
Marital status									
Single (never married)	20	19	0.95	21	20	0.95	18	17	0.95
Common-law	16	16	1.01	15	15	0.97	17	17	1.06
Married	40	43	1.08	38	41	1.08	42	46	1.08
Previously married	25	22	0.91	25	23	0.93	24	20	0.86
Lone parent									
Yes	23	22	0.95	23	23	0.96	23	22	0.93
No	77	78	1.01	77	77	1.01	77	78	1.02
Educational attainment									
Less than high school diploma	46	43	0.93	45	42	0.93	50	45	0.92
High school diploma	34	36	1.05	35	37	1.05	32	35	1.07
Post-secondary diploma	15	16	1.08	15	16	1.06	15	17	1.12
University degree	5	5	1.07	5	6	1.12	3	3	0.99
Labour force status									
Employed	46	50	1.10	47	51	1.08	43	49	1.14
Unemployed	11	11	0.98	10	10	0.97	13	13	0.98
Not in labour force	43	39	0.90	43	40	0.92	44	38	0.87
Income quintile									
Quintile 1 - lowest	47	43	0.93	47	44	0.93	46	42	0.92
Quintile 2	20	21	1.05	20	20	1.03	21	23	1.08
Quintile 3	15	16	1.06	14	15	1.06	15	16	1.05
Quintile 4	11	12	1.08	11	12	1.11	11	11	1.04
Quintile 5 - highest	8	8	1.08	8	8	1.09	7	8	1.08
Activity limitation									
Not stated	1	0	0.54	1	0	0.51	1	0	0.60
No	84	85	1.02	84	85	1.02	84	86	1.02
Yes	15	14	0.91	15	14	0.92	16	14	0.90

Source: 1991 census and 1991–2001 Canadian census mortality follow-up study.

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE C

Demographic and socio-economic characteristics of the in-scope (eligible) urban non-Aboriginal census respondents compared to urban non-Aboriginal cohort members, by sex and place of residence, non-institutional population aged 25 years or older at baseline, 1991

	All urban areas			Metropolitan areas[a]			Smaller urban centres[b]		
	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio
	%	%		%	%		%	%	
Men									
Number	6 470 600	1 013 300		5 159 500	799 800		1 311 100	213 400	
Age group (years)									
25 to 34	29	28	0.95	30	28	0.94	27	26	0.97
35 to 44	26	26	1.02	26	26	1.01	26	26	1.03
45 to 54	18	18	1.03	18	18	1.03	17	18	1.02
55 to 64	14	14	1.03	13	14	1.04	14	14	1.01
65 to 74	9	10	1.04	9	10	1.05	11	11	1.01
75 +	5	5	0.97	4	4	0.97	6	5	0.93
Marital status									
Single (never married)	16	14	0.84	17	15	0.84	13	11	0.85
Common-law	7	7	0.90	7	6	0.90	8	7	0.88
Married	67	72	1.08	66	72	1.08	70	75	1.07
Previously married	9	7	0.81	9	7	0.80	9	8	0.81
Lone parent									
Yes	98	98	1.00	98	98	1.00	98	98	1.00
No	2	2	0.86	2	2	0.86	2	2	0.85
Educational attainment									
Less than high school diploma	32	31	0.97	30	29	0.97	38	36	0.96
High school diploma	38	38	1.01	37	37	1.00	40	40	1.02
Post-secondary diploma	13	14	1.03	14	14	1.02	12	12	1.04
University degree	17	18	1.03	19	19	1.03	11	12	1.05
Labour force status									
Employed	72	74	1.02	73	75	1.02	69	71	1.03
Unemployed	6	6	0.91	6	6	0.90	7	6	0.93
Not in labour force	21	20	0.96	20	20	0.96	24	23	0.95
Income quintile									
Quintile 1 – lowest	16	14	0.87	17	14	0.87	15	13	0.88
Quintile 2	20	19	0.99	20	19	0.99	19	19	0.99
Quintile 3	21	21	1.02	21	21	1.02	21	22	1.03
Quintile 4	21	22	1.04	21	22	1.04	22	23	1.04
Quintile 5 – highest	22	23	1.05	22	23	1.05	22	23	1.03
Activity limitation									
Not stated	1	0	0.77	1	0	0.74	1	1	0.88
No	89	89	1.01	89	90	1.01	86	87	1.01
Yes	11	10	0.96	10	10	0.97	13	12	0.94

TABLE C (Continued)

Demographic and socio-economic characteristics of the in-scope (eligible) urban non-Aboriginal census respondents compared to urban non-Aboriginal cohort members, by sex and place of residence, non-institutional population aged 25 years or older at baseline, 1991

	All urban areas			Metropolitan areas[a]			Smaller urban centres[b]		
	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio	In-scope	Cohort	Ratio
	%	%		%	%		%	%	
Women									
Number	6 983 500	1 049 400	1.00	5 574 600	833 700	1.00	1 408 900	215 700	1.00
Age group (years)									
25 to 34	27	29	1.04	28	29	1.03	26	28	1.09
35 to 44	25	26	1.08	25	26	1.07	24	26	1.10
45 to 54	16	17	1.01	16	17	1.01	16	16	1.00
55 to 64	13	12	0.89	13	12	0.90	14	12	0.85
65 to 74	11	10	0.91	11	10	0.92	12	11	0.86
75 +	7	6	0.89	7	6	0.90	8	7	0.89
Marital status									
Single (never married)	12	13	1.01	13	13	1.00	9	10	1.07
Common-law	6	6	1.00	6	6	1.00	6	6	1.01
Married	61	62	1.02	60	61	1.02	63	63	1.00
Previously married	21	20	0.95	21	20	0.94	21	21	0.97
Lone parent									
Yes	91	92	1.00	91	92	1.00	92	92	1.00
No	9	8	0.96	9	8	0.95	8	8	0.99
Educational attainment									
Less than high school diploma	35	32	0.91	34	31	0.91	40	36	0.89
High school diploma	34	36	1.03	34	35	1.03	34	36	1.04
Post-secondary diploma	18	19	1.08	18	19	1.07	18	19	1.10
University degree	12	13	1.08	14	15	1.07	8	9	1.14
Labour force status									
Employed	55	60	1.09	56	61	1.08	51	57	1.12
Unemployed	5	5	1.01	5	5	1.01	5	6	1.03
Not in labour force	40	35	0.88	39	34	0.88	44	38	0.86
Income quintile									
Quintile 1 – lowest	21	20	0.92	21	19	0.91	22	20	0.94
Quintile 2	21	20	0.96	21	20	0.97	20	19	0.94
Quintile 3	20	20	1.02	20	20	1.02	20	20	1.02
Quintile 4	19	20	1.05	19	20	1.05	19	20	1.04
Quintile 5 – highest	19	20	1.07	19	20	1.06	19	20	1.07
Activity limitation									
Not stated	1	0	0.89	1	0	0.87	0	0	0.97
No	88	89	1.01	89	90	1.01	87	88	1.02
Yes	11	10	0.90	11	10	0.91	13	11	0.89

Source: 1991 census and 1991–2001 Canadian census mortality follow-up study.

[a] Population ≥ 100 000

[b] Population ≥ 10 000

TABLE D

Deaths and age-standardized mortality rates per 100 000 person-years at risk for urban non-Aboriginal adults by sex and place of residence, non-institutional population aged 25 years or older at baseline, Canada, 1991–2001

	All urban areas			Metropolitan areas[a]			Smaller urban centres[b]		
	Deaths	ASMR	95% CI	Deaths	ASMR	95% CI	Deaths	ASMR	95% CI
Men									
All causes	111 126	561.7	(558.1-565.4)	84 836	553.2	(549.1-557.3)	26 290	592.8	(584.6-601.0)
Infectious diseases	1 875	15.6	(14.9-16.4)	1 654	18.0	(17.0-19.0)	221	6.7	(5.7-7.8)
HIV/AIDS	919	10.5	(9.8-11.2)	876	12.6	(11.7-13.5)	43	2.4	(1.8-3.3)
Other infectious diseases	956	5.1	(4.8-5.5)	778	5.4	(5.0-5.8)	178	4.2	(3.6-5.0)
Cancer	37 073	186.7	(184.7-188.8)	28 544	184.8	(182.5-187.1)	8 529	194.1	(189.5-198.7)
Trachea/bronchus/lung cancers	11 315	56.0	(54.9-57.1)	8 624	54.7	(53.5-55.9)	2 691	60.7	(58.3-63.3)
Other cancers	25 758	130.7	(129.0-132.5)	19 920	130.1	(128.1-132.1)	5 838	133.3	(129.6-137.2)
Endocrine diseases	3 463	17.1	(16.4-17.7)	2 647	16.8	(16.1-17.5)	816	18.0	(16.7-19.5)
Circulatory system	40 955	190.0	(188.0-192.0)	30 951	185.2	(183.0-187.4)	10 004	207.1	(202.6-211.6)
Ischemic heart disease	25 856	121.7	(120.1-123.3)	19 645	118.7	(117.0-120.5)	6 211	132.5	(128.9-136.2)
Other circulatory diseases	15 099	68.3	(67.1-69.5)	11 306	66.5	(65.2-67.8)	3 793	74.6	(72.0-77.2)
Respiratory diseases	9 390	39.2	(38.3-40.0)	6 971	37.8	(36.9-38.7)	2 419	43.8	(42.0-45.7)
Digestive system diseases	3 886	20.1	(19.4-20.8)	3 004	20.0	(19.3-20.8)	882	20.2	(18.7-21.7)
External causes	5 710	49.3	(47.9-50.8)	4 279	46.9	(45.3-48.4)	1 431	58.8	(55.5-62.4)
Suicide	2 063	20.5	(19.6-21.5)	1 547	19.4	(18.4-20.5)	516	24.6	(22.4-27.0)
Motor vehicle	1 031	9.4	(8.8-10.1)	746	8.7	(8.0-9.4)	285	12.5	(11.0-14.3)
Other external causes	2 616	19.4	(18.6-20.3)	1 986	18.8	(17.9-19.8)	630	21.7	(19.8-23.8)
All other causes	8 774	43.8	(42.8-44.8)	6 786	43.7	(42.6-44.9)	1 988	44.1	(41.9-46.5)
Smoking-related diseases	18 829	89.4	(88.0-90.8)	14 182	86.8	(85.3-88.3)	4 647	98.9	(95.8-102.0)
Alcohol-related diseases	1 433	9.2	(8.6-9.7)	1 145	9.3	(8.7-9.9)	288	8.5	(7.5-9.7)
Drug-related diseases	513	5.1	(5.6-5.1)	410	5.6	(5.1-6.2)	103	5.4	(4.4-6.6)
Amenable to medical intervention (< 75 years)	5 540	38.1	(37.0-39.2)	4 473	40.1	(38.8-41.4)	1 067	30.4	(28.5-32.5)
Women									
All causes	81 806	317.6	(314.9-320.2)	63 646	313.6	(310.7-316.6)	18 160	332.5	(326.6-338.6)
Infectious diseases	834	3.6	(3.3-4.0)	685	3.8	(3.5-4.2)	149	2.9	(2.4-3.6)
HIV/AIDS	54	0.6	(0.4-0.8)	51	0.7	(0.5-0.9)	3	0.2	(0.1-0.6)
Other infectious diseases	780	3.1	(2.8-3.3)	634	3.1	(2.9-3.5)	146	2.7	(2.2-3.3)
Cancer	27 256	134.3	(132.5-136.2)	21 495	133.6	(131.5-135.7)	5 761	137.2	(133.1-141.5)
Trachea/bronchus/lung cancers	5 687	29.1	(28.2-29.9)	4 433	28.4	(27.5-29.4)	1 254	31.7	(29.8-33.8)
Breast cancers	5 158	29.7	(28.8-30.6)	4 111	29.9	(28.9-31.0)	1 047	28.8	(26.8-30.9)
Other cancers	16 411	75.5	(74.2-76.9)	12 951	75.2	(73.7-76.8)	3 460	76.7	(73.7-79.9)
Endocrine	2 696	9.6	(9.2-10.1)	2 110	9.5	(9.0-10.0)	586	10.2	(9.2-11.2)
Circulatory system	30 369	92.4	(91.1-93.6)	23 298	90.0	(88.6-91.3)	7 071	101.4	(98.6-104.3)
Ischemic heart disease	16 007	48.2	(47.3-49.0)	12 328	47.1	(46.2-48.1)	3 679	52.0	(50.1-54.0)
Other circulatory diseases	14 362	44.2	(43.3-45.1)	10 970	42.8	(41.9-43.8)	3 392	49.3	(47.3-51.4)
Respiratory diseases	6 421	19.8	(19.2-20.4)	4 953	19.3	(18.7-20.0)	1 468	21.6	(20.3-22.9)
Digestive system diseases	3 070	11.6	(11.1-12.1)	2 376	11.6	(11.0-12.1)	694	11.7	(10.6-12.8)
External causes	2 995	17.8	(17.0-18.6)	2 273	17.1	(16.3-18.0)	722	20.4	(18.6-22.4)
Suicide	610	5.8	(5.3-6.3)	490	5.8	(5.3-6.4)	120	5.8	(4.8-7.0)
Motor vehicle	491	3.8	(3.4-4.2)	356	3.4	(3.1-3.9)	135	5.1	(4.2-6.2)
Other external (excluding suicide)	1 894	8.2	(7.7-8.7)	1 427	7.9	(7.3-8.4)	467	9.5	(8.4-10.7)
Other external (including suicide)	2 504	14.0	(13.3-14.7)	1 917	13.6	(12.9-14.4)	587	15.3	(13.8-17.0)
All other causes	8 165	28.5	(27.7-29.2)	6 456	28.8	(27.9-29.7)	1 709	27.2	(25.6-28.9)
Smoking-related diseases	9 530	42.6	(41.6-43.6)	7 430	41.8	(40.7-42.9)	2 100	45.8	(43.6-48.2)
Alcohol-related diseases	484	3.0	(2.7-3.3)	382	3.1	(2.7-3.4)	102	2.7	(2.2-3.4)
Drug-related diseases	413	3.8	(3.4-4.2)	326	3.7	(3.3-4.2)	87	4.2	(3.4-5.3)
Amenable to medical intervention (< 75 years)	6 595	46.2	(45.0-47.4)	5 230	46.0	(44.7-47.4)	1 365	46.8	(44.2-49.6)

Source: 1991–2001 Canadian census mortality follow-up study.

