Health Promotion and Chronic Disease Prevention in Canada

Research, Policy and Practice

Volume 38 · Number 3 · March 2018

Inside this issue

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 2368-738X
Pub. 170295
Journal_HPCDP-Revue_PSPMC@phac-aspc.gc.ca

Également disponible en français sous le titre : *Promotion de la santé et prévention des maladies chroniques au Canada : Recherche, politiques et pratiques*

Submission guidelines and information on article types are available at:
https://www.canada.ca/en/public-health/services/reports-publications/health-promotion-chronic-disease-prevention-canada-research-policy-practice/information-authors.html

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

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

Canada

https://doi.org/10.24095/hpcdp.38.3.01

Childhood cancer incidence in Canada: demographic and geographic variation of temporal trends (1992–2010)

Lin Xie, MSc; Jay Onysko, MA; Howard Morrison, PhD

This original quantitative research article has been peer reviewed.

Abstract

Introduction: Surveillance of childhood cancer incidence trends can inform etiologic research, policy and programs. This study presents the first population-based report on demographic and geographic variations in incidence trends of detailed pediatric diagnostic groups in Canada.

Methods: The Canadian Cancer Registry data were used to calculate annual age-standardized incidence rates (ASIRs) from 1992 to 2010 among children less than 15 years of age by sex, age and region for the 12 main diagnostic groups and selected subgroups of the International Classification of Childhood Cancer (ICCC), 3rd edition. Temporal trends were examined by annual percent changes (APCs) using Joinpoint regression.

Results: The ASIRs of childhood cancer among males increased by 0.5% (95% confidence interval (CI) = 0.2–0.9) annually from 1992 to 2010, whereas incidence among females increased by 3.2% (CI = 0.4–6.2) annually since 2004 after an initial stabilization. The largest overall increase was observed in children aged 1–4 years (APC = 0.9%, CI = 0.4–1.3). By region, the overall rates increased the most in Ontario from 2006 to 2010 (APC = 5.9%, CI = 1.9–10.1), and increased non-significantly in the other regions from 1992 to 2010. Average annual ASIRs for all cancers combined from 2006 to 2010 were lower in the Prairies (149.4 per million) and higher in Ontario (170.1 per million). The ASIRs increased for leukemias, melanoma, carcinoma, thyroid cancer, ependymomas and hepatoblastoma for all ages, and neuroblastoma in 1–4 year olds. Astrocytoma decreased in 10–14 year olds (APC = −2.1%, CI = −3.7 to −0.5), and among males (APC = −2.4%, CI = −4.6 to −0.2) and females (APC = −3.7%, CI = −5.8 to −1.6) in Ontario over the study period.

Conclusion: Increasing incidence trends for all cancers and selected malignancies are consistent with those reported in other developed countries, and may reflect the changes in demographics and etiological exposures, and artefacts of changes in cancer coding, diagnosis and reporting. Significant decreasing trend for astrocytoma in late childhood was observed for the first time.

Keywords: *childhood cancer, ICCC, age-standardized incidence rate, annual percent change*

Highlights

- Childhood cancer incidence increased by 0.5% annually from 1992 to 2010 among males, and increased by 3.2% from 2004 to 2010 among females.
- The overall increase was observed in the most recent decade, and among children aged 1–4.
- The overall incidence tended to increase in each region from 1992 to 2010. The rates were lower in the Prairies and higher in Ontario from 2006 to 2010.
- Significant increases were observed for leukemias, melanoma, carcinoma, thyroid cancer, ependymomas and hepatoblastoma for all ages combined, and neuroblastoma in children aged 1–4.
- Astrocytoma incidence decreased among children aged 10–14 years.
- The findings can help inform etiologic research, public health policy and programs.

Introduction

While cancer in children is rare and represents less than 1% of all new cancer cases in Canada, it is the most common cause of death (following accidents) among children > 1 year of age in Canada.[1,2] Although treatment advances have increased the overall five-year survival rate from 71% to 83% over the last three decades, childhood cancer has a lifelong health, psychosocial, and financial impact on children and their families.[1,3] Patients who survive five years remain at risk of recurrence or progression of their primary cancer and are at an increased risk of developing subsequent malignancies, chronic diseases, and functional impairments as a result of treatment.

A Statistics Canada report has documented a statistically significant increase of 0.4% per year in overall incidence of pediatric cancers from 1992 to 2010 at the national level.[4] In recent years, the possibility that the incidence rates of certain pediatric malignancies are increasing has become a topic of public and scientific concern.[5-8] Reasons for such changes are not yet understood. Surveillance of cancer incidence trends may provide insight to develop new hypotheses for future etiologic studies, and may inform the need for health services in particular populations. However, the recent temporal trends in incidence have not been examined in

Author reference:

Public Health Agency of Canada, Ottawa, Ontario, Canada

Correspondence: Lin Xie, Centre for Surveillance and Applied Research, Public Health Agency of Canada, 785 Carling Avenue, AL 6807A, Ottawa, ON K1A 0K9; Tel: 613-513-6357; Fax: 613-960-0944; Email: lin.xie@canada.ca

detail by pediatric diagnostic groups or in regional contexts. This study presents detailed recent population-based data on demographic and geographic variations in childhood cancer incidence trends in Canada.

Methods

Data sources

The cancer incidence data were extracted from the Canadian Cancer Registry (CCR),[9] except for Quebec where, from 2008 to 2010, data were obtained in a summary format from the province directly. The incidence data are collected by the provincial and territorial cancer registries, which report data annually to the CCR at Statistics Canada. The CCR is a dynamic, person-oriented, population-based database with cases newly diagnosed from 1992 onward.

Cancer diagnoses were coded according to topography, morphology and behaviour using the International Classification of Diseases for Oncology, Third Edition (ICD-O-3)[10] and were converted to the International Classification of Childhood Cancer, Third Edition (ICCC-3).[11,12] All primary malignancies diagnosed during the period 1992 through 2010 among those aged 0–14 years were included. The ICCC-3 includes non-malignant intracranial and intraspinal tumours in categories III and X. In accordance with this classification, non-malignant central nervous system (CNS) tumours were also included as a separate analysis.

Population estimates for Canada and the provinces/territories used in the calculation of incidence rates were based on quinquennial censuses conducted from 1991 to 2011. We used intercensal estimates prepared by Statistics Canada for the years between these censuses.[13]

Statistical analysis

Cancer incidence counts and population estimates were summarized by age group (< 1 [infants], 1–4, 5–9, and 10–14 [late childhood] years), year of diagnosis, sex, and geographical region at diagnosis (British Columbia, the Prairie provinces [Alberta, Saskatchewan and Manitoba], Ontario, Quebec, the Atlantic provinces [New Brunswick, Prince Edward Island, Nova Scotia, and Newfoundland and Labrador], and the Territories [Yukon, Northwest Territories and Nunavut]). Given that the number of cancer cases was too small to provide stable estimates for some cancers for each of the Prairie provinces, the Atlantic provinces or the Territories, aggregated regions were created for analysis. Rates for each category were calculated by dividing the number of cases in each category by the corresponding population figure. These age-specific rates were standardized to the 2011 Canadian population, using the direct method, to obtain age-standardized incidence rates (ASIRs) per million children.

Joinpoint Regression Program, which is a statistical software for the analysis of trends, was used to identify changes in the trends of annual age-standardized incidence rates of selected cancers over the period from 1992 to 2010.[14] The response variable was the natural logarithm of the ASIR, and the independent variable was the year of cancer diagnosis. Separate analyses were run by cancer type, sex, age and region. The annual percent change (APC) in cancer incidence rates was calculated by fitting a piecewise linear regression model, assuming a constant rate of change in the logarithm of the annual ASIR in each segment.[15] The estimated slope from this model was then transformed back to represent an annual percentage increase or decrease in the rate. The test of APC is based on asymptotic t-test. The APC was considered statistically significant if its 95% confidence interval (CI) did not include zero ($p < 0.05$). The connecting points of the linear segments are referred to as changepoints or joinpoints. The models incorporated estimated standard errors of the ASIRs. To reduce the likelihood of reporting spurious changes in trends, we used a minimum of five observations from a joinpoint to either end of the data and a minimum of four observations between joinpoints. Statistical significance in changes of the trends (joinpoints) was determined using Monte Carlo permutation tests with the Bonferroni adjustment to control the overfitting probability of the multiple tests (the overall significance level was 0.05).

To ensure confidentiality and limit the possibility of residual disclosure, in keeping with CCR reporting requirements, incidence counts presented in the tables and Figure 1 have been randomly rounded either up or down to a multiple of 5. As a result, when these data are grouped, the totals may not equal the sums of individual values. ASIRs were derived using the actual counts. The ASIRs and APCs are not reported when the corresponding rounded counts are less than 30. In addition, the extended classifications of lymphoid leukemias, except for precursor cell lymphoblastic leukemia, are not presented, as the cases in these subgroups originally coded in ICD-O-2 do not have the required information to be converted to ICD-O-3.[10] Also, the results by region are only reported for the 12 major diagnostic categories and the subtypes with significant APCs.

Results

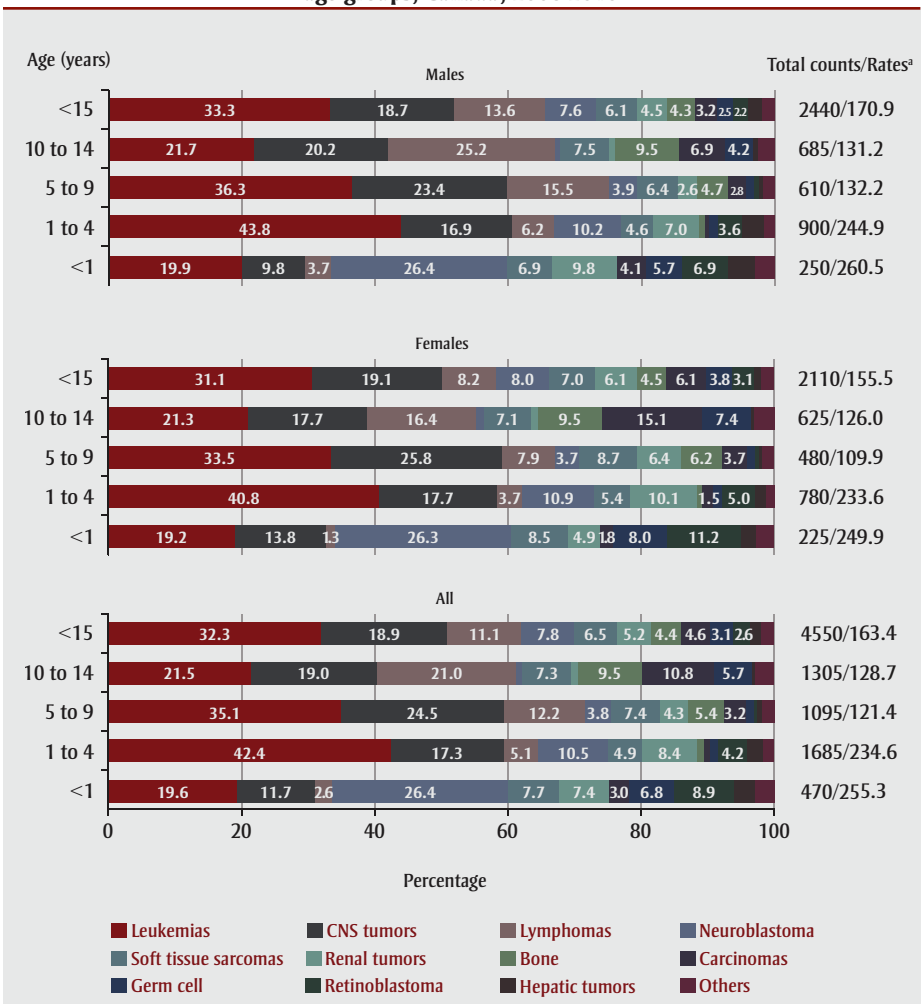
Since the completeness of non-malignant CNS tumor data collection varied by province (data not shown), which may have an impact on comparisons across region and time (see Discussion), the results addressed in this section for all cancers combined and CNS tumors are based on malignancies only, whereas results of the best fit joinpoint regression models for these two categories including non-malignant CNS tumors are also provided in Tables 1–5.

Recent incidence counts and rates (2006–2010)

Figure 1 summarizes the distribution of primary cancers for Canada from 2006 to 2010 by age groups for males and females combined and separately. During this period, an average of 910 new diagnoses each year; i.e., a total of 4550 new cases, were reported among children 14 years and under in Canada: 2440 (53.6%) in males and 2110 (46.4%) in females, which amounts to a male:female ratio of 1.2:1. The average annual ASIR was 163.4 per million children, with males having a higher rate than females (170.9 vs. 155.5 per 10^6 children). Average annual ASIRs for all cancers combined from 2006 to 2010 were lower in the Prairies (149.4 per 10^6) and higher in Ontario (170.1 per 10^6) (Figure 2).

While most adult cancers are carcinomas, childhood cancers show much histologic and biologic diversity, and are mainly not of epithelial origin. Overall, the most common childhood cancers diagnosed from 2006 to 2010 were leukemias (32.3%), CNS tumors (18.9%), and lymphomas (11.1%) (Figure 1). Next most common were neuroblastoma (7.8%), soft tissue sarcoma (6.5%), and renal tumors (5.2%).

FIGURE 1
Distribution of new cancer cases diagnosed in children less than 15 years of age by sex and age groups, Canada, 2006-2010



Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
Note: The rates were standardized to the 2011 Canadian population for all ages combined.
[a] The number of new cases were randomly rounded either up or down to a multiple of 5.

The top 5 most common cancers were similarly distributed within each region, with some variations in proportions and ranking in the Atlantic region (Figure 2), possibly due to Type I error from the small population in the region. The distribution of the most frequent childhood cancers was generally the same for males and females, except lymphomas were more common in males (13.6% compared to 8.2%), and carcinomas (especially, thyroid carcinoma) were more common in females (6.1% vs. 3.2%) (Figure 1).

Around half of children's cancer cases (47.4%) were diagnosed among those under the age of five years (Figure 1). The age-specific incidence rates in children aged less than 5 years were around twice those of their older counterparts. The highest incidence was observed in infants under the age of one year and generally declined with age. Patterns of diagnoses varied considerably by age group. In infants, neuroblastoma formed the most commonly diagnosed cancers and accounted for nearly a third of all cases (26.4%), followed by leukemias (19.6%) and CNS tumours (11.7%). The embryonal tumors of neuroblastoma, retinoblastoma, and nephroblastoma jointly accounted for 42.6% of all diagnoses in infants. Leukemias prevailed among 1–4 year olds, accounting for 42.4% of all diagnoses, while in 5–9 year olds and 10–14 year olds, lymphomas and bone tumours became increasingly common (lymphomas: 12.2% and 21.0%; bone cancers: 5.4% and 9.5%, respectively). Also in children aged 10–14 years, leukemias (21.5%) and CNS tumours (19.0%) predominated.

Overall temporal trends (1992–2010)

Trends varied greatly by cancer type, although the small numbers of some types may have resulted in extensive random fluctuations in rates even when the trend was statistically significant. The incidence rates of childhood cancer increased by an average of 0.4% per year (95% CI = 0.1–0.8), from 154.8 per million children in 1992 to 169.7 per million in 2010 (Table 1). Leukemia overall and lymphoid leukemia specifically had an equally increase from 1992 through 2010 (APC = 0.6%, CI = 0.1–1.2). Lymphoid leukemia is the most common type in children, accounting for nearly four-fifths (78.5%) of all leukemias and as such largely determined the incidence pattern for leukemia overall. Rates which increased by at least 2% annually over the study period included: unspecified lymphomas (APC = 3.4%; CI = 0.7–6.2), ependymomas (APC = 2.3%, CI = 0.2–4.3), hepatoblastoma (APC = 2.4%, CI = 0.4–4.4), carcinoma (APC = 2.5%, CI = 0.2–4.7), thyroid cancer (APC = 4.2, CI = 1.4–7.1) and melanoma (APC = 2.7%, CI = 0.1–5.4). The data suggested a decrease for malignant gonadal germ cell tumors (APC = −2.3%, CI = −4.4 to −0.03). Figure 3 highlights the trends for all cancers combined and the most common five cancers in children under 15 years of age.

Trends by sex

The trends for all cancers combined (APC = 0.5%, CI = 0.2–0.9) and leukemias (APC = 0.8%, CI = 0.03–1.6) among males paralleled the increases observed overall (Table 1). A break in trend was observed for all cancers combined among females; the rate increased by 3.2% per year (CI = 0.4–6.2) from 2004 to 2010, which followed an initial period of stable rates. Positive trends were also observed for other hematologic malignancies over the entire period: miscellaneous lymphoreticular neoplasms in both males (APC = 6.8%, CI = 2.2–11.7) and females (APC = 4.6%, CI = 0.7–8.6), and unspecified lymphomas among males (APC = 3.3%, CI = 0.5–6.2). Some embryonal tumors demonstrated increasing trends in males. An increase occurred for neuroblastoma overall in males (APC = 1.4%, CI = 0.2–2.6), as did its subgroup of neuroblastoma and ganglioneuroblastoma (IV(A)) which comprised nearly all male neuroblastoma

TABLE 1
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by sex, Canada, 1992–2010

Both sexes combined									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	16955	97.54	100.00	890	155.71	0.45	1992–2010	0.08 to 0.81	0.02
All childhood cancers including non-malignancies brain	17380	100.00	102.52	915	159.55	0.40	1992–2010	0.08 to 0.73	0.02
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	5485	31.57	32.36	285	50.74	0.64	1992–2010	0.08 to 1.20	0.03
I(A) Lymphoid leukemias	4305	24.78	25.41	225	39.86	0.61	1992–2010	0.09 to 1.13	0.02
Lymphoid leukemias, precursor cell leukemias	4075	23.45	24.04	215	37.68	0.02	1992–2010	−0.78 to 0.83	0.95
I(B) Acute myeloid leukemias	755	4.36	4.47	40	6.98	−0.49	1992–2010	−2.20 to 1.25	0.56
I(C) Chronic myeloproliferative diseases	140	0.81	0.83	10	1.29	0.01	1992–2010	−2.63 to 2.71	1.00
I(D) Myelodysplastic syndrome and other myeloproliferative diseases	85	0.48	0.49	5	0.78	4.06	1992–2010	−0.08 to 8.36	0.05
I(E) Unspecified and other specified leukemias	200	1.13	1.16	10	1.84	17.13	1992–2001	6.18 to 29.21	< 0.01
						−31.47	2001–2004	–	0.45
						18.36	2004–2010	0.99 to 38.72	0.04
Lymphomas and reticuloendothelial neoplasms	1905	10.94	11.22	100	17.01	0.48	1992–2010	−0.35 to 1.32	0.24
II(A) Hodgkin lymphomas	715	4.10	4.21	35	6.28	0.34	1992–2010	−1.00 to 1.70	0.60
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	600	3.45	3.53	35	5.39	0.14	1992–2010	−1.58 to 1.89	0.86
II(C) Burkitt lymphoma	270	1.55	1.59	15	2.41	−2.54	1992–2010	−5.82 to 0.85	0.13
II(D) Miscellaneous lymphoreticular neoplasms	130	0.75	0.77	5	1.22	−0.40	1992–2006	−5.98 to 5.52	0.88
						38.05	2006–2010	7.48 to 77.31	0.02
II(E) Unspecified lymphomas	190	1.09	1.12	10	1.71	3.41	1992–2010	0.72 to 6.18	0.02
CNS and miscellaneous intracranial and intraspinal neoplasms	3345	19.22	19.71	175	30.41	0.13	1992–2010	−0.46 to 0.71	0.65
III(A) Ependymomas and choroid plexus tumor	325	1.86	1.91	15	3.02	2.25	1992–2010	0.23 to 4.31	0.03
III(B) Astrocytomas	1505	8.64	8.86	80	13.59	−0.97	1992–2010	−2.10 to 0.16	0.09
III(C) Intracranial and intraspinal embryonal tumors	805	4.61	4.73	45	7.33	−0.45	1992–2010	−1.77 to 0.89	0.49
III(D) Other gliomas	475	2.72	2.79	25	4.29	1.27	1992–2010	−0.82 to 3.41	0.22
III(E) Other specified intracranial and intraspinal neoplasms	45	0.25	0.26	5	0.40	11.39	1992–2010	7.70 to 15.20	< 0.01
III(F) Unspecified intracranial and intraspinal neoplasms	195	1.13	1.16	10	1.79	2.37	1992–2010	−2.96 to 7.98	0.37
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	3770	21.69	22.23	195	34.25	−0.03	1992–2010	−0.51 to 0.45	0.89
III(A) Ependymomas and choroid plexus tumor including non-malignancies	360	2.07	2.12	15	3.34	1.58	1992–2010	−0.22 to 3.42	0.08
III(B) Astrocytomas including non-malignancies	1545	8.90	9.12	85	13.98	−0.98	1992–2010	−2.04 to 0.09	0.07
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	805	4.61	4.73	40	7.33	−0.45	1992–2010	−1.77 to 0.89	0.49
III(D) Other gliomas including non-malignancies	475	2.73	2.80	25	4.29	1.29	1992–2010	−0.80 to 3.43	0.21

Continued on the following page

TABLE 1 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by sex, Canada, 1992–2010

	Both sexes combined								
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
III(E) Other specified intracranial and intraspinal neoplasms including non-malignancies	320	1.85	1.90	20	2.89	1.88	1992–2010	−1.08 to 4.93	0.20
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	265	1.52	1.56	15	2.42	0.25	1992–2010	−4.24 to 4.95	0.91
Neuroblastoma and other peripheral nervous cell tumors	1260	7.26	7.44	65	12.03	0.74	1992–2010	−0.45 to 1.95	0.21
IV(A) Neuroblastoma and ganglioneuroblastoma	1245	7.15	7.33	65	11.86	0.83	1992–2010	−0.30 to 1.98	0.14
Retinoblastoma	430	2.47	2.54	25	4.13	0.30	1992–2010	−1.66 to 2.30	0.75
Renal tumours	950	5.47	5.60	50	8.90	−0.68	1992–2010	−2.07 to 0.73	0.32
VI(A) Nephroblastoma and other nonepithelial renal tumors	895	5.16	5.29	50	8.42	−0.71	1992–2010	−2.16 to 0.77	0.33
VI(B) Renal carcinomas	30	0.18	0.19	5	0.28	−5.27	1992–2010	−9.53 to −0.81	0.02
Hepatic tumours	260	1.49	1.53	15	2.47	1.35	1992–2010	−0.13 to 2.86	0.07
VII(A) Hepatoblastoma	210	1.23	1.26	10	2.07	2.42	1992–2010	0.44 to 4.42	0.02
VII(B) Hepatic carcinomas	35	0.21	0.22	5	0.33	−3.00	1992–2010	−7.92 to 2.18	0.23
Malignant bone tumours	760	4.39	4.50	40	6.76	−0.50	1992–2010	−1.66 to 0.66	0.37
VIII(A) Osteosarcomas	380	2.19	2.24	20	3.35	−0.89	1992–2010	−2.61 to 0.85	0.29
VIII(C) Ewing tumor and related sarcomas of bone	300	1.73	1.77	15	2.66	0.63	1992–2010	−1.27 to 2.57	0.49
VIII(D) Other specified malignant bone tumors	30	0.17	0.18	0	0.27	−3.39	1992–2010	−7.70 to 1.12	0.13
VIII(E) Unspecified malignant bone tumors	35	0.22	0.22	5	0.34	−0.89	1992–2010	−5.83 to 4.31	0.72
Soft tissue and other extraosseous sarcomas	1060	6.08	6.23	55	9.60	−0.08	1992–2010	−1.46 to 1.31	0.90
IX(A) Rhabdomyosarcomas	530	3.04	3.12	30	4.83	−0.52	1992–2010	−2.37 to 1.36	0.56
IX(B) Fibrosarcomas, peripheral nerve sheath tumors, and other fibrous neoplasms	100	0.57	0.58	5	0.91	0.41	1992–2010	−3.51 to 4.49	0.83
IX(D) Other specified soft tissue sarcomas	305	1.75	1.79	15	2.73	0.25	1992–2010	−2.18 to 2.74	0.83
IX(E) Unspecified soft tissue sarcomas	125	0.71	0.73	5	1.13	0.40	1992–2010	−3.38 to 4.33	0.83
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	555	3.19	3.27	30	5.05	−0.38	1992–2010	−1.64 to 0.90	0.54
X(A) Intracranial and intraspinal germ cell tumors	155	0.91	0.94	10	1.42	1.91	1992–2010	−0.88 to 4.79	0.17
X(B) Malignant extracranial and extragonadal germ cell tumors	130	0.76	0.78	10	1.26	1.20	1992–2010	−1.12 to 3.57	0.29
X(C) Malignant gonadal germ cell tumors	230	1.33	1.36	10	2.07	−2.25	1992–2010	−4.42 to −0.03	0.05
Other malignant epithelial neoplasms and malignant melanomas	670	3.86	3.95	35	5.99	2.45	1992–2010	0.23 to 4.72	0.03
XI(A) Adrenocortical carcinomas	35	0.20	0.20	5	0.32	4.22	1992–2010	−0.95 to 9.66	0.10
XI(B) Thyroid carcinomas	260	1.49	1.53	10	2.29	4.20	1992–2010	1.37 to 7.11	0.01
XI(D) Malignant melanomas	155	0.87	0.89	5	1.36	2.68	1992–2010	0.08 to 5.35	0.04
XI(F) Other and unspecified carcinomas	205	1.19	1.22	10	1.85	−0.84	1992–2010	−3.73 to 2.13	0.56
Other and unspecified malignant neoplasms	280	1.61	1.65	15	2.61	2.78	1992–2010	−0.02 to 5.65	0.05

Continued on the following page

TABLE 1 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by sex, Canada, 1992–2010

Both sexes combined									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
XII(A) Other specified malignant tumors	35	0.21	0.22	5	0.35	15.83	1992–1999	5.45 to 27.22	0.01
						−10.31	1999–2006	−33.63 to 21.19	0.44
						32.16	2006–2010	12.48 to 55.27	< 0.01
XII(B) Other unspecified malignant tumors	245	1.40	1.43	10	2.26	1.68	1992–2010	−1.42 to 4.88	0.27
Males									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	9135	97.36	100.00	480	163.68	0.52	1992–2010	0.16 to 0.88	0.01
All childhood cancers including non-malignancies brain	9380	100.00	102.72	495	168.01	0.45	1992–2010	0.09 to 0.81	0.02
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	3000	32.03	32.90	160	54.26	0.82	1992–2010	0.03 to 1.62	0.04
I(A) Lymphoid leukemias	2420	25.80	26.50	125	43.73	0.82	1992–2010	0.05 to 1.60	0.04
Lymphoid leukemias, precursor cell leukemias	2280	24.31	24.97	120	41.14	0.19	1992–2010	−0.76 to 1.16	0.68
I(B) Acute myeloid leukemias	375	3.99	4.10	20	6.74	−0.50	1992–2010	−2.71 to 1.75	0.64
I(C) Chronic myeloproliferative diseases	75	0.80	0.82	5	1.35	−0.43	1992–2010	−4.02 to 3.28	0.80
I(D) Myelodysplastic syndrome and other myeloproliferative diseases	45	0.50	0.51	0	0.86	7.68	1992–2010	2.51 to 13.11	0.01
I(E) Unspecified and other specified leukemias	90	0.94	0.96	5	1.58	0.97	1992–2010	−4.92 to 7.24	0.74
Lymphomas and reticuloendothelial neoplasms	1260	13.46	13.82	70	22.04	0.51	1992–2010	−0.50 to 1.53	0.30
II(A) Hodgkin lymphomas	415	4.45	4.57	25	7.18	0.61	1992–2010	−1.39 to 2.66	0.53
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	400	4.28	4.39	20	7.03	0.47	1992–2010	−1.32 to 2.30	0.59
II(C) Burkitt lymphoma	230	2.42	2.49	15	3.94	−2.72	1992–2010	−6.22 to 0.90	0.13
II(D) Miscellaneous lymphoreticular neoplasms	90	0.95	0.97	5	1.63	6.82	1992–2010	2.15 to 11.71	0.01
II(E) Unspecified lymphomas	130	1.36	1.40	10	2.25	3.29	1992–2010	0.50 to 6.15	0.02
CNS and miscellaneous intracranial and intraspinal neoplasms	1795	19.13	19.65	95	31.86	0.12	1992–2010	−0.85 to 1.11	0.79
III(A) Ependymomas and choroid plexus tumor	185	1.96	2.02	10	3.32	1.71	1992–2010	−0.81 to 4.29	0.17
III(B) Astrocytomas	770	8.18	8.40	40	13.56	−0.83	1992–2010	−2.71 to 1.09	0.37
III(C) Intracranial and intraspinal embryonal tumors	480	5.16	5.30	25	8.62	−0.38	1992–2010	−1.96 to 1.23	0.62
III(D) Other gliomas	240	2.55	2.62	10	4.23	0.99	1992–2010	−1.77 to 3.82	0.46
III(F) Unspecified intracranial and intraspinal neoplasms	100	1.07	1.10	5	1.78	1.95	1992–2010	−2.46 to 6.56	0.37
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	2040	21.77	22.36	105	36.19	−0.12	1992–2010	−0.93 to 0.69	0.75
III(A) Ependymomas and choroid plexus tumor including non-malignancies	205	2.20	2.26	10	3.70	0.94	1992–2010	−1.62 to 3.56	0.45
III(B) Astrocytomas including non-malignancies	795	8.48	8.71	45	14.05	−0.83	1992–2010	−2.61 to 0.98	0.34