Abbreviations: —, not applicable; AIDS, acquired immune deficiency syndrome; ASMR, age-standardized mortality rates; CI, confidence interval; HIV, human immunodeficiency virus.

Reference population (person-years at risk) for age standardization was taken from the Aboriginal age distribution (5-year age groups).

[a] Population ≥ 100 000

[b] Population ≥ 10 000

Arthritis in the Canadian Aboriginal population: north-south differences in prevalence and correlates*

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* This article is part of a joint publication initiative between *Preventing Chronic Disease* and *Chronic Diseases in Canada*. *Preventing Chronic Disease* is the secondary publisher, while *Chronic Diseases in Canada* is the primary publisher.

Abstract

Background: Information on arthritis and other musculoskeletal disorders among Aboriginal people is sparse. Survey data show that arthritis and rheumatism are among the most commonly reported chronic conditions and their prevalence is higher than among non-Aboriginal people.

Objective: To describe the burden of arthritis among Aboriginal people in northern Canada and demonstrate the public health significance and social impact of the disease.

Methods: Using cross-sectional data from more than 29 000 Aboriginal people aged 15 years and over who participated in the Aboriginal Peoples Survey 2006, we assessed regional differences in the prevalence of arthritis and its association with other risk factors, co-morbidity and health care use.

Results: The prevalence of arthritis in the three northern territories ("North") is 12.7% compared to 20.1% in the provinces ("South") and is higher among females than males in both the North and South. The prevalence among Inuit is lower than among other Aboriginal groups. Individuals with arthritis are more likely to smoke, be obese, have concurrent chronic diseases, and are less likely to be employed. Aboriginal people with arthritis utilized the health care system more often than those without the disease.

Conclusion: Aboriginal-specific findings on arthritis and other chronic diseases as well as recognition of regional differences between North and South will enhance program planning and help identify new priorities in health promotion.

Keywords: *arthritis, Aboriginal people, Northern Canada, Inuit, First Nations, Métis, North American Indians, Aboriginal Peoples Survey*

Introduction

Information on arthritis and other musculoskeletal disorders among Aboriginal people is sparse and geographically limited—mainly to Alaska, British Columbia and Manitoba.[1-3] Several national surveys—the First Nations Regional Longitudinal Health Survey (RHS),[4,5] the Canadian Community Health Survey (CCHS)[6,7] and the Aboriginal Peoples Survey (APS)[8,9]—have provided some data on the prevalence of arthritis, rheumatism and other musculoskeletal conditions, such as back pain, among adults in the Aboriginal population. These surveys generally show that arthritis and rheumatism are among the most commonly reported chronic conditions, that prevalence is higher than among non-Aboriginal people in Canada, and that prevalence is increasing; for example, the crude prevalence was 15% in 1991 and 19% in 2001 according to the APS,[8,9] while the age-adjusted prevalence was 22% in 1997 and 25% in 2002/03 according to the RHS.[4,5] (Note that these surveys are not directly comparable with one another due to the inclusion of different Aboriginal groups and the use of different standard populations in age-adjustment of rates.) Arthritis also contributes to more than half of the self-reported disability among First Nations people in Canada.[5]

Disability resulting from arthritis can be exacerbated in the north of Canada by severe weather, inadequate infrastructure and unreliable transportation. Arthritis compromises the ability of Aboriginal people to pursue traditional activities, such as harvesting country foods, and traditional crafts. The geographical isolation of many communities reduces access to specialist services. Cultural context is an additional dimension and requires region-specific directions and broad partnerships to plan and implement culturally appropriate health services and support systems; specific considerations include, but are not limited to, access to traditional healers and medicines, languages spoken, and the design of support services in communities.

This paper describes the burden of arthritis among Aboriginal people in Yukon, Northwest Territories and Nunavut—the three northern territories of Canada. We assess regional differences in the prevalence of arthritis and its association with other risk factors, co-morbidity and health care use between these three northern territories (the "North") and the ten provinces of southern Canada (the "South") using data from the recently released APS 2006.[10]

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Methods

We used cross-sectional data from more than 29 000 Aboriginal respondents (off-reserve First Nations, Métis, Inuit) aged 15 years and over who participated in the APS 2006 (Table 1). Statistics Canada conducted the APS as a post-censal survey to collect information on the social and economic conditions of Aboriginal people living in Canada.

The APS 2006 asked respondents whether a doctor, nurse or other health professional had ever told them that they have arthritis or rheumatism. We separately examined associations between arthritis and various demographic, socioeconomic, behavioural and health care correlates for the three territories and the 10 provinces, not to test specific etiological hypotheses but to demonstrate the public health significance and social impact of the burden of arthritis. Obesity, defined as body mass index (BMI) of 30 kg/m^2 or higher, and smoking are well-established risk factors for arthritis,[11,12] and the APS 2006 asked respondents about their height, weight and smoking experience and habits. Arthritis is also associated with reduced employment and work limitations among adults;[13] the APS asked respondents, "Last week, did you work for pay or in self-employment?"

We determined prevalence of arthritis for three separate groups based on the question "Do any of your ancestors belong to the following Aboriginal groups? (Can check more than one): North American Indian, Métis or Inuit." Individuals who checked only "North American Indian" constitute the "First Nations" group, individuals who checked only "Inuit" constitute the "Inuit" group, and all others including Métis and those who checked multiple Aboriginal groups were combined into an "Other" category as each of these groups have small sample sizes in the North. We report only crude prevalence proportions; we did not compute age-adjusted prevalence as the dataset did not include non-Aboriginal people for comparison, and comparing this study with published age-adjusted rates is difficult due to the different standard populations that have been used; further, the crude prevalence more accurately reflects the burden of disease needed to plan public health programs.

We performed all analyses using SAS version 9.2 (SAS Institute Inc, Cary, North Carolina). Since the APS 2006 was based on a complex survey design, we used survey weights in all analyses and calculated variance estimates using the bootstrap technique with the 1000 bootstrap weights provided by Statistics Canada. We determined all proportions in accordance with rounding guidelines suggested by Statistics Canada and calculated confidence intervals (CIs) from unrounded components. Detailed survey methodology is available from Statistics Canada.[10] We used age- and sex-adjusted logistic regression analyses to assess associations between arthritis and various correlates.

TABLE 1
Number of respondents aged 15 years and over by geographic region and Aboriginal group in the North[a] and South[b] of Canada

	North[a]		South[b]	
	Male	Female	Male	Female
First Nations	380	420	5260	6680
Inuit	1630	1650	830	880
Other[c]	270	260	4990	5870

Note: numbers are unweighted and have been rounded to the nearest ten.

[a] The three Canadian northern territories: Yukon, Northwest Territories, Nunavut.

[b] The 10 Canadian provinces: British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, Quebec, New Brunswick, Prince Edward Island, Nova Scotia, Newfoundland and Labrador.

[c] Respondents of Métis or multiple Aboriginal ancestry.

Results

Prevalence

The crude prevalence of arthritis or rheumatism for the three combined Aboriginal groups in the territories is 12.7% (95% CI: 12.5-13.0) compared to 20.1% in the provinces (95% CI: 19.9-20.3). Arthritis is more prevalent among females than males in both the North and South. The prevalence among Inuit is lower than among First Nations and other Aboriginal groups. As expected, prevalence increases with age (Table 2).

Health risks

For a comparison of the proportion of respondents with and without arthritis who are daily smokers, are obese or have co-morbid conditions, see Table 3.

Smoking is more prevalent among Aboriginal people in the North than in the South. In the South, there is an association between daily smoking and arthritis (age-sex-adjusted odds ratio [OR] = 1.58, 95% CI: 1.53-1.62), but daily smoking is not a significant factor in the North (OR = 1.05, 95% CI: 0.98-1.12). Obesity is more prevalent among individuals with arthritis, and the association between obesity and arthritis is stronger among Aboriginal people in the South (OR = 1.59, 95% CI: 1.54-1.64) than in the North (OR = 1.36, 95% CI: 1.26-1.47).

In both the South and the North, a higher proportion of individuals with arthritis than those without report having at least one other chronic condition such as diabetes, heart disease, hypertension, stroke, asthma, chronic bronchitis, emphysema or cancer (for the North, OR = 1.88, 95% CI: 1.77-2.00; for the South, OR = 2.55, 95% CI: 2.48-2.61).

Health care use

The proportion of individuals who report consulting a health professional (primary care physician or nurse) or traditional

[†] In the APS 2006, a traditional healer refers to someone who is recognized by the community as a traditional counsellor, or someone who provides traditional medicines such as herbs, or is a traditional or spiritual leader.

TABLE 2

Crude prevalence (%) of arthritis and rheumatism among Aboriginal people aged 15 years and over in the North[a] and South[b] of Canada

	North[a]						South[b]					
	Male		Female		Both		Male		Female		Both	
	Prevalence (%) (95% CI)		Prevalence (%) (95% CI)		Prevalence (%) (95% CI)		Prevalence (%) (95% CI)		Prevalence (%) (95% CI)		Prevalence (%) (95% CI)	
All Aboriginal groups	10.2	(9.8-10.6)	15.2	(14.7-15.6)	12.7	(12.5-13.0)	16.2	(16.0-16.5)	23.3	(23.0-23.5)	20.1	(19.9-20.3)
First Nations	12.0	(11.1-12.8)	16.0	(15.1-16.9)	14.2	(13.5-14.8)	14.9	(14.6-15.3)	23.2	(22.9-23.6)	19.6	(19.3-19.8)
Inuit	8.8	(8.4-9.2)	14.5	(14.0-15.0)	11.7	(11.4-12.0)	9.7	(8.7-10.8)	18.3	(16.9-19.7)	14.0	(13.1-14.9)
Other	10.7	(9.6-11.8)	15.4	(14.2-16.6)	12.9	(12.1-13.8)	18.0	(17.7-18.4)	23.5	(23.2-23.9)	21.0	(20.7-21.2)
15-24 years	1.8	(1.5-2.1)	2.6	(2.3-3.0)	2.2	(2.0-2.4)	3.0	(2.7-3.2)	4.5	(4.2-4.8)	3.8	(3.5-4.0)
25-44 years	6.7	(6.3-7.2)	10.6	(9.9-11.2)	8.7	(8.3-9.1)	10.5	(10.2-10.8)	15.0	(14.7-15.4)	13.1	(12.9-13.3)
45-64 years	20.9	(19.7-22.0)	29.5	(28.3-30.6)	25.3	(24.5-26.1)	29.0	(28.4-29.6)	40.0	(39.5-40.6)	35.0	(34.6-35.4)
65+ years	34.4	(32.0-36.7)	51.8	(49.3-54.3)	43.5	(41.8-45.2)	40.1	(39.0-41.3)	60.8	(59.7-61.9)	51.8	(50.9-52.6)

Abbreviations: CI, confidence interval.

[a] The three Canadian northern territories: Yukon, Northwest Territories, Nunavut.

[b] The 10 Canadian provinces: British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, Quebec, New Brunswick, Prince Edward Island, Nova Scotia, Newfoundland and Labrador.

TABLE 3

Crude prevalence (%) of health risks associated with arthritis among Aboriginal people aged 15 years and over in the North[a] and South[b] of Canada

	North[a]				South[b]			
	Arthritis		No Arthritis		Arthritis		No Arthritis	
	Prevalence (%) (95% CI)		Prevalence (%) (95% CI)		Prevalence (%) (95% CI)		Prevalence (%) (95% CI)	
Daily smoking	44.8	(43.6-46.1)	51.6	(51.1-52.1)	36.2	(35.8-36.7)	29.6	(29.4-29.8)
Obese (BMI ≥ 30)	36.7	(35.4-38.0)	23.7	(23.2-24.2)	33.4	(32.9-33.8)	22.6	(22.4-22.9)
Co-morbid conditions[c]	47.0	(45.8-48.2)	20.4	(20.0-20.8)	61.6	(61.1-62.1)	29.1	(28.9-29.3)

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

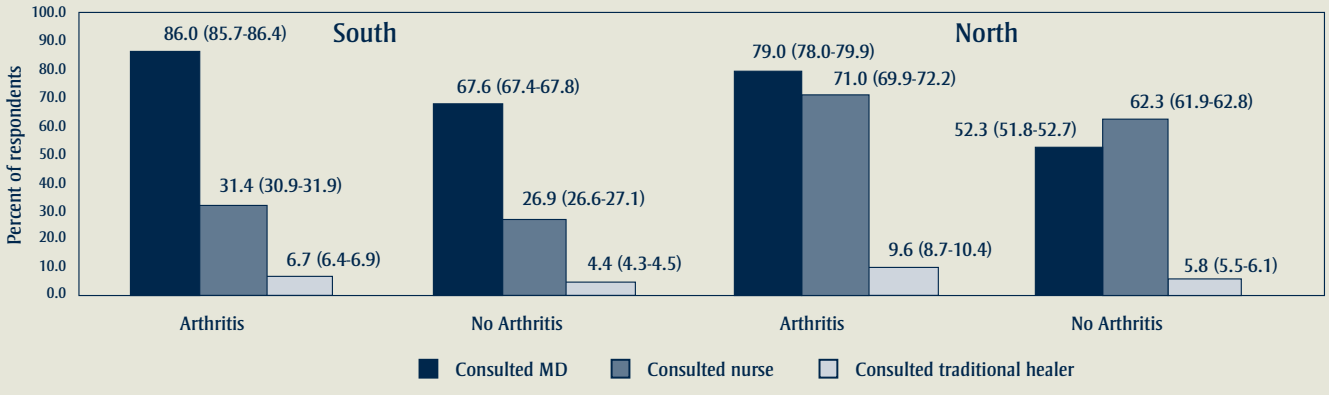
[a] The three Canadian northern territories: Yukon, Northwest Territories, Nunavut.

[b] The 10 Canadian provinces: British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, Quebec, New Brunswick, Prince Edward Island, Nova Scotia, Newfoundland and Labrador.

[c] Co-morbid conditions include diabetes, hypertension, heart disease, asthma, chronic bronchitis, emphysema, cancer and stroke.

FIGURE 1

Utilization of health services by Aboriginal people aged 15 years and over in the North[a] and South[b] of Canada by type of provider and by arthritis status



[a] The three Canadian northern territories: Yukon, Northwest Territories, Nunavut.

[b] The 10 Canadian provinces: British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, Quebec, New Brunswick, Prince Edward Island, Nova Scotia, Newfoundland and Labrador.

healer† anytime in the 12 months preceding the survey was higher among individuals with arthritis than those without the condition. (In the North, OR = 2.32, 95% CI: 2.10-2.56; in the South, OR = 2.25, 95% CI: 2.17-2.33). In the North, arthritis patients consulted nurses and traditional healers more and physicians less frequently than those in the South. (See Figure 1.)

Social conditions

A lower proportion of individuals with arthritis report being employed in the week before the survey (either self-employed or otherwise working for pay) compared to those without arthritis. The association was stronger in the South (OR = 0.58, 95% CI: 0.57-0.59) than in the North (OR = 0.75, 95% CI: 0.70-0.79).

Discussion

The lower prevalence estimates among Aboriginal people in the North compared to those in the South obtained from the APS 2006 are comparable to those from other surveys.[6,7] In Tjepkema's analyses of CCHS 2000/01,[6] the prevalence for Aboriginal people in the North is 10% while that in the South is 19% for rural residents and 20% for urban residents; Lix et al. obtain a prevalence of 12% in the North and 20% in the South from the CCHS 2005/06.[7] Both these studies also show that the prevalence among Aboriginal people is higher than non-Aboriginal people in the South but not in the North. Note that both the CCHS and APS cover the same Aboriginal groups—off-reserve First Nations, Inuit and Métis. Although less access to specialist care may be responsible for the lower detection rate of arthritis in the North, the prevalence of arthritis is based on self-report and not on clinically verified diagnoses by rheumatologists; further, as a chronic disease arthritis is likely to have been diagnosed sometime in the past over the long term even with limited specialist health care.

In surveys such as the APS, CCHS and RHS, self-reports under the rubric "arthritis and rheumatism" lack clinical accuracy. These self-reports are also limited by the inability to differentiate between different types of arthritides—rheumatoid arthritis, osteoarthritis, etc. However, as a tool for assessing population health and the need for health care, such crude measures are nevertheless useful, particularly to describe the patterns in different population subgroups.

The lower prevalence of arthritis among Aboriginal people in the North can also be attributed to the high proportion of Inuit in the population. (According to the 2006 Census, approximately 54% of Aboriginal residents of the northern territories report some Inuit ancestry, compared to 4% of Aboriginal people in Canada as a whole.[14]) A lower prevalence of arthritis among Inuit relative to other Aboriginal people has been shown nationally in APS 2001[9] and CCHS 2000/01.[6] In this study we demonstrate that, within the North, the prevalence of arthritis among all Aboriginal groups—Inuit, First Nations, and Other—is also lower than the corresponding group in the South (Table 2).

It is unclear as to why Canadian Inuit have lower prevalence of arthritis than First Nations people. The self-reported arthritis rubric is a mixed bag of clinical entities with different etiologies. A review of North American indigenous populations found that Inuit tend to have high rates of spondyloarthropathies whereas Native Americans have high rates of rheumatoid arthritis.[1] A study based on clinical records indicates that the Inupiat in the Alaska North Slope region (who are culturally and linguistically related to the Inuvialuit in the Northwest Territories) have high rates of rheumatoid arthritis compared to some Native American tribes, and much higher than the Yupik in western Alaska.[15] A recent study from Alaska that estimated the prevalence of self-reported and clinically undifferentiated arthritis showed that it is higher among Alaska Natives than the general U.S. population, but the Alaskan sample is a mix of Yupik and Native American tribes in the southeastern part of the state.[16]

Aboriginal people suffering from arthritis have unfavourable health profiles; they are more likely to be daily smokers, be obese and have concurrent chronic diseases, although the magnitude differs between the North and South, reflecting the background prevalence of these associated traits and conditions. Arthritis can limit the opportunity for employment, although this survey does not provide evidence that the lower employment rate is the direct result of the disease.

As expected, Aboriginal people with arthritis are more likely to utilize the health care system, with higher proportions reporting visits to physicians, nurses and traditional healers. The pattern of use reflects the different systems in place in the North and South. We cannot, however, determine if the higher health service use is the direct result of arthritis, but it is a plausible explanation given the nature of the disease, the presence of other risk factors such as smoking and obesity, and co-morbidities. In the North, primary care is predominantly delivered by nurses in health centres in the communities, and individuals have only periodic contact with visiting physicians. For many, visits to specialists such as rheumatologists requires air travel away from home.

Further research is required to explore North-South disparities in the burden of arthritis in Aboriginal populations. Also needed are more refined diagnoses, including rheumatoid arthritis, osteoarthritis and other musculoskeletal disorders, as well as separate analyses of Inuit and First Nations samples, which are sufficiently large within the North. Aboriginal-specific findings on arthritis and other chronic diseases, as well as recognition of regional differences between North and South, will enhance program planning and help identify new priorities in health promotion. The creation and transmission of quality evidence to appropriate stakeholders to ensure uptake and application of study findings will help reduce health disparities.

Acknowledgment

Funding for this study was provided by the Canadian Arthritis Network's National Aboriginal Arthritis Research Initiative (project code: 07-NAARI-04).

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Obesity and obesity-related comorbidities in a Canadian First Nation population*

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* This article is part of a joint publication initiative between *Chronic Diseases in Canada* and *Preventing Chronic Disease*. *Chronic Diseases in Canada* is the secondary publisher, while *Preventing Chronic Disease* is the primary publisher. The primary publication can be found at Bruce SG, Riediger ND, Zacharias JM, Young TK. Obesity and obesity-related comorbidities in a Canadian First Nation population. Prev Chronic Dis 2011;8(1). http://www.cdc.gov/pcd/issues/2011/jan/09_0212.htm

Abstract

Introduction: Rates of obesity are higher among Canada's Aboriginal First Nations populations than among non-First Nations populations. We studied obesity and obesity-related illness in a Manitoba First Nation community.

Methods: We conducted a screening study of diabetes and diabetes complications in 2003, from which we drew a representative sample of Manitoba First Nation adults (N = 483). We assessed chronic disease and chronic disease risk factors.

Results: Prevalence of obesity and associated comorbidities was higher among women than men. By using multivariate analysis, we found that factors significantly associated with obesity among women were diastolic blood pressure, insulin resistance, and employment status. Among men, factors were age, apolipoprotein A1 level, apolipoprotein B level, and insulin resistance. Seventy-five percent of study participants had at least 1 of the following conditions: obesity, dyslipidemia, hypertension, or diabetes. Comorbidity was high even among the youngest age groups; 22% of men and 43% of women aged 18 to 29 had 2 or more chronic conditions. Twenty-two percent of participants had undiagnosed hypertension. Participants with undiagnosed hypertension had significantly more chronic conditions and were more likely to have microalbuminuria than were those without hypertension. The number of chronic conditions was not significantly different for participants with newly diagnosed hypertension than for those with previously diagnosed hypertension.

Conclusions: The prevalence of obesity and other chronic conditions in the study community is high, especially considering the number of young people. Community-based interventions are being undertaken to reduce the excessive rate of illness.

Keywords: *cardiovascular, community health, diabetes, minority, obesity, screening, Type 2 diabetes, First Nations, North American Indians*

Introduction

The Canadian First Nations population has poorer overall health than does the general Canadian population,[1] specifically in terms of chronic diseases, chronic disease risk factors,[2] and injuries and accidents.[3] In Canada, First Nations peoples are 1 of 3 constitutionally recognized Aboriginal groups; the other 2 are the Métis and the Inuit. In this article, we use the term *Aboriginal* to report on research that included 2 or more of these distinct groups if no distinction was made between the groups in the analysis. However, if the research included only 1 group, we have identified that group. According to the 2005-2006 Canadian Community Health Survey, the prevalence of obesity among people who self-identified as Aboriginal and who did not live on reserve land was 20% in Canada's north (Yukon, Northwest Territories, and Nunavut) and 23% in the rest of Canada.[4]

Obesity prevalence appears to be higher among First Nations peoples living on reserves. In Sandy Lake, Ontario, the prevalence of obesity (body mass index [BMI] ≥30 kg/m^2) was 50% for men and 65% for women.[5] Furthermore, in a First Nation community in Quebec, 91% of study participants from a sample of 172 were abdominally obese.[6] The prevalence of obesity was 55% among a sample of Alberta First Nation people and 49% among a sample of Métis people.[7]

Prevalence of obesity-related comorbidities is also high among Canadian First Nations people. The prevalence of diabetes among Canadian First Nations populations is 3 to 5 times higher than among the general Canadian population.[5-8] Hypertension, dyslipidemia, metabolic syndrome, and diabetes complications such as cardiovascular disease (CVD), stroke, retinopathy, neuropathy, and nephropathy are also major contributors to poor health.[2,5-10] CVD is the leading cause of death in Canada, and Aboriginal populations have twice the CVD death rate of non-Aboriginal populations.[2] In a random sample, the rate of CVD was 18% among Canadian Aboriginal people and 8% among people of European ancestry.[2]

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Despite the evidence of excess obesity, diabetes, and related metabolic conditions among Canada's First Nations populations, few researchers have investigated their coexistence in this population. Our purpose was to explore the magnitude and effect of obesity and obesity-related comorbidities in a Manitoba First Nation.

Methods

Our methods have been previously described.[10] Briefly, 483 eligible residents of a Manitoba First Nation community volunteered in 2003 to participate in a screening study for diabetes and diabetes complications. A total of 1356 eligible participants included nonpregnant adults aged 18 years or older who were Registered Indians and who were residents of the community. Our sample (36%, 483 of 1356) is representative of eligible participants by age and sex.[10] A registered nurse drew venous samples to measure glucose, hemoglobin A1c, insulin, total cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, apolipoprotein A1 (apoA1), total apolipoprotein B (apoB), and homocysteine levels from fasting participants (low-density lipoprotein [LDL] cholesterol was calculated).

A registered nurse or trained research assistant administered a 17-item questionnaire that included standard demographic data (age, sex, employment status, education level), current and past smoking status, number of cigarettes smoked per day, previous diagnosis of diabetes and hypertension ("Have you ever been told by a doctor that you have diabetes? How long have you had diabetes?"), and current medication use. Standard techniques were used to obtain anthropometric measures.[11] Height was measured via metric wall tape and set square to the nearest 0.5 cm; weight was measured on a balance scale to the nearest 0.1 kg; waist circumference was measured at noticeable waist narrowing or at the level of the 12th rib, to the nearest 0.5 cm; and hip circumference was measured at the level of the symphysis pubis and the largest area of the buttocks to the nearest 0.5 cm.[11]

Abdominal obesity was defined as waist circumference greater than 102 cm for men and greater than 88 cm for women.[10] Diabetes was defined as a fasting plasma glucose of 7.0 mmol/L or higher, or a previous diagnosis; impaired fasting glucose was defined as a fasting plasma glucose of 6.1 to 6.9 mmol/L.[12] Hypertension was defined as systolic blood pressure higher than 140 mmHg or diastolic blood pressure higher than 90 mmHg, or a previous diagnosis. Dyslipidemia was defined as a plasma triglyceride level of 1.7 mmol/L or higher and HDL cholesterol level of 1.03 mmol/L or less for men or 1.30 mmol/L or less for women. Metabolic syndrome was defined using Adult Treatment Panel III criteria.[13] Insulin resistance was estimated through the homeostasic model assessment (HOMA), which is calculated as follows: [(insulin [pmol] x 0.139) x (glucose [mmol/L]/22.5)]. Microalbuminuria was defined as an albumin-to-creatinine ratio higher than 2.0 mg/mmol for men and higher than 2.8 mg/mmol for women. Neuropathy was defined as presence of numbness, tingling, pain, and loss of protective sensation determined through application of the 10-g Semmes-Weinstein monofilament wire system (Sensory Testing Systems, Baton Rouge, Louisiana).[14] A registered nurse completed the foot examination and applied the 10-g monofilament. The University of Manitoba Health Research Ethics Board approved the project.