Continued on the following page

TABLE 1 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by sex, Canada, 1992–2010

	Males								
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	485	5.16	5.30	25	8.62	−0.38	1992–2010	−1.96 to 1.23	0.62
III(D) Other gliomas including non-malignancies	240	2.56	2.63	10	4.25	1.02	1992–2010	−1.70 to 3.81	0.44
III(E) Other specified intracranial and intraspinal neoplasms including non-malignancies	170	1.86	1.91	10	3.04	−0.17	1992–2010	−3.67 to 3.45	0.92
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	145	1.52	1.57	10	2.53	−1.41	1992–2010	−4.66 to 1.94	0.38
Neuroblastoma and other peripheral nervous cell tumors	650	6.94	7.13	35	12.15	1.37	1992–2010	0.15 to 2.60	0.03
IV(A) Neuroblastoma and ganglioneuroblastoma	645	6.86	7.04	35	12.01	1.36	1992–2010	0.14 to 2.60	0.03
Retinoblastoma	215	2.30	2.37	10	4.04	−1.30	1992–2010	−3.76 to 1.23	0.29
Renal tumours	425	4.53	4.65	25	7.80	−0.25	1992–2010	−2.38 to 1.93	0.81
VI(A) Nephroblastoma and other nonepithelial renal tumors	400	4.24	4.36	20	7.33	−0.36	1992–2010	−2.60 to 1.94	0.74
Hepatic tumours	160	1.73	1.77	5	3.00	2.18	1992–2010	0.01 to 4.40	0.05
VII(A) Hepatoblastoma	130	1.41	1.45	10	2.47	3.22	1992–2010	0.60 to 5.91	0.02
Malignant bone tumours	390	4.16	4.27	20	6.74	0.11	1992–2010	−1.86 to 2.12	0.91
VIII(A) Osteosarcomas	195	2.04	2.09	10	3.29	−1.52	1992–2010	−4.19 to 1.22	0.25
VIII(C) Ewing tumor and related sarcomas of bone	160	1.70	1.74	10	2.76	2.28	1992–2010	−0.81 to 5.47	0.14
Soft tissue and other extraosseous sarcomas	565	6.05	6.21	25	10.02	−0.84	1992–2010	−2.46 to 0.82	0.30
IX(A) Rhabdomyosarcomas	290	3.13	3.22	15	5.22	−0.87	1992–2010	−2.96 to 1.26	0.40
IX(B) Fibrosarcomas, peripheral nerve sheath tumors, and other fibrous neoplasms	50	0.53	0.55	5	0.90	0.12	1992–2010	−4.22 to 4.65	0.96
IX(D) Other specified soft tissue sarcomas	155	1.66	1.71	5	2.72	−1.02	1992–2010	−4.58 to 2.66	0.56
IX(E) Unspecified soft tissue sarcomas	70	0.71	0.73	5	1.18	0.16	1992–2010	−3.19 to 3.62	0.92
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	255	2.72	2.79	10	4.58	−0.49	1992–2010	−2.41 to 1.46	0.60
X(A) Intracranial and intraspinal germ cell tumors	110	1.17	1.20	5	1.92	1.89	1992–2010	−1.80 to 5.72	0.30
X(B) Malignant extracranial and extragonadal germ cell tumors	45	0.50	0.51	0	0.88	1.15	1992–2010	−4.30 to 6.91	0.67
X(C) Malignant gonadal germ cell tumors	90	0.93	0.95	5	1.57	−4.02	1992–2010	−6.71 to −1.24	0.01
Other malignant epithelial neoplasms and malignant melanomas	270	2.87	2.95	15	4.70	1.61	1992–2010	−1.37 to 4.69	0.27
XI(B) Thyroid carcinomas	75	0.77	0.79	5	1.24	2.88	1992–2010	−1.05 to 6.98	0.14
XI(D) Malignant melanomas	75	0.84	0.87	5	1.38	3.19	1992–2010	−0.89 to 7.44	0.12
XI(F) Other and unspecified carcinomas	95	1.03	1.06	5	1.71	−0.22	1992–2010	−4.22 to 3.94	0.91
Other and unspecified malignant neoplasms	135	1.45	1.49	10	2.49	3.86	1992–2010	−0.55 to 8.46	0.08
XII(B) Other unspecified malignant tumors	120	1.28	1.31	5	2.19	2.31	1992–2010	−2.24 to 7.08	0.30

Continued on the following page

TABLE 1 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by sex, Canada, 1992–2010

	Females								
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	7820	97.75	100.00	415	147.32	−0.72	1992–2004	−1.71 to 0.29	0.15
						3.23	2004–2010	0.35 to 6.20	0.03
All childhood cancers including non-malignancies brain	8000	100.00	102.30	420	150.65	0.35	1992–2010	−0.22 to 0.92	0.22
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	2480	31.02	31.74	130	47.04	0.43	1992–2010	−0.32 to 1.19	0.24
I(A) Lymphoid leukemias	1885	23.58	24.13	100	35.79	0.36	1992–2010	−0.57 to 1.30	0.43
Lymphoid leukemias, precursor cell leukemias	1795	22.45	22.96	95	34.04	−0.18	1992–2010	−1.31 to 0.96	0.74
I(B) Acute myeloid leukemias	380	4.80	4.91	25	7.22	−0.50	1992–2010	−2.55 to 1.60	0.62
I(C) Chronic myeloproliferative diseases	70	0.82	0.84	5	1.22	0.71	1992–2010	−3.90 to 5.55	0.75
I(D) Myelodysplastic syndrome and other myeloproliferative diseases	40	0.45	0.46	0	0.70	1.65	1992–2010	−3.05 to 6.59	0.48
I(E) Unspecified and other specified leukemias	105	1.36	1.39	5	2.10	16.69	1992–2001	2.75 to 32.52	0.02
						−34.96	2001–2004	–	0.45
						27.43	2004–2010	4.88 to 54.84	0.02
Lymphomas and reticuloendothelial neoplasms	640	8.00	8.18	35	11.72	0.31	1992–2010	−1.08 to 1.72	0.64
II(A) Hodgkin lymphomas	295	3.70	3.78	15	5.34	−0.25	1992–2010	−1.88 to 1.41	0.75
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	200	2.47	2.53	10	3.66	−0.61	1992–2010	−3.66 to 2.53	0.68
II(C) Burkitt lymphoma	40	0.54	0.55	0	0.79	−1.13	1992–2010	−5.41 to 3.34	0.59
II(D) Miscellaneous lymphoreticular neoplasms	40	0.51	0.52	5	0.79	4.57	1992–2010	0.72 to 8.57	0.02
II(E) Unspecified lymphomas	65	0.77	0.79	0	1.14	3.29	1992–2010	−1.97 to 8.83	0.21
CNS and miscellaneous intracranial and intraspinal neoplasms	1545	19.34	19.78	80	28.89	0.07	1992–2010	−0.96 to 1.10	0.89
III(A) Ependymomas and choroid plexus tumor	140	1.75	1.79	5	2.70	2.99	1992–2010	0.60 to 5.43	0.02
III(B) Astrocytomas	735	9.19	9.40	40	13.62	−1.19	1992–2010	−2.76 to 0.39	0.13
III(C) Intracranial and intraspinal embryonal tumors	315	3.97	4.07	15	5.97	−0.57	1992–2010	−2.84 to 1.76	0.61
III(D) Other gliomas	235	2.92	2.99	15	4.34	0.88	1992–2010	−1.98 to 3.81	0.53
III(E) Other specified intracranial and intraspinal neoplasms	25	0.30	0.31	0	0.45	11.38	1992–2010	5.49 to 17.60	< 0.01
III(F) Unspecified intracranial and intraspinal neoplasms	95	1.20	1.23	5	1.80	58.43	1992–1996	–	0.03
						−16.38	1996–2001	−40.10 to 16.75	0.25
						65.74	2001–2004	–	0.28
						−28.95	2004–2010	−40.97 to −14.47	< 0.01
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	1730	21.58	22.08	90	32.22	0.03	1992–2010	−0.96 to 1.04	0.95
III(A) Ependymomas and choroid plexus tumor including non-malignancies	155	1.92	1.97	10	2.96	2.37	1992–2010	0.27 to 4.51	0.03
III(B) Astrocytomas including non-malignancies	750	9.39	9.60	40	13.91	−1.21	1992–2010	−2.71 to 0.32	0.11

Continued on the following page

TABLE 1 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by sex, Canada, 1992–2010

	Females								
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	315	3.97	4.07	20	5.97	−0.57	1992–2010	−2.84 to 1.76	0.61
III(D) Other gliomas including non-malignancies	235	2.92	2.99	15	4.34	0.88	1992–2010	−1.98 to 3.81	0.53
III(E) Other specified intracranial and intraspinal neoplasms including non-malignancies	150	1.85	1.89	10	2.74	4.03	1992–2010	0.08 to 8.14	0.05
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	120	1.52	1.56	5	2.29	8.90	1992–2005	1.10 to 17.31	0.03
						−31.76	2005–2010	−57.68 to 10.04	0.11
Neuroblastoma and other peripheral nervous cell tumors	610	7.62	7.80	35	11.91	0.04	1992–2010	−1.62 to 1.74	0.96
IV(A) Neuroblastoma and ganglioneuroblastoma	595	7.49	7.66	30	11.71	0.22	1992–2010	−1.38 to 1.85	0.77
Retinoblastoma	215	2.67	2.74	10	4.23	2.23	1992–2010	−0.63 to 5.16	0.12
Renal tumours	525	6.56	6.71	30	10.05	−0.85	1992–2010	−2.43 to 0.75	0.28
VI(A) Nephroblastoma and other nonepithelial renal tumors	500	6.24	6.38	25	9.56	−0.76	1992–2010	−2.48 to 0.99	0.37
Hepatic tumours	100	1.21	1.24	5	1.92	0.29	1992–2010	−3.04 to 3.75	0.86
VII(A) Hepatoblastoma	85	1.02	1.05	5	1.64	1.58	1992–2010	−2.13 to 5.44	0.39
Malignant bone tumours	375	4.66	4.77	15	6.77	−1.21	1992–2010	−2.76 to 0.37	0.13
VIII(A) Osteosarcomas	190	2.36	2.42	10	3.42	−0.43	1992–2010	−2.60 to 1.79	0.68
VIII(C) Ewing tumor and related sarcomas of bone	140	1.76	1.80	5	2.55	−0.63	1992–2010	−2.96 to 1.77	0.59
Soft tissue and other extraosseous sarcomas	490	6.11	6.25	25	9.17	0.82	1992–2010	−0.66 to 2.33	0.26
IX(A) Rhabdomyosarcomas	235	2.94	3.00	10	4.42	−0.12	1992–2010	−2.14 to 1.94	0.90
IX(B) Fibrosarcomas, peripheral nerve sheath tumors, and other fibrous neoplasms	50	0.61	0.63	5	0.92	0.58	1992–2010	−3.10 to 4.39	0.75
IX(D) Other specified soft tissue sarcomas	150	1.85	1.89	10	2.75	2.07	1992–2010	−0.45 to 4.65	0.10
IX(E) Unspecified soft tissue sarcomas	60	0.71	0.73	0	1.07	0.86	1992–2010	−4.36 to 6.37	0.74
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	300	3.74	3.82	20	5.55	−0.35	1992–2010	−2.58 to 1.93	0.75
X(A) Intracranial and intraspinal germ cell tumors	50	0.61	0.63	0	0.89	3.43	1992–2010	−0.58 to 7.59	0.09
X(B) Malignant extracranial and extragonadal germ cell tumors	85	1.06	1.09	5	1.67	1.10	1992–2010	−1.49 to 3.75	0.39
X(C) Malignant gonadal germ cell tumors	145	1.80	1.84	10	2.60	−1.39	1992–2010	−4.72 to 2.06	0.40
Other malignant epithelial neoplasms and malignant melanomas	405	5.01	5.13	20	7.34	2.93	1992–2010	0.56 to 5.36	0.02
XI(B) Thyroid carcinomas	185	2.34	2.39	10	3.40	4.85	1992–2010	1.80 to 7.99	< 0.01

Continued on the following page

TABLE 1 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by sex, Canada, 1992–2010

	Females								
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
XI(D) Malignant melanomas	75	0.90	0.92	5	1.34	1.27	1992–2010	−2.84 to 5.57	0.53
XI(F) Other and unspecified carcinomas	110	1.36	1.39	5	1.99	−0.80	1992–2010	−4.17 to 2.68	0.63
Other and unspecified malignant neoplasms	145	1.80	1.84	5	2.73	1.46	1992–2010	−1.48 to 4.50	0.31
XII(B) Other unspecified malignant tumors	125	1.54	1.57	5	2.34	1.00	1992–2010	−2.56 to 4.69	0.57

Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
[a]The ASIRs were standardized to the 2011 Canadian population.

TABLE 2
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

	Age <1 year								
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	1705	98.10	100.00	85	248.64	−0.16	1992–2010	−1.21 to 0.90	0.75
All childhood cancers including non-malignancies brain	1735	100.00	101.94	95	253.47	−0.20	1992–2010	−1.23 to 0.84	0.69
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	340	19.38	19.75	20	49.23	−0.27	1992–2010	−1.95 to 1.44	0.74
I(A) Lymphoid leukemias	125	7.15	7.29	5	18.22	−1.83	1992–2010	−4.62 to 1.04	0.19
Lymphoid leukemias, precursor cell leukemias	120	6.81	6.94	5	17.30	−2.54	1992–2010	−5.45 to 0.46	0.09
I(B) Acute myeloid leukemias	110	6.40	6.53	5	16.19	−2.06	1992–2010	−4.49 to 0.43	0.10
I(C) Chronic myeloproliferative diseases	35	2.13	2.18	0	5.37	−0.10	1992–2010	−4.01 to 3.97	0.96
I(E) Unspecified and other specified leukemias	45	2.60	2.65	5	6.67	0.97	1992–2010	−4.41 to 6.66	0.71
Lymphomas and reticuloendothelial neoplasms	55	3.17	3.23	5	8.05	−2.81	1992–2010	−6.55 to 1.08	0.14
II(D) Miscellaneous lymphoreticular neoplasms	35	1.90	1.94	0	4.78	−4.43	1992–2010	−8.14 to −0.58	0.03
CNS and miscellaneous intracranial and intraspinal neoplasms	185	10.78	10.99	10	27.33	0.41	1992–2010	−2.29 to 3.18	0.75
III(A) Ependymomas and choroid plexus tumor	35	2.02	2.06	0	5.15	5.60	1992–2010	1.94 to 9.38	< 0.01
III(B) Astrocytomas	60	3.69	3.76	5	9.26	−0.24	1992–2010	−4.22 to 3.91	0.90
III(C) Intracranial and intraspinal embryonal tumors	50	2.88	2.94	0	7.40	−1.01	1992–2010	−5.92 to 4.15	0.68

Continued on the following page

TABLE 2 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

Age <1 year									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	220	12.69	12.93	10	32.16	0.19	1992–2010	−1.64 to 2.06	0.83
III(A) Ependymomas and choroid plexus tumor including non-malignancies	40	2.25	2.29	5	5.73	5.04	1992–2010	1.20 to 9.03	0.01
III(B) Astrocytomas including non-malignancies	65	3.81	3.88	5	9.57	−0.16	1992–2010	−4.06 to 3.90	0.94
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	50	2.88	2.94	5	7.40	−1.01	1992–2010	−5.92 to 4.15	0.68
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	30	1.90	1.94	0	4.78	−5.87	1992–2010	−8.37 to −3.30	< 0.01
Neuroblastoma and other peripheral nervous cell tumors	445	25.61	26.10	25	64.56	−14.00	1992–1996	−28.97 to 4.13	0.11
						2.78	1996–2010	−0.50 to 6.17	0.09
IV(A) Neuroblastoma and ganglioneuroblastoma	440	25.55	26.04	25	64.41	−14.22	1992–1996	−29.13 to 3.83	0.11
						2.86	1996–2010	−0.43 to 6.25	0.08
Retinoblastoma	150	8.59	8.76	5	21.70	−0.60	1992–2010	−4.08 to 3.02	0.73
Renal tumours	130	7.38	7.52	5	18.89	0.38	1992–2010	−2.72 to 3.58	0.80
VI(A) Nephroblastoma and other nonepithelial renal tumors	120	6.98	7.11	5	17.86	0.65	1992–2010	−2.34 to 3.74	0.65
Hepatic tumours	60	3.58	3.64	5	9.16	1.95	1992–2010	−3.15 to 7.31	0.44
VII(A) Hepatoblastoma	60	3.46	3.53	0	8.85	1.93	1992–2010	−3.02 to 7.12	0.43
Soft tissue and other extraosseous sarcomas	110	6.29	6.41	5	15.93	0.40	1992–2010	−2.95 to 3.87	0.81
IX(A) Rhabdomyosarcomas	30	1.79	1.82	5	4.53	−4.82	1992–2010	−9.23 to −0.19	0.04
IX(B) Fibrosarcomas, peripheral nerve sheath tumors, and other fibrous neoplasms	30	1.85	1.88	0	4.70	0.73	1992–2010	−4.53 to 6.27	0.78
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	105	6.00	6.11	5	15.20	1.86	1992–2010	−1.38 to 5.21	0.25
X(B) Malignant extracranial and extragonadal germ cell tumors	65	3.81	3.88	5	9.71	3.44	1992–2010	0.17 to 6.82	0.04
X(C) Malignant gonadal germ cell tumors	30	1.56	1.59	5	3.87	−0.28	1992–2010	−5.42 to 5.15	0.91
Other malignant epithelial neoplasms and malignant melanomas	50	2.88	2.94	5	7.34	1.03	1992–2010	−3.63 to 5.92	0.65
XI(F) Other and unspecified carcinomas	30	1.73	1.76	5	4.36	−2.09	1992–2010	−6.45 to 2.47	0.34
Other and unspecified malignant neoplasms	70	4.15	4.23	0	10.52	−5.42	1992–2010	−10.35 to −0.21	0.04
XII(B) Other unspecified malignant tumors	65	3.81	3.88	5	9.63	−6.47	1992–2010	−11.68 to −0.96	0.02
Age 1 to 4 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	6165	98.69	100.00	325	219.32	0.89	1992–2010	0.44 to 1.34	< 0.01
All childhood cancers including non-malignancies brain	6245	100.00	101.33	325	222.24	0.87	1992–2010	0.42 to 1.32	< 0.01

Continued on the following page

TABLE 2 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

Age 1 to 4 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	2640	42.27	42.83	135	93.95	0.82	1992–2010	−0.10 to 1.74	0.08
I(A) Lymphoid leukemias	2265	36.25	36.73	120	80.59	0.92	1992–2010	0.09 to 1.76	0.03
Lymphoid leukemias, precursor cell leukemias	2160	34.63	35.09	115	76.82	0.43	1992–2010	−0.61 to 1.49	0.39
I(B) Acute myeloid leukemias	260	4.13	4.19	15	9.15	−0.11	1992–2010	−2.80 to 2.65	0.93
I(D) Myelodysplastic syndrome and other myeloproliferative diseases	30	0.43	0.44	0	0.98	17.66	1992–2002	8.49 to 27.61	< 0.01
						−7.10	2002–2010	−20.03 to 7.91	0.31
I(E) Unspecified and other specified leukemias	65	1.04	1.05	0	2.32	2.60	1992–2010	−2.41 to 7.86	0.29
Lymphomas and reticuloendothelial neoplasms	300	4.77	4.83	15	10.63	2.22	1992–2010	−0.03 to 4.52	0.05
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	130	2.05	2.08	5	4.59	3.03	1992–2010	−0.39 to 6.56	0.08
II(C) Burkitt lymphoma	60	0.94	0.96	5	2.08	−2.82	1992–2010	−5.91 to 0.36	0.08
II(D) Miscellaneous lymphoreticular neoplasms	50	0.82	0.83	5	1.82	6.91	1992–2010	2.31 to 11.73	0.01
II(E) Unspecified lymphomas	35	0.54	0.55	0	1.23	2.09	1992–2010	−2.89 to 7.34	0.40
CNS and miscellaneous intracranial and intraspinal neoplasms	1070	17.11	17.34	55	38.10	1.20	1992–2010	−0.09 to 2.51	0.07
III(A) Ependymomas and choroid plexus tumor	170	2.69	2.73	10	5.97	1.59	1992–2010	−0.70 to 3.94	0.16
III(B) Astrocytomas	415	6.61	6.70	20	14.70	0.52	1992–2010	−1.59 to 2.67	0.61
III(C) Intracranial and intraspinal embryonal tumors	290	4.64	4.70	15	10.32	1.20	1992–2010	−1.22 to 3.68	0.31
III(D) Other gliomas	135	2.15	2.17	5	4.80	1.46	1992–2010	−1.37 to 4.38	0.29
III(F) Unspecified intracranial and intraspinal neoplasms	55	0.88	0.89	5	1.98	1.15	1992–2010	−4.95 to 7.65	0.70
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	1155	18.43	18.67	65	41.02	1.06	1992–2010	−0.22 to 2.35	0.10
III(A) Ependymomas and choroid plexus tumor including non-malignancies	170	2.79	2.82	10	6.18	1.77	1992–2010	−0.57 to 4.16	0.13
III(B) Astrocytomas including non-malignancies	425	6.76	6.85	20	15.03	0.50	1992–2010	−1.53 to 2.57	0.61
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	290	4.64	4.70	15	10.32	1.20	1992–2010	−1.22 to 3.68	0.31
III(D) Other gliomas including non-malignancies	135	2.15	2.17	10	4.80	1.46	1992–2010	−1.37 to 4.38	0.29
III(E) Other specified intracranial and intraspinal neoplasms including non-malignancies	55	0.90	0.91	0	2.02	1.12	1992–2010	−2.63 to 5.02	0.54
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	75	1.20	1.22	5	2.68	−0.52	1992–2010	−5.58 to 4.81	0.84
Neuroblastoma and other peripheral nervous cell tumors	620	9.97	10.11	35	22.22	1.62	1992–2010	0.20 to 3.05	0.03
IV(A) Neuroblastoma and ganglioneuroblastoma	620	9.88	10.01	30	22.01	1.66	1992–2010	0.28 to 3.07	0.02
Retinoblastoma	260	4.18	4.23	10	9.26	0.85	1992–2010	−1.56 to 3.32	0.47

Continued on the following page

TABLE 2 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

Age 1 to 4 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Renal tumours	555	8.84	8.96	25	19.53	−0.86	1992–2010	−2.50 to 0.80	0.29
VI(A) Nephroblastoma and other nonepithelial renal tumors	540	8.61	8.73	30	19.04	−0.88	1992–2010	−2.62 to 0.89	0.31
Hepatic tumours	145	2.34	2.37	5	5.21	2.83	1992–2010	0.04 to 5.70	0.05
VII(A) Hepatoblastoma	135	2.18	2.21	5	4.87	3.73	1992–2010	1.10 to 6.43	0.01
Malignant bone tumours	60	1.02	1.04	5	2.27	−0.89	1992–2010	−4.79 to 3.18	0.65
VIII(C) Ewing tumor and related sarcomas of bone	35	0.61	0.62	5	1.35	1.67	1992–2010	−3.41 to 7.03	0.50
Soft tissue and other extraosseous sarcomas	280	4.48	4.54	15	9.90	−4.09	1992–2003	−8.31 to 0.33	0.07
						8.78	2003–2010	−0.57 to 19.01	0.06
IX(A) Rhabdomyosarcomas	215	3.41	3.46	10	7.51	−5.42	1992–2004	−10.76 to 0.24	0.06
						15.86	2004–2010	−1.97 to 36.94	0.08
IX(D) Other specified soft tissue sarcomas	40	0.64	0.65	5	1.44	−1.06	1992–2010	−5.28 to 3.34	0.61
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	110	1.71	1.74	10	3.78	−1.74	1992–2010	−4.81 to 1.43	0.26
X(B) Malignant extracranial and extragonadal germ cell tumors	45	0.75	0.76	0	1.66	−1.78	1992–2010	−4.77 to 1.30	0.24
X(C) Malignant gonadal germ cell tumors	35	0.61	0.62	0	1.33	−4.20	1992–2010	−10.68 to 2.75	0.21
Other malignant epithelial neoplasms and malignant melanomas	50	0.77	0.78	0	1.71	6.00	1992–2010	0.61 to 11.69	0.03
Other and unspecified malignant neoplasms	75	1.22	1.23	5	2.75	3.90	1992–2010	−0.81 to 8.83	0.10
XII(B) Other unspecified malignant tumors	60	1.01	1.02	5	2.28	2.71	1992–2010	−3.05 to 8.81	0.34
Age 5 to 9 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	4335	97.31	100.00	225	118.00	0.37	1992–2010	−0.01 to 0.76	0.05
All childhood cancers including non-malignancies brain	4460	100.00	102.77	235	121.22	0.36	1992–2010	0.00 to 0.73	0.05
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	1485	33.30	34.22	80	40.42	0.85	1992–2010	−0.14 to 1.86	0.09
I(A) Lymphoid leukemias	1235	27.70	28.46	65	33.62	0.60	1992–2010	−0.45 to 1.66	0.25
Lymphoid leukemias, precursor cell leukemias	1165	26.06	26.78	60	31.57	−0.14	1992–2010	−1.27 to 1.01	0.80
I(B) Acute myeloid leukemias	160	3.57	3.66	10	4.33	0.50	1992–2010	−2.89 to 4.01	0.76
I(E) Unspecified and other specified leukemias	45	1.08	1.11	0	1.31	31.75	1992–1999	−2.28 to 77.63	0.07
						−2.34	1999–2010	−10.19 to 6.20	0.55