Statistical analyses were completed by using SPSS version 16 for Windows (IBM, Chicago, Illinois). We used χ^2 tests to detect differences between the sexes for chronic disease prevalence, risk factors, and sociodemographic variables. We compared differences between the sexes on variables that were continuously distributed by using *t* tests or Mann-Whitney tests for variables with a nonnormal distribution. Differences in the number of chronic health conditions by age group and sex and number of comorbidities by hypertensive status were determined by using χ^2 tests. Tests were 2-tailed and differences were considered significant at $p < .05$. We used logistic regression to estimate odds ratios (ORs) for obesity and microalbuminuria with 95% confidence intervals (CIs). Participants with missing values were excluded from analyses. No pattern was found for missing values by sex, age group, chronic disease, or risk factor variables.

Results

The demographic and health status characteristics of the study sample describe a young population with low education and high unemployment (Table 1). The prevalence of smoking, diabetes, hypertension, and overweight and obesity was high among study participants. Waist circumference was available for 259 of the 264 obese participants; 96% (250 of 259) had waist circumferences that placed them at high risk for adverse health outcomes.[9] We found no significant differences between men and women in prevalence of diabetes or hypertension. However, the prevalence of dyslipidemia among women (38%) was significantly higher than among men (26%).

Overall obesity and abdominal obesity

We used BMI and waist circumference to classify participants as obese by age and sex (Figure 1). Almost 50% of men and 65% of women were obese as defined by BMI, and 53% of men and 81% of women had abdominal obesity. Obesity was more common among women than men according to BMI ($\chi^2 = 14.62$, $p < .001$) and abdominal obesity ($\chi^2 = 41.38$, $p < .001$). The prevalence of BMI ≥ 30 kg/m^2 was higher among women aged 18 to 29 years than among men of the same age group ($\chi^2 = 9.06$, $p < .01$). Abdominal obesity was significantly more common for women than for men in all age groups except 40 to 49 years. Three-quarters of women aged 18 to 29 years had abdominal obesity (Figure 1).

TABLE 1
Characteristics of First Nation population (N = 483), Manitoba, Canada, 2003

Characteristic[a]	Value
Sex, n (%)	
Men	230 (48)
Women	253 (52)
Age, y, mean (SD)	37.8 (12.3)
Education (n = 469), n (%)	
Grade 9 or higher	220 (47)
Lower than grade 9	249 (53)
Employment status (n = 476), n (%)	
Employed	137 (29)
Unemployed	339 (71)
Ever smoked (n = 477), n (%)	
Yes	391 (82)
No	86 (18)
Current smoker (n = 471), n (%)	
Yes	349 (74)
No	122 (26)
BMI, kg/m^2 (n = 468), n (%)	
<25.0	76 (16)
25.0-29.9	128 (27)
≥30.0	264 (56)
Metabolic syndrome[b] (n = 475), n (%)	252 (53)
Abdominal obesity[c] (n = 464)	313 (68)
Diabetes[d] (n = 483), n (%)	140 (29)
Hypertension[e] (n = 472), n (%)	201 (43)
Dyslipidemia[f] (n = 483), n (%)	155 (32)
Microalbuminuria[g] (n = 466), n (%)	94 (20)

Abbreviations: SD, standard deviation; BMI, body mass index.

[a] Numerators vary from 464 to 483 because not all participants completed the full protocol.

[b] Defined using Adult Treatment Panel III criteria.[13]

[c] Defined as > 102 cm for men and > 88 cm for women.

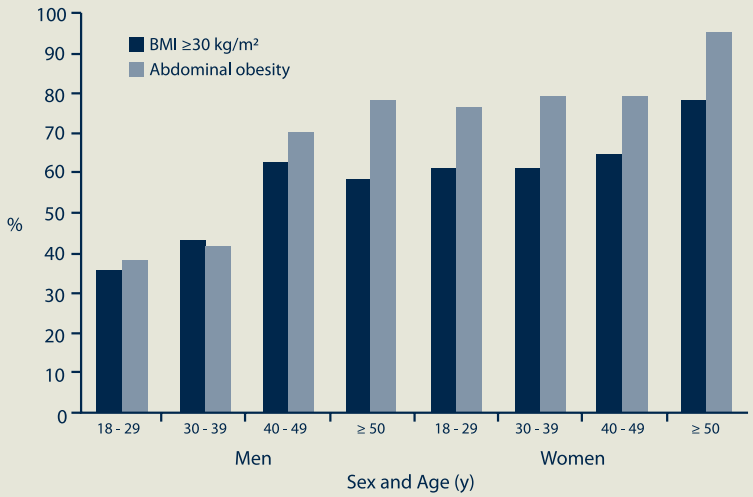
[d] Defined as a previous diagnosis or fasting blood glucose ≥ 7.0 mmol/L.

[e] Defined as systolic blood pressure > 140 mm Hg or diastolic blood pressure > 90 mm Hg or previous diagnosis.

[f] Defined as plasma triglyceride level ≥ 1.7 mmol/L and HDL cholesterol level ≤ 1.03 mmol/L for men or ≤ 1.30 mmol/L for women.

[g] Defined as an albumin-to-creatinine ratio > 2.0 mg/mmol for men and > 2.8 mg/mmol for women.

FIGURE 1
Prevalence of obesity by age and sex in a Canadian First Nation population.



Abbreviations: BMI, body mass index.

Abdominal obesity was defined as waist circumference greater than 102 cm for men and greater than 88 cm for women.

[A tabular version of this figure is also available.]

Given the differences in obesity between men and women and the high prevalence of abdominal obesity, we determined factors associated with abdominal obesity for each sex by using multivariable backward stepwise logistic regression. Variables included in the models were those that were significantly associated with abdominal obesity in bivariate analyses. For women those variables were age; systolic and diastolic blood pressure; triglyceride, apoA1, and apoB levels; insulin resistance; education; and employment status. For men variables included in the model were age; systolic and diastolic blood pressure; triglyceride, apoA1, and apoB levels; insulin resistance; and microalbuminuria (Table 2).

For women, the odds of abdominal obesity increased with diastolic blood pressure and insulin resistance. In addition, the odds of obesity were lower for women who were employed than for those who were unemployed. Among men, abdominal obesity was associated with increasing age, insulin resistance, lower apoA1, and higher apoB levels.

Comorbidities

We determined the extent of comorbidity among this population for 4 chronic conditions: obesity, diabetes, hypertension, and dyslipidemia. The distribution of chronic conditions by age and sex (Figure 2) showed that women aged 18 to 29 and aged 50 or older had significantly more chronic conditions than men of the same age groups. Twenty-two percent (16 of 73) of men and 43% (30 of 69) of women aged 18 to 29 had 2 or more preventable chronic conditions. Among participants with abdominal obesity, 48% (147 of 303) had hypertension and 35% (111 of 313) had diabetes. Thirty-seven percent (54 of 147) of the hypertension and 26% (29 of 111) of diabetes cases among these participants were undiagnosed.

Undiagnosed hypertension

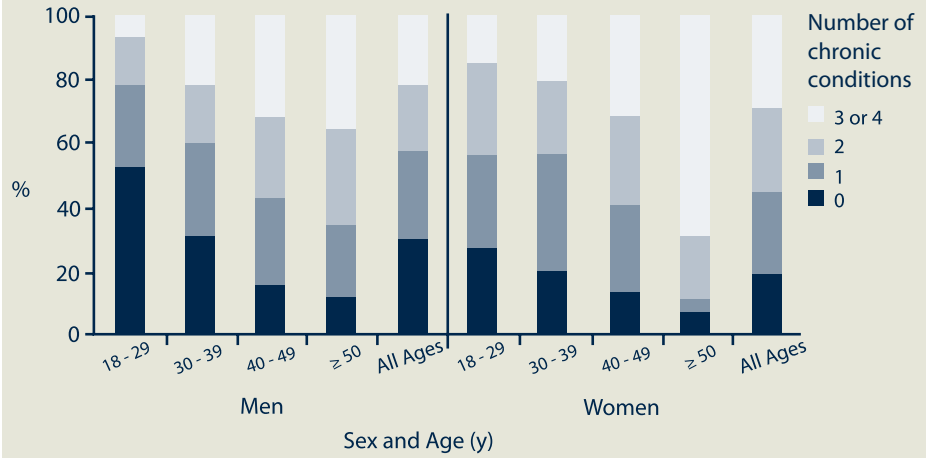
Overall, 22% (72 of 337) of study participants had undiagnosed hypertension. We compared the extent of comorbidity for participants with newly diagnosed hypertension and 2 groups: 1) participants who were not hypertensive and 2)

TABLE 2
Odds of abdominal obesity by sex, First Nation population (N = 483), Manitoba, Canada, 2003

Sex	Risk Factor	β (SE)		OR (95% CI)		*p*-value[a]
Women	Currently employed	- 1.16	(0.45)	0.31	(0.13-0.76)	.01
	Diastolic blood pressure	0.05	(0.02)	1.05	(1.01-1.10)	.03
	Insulin resistance	1.14	(0.205)	0.31	(0.13-0.76)	.01
Men	Age	0.05	(0.01)	1.05	(1.02-1.08)	.001
	ApoA1	- 3.06	(1.20)	0.05	(0-0.49)	.01
	ApoB	1.54	(0.68)	4.64	(1.22-17.65)	.02
	Insulin resistance	0.33	(0.08)	1.40	(1.19-1.63)	<.001

Abbreviations: SE, standard error; OR, odds ratio; CI, confidence interval; apo, apolipoprotein.
[a] Calculated by logistic regression.

FIGURE 2
Percentage of sample with chronic conditions (obesity, diabetes, hypertension, dyslipidemia) by age and sex in a Canadian First Nation population.



[A tabular version of this figure is also available.]

participants with a previous diagnosis of hypertension (Table 3). Participants with newly diagnosed hypertension were significantly more likely to have more chronic conditions than were the normotensive participants. However, we found no significant differences in extent of comorbidity between those with newly diagnosed hypertension and those with previously diagnosed hypertension. In terms of outcomes, the adjusted odds of microalbuminuria among people with newly diagnosed hypertension were almost 2 times higher than among those without hypertension. The adjusted odds of microalbuminura among those with previously diagnosed hypertension were almost 5 times higher than among those without hypertension [< .001 in Table].

Discussion

The prevalence of obesity in the study population is among the highest reported for a Canadian First Nation community on a reserve[6,7] and is substantially higher than that among the general Canadian[4] and off-reserve Aboriginal populations.[4,15] The high prevalence of obesity in the study population is concerning given the etiologic role of obesity in diabetes, heart disease, stroke, and some cancers. The prevalence of diabetes that we found is one of the highest reported among Canadian First Nations populations.[6,7,16]

One finding of concern is the high prevalence of obesity among young adults, especially young women of reproductive age. The relationships between maternal obesity and gestational diabetes, type 2 diabetes, poor birth outcomes, and development of obesity and type 2 diabetes among offspring are well documented.[17-20] Thus, the prevalence of obesity in this young study population warrants intervention. These findings are important for 2 reasons: 1) participants developed chronic conditions at young ages, and 2) hypertension and diabetes cases were undiagnosed among a large proportion of obese participants.

Results from logistic regression confirmed established associations between obesity and plasma lipid levels, hypertension, insulin resistance, and sociodemographic factors in the study population. The sex-specific regression analyses did not include lipids as a predictive factor for abdominal obesity among women. We offer 2 possible reasons for this. First, the prevalence of abdominal obesity was high among women in all age groups but the presence of abnormal lipid levels was not. These age differences may have been blunted because our outcome (obesity) was present in all age groups. Second, previous research has shown significant sex differences in the relationship between adiposity and plasma lipids.[21] Because abnormal lipid levels did occur among women, this finding warrants further examination.