Continued on the following page

TABLE 2 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

Age 5 to 9 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Lymphomas and reticuloendothelial neoplasms	540	12.13	12.47	30	14.71	0.10	1992–2010	−1.40 to 1.62	0.89
II(A) Hodgkin lymphomas	135	2.96	3.04	5	3.59	−1.62	1992–2010	−4.65 to 1.50	0.29
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	210	4.69	4.82	15	5.66	−0.59	1992–2010	−3.02 to 1.90	0.62
II(C) Burkitt lymphoma	110	2.44	2.51	5	2.95	−0.41	1992–2010	−3.84 to 3.14	0.81
II(E) Unspecified lymphomas	65	1.46	1.50	5	1.77	2.78	1992–2010	−1.89 to 7.67	0.23
CNS and miscellaneous intracranial and intraspinal neoplasms	1140	25.57	26.27	60	30.95	−0.24	1992–2010	−1.38 to 0.92	0.67
III(A) Ependymomas and choroid plexus tumor	60	1.35	1.38	5	1.62	−1.11	1992–2010	−5.38 to 3.36	0.60
III(B) Astrocytomas	530	11.89	12.21	30	14.39	−0.85	1992–2010	−2.44 to 0.77	0.28
III(C) Intracranial and intraspinal embryonal tumors	290	6.53	6.71	15	7.87	−0.67	1992–2010	−3.01 to 1.73	0.56
III(D) Other gliomas	190	4.19	4.31	10	5.09	0.93	1992–2010	−2.26 to 4.23	0.55
III(F) Unspecified intracranial and intraspinal neoplasms	55	1.28	1.31	5	1.56	14.98	1992–2005	5.25 to 25.61	< 0.01
						−32.87	2005–2010	−55.39 to 1.03	0.06
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	1260	28.26	29.04	65	34.18	−0.24	1992–2010	−1.34 to 0.88	0.66
III(A) Ependymomas and choroid plexus tumor including non-malignancies	70	1.53	1.57	5	1.84	−2.04	1992–2010	−6.20 to 2.30	0.33
III(B) Astrocytomas including non-malignancies	550	12.27	12.61	25	14.84	−0.81	1992–2010	−2.38 to 0.78	0.30
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	290	6.53	6.71	15	7.87	−0.67	1992–2010	−3.01 to 1.73	0.56
III(D) Other gliomas including non-malignancies	185	4.19	4.31	10	5.09	0.93	1992–2010	−2.26 to 4.23	0.55
III(E) Other specified intracranial and intraspinal neoplasms including non-malignancies	100	2.15	2.21	5	2.60	17.06	1992–2001	4.32 to 31.35	0.01
						−4.60	2001–2010	−13.20 to 4.85	0.30
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	70	1.59	1.64	5	1.94	11.58	1992–2005	2.22 to 21.81	0.02
						−34.00	2005–2010	−60.57 to 10.46	0.11
Neuroblastoma and other peripheral nervous cell tumors	150	3.41	3.50	10	4.14	−0.32	1992–2010	−2.89 to 2.31	0.80
IV(A) Neuroblastoma and ganglioneuroblastoma	150	3.34	3.43	5	4.06	−0.01	1992–2010	−2.61 to 2.67	1.00
Renal tumours	220	4.84	4.98	15	5.83	−0.83	1992–2010	−3.55 to 1.97	0.54
VI(A) Nephroblastoma and other nonepithelial renal tumors	205	4.60	4.72	10	5.54	−0.97	1992–2010	−3.77 to 1.91	0.48
Malignant bone tumours	225	4.98	5.12	10	6.03	−0.95	1992–2010	−4.17 to 2.38	0.55
VIII(A) Osteosarcomas	105	2.33	2.40	5	2.84	−0.75	1992–2010	−4.41 to 3.04	0.68
VIII(C) Ewing tumor and related sarcomas of bone	95	2.20	2.26	5	2.66	−1.26	1992–2010	−6.25 to 3.99	0.61

Continued on the following page

TABLE 2 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

Age 5 to 9 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Soft tissue and other extraosseous sarcomas	295	6.71	6.89	15	8.15	0.15	1992–2010	−1.72 to 2.05	0.87
IX(A) Rhabdomyosarcomas	170	3.79	3.89	10	4.59	0.00	1992–2010	−1.82 to 1.85	1.00
IX(D) Other specified soft tissue sarcomas	85	1.86	1.91	5	2.26	−0.16	1992–2010	−4.18 to 4.03	0.94
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	70	1.59	1.64	5	1.93	1.65	1992–2010	−2.95 to 6.47	0.47
X(A) Intracranial and intraspinal germ cell tumors	35	0.76	0.78	0	0.93	16.29	1992–1999	2.48 to 31.96	0.02
						−10.62	1999–2006	−23.06 to 3.83	0.13
						18.51	2006–2010	−7.47 to 51.80	0.16
X(C) Malignant gonadal germ cell tumors	35	0.76	0.78	0	0.91	−1.20	1992–2010	−6.37 to 4.26	0.64
Other malignant epithelial neoplasms and malignant melanomas	120	2.69	2.77	5	3.28	3.24	1992–2010	−0.06 to 6.65	0.05
XI(B) Thyroid carcinomas	55	1.23	1.27	0	1.51	3.19	1992–2010	−0.33 to 6.84	0.07
XI(D) Malignant melanomas	35	0.74	0.76	0	0.92	6.13	1992–2010	3.16 to 9.19	< 0.01
Other and unspecified malignant neoplasms	55	1.19	1.22	0	1.46	2.07	1992–2010	−1.70 to 5.98	0.27
XII(B) Other unspecified malignant tumors	45	1.03	1.06	0	1.26	0.63	1992–2010	−3.58 to 5.03	0.76
Age 10 to 14 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	4750	96.09	100.00	250	122.57	0.17	1992–2010	−0.54 to 0.88	0.62
All childhood cancers including non-malignancies brain	4945	100.00	104.06	260	127.53	0.08	1992–2010	−0.57 to 0.72	0.80
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	1025	20.74	21.59	50	26.47	0.36	1992–2010	−0.85 to 1.57	0.54
I(A) Lymphoid leukemias	685	13.84	14.41	35	17.67	0.09	1992–2010	−1.06 to 1.25	0.87
Lymphoid leukemias, precursor cell leukemias	630	12.81	13.33	30	16.38	−0.54	1992–2010	−2.10 to 1.04	0.48
I(B) Acute myeloid leukemias	230	4.65	4.84	10	5.95	−0.34	1992–2010	−2.87 to 2.26	0.78
I(C) Chronic myeloproliferative diseases	50	1.03	1.07	0	1.31	3.27	1992–2010	−1.35 to 8.11	0.16
I(E) Unspecified and other specified leukemias	40	0.79	0.82	5	1.01	4.39	1992–2010	−1.28 to 10.38	0.12
Lymphomas and reticuloendothelial neoplasms	1010	20.40	21.23	55	26.00	0.36	1992–2010	−0.94 to 1.68	0.56
II(A) Hodgkin lymphomas	555	11.21	11.67	30	14.29	0.82	1992–2010	−0.87 to 2.53	0.32
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	250	5.02	5.22	15	6.41	−1.06	1992–2010	−4.20 to 2.17	0.49
II(C) Burkitt lymphoma	100	2.06	2.15	5	2.63	−4.35	1992–2010	−9.35 to 0.94	0.10
II(E) Unspecified lymphomas	85	1.70	1.77	5	2.16	5.25	1992–2010	2.30 to 8.28	< 0.01

Continued on the following page

TABLE 2 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

Age 10 to 14 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
CNS and miscellaneous intracranial and intraspinal neoplasms	945	19.13	19.90	50	24.40	−0.77	1992–2010	−1.98 to 0.45	0.20
III(A) Ependymomas and choroid plexus tumor	60	1.23	1.28	0	1.56	5.11	1992–2010	1.45 to 8.91	0.01
III(B) Astrocytomas	495	10.02	10.43	25	12.80	−2.07	1992–2010	−3.67 to −0.45	0.02
III(C) Intracranial and intraspinal embryonal tumors	170	3.46	3.60	10	4.43	−2.34	1992–2010	−4.64 to 0.02	0.05
III(D) Other gliomas	140	2.83	2.95	5	3.62	0.69	1992–2010	−2.82 to 4.34	0.69
III(F) Unspecified intracranial and intraspinal neoplasms	65	1.30	1.35	5	1.63	4.34	1992–2010	0.19 to 8.67	0.04
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	1135	23.03	23.97	60	29.36	−1.02	1992–2010	−2.31 to 0.28	0.12
III(A) Ependymomas and choroid plexus tumor including non-malignancies	80	1.60	1.66	5	2.02	3.82	1992–2010	0.37 to 7.38	0.03
III(B) Astrocytomas including non-malignancies	510	10.34	10.76	25	13.22	−2.11	1992–2010	−3.69 to −0.50	0.01
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	170	3.46	3.60	10	4.43	−2.34	1992–2010	−4.64 to 0.02	0.05
III(D) Other gliomas including non-malignancies	145	2.85	2.97	10	3.64	0.73	1992–2010	−2.70 to 4.28	0.66
III(E) Other specified intracranial and intraspinal neoplasms including non-malignancies	150	3.04	3.16	10	3.86	−0.30	1992–2010	−3.85 to 3.38	0.86
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	85	1.74	1.81	5	2.20	1.79	1992–2010	−1.93 to 5.65	0.33
Neuroblastoma and other peripheral nervous cell tumors	40	0.85	0.88	5	1.08	−1.01	1992–2010	−4.58 to 2.68	0.56
IV(A) Neuroblastoma and ganglioneuroblastoma	35	0.67	0.70	0	0.85	−0.01	1992–2010	−4.72 to 4.92	0.99
Renal tumours	55	1.09	1.14	0	1.39	−1.32	1992–2010	−5.91 to 3.50	0.56
VI(A) Nephroblastoma and other nonepithelial renal tumors	35	0.67	0.70	0	0.85	−0.32	1992–2010	−5.27 to 4.89	0.90
Hepatic tumours	30	0.55	0.57	5	0.70	−5.02	1992–2010	−8.68 to −1.21	0.01
Malignant bone tumours	475	9.55	9.94	25	12.18	−0.30	1992–2010	−1.59 to 1.01	0.64
VIII(A) Osteosarcomas	260	5.32	5.54	15	6.79	−0.56	1992–2010	−2.90 to 1.85	0.63
VIII(C) Ewing tumor and related sarcomas of bone	160	3.30	3.43	10	4.19	1.39	1992–2010	−0.94 to 3.78	0.23
Soft tissue and other extraosseous sarcomas	370	7.45	7.75	15	9.49	−0.54	1992–2010	−3.05 to 2.04	0.66
IX(A) Rhabdomyosarcomas	115	2.35	2.44	5	3.00	−1.54	1992–2010	−5.18 to 2.23	0.40
IX(B) Fibrosarcomas, peripheral nerve sheath tumors, and other fibrous neoplasms	35	0.73	0.76	0	0.93	−0.81	1992–2010	−4.85 to 3.41	0.69
IX(D) Other specified soft tissue sarcomas	155	3.20	3.33	10	4.07	−1.03	1992–2010	−4.40 to 2.45	0.54
IX(E) Unspecified soft tissue sarcomas	55	1.17	1.22	0	1.50	1.80	1992–2010	−2.11 to 5.86	0.35
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	270	5.50	5.73	15	7.03	−0.80	1992–2010	−3.31 to 1.78	0.52
X(A) Intracranial and intraspinal germ cell tumors	100	2.04	2.13	5	2.62	−0.16	1992–2010	−3.37 to 3.15	0.92
X(C) Malignant gonadal germ cell tumors	130	2.67	2.78	10	3.41	−0.61	1992–2010	−3.81 to 2.71	0.70

Continued on the following page

TABLE 2 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnoses by age, Canada, 1992–2010

Age 10 to 14 years									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Other malignant epithelial neoplasms and malignant melanomas	455	9.15	9.52	25	11.67	1.72	1992–2010	−0.77 to 4.27	0.16
XI(B) Thyroid carcinomas	190	3.93	4.09	15	5.01	3.47	1992–2010	−0.37 to 7.47	0.07
XI(D) Malignant melanomas	95	1.92	2.00	5	2.45	−1.34	1992–2010	−4.01 to 1.41	0.32
XI(F) Other and unspecified carcinomas	135	2.73	2.84	10	3.49	−0.28	1992–2010	−3.78 to 3.34	0.87
Other and unspecified malignant neoplasms	80	1.60	1.66	5	2.03	7.20	1992–2010	3.71 to 10.80	< 0.01
XII(B) Other unspecified malignant tumors	70	1.38	1.43	5	1.75	7.52	1992–2010	3.84 to 11.32	< 0.01

Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
[a] The ASIRs were standardized to the 2011 Canadian population.

TABLE 3
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males and females combined, Canada, 1992–2010

British Columbia									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	2030	96.26	100.00	105	152.22	0.23	1992–2010	−0.80 to 1.27	0.65
All childhood cancers including non-malignancies brain	2110	100.00	103.89	110	158.01	0.32	1992–2010	−0.67 to 1.31	0.51
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	695	32.92	34.20	40	52.82	0.33	1992–2010	−1.14 to 1.83	0.64
Lymphomas and reticuloendothelial neoplasms	220	10.37	10.78	10	15.81	1.03	1992–2010	−1.92 to 4.06	0.48
CNS and miscellaneous intracranial and intraspinal neoplasms	380	18.00	18.70	20	28.30	0.43	1992–2010	−1.29 to 2.18	0.61
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	460	21.74	22.59	25	34.09	0.79	1992–2010	−0.87 to 2.47	0.33
Neuroblastoma and other peripheral nervous cell tumors	155	7.34	7.63	5	12.19	2.44	1992–2010	−0.11 to 5.06	0.06
Retinoblastoma	50	2.32	2.41	5	3.87	−1.54	1992–2010	−4.88 to 1.92	0.36
Renal tumours	110	5.07	5.27	5	8.15	−1.32	1992–2010	−3.89 to 1.32	0.30
Hepatic tumours	35	1.61	1.67	0	2.61	9.86	1992–1999	−0.72 to 21.56	0.06
						−32.48	1999–2002	−71.72 to 61.22	0.33
						43.69	2002–2005	–	0.37
						−14.53	2005–2010	−29.37 to 3.43	0.09

Continued on the following page

TABLE 3 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males and females combined, Canada, 1992–2010

British Columbia									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Malignant bone tumours	115	5.45	5.66	5	8.18	−1.12	1992–2010	−4.46 to 2.34	0.50
Soft tissue and other extraosseous sarcomas	135	6.30	6.55	5	9.74	−0.69	1992–2010	−3.58 to 2.29	0.63
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	75	3.55	3.69	5	5.44	−2.11	1992–2010	−6.51 to 2.49	0.34
Other malignant epithelial neoplasms and malignant melanomas	60	2.98	3.10	5	4.56	0.49	1992–2010	−3.65 to 4.79	0.81
Prairies									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	2885	94.97	100.00	155	141.12	0.35	1992–2010	−0.39 to 1.10	0.33
All childhood cancers including non-malignancies brain	3040	100.00	105.30	160	148.50	0.26	1992–2010	−0.47 to 1.00	0.47
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	945	31.16	32.81	50	46.71	0.84	1992–2010	−0.55 to 2.25	0.22
Lymphomas and reticuloendothelial neoplasms	325	10.73	11.30	15	15.64	1.86	1992–2010	−0.17 to 3.94	0.07
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	115	3.82	4.02	5	5.60	4.81	1992–2010	1.58 to 8.14	0.01
CNS and miscellaneous intracranial and intraspinal neoplasms	610	20.04	21.10	30	29.47	−0.09	1992–2010	−1.49 to 1.33	0.90
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	760	25.07	26.40	40	36.85	−0.39	1992–2010	−1.69 to 0.92	0.53
Neuroblastoma and other peripheral nervous cell tumors	205	6.68	7.03	10	10.22	0.17	1992–2010	−1.43 to 1.79	0.83
Retinoblastoma	75	2.44	2.56	5	3.71	−0.31	1992–2010	−4.14 to 3.68	0.87
Renal tumours	185	6.02	6.34	10	9.10	−1.65	1992–2010	−4.29 to 1.07	0.22
Hepatic tumours	55	1.68	1.77	0	2.57	3.48	1992–2010	−1.25 to 8.44	0.14
Malignant bone tumours	125	4.15	4.37	10	5.98	1.25	1992–2010	−1.90 to 4.50	0.42
VIII(C) Ewing tumor and related sarcomas of bone	45	1.55	1.63	0	2.23	4.14	1992–2010	0.72 to 7.68	0.02
Soft tissue and other extraosseous sarcomas	175	5.69	5.99	5	8.40	−0.12	1992–2010	−2.68 to 2.52	0.92
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	105	3.49	3.67	5	5.12	−0.68	1992–2010	−3.56 to 2.28	0.63
X(C) Malignant gonadal germ cell tumors	40	1.32	1.39	0	1.93	−5.85	1992–2010	−9.49 to −2.06	0.01
Other malignant epithelial neoplasms and malignant melanomas	80	2.67	2.81	0	3.87	−1.39	1992–2010	−5.45 to 2.85	0.49
Ontario[b]									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	6655	100.00	100.00	350	157.62	−0.05	1992–2006	−0.67 to 0.56	0.85
						5.91	2006–2010	1.90 to 10.08	0.01

Continued on the following page

TABLE 3 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males and females combined, Canada, 1992–2010

	Ontario[b]								
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	2110	31.69	31.69	110	50.12	0.78	1992–2010	−0.34 to 1.92	0.16
I(A) Lymphoid leukemias	1675	25.17	25.17	90	39.87	1.33	1992–2010	0.24 to 2.44	0.02
Lymphoid leukemias, precursor cell leukemias	1480	22.29	22.29	80	35.26	1.12	1992–2000	−2.78 to 5.17	0.55
						−13.91	2000–2004	−29.82 to 5.62	0.14
						14.23	2004–2010	7.50 to 21.37	< 0.01
I(C) Chronic myeloproliferative diseases	30	0.45	0.45	0	0.70	−9.02	1992–2010	−13.48 to −4.33	< 0.01
I(E) Unspecified and other specified leukemias	90	1.40	1.40	5	2.21	18.50	1992–2001	5.61 to 32.96	0.01
						−12.68	2001–2010	−22.51 to −1.59	0.03
Lymphomas and reticuloendothelial neoplasms	775	11.63	11.63	40	17.91	0.64	1992–2010	−0.83 to 2.12	0.37
II(D) Miscellaneous lymphoreticular neoplasms	65	0.98	0.98	0	1.55	7.59	1992–2010	3.09 to 12.29	< 0.01
II(E) Unspecified lymphomas	155	2.28	2.28	5	3.50	4.34	1992–2010	1.27 to 7.51	0.01
CNS and miscellaneous intracranial and intraspinal neoplasms	1340	20.14	20.14	70	31.49	−1.40	1992–2004	−2.82 to 0.05	0.06
						4.99	2004–2010	0.96 to 9.18	0.02
III(A) Ependymomas and choroid plexus tumor	115	1.68	1.68	5	2.69	3.33	1992–2010	0.68 to 6.05	0.02
III(B) Astrocytomas	575	8.65	8.65	30	13.47	−5.90	1992–2004	−7.96 to −3.80	< 0.01
						5.65	2004–2010	−1.56 to 13.39	0.12
III(D) Other gliomas	185	2.76	2.76	5	4.32	4.52	1992–2010	2.34 to 6.74	< 0.01
III(F) Unspecified intracranial and intraspinal neoplasms	145	2.16	2.16	5	3.35	12.13	1992–2005	3.76 to 21.17	0.01
						−21.74	2005–2010	−40.25 to 2.51	0.07
Neuroblastoma and other peripheral nervous cell tumors	445	6.67	6.67	25	10.94	1.85	1992–2010	−0.35 to 4.09	0.09
Retinoblastoma	165	2.43	2.43	10	4.03	1.10	1992–2010	−1.26 to 3.52	0.34
Renal tumours	350	5.29	5.29	15	8.45	−0.63	1992–2010	−2.40 to 1.17	0.47
Hepatic tumours	105	1.59	1.59	5	2.60	1.15	1992–2010	−2.01 to 4.42	0.46
Malignant bone tumours	285	4.28	4.28	15	6.58	−0.90	1992–2010	−3.02 to 1.28	0.39
Soft tissue and other extraosseous sarcomas	395	5.93	5.93	25	9.32	0.28	1992–2010	−1.49 to 2.08	0.75
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	205	3.06	3.06	10	4.82	1.46	1992–2010	−0.88 to 3.85	0.21
Other malignant epithelial neoplasms and malignant melanomas	290	4.33	4.33	15	6.66	3.85	1992–2010	1.07 to 6.71	0.01
XI(B) Thyroid carcinomas	110	1.65	1.65	10	2.49	6.34	1992–2010	2.98 to 9.81	< 0.01

Continued on the following page

TABLE 3 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males and females combined, Canada, 1992–2010

Ontario[b]									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Other and unspecified malignant neoplasms	195	2.96	2.96	10	4.69	2.17	1992–2010	−2.01 to 6.54	0.29
XII(B) Other unspecified malignant tumors	180	2.70	2.70	10	4.27	65.13	1992–1996	5.53 to 158.41	0.03
						−2.18	1996–2010	−6.07 to 1.87	0.26
Quebec									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	4140	96.60	100.00	220	168.59	0.13	1992–2010	−0.53 to 0.79	0.69
All childhood cancers including non-malignancies brain	4290	100.00	103.52	225	174.36	0.05	1992–2010	−0.60 to 0.71	0.87
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	1310	30.54	31.61	70	53.70	0.57	1992–2010	−0.52 to 1.67	0.29
I(D) Myelodysplastic syndrome and other myeloproliferative diseases	35	0.77	0.80	0	1.38	18.53	1992–2004	4.42 to 34.55	0.01
						−12.36	2004–2010	−24.97 to 2.35	0.09
Lymphomas and reticuloendothelial neoplasms	460	10.72	11.10	25	18.16	−1.06	1992–2010	−2.97 to 0.90	0.27
CNS and miscellaneous intracranial and intraspinal neoplasms	770	17.88	18.51	40	31.01	0.50	1992–2010	−0.75 to 1.77	0.41
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	910	21.28	22.03	45	36.78	2.58	1992–2005	1.19 to 3.99	< 0.01
						−10.14	2005–2010	−16.05 to −3.83	< 0.01
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	80	1.84	1.91	5	3.14	−14.70	1992–2001	−25.16 to −2.77	0.02
						47.35	2001–2004	–	0.55
						−33.06	2004–2010	−54.59 to −1.34	0.04
Neuroblastoma and other peripheral nervous cell tumors	385	8.97	9.29	20	16.08	−1.27	1992–2010	−3.56 to 1.08	0.27
Retinoblastoma	115	2.66	2.75	5	4.79	−0.20	1992–2010	−3.46 to 3.17	0.90
Renal tumours	235	5.57	5.77	15	10.01	0.08	1992–2010	−2.61 to 2.85	0.95
Hepatic tumours	45	1.10	1.13	5	1.99	4.06	1992–2010	−0.33 to 8.64	0.07
Malignant bone tumours	185	4.31	4.46	5	7.21	−4.88	1992–2002	−8.45 to −1.16	0.01
						6.19	2002–2010	0.39 to 12.32	0.04
Soft tissue and other extraosseous sarcomas	265	6.25	6.47	15	10.78	0.04	1992–2010	−2.27 to 2.41	0.97
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	130	3.05	3.16	10	5.31	−1.21	1992–2010	−4.90 to 2.63	0.51
X(B) Malignant extracranial and extragonadal germ cell tumors	35	0.77	0.80	0	1.41	9.46	1992–2004	2.62 to 16.75	0.01
						−26.73	2004–2010	−40.96 to −9.06	0.01

Continued on the following page

TABLE 3 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males and females combined, Canada, 1992–2010

Quebec									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Other malignant epithelial neoplasms and malignant melanomas	180	4.22	4.37	10	7.20	3.47	1992–2010	0.40 to 6.63	0.03
XI(B) Thyroid carcinomas	75	1.72	1.79	5	2.94	8.21	1992–2010	3.61 to 13.02	< 0.01
Other and unspecified malignant neoplasms	60	1.33	1.38	0	2.36	1.09	1992–2010	−4.07 to 6.53	0.67
Atlantic provinces									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	1180	96.00	100.00	60	152.27	0.34	1992–2010	−0.90 to 1.59	0.57
All childhood cancers including non-malignancies brain	1225	100.00	104.16	65	158.19	0.23	1992–2010	−1.01 to 1.49	0.70
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	410	33.44	34.83	20	53.90	0.90	1992–2010	−1.20 to 3.05	0.38
Lymphomas and reticuloendothelial neoplasms	120	9.71	10.11	5	14.72	1.38	1992–2010	−1.47 to 4.30	0.33
CNS and miscellaneous intracranial and intraspinal neoplasms	230	18.68	19.46	10	28.77	8.63	1992–1998	−1.07 to 19.29	0.08
						−6.38	1998–2010	−10.19 to −2.40	< 0.01
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	280	22.68	23.62	15	34.69	10.74	1992–1998	1.84 to 20.43	0.02
						−7.22	1998–2010	−10.52 to −3.79	< 0.01
Neuroblastoma and other peripheral nervous cell tumors	75	5.87	6.12	5	10.12	2.22	1992–2010	−0.60 to 5.11	0.12
Retinoblastoma	30	2.45	2.55	0	4.19	55.50	1992–1996	–	0.23
						−30.80	1996–2002	−44.84 to −13.17	0.01
						163.12	2002–2006	–	0.04
						−55.12	2006–2010	−71.86 to −28.43	< 0.01
Renal tumours	65	5.22	5.44	5	8.81	1.27	1992–2010	−2.62 to 5.32	0.51
Malignant bone tumours	50	4.00	4.16	5	5.91	3.84	1992–2010	−1.65 to 9.64	0.16
Soft tissue and other extraosseous sarcomas	90	7.01	7.31	5	10.77	0.07	1992–2010	−4.28 to 4.62	0.97
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	35	3.10	3.23	0	4.88	−0.87	1992–2010	−3.88 to 2.23	0.56
Other malignant epithelial neoplasms and malignant melanomas	50	4.32	4.50	0	6.43	0.63	1992–2010	−2.27 to 3.61	0.66
Territories									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	55	98.21	100.00	5	106.35	0.51	1992–2010	−2.90 to 4.04	0.76
All childhood cancers including non-malignant CNS tumors	55	100.00	101.82	5	108.15	0.53	1992–2010	−2.88 to 4.07	0.75

Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
[a] The ASIRs were standardized to the 2011 Canadian population.
[b] There were no non-malignant cases in Ontario.

TABLE 4
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males, Canada, 1992–2010

British Columbia									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	1095	96.21	100.00	55	158.93	0.41	1992–2010	−1.06 to 1.90	0.56
All childhood cancers including non-malignancies brain	1135	100.00	103.94	60	165.02	0.53	1992–2010	−0.90 to 1.98	0.45
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	380	33.66	34.98	20	56.45	0.94	1992–2010	−0.84 to 2.74	0.28
Lymphomas and reticuloendothelial neoplasms	150	13.04	13.55	10	20.83	2.72	1992–2010	−0.77 to 6.32	0.12
CNS and miscellaneous intracranial and intraspinal neoplasms	190	16.92	17.58	10	27.64	−0.24	1992–2010	−2.61 to 2.18	0.83
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	235	20.70	21.52	10	33.73	0.48	1992–2010	−2.05 to 3.08	0.70
Neuroblastoma and other peripheral nervous cell tumors	70	6.17	6.41	5	10.70	3.71	1992–2010	−0.41 to 8.00	0.08
Retinoblastoma	30	2.47	2.56	5	4.26	−3.92	1992–2010	−6.78 to −0.97	0.01
Renal tumours	60	5.02	5.22	0	8.48	0.68	1992–2010	−2.70 to 4.19	0.68
Malignant bone tumours	55	4.85	5.04	0	7.64	−1.88	1992–2010	−7.61 to 4.22	0.52
Soft tissue and other extraosseous sarcomas	70	5.99	6.23	5	9.66	−0.62	1992–2010	−4.66 to 3.59	0.76
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	40	3.52	3.66	0	5.72	−4.24	1992–2010	−8.65 to 0.37	0.07
Other malignant epithelial neoplasms and malignant melanomas	30	2.29	2.38	5	3.68	0.15	1992–2010	−4.13 to 4.63	0.94
Prairies									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	1580	95.13	100.00	85	150.77	0.85	1992–2010	−0.09 to 1.79	0.07
All childhood cancers including non-malignancies brain	1660	100.00	105.12	85	158.35	0.76	1992–2010	−0.12 to 1.64	0.09
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	535	32.35	34.01	30	51.70	1.54	1992–2010	−0.40 to 3.52	0.11
Lymphomas and reticuloendothelial neoplasms	210	12.75	13.40	15	19.78	1.16	1992–2010	−1.15 to 3.52	0.31
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	75	4.45	4.68	0	6.91	3.84	1992–2010	0.29 to 7.51	0.04
CNS and miscellaneous intracranial and intraspinal neoplasms	335	19.96	20.99	15	31.36	0.99	1992–2010	−0.72 to 2.74	0.24
III(C) Intracranial and intraspinal embryonal tumors	75	4.63	4.87	5	7.27	−18.75	1992–1996	−43.00 to 15.83	0.23
						6.34	1996–2010	0.33 to 12.71	0.04
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	410	24.83	26.11	20	38.94	0.64	1992–2010	−0.80 to 2.10	0.36
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	80	4.63	4.87	5	7.27	−18.75	1992–1996	−43.00 to 15.83	0.23
						6.34	1996–2010	0.33 to 12.71	0.04
Neuroblastoma and other peripheral nervous cell tumors	100	6.01	6.32	5	9.84	1.04	1992–2010	−1.98 to 4.15	0.48
Retinoblastoma	30	1.98	2.09	0	3.27	−2.99	1992–2010	−7.49 to 1.73	0.20

Continued on the following page

TABLE 4 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males, Canada, 1992–2010

Prairies									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Renal tumours	90	5.29	5.56	5	8.50	−3.27	1992–2010	−6.35 to −0.09	0.04
Hepatic tumours	35	2.10	2.21	0	3.43	4.34	1992–2010	−0.78 to 9.71	0.09
Malignant bone tumours	65	3.67	3.86	5	5.67	1.32	1992–2010	−2.64 to 5.45	0.50
Soft tissue and other extraosseous sarcomas	95	5.65	5.94	5	8.87	−1.35	1992–2010	−4.96 to 2.40	0.45
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	50	2.89	3.03	0	4.55	−2.61	1992–2010	−6.80 to 1.76	0.22
Other malignant epithelial neoplasms and malignant melanomas	40	2.22	2.34	0	3.44	1.75	1992–2010	−2.90 to 6.62	0.44
Ontario[b]									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	3585	100.00	100.00	190	165.70	1.55	1992–2002	0.45 to 2.66	0.01
						−5.12	2002–2005	−17.46 to 9.05	0.42
						5.00	2005–2010	1.87 to 8.23	< 0.01
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	1150	32.11	32.11	60	53.46	0.89	1992–2010	−0.06 to 1.84	0.06
I(A) Lymphoid leukemias	935	26.05	26.05	45	43.43	1.36	1992–2010	0.28 to 2.45	0.02
Lymphoid leukemias, precursor cell leukemias	820	22.93	22.93	45	38.16	1.28	1992–2001	−2.63 to 5.34	0.49
						−18.31	2001–2004	−50.22 to 34.07	0.39
						13.60	2004–2010	5.26 to 22.61	< 0.01
Lymphomas and reticuloendothelial neoplasms	505	14.06	14.06	25	22.79	1.15	1992–2010	−0.76 to 3.09	0.22
II(E) Unspecified lymphomas	100	2.79	2.79	5	4.51	4.46	1992–2010	1.69 to 7.30	< 0.01
CNS and miscellaneous intracranial and intraspinal neoplasms	730	20.31	20.31	40	33.34	0.26	1992–2010	−1.07 to 1.61	0.68
III(B) Astrocytomas	285	8.01	8.01	15	13.09	−2.42	1992–2010	−4.58 to −0.22	0.03
III(D) Other gliomas	90	2.51	2.51	5	4.14	4.71	1992–2010	1.01 to 8.54	0.02
Neuroblastoma and other peripheral nervous cell tumors	250	6.89	6.89	10	11.87	1.30	1992–2010	−1.16 to 3.83	0.28
Retinoblastoma	80	2.26	2.26	0	3.92	−1.36	1992–2010	−5.10 to 2.53	0.47
Renal tumours	150	4.13	4.13	10	7.00	−0.15	1992–2010	−2.75 to 2.53	0.91
Hepatic tumours	65	1.84	1.84	5	3.13	3.58	1992–2010	0.32 to 6.94	0.03
VII(A) Hepatoblastoma	55	1.48	1.48	5	2.54	5.80	1992–2010	2.68 to 9.02	< 0.01
Malignant bone tumours	145	4.02	4.02	5	6.47	−0.71	1992–2010	−3.66 to 2.32	0.62
Soft tissue and other extraosseous sarcomas	220	6.19	6.19	10	10.17	−0.74	1992–2010	−3.30 to 1.89	0.56

Continued on the following page

TABLE 4 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males, Canada, 1992–2010

Ontario[b]									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	85	2.48	2.48	5	4.14	1.31	1992–2010	−2.62 to 5.39	0.50
Other malignant epithelial neoplasms and malignant melanomas	115	3.18	3.18	5	5.16	1.36	1992–2010	−2.09 to 4.93	0.42
XI(B) Thyroid carcinomas	35	0.86	0.86	0	1.36	5.68	1992–2010	1.91 to 9.58	0.01
Other and unspecified malignant neoplasms	95	2.54	2.54	5	4.25	1.20	1992–2010	−4.34 to 7.06	0.66
Quebec									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	2230	95.92	100.00	115	177.37	0.14	1992–2010	−0.63 to 0.92	0.71
All childhood cancers including non-malignancies brain	2325	100.00	104.25	125	184.65	−0.05	1992–2010	−0.79 to 0.70	0.89
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	715	30.63	31.93	35	56.99	0.14	1992–2010	−1.49 to 1.80	0.86
I(C) Chronic myeloproliferative diseases	30	1.25	1.30	0	2.35	5.96	1992–2010	1.38 to 10.75	0.01
Lymphomas and reticuloendothelial neoplasms	320	13.57	14.15	20	24.37	−1.80	1992–2010	−3.96 to 0.40	0.10
CNS and miscellaneous intracranial and intraspinal neoplasms	425	18.08	18.85	20	33.30	0.35	1992–2010	−1.50 to 2.24	0.69
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	515	22.16	23.11	25	40.59	2.06	1992–2004	0.14 to 4.01	0.04
						−7.38	2004–2010	−12.91 to −1.50	0.02
III(E) Other specified intracranial and intraspinal neoplasms including non-malignancies	55	2.28	2.37	0	4.05	−13.60	1992–2000	−22.56 to −3.60	0.01
						14.61	2000–2006	−2.67 to 34.97	0.09
						−47.06	2006–2010	−73.25 to 4.77	0.06
III(F) Unspecified intracranial and intraspinal neoplasms including non-malignancies	50	2.10	2.19	5	3.78	−7.94	1992–2010	−12.27 to −3.41	< 0.01
Neuroblastoma and other peripheral nervous cell tumors	195	8.29	8.64	10	15.87	0.38	1992–2010	−2.10 to 2.93	0.75
Retinoblastoma	60	2.58	2.69	5	4.89	−0.92	1992–2010	−4.05 to 2.32	0.55
Renal tumours	105	4.38	4.57	5	8.40	0.97	1992–2010	−3.36 to 5.50	0.65
Hepatic tumours	30	1.29	1.34	0	2.49	2.12	1992–2010	−2.37 to 6.81	0.34
Malignant bone tumours	100	4.17	4.34	5	7.38	0.99	1992–2010	−1.95 to 4.02	0.49
Soft tissue and other extraosseous sarcomas	140	6.01	6.27	10	10.93	−0.58	1992–2010	−3.89 to 2.85	0.72
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	60	2.66	2.78	5	4.94	1.02	1992–2010	−2.55 to 4.73	0.56
Other malignant epithelial neoplasms and malignant melanomas	70	2.92	3.05	5	5.28	2.62	1992–2010	−2.05 to 7.51	0.26
Other and unspecified malignant neoplasms	30	1.33	1.39	5	2.54	4.58	1992–2010	0.13 to 9.23	0.04

Continued on the following page

TABLE 4 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, males, Canada, 1992–2010

Atlantic provinces									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	615	95.49	100.00	35	154.99	0.06	1992–2010	−1.81 to 1.96	0.95
All childhood cancers including non-malignancies brain	645	100.00	104.72	35	161.76	−0.12	1992–2010	−2.08 to 1.89	0.90
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	210	33.13	34.69	10	54.85	1.61	1992–2010	−1.38 to 4.68	0.27
Lymphomas and reticuloendothelial neoplasms	80	12.29	12.87	5	19.19	1.72	1992–2010	−2.17 to 5.77	0.37
CNS and miscellaneous intracranial and intraspinal neoplasms	120	18.20	19.06	5	28.53	−2.78	1992–2010	−5.99 to 0.54	0.09
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	145	22.71	23.78	5	35.30	12.16	1992–1997	−5.79 to 33.52	0.18
						−7.45	1997–2010	−12.19 to −2.45	0.01
Neuroblastoma and other peripheral nervous cell tumors	40	6.22	6.51	5	10.96	0.35	1992–2010	−3.31 to 4.15	0.84
Malignant bone tumours	30	4.67	4.89	5	7.12	1.69	1992–2010	−4.36 to 8.13	0.57
Soft tissue and other extraosseous sarcomas	45	6.53	6.84	5	10.22	−24.40	1992–1998	−37.07 to −9.17	0.01
						21.86	1998–2006	0.80 to 47.31	0.04
						−25.26	2006–2010	−49.89 to 11.46	0.14
Territories									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	25	100.00	100.00	0	95.99	0.42	1992–2010	−3.46 to 4.45	0.82
All childhood cancers including non-malignant CNS tumors	25	100.00	100.00	0	95.99	0.42	1992–2010	−3.46 to 4.45	0.82

Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
[a] The ASIRs were standardized to the 2011 Canadian population.
[b] There were no non-malignant cases in Ontario.

TABLE 5
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, females, Canada, 1992–2010

British Columbia									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	940	96.31	100.00	50	145.09	−0.07	1992–2010	−1.56 to 1.43	0.92
All childhood cancers including non-malignancies brain	975	100.00	103.83	50	150.57	0.00	1992–2010	−1.50 to 1.52	1.00
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	310	32.07	33.30	20	48.97	−0.57	1992–2010	−2.78 to 1.70	0.60
I(B) Acute myeloid leukemias	50	5.02	5.21	5	7.50	−17.65	1992–1996	−37.50 to 8.50	0.15
						38.45	1996–1999	–	0.38
						−10.68	1999–2010	−16.27 to −4.72	< 0.01
Lymphomas and reticuloendothelial neoplasms	70	7.27	7.55	5	10.49	−1.96	1992–2010	−6.17 to 2.43	0.35
CNS and miscellaneous intracranial and intraspinal neoplasms	190	19.26	20.00	10	29.01	0.95	1992–2010	−1.80 to 3.77	0.48
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	220	22.95	23.83	15	34.49	0.93	1992–2010	−1.73 to 3.67	0.47
Neuroblastoma and other peripheral nervous cell tumors	85	8.71	9.04	5	13.76	1.44	1992–2010	−2.01 to 5.01	0.40
Renal tumours	50	5.12	5.32	0	7.80	−2.06	1992–2010	−6.62 to 2.73	0.37
Malignant bone tumours	60	6.15	6.38	5	8.75	−1.80	1992–2010	−5.81 to 2.37	0.37
Soft tissue and other extraosseous sarcomas	65	6.66	6.91	5	9.83	0.67	1992–2010	−3.22 to 4.73	0.72
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	35	3.59	3.72	5	5.14	2.86	1992–2010	−2.08 to 8.04	0.24
Other malignant epithelial neoplasms and malignant melanomas	40	3.79	3.94	5	5.48	0.30	1992–2010	−4.31 to 5.14	0.89
Prairies									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	1305	94.77	100.00	70	130.92	−0.24	1992–2010	−1.18 to 0.72	0.61
All childhood cancers including non-malignancies brain	1375	100.00	105.52	75	138.09	−0.35	1992–2010	−1.31 to 0.62	0.45
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	410	29.72	31.37	20	41.42	−0.02	1992–2010	−1.47 to 1.46	0.98
Lymphomas and reticuloendothelial neoplasms	110	8.28	8.74	5	11.28	3.50	1992–2010	0.26 to 6.84	0.04
II(B) Non-Hodgkin lymphomas (except Burkitt lymphoma)	45	3.05	3.22	0	4.21	6.03	1992–2010	1.64 to 10.61	0.01
CNS and miscellaneous intracranial and intraspinal neoplasms	280	20.13	21.24	15	27.47	−1.38	1992–2010	−3.43 to 0.72	0.18
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	350	25.36	26.76	15	34.64	−1.62	1992–2010	−3.76 to 0.56	0.13
Neuroblastoma and other peripheral nervous cell tumors	100	7.49	7.90	5	10.61	−1.29	1992–2010	−4.42 to 1.93	0.40
Retinoblastoma	45	2.98	3.14	0	4.18	−1.50	1992–2010	−4.86 to 1.99	0.37
Renal tumours	95	6.90	7.29	5	9.73	−0.07	1992–2010	−3.92 to 3.94	0.97

Continued on the following page

TABLE 5 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, females, Canada, 1992–2010

Prairies									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Malignant bone tumours	65	4.72	4.98	5	6.30	0.64	1992–2010	−4.14 to 5.66	0.79
Soft tissue and other extraosseous sarcomas	75	5.74	6.06	0	7.92	1.72	1992–2010	−1.76 to 5.33	0.32
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	60	4.22	4.45	0	5.73	−4.00	1992–2005	−8.39 to 0.60	0.08
						11.42	2005–2010	−7.78 to 34.62	0.24
Other malignant epithelial neoplasms and malignant melanomas	40	3.20	3.37	0	4.32	−4.46	1992–2010	−8.19 to −0.59	0.03
Ontario[b]									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	3070	100.00	100.00	165	149.15	−0.48	1992–2006	−1.82 to 0.87	0.45
						8.96	2006–2010	0.15 to 18.56	0.05
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	960	31.21	31.21	50	46.64	0.58	1992–2010	−1.29 to 2.48	0.53
Lymphoid leukemias, precursor cell leukemias	665	21.54	21.54	35	32.22	10.41	1992–1996	−8.74 to 33.58	0.28
						−8.94	1996–2004	−16.60 to −0.57	0.04
						14.42	2004–2010	3.53 to 26.46	0.01
I(E) Unspecified and other specified leukemias	45	1.50	1.50	5	2.26	17.14	1992–1999	1.88 to 34.69	0.03
						−5.28	1999–2010	−10.93 to 0.73	0.08
Lymphomas and reticuloendothelial neoplasms	270	8.79	8.79	15	12.79	−0.41	1992–2010	−2.34 to 1.56	0.66
CNS and miscellaneous intracranial and intraspinal neoplasms	615	19.95	19.95	35	29.54	0.34	1992–2010	−1.17 to 1.86	0.64
III(A) Ependymomas and choroid plexus tumor	50	1.56	1.56	5	2.38	−4.60	1992–2003	−11.72 to 3.08	0.21
						17.38	2003–2010	3.29 to 33.38	0.02
III(B) Astrocytomas	285	9.40	9.40	15	13.86	−3.74	1992–2010	−5.84 to −1.59	< 0.01
III(C) Intracranial and intraspinal embryonal tumors	100	3.29	3.29	5	4.91	4.01	1992–2010	1.83 to 6.24	< 0.01
III(D) Other gliomas	95	3.06	3.06	5	4.52	3.26	1992–2010	0.62 to 5.98	0.02
III(F) Unspecified intracranial and intraspinal neoplasms	75	2.41	2.41	5	3.54	15.32	1992–2005	6.45 to 24.93	< 0.01
						−36.13	2005–2010	−57.62 to −3.75	0.03
Neuroblastoma and other peripheral nervous cell tumors	195	6.41	6.41	10	9.97	2.50	1992–2010	−0.51 to 5.60	0.10
Retinoblastoma	80	2.64	2.64	5	4.15	3.98	1992–2010	0.91 to 7.15	0.01
Renal tumours	205	6.64	6.64	15	9.98	10.47	1992–1998	0.76 to 21.12	0.04
						−19.07	1998–2002	−38.41 to 6.33	0.12
						7.57	2002–2010	1.47 to 14.05	0.02

Continued on the following page

TABLE 5 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, females, Canada, 1992–2010

Ontario[b]									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
VI(A) Nephroblastoma and other nonepithelial renal tumors	180	5.92	5.92	10	8.91	12.18	1992–1998	0.83 to 24.81	0.04
						−20.40	1998–2002	−42.21 to 9.65	0.15
						8.81	2002–2010	1.69 to 16.43	0.02
Hepatic tumours	40	1.30	1.30	0	2.04	−1.61	1992–2010	−6.45 to 3.48	0.51
Malignant bone tumours	140	4.59	4.59	5	6.70	−1.37	1992–2010	−3.93 to 1.26	0.28
Soft tissue and other extraosseous sarcomas	170	5.63	5.63	10	8.43	1.43	1992–2010	−0.58 to 3.48	0.15
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	115	3.74	3.74	10	5.54	1.28	1992–2010	−1.79 to 4.45	0.40
X(B) Malignant extracranial and extragonadal germ cell tumors	35	1.11	1.11	0	1.74	4.76	1992–2010	0.38 to 9.34	0.03
Other malignant epithelial neoplasms and malignant melanomas	175	5.66	5.66	10	8.23	5.25	1992–2010	1.67 to 8.97	0.01
XI(B) Thyroid carcinomas	75	2.57	2.57	5	3.69	7.33	1992–2010	2.42 to 12.47	0.01
Other and unspecified malignant neoplasms	105	3.45	3.45	5	5.14	2.82	1992–2010	−1.04 to 6.83	0.14
Quebec									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	1910	97.40	100.00	100	159.41	0.12	1992–2010	−0.73 to 0.99	0.76
All childhood cancers including non-malignancies brain	1965	100.00	102.67	105	163.57	0.18	1992–2010	−0.68 to 1.04	0.67
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	600	30.43	31.24	30	50.25	1.06	1992–2010	−0.30 to 2.43	0.12
Lymphomas and reticuloendothelial neoplasms	145	7.34	7.54	5	11.64	0.39	1992–2010	−3.03 to 3.93	0.82
CNS and miscellaneous intracranial and intraspinal neoplasms	345	17.64	18.11	15	28.61	0.60	1992–2010	−1.48 to 2.73	0.55
III(C) Intracranial and intraspinal embryonal tumors	75	3.87	3.98	5	6.24	−4.69	1992–2010	−8.61 to −0.61	0.03
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	395	20.23	20.77	25	32.77	0.92	1992–2010	−1.54 to 3.44	0.44
III(C) Intracranial and intraspinal embryonal tumors including non-malignancies	75	3.87	3.98	0	6.24	−4.69	1992–2010	−8.61 to −0.61	0.03
Neuroblastoma and other peripheral nervous cell tumors	195	9.79	10.05	10	16.30	−16.35	1992–1997	−28.80 to −1.73	0.03
						2.01	1997–2010	−2.54 to 6.78	0.37
Retinoblastoma	55	2.75	2.83	0	4.68	1.03	1992–2010	−3.59 to 5.87	0.65
Renal tumours	135	6.98	7.17	10	11.71	−1.30	1992–2010	−3.55 to 1.00	0.25
Malignant bone tumours	90	4.49	4.60	5	7.02	−2.39	1992–2010	−5.30 to 0.60	0.11
Soft tissue and other extraosseous sarcomas	130	6.52	6.70	5	10.63	0.83	1992–2010	−1.51 to 3.24	0.47
Germ cell tumors, trophoblastic tumors, and neoplasms of gonads	65	3.52	3.61	5	5.69	−2.02	1992–2010	−6.55 to 2.73	0.37

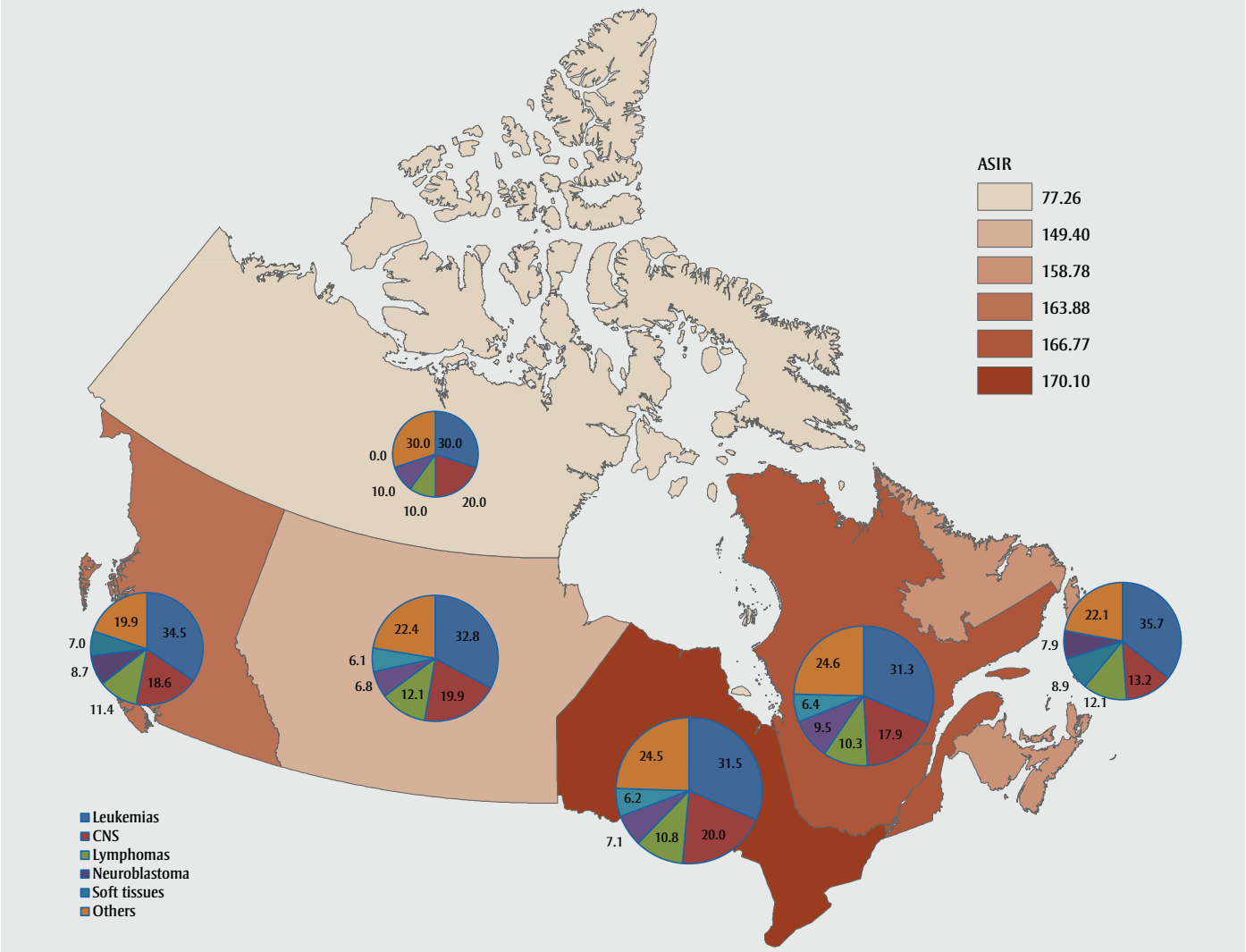
Continued on the following page

TABLE 5 (continued)
Annual percent changes (APC) of age standardized incidence rates (ASIRs)[a] (per million) of selected ICCC diagnosis categories by geographic region, females, Canada, 1992–2010

Quebec									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
Other malignant epithelial neoplasms and malignant melanomas	110	5.76	5.91	5	9.22	3.63	1992–2010	0.25 to 7.12	0.04
XI(B) Thyroid carcinomas	50	2.70	2.77	5	4.32	8.43	1992–2010	3.96 to 13.09	< 0.01
Other and unspecified malignant neoplasms	30	1.33	1.36	0	2.19	26.90	1992–1996	−4.07 to 67.87	0.09
						−66.08	1996–1999	–	0.98
						88.43	1999–2003	–	0.39
						−13.52	2003–2010	−29.24 to 5.70	0.13
Atlantic provinces									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	565	96.57	100.00	30	149.37	0.42	1992–2010	−1.69 to 2.58	0.68
All childhood cancers including non-malignancies brain	585	100.00	103.55	30	154.41	0.35	1992–2010	−1.67 to 2.41	0.72
Leukemias, myeloproliferative diseases, and myelodysplastic diseases	195	33.79	34.99	15	52.90	0.30	1992–2010	−2.58 to 3.27	0.83
I(B) Acute myeloid leukemias	35	6.52	6.75	0	10.43	14.42	1992–2003	4.23 to 25.60	0.01
						−13.02	2003–2010	−24.21 to −0.17	0.05
Lymphomas and reticuloendothelial neoplasms	40	6.86	7.10	5	10.02	−0.94	1992–2010	−4.82 to 3.09	0.62
CNS and miscellaneous intracranial and intraspinal neoplasms	115	19.21	19.89	10	29.00	−0.32	1992–2010	−4.18 to 3.69	0.87
CNS and miscellaneous intracranial and intraspinal neoplasms including non-malignancies	130	22.64	23.45	5	34.04	−0.49	1992–2010	−4.13 to 3.29	0.79
Neuroblastoma and other peripheral nervous cell tumors	35	5.49	5.68	5	9.25	−38.68	1992–1996	−57.57 to −11.37	0.01
						71.74	1996–1999	–	0.46
						2.00	1999–2010	−5.28 to 9.83	0.57
Renal tumours	35	6.17	6.39	0	10.08	−0.16	1992–2010	−3.71 to 3.51	0.92
Soft tissue and other extraosseous sarcomas	45	7.55	7.82	0	11.35	2.60	1992–2010	−2.42 to 7.88	0.29
Other malignant epithelial neoplasms and malignant melanomas	30	5.15	5.33	5	7.50	4.07	1992–2010	−0.19 to 8.52	0.06
Territories									
	Total cases	% (including non-malignancies)	% (malignancies only)	Average annual cases	Average ASIR	APC	Year	95% CI	*p*-value
All childhood cancers (malignancies only)	30	96.77	100.00	0	117.51	−1.43	1992–2010	−6.31 to 3.72	0.56
All childhood cancers including non-malignant CNS tumors	35	100.00	103.33	5	121.20	−1.33	1992–2010	−6.34 to 3.96	0.60

Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
[a] The ASIRs were standardized to the 2011 Canadian population.
[b] There were no non-malignant cases in Ontario.

FIGURE 2
Average annual age-standardized incidence rates (ASIRs) (per million) of all cancers combined and most common cancers (%) by region, age < 15, Canada, 2006-2010



Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
Notes: 1. The pie charts represent the percentage distribution of new cancer cases in each region.
2. The ASIRs were standardized to the 2011 Canadian population.

cases. Hepatoblastoma constituted four-fifths (81.3%) of all hepatic cancer cases in males; rates of hepatoblastoma increased by 3.2% per year (CI = 0.6–5.9), and drove the increase of 2.2% per year for hepatic cancers overall (CI = 0.01–4.4).

While incidence rates for CNS tumors have remained stable, some of its divisions showed significant changes. Notably, ependymomas increased among females (APC = 3.0%, CI = 0.6–5.4), echoing the rate transition of this disease overall. Incidence of carcinoma among females increased (APC = 2.9%, CI = 0.6–5.4), as did its subgroup of thyroid cancer (APC = 4.9%, CI = 1.8–8.0). For malignant gonadal germ cell tumors, the rate in males decreased (APC = −4.0%, CI = −6.7 to −1.2), with a non-significant less rapid decline in rates noted in females (APC = −1.4%, CI = −4.7 to 2.1).

Trends by age group

The overall increasing trend for all cancers combined was suggested among children aged 1–4 years (APC = 0.9%, CI = 0.4–1.3), whereas the rates appeared stable in other age groups (Table 2). Specifically, the incidence rate of lymphoid leukemias increased among children aged 1–4 years (APC = 0.9%, CI = 0.1–1.8).

Astrocytoma formed the largest subgroup of all CNS tumors, constituting more than two-fifths (45.0%) of the total. The incidence proportion of astrocytoma increased with age, from 32.4% in infants to 52.4% in late childhood. The rates of astrocytoma decreased by 2.1% annually among children aged 10–14 years (CI = −3.7 to −0.5) and appeared stable in the under-tens over the entire study period. In line with the trend observed overall and in females, the rates of ependymomas increased in infants and late childhood (APC = 5.6%, CI = 1.9–9.4 and APC = 5.1%, CI = 1.5–8.9, respectively), although the rates were based on small numbers of cases.

Several types of embryonal tumors demonstrate age difference in incidence trends.

FIGURE 3
Age-standardized incidence rates (ASIRs) for all cancers combined and top five most common cancers in children under 15 years of age, Canada, 1992-2010

Data sources: Canadian Cancer Registry (CCR) database at Statistics Canada and Quebec Cancer Registry (2008-2010).
Abbreviations: ASIR, age-standardized incidence rate; CNS, central nervous system.
Note: The ASIRs were standardized to the 2011 Canadian population.

The rates increased by 1.6% per year (CI = 0.2–3.1) for neuroblastoma overall and equally for neuroblastoma and ganglioneuroblastoma (IV(A)) for children ages 1–4 years. Hepatoblastoma comprised nearly all hepatic cancer cases in children under 5 years of age. In children aged 1–4 years, rates of hepatoblastoma increased by 3.7% per year (CI = 1.1–6.4).