We found a high prevalence of comorbidity even among the youngest age groups. The Diabetes and Related conditions in Urban Indigenous people in the Darwin region (DRUID) study also found high numbers of cardiovascular comorbidities among Australian Aborigines, and a higher number of comorbidities with increasing age.[22] A large proportion of the study participants had undiagnosed diabetes and hypertension, despite the known strong correlations among obesity, diabetes, dyslipidemia, and hypertension[23] (we could not determine the extent of undiagnosed dyslipidemia among study participants because we did not ask them to self-report abnormal lipid levels). In a previous study, risk factors for not having blood pressure measured included male sex, never being married, not having a regular physician, being younger, and belonging to an Aboriginal or other ethnic minority group.[24] In our study, the likelihood of not having hypertension

Table 3
Comorbidities and risk for microalbuminuria by hypertension status, First Nation population, Manitoba, Canada, 2003

Participants' hypertension status (N = 453)[b]	No. of participants (%)					Risk for microalbuminuria[a]		
	No. of comorbidities				*p*-value[c]	β (SE)	Odds Ratio	*p*-value[c]
	0	1	2	3				
No hypertension (n = 263), n (%)	111 (42)	88 (33)	50 (19)	14 (5)	1 [Reference]	1 [Reference]	1.000	1 [Reference]
Newly diagnosed hypertension (n = 72), n (%)	17 (24)	20 (28)	19 (26)	16 (22)	< .001	0.653 (0.48-0.82)	1.921	< .001
Previously diagnosed hypertension (n = 118), n (%)	18 (15)	36 (31)	38 (32)	26 (22)	.510	1.542 (1.22-1.86)	4.673	< .001

Abbreviation: SE, standard error.

[a] Adjusted for age and sex. Those with newly diagnosed hypertension had no significant difference in risk for microalbuminuria compared with those with previously diagnosed hypertension.

[b] In this analysis, we included only participants for whom values were available for all variables.

[c] Calculated by using $\chi 2$ test.

diagnosed was higher for men (OR, 3.27; 95% CI, 1.74-6.10; $p < .01$) and younger participants (OR, 1.04; 95% CI, 1.01-1.07; $p < .001$).

In our study, the undiagnosed hypertension was not benign. The extent of comorbidity among participants with newly diagnosed hypertension was similar to that for those with previously diagnosed hypertension. In addition, the risk for microalbuminuria was significantly higher among participants with newly diagnosed hypertension compared with those without hypertension but not significantly different between those with newly diagnosed hypertension and those with previously diagnosed hypertension. This suggests that newly diagnosed hypertension among participants had existed for some time. The association between hypertension and outcomes such as CVD and stroke warrants vigilant screening on the part of health care providers, especially in high-risk populations. Some participants in our "newly diagnosed" group may have been told by a physician that they did have hypertension, but they may not have remembered or they may have not understood. However, none were receiving antihypertensive treatment, so they probably had not received a hypertension diagnosis before our study.

The study is subject to limitations. First, our sample was based on volunteers and therefore may not be representative of the community as a whole or of other Canadian First Nations communities. A screening study based on a volunteer sample may attract primarily healthy people who are motivated to learn more about their health, resulting in an underestimation of illness. On the other hand, a screening study can attract people who already have health problems and are seeking additional medical assistance, which may result in an overestimation of the prevalence of illness in a population. We do not think our sample was overrepresented by either group because men and women were equally represented, and the age distribution of our sample matched that of the eligible population.[10] Another indication that the prevalence of illness in the community was not overstated is that only half of the community members known to have diabetes participated in the study. None of the 15 people with end-stage renal disease participated, and only 3 of 10 community members with amputations participated.[10] The prevalence of chronic disease and risk factors that we report are not substantially out of line with previous research.

A second limitation is the use of a fasting glucose test rather than a glucose tolerance test. More people with diabetes may have been identified if 2-hr glucose tolerance tests were conducted. However, our protocol is acceptable for epidemiologic research. A third limitation is that we did not validate the self-reported hypertension or diabetes status measures with local health care providers, so we may have underestimated self-reported prevalence and therefore overestimated undiagnosed cases. However, we have previously reported lack of adherence with standards of care in this community in relation to foot examinations among people with diabetes,[10] so participants may not have been tested for diabetes and hypertension even when indicated. Finally, the study is cross-sectional, so we cannot infer the temporal sequence of events.

The prevalence of obesity in this population is among the highest reported among Canadian First Nations populations, particularly among women in their reproductive years. The extent of obesity-related comorbidity in this population is high even among young adults, and women at almost every age have a significantly higher rate of comorbidity than do men. A sizable proportion of participants have undiagnosed hypertension that may have been present for some time, given the significant associations with the other chronic diseases and microalbuminuria. The prevalence of cardiovascular and renal disease risk factors in this population may portend a larger prevalence of cardiovascular and renal disease. In addition, given the influence of maternal obesity and diabetes on the health of offspring, an increase in childhood obesity and type 2 diabetes could occur in the community.

An increasing prevalence of obesity and obesity-related conditions is not inevitable, however. Many prevention activities are under way. First, a research intervention in the community is focused on preventing gestational diabetes through controlling weight gain during pregnancy with exercise and diet. Second, the community operates a fitness center that has good equipment and instruction. Third, the health center offers education on diet, exercise, and wellness. Fourth, walking groups for youth and adults are organized through the health center. Fifth, activity programs for young people operate out of the local schools. However, given the well-established effect of obesity on health, continued surveillance of chronic disease and risk factors is warranted, as are further health promotion and health education

initiatives. We continue to work with the community to develop and evaluate primary and secondary prevention activities.

Acknowledgments

We thank the Canadian Institutes of Health Research (CIHR) and the Manitoba Health Research Council for their funding for this project. Dr Riediger is the recipient of a CIHR Doctoral Canada Graduate Scholarship. We are grateful for the statistical assistance of Mary Cheang. Finally, we thank the study community, staff, and leadership for their participation and ongoing commitment.

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Cardiovascular risk according to plasma apolipoprotein and lipid profiles in a Canadian First Nation*

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* This article is part of a joint publication initiative between *Chronic Diseases in Canada* and *Preventing Chronic Disease*. *Chronic Diseases in Canada* is the secondary publisher, while *Preventing Chronic Disease* is the primary publisher. The primary publication can be found at Riediger ND, Bruce SG, Young TK. Cardiovascular risk according to plasma apolipoprotein and lipid profiles in a Canadian First Nation. Prev Chronic Dis 2011;8(1). http://www.cdc.gov/pcd/issues/2011/jan/09_0216.htm

Abstract

Introduction: Despite high diabetes rates among Canadian First Nations people, little is known about their cardiovascular disease risk. Our aim was to describe the apolipoprotein profile with respect to cardiovascular risk in a Canadian First Nation community.

Methods: In 2003, a representative sample of adult members of a Manitoba First Nation (N = 483) participated in a screening study for diabetes and diabetes complications. We assessed their cardiovascular risk factors.

Results: Sixty percent of women were at increased cardiovascular risk because of low apolipoprotein A1 (apoA1) levels, compared with 35% of men. The proportion of women with low apoA1 levels decreased with age, but the proportion with low high-density lipoprotein levels remained stable across age groups. Both apoB and apoA1 were significantly associated with obesity when age, sex, diastolic blood pressure, homocysteine, diabetes, and insulin resistance were controlled for.

Conclusion: Apolipoprotein and lipid profiles in this First Nation population suggest high cardiovascular risk. Future research should characterize the lipoprotein particle size in this population.

Keywords: *cardiovascular, community health, diabetes, epidemiology, obesity, screening, minority, First Nations, North American Indians*

Introduction

In recent years, the prevalence of cardiovascular disease among Aboriginal people in Canada has been increasing and is now higher than the prevalence among non-Aboriginal people. In a random sample of Canadian Aboriginal people, the prevalence of cardiovascular disease was 18%, compared with 8% in Canadians of European descent.[1]

Apolipoprotein A1 (apoA1) is the major protein of high-density lipoprotein (HDL), and apoB is among the major proteins of very low-, low- (LDL), and intermediate-density lipoproteins. Because of their associations with the respective lipoproteins, apoA1 is inversely and apoB is positively associated with cardiovascular risk.[2] In fact, evidence suggests that apoA1 and apoB are better predictors of heart disease risk than are HDL and LDL cholesterol levels.[3-5] Apolipoproteins may also offer advantages over lipoprotein cholesterol measurements because they are direct measurements, whereas LDL, for example, is calculated from other lipoproteins from a fasting blood sample.

Despite the high rate of diabetes and cardiovascular disease among Canadian Aboriginal people, little research has gone beyond examining traditional risk factors. In addition, research has been mostly based on chart review, and the few population-based studies that have been conducted have been limited to a single First Nation community.[6-8] Our objective was to describe the apolipoprotein profile and its relationship to cardiovascular risk factors in a Canadian First Nation community.

Methods

We conducted this study with data from a larger community-based screening study on diabetes complications.[9] The sample of 483 men and women from a Manitoba First Nation was representative in terms of age and sex. Eligible participants (n = 1356) were nonpregnant adults aged 18 years or older who were Registered Indians and lived in the community. The community is approximately 200 km northwest of Winnipeg, Manitoba. Data were collected from January through December 2003, and each participant had all of his or her data collected on the same day. Further details on the study can be found elsewhere.[9] The study was approved by the University of Manitoba Health Research Ethics Board.

A registered nurse collected a fasting blood sample, and we assessed levels of plasma glucose, insulin, triglyceride, HDL cholesterol, LDL cholesterol, total cholesterol, total apoB, and apoA1. The nurse also measured blood pressure,

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urinary albumin and creatinine levels, and anthropometric characteristics.[9] To assess insulin resistance, we used the homeostatic model assessment with glucose and insulin results in the following equation: (insulin [pmol] × 0.139) × (glucose [mmol/L]/22.5). Risk factors assessed in this study are defined in Table 1.

We analyzed all data by using SPSS version 16.0 for Windows (IBM, Chicago, Illinois). We compared plasma lipid levels by using *t* tests and Mann-Whitney U nonparametric tests. We compared differences in apolipoprotein levels by cardiovascular risk factors by using both *t* tests and Mann-Whitney U tests for variables with a nonnormal distribution or unequal variances. We used χ^2 tests to detect differences in risk for cardiovascular disease by apolipoprotein category. Tests were 2-tailed, and *p*-values < .05 were considered significant. To determine linear trends for mean apolipoprotein values by age group, we used 1-way analysis of variance with linear contrast. We estimated odds ratios for obesity by using backward stepwise multivariate logistic regression. We included variables in the model that were significantly associated with obesity in bivariate analyses. Those variables were age, sex, ever having smoked, systolic and diastolic blood pressure, presence of diabetes, triglyceride level, apoA1 level, apoB level, insulin resistance, homocysteine level, and microalbuminuria.

TABLE 1
Risk factors assessed in a study of cardiovascular risk in a Canadian First Nation, 2003

Risk Factor	Definition	
	Men	Women
Obesity	BMI ≥ 30.0 kg/m^2	
High-risk WC	WC > 102 cm	WC > 88 cm
Diabetes	Self-report diagnosis, taking an oral hypoglycemic agent, or fasting glucose ≥ 7.0 mmol/L	
Hypertension	Self-report of diagnosis, SBP >140 mm Hg, or DBP > 90 mm Hg	
Dyslipidemia	Fasting plasma TG ≥ 1.7 mmol/L and fasting plasma HDL cholesterol ≤ 1.03 mmol/L	Fasting plasma TG ≥ 1.7 mmol/L and fasting plasma HDL cholesterol ≤ 1.3 mmol/L
Microalbuminuria[a]	ACR > 2.0 mg/mmol	ACR > 2.8 mg/mmol
Metabolic syndrome	Adult Treatment Panel III criteria[10]	
Cardiometabolic risk	At-risk WC plus plasma TG ≥ 1.7 mmol/L	
Low apoA1	ApoA1 < 1.07 g/L	ApoA1 < 1.22 g/L
High apoB	ApoB > 1.2 g/L	
High apoB:apoA1 ratio[5]	> 0.8	> 0.7

Abbreviations: BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triglyceride; HDL, high-density lipoprotein; ACR, albumin-to-creatinine ratio; apo, apolipoprotein.

[a] Determined by using the Bayer DCA 2000 point-of-care analyzer (Elkhart, Indiana).

TABLE 2
Plasma lipid levels among 483 Canadian First Nations adults, 2003

Lipid[a]	Men (n = 230), Mean (SD)	Women (n = 253), Mean (SD)	*p*-value[b]	Men and Women, Mean (SD)
Triglyceride, mmol/L	2.3 (2.5)	2.1 (2.0)	.86	2.2 (2.3)
	1.7 (1.1-2.6)[c]	1.7 (1.2-2.5)[c]		1.7 (1.2-2.6)[c]
LDL cholesterol, mmol/L	2.9 (0.9)	2.6 (0.9)	< .001	2.7 (0.9)
HDL cholesterol, mmol/L	1.2 (0.3)	1.2 (0.3)	< .04[d]	1.2 (0.3)
Total cholesterol, mmol/L	5.0 (1.2)	4.8 (1.1)	.07	4.9 (1.2)

Abbreviations: SD, standard deviation; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

[a] Mean LDL and total cholesterol values are provided, although these were not included in the definition of dyslipidemia because their recommended levels vary according to other risk factors (http://www.cfpc.ca/english/cfpc/programs/patient%20education/cholesterol/default.asp).

[b] Independent *t* test for differences between sex, unless otherwise noted.

[c] Data presented as median (interquartile range) because of skewed distribution; statistical analysis with Mann-Whitney test.

[d] Mann-Whitney test (unequal variances).

Results

Risk for cardiovascular disease was high according to traditional cardiovascular risk factors such as HDL cholesterol and triglyceride levels (Table 2). Rates of obesity, diabetes, hypertension, and microalbuminuria in this population were also high (Table 3).