Trends by geographic area

Trends by geographic area are presented for both sexes combined (Table 3) and individually (Table 4 and 5). The rates of all cancers combined increased the most in Ontario from 2006 (APC = 5.9%, CI = 1.9–10.1) after a preceding stable period, and increased non-significantly in the other regions from 1992 to 2010. Positive trends in Ontario were noted for both sexes: while the trend among females was very similar to those observed overall, increases in trends in males occurred between 1992 and 2002 (APC = 1.6%, CI = 0.5–2.7), and more rapidly between 2005 and 2010 (APC = 5.0%, CI = 1.9–8.2).

Some lymphohematopoietic malignancies demonstrated increasing trends in Ontario and the Prairies: lymphoid leukemias among males (APC = 1.4%, CI = 0.3–2.5) and among all children (APC = 1.3%, CI = 0.2–2.4), and unspecified lymphomas (APC = 4.3%, CI = 1.3–7.5) in Ontario; as well as lymphomas in females (APC = 3.5%, CI = 0.3–6.8), and non-Hodgkin lymphomas (except Burkitt lymphoma) in males and females combined (APC = 4.8%, CI = 1.6–8.1) and separately (males: APC = 3.8%, CI = 0.3–7.5; females: APC = 6.0%, CI = 1.6–10.6) in the Prairies. Two joinpoints suggest shifts in the direction of the trend for a subgroup of lymphoid leukemia, precursor cell lymphoblastic leukemia in Ontario for both sexes individually and combined: an early non-significant rise and a recent significant more rapid increase since 2004.

Amphi-directional incidence trends of CNS tumors were noted in some regions. Rates of CNS tumors in Ontario decreased non-significantly by 1.4% per year from 1992 to 2004 (CI = −2.8 to 0.1), and subsequently increased significantly by 5.0% per year from 2004 to 2010 (CI = 1.0–9.2). In comparison, the rates in the Atlantic region displayed a reverse trend. The ASIRs of CNS tumors in the Atlantic region were the highest in the country during 2002–2004 and then dropped to the lowest in 2005, and 2007–2010 (data not shown). Incidence of astrocytoma in Ontario decreased consistently over the study horizon in males (APC = −2.4%, CI = −4.6 to −0.2) and females (APC = −3.7%, CI = −5.8 to −1.6), while increases were observed for ependymomas (APC = 3.3%, CI = 0.7–6.1), intracranial and intraspinal embryonal tumors among females (APC = 4.0%, CI = 1.8–6.2), and other gliomas in males and females combined (APC = 4.5%, CI = 2.3–6.7) and separately (males: APC = 4.7%, CI = 1.0–8.5; females: APC = 3.3%, CI = 0.6–6.0).

Significant changes were also observed for other embryonal tumors in central Canada. Neuroblastoma in females in Quebec decreased significantly by 16.4% per year from 1992 to 1997, but increased non-significantly by 2% thereafter. For neuroblastoma in males in Quebec, a joinpoint was not suggested for the best fitted model, but an one-joinpoint model showed a similar but non-significant trend as that in females: the rates dropped by 7.0% (CI = −22.6 to 11.7) per year during 1992–1997, and then rose by 2.6% (CI = −2.1 to 7.5) (data not shown). Retinoblastoma increased by 4% annually (CI = 0.9–7.2) over the entire period in females in Ontario. Two breaks in trend show that there have been early (in the 1990s) and recent (since 2002), significant increases in the incidence of nephroblastomas in females in Ontario, and a corresponding trend was evident in renal tumors as a whole. There is a suggestion, however, that renal tumors among males decreased by 3.3% per year (CI = −6.4 to −0.1) in the Prairies.

The increases were similar for carcinoma in Ontario and Quebec, mainly driven by the increases in thyroid cancers more specifically among females. Bone cancer in Quebec decreased by 4.9% (CI = −8.5 to −1.2) per year from 1992 to 2002 for males and females combined and increased by 6.2% (CI = 0.4–12.3) thereafter.

Discussion

Our study found that the incidence rates of childhood cancer increased by an average of 0.4% per year from 1992 to 2010. Similar increases have been documented in the United States,[5] Australia,[6] in European countries,[7] in Asian nations,[8] and internationally.[16] A study using data from the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program indicated that the overall cancer incidence rates increased non-significantly by 0.4% per year between 1992 and 2004 in the US,[5] consistent with our change magnitude. The non-significant increase was updated to continue (APC = 0.3%, CI = −0.1 to 0.7) during 2001–2009 based on data which provided greater population coverage.[17] Considering the findings on all cancers combined from Ellison et al. who examined the top 5 most common cancers at the national level over the same time frame,[4] our study had comparable results for males and both sexes combined; however, it also revealed a recent substantial increase among females.

For the period from 2001 to 2010, our study showed an annual increase in overall rates of 1.5% (CI = 0.6–2.4), driven mainly by the increase in cancer rates in females (APC = 2.5%, CI = 1.2–3.8) (data not shown). The overall trend in females is due in large part to the rate increases in leukemias (APC = 2.3%, CI = 0.5–4.2), lymphomas (APC = 1.8%, CI = −1.9 to 5.6), neuroblastoma (APC = 3.7%, CI = −0.8 to 8.5), soft tissue sarcoma (APC = 3.9%, CI = −0.8 to 8.8), and most pronouncedly in thyroid cancer (APC = 10.4%, CI = 3.4–17.8) (data not shown). Regarding an earlier period (1985–1992), Health Canada reported that the incidence rates for all cancers combined in children and teenagers aged under 20 tended to increase slightly.[18]

Broad similarities in the increase of ASIRs for some cancers raise questions as to the potential for common etiologies, given the etiology of pediatric cancer is largely unknown. Several hypotheses have been put forward to explain the trends. The changes may partially be artefacts of changes in classification, increased use of advanced diagnostic technology, and improved cancer reporting. The overall increases were confined to 1992–1999 and 2003–2010, mirrored trends in leukemias, lymphomas, soft tissue sarcoma, and CNS tumors (data not shown). The increases which occurred in 1992–1999 coincided with the introduction of ICCC in 1996 and the increased use of magnetic resonance imaging (MRI) during 1990–2001, whereas the increase which occurred in 2003–2010 coincided with the introduction of ICD-O-3 in 2001 and the increased use of molecular tests to supplement pathological diagnosis in an attempt to improve the precision and objectivity of the histopathological diagnosis. The incidence trends in

children have also been associated with changes in environmental exposures or gene-environment interactions, parental lifestyle, changes in birth weight, or changes in social structures.[7]

The observed increased incidence trends could, in part, be explained as an artefact of increases in survival. Prognosis has been improving in the last three decades as a result of more accurate diagnoses and improved treatment strategies. Research has shown that risk for subsequent malignant neoplasms is higher for childhood cancer survivors than is the risk for cancer in people of the same age in the general population.[19] Our data show that the percentage of second or third cancers increased from 0.7% in 1992 to 4.1% in 2006 (with an interruption in 2004), and then dropped sharply in males; as it did in females but with a smaller increase (data not shown). These increases of subsequent malignant neoplasms in Canada coincide with the magnitude and significance of the increases in the overall incidence trends.

Risk of pediatric cancer has been linked to maternal age at birth. A large US case–control study reported an increase of 8% in overall childhood cancer risk for each quinquennial increase in maternal age, with similar increases for most of the frequent cancers.[20] Maternal age could also be a marker for unknown environmental exposures which may have changed over time.[6] As in most developed countries, the average maternal ages at both first and all childbirths have risen since the mid-1970s in Canada.[21] During our study period, the average age at all childbirths increased from 27.9 years in 1992 to 30.1 years in 2010.[21] The rise of maternal age might have contributed to the incidence increase, but the extent to which this might occur is unknown.

Childhood cancer is characterized by heterogeneity, different cancers likely have different etiologies. To follow up our findings, it would be useful to identify the tumour types and population groups that were specifically affected by these trends. The strongest increase of ASIRs for all cancers combined is seen in children aged 1–4 years. The rise is driven, in large part, by an increase in leukemia, which is the most common cancer (accounting for a third of all cancers) in children. Ontario experienced the most pronounced increase from 2006 to 2010 for all cancers combined, and for leukemia, subgroups of lymphomas, CNS tumors, embryonal tumors, carcinoma and thyroid cancer. While demographic and/or etiologic differences could potentially exist between the geographic regions, the variation in cancer registry practices could also explain the geographical differences in cancer incidence.

Leukemia overall and lymphoid leukemia specifically had an equally significant increase. The incidence rate of lymphoid leukemia also increased significantly in those aged 1–4 years. Similar increases in leukemia have been reported in other developed countries.[5,6,22] Previous studies have shown that ionizing radiation, certain genetic disorders, high birth weight, cytotoxic alkylating agents, parental age, parental smoking, prenatal and postnatal pesticide exposures, residential traffic-related air pollution and prenatal exposure to infectious agents such as John Cunningham virus have been associated with leukemia in children.[23-27] Fetuses and young children might be more susceptible to the exposures because of their underdeveloped detoxification mechanisms or higher intake rates relative to their body weight compared with older children. There is considerable evidence of a positive association between improving socioeconomic status and a peak incidence of precursor B-cell acute lymphoblastic leukemia (ALL) in children aged 2–3.[28] It has also been suggested that aberrant immune response to delayed infection by unknown agents may play a role in conversion of preleukemic clones into overt precursor B-cell ALL.[23] Precursor cell lymphoblastic leukemia increased non-significantly by 0.4% per year (CI = −0.6 to 1.5) among Canadian children aged 1–4 years from 1992 to 2010 (Table 2), whereas a significant increase of the disease in Ontario was confined to 2004–2010 (Table 3). A Canadian spatial study found that areas with a higher proportion of immigrants had higher childhood leukemia incidence rates.[29] The proportion of immigrants in Canada steadily increased from 16.1% of the total population in 1991 to 18.4% in 2001 and 20.6% in 2011.[30] The percentage of immigrants who settled in Ontario was over 50% from 1992 to 2006,[31] with the proportion of immigrants increasing from 25.6% of the total provincial population in 1996, to 26.8% in 2001 and 28.3% in 2006.[32] The increased immigrant population may play a role in the observed increases in cancer incidence. However this association is from a single study.

The stable rate of CNS tumors was also observed in the US for similar reporting periods (1992–2004[5] and 1987–2009[33]). The increase of CNS tumors in the US confined to 2000–2010 is comparable to the Ontario trend.[22] Also, a significant change in rate was found for non-malignant brain tumors in the US population. It has been suggested that the increase is likely attributable to changes in the detection and reporting of these diseases.[34] The recent increase of CNS tumors in Ontario may reflect the increased use of molecular markers to supplement pathological diagnosis.

The International Agency for Research on Cancer (IARC) stated that X-radiation and gamma-radiation, forms of ionizing radiation, are the only established risk factors for CNS cancers.[35] IARC also groups radiofrequency non-ionizing radiation from telecommunications as a possible cause of CNS malignancies, with limited evidence.[35,36] Genetic and hereditary conditions are associated with an increased risk. Changes in environmental and medical exposures or gene-environment interactions, such as ionizing radiation and pesticides have been linked to the recent increases in incidence of CNS tumors.[37] A Canadian study found a positive association between astrocytoma and maternal exposure to residential air pollution.[24]

Our study shows that incidence of hepatoblastoma has risen 2.4% per year between 1992 and 2010. An annual increase of 4% was observed in the US between 1992 and 2004.[5] Although few causes of hepatoblastoma have been established, several clues have emerged. Studies[38-40] have found a strong association between hepatoblastoma and very low birth weight (VLBW) (< 1500 g), suggesting an iatrogenic etiology. Risk of hepatoblastoma was elevated 20-fold in Children with VLBW, and doubled in children with moderately low birth weight (1500–2500 g).[38] It has been previously noted that the rise in hepatoblastoma corresponds to the increase in the frequency of low or very low weight births in the US.[41] The Public Health Agency of Canada reported that the low birth weight rate generally increased from 2001 to 2010 in Canada.[42] Furthermore, the survival rate of low birth weight babies in Canada has increased with improved neonatal care. These together may, in part, account for the increased trend in hepatoblastoma in this study.

As presented in our data, neuroblastoma is the most common pediatric cancer diagnosed in infants,[43] accounting for 26.4% of all diagnoses in Canada. It is the third most frequent cancer in children 1–4 year olds, accounting for 10.5% of all cases (Figure 1). The incidence of neuroblastoma increased significantly in children 1–4 year olds during 1992–2010, similar to patterns observed in Europe.[43] Increased use of advanced diagnostic techniques, detecting latent or asymptomatic tumours, may have contributed to the observed increase in incidence.[44] The large declines in neuroblastoma in Quebec noted in the 1992–1997 period reflects the ending of a large screening trial in 1994 which resulted in the identification of many cases of neuroblastoma which may otherwise never have been clinically detected.[45]

The rapid increase of pediatric thyroid cancer was confirmed by other studies.[17,46] Siegel et al. reported that thyroid cancer incidence rates increased by 4.9% per year (CI = 3.2–6.6) among US children and adolescents (less than 20 years of age) during 2001–2009.[17] Previous studies have also revealed increased rates of thyroid cancers among adults in Canada and other countries.[1,47,48] It is unknown if causes for the increase in thyroid carcinomas in children are the same as those in adults. Increased use of advanced diagnostic technologies has contributed to the detection of small, subclinical thyroid tumors.[49] More frequent use of imaging to diagnose benign thyroid diseases, which are more common in females than males, may explain the more increase of thyroid cancer in females.[49] On the other hand, it has been shown that exposure to radiation by increased use of CT scans[50] may increase risk of thyroid cancer.[51,52] There is also evidence of a positive association between obesity and adult thyroid cancer risk.[53-54] The increased obesity prevalence among the pediatric population[55-57] may be responsible for some of the increases in thyroid cancer.

The annual significant decrease of 2.1% in astrocytoma incidence among children aged 10–14 years is similar to the non-significant decrease (APC = −1.9, CI = −4.4 to 0.8) in the same age group between 1992 and 2004 observed in the US.[5] The decrease of astrocytoma could be partially explained by improvements in diagnosis and classification with implementation of the ICD-O-3 in 2001. As per ICD-O-3, pilocytic astrocytomas are coded as uncertain/borderline tumors (morphological code 9421/1), and thus, were excluded from analysis of the malignant cases. In addition, the decrease of astrocytomas not otherwise specified (NOS) suggests improvements in precise diagnostic classification of CNS tumors.[33] Declining incidence trends for malignant gonadal germ cell tumors accords with the reduction in prevalence of congenital anomalies.[20,58]

Strengths and limitations

Our findings should be interpreted in the context of study limitations and strengths. Although the provincial and territorial cancer registries strive to find and define new cancer cases according to the national standard, reporting procedures and completeness remain inconsistent across the registries.[1] The incidence of some cancers in Quebec, particularly for those that rely more heavily on pathological diagnosis, are underestimated as a result of the registry's dependence on hospitalization data during the study period. Although all provincial and territorial cancer registries now record cancers according to the SEER rules for multiple primaries, not all registries were able to report according to the new requirements beginning in 2007.[9]

Cancer incidence may be under-reported in some provinces due to missing information on "death certificate only" (DCO) cases or incomplete linkage of cancer data with vital statistics information for the data used in this study. The number of DCO cases from 2008 to 2010 in Newfoundland and Labrador (NL) was estimated based on 2007 data. NL has recently implemented death clearance processes to improve case ascertainment and have also improved the case reporting from areas that previously under-registered cases. In Quebec, DCO cases were incompletely recorded before 2000. The number of DCO cases for 2010 in Quebec was calculated as the average of 2005 to 2009 data. Ontario did not report DCO cases for 2008 to 2010. Their number of DCO cases for these three years was estimated by averaging the DCO cases in 2003 to 2007. The number of DCO cases is below 2% of total new cases.

Non-malignant brain tumors are not routinely captured or reported to CCR, and these cases in CCR are underreported based on our analysis (data not shown). Inclusion of benign brain tumors in the analysis could result in an artefact when comparing incidence across time and geographic area, given the incompleteness of the data collection. For example, the analysis based on the dataset comprising non-malignant along with malignant CNS tumors did not detect the statistically significant break in the ASIR trend for all cancers combined in females. Another example is that the addition of a preponderance of non-malignant cases (86%) to the total of other specified intracranial and intraspinal neoplasms (III(E)) resulted in a significant joinpoint trend in the 5–9 year age group (Table 2).

A Type I error may have biased the results for the diagnostic groups with only a small number of cases. Multiple tests were performed with adjustment to control the overall over-fitting error probability of 0.05; because of small numbers, random fluctuations in rates may erroneously show as significant certain trends. Therefore, trends involving a small number of cases and those with wide confidence intervals should be interpreted critically. For example, the increase of non-Hodgkin lymphomas (except Burkitt lymphoma) among females in the Prairies involved a small number of cases (45) between 1992 and 2010. Some significant findings show significance that is close to the cut-off of 0.05, e.g. decreasing malignant gonadal germ cell tumors, and increasing hepatic cancers in males. These trends should be further validated.

The increases of all cancers and selected malignancies varied in magnitude and significance among regions. The statistical significance achieved in Ontario may be a reflection of the size of its population.

Differences in trends by tumor type, sex, age, and region were described in this study but the relationships among the trends were not tested statistically. The results therefore may include spurious associations.

The principal strength of CCR is the complete population coverage and high data quality. Our analysis provides current trends in childhood cancer incidence, and to our knowledge represents the first report for the detailed diagnostic groups in demographic and geographic context.

Conclusion

In summary, overall incidence rates of childhood cancer have slowly increased

since 1992. Statistically significant increases were observed in several malignancies such as leukemia, unspecified lymphoma, ependymoma, hepatoblastoma, thyroid and melanoma. The differences in the temporal trends were also registered by sex, age, and geographic area. The rates for all cancers combined increased the most in Ontario, and increased non-significantly in the other regions from 1992 to 2010. Another new finding is that astrocytoma incidence decreased significantly among children aged 10–14 years. Given the limited understanding of pediatric cancer etiology, this study underscores the value of surveillance in creating opportunities to seek insights into the factors driving incidence trends. This knowledge may ultimately help inform public health policy and programs.

Acknowledgements

We acknowledge the collaboration between the provincial and territorial cancer registries and the Health Statistics Division of Statistics Canada for providing the Canadian Cancer Registry data. We thank the Quebec Cancer Registry for providing their aggregate data for 2008 to 2010. We thank Mr. Robert Semenciw, formerly from the Public Health Agency of Canada (PHAC), for providing expert advice on cancer surveillance, and reviewing the manuscript. We thank Dr. Dianne Zakaria of PHAC for discussions on analysis method, and Dr. Shiliang Liu for discussions on risk factors. We also thank Ms. Judy Snider for reviewing the manuscript.

Conflicts of interest

The authors declare no conflicts of interest.

Author contributions and statement

All authors contributed to study design, interpretation of the data, and drafting and/or revising the paper. LX performed the analysis.

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada.

References

1. Canadian Cancer Society's Advisory Committee on Cancer Statistics. Canadian Cancer Statistics 2015. Toronto, ON: Canadian Cancer Society; 2015.

2. Statistics Canada. Leading causes of death, total population, by age group and sex, Canada. Ottawa (ON): Statistics Canada; 2017 [CANSIM Table 102-0561]. Available from: http://www5.statcan.gc.ca/cansim/a26?lang=eng&retrLang=eng&id=1020561&tabMode=dataTable&p1=-1&p2=9&srchLan=-1

3. Canadian Cancer Society/National Cancer Institute of Canada. Canadian cancer statistics 2008. Toronto, Canada, 2008.

4. Ellison L, Janz T. Childhood cancer incidence and mortality in Canada. Health at a Glance. Ottawa (ON): Statistics Canada; 2015 [Statistics Canada, Catalogue no.82-624-X ISSN 1925-6493]. Available from: http://www.statcan.gc.ca/pub/82-624-x/2015001/article/14213-eng.pdf

5. Linabery AM, Ross JA. Trends in childhood cancer incidence in the U.S. (1992-2004). Cancer. 2008;112(2):416-32. doi: 10.1002/cncr.23169.

6. Baade PD, Youlden DR, Valery PC, et al. Trends in incidence of childhood cancer in Australia, 1983-2006. Br J Cancer. 2010;102(3):620-6. doi: 10.1038/sj.bjc.6605503.

7. Kaatsch P. Epidemiology of childhood cancer. Cancer Treat Rev. 2010;36(4):277-85. doi: 10.1016/j.ctrv.2010.02.003.

8. Liu YL, Lo WC, Chiang CJ, et al. Incidence of cancer in children aged 0-14 years in Taiwan, 1996-2010. Cancer Epidemiol. 2015;39(1):21-8. doi: 10.1016/j.canep.2014.11.010.

9. Statistics Canada. Canadian Cancer Registry (CCR) [Internet]. Ottawa (ON): Statistics Canada. Available at: http://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&SDDS=3207

10. Fritz A, Percy C, Jack A, et al. International Classification of Diseases for Oncology. Third Edition, First Revision. Geneva, Switzerland: World Health Organization; 2013. Available at: http://apps.who.int/iris/bitstream/10665/96612/1/9789241548496_eng.pdf

11. Steliarova-Foucher E, Stiller C, Lacour B, et al. International Classification of Childhood Cancer, third edition. Cancer. 2005;103(7):1457-67. doi: 10.1002/cncr.20910.

12. National Cancer Institute's Surveillance, Epidemiology, and End Results Program. Main Classification Table from the ICCC-3 based on ICD-O-3. Available at: https://seer.cancer.gov/iccc/iccc3.html

13. Statistics Canada. Annual Demographic Estimates: Canada, Provinces and Territories, 2015. Ottawa (ON): Statistics Canada; 2015 [Catalogue no. 91-215-X]. CANSIM Table 051-0001 released in September 2015. Available at: http://www.statcan.gc.ca/pub/91-215-x/91-215-x2015000-eng.pdf

14. Joinpoint Regression Program, Version 4.3.1.0 - April 2016; Statistical Methodology and Applications Branch, Surveillance Research Program, National Cancer Institute, Bethesda, MD, USA.

15. Kim HJ, Fay MP, Feuer EJ, et al. Permutation tests for joinpoint regression with applications to cancer rates. Stat Med. 2000;19(3):335-51. Erratum in: Stat Med 2001;20(4):655.

16. Steliarova-Foucher E, Colombet M, Ries LAG, et al. International incidence of childhood cancer, 2001–10: a population-based registry study. Lancet Oncol. 2017;18(6):719-31. doi: 10.1016/S1470-2045(17)30186-9.

17. Siegel DA, King J, Tai E, et al. Cancer incidence rates and trends among children and adolescents in the United States, 2001-2009. Pediatrics. 2014;134(4):e945-55. doi: 10.1542/peds.2013-3926.

18. Huchcroft S, Clarke A, Y Mao, et al. This Battle Which I Must Fight. Cancer in Canada's Children and Teenagers. Ottawa: Supply and Services Canada, 1996. ISBN 0-662-24310-2.

19. Neglia JP, Friedman DL, Yasui Y, et al. Second malignant neoplasms in five-year survivors of childhood cancer: childhood cancer survivor study. J Natl Cancer Inst. 2001;93(8):618-29. doi: 10.1093/jnci/93.8.618.

20. Johnson KJ, Carozza SE, Chow EJ, et al. Parental age and risk of childhood cancer: a pooled analysis. Epidemiology. 2009;20(4):475-83. doi: 10.1097/EDE.0b013e3181a5a332.

21. Statistics Canada. Fertility: Fewer children, older moms. Date modified: 2016-09-28. Available at: http://www.statcan.gc.ca/pub/11-630-x/11-630-x2014002-eng.htm

22. Gittleman HR, Ostrom QT, Rouse CD, et al. Trends in central nervous system tumor incidence relative to other common cancers in adults, adolescents, and children in the United States, 2000 to 2010. Cancer. 2015; 121(1):102-12. doi: 10.1002/cncr.29015.

23. Eden T. Aetiology of childhood leukaemia. Cancer Treat Rev. 2010;36(4): 286-97. doi: 10.1016/j.ctrv.2010.02.004.

24. Lavige É, Bélair MA, Do MT, et al. Maternal exposure to ambient air pollution and risk of early childhood cancers: A population-based study in Ontario, Canada. Environ Int. 2017; 100:139-147. doi: 10.1016/j.envint.2017.01.004.

25. Carlos-Wallace FM, Zhang L, Smith MT, et al. Parental, In Utero, and Early-Life Exposure to Benzene and the Risk of Childhood Leukemia: A Meta-Analysis. Am J Epidemiol. 2016; 183(1):1-14. doi: 10.1093/aje/kwv120.

26. Chen M, Chang CH, Tao L, et al. Residential Exposure to Pesticide During Childhood and Childhood Cancers: A Meta-Analysis. Pediatrics. 2015;136(4):719-29. doi: 10.1542/peds.2015-0006.

27. Ross JA, Swensen AR. Prenatal epidemiology of pediatric tumors. Curr Oncol Rep. 2000;2(3):234-41.

28. Linet MS, Brown LM, Mbulaiteye SM, et al. International long-term trends and recent patterns in the incidence of leukemias and lymphomas among children and adolescents ages 0-19 years. Int J Cancer. 2016;138(8):1862-74. doi: 10.1002/ijc.29924.

29. Torabi M, Singh H, Galloway K, et al. Geographical variation in the incidence of childhood leukaemia in Manitoba. J Paediatr Child Health. 2015;51(11):1121-6. doi: 10.1111/jpc.12930.

30. Statistics Canada. 150 years of immigration in Canada. Ottawa (ON): Statistics Canada; 2017. Available at: https://www.statcan.gc.ca/pub/11-630-x/11-630-x2016006-eng.htm

31. Jonathan Chagnon. Migration: International, 2010 and 2011. Ottawa (ON): Statistics Canada;2013 [Catalogue no. 91-209-X]. Available at: http://www.statcan.gc.ca/pub/91-209-x/2013001/article/11787-eng.pdf

32. Statistics Canada. Census Trends: Geography selection for Canada, provinces and territories. Ottawa (ON): Statistics Canada; 2007. Available at: http://www12.statcan.gc.ca/census-recensement/2006/dp-pd/92-596/P1-2.cfm?Lang = eng&T = PR&PRCODE = 35&GEOCODE = 35&GEOLVL = PR&TID = 800

33. McKean-Cowdin R, Razavi P, Barrington-Trimis J, et al. Trends in childhood brain tumor incidence, 1973-2009. J Neurooncol. 2013;115(2): 153-60. doi: 10.1007/s11060-013-1212-5.

34. Papathoma P, Thomopoulos TP, Karalexi MA, et al. Childhood central nervous system tumours: Incidence and time trends in 13 Southern and Eastern European cancer registries. Eur J Cancer. 2015;51(11):1444-55. doi: 10.1016/j.ejca.2015.04.014.

35. International Agency for Research on Cancer. Agents classified by the IARC monographs, Volumes 1-109 IARC Monographs on the evaluation of carcinogenic risks to humans [Internet]. Lyon (FR): International Agency for Research on Cancer; 2013. Available at: http://monographs.iarc.fr/ENG/Classification/index.php

36. Boyle P, Levin B, editors. World Cancer report 2008 [Internet]. Lyon (FR): International Agency for Research on Cancer; 2008. Available at: http://www.iarc.fr/en/publications/pdfs-online/wcr/2008/wcr_2008.pdf

37. Kaatsch P, Steliarova-Foucher E, Crocetti E, et al. Time trends of cancer incidence in European children (1978-1997): report from the Automated Childhood Cancer Information System project. Eur J Cancer. 2006;42(13): 1961-71. doi: 10.1016/j.ejca.2006.05.014.

38. Spector LG, Birch J. The epidemiology of hepatoblastoma. Pediatr Blood Cancer. 2012;59(5):776-9. doi: 10.1002/pbc.24215.

39. McLaughlin CC, Baptiste MS, Schymura MJ, et al. Maternal and infant birth characteristics and hepatoblastoma. Am J Epidemiol. 2006;163(9):818-28. doi: 10.1093/aje/kwj104.

40. Ansell P, Mitchell CD, Roman E, et al. Relationships between perinatal and maternal characteristics and hepatoblastoma: a report from the UKCCS. Eur J Cancer. 2005;41(5):741-8. doi: 10.1016/j.ejca.2004.10.024.

41. Spector LG, Feusner JH, Ross JA. Hepatoblastoma and low birth weight. Pediatr Blood Cancer. 2004; 43(6):706. doi: 10.1002/pbc.20122.

42. Public Health Agency of Canada. Perinatal Health Indicators for Canada 2013: a Report of the Canadian Perinatal Surveillance System. Ottawa (ON): Public Health Agency of Canada;2013. Available at: http://publications.gc.ca/collections/collection_2014/aspc-phac/HP7-1-2013-eng.pdf

43. Larrañaga N, Sanchez MJ, Ardanaz E, et al. Incidence Patterns and Trends of non-Central Nervous System Solid Tumours in Children and Adolescents. A Collaborative Study of the Spanish Population Based Cancer Registries. J Cancer. 2016;7(3):335-43. doi: 10.7150/jca.12943.

44. Park JR, Eggert A, Caron H. Neuroblastoma: biology, prognosis, and treatment. Hematol Oncol Clin North Am. 2010;24(1):65-86. doi: 10.1016/j.hoc.2009.11.011.

45. Woods WG, Gao Rn, Shuster JJ, et al. Screening of infants and mortality due to neuroblastoma. N Engl J Med. 2002;346(14):1041-6. doi: 10.1056/NEJMoa012387.

46. Holmes L Jr, Hossain J, Opara F. Pediatric thyroid carcinoma incidence and temporal trends in the USA (1973-2007): race or shifting diagnostic paradigm? ISRN Oncol. 2012; 2012:906197. doi: 10.5402/2012/906197.

47. Xie L, Semenciw R, Mery L. Cancer incidence in Canada: trends and projections (1983-2032). Health Promot Chronic Dis Prev Can. 2015;35 Suppl 1:2-186. doi: 10.24095/hpcdp.35.S1.02.

48. Siegel R, Ma J, Zou Z, et al. Cancer statistics, 2014. CA Cancer J Clin. 2014;64(1):9-29. doi: 10.3322/caac.21208.

49. Kent WD, Hall SF, Isotalo PA, et al. Increased incidence of differentiated thyroid carcinoma and detection of subclinical disease. CMAJ. 2007;177(11): 1357-61. doi: 10.1503/cmaj.061730.

50. Linet MS, Kim KP, Rajaraman P. Children's exposure to diagnostic medical radiation and cancer risk: epidemiologic and dosimetric considerations. Pediatr Radiol. 2009;39 Suppl 1:S4-26. doi: 10.1007/s00247-008-1026-3.

51. Hammer GP, Seidenbusch MC, Regulla DF, et al. Childhood cancer risk from conventional radiographic examinations for selected referral criteria: results from a large cohort study. AJR Am J Roentgenol. 2011;197(1):217-23. doi: 10.2214/AJR.10.4979.

52. Schonfeld SJ, Lee C, Berrington de González A. Medical exposure to radiation and thyroid cancer. Clin Oncol (R Coll Radiol). 2011;23(4):244-50. doi: 10.1016/j.clon.2011.01.159.

53. Peterson E, De P, Nuttall R. BMI, diet and female reproductive factors as risks for thyroid cancer: a systematic review. PLoS One. 2012;7(1):e29177. doi: 10.1371/journal.pone.0029177.

54. Meinhold CL, Ron E, Schonfeld SJ, et al. Nonradiation risk factors for thyroid cancer in the US Radiologic Technologists Study. Am J Epidemiol. 2010;171(2):242-52. doi: 10.1093/aje/kwp354.

55. Statistics Canada. Health trends 2013 [Internet]. Ottawa (ON): Statistics Canada; 2013. [Statistics Canada, Catalogue No. 82-213-XWE]. Available at: http://www12.statcan.gc.ca/health-sante/82-213/index.cfm?Lang=eng

56. Public Health Agency of Canada. Obesity in Canada [Internet]. Ottawa (ON): Public Health Agency of Canada; 2011. Available at: http://www.phac-aspc.gc.ca/hp-ps/hl-mvs/oic-oac/adult-eng.php#figure-1

57. Tjepkema, M. Adult obesity in Canada: measured height and weight. In: Nutrition: findings from the Canadian Community Health Survey, no. 1. [Statistics Canada Catalogue No. 82-620-MWE]. Ottawa (ON): Statistics Canada; 2006.

58. Irvine B, Luo W, León JA. Congenital anomalies in Canada 2013: a perinatal health surveillance report by the Public Health Agency of Canada's Canadian Perinatal Surveillance System. Health Promot Chronic Dis Prev Can. 2015;35(1):21-2.

https://doi.org/10.24095/hpcdp.38.3.02

Equity reporting: a framework for putting knowledge mobilization and health equity at the core of population health status reporting

Lesley Ann Dyck, MA (1); Susan Snelling, PhD (2); Val Morrison, MA (3); Margaret Haworth-Brockman, MSc (4); Donna Atkinson, MA (5)

This evidence synthesis has been peer reviewed.

Abstract

The National Collaborating Centres for Public Health (NCCPH) collaborated on the development of an action framework for integrating equity into population health status reporting. This framework integrates the research literature with on-the-ground experience collected using a unique collaborative learning approach with public health practitioners from across Canada.

This article introduces the *Action Framework*, describes the learning process, and then situates population health status reporting (PHSR) in the current work of the public health sector. This is followed by a discussion of the nature of evidence related to the social determinants of health as a key aspect of deciding what and how to report. Finally, the connection is made between data and implementation by exploring the concept of actionable information and detailing the *Action Framework* for equity-integrated population health status reporting. The article concludes with a discussion of the importance of putting knowledge mobilization at the core of the PHSR process and makes suggestions for next steps. The purpose of the article is to encourage practitioners to use, discuss, and ultimately strengthen the framework.

Keywords: *population health status reporting, health equity, inequity, social determinants of health, knowledge mobilization*

Highlights

- Population health status reporting is a core public health practice, but in Canada it does not tend to explicitly describe health inequities or make recommendations for action to improve health equity.
- This project used a unique collaborative learning circle approach to examine how to better integrate a focus on health equity in population health status reporting processes.
- The result of the project is an action framework that puts knowledge mobilization at the centre to support the implementation of a population health status reporting process that is more likely to result in action to improve health equity.

Introduction

Describing differences in health status between and within populations or groups is central to population health status reporting in Canada.[1] However, we are particularly interested in differences in health status that can be judged as systematic, unfair and avoidable. These differences in health outcomes are often described as social inequalities, or inequities, and are rooted in unequal power relationships and structures across societies.[2,3] In order to address inequities and improve health equity, we must therefore take collaborative action to improve the social determinants at the root of the health disparity, which include a range of social, political and economic factors.[4] This is at the heart of an equity-integrated population health status reporting process.

The public health sector has a number of roles in addressing the social determinants of health and improving health equity.[5] The role we focus on in this article is 'assess and report'. Reporting purposefully on differences in health status between socio-economic groups, rather than adjusting for the effect of this difference on health status, has been identified as a promising practice for improving health equity.[6] Purposeful reporting of health inequities leverages both the core public health function of surveillance and the common practice of population health status reporting (PHSR). By assessing and reporting on health inequities, including effective strategies to reduce these inequities, the argument has been made that public health organizations are more likely to take action and be better able to support others to collaborate to decrease health inequities.[7]

We went looking for population health status reports in Canada that demonstrate the effective integration of health equity issues and the social determinants of

Author references:

1. National Collaborating Centre for Determinants of Health, St. Francis Xavier University, Antigonish, Nova Scotia, Canada
2. National Collaborating Centre for Methods and Tools, McMaster University, Hamilton, Ontario, Canada
3. Centre de collaboration nationale sur les politiques publiques et la santé, Institut national de santé publique du Québec, Montréal, Quebec, Canada
4. National Collaborating Centre for Infectious Diseases, University of Manitoba, Winnipeg, Manitoba, Canada
5. National Collaborating Centre for Aboriginal Health, University of Northern British Columbia, Prince George, British Columbia, Canada

Correspondence: National Collaborating Centre for Determinants of Health, St. Francis Xavier University, PO Box 5000, Antigonish, NS B2G 2W5; Tel: 902-867-6133; Email: nccdh@stfx.ca

health and found they were not common. When we did find them, there did not seem to be a consistent or standard approach.[8-11] This led us to ask: What does the effective integration of health equity look like in a PHSR process? What do we need to pay attention to in order to do it well? How does such a process contribute to action on the social determinants of health to improve health equity? While exploring these questions we developed the *Equity-Integrated Population Health Status Reporting: Action Framework,*[12] an action framework for the PHSR process that we thought might help to guide public health organizations in their work of 'assessing and reporting' in a manner that would drive action on the social determinants of health and health inequity.

This article introduces the *Action Framework* and provides the context for its development. We start by briefly describing our learning process, and then situate PHSR in the work of the public health sector. This is followed by a discussion of the nature of evidence related to the social determinants of health as a key aspect of deciding what and how to report. Finally, we make the connection between data and implementation by exploring the concept of actionable information, and then introduce our *Action Framework* for equity-integrated PHSR. We conclude with a discussion of the importance of putting knowledge mobilization at the core of the PHSR process and make suggestions for next steps.

Methods: our framework development process

Our learning process was led by the National Collaborating Centre for the Determinants of Health (NCCDH), one of six national collaborating centres for public health established in 2005 to strengthen knowledge translation and exchange for public health in Canada.[13] A learning circle of health equity champions from across Canada was established by the NCCDH, representing a diversity of perspectives from ten different public health organizations (such as program managers, medical health officers, policy analysts and epidemiologists from health units/regional health authorities, and provincial public health departments) and universities (researchers specifically). They were tasked with identifying and exploring the core issues associated with integrating health equity into population health status reporting, and identifying promising practices in the Canadian context. This resulted in the *Learning Together Series,*[14] a collection of documents describing the learning circle process and the key questions explored during each meeting of the circle. This became the foundation for a collaboration with the other five NCCPH centres to develop the *Equity-Integrated Population Health Status Reporting: Action Framework.*[12] This *Action Framework* was developed and refined through interviews with ten key stakeholders at local, provincial and national levels in Canada. Iterations of the *Action Framework* were also presented and discussed during workshops at three Canadian public health conferences* and a webinar.† Feedback from over 100 public health practitioners attending these events was collected via notes of the proceedings and evaluations, and used to inform the final version of the framework.

Results: production of an equity-integration population health status report

What is a population health status report?

The six core functions of public health in Canada include: population health assessment, health promotion, disease and injury control and prevention, health protection, surveillance, and emergency preparedness and epidemic response.[15,16] All levels of government (federal, provincial, territorial and their delegated authorities including regional health authorities) perform some or all of these functions. All governments appoint a chief public or medical health officer to provide leadership to their public health efforts in their respective jurisdictions,[15] with the legislation and roles varying somewhat across provinces and territories.

Reporting is not a core function but is an essential tool for fulfilling the public health mandate across the six core functions. In a summary of the structural profile of public health in Canada, the National Collaborating Centre for Healthy Public Policy found that the mandate to report on population health assessment and surveillance (as the key functions most relevant to population health status reporting) varied across jurisdictions. At the federal level "[t]he Chief Public Health Officer shall, within six months after the end of each fiscal year, submit a report to the Minister on the state of public health in Canada."[17] An example from the provincial level comes from British Columbia (BC), where the BC Public Health Act stipulates that population health assessment is mainly the responsibility of the Provincial Health Officer (PHO). At the regional level, an example from Manitoba positions population health assessment as a public health function that is partially assumed on the regional level with some of its components instituted by the Regional Health Authorities Act.[17]

Integrating the concept of equity

For our project, we used a definition of equity-integrated population health status reports to include "any instrument that uses existing scientific and local knowledge to inform decisions, improve health programs, and reduce health inequities."[1,p.2] Population health status reports generally include surveillance and other data, and tend to be used to highlight specific public health issues or topics.[1] Having said that, one of the challenges of examining health equity in the context of a population health status report is that there is no standardized format, content or process for this report. If we consider PHSR at the broadest level to be a type of population health assessment, we can frame it within the larger context of health knowledge (Figure 1).[18] Based on this, a population health status report can be understood as a product (e.g., print document, electronic file, or webpage) that provides an assessment of the health of the population and generates actionable public health knowledge. It is based on the same multiple data sources that inform both public health surveillance and public health research (Figure 1).[18]

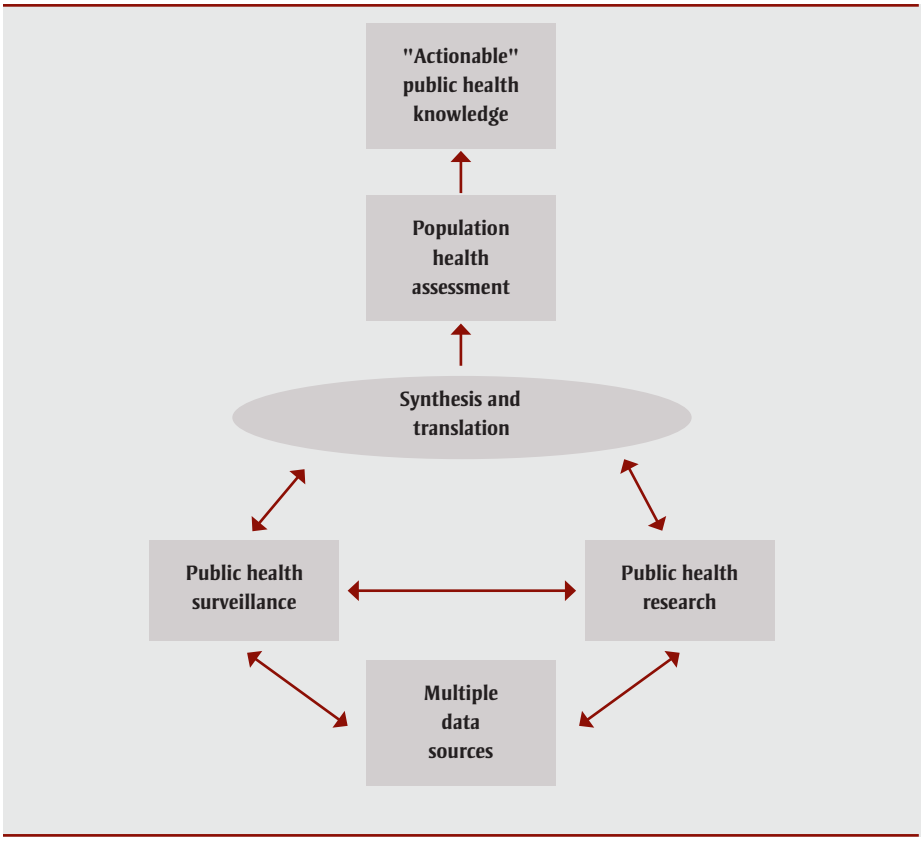
Characterizing the assessment process and its objectives

Information about how to undertake an effective and actionable population health status reporting/assessment process is

* Public Health Association of British Columbia Annual Conference (PHABC), Vancouver, BC, November 2013; The Ontario Public Health Convention (TOPHC), Toronto, ON, April 2014; Canadian Public Health Association Annual Conference (CPHA), Toronto, ON, May 2014.

† Hosted by CHNET-Works!, March 2014.

FIGURE 1
Public health surveillance in the larger context of health knowledge



Source: Lexicon, definitions, and conceptual framework for public health surveillance.[18,p.13]

hindered by the lack of established reporting and process guidelines. Community health assessment is a comprehensive community development approach, which is normally part of a larger community health improvement process.[19] It is often led by community organizations in partnership with the health sector and is most commonly found in the United States.[20] Like community health assessment, population health status reporting (PHSR) is both the activity and product of identifying and prioritizing population health issues, and it varies according to the size and nature of the community, the lead organization or partners and their goals, the resources available and other local factors.[20] However, PHSR as a process in Canada is led by the public health sector and is therefore more likely to be undertaken as a method of generating actionable information for the public health sector, not the wider community. As we shall explain, this presents a particular challenge for reporting on health inequities with the objective of generating action to improve the social determinants of health.

In our review of public health reports across Canada, we found that the intended purpose of any particular report was context/topic specific and could include any or all of: a) a program/service focus around improving accountability and assessing quality/effectiveness; b) a population focus to assess changes in health status over time and across geographic regions; and c) a health disparity focus to identify or quantify health differences between groups.[21] We concluded that the evidence-based and public nature of population health status reports, while not standardized or necessarily inclusive of equity issues, has helped them become a key building block for the construction and realignment of public health policies and programs.[1]

However, there is a second audience of healthy public policy stakeholders outside of the public health sector (including other government departments, municipalities, and community organizations) that is often targeted by these reports, usually through the inclusion of cross-sectoral intervention examples and recommendations for action.[21] With respect to action on the social determinants of health, this second "external" audience is critical. Health equity is determined by social factors related to broad public policy, norms and values—most of which are outside the influence of the health sector. Therefore, if the information is only actionable by the public health sector, it will be insufficient to reduce systemic health inequities.

Our learning circle of public health practitioners and researchers came to the conclusion that a successful report is one that is used. What makes the information in the report actionable is the critical consideration for how to best integrate health equity into a population health status report. What we learned from public health practitioners across Canada is that, in order to ensure a report is used, we need to attend to the format and content of a population health status report, as well as how we engage with stakeholders in the community as part of the data gathering and reporting process.

We will more closely examine engagement as a key principle of PHSR, but first we need to apply an equity lens to what is considered valid evidence for a population health status report.

The evidence base for "health inequity" as a public health issue and area for action

The evidence base for health inequity as a public health issue and area for action is growing; helped considerably by the establishment of the World Health Organization (WHO) Commission on Social Determinants of Health (CSDH). One of the CSDH knowledge networks created a guide for constructing the evidence base on the social determinants of health, including six conceptual and theoretical problems.[22] One of the most important points they make for translating knowledge about health inequities into action is that evidence on its own does not ensure success or provide an imperative for action. It needs refinement and engagement of all the players involved in generating evidence, turning it into policy, and turning policy into action and practice. The guide concludes with a recognition that—although we know a lot about the social factors that affect health—what is known is not universal in its applicability. What is known "… must therefore be read through a lens which deals with its

salience, meaning and relevance in particular local contexts."[22,p.218] This underscores the importance of engaging those who understand the local context in the process of gathering, analyzing and reporting data on population heath status in order to effectively integrate health equity considerations.

There are a number of population health status reports in Canada that have tackled the conceptual challenges in different ways in order to effectively integrate a health equity lens.[8-11,23-26] These reports share the distinction of being explicit about their focus on equity and intention to drive action to improve health equity, and referencing the collaborations and consultations with both organizations and citizens that were necessary to produce the reports. However, these reports do not share a standardized approach, and most are one-time-only reports making it difficult to track change over time and evaluate their collective impact on reducing health inequities. Notable exceptions to the one-time-only reports are the Toronto *Unequal City* reports from 2010 and 2015,[11] and the Community Health Assessments from Brandon 2004, 2009, and 2015[23] and Winnipeg 2004, 2009-10 and 2015.[26]

Part of the challenge of tracking change over time has been the diversity of measures and indicators used to assess and monitor health equity. This challenge has been of particular interest over the past decade or so in Canada, resulting in collaborative equity indicator development processes,[27,28] the development and application of a variety of socio-economic deprivation indices[29-31] and an equity indicator trend report.[32] Epidemiologists continue to discuss the best methods to measure and track health equity and inequity,[33-36] but some argue that it is not the quality of the measures that are the issue, but establishing agreement on which indicators to use and encouraging consistent collection and reporting over time.[37,38]

There continue to be significant conceptual and methodological issues that create barriers to accessing appropriate and high-quality data. For example, administrative health data do not normally include income, ethnicity, employment and education data that would allow us to disaggregate population data in a manner that would support a health equity assessment. This makes it very difficult to look at differences in health status between populations; particularly change over time for groups who have been traditionally marginalized and oppressed (e.g. people with disabilities, members of the LGBTQ community). We can also see these challenges in the poor quality and lack of Indigenous health information in Canada, the United States, New Zealand and Australia. Only recently have health surveys in Canada and elsewhere made it possible for people to self-identify as Indigenous, allowing analysts to better understand health inequities for Indigenous people living off-reserve and in urban settings. Finally, causal pathways between interventions and impacts on health inequities are not clearly understood,[22] making it difficult to know how and what data to collect as part of standard program evaluations. All of this has impeded "… the strategic implementation of evidence-based public health interventions aimed at preventing avoidable mortality."[39,p.644]

In Canada, data associated with First Nation, Inuit and Métis populations are often not available, incomplete, culturally inappropriate, and impacted by fundamental power and control issues, including jurisdictional arguments among different levels of government.[40,41] There have been attempts to overcome these challenges, for example through the work of the First Nations Information Governance Centre (FNIGC). The FNIGC has worked to put communities at the heart of the population health status reporting process by developing the First Nations Regional Health Survey (RHS). This has given communities control over the PHSR process, including decisions about participation, choice of indicators, ownership of data and the information reported.[42] However, this is only a first step as the First Nations RHS does not include the large number of Indigenous people living in urban settings across Canada or other Indigenous groups (e.g. Métis people).

Engagement and actionable health information

Corburn and Cohen make the case that "drafting, measuring, tracking, and reporting of indicators can be viewed not as a technical process for experts alone, but rather as an opportunity to develop new participatory science policy making, or what we call governance."[38,p.2] They refer to governance not just of formal institutions, but also "norms, routines and practices" that help shape issues that get onto the health research and policy agenda, the evidence base that is used, and the social actors who are deemed expert enough to participate in these decisions. As a result, it is the process of developing and using indicators of health equity/inequities that create opportunities for new healthy and equitable governance.[38] This is reinforced in the WHO Europe report on governance for health equity,[43] where they recommend equity and health equity as essential markers of a fair and sustainable society, requiring evidence and analysis connected to broad sectoral goals and joint assessment methods across sectors and stakeholders.

For public health institutions, using indicators to shape policy and drive action to improve health equity requires capacity to move beyond traditional indicators and engage with a broad range of stakeholders in non-traditional ways. It is important to recognize that

> [t]raditional indicators that measure morbidity and mortality tend to either place responsibility for improving health on the medical or public health communities or on vaguely identified institutions such as the economy, education, or built environment. The result is an overemphasis on medical and public health solutions while failing to articulate the specific institutions and policies that might need to change to promote greater health equity.[38,p.5]

A community-engaged approach to PHSR is critical for integrating health equity in a manner that informs the development and delivery of public health programs and services, but also drives intersectoral action on health inequity. This requires that the public health sector move beyond traditional monitoring and surveillance approaches and not be limited to population health status defined by aggregating individual-level health data. Given that evidence is never free of values, if we do not apply an equity lens to collecting, analyzing and synthesizing evidence, we run the risk of ignoring systemic power and oppression issues potentially embedded in population health status measures.

By adopting a community-engaged approach to the 'assess and report' role, the public health sector can benefit from the power of PHSR to blend evidence with values—in this case values of equity and fairness. A community-engaged approach to PHSR

makes these values explicit in evidence and increases the potential of the evidence to be actionable. The *Action Framework* identifies three essential components guiding the engagement process for equity-integrated PHSR, including communicate, collaborate, and apply a health-equity values lens[12] (see the "knowledge mobilization core" in Figure 2).

Discussion: an action framework for PHSR

In traditional population health status reports, the knowledge to action process emphasizes evidence and concludes with a summary of health status. In reporting processes oriented to action, however, the knowledge mobilization approach combines research knowledge with other types of knowledge and turns them into policy recommendations to drive practice. Although this action-oriented approach to PHSR is less common, there are increasing numbers of examples in Canada.[9,11,24]

Orientation to action

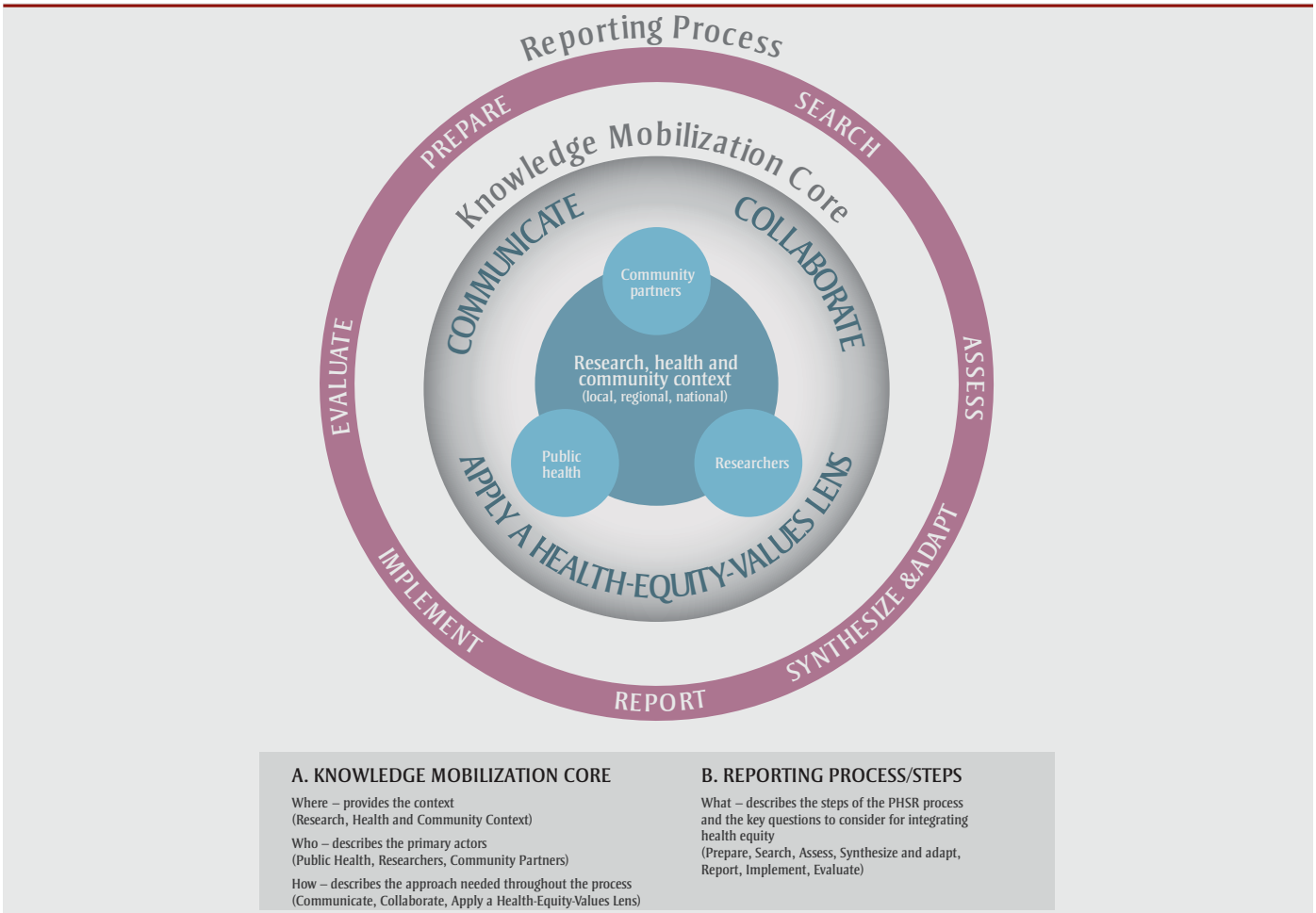
We are proposing a PHSR framework that is oriented to action, putting equity-informed knowledge mobilization at the core and surrounded by population health status reporting steps, as depicted in Figure 2. Although improved equity in population health status is the intended long-term outcome, the framework is unique in that it includes outcomes to ensure "the community is better equipped to take action to address health equity issues"[12,p.9] and therefore puts local intersectoral leadership at the very centre. The framework also identifies roles and specific outcomes for each of the three core stakeholder groups as a result of engaging in this process, including public health, community partners, and researchers.

The *Action Framework* draws from two similar evidence-driven frameworks: the *Evidence informed public health model* from the National Collaborating Centre for Methods and Tools (NCCMT)[44] and the *Action Cycle* developed by the Robert Wood Johnson Foundation (RWJF).[45] A brief summary of our framework is provided here, but a complete description—including promising-practice examples—can be found in the document *Equity-Integrated Population Health Status Reporting: Action Framework*[12] available from the NCCDH website.