Significantly more women than men had apoA1 values that indicated cardiovascular risk (60% vs 35%; $p < .001$). Almost 18% of men and 12% of women had apoB values that indicated cardiovascular risk, but the difference was not significant. The proportion of participants with increased risk according to the apoB:apoA1 ratio was 54% for men and 57% for women, and the difference did not reach significance.

Mean apoB concentrations and the apoB:apoA1 ratio were significantly higher in men and participants with any cardiovascular risk factor (Table 3). Mean apoA1 concentrations were lower in patients with most cardiovascular risk factors, but the difference did not reach significance in patients with diabetes, hypertension, microalbuminuria, or cardiometabolic risk.

Cardiovascular risk tended to increase with age (Tables 4 and 5). We found a significant linear trend for age in men for apoB and apoB:apoA1 ratio. We also found a significant and positive linear trend for age in women for apoB and apoB:apoA1 ratio. Conversely, cardiovascular risk according

TABLE 3
Plasma apolipoprotein levels by sex and risk factors for cardiovascular disease among 483 Canadian First Nations adults, 2003[a]

Characteristic[b]	n (%)	ApoB (g/L)	*p*-value	ApoA1 (g/L)	*p*-value	ApoB:ApoA1 ratio	*p*-value
Sex							
Men	230 (48)	0.94 (0.28)	.046	1.14 (1.03-1.23)[c]	.004[d]	0.84 (0.63-1.02)[c]	.001[d]
Women	253 (52)	0.89 (0.26)		1.17 (1.05-1.31)[c]		0.75 (0.59-0.91)[c]	
Obese							
Yes	265 (56)	0.97 (0.96)	< .001	1.13 (0.17)	< .001	0.87 (0.24)	< .001
No	204 (44)	0.83 (0.79)		1.20 (0.19)		0.71 (0.24)	
At-risk waist circumference							
Yes	313 (68)	0.96 (0.26)	< .001	1.15 (0.18)	.006	0.85 (0.25)	< .001
No	151 (32)	0.82 (0.26)		1.20 (0.18)		0.70 (0.23)	
Diabetes							
Yes	140 (29)	1.05 (0.29)	< .001	1.16 (0.19)	.92	0.91 (0.26)	< .001
No	343 (71)	0.86 (0.25)		1.17 (0.18)		0.75 (0.23)	
Hypertension							
Yes	201 (43)	0.99 (0.28)	< .001	1.18 (0.19)	.10	0.85 (0.27)	< .001
No	271 (57)	0.86 (0.25)		1.15 (0.18)		0.76 (0.23)	
Microalbuminuria							
Yes	94 (20)	1.01 (0.81-1.26)[c]	< .001[d]	1.15 (0.17)	.49	0.90 (0.26)	< .001
No	372 (80)	0.86 (0.68-1.05)[c]		1.17 (0.18)		0.77 (0.24)	
Cardiometabolic risk							
Yes	212 (45)	1.05 (0.25)	< .001	1.16 (0.19)	.54	0.93 (0.24)	< .001
No	255 (55)	0.80 (0.23)		1.17 (0.18)		0.70 (0.21)	
Dyslipidemia							
Yes	155 (32)	1.03 (0.90-1.19)[c]	< .001[d]	1.07 (0.98-1.18)[c]	< .001[d]	0.98 (0.23)	< .001
No	328 (68)	0.81 (0.65-1.01)[c]		1.19 (1.09-1.31)[c]		0.72 (0.22)	
Metabolic syndrome							
Yes	252 (53)	1.02 (0.26)	< .001	1.13 (0.17)	< .001	0.91 (0.75-1.05)[c]	< .001[d]
No	223 (47)	0.80 (0.23)		1.20 (0.18)		0.64 (0.53-0.80)[c]	

Abbreviation: apo, apolipoprotein.

[a] Values for apoA1, apoB, and apoB:apoA1 ratio are given as mean (standard deviation), and differences were assessed by using independent-samples *t* tests, unless otherwise noted.

[b] Definitions for characteristics are provided in Table 1. Data were not available for all participants for every characteristic.

[c] Results reported are median (interquartile range) because of skewed distribution.

[d] Mann-Whitney U (nonparametric) test.

to apoA1 decreased with age among women, and mean apoA1 levels increased with age. HDL cholesterol levels did not significantly increase or decrease with age among women (data not shown).

The final logistic model for presence of obesity included age, sex, diastolic blood pressure, diabetes, homocysteine, insulin resistance, apoA1, and apoB. A person with an apoA1 value of 1.14 g/L was 1.2 times as likely to be obese as was a person with an apoA1 of 1.20 g/L. Furthermore, the odds of obesity were 1.35 times as high for a person with an apoB level of 1.00 g/L as for a person with a level of 0.80 g/L.

Discussion

According to plasma lipid levels and apolipoprotein profiles, the risk for cardiovascular disease is high among Canadian First Nations people. The abnormal plasma apolipoprotein concentrations we found are consistent with the high prevalence of obesity and diabetes in the community. Generally, participants had low HDL cholesterol, low apoA1, and high triglyceride levels, which typically coexist in people with insulin resistance.

The average lipid profile in the study community differed dramatically from that of the US population in general,[11] most likely because of the high prevalence of diabetes in the community. For example, mean plasma LDL cholesterol levels among study participants were lower than those among NHANES[†] participants for both sexes. However, plasma HDL cholesterol levels were lower and plasma triglyceride

[†] National Health and Nutrition Examination Survey

TABLE 4
Plasma apolipoprotein levels by sex and age among 481[a] Canadian First Nations adults, 2003

Sex and Age, y	Mean (SD) ApoB	*p*- value[b]	Mean (SD) ApoA1	*p*- value[b]	Mean (SD) ApoB:ApoA1 ratio	*p*- value[b]
Men (n = 229)						
18-29 (n = 72)	0.79 (0.26)	< .001	1.12 (0.14)	.12	0.71 (0.25)	< .001
30-39 (n = 65)	0.95 (0.25)		1.14 (0.15)		0.85 (0.25)	
40-49 (n = 49)	1.09 (0.26)		1.13 (0.16)		0.97 (0.24)	
≥ 50 (n = 43)	1.01 (0.25)		1.17 (0.17)		0.88 (0.24)	
Women (n = 252)						
18-29 (n = 70)	0.78 (0.24)	< .001	1.15 (0.20)	.03	0.69 (0.22)	.001
30-39 (n = 78)	0.89 (0.22)		1.20 (0.17)		0.75 (0.21)	
40-49 (n = 59)	0.96 (0.27)		1.21 (0.21)		0.81 (0.25)	
≥ 50 (n = 45)	0.99 (0.29)		1.22 (0.22)		0.83 (0.28)	

Abbreviations: SD, standard deviation; apo, apolipoprotein.

[a] For 2 participants, the blood sample was insufficient to assess apoA1 and apoB; priority was given to measuring the other plasma lipids (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglyceride).

[b] Analysis of variance with linear contrast.

TABLE 5
Canadian First Nations adults at risk for cardiovascular disease by plasma apolipoprotein levels, 2003 (N = 481)[a]

Sex and Age, y	n (%) ApoB	*p*- value[b]	n (%) ApoA1	*p*- value[b]	n (%) ApoB:ApoA1 Ratio	*p*- value[b]
Men (n = 229)						
18-29 (n = 72)	5 (7)	.003	25 (35)	.87	20 (29)	< .001
30-39 (n = 65)	10 (15)		24 (37)		37 (57)	
40-49 (n = 49)	15 (31)		18 (37)		40 (82)	
≥ 50 (n = 43)	10 (23)		14 (33)		25 (58)	
Women (n = 252)						
18-29 (n = 70)	5 (7)	.006	52 (74)	.006	30 (43)	.01
30-39 (n = 78)	6 (8)		43 (55)		45 (58)	
40-49 (n = 59)	10 (17)		37 (63)		38 (65)	
≥ 50 (n = 45)	10 (22)		20 (44)		29 (64)	

Abbreviation: apo, apolipoprotein.

[a] Cutoffs for increased cardiovascular risk are shown in Table 1. For 2 participants, the blood sample was insufficient to assess apoA1 and apoB; priority was given to measuring the other plasma lipids (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglyceride).

[b] χ^2 Test with linear association.

levels were much higher among our study participants than among the general American population. Compared with Australian Aboriginal people and Torres Strait Islanders,[12] our study participants had slightly lower triglyceride and higher HDL cholesterol levels. However, these levels are still worse than those among other Canadian Cree-Ojibwa peoples, Inuit, or non-Aboriginal people, as assessed in the early 1990s.[13] In addition, triglyceride and HDL cholesterol levels were higher and LDL cholesterol levels were lower in the study community than among another Canadian Aboriginal community;[1] the other Aboriginal community, however, included only people aged 35 to 75 years, whereas our study sample included people aged 18 years or older, and the mean age was 38 years. Neither our lipid values nor the others described in this paragraph were age- or sex-standardized.

Paradoxically, the proportion of women with low apoA1 levels (higher cardiovascular risk) significantly decreased with age, and mean apoA1 levels had a significant positive linear trend with older age. The change in apoA1 levels by age in women seems to indicate more dyslipidemia in younger women. However, the proportion of women with HDL cholesterol levels that indicated increased risk remained stable across age groups. Therefore, for the same HDL cholesterol levels, older women (≥ 50 y) had lower apoA1 levels than did younger women (< 50 years). This phenomenon may reflect a shift in HDL particle size in the older age groups, in which the proportion of small, dense HDL particles increases relative to large HDL particles. This explanation is further

supported by the fact that the prevalence of diabetes increased with increasing age in our study.

Despite a likely genetic susceptibility to diabetes and its comorbidities in this First Nation community, we hypothesize that much of the dyslipidemia can be attributed to poor diet and inactivity. In the past, other indigenous communities, such as the Greenland Inuit, actually had more favorable lipid profiles than did nonindigenous people, most likely because of their traditional lifestyle.[14] When compared with Danish controls, the Inuit had significantly higher apoA1 and significantly lower apoB, LDL cholesterol, total cholesterol, and triglyceride levels. Although apoA1 levels were significantly higher among the Inuit, HDL cholesterol levels were not, which indicates potential differences in the types of HDL between the 2 groups (differences in particle size). The Inuit may have a disproportionate number of atherogenic small, dense HDL particles, as opposed to the more beneficial large HDL particles, which is what we hypothesize in our study community, particularly among women. In a Canadian Oji-Cree First Nation community, although apoA1 levels were significantly lower among men with hypertriglyceridemic waist, apoA1 levels were actually nonsignificantly higher among women with hypertriglyceridemic waist.[15] This apoA1 sex difference may partially explain the higher risk for coronary heart disease for women with diabetes than men with diabetes.[16]

A preponderance of small, dense LDL and small HDL particles is associated with obesity,[17] increased risk of coronary artery disease,[18] and insulin resistance, regardless of diabetes status.[19] Low HDL cholesterol concentrations among people with diabetes may indicate a specific reduction in large HDL particles (as well as a possible increase in small HDL particles), which may not necessarily significantly reduce apoA1 levels.[19] In our study, because of the high rate of diabetes, we suspect that the low HDL cholesterol levels reported are due to the loss of large HDL particles, especially among older women. Women may also have lower HDL cholesterol levels than men in response to obesity,[20] so apoA1 levels may also differ between men and women in response to obesity.

The apparent conflicting results of apoA1 and HDL cholesterol levels may affect the predictive nature of apoA1 and the apoB:apoA1 ratio for cardiovascular risk in this population. Future research should determine the association of apoA1 and apoB:apoA1 ratio on cardiovascular outcomes in First Nations peoples with diabetes. The apoB:apoA1 ratio was found to predict metabolic syndrome in nonobese but not obese participants,[21] perhaps because obese participants are more likely to have diabetes. Mean apoA1 values were virtually identical in our study community, regardless of diabetes status, but mean HDL cholesterol levels were significantly lower in participants with diabetes. This finding supports the notion of an HDL profile of mostly small HDL particles in people in the community, which would keep plasma HDL levels low while increasing apoA1 levels.

Our study has several limitations. Although we cannot say whether the sample was completely representative of the population, it was representative according to age and sex and did not consist of those in the poorest health. Only 105 of the 275 community members with previously diagnosed diabetes participated. In addition, 3 of 10 community members with amputations and none of 15 members with end-stage renal disease participated. We also did not assess lipoprotein particle size and the distribution of sizes among the various types of lipoproteins. Finally, because of the cross-sectional nature of the data, no outcome data are available.

In conclusion, this community is at high cardiovascular risk, according to both plasma lipid and apolipoprotein profiles. Much of this risk is mediated by the high number of community members with diabetes and obesity and the associated changes in lipoprotein profile. More data regarding lipoprotein particle size and the distribution of small, medium, and large HDL and LDL particles are needed to confirm our hypotheses, but these preliminary data can be used to guide interventions that reduce the prevalence of chronic disease in Canadian First Nations people.

Acknowledgments

We acknowledge financial support for this project from the Canadian Institutes of Health Research (CIHR) and the Manitoba Health Research Council. Natalie Riediger is the recipient of a CIHR Doctoral Canada Graduate Scholarship.

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The role of public health in addressing child maltreatment in Canada

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Abstract

Child maltreatment is a significant health and social issue given its prevalence across the general population and the significant short- and long-term outcomes associated with maltreatment in childhood. There is a need for a comprehensive, collaborative and multisectoral approach for identification, prevention and intervention of this complex issue. Within this multisectoral collaboration, it is essential for public health in Canada to define its role in addressing and preventing child maltreatment. This commentary summarizes how public health can address the issue of child maltreatment in Canada by specifically: 1) measuring the magnitude of maltreatment through public health surveillance systems such as the Canadian Incidence Study of Reported Child Abuse and Neglect; 2) identifying modifiable risk factors; 3) identifying and evaluating community-based interventions to prevent violence; and 4) implementing evidence-based primary prevention strategies.

Keywords: *primary prevention, public health, child abuse, intervention, nurse home visitation, Nurse-Family Partnership, child maltreatment, surveillance*

Introduction

Child maltreatment involves the harm, or the potential for harm, to a child or youth by an adult who they trust or depend upon.[1] This harm may occur through either an act of commission (e.g. physical, sexual or emotional abuse) or an act of omission (e.g. physical, emotional or medical neglect, failure to supervise or exposure to violence).[1] Maltreatment in childhood is associated with short- and long-term physical, social, emotional and cognitive impairment that can last a lifetime.[2,3] Exposure to maltreatment in childhood is common: approximately one-third of adult Canadians report histories of physical or sexual abuse or both during childhood.[4]

Child maltreatment can be difficult to identify, as there is a lack of consensus across jurisdictions and sectors (health care, law, education, justice) about which "acts" constitute abuse. As a health and social issue, child maltreatment is difficult to prevent, as there are risk indicators at individual, family and societal levels to address. It is also difficult to intervene or treat child maltreatment as different sectors have distinct roles and responsibilities for responding to maltreatment. Given this complexity, there is a need for a comprehensive, collaborative and multisectoral approach for identification, prevention and intervention.

While all professionals working in the different sectors have a legal responsibility to report suspected or observed maltreatment to child welfare services, the different sectors have unique roles in responding to the issue (Table 1). It is essential that public health in Canada defines its role in addressing child maltreatment within such a multisectoral collaboration as it is the sector that connects the biological and individual determinants of impairment with the social, economic and political determinants that influence population health.[5]

Public health approach to child maltreatment

This commentary summarizes how a public health approach to child maltreatment can be applied in Canada by specifically: 1) measuring the magnitude of maltreatment through public health surveillance systems; 2) identifying modifiable risk factors; 3) identifying and evaluating community-based interventions to prevent violence; and 4) implementing evidence-based primary prevention strategies.

Public health approach to addressing problems

The public health approach[5-7] to addressing problems has four distinct steps: 1) measurement of the scope and magnitude of the problem using surveillance and epidemiological methods; 2) identification of the causes and correlates associated with the problem, including any risk or protective indicators that may be modified through intervention or prevention programs; 3) development, implementation and subsequent evaluation of interventions; and 4) implementation of those evidence-based interventions that have been determined to affect relevant and clinically important outcomes.

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Measurement of child maltreatment in Canada

At the federal level, the Public Health Agency of Canada (PHAC) defines and measures the incidence of and risk indicators associated with child maltreatment, and the service outcomes of child maltreatment investigations. An emerging priority is to ensure that these data are accessible to provincial/territorial and regional public health decision makers responsible for defining public health issues and implementing primary prevention programs. In public health, the scope and magnitude of an issue is measured using epidemiological and surveillance data; PHAC coordinates the Canadian Incidence Study of Reported Child Abuse and Neglect (CIS),[8] a national public health surveillance system conducted every five years to capture data on five categories of maltreatment in children from birth to 15 years of age: neglect, emotional abuse, exposure to domes tic violence, sexual and physical abuse. In the fall of 2003, child protection workers collected data for the CIS-2003 from a representative sample of 63 child welfare service areas across Canada that followed a total of 14 200 child maltreatment investigations. In Quebec, data were extracted from an administrative information system; in all other provinces and territories, child protection workers completed a standardized assessment form.[8] Data collected included the characteristics of the investigated child(ren), details of the maltreatment investigation, level of substantiation, child health outcomes, service dispositions, and family and household characteristics.

However, Pless argues that public health surveillance systems are often focussed on the diligent collection and analysis of data and that these findings are rarely shared in a timely fashion with those decision makers motivated to and reponsible for taking action and implementing prevention programs.[9] He concludes by stating that "the ideal solution is to make surveillance serve the goal of prevention." Although the CIS is coordinated through PHAC, the CIS findings are primarily communicated to the general public or audiences of child welfare decision makers[10] mandated to identify maltreated children and to deliver secondary prevention programs to reduce rates of recurrence and impairment associated with abuse and neglect.[11] To support the planning, implementation and evaluation of primary prevention programs, the CIS surveillance data should be disseminated to decision makers and public health researchers in those provincial and local-level public health agencies responsible for implementing such programs. Increased access to child maltreatment surveillance data would give targeted public health decision makers: 1) increased awareness of the CIS findings; 2) statistics to position child maltreatment as a public health priority and thus prioritize resource allocation towards primary prevention programs; 3) information on child maltreatment trends; 4) data on risk indicators that can be modified through public health interventions; 5) augmented understanding of referral patterns to child welfare by public health professionals; 6) opportunites to identify research priorities related to maltreatment; and 7) the ability to identify populations for targeted primary prevention programs.

At present, the CIS provides the best available snapshot on the incidence of child maltreatment in Canada; however, there are several limitations to this national surveillance system. First, the utility of the results are limited in that the findings provide national level data and are not valid at the local level due to sampling procedures.[8] Within each cycle, however, provinces and territories can provide resources for oversampling to obtain jurisdictional estimates of the magnitude of child maltreatment. Second, there has been no formal evaluation of the CIS surveillance system, so its effectiveness at collecting, analyzing and disseminating the data is unknown. Third, to plan culturally relevant prevention programs, it is essential to collect accurate data, particularly from the groups determined to be at risk. The CIS has not yet obtained a representative sample of First Nations agencies, although the number of participating agencies is increasing with each subsequent CIS cycle—a promising finding.

Fourth, the most significant limitation is that the surveillance findings are determined by a single source of data—child welfare agency reports. Thus, while the CIS is a rich source of information on child welfare investigations, because it does not include information on police investigations of maltreatment or unreported cases of abuse or neglect, the true burden of maltreatment in the population is underestimated. To garner a comprehensive understanding of the health of Canadian children, what is required is a network of public health surveillance systems linked to other health information surveys and sources characterized by common data elements, mechanisms for timely data collection and distribution, and ease of access to the data.[12] In the United States, the Centers for Disease Control and Prevention is developing alternate systems to collect information from hospital and emergency departments on fatal and non-fatal child maltreatment and on victims of violence to develop a National Violent Death Reporting System.[13] These data sources will be used together with findings from the National Child Abuse and Neglect Data System and the US National Incidence Study of Child Abuse and Neglect, which includes sentinel surveys of community professionals working with children and families outside of the child welfare system,[14] to more accurately measure child maltreatment. In Australia, there is a system that links data from different health and social service administrative datasets for all children to enhance the quality of child protection data.[15] In Canada, PHAC, given its establishment and coordination of the CIS and other health surveillance systems, is optimally positioned to collaborate in the development of an integrated surveillance system that would be informed by more than just reports investigated by child welfare.

Identification of determinants of child maltreatment

Multiple individual, household and community risk indicators are associated with physical abuse, sexual abuse and neglect.[16] The CIS provides information on risk factors for public health researchers and decision makers to conduct secondary analyses to answer relevant questions around child maltreatment and to organize into conceptual frameworks as foundations for developing child maltreatment

prevention strategies;[5] these risk factors include child characteristics (e.g. age, gender, ethnic background), caregiver and child functioning (e.g. exposure to intimate partner violence, substance abuse, mental health issues, parental history of childhood maltreatment) and household characteristics (e.g. structure, family size, income, employment). While not intended for collecting community level data, the CIS also provides some insight into social determinants of maltreatment such as housing problems, low employment rates and poverty.

Identification and development of prevention strategies

Across health and social service sectors, public health departments are responsible for identifying or developing primary prevention interventions or programs to address core public health issues at a population health level. The challenge is that each specific category of child maltreatment is associated with unique but sometimes overlapping risk indicators that require abuse-specific interventions. In a recent review of child maltreatment interventions, only one parenting program was identified as effective at preventing the recurrence of physical abuse and no interventions were identified as preventing the recurrence of neglect;[11] a small number of interventions resulted in improved behavioural or mental health outcomes in children who had been neglected, exposed to intimate partner violence or sexually abused.[11] A systematic review of parenting interventions identified complex, multifaceted home visitation programs targeting at-risk families as effective at preventing unintentional injuries in children, a proxy measure of neglect.[17] Two recent integrative reviews identified the Nurse-Family Partnership (NFP) program as the best available means of preventing child maltreatment;[11,18] nurses frequently visit targeted young, low-income, first-time mothers from pregnancy (less than 29 weeks gestation) until the child is 2 years of age. Three randomized controlled trials (RCT) have demonstrated multiple consistent and enduring beneficial maternal and child health outcomes.[19-21] Further, the NFP program results in significant cost savings; the economic benefits are most likely due to the ability of the program to increase high school graduation rates and help mothers find employment, while also reducing rates of child physical abuse and neglect, substance abuse, crime and use of social welfare.[22] The NFP is consistently identified as having higher benefit-to-cost ratios per participant than most other prevention programs for parents of infants and young children in the United States.[22-24]

If there are no rigorously evaluated effective interventions, public health researchers can build upon identified risk and protective factors and established theoretical models to develop and test primary prevention interventions. Mrazek and Haggerty[25] developed a comprehensive framework that many consider the "gold standard" for guiding the development of such interventions. This framework complements the steps of the public health approach and includes five fundamental stages: 1) problem identification and measurement; 2) identification of risk and protective factors and theoretical models from multiple fields; 3) intervention development, training of interveners and conduct of small-scale pilot or feasibility studies leading to an RCT that replicates the intervention; 4) conduct of large-scale RCT to establish effectiveness; and 5) broad implementation of the intervention and ongoing program evaluation.

Implementation and evaluation of evidence-based interventions and policies

There is a pressing need, especially when resources are limited, for all public health departments to implement effective interventions for at-risk families, rather than providing programs that have not been proved adequate or sufficient. The NFP program is internationally recognized as the intervention most capable of preventing child maltreatment.[11] Widely implemented across the United States, this innovative program is currently being evaluated and replicated in England, Scotland, Germany, the Netherlands and Australia.[26] The NFP program is an example of a primary prevention intervention that falls within the scope of public health nurse practice; given the provincial/territorial responsibilities for coordinating and funding direct health services, it would be suitable for delivery by a regional public health agency.

Currently, all provinces and territories (with the exception of Nunavut) have implemented home visitation programs, some with a universal postpartum component and all targeting parents and households characterized by risk indicators associated with poor child health and development outcomes.[27] These programs are primarily offered through or in collaboration with community public health agencies. The home visitation programs have similar goals—to promote healthy child growth and development by increasing parenting capacity through education, community referrals and social support—though not all are created equal and they vary considerably around program objectives, qualifications of the home visitor (professional, paraprofessional or layperson), intensity and frequency of visits, use of a structured curriculum, timing of enrollment (pregnancy or postpartum) and length of program. To date, no peer-reviewed data are available from provincial/territorial evaluations to estimate the impact of these home-visiting strategies on maternal-child health outcomes. Further, most evaluations have not been conducted using designs that included comparisons of treatment and control groups,[27] with the exception of Manitoba's *Families First* program.[28] Without the use of such rigorous study designs, it is difficult to conclude that home visitation interventions cause any observed changes.

Within the paradigm of evidence-based health care and in the public health sector where resources are often scarce, local public health departments are ethically responsible for implementing those interventions that have been evaluated to significantly affect key maternal-child outcomes, including preventing abuse and neglect. However, since a program may not necessarily demonstrate the same magnitude of positive outcomes in the applied environment as in research contexts, implementation must be preceded by a pilot study and an evaluation using an RCT.[16,25] This approach is being used to test the feasibility and acceptability of the NFP program within Canadian health and social

care systems. In 2008, the City of Hamilton Public Health Services and a multidisciplinary team of researchers from McMaster University adapted the NFP curriculum materials for use with young, low-income, first-time mothers living in Hamilton, Ontario. They are conducting a pilot study to test procedures for recruitment; strategies for retention and the feasibility of and methods for collecting child maltreatment data from local child welfare agencies, hospital visit data for mothers and children, and clinical and interview data from participants. Additionally, they are undertaking a qualitative study to explore the acceptability of this targeted intervention to clients and their families, to the Public Health Nurses conducting the home visits and to community professionals involved in referring and providing auxiliary health and social services to clients. Only if the results of these pilot studies are favourable will an RCT be undertaken to measure the impact and cost-benefit of the program in Ontario and other potential communities across Canada.

Conclusion

As part of a multisectoral response to child maltreatment, the field of public health—building on its strong foundation—is providing leadership and measuring the magnitude and scope of the issue; identifying individual, family and community-level risk indicators for maltreatment; and identifying, implementing and evaluating primary prevention strategies. The next priorities should be to: 1) disseminate the CIS surveillance findings in a timely fashion to those decision makers who are responsible for implementing child and youth injury prevention programs or parenting programs at the provincial/territorial level; 2) engage public health researchers to conduct secondary analyses of the CIS dataset to answer relevant questions; and 3) establish a process for evaluating the CIS.