Knowledge mobilization

The knowledge mobilization core of the framework is the foundation for the essential knowledge synthesis, translation and exchange that happens throughout the PHSR process. It is specific to the intended users of the framework (intersectoral community leadership) and is based on a

FIGURE 2
Equity-Integrated Population Health Status Reporting: Action Framework



Source: Summary – *Equity-Integrated Population Health Status Reporting: Action Framework*.[49,p.2]

collaborative approach that integrates health equity throughout. It includes three main elements related to *where, who,* and *how* (see Box 1). Concrete examples of strong knowledge mobilization for an equity-integrated PHSR approach in Canada and internationally can be found in the *Action Framework* document.[12] These include reports that apply an explicit health equity lens, as well as those that provide good examples of collaboration and communication practices around health equity and PHSR.

Steps for developing and implementing reports

The 'reporting process/steps' in our framework include seven steps for developing and implementing PHSR. Each step includes key questions to guide activities to ensure the right structures are implemented to support the work of the equity-integrated PHSR process (see Box 2). Just as we did for the knowledge mobilization core of the framework, we identified a number of promising practices in association with one or more of the seven steps of the reporting process. These can also be found in the *Action Framework* document.[12]

As a side note, the one step in the process that we were unable to find a promising practice for is the 'evaluate' step. One of the challenges around evaluating outcomes such as the impact of policy changes is the long-term nature of the process. As Hilary Graham has pointed out, this has an impact on political commitment to greater health equity, which "… may quickly wane, particularly if the policy changes … prove insufficient to secure a narrowing of inequalities … within the short time periods that governments typically set for their policy goals."[46,p.475] Through our consultation process we learned from some informants that they are either evaluating or planning to evaluate their PHSR activities, but we were not able to document concrete examples. As a next step in developing the

BOX 1
Knowledge mobilization core

Where – a PHSR process can be done at any level: local, regional, or national. At each level there are different people, organizations, political cultures, and available data. Ultimately, however, the community context and local issues inform the reporting process, and are impacted by it as part of the larger system(s). Over time, the community is better equipped to take action to address health equity issues, and the outcome is improvement in health equity within the local community context.

Who – the primary actors in a strong equity-integrated population health status reporting process are the public health sector, community partners, and researchers; a process led by any actor alone is less likely to result in action. The capacity for leadership and action of each is critical to being able to effectively integrate health equity into a PHSR process. The public health sector is essential in implementing PHSR, and public health actors and advocates are well positioned to provide leadership for an effective PHSR process. Community partners (including government, community organizations and other grassroots leaders) are critical throughout the entire process, and researchers working in a variety of settings and disciplines are important at different points in the process.

How – There is no 'one size fits all' approach to mobilizing knowledge in a PHSR process. However, there are principles that are essential to apply throughout the process, which have been captured in the framework as a series of questions that must be considered. These questions can be clustered into three groups: a) Apply a health-equity-values lens, b) Collaborate, and c) Communicate.

Source: Adapted from Summary - *Equity-Integrated Population Health Status Reporting: Action Framework*.[49,p.3]

BOX 2
Key questions for each of the seven steps of the equity-integrated PHSR process

1. *Prepare* - Who needs to be part of the process? What are the key questions and issues/problems? In what ways are equity values integrated into our investigation questions?

2. *Search* - What is the best way to find the relevant research evidence? What indicators will help us answer the research question? What other data are available? Do we need to develop a plan to collect additional data?

3. *Assess* - What are the data sources and the quality of the data? What limitations are inherent in the sources and data? Is there evidence available from other quantitative, qualitative or participatory research that can be used to complement the data? How do research approaches, data collection and analysis integrate health equity values? Do the various indicators adequately measure both assets and deficits? How well are population demographics disaggregated by geographic, economic and social characteristics?

4. *Synthesize and adapt* - How can we synthesize, adapt and integrate different types of evidence to paint a more complete picture of inequities? What recommendations can we make for practice based on the available evidence? How are health equity values integrated into our recommendations? How do the recommendations relate to the local context?

5. *Report* - Who is our audience and what is the best way to communicate what we have learned?

6. *Implement* - How can we frame the findings so that they engage everyone? What is the best way to explore potential actions, spanning from community mobilization to policy development? How can we collaborate to implement these potential actions?

7. *Evaluate* - How well did the PHSR process contribute to achieving our organizational goals for the report, where improved equity is included and integrated among those goals? In what ways did increased community capacity to take action on the social determinants of health and health equity result from the process?

Source: Adapted from: *Equity-Integrated Population Health Status Reporting: Action Framework*.[12,p.35]

equity-integrated PHSR framework, it will be important to seek out and learn from any evaluations that have been undertaken.

Conclusion: contribution and further development of the action framework

As our learning circle and other public health informants told us, a report that doesn't get used won't help us to improve health equity.[21] As a result, knowledge mobilization is a central feature of an equity-integrated PHSR framework. We learned that an equity-integrated PHSR process needs to be built around an iterative process that can be applied to fit the context and capacity of stakeholders, and that can draw on promising practices from other disciplines and jurisdictions. In our conversations with a range of public health practitioners, we also learned that to do it well, equity-integrated PHSR must be transparent in how it brings together evidence and social justice values. This makes it more likely that the data will be used to inform other larger processes, including community health assessment and improvement, anti-poverty initiatives and sustainable development work, all of which will contribute to improved health equity.

Although 'evaluate' is an important step in the framework, we were not able to find evaluations describing how PHSR contributes to action on the social determinants of health specifically. Collectively, we need to strengthen the evidence base for 'assess and report' as a promising public health practice to address health inequities.[14] We propose two main areas of inquiry and look forward to supporting research-to-practice collaborations in these areas:

(1) an assessment of current PHSR processes being implemented by public health in Canada, with the objective of evaluating both the processes and outcomes, including policy change[47]

(2) the development of clear performance guidelines for PHSR that effectively integrate health equity, as well as organizational and healthy public policy objectives[48]

Our hope is that this framework will contribute to the improvement and application of population health status reporting to advance health equity in Canada. We look forward to hearing from public health organizations about how they are using their own PHSR processes to improve health equity in their communities.

Acknowledgments

Funding for this project was provided from the operating budget of the National Collaborating Centre for the Determinants of Health, which is funded by the Public Health Agency of Canada. Special thanks to Tannis Cheadle for her assistance in developing and writing the *Action Framework* document. And thanks as always to the many public health practitioners who participated in the learning circle meetings, workshops and webinars, and reviewed many drafts along the way.

Conflicts of interest

The authors declare that they have no competing interests.

Authors' contributions

LAD provided leadership to project and conceptualized and wrote the initial draft of the paper. SS, VM, MHB and DA contributed relevant literature from each NCC program and provided feedback to drafts of the paper. All authors approved the final manuscript.

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada.

References

1. National Collaborating Centre for Determinants of Health. Learning together: what is a population health status report and why is it important? [Internet]. Antigonish (NS): NCCDH, St Francis Xavier University; 2012 [cited 2017 Jul 31]. Available from: http://nccdh.ca/images/uploads/Learning_Circle_3.pdf

2. Braveman PA, Kumanyika S, Fielding J, et al. Health, disparities and health equity: the issue is justice. Am J Public Health. 2011;101:S149-S155. doi: 10.2105/AJPH.2010.300062.

3. Whitehead M, Dahlgren G. Concepts and principles for tackling social inequities in health: levelling up part 1 [Internet]. Copenhagen (Denmark): World Health Organization; 2006 [cited 2017 Jul 31]. Available from: http://www.euro.who.int/__data/assets/pdf_file/0010/74737/E89383.pdf

4. National Collaborating Centre for Determinants of Health. Glossary of essential health equity terms [Internet]. Antigonish (NS): NCCDH, St. Francis Xavier University; 2014 [cited 2017 Jul 31]. Available from: http://nccdh.ca/images/uploads/comments/Glossary_EN.pdf

5. National Collaborating Centre for Determinants of Health. Let's talk: public health roles for improving health equity [Internet]. Antigonish (NS): NCCDH, St. Francis Xavier University; 2013 [cited 2017 Jul 31]. Available from: http://nccdh.ca/images/uploads/PHR_EN_Final.pdf

6. Sudbury & District Health Unit. 10 promising practices to guide local public health practice to reduce social inequities in health: technical briefing [Internet]. Sudbury (ON): Sudbury & District Health Unit; 2016 [cited 27 Jul 2017]. Available from: http://www.nmpha.org/Resources/Documents/10PromisingPractices.pdf

7. National Collaborating Centre for Determinants of Health. Let's talk: health equity [Internet]. Antigonish (NS): NCCDH, St. Francis Xavier University; 2013 [cited 2017 Jul 31]. Available from: http://nccdh.ca/images/uploads/Lets_Talk_Health_Equity_English.pdf

8. Public Health Agency of Canada. The Chief Public Health Officer's report on the state of public health in Canada: addressing health inequalities [Internet]. Ottawa (ON): PHAC; 2008 [cited 2017 Jul 31]. Available from: http://www.phac-aspc.gc.ca/cphorsphc-respcacsp/2008/fr-rc/pdf/CPHO-Report-e.pdf

9. Lemstra M, Neudorf C. Health disparity in Saskatoon: analysis to intervention [Internet]. Saskatoon (SK): Saskatoon Health Region; 2014 [cited 2017 Jul 31]. Available from: http://www.caledoninst.org/Special%20Projects/CG-COP/Docs/HealthDisparityRept-complete.pdf

10. Sudbury & District Health Unit. Opportunity for all: the path to health equity [Internet]. Sudbury (ON): Sudbury & District Health Unit; 2013 [cited 2017 Jul 31]. Available from: https://www.sdhu.com/wp-content/uploads/2015/10/Opportunity-for-All-Report_FINAL_for-web_FileInfo.pdf

11. Toronto Public Health. The unequal city 2015: Income and health inequities in Toronto: technical report [Internet]. Toronto (ON): Toronto Public Health; 2015 [cited 2017 Jul 31]. Available from: http://www1.toronto.ca/City%20Of%20Toronto/Toronto%20Public%20Health/Performance%20&%20Standards/Health%20Surveillance%20and%20Epidemiology/Files/pdf/Technical%20Report%20FINAL%20PRINT_AODA.pdf

12. National Collaborating Centre for Public Health and National Collaborating Centre for Determinants of Health. Equity-integrated population health status reporting: action framework [Internet]. Antigonish (NS): NCCDH, St. Francis Xavier University; 2016 [cited 2017 Jul 31]. Available from: http://nccph.ca/images/uploads/general/PHSR_Action_Framework_EN_final.pdf

13. National Collaborating Centres for Public Health. About us [Internet]. NCCPH; [cited 2017 Jul 31]. Available from: http://nccph.ca/about-us

14. National Collaborating Centre for Determinants of Health. Learning together: how and what we learned about equity-integrated population health status reporting [Internet]. Antigonish (NS): NCCDH, St Francis Xavier University; 2013 [cited 2017 Jul 31]. Available from: http://nccdh.ca/images/uploads/Equity_Integration_EN_Final_En.pdf

15. Marchildon GP. Canada: health system review 2013 [Internet]. Copenhagen: European Observatory on Health Systems and Policies; 2013 [cited 2017 Jul 31]. Available from: http://www.euro.who.int/en/about-us/partners/observatory/publications/health-system-reviews-hits/full-list-of-country-hits/canada-hit-2013

16. Pan-Canadian Public Health Network. Blueprint for a federated system for public health surveillance in Canada: vision and action plan [Internet]. Ottawa (ON): Pan-Canadian Public Health Network; 2016 [cited 2017 Jul 31]. Available from: http://www.phn-rsp.ca/pubs/bfsph-psfsp-2016/index-eng.php

17. National Collaborating Centre for Healthy Public Policy. Structural profile of public health in Canada [Internet]. Montréal (QC): NCCHPP; [cited 2017 Jul 27]. Available from: http://www.ncchpp.ca/710/Structural_Profile_of_Public_Health_in_Canada.ccnpps

18. Hall HI, Correa A, Yoon PW, Braden CR. Lexicon, definitions, and conceptual framework for public health surveillance. MMWR Surveill Summ. 2012;61(Suppl):10-14.

19. Fawcett S, Holt C, Schultz J. Some recommended practice areas for community health improvement (report to the CDC, office of prevention through healthcare) [Internet]. Kansas: Work Group for Community Health and Development, University of Kansas; 2011 [cited 2017 Jul 31]. Available from: http://ctb.ku.edu/sites/default/files/site_files/recommended_practices_for_enhancing_community_health_improvement.pdf

20. Association for Community Health Improvement. ACHI community health assessment tool kit [Internet]. Health Research and Educational Trust; 2007 [cited 2017 Jul 31]. Available from: http://www.assesstoolkit.org/

21. National Collaborating Centre for Determinants of Health. Learning together: reviewing evidence on the purpose of population health status reporting [Internet]. Antigonish (NS): NCCDH, St Francis Xavier University; 2012 [cited 2017 Jul 31]. Available from: http://nccdh.ca/images/uploads/Learning_Circle_4.pdf

22. Bonnefoy J, Morgan A, Kelly MP, Butt J, Bergman V. Constructing the evidence base on the social determinants of health: a guide [Internet]. Measurement and Evidence Knowledge Network (MEKN) of the WHO Commission on Social Determinants of Health; 2007 [cited 2017 Jul 31]. Available from: www.who.int/social_determinants/knowledge_networks/add_documents/mekn_final_guide_112007.pdf

23. Allan J, McPherson N, Williams C, et al. Prairie mountain health community health assessment 2015 [Internet]. Souris (MB): Prairie Mountain Health; 2015 [cited 2017 Jul 31]. Available from: http://www.prairiemountainhealth.ca/images/Community_Health_Assessment.pdf

24. Winnipeg Regional Health Authority. Health for all: building Winnipeg's health equity action plan [Internet]. Winnipeg (MB): Winnipeg Regional Health Authority; 2013 [cited 2017 Jul 31]. Available from: http://www.wrha.mb.ca/about/healthequity/files/HealthForAll_Documentwithlinks.pdf

25. Vancouver Coastal Health. Reducing health disparities in Vancouver Coastal Health communities: population health priorities [Internet]. Vancouver (BC): Vancouver Coastal Health; 2008 [cited 2016 Jan 11].

26. Centre for Healthcare Innovation and Winnipeg Regional Health Authority. 2014 community health assessment [Internet]. Winnipeg, MB: WRHA & CHI Evaluation Platform; 2015 [cited 2017 Jul 31]. Available from: http://www.wrha.mb.ca/research/cha2014/index.php

27. Provincial Health Services Authority. Development of priority health equity indicators for British Columbia: process and outcome report [Internet]. Vancouver (BC): Provincial Health Services Authority, Population and Public Health Program; 2014 [cited 2017 Jul 31]. Available from: http://www.phsa.ca/Documents/developmentofpriorityhealthequityindicatorsreportw.pdf

28. Pan-Canadian Public Health Network. Indicators of health inequalities. A report from the population health promotion expert group and the healthy living issue group for the Pan-Canadian public health network [Internet]. Pan-Canadian Public Health Network; 2010 [cited 2017 Jul 31]. Available from: http://www.phn-rsp.ca/pubs/ihi-idps/pdf/Indicators-of-Health-Inequalities-Report-PHPEG-Feb-2010-EN.pdf

29. Bell N, Hayes MV. The Vancouver area neighbourhood deprivation index (vandix): a census-based tool for assessing small-area variations in health status. Can J Public Health. 2012;103(Suppl 2):S32.

30. Chateau D, Metge C, Prior H, Soodeen R. Learning from the census: the socio-economic factor index (SEFI) and health outcomes in Manitoba. Can J Public Health. 2012;103(Suppl 2):S27.

31. Pampalon R, Hamel D, Gamache P, Philibert MD, Raymond G, Simpson A. An area-based material and social deprivation index for public health in Québec and Canada. Can J Public Health. 2012;103(Suppl 2):S22.

32. Canadian Institute for Health Information. Trends in income related health inequalities in Canada: technical report [Internet]. Ottawa (ON): CIHI; 2015 [cited 2017 Jul 31]. Available from: https://secure.cihi.ca/free_products/trends_in_income_related_inequalities_in_canada_2015_en.pdf

33. Norheim OF. Gini impact analysis: measuring pure health inequity before and after interventions. Public Health Ethics. 2010;3(3):282-292. doi: 10.1093/phe/phq017.

34. Harper S, King NB, Meersman SC, Reichman ME, Breen N, Lynch J. Implicit value judgments in the measurement of health inequalities. Milbank Q. 2010;88(1):4-29. doi: 10.1111/j.1468-0009.2010.00587.x.

35. Asada Y, Hurley J, Norheim OF, Johri M. A three-stage approach to measuring health inequalities and inequities. Int J Equity Health. 2014;13:98. doi: 10.1186/s12939-014-0098-y.

36. Moonesinghe R, Bouye K, Penman-Aguilar A. Difference in health inequity between two population groups due to a social determinant of health. Int J Environ Res Public Health. 2014;11(12):13083. doi: 10.3390/ijerph111213074.

37. Gakidou E, Fullman N. Monitoring health inequalities: measurement considerations and implications [Internet]. Brisbane (AU): Health Information Systems Knowledge Hub, School of Population Health, University of Queensland; 2012 [cited 2017 Jul 31]. Available from: http://www.healthdata.org/research-article/monitoring-health-inequalities-measurement-considerations-and-implications

38. Corburn J, Cohen AK. Why we need urban health equity indicators: integrating science, policy, and community. PLoS Med. 2012;9(8):e1001285. doi: 10.1371/journal.pmed.1001285.

39. Freemantle J, Ring I, Arambula Solomon TG, et al. Indigenous mortality (revealed): the invisible illuminated. Am J Public Health. 2015;105(4):644-652. doi: 10.2105/AJPH.2014.301994.

40. Adelson N. The embodiment of inequity: health disparities in aboriginal Canada. Can J Public Health. 2005;6(Suppl 2):S61.

41. King M, Smith A, Gracey M. Indigenous health part 2: the underlying causes of the health gap. Lancet. 2009;374(9683):76-85. doi: 10.1016/S0140-6736(09)60827-8.

42. First Nations Information Governance Centre. First Nations regional health survey (RHS) 2008/10: national report on adults, youth and children living in First Nations communities [Internet]. Ottawa (ON): FNIGC; 2012 [cited 2017 Jul 31]. Available from: http://fnigc.ca/sites/default/files/First%20Nations%20Regional%20Health%20Survey%20(RHS)%202008-10%20-%20National%20Report.pdf

43. Brown C, Harrison D, Burns H, Ziglio E. Governance for health equity: taking forward the equity values and goals of health 2020 in the WHO European region [Internet]. Copenhagen (Denmark): World Health Organization Regional Office for Europe; 2013 [cited 2017 Jul 31]. Available from: http://www.euro.who.int/__data/assets/pdf_file/0020/235712/e96954.pdf

44. National Collaborating Centre for Methods and Tools. Evidence-informed public health [Internet]. Hamilton (ON): NCCMT, McMaster University; 2016 [cited 2017 Jul 31]. Available from: http://www.nccmt.ca/professional-development/eiph

45. Robert Wood Johnson Foundation. County health rankings and roadmaps - action center [Internet]. Robert Wood Johnson Foundation; [cited 2017 Jul 31]. Available from: http://www.countyhealthrankings.org/roadmaps/action-center

46. Graham H. Health inequalities, social determinants and public health policy. Policy Polit. 2009;37(4):463-479. doi: 10.1332/030557309X445618.

47. Embrett MG, Randall GE. Social determinants of health and health equity policy research: exploring the use, misuse, and non-use of policy analysis theory. Soc Sci Med. 2014;108:147-155. doi: 10.1016/j.socscimed.2014.03.004.

48. Nakaima A, Sridharan S, Gardner B. Towards a performance measurement system for health equity in a local health integration network. Eval Program Plann. 2013;36(1):204-212. doi: 10.1016/j.evalprogplan.2012.03.009.

49. National Collaborating Centres for Public Health and National Collaborating Centre for Determinants of Health. Summary - equity-integrated population health status reporting: action framework [Internet]. Antigonish (NS): NCCDH; 2016 [cited 2017 Jul 31]. Available from: http://nccdh.ca/images/uploads/comments/PHSR_Action_Framework_Summary_EN_v3.pdf

https://doi.org/10.24095/hpcdp.38.3.03

Addition of food group equivalents to the Canadian Diet History Questionnaire II for the estimation of the Canadian Healthy Eating Index-2005

Maria McInerney, MSc (1); Vikki Ho, PhD (2); Anita Koushik, PhD (2); Isabelle Massarelli, RD, (3); Isabelle Rondeau, RD, (3); Gavin R. McCormack, PhD (1); Ilona Csizmadi, PhD (1)

This original quantitative research article has been peer reviewed.

Abstract

Introduction: Poor diet quality has been shown to increase the risk of common chronic diseases that can negatively impact quality of life and burden the healthcare system. Canada's Food Guide evidence-based recommendations provide dietary guidance aimed at increasing diet quality. Compliance with Canada's Food Guide can be assessed with the Canadian Healthy Eating Index (C-HEI), a diet quality score. The recently designed Canadian Diet History Questionnaire II (C-DHQ II), a comprehensive food frequency questionnaire could be used to estimate the C-HEI in Canadian populations with the addition of food group equivalents (representing Canada's Food Guide servings) to the C-DHQ II nutrient database. We describe methods developed to augment the C-DHQ II nutrient database to estimate the C-HEI.

Methods: Food group equivalents were created using food and nutrient data from existing published food and nutrient databases (e.g. the Canadian Community Health Survey — Cycle 2.2 Nutrition [2004]). The variables were then added to the C-DHQ II companion nutrient database. C-HEI scores were determined and descriptive analyses conducted for participants who completed the C-DHQ II in a cross-sectional Canadian study.

Results: The mean (standard deviation) C-HEI score in this sample of 446 adults aged 20 to 83 was 64.4 (10.8). Women, non-smokers, and those with more than high school education had statistically significant higher C-HEI scores than men, smokers and those with high school diplomas or less.

Conclusion: The ability to assess C-HEI using the C-DHQ II facilitates the study of diet quality and health outcomes in Canada.

Keywords: *diet quality, healthy eating index, food frequency questionnaire, dietary assessment, nutrient database*

Highlights

- High diet quality is important for chronic disease prevention.
- The Canadian Healthy Eating Index (C-HEI), a dietary pattern score which reflects age- and sex-specific dietary recommendations in the 2007 Eating Well with Canada's Food Guide (CFG), can be used to measure and monitor diet quality.
- Food group equivalents (representing CFG servings) are required to derive the C-HEI, however, these variables are absent from most Canadian nutrient databases.
- This study demonstrates a rigorous yet feasible approach for adding food group equivalents to the Canadian Diet History Questionnaire II nutrient database.
- Now, the Canadian Diet History Questionnaire II can be used to derive the C-HEI to quantify diet quality in Canadian populations.

Introduction

Dietary pattern indices are multidimensional measures that capture several components of diet and can be used to assess diet quality.[1] There is growing interest in the development, estimation, and application of dietary pattern indices because of the relation between diet and chronic diseases[2–5] and mortality risk.[6] From a surveillance perspective, some dietary pattern indices are useful in determining how well populations meet national dietary recommendations.[7,8]

The Healthy Eating Index (HEI)[9] is a diet quality score originally developed by the United States Department of Agriculture. Similarities between the dietary recommendations for Canada and the United States facilitate the adaptation of the American HEI for Canada.[8,10–13] In one adaptation[8], the Canadian Healthy Eating Index 2005 (C-HEI) was created using the 2007 Eating Well with Canada's Food Guide (CFG)[14] recommendations and serving equivalents, thus reflecting the Canadian age- and sex-specific dietary recommendations. The C-HEI is therefore appropriate for monitoring and evaluating the diet quality of Canadians.[8,10]

C-HEI scores range from 0 to 100, representing total diet quality through adequacy

Author references:

1. Department of Community Health Sciences, Cumming School of Medicine, University of Calgary, Calgary, Alberta, Canada
2. CRCHUM (Centre de recherche du CHUM) and Département de médecine sociale et préventive, Université de Montréal, Montréal, Quebec, Canada
3. Bureau of Food Surveillance and Science Integration, Food Directorate, Health Canada, Ottawa, Ontario, Canada

Correspondence: Maria McInerney, Department of Community Health Sciences, Cumming School of Medicine, University of Calgary, 3280 Hospital Drive NW, Calgary, AB T2N 4Z6; Email: mcmciner@ucalgary.ca

and moderation components[8] with higher scores representing closer alignment with CFG recommendations. The C-HEI adequacy components reflect intakes of total fruits and vegetables, whole fruit, dark green and orange vegetables, milk and alternatives, meat and alternatives, total grain products, whole grain products and unsaturated fats. The moderation components reflect intakes of sodium, saturated fats, and other foods not recommended in CFG. Table 1 outlines the C-HEI[8] scoring criteria.

The Canadian Diet History Questionnaire II (C-DHQ II), a comprehensive food frequency questionnaire, was recently adapted for use in Canada from the US National Cancer Institute's DHQ II, using dietary intake data reported in the Canadian Community Health Survey, Cycle 2.2, Nutrition (2004)[15] to create the 331 C-DHQ II database food categories. The C-DHQ II is available online[16,17] and is used in numerous Canadian studies currently in progress[18,19] and there is growing interest in using it to assess diet quality as was an earlier version of the questionnaire.[20]

We describe methods used to create food group equivalents (representing CFG servings) required to derive the C-HEI (total fruits and vegetables, whole fruit, dark green and orange vegetables, milk and alternatives, meat and alternatives, total grain products, whole grain products and other foods) for foods queried on the C-DHQ II. We also present food group equivalents and C-HEI scores derived from the C-DHQ II for a sample of Canadian adults enrolled in the "Pathways to Health Study."[21] This article is based on a larger study reported in the graduate thesis *Associations between the Neighbourhood Food Environment, Neighbourhood Socioeconomic Status and Diet Quality in Canadian Adults.*[22]

TABLE 1
Scoring criteria for Canadian adapted Healthy Eating Index (C-HEI)

Component (food group)	Range of scores	Scoring criteria
Adequacy[a]	**0 to 60 points**	
Total vegetables and fruit	0 to 10 points	Minimum: 0 Maximum: 4 to 10 servings[b]
Whole fruit	0 to 5 points	Minimum: 0 Maximum: 0.8 to 2.1 servings (21% of recommendation for total vegetables and fruit)[b]
Dark green and orange vegetables	0 to 5 points	Minimum: 0 Maximum: 0.8 to 2.1 servings (21% of recommendation for total vegetables and fruit)[b]
Total grain products	0 to 5 points	Minimum: 0 Maximum: 3 to 8 servings[b]
Whole grain products	0 to 5 points	Minimum: 0 Maximum: 1.5 to 4 servings (50% of recommendation for total grain products)[b]
Milk and alternatives	0 to 10 points	Minimum: 0 Maximum: 2 to 4 servings[b]
Meat and alternatives	0 to 10 points	Minimum: 0 Maximum: 1 to 3 servings (75 to 225 grams)[b]
Unsaturated fats	0 to 10 points	Minimum: 0 Maximum: 30 to 45 grams[b]
Moderation[c]	**0 to 40 points**	
Saturated fats	8 to 10 points 0 to 8 points	Maximum 7% to 10% of total energy intake
Sodium	8 to 10 points 0 to 8 points	Adequate intake to tolerable upper intake level
"Other food"	0 to 20 points	Minimum: 5% or less of total energy intake Maximum: 40% or total energy intake

Source: Garriguet D. Diet quality in Canada. Health Rep. 2009;20(3):41-52.

[a] For adequacy components, 0 points for minimum or less, 5 or 10 maximum or more, and proportional for amounts between minimum and maximum.

[b] According to age and sex, as specified in *Canada's Food Guide*.

[c] For moderation components, 10 or 20 points for minimum or less, 0 points for maximum or more, and proportionally between minimum and maximum.