The refinement and subsequent dissemination of the CIS surveillance data to public health decision makers will increase their awareness of risk indicators associated with child maltreatment; these risk indicators can then be addressed by prioritizing the implementation of interventions. There is a significant body of literature that identifies the NFP program as the most cost-effective approach to preventing child maltreatment as well as to improving other important maternal-child outcomes. After appropriate evaluation, public health departments could implement the NFP program to enhance the current universal parenting programs.

With the emergence of prevention programs that are known to be effective, public health researchers and decision makers need to continue to advocate for collaborations and resources that facilitate rigorous evaluations of programs. As with any other health care intervention, we should be able to clearly articulate to clients the benefits and potential harms they may experience as a result of consenting to participate in any public health program.

Acknowledgments

Dr. Jack received support for the preparation of this commentary from the Injury and Child Maltreatment Surveillance Section, Public Health Agency of Canada. She also

TABLE 1
Sector responses to child maltreatment responses to child maltreatment

Sector	General response to child maltreatment
1. Child Welfare	• Respond and investigate reports of observed or suspected maltreatment • Offer counselling and other services for perpetrators, children at-risk and maltreated children • Provide care for children in out-of-home placements • Prevent the recurrence of child maltreatment (secondary prevention)
2. Justice	• Legal reform • Prosecution of perpetrators • Protection of child witnesses
3. Education	• Teach students and parents about recognizing, preventing and responding to different types of maltreatment
4. Primary and acute care health services	• Identification and documentation of signs and symptoms of maltreatment • Assess children exposed to maltreatment • Treat physical injuries or emotional harm that occurs as a result of maltreatment (tertiary prevention)
5. Law enforcement	• Respond and investigate reports of suspected or observed maltreatment Determine criminal law violations • Identify and apprehend alleged perpetrators • File, if necessary, appropriate criminal charges
6. Public health	• Coordinate surveillance systems, including the collection, analysis and dissemination of findings • Identify, implement and evaluate primary prevention programs

holds the Institute of Human Development, Child and Youth Health, Reproduction and Child Health New Investigator Personnel Award from the Canadian Institutes of Health Research. Thank you to Lil Tonmyr and Wendy Hovdestad, Injury and Child Maltreatment Section, Health Surveillance and Epidemiology Division, Public Health Agency of Canada, for their feedback on drafts of this manuscript.

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Executive summary

Performance monitoring for cervical cancer screening programs in Canada

Screening Performance Indicators Working Group, Cervical Cancer Prevention and Control Network

The purpose of this report is to define a core set of performance indicators for organized cervical cancer screening programs in Canada.

The goals for establishing a pan-Canadian set of performance indicators are to promote high quality screening through monitoring and evaluation. Over time, with regular monitoring and reporting of these indicators, an evidence base will grow which will permit the setting of pan-Canadian targets.

Cervical cancer control is undergoing tremendous development as knowledge of the causal relationship between the human papillomavirus (HPV) and cervical cancer continues to increase. Regular monitoring and reporting of these indicators will facilitate the evaluation of the impact of new technologies and interventions.

The program performance indicators described were selected by the Screening Performance Indicators Working Group (SPIWG) through a consensus-based, iterative process. Feedback from content experts including researchers, clinicians and administrators across Canada was also sought.

The program performance indicators reflect the current pan-Canadian screening practices and include the following: coverage (i.e. participation and retention rates), cytology performance (i.e. specimen adequacy and Pap test results), system capacity (i.e. cytology turn-around time and time to colposcopy), follow-up (i.e. biopsy rate, cytology-histology agreement) and outcomes (i.e. pre-cancer detection rate, cancer incidence, disease extent at diagnosis: cancer stage, screening history in cases of invasive cancer).

The ongoing implementation of HPV immunization programs will have a significant future impact on cervical cancer in Canada. To detect changes in cervical cancer and cervical cancer screening attributable to HPV vaccine programs, the SPIWG recommends that relevant core performance indicators be monitored by 10-year age groups to detect early changes, and eventually by various HPV vaccination parameters (e.g. type of vaccine, fully/partially/not vaccinated, time since vaccination) to detect differences.

It is challenging to define quantifiable performance indicators over the entire spectrum of activity for an organized screening program especially given that the body of literature is continually evolving, as are the technologies and methods used to screen, diagnose and treat cervical cancer. In light of this, this core set of performance indicators is expected to be updated as pan-Canadian screening policy and management guidelines evolve over time. Future indicators should include areas such as professional education initiatives, public education initiatives, letters of invitation, recruitment initiatives, program efficiency, HPV testing protocols, HPV immunization, among others.

The implementation of HPV vaccine programs and the consideration of HPV testing as a primary screening test will require pan-Canadian experts to convene to develop new cervical screening policy and management guidelines. The SPIWG urges that the identification of performance indicators be included within the development of screening policy and management guidelines. This emphasizes the integral role of performance monitoring and evaluation in policy implementation.

Much of this document is highly technical; however, the *Background* and *Future Directions* sections and *Appendix C* provide a general overview of cervical screening in Canada and its evaluation.

For more information, the *Report from the Screening Performance Indicators Working Group, Cervical Cancer Prevention and Control Network (CCPCN): January 2009* is available on the PHAC website at: http://www.phac-aspc.gc.ca/cd-mc/cancer/pmc-cspc-srpdccuc/index-eng.php

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Executive summary

Report from the Canadian Chronic Disease Surveillance System: Hypertension in Canada, 2010

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Introduction

Hypertension is a common and serious health problem among Canadians, and tracking hypertension leads to understanding how the condition can be prevented and treated. The *Report from the Canadian Chronic Disease Surveillance System: Hypertension in Canada, 2010* provides a current and comprehensive picture of hypertension in Canada. Written in collaboration with the provincial and territorial governments, it is the Public Health Agency of Canada's first national surveillance report on hypertension from the Canadian Chronic Disease Surveillance System (CCDSS), which was initially used to track diabetes.

The main purpose of the report is to provide governments and the public with new knowledge in order to help reduce the risk of developing hypertension and to improve its outcomes among Canadians.

The report shows that hypertension—defined by CCDSS as a minimum of one hospitalization or of two physician claims with a diagnosis of hypertension within a two-year period—is highly prevalent. The number of Canadian adults living with hypertension has increased between 1998/99 and 2006/07 and is projected to continue to increase, with a resultant major impact on Canada's health system. Moreover, a substantial number of Canadians are living with both hypertension and diabetes; for them, mortality rates from any causes are higher than among people with only one of these conditions.

Highlights

The *Report from the Canadian Chronic Disease Surveillance System: Hypertension in Canada, 2010* features the most recent data available, from fiscal year 2006/07, as well as trend data from 1998/99 to 2006/07. Where data on both diagnosed hypertension and diabetes are presented, trend data are from 2000/01 onwards as data for diabetes were not available prior to this. The report also provides provincial/territorial comparisons. However, data for Nunavut and Quebec were unavailable, though these will likely be available in future reports. Data were reported for adults aged 20 years and older.

Prevalence

- Nearly 6 million Canadians—or more than one in five adults over the age of 20—were living with diagnosed hypertension in 2006/07 (24.0% of women and 21.3% of men, crude prevalence).
- The age-standardized prevalence of diagnosed hypertension increased from 12.9% in 1998/99 to 19.6% in 2006/07.
- Projections indicate that, if current age and sex trends continue, by 2011/12 about 7.3 million Canadians will have been diagnosed with hypertension—an estimated increase of 25.5% from 2006/07.

Incidence

- Age-standardized incidence rates of diagnosed hypertension remained stable throughout the study period with overall incidence rates (age-standardized to the 1991 Canadian population) of 26.2 per 1000 in 1998/99 and 25.8 per 1000 in 2006/07.
- Almost half a million (450 000) Canadians were newly diagnosed with hypertension in 2006/07 (22.1 per 1000 population aged 20 years and older, 21.6 per 1000 women and 22.7 per 1000 men, crude incidence).

Provincial and territorial comparisons

- The age-standardized prevalence of diagnosed hypertension was above the national average in the Atlantic provinces and below the national average in the west and north (Yukon, Northwest Territories and British Columbia).
- Yukon has the highest age-standardized incidence rate of diagnosed hypertension, closely followed

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by Newfoundland and Labrador. The lowest rates were observed in Ontario and the Northwest Territories.

Hypertension and diabetes

- In 2006/07, 5.1% of Canadians aged 20 years and older (1 million) were living with both diagnosed diabetes and hypertension.
- 22.7% of adults with diagnosed hypertension also had diagnosed diabetes, and 62.8% of adults with diagnosed diabetes also had diagnosed hypertension.
- Age-standardized prevalence of diagnosed diabetes among adults with diagnosed hypertension increased from 10.9% in 2000/01 to 14.3% in 2006/07.

Mortality

- Between 1998/99 and 2006/07, all-cause mortality rates for adults with diagnosed hypertension decreased from 7.3 per 1000 to 6.7 per 1000 among women and from 12.2 per 1000 to 10.2 per 1000 among men.
- In 2006/07, all-cause mortality rates were, respectively, 34% and 44% higher among women and men with diagnosed hypertension than among those without diagnosed hypertension.
- In 2006/07, age-standardized all-cause mortality rates were about 2 times higher for adults with both diagnosed hypertension and diabetes compared to adults with diagnosed hypertension only.

Summary

The *Report from the Canadian Chronic Disease Surveillance System: Hypertension in Canada, 2010* provides an up-to-date picture of hypertension in Canada. Although the overall incidence rate has been stable, the prevalence has been increasing steadily over the last decade, meaning that the number of Canadians who are living with hypertension has increased.

Known as the "silent killer," hypertension is a leading modifiable risk factor for cardiovascular disease (CVD) and mortality in the world. In most cases, hypertension has no symptoms and can only be diagnosed through proper blood pressure measurement. If left untreated, hypertension can increase a person's risk of stroke, coronary heart disease, dementia, diabetes, heart and kidney failure and other chronic diseases.

Hypertension affects all age groups, but the risk of hypertension increases with age. The Canadian population is aging, and with increasing rates of obesity and diabetes, the risk of developing hypertension is projected to increase in Canada.

The risk of developing hypertension can be reduced through eating a healthy diet, limiting sodium intake, avoiding excessive alcohol consumption, losing excess weight and through regular physical activity.

Hypertension can be controlled with lifestyle modifications and/or use of blood pressure lowering medication. Moreover, it is important that individuals with hypertension have their cholesterol and blood sugar levels and kidney function checked regularly as the presence of these risk factors increases the risk of damage from hypertension. Improved management of hypertension can prevent heart disease, kidney disease and stroke in the population.

For more information, the *Report from the Canadian Chronic Disease Surveillance System: Hypertension in Canada, 2010* is now available on the PHAC website at: http://phac-aspc.gc.ca/cd-mc/cvd-mcv/ccdss-snsmc-2010/index-eng.php

What Chronic Disease Infobase Data Cubes can do for you

Background

Public health is complex, largely due to the diversity of the population under study. Because of this, characterizing the relationships that exist between exposure(s) and disease requires comprehensive analyses of many variables. Traditional methods of data analysis using statistical software (e.g. SAS® and STATA®) are best for complex analyses. However, for routine queries, for instance, the prevalence of a chronic disease by age and geography, the Public Health Agency of Canada has developed an online tool to enhance the efficiency of this type of data analysis.

An interactive tool

Chronic Disease Infobase data cubes display chronic disease health indicator data interactively. This online data analysis tool is very flexible; you can explore many different variables and look at associations between them; you can combine, nest and change the variables instantly; and you can change the appearance of your figure quickly and easily by changing the figure type and adjusting the series' colours. The efficiency and utility of this approach for generating cross-tabulations and figures is unparalleled.

Accessing and saving data

Access to the Chronic Disease Infobase data cubes is via your web browser; no additional software or downloads are required. In addition, each user is able to save queries for future access and send queries to colleagues. You can also export figures and/or tables to several file formats (e.g. portable document format, Microsoft Excel spreadsheet).

Easy to use: a Nova Scotia example

Scenario: you are a regional surveillance analyst in Halifax, Nova Scotia. A provincial government employee asks you for the prevalence of smoking in your region, by age group, gender and occupation. You complete the analysis immediately by accessing Chronic Disease Infobase data cubes online. From the list of cubes, you choose the most appropriate one for your analysis. You then simply orient your table to include the four variables required, choose the chart type that best represents the data (e.g. pie chart vs. clustered bar chart) and export the table to the format of your choice.

A rich palette of data

Infobase data cubes contain various types of data, including mortality, morbidity and risk factor data. These can be compared across various demographic data to generate statistics such as prevalence estimates, crude and adjusted incidence rates, and trends over time. The chronic disease indicator data come from many different sources, including the Canadian Community Health Survey, the Canadian Health Measures Survey, Vital Statistics and cancer incidence data from Statistics Canada, the Canadian Chronic Diseases Surveillance System and the Canadian Census. All data contained in the Chronic Disease Infobase data cubes are pre-summarized and meet the requirements of all applicable user agreements.

To access this new online surveillance tool, enter the following Web URL into your browser, http://www.infobase.phac-aspc.gc.ca

For more information about the tool, please contact infobase@phac-aspc.gc.ca