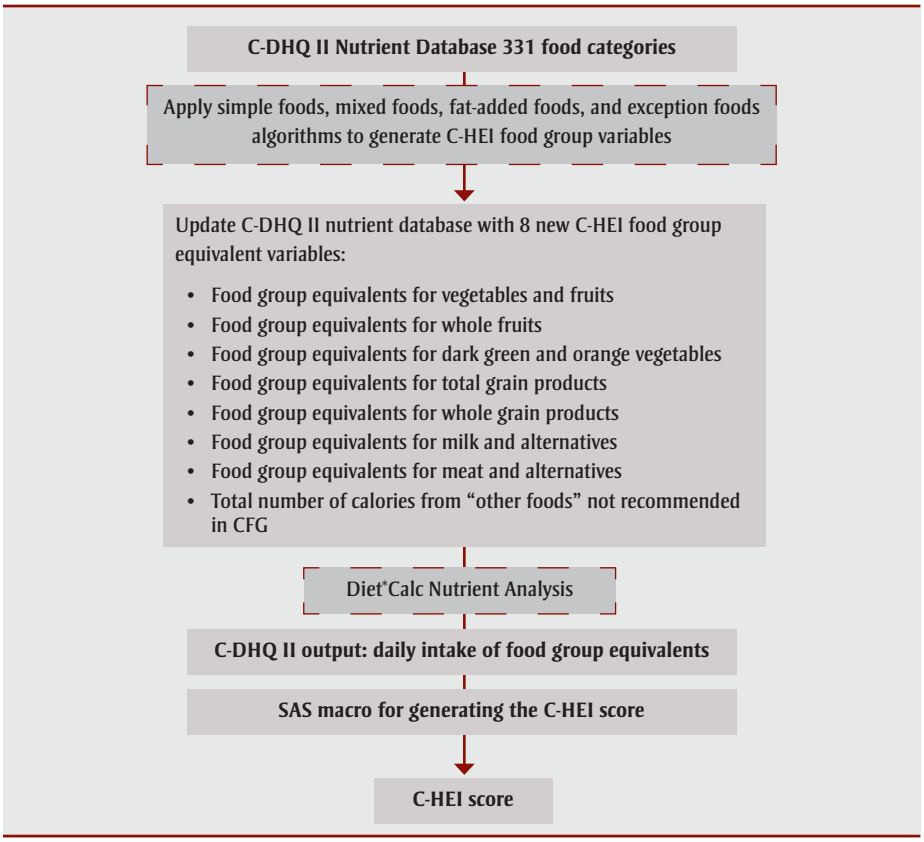
Methods

An overview of the steps required to derive the C-HEI from the C-DHQ II is presented in Figure 1. Eight new variables representing food group equivalents (CFG servings) for total fruits and vegetables, whole fruit, dark green and orange vegetables, milk and alternatives, meat and alternatives, total grain products, whole grains and other foods, were created for each of the 331 C-DHQ II nutrient database food categories. Algorithm steps for creating the new variables from Canadian Community Health Survey foods are shown in Figure 2.

Multiple data sources were used to compute the new variables: the C-DHQ II nutrient database, the most recent US DHQ II nutrient database, Health Canada's Classification of Foods in the Canadian Nutrient File According to Eating Well with Canada's Food Guide report and accompanying database (hereafter referred to as CNF/CFG Classification)[23,24], the detailed food and recipe file derived from the Canadian Community Health Survey[15] and used in the development of the C-DHQ II nutrient database, the National Health and Nutrition Examination Survey food and nutrient database used to create the US DHQ II nutrient database, and the US Food Patterns Equivalent Database.[25]

Of the 331 food categories in the C-DHQ II nutrient database, 302 were divided into four broad groups based on their composition (ingredients). Algorithms of varying complexity were required to disaggregate the food categories according to the components of the C-HEI (food group equivalents). The four algorithm groups were: 1) simple foods; 2) mixed foods; 3) fat-added foods; and 4) exception foods (Figure 2). The 29 remaining food groups

FIGURE 1
Process overview



Abbreviations: C-DHQ II, Canadian Diet History Questionnaire II; CFG, Eating Well with Canada's Food Guide; C-HEI, Canadian Healthy Eating Index; SAS, Statistical Analysis System Software.

were allocated on an individual basis. The *simple foods* category represented C-DHQII foods that comprise mainly single food items (e.g. milk, bananas, peppers, breads, and meats). The *mixed foods* category represented C-DHQII foods that comprise multiple foods (e.g. pasta with meat sauce, chicken mixtures [sandwiches], pizza with vegetables and meat). The *fat-added foods* category represented C-DHQII foods that comprise simple food items identified in the C-DHQII as having fat added in the cooking or preparation process (e.g. peppers with fat added). The *exception foods* category represented C-DHQII foods that were usually not consumed as stand-alone foods (e.g. sauces, spreads, and condiments).

For each of the 302 food categories we identified the top one to three most frequently reported foods in the Canadian Community Health Survey food and recipe file (hereafter referred to as the primary foods) to represent the corresponding C-DHQ II food category for deriving food group equivalents. The top two or three foods were examined if multiple foods with similar nutrient profiles were reported at similar frequencies of intake. In cases of multiple primary foods, we estimated the average standard CFG serving weight in grams and then weighted the food group allocations accordingly.

The primary food(s) identified in the Canadian Community Health Survey food and recipe file were further examined to confirm that they were sufficiently representative of corresponding C-DHQ II food categories by comparing their nutrient profiles to the category-specific nutrient profiles in the C-DHQ II nutrient database. Nutrient content for key nutrients [energy (kcal), total sugar (g), total fat (g), total saturated fat (g), and sodium (mg)] were compared for 100 gram equivalents of food. The primary food was deemed sufficiently representative if the key nutrient values from the Canadian Community Health Survey file and the C-DHQ II nutrient database profile differed by ≤ 5%. In cases where the difference was > 5%, the next most frequently reported foods were additionally considered until a sufficient representation was found (exploring up to three top reported foods per C-DHQ II food category). This approach, while conservative, was considered reasonable based on the expert opinion of the authors (MM, IR, IM and IC).

In reality, the number of nutrient profiles examined to identify a primary food varied when the top most frequently reported foods were reported at similar frequencies. In such cases, the key nutrients of all the top reported foods were examined to determine similarities amongst their nutrient profiles. If the nutrient profiles differed by ≥ 10%, then up to five foods were considered for analysis. We then calculated food group equivalents for each food and then averaged the nutrient values to determine a final value. Other exceptions occurred in the mixed foods algorithm where up to 20 nutrient profiles could be examined to determine the most representative primary food(s) by consensus established amongst co-authors. This was done to account for the diversity of foods included in C-DHQ II mixed foods. Two co-authors (IM and IR) randomly reviewed 10% of the food group equivalent variables as a quality assurance check by manually recalculating the food group equivalents to ensure the algorithms were correctly applied.

The CNF/CFG classification was designed by Health Canada to assess the Canadian population's adherence to the CFG recommendation for food group intakes for healthy eating.[23,24] The CFG classification was applied to the Canadian Nutrient File (2001b)[26] which was the nutrient composition database linked to reported foods in the Canadian Community Health Survey. The CNF/CFG classification assigns each CNF food to a CFG food group and subgroup. For the four major CFG food groups (vegetables and fruits, grain products, milk and alternatives, meat and alternatives) CNF foods are classified into "Tiers" according to the quality of their alignment with CFG recommendations with additional consideration given to fat, sugar, and sodium content.[23,24] Tier 1 and 2 are "foods in line with CFG guidance"; Tier 3 foods are "foods partially in line with CFG guidance"; and Tier 4 food are "foods not in line with CFG guidance."[23,24]

We used the CNF/CFG classification and accompanying database to identify the weight of one standard CFG serving size of the primary food and the CFG food group(s) to which the primary food

FIGURE 2
Basic algorithms

All 331 C-DHQ II food categories
N=331

All C-DHQ II food groups are categorized into 4 mutually exclusive groups.

Simple foods N=208	Mixed foods N=38	Fat-added foods N=19	Exception foods N=37
Step 1: Identify primary foods in CCHS detailed file.	Step 1: Identify primary foods in CCHS detailed file.	Step 1: Identify primary foods in CCHS detailed file using C-DHQ II food groups *without* the fat added.	Step 1: Complete steps 1 to 4 as in "simple foods" for foods with 7000, 8000 and 9999 classification in CNF/CFG classification.
Step 2: Query primary food in the CNF/CFG classification to determine weight in grams (g) of 1 CFG serving.	Step 2: Query primary food in US DHQ II MPED/FPED detailed file and determine up to 3 primary MPED/FPED equivalents (measured in US cup or oz/100g).	Step 2: Determine the CFG food group(s) from Tier allocation for primary food in the CNF/CFG classification.	Step 2: 7000 and 9999 coded foods are not considered for allocation toward any of the CFG equivalent variables.
Step 3: Compare nutrient profile of food identified in the CNF/CFG classification to nutrient profile in the C-DHQ II nutrient database.	Step 3: Adjust the MPED/FPED equivalent to be MPED/FPED value per gram weight of each C-DHQ II portion weight (g).	Step 3: Determine grams of fat added by subracting the weight (g) of the non-fat-added C-DHQ II group weight from the fat-added C-DHQ II group in the C-DHQ II nutrient database.	Step 3: 8000 coded foods: assess sugar, fat, and sodium content of C-DHQ II group using RA values, the method and thresholds described in CNF/CFG classification for Tiers.
Step 4: Determine the CFG food group(s) allocation for primary food in the CNF/CFG classification database.	Step 4: Query the weight (g) of 1 CFG serving and 1 US serving for each food that constitutes the primary MPEDs/FPEDs identified in step 2, in the CNF/CFG classification tool and the MPED/FPED User Guide, respectively.	Step 4: Determine the primary type of fat added (saturated vs unsaturated) using C-DHQ II nutrient database.	Step 4: if the food is determined to be Tier 4, allocate the caloric value to the "other foods" variable. All other variables receive value of 0. If the food is determined to be Tier 1-3, repeat steps 2 to 6 as described in "mixed foods".
Step 5: Divide the weight (g) of each C-DHQ II portion size by the weight (g) of 1 CFG serving of the primary food identified in step 2.	Step 5: Using values determined in step 4, adjust the US standard serving weight to reflect the CFG standard weight for MPEDs/FPEDs value in step 3. Product is the number of CFG serving equivalents.	Step 5: For primarily unsaturated fats as the added fat, use the gram weight of fat the fat added C-DHQ II food group to repeat steps 3 to 6 as described in "simple foods".	
Step 6: In the C-DHQ II nutrient database, allocate the value to the appropriate CFG serving equivalents variable(s) (identified in step 4). All other CFG serving equivalent variables receive a value of 0.	Step 6: In the C-DHQ II nutrient database, allocate CFG serving equivalents to appropriate CFG serving equivalent variable (s) which align with the MPED/FPED allocations. All other CFG serving equivalent variables receive a value of 0.	Step 6: For primarily saturated fats as added fat, use the gram weight of no fat added C-DHQ II food group to repeat steps 3 to 6 as described in "simple foods". In addition, calculate the caloric value of added saturated fat and allocated the caloric value to the "other foods" CFG equivalent variable.	

Abbreviations: CCHS, Canadian Community Health Survey; C-DHQ II, Canadian Diet History Questionnaire II; CFG, Eating Well with Canada's Food Guide; C-HEI, Canadian Healthy Eating Index; CNF/CFG, Health Canada's Canadian Nutrient File/Canada's Food Guide Classification Tool; DHQ II, Diet History Questionnaire II; FPED, Food Patterns Equivalents Database; MPED, MyPyramid Equivalents Database; RA, Reference Amounts (from CNF/CFG).

Notes: N = 29 food groups did not follow any of the algorithms because the foods were either not found in the CNF/CFG, the foods comprising the C-DHQ II group could not be determined, or there was substantial conflict in Tier allocation for the most frequently reported foods. These food groups were considered on an individual basis and reviewed by at least 3 reviewers. According to the CNF/CFG Tool: 7000 = "other foods recommended in the CFG"; 8000 = "recipes not classified"; 9999 = "food and beverage not classified".

belonged (i.e. where the food group equivalent should be allocated). All Tier 4 foods and those coded in the CNF/CFG classification as "other foods"; "meal replacements and supplements" and, "foods and beverages not classified" were allocated to the food group equivalent for "other foods". In cases where the primary food was not found in the CNF/CFG classification, or if the exact type of food was unknown (e.g. "bread" with no further descriptors) the standard weight for such foods in the CFG[14] was used (e.g. in CFG the serving weight for bread is 30 g). For primary food(s) not found in the CNF/CFG classification or in the US DHQII or National Health and Nutrition Examination Survey database, consultation among authors determined the most appropriate CFG food group allocation(s).

For multiple primary foods and where there was discrepancy in Tier classification between the primary foods, the Tier classification that best represented the primary food was assumed (i.e. the Tier allocation that appeared most frequently). When the predefined algorithms could not be used due to missing information (e.g. primary food was not found in the CNF/CFG classification database), and for foods coded in the CNF/CFG classification as "recipes", similar foods or ingredients, were identified to determine an appropriate Tier allocation for the C-DHQ II category. In such cases, authors discussed and decided on appropriate group allocation.

For the mixed food algorithm, we employed the US Food Patterns Equivalents (FPED) and MyPyramid Equivalent Database (MPED) food group allocations in the National Health and Nutrition Examination Survey foods and recipes database to guide food group equivalent allocation. MPEDs were used for grain products (instead of FPEDs) since the methods employed to derive their values aligned more closely with the current method for deriving food group equivalents.[26]

To determine the ingredients of mixed foods (e.g. type of meat, vegetable, grain), the descriptors of the primary food(s) found in the Canadian Community Health Survey food and recipe file were used. In some cases, assumptions were based on the authors' knowledge of ingredients typically included in mixed food recipes (e.g. for vegetable spring rolls, cabbage was assumed as the primary contributor to the vegetables and fruit CFG food group). To confirm acceptability of assumptions, we compared the nutrient profiles of primary food(s) in the National Health and Nutrition Examination Survey database to nutrient profiles for the food category in the C-DHQ II nutrient database, with attention to key nutrients previously mentioned. An approximate threshold of 20% difference between the two nutrient profiles was deemed acceptable given the difference in food composition between the US and Canada.[17] For each primary food, the three highest contributing FPED (and in the case of grains, MPED) values were adjusted to reflect the total gram weight of each ingredient of the mixed food found in each of the six C-DHQ II portion sizes. The gram weights were then adjusted from US standard serving weights[26] to standard CFG serving weights.[24]

There were 29 C-DHQ II food categories that could not be allocated according to the four mutually exclusive food groups because either the foods were not found in the CNF/CFG classification, the foods comprising the C-DHQ II food categories could not be determined, there was substantial conflict in Tier allocation for the most frequently reported foods, or the food was not relevant for C-HEI estimation (e.g. spices, coffee, tea). We reviewed these 29 C-DHQ II food categories on an individual basis.

Because no brand names were available in the Canadian Community Health Survey foods and recipes file for ready-to-eat breakfast cereals, they could not be identified in the CNF/CFG classification to determine if they contributed to whole grains. As such, an algorithm was designed to minimize the under-estimation of whole grains. To determine a standard gram weight for 'ready-to-eat' breakfast cereals in the C-DHQ II food categories, an average weight from all 'ready-to-eat' breakfast cereals in the CNF/CFG classification database was calculated. To ensure that an accurate amount of whole grains were allocated, the US DHQ II databases allocation of whole grains for 'ready-to-eat' breakfast cereals was used. The MPED groups considered were "total grains" and "whole grains" and used in the formula:

$$\frac{Whole\ grain\ MPED}{Total\ grain\ MPED}\ X\ 100\ =\ \%\ whole\ grain\ in\ total\ grain\ MPED$$

We then multiplied the percent whole grain in total grain MPED by the Canadian total grains value (using the simple foods algorithm) to estimate the value to be assigned to whole grains food group equivalent variable.

Completed food group equivalents for all C-DHQ II food categories were added to the C-DHQ II nutrient database. Daily intake of food group equivalents for each food group was estimated by the DietCalc software (version 1.5.1), the nutrient analysis program for the C-DHQ II. SAS was used to analyze the C-DHQ II output and to derive the C-HEI *(the SAS code to derive the C-HEI is available from authors)*.

Test sample and procedure

Participants recruited from the "Pathways to Health Study"[21], a study aimed at investigating the relation between diet quality and neighbourhood environments in adults, completed the C-DHQ II. The study began in April 2014 and included a two-staged stratified random sample of adults from 12 Calgary neighbourhoods, in Alberta, Canada. The 446 participants who responded to mailed invitations to complete the online C-DHQ II are included in this analysis.

All analyses were stratified by sex. Descriptive statistics (means, standard deviations, median, minimums, and maximum) were computed for: C-HEI total score, C-HEI component scores, energy intake, and the number of CFG servings. Mean energy intake and CFG servings were examined to determine the plausibility of results based on 'a priori' knowledge of food intakes in comparable populations.

Previous reports suggest that C-HEI scores vary by socio-demographic characteristics (e.g. sex, age, ethnicity, immigrant status, household income, education level, and smoking status).[8] We used ANOVA with *post-hoc* Bonferroni adjustments to estimate differences in total C-HEI scores by: age group, marital status, level of education, gross household income, smoking status, and ethnicity in the total population and by sex. In addition, we assessed the linear trend in C-HEI scores for household income category and education level in the total population and by sex. We used independent t-tests to estimate differences in C-HEI component scores, by sex. Statistical significance was set at

$\alpha < 0.05$. All significance testing was completed using Stata statistical software: Release 13.0 (StataCorp LP, College Station, TX, US).

Results

The study sample consisted of 172 men and 274 women. A majority of the participants were married/living with a partner, had at least a high-school diploma, had a household income above $60 000, were non-smokers, and were white (Table 2).

The distribution of C-HEI scores was approximately normal (data not shown). The mean daily C-HEI score was 64.4 (standard deviation: 10.8) (Table 2). Men (61.5 [10.5]) and smokers (56.9 [11.8]) had lower C-HEI scores than women (66.3 [10.6]), and non-smokers (64.7[10.6]), respectively. Further, there was a statistically significant increasing linear trend with level of education. Non-smoking status and higher education was also associated with higher C-HEI scores amongst women but only non-smoking status was associated with a higher C-HEI score in men.

The mean (standard deviation) daily energy intake was 1650 (717) kcal (Table 3). The highest mean number of average daily CFG servings was reported for total vegetables and fruit 6.3 (3.2) and the lowest was for whole grains 0.4 (0.4). For every CFG food group, the mean number of daily servings was below the CFG age and sex-specific recommendations.

C-HEI component scores are presented in Table 3. Compared to men, women had significantly ($p < .05$) higher 'unsaturated fat', 'sodium', 'meat and alternatives', and 'other food' component scores while men had significantly ($p < .05$) higher 'saturated fat' component scores.

Figure 3 illustrates the multidimensional aspect of the C-HEI and the variation in compliance with recommendations for adequacy of food group intakes and moderation in the intake of not recommended dietary components. On average, none of the recommendations for these components are met 100%, and men and women follow similar patterns of alignment with C-HEI component intakes. The lowest scoring component was whole grains while

TABLE 2
Demographic characteristics of participants and distribution of average daily total C-HEI scores, by sex

	Total				Men				Women			
	%	Mean (SD)	Median	Min, max	n	Mean (SD)	Median	Min, max	n	Mean (SD)	Median	Min, max
Overall C-HEI scores	N = 446	64.4 (10.8)	65.3	30.2, 88.5	172	61.5 (10.5)	62.9	31.4, 79.2	274	66.3 (10.6)[a]	67.5	30.2, 88.5
Age (years)												
21–39	20.4	65.6 (10.1)	66.4	37.1, 88.5	23	60.0 (10.5)	62.9	37.1, 77.4	68	67.4 (9.4)[b]	68.4	46.1, 88.5
40–59	44.8	64.2 (10.8)	65.4	30.2, 85.1	68	61.6 (10.2)	62.9	31.5, 78.6	132	65.6 (10.9)	67.0	30.2, 85.1
60+	34.8	64.1 (11.0)	65.1	31.4, 84.2	81	61.9 (11.0)	63.0	31.4, 79.2	74	66.5 (10.7)	67.6	7.1, 84.2
Marital status												
Married or living with partner	78.5	65.1 (10.6)	65.7	31.4, 88.5	141	61.8 (10.3)	62.1	31.4, 79.2	209	67.3 (10.3)[b]	68.5	36.9, 88.5
All other arrangements	21.5	62.1 (11.0)	64.2	30.2, 84.0	31	60.1 (11.8)	63.9	31.5, 75.8	65	62.1 (11.0)	64.2	30.1, 84.0
Education												
High school diploma or less	12.6	60.9 (9.8)	60.3	39.9, 79.6	21	59.4 (9.4)	59.9	41.0, 79.2	35	61.7 (10.1)	60.8	39.9, 79.6
College/vocation/ trade/certificate	17.5	64.2 (11.8)	65.5	31.4, 84.3	31	61.7 (11.6)	63.2	31.4, 78.0	47	65.9 (11.7)	67.0	37.0, 84.3
University	70.0	65.2 (10.6)[c]	66.3	30.2, 88.5	120	61.8 (10.5)	63.3	31.5, 79.2	192	67.2 (10.1)[b,c]	68.5	30.2, 88.5
Gross household income												
0–$59 999	8.7	63.1 (10.4)	63.0	43.3, 84.2	18	64.7 (10.6)	64.7	43.3, 79.2	21	61.7 (10.3)	59.6	45.0, 59.6
$60 000–$119 999	33.9	63.5 (10.7)	65.5	31.4, 85.1	61	59.0 (10.6)	60.1	31.4, 79.2	90	66.6 (9.6)[b]	68.2	31.4, 79.2
≥ $120 000	43.3	65.7 (10.7)	67.0	30.2, 88.5	76	62.6 (10.4)	63.5	31.4 ,78.9	117	67.7 (10.5)[b]	68.9	30.2, 88.5
Refused	14.1	65.6 (10.1)	62.8	40.5, 84.8	17	62.3 (10.0)	58.9	3.5, 75.7	46	64.0 (11.5)	63.2	40.5, 84.8
Smoking status												
Non-smoker	6.4	64.7 (10.6)[d]	65.5	30.2, 88.5	166	61.8 (10.6)	63.0	31.4, 79.2	264	66.6 (10.3)[b,d]	67.5	30.2, 88.5
Smoker	.6	56.9 (11.8)	54.2	36.9, 73.2	6	54.7 (8.6)	51.6	44.6, 67.0	10	58.2 (13.7)	61.8	36.9, 73.2
Race												
White	93.5	64.4 (10.8)	65.3	30.2, 88.5	5	61.3 (10.8)	62.9	31.4, 79.2	14	66.2 (10.4)[b]	67.5	30.2, 88.5
All other	6.5	65.4 (10.0)	66.1	40.0, 79.3	57	63.8 (7.8)	64.2	49.6, 76.6	260	67.1 (12.0)	69.7	40.0, 73.3

Abbreviations: C-HEI, Canadian Healthy Eating Index; SD, standard deviation.
[a] Significant difference by sex $p < .05$, independent t-test.
[b] Significant difference by sex within socio-demographic groups $p < .05$, ANOVA post-hoc bonferonni.
[c] Significant $p < .05$ linear trend across socio-demographic groups, ANOVA post-hoc test.
[d] Significant difference between socio-demographic groups $p < .05$, ANOVA post-hoc bonferonni.

TABLE 3
Distribution of daily reported intake of food group equivalents (in Canada's Food Guide servings), Canadian Healthy Eating Index component scores and energy, by sex

	Total (n = 446)				Men (n = 172)				Women (n = 274)			
CFG serving	Mean (SD)	Median	Min, max	CFG range[a]	Mean (SD)	Median	Min, max	CFG range[a]	Mean (SD)	Median	Min, max
Total vegetables and fruits	6.3 (3.2)	5.7	0.7, 22.8	7–10	6.3 (3.3)	5.5	1.6, 22.8	7–8	6.3 (3.2)	5.8	0.7, 19.1
Whole fruits	1.8 (1.1)	1.7	0.0, 5.9		1.7 (1.1)	1.4	0.1, 5.9		1.9 (1.1)	1.7	0.0, 5.8
Dark green and orange vegetables	2.0 (1.8)	1.5	0.0, 15.8		1.6 (1.2)	1.3	0.2, 7.7		2.2 (2.1)	1.6	0.0, 15.8
Total grains	3.2 (1.9)	3.0	0.1, 12.8	7–8	3.9 (2.2)	3.6	0.4, 12.8	6–7	2.8 (1.6)	2.6	0.1, 9.11
Whole grains	0.4 (0.4)	0.3	0.0, 2.7	3.5–4	0.5 (0.5)	0.4	0.0, 2.7	3–3.5	0.4 (0.4)	0.3	0.0, 2.6
Milk and alternatives	1.6 (1.2)	1.3	0.0, 7.0	2–3	1.7 (1.1)	1.4	0.1, 1.4	2–3	1.5 (1.2)	1.2	0.0, 7.0
Meat and alternatives	1.9 (1.1)	1.6	0.1, 10.3	2–3	2.1 (1.3)	1.8	0.3, 10.3	2	1.7 (1.0)	1.5	0.1, 9.4
Sodium (mg)	2344 (922)	2158	570, 7384		2690 (1180)	2373	723, 7384		2128 (781)	2004	570, 4893
Saturated fat (g)	19.9 (9.4)	17.6	4.2, 64.7		22.5 (10.2)	18.5	5.4, 64.7		18.6 (8.5)	16.3	4.2, 60.9
Energy (calories)	1650 (717)	1545	483, 4514		1989 (717)	1734	538, 4514		1440 (505)	1433	483, 3899
Calories from other foods	339 (219)	283	21, 1926		422 (253)	356	43, 1721		287 (177)	255.3	21, 1926
Calories from other foods (%)	20.3 (8.8)	19.1	3.3, 55.8		22.1 (9.6)	21.1	3.7, 55.8		19.1 (8.1)	17.7	3.3, 49.5
C–HEI components scores	**Mean (SD)**	**Median**	**Min, max**	**Scale**	**Mean (SD)**	**Median**	**Min, max**	**Scale**	**Mean (SD)**	**Median**	**Min, max**
Total vegetables and fruits	7.6 (2.4)	7.8	0.8, 10.0	0–10	7.4 (2.5)	7.6	2.1, 10.0	0–10	7.7 (2.3)	8.1	0.8, 10.0
Whole fruits	4.1 (1.3)	5.0	0.1, 5.0	0–5	3.9 (1.4)	4.5	0.2, 5.0	0–5	4.3 (1.2)	5.0	0.1, 5.0
Dark green and orange vegetables	3.9 (1.4)	4.8	0.1, 5.0	0–5	3.7 (1.5)	4.0	0.5, 5.0	0–5	4.0 (1.3)	5.0	0.1, 5.0
Total grains	2.4 (1.2)	2.3	0.1, 5.0	0–5	2.6 (1.2)	2.5	0.3, 5.0	0–5	2.3 (1.2)	2.1	0.1, 5.0
Whole grains	0.6 (0.6)	0.5	0.0, 4.3	0–5	0.7 (0.6)	0.5	0.0, 3.7	0–5	0.6 (0.6)	0.4	0.0, 4.3
Milk and alternatives	5.5 (3.1)	5.2	0.0, 10.0	0–10	5.5 (3.0)	5.4	0.3, 10.0	0–10	5.4 (3.1)	5.1	0.0, 10.0
Meat and alternatives	6.9 (2.5)	6.9	0.7, 10.0	0–10	6.3 (2.5)	6.0	1.1, 10.0	0–10	7.3 (2.5)[b]	7.6	0.7, 10.0
Unsaturated fats	8.5 (1.9)	10.0	1.8, 10.0	0–10	7.9 (2.2)	8.4	1.8, 10.0	0–10	8.9 (1.7)[b]	10.0	2.8, 10.0
Saturated fats	6.3 (2.9)	7.0	0.0, 10.0	0–10	6.7 (2.6)	7.4	0.0, 10.0	0–10	6.0 (3.0)	6.7	0.0, 10.0
Sodium	7.3 (2.6)	8.3	0.0, 10.0	0–10	6.5 (3.0)	7.7	0.0, 10.0	0–10	7.9 (2.1)[b]	8.5	0.0, 10.0
Other foods	11.4 (4.7)	12.0	0.0, 20.0	0–20	10.5 (4.9)	10.8	0.0, 20.0	0–20	12.0 (4.5)[b]	12.7	0.0, 10.0

Abbreviations: CFG, Eating Well with Canada's Food Guide; C-HEI, Canadian Healthy Eating Index; SD, standard deviation.
Note: CFG range = recommended number of food guide servings per day, adults 19 years and older.
[a] Canada's Food Guide recommendation range for adults 19 years and older.
[b] Significant difference by sex $p < .05$.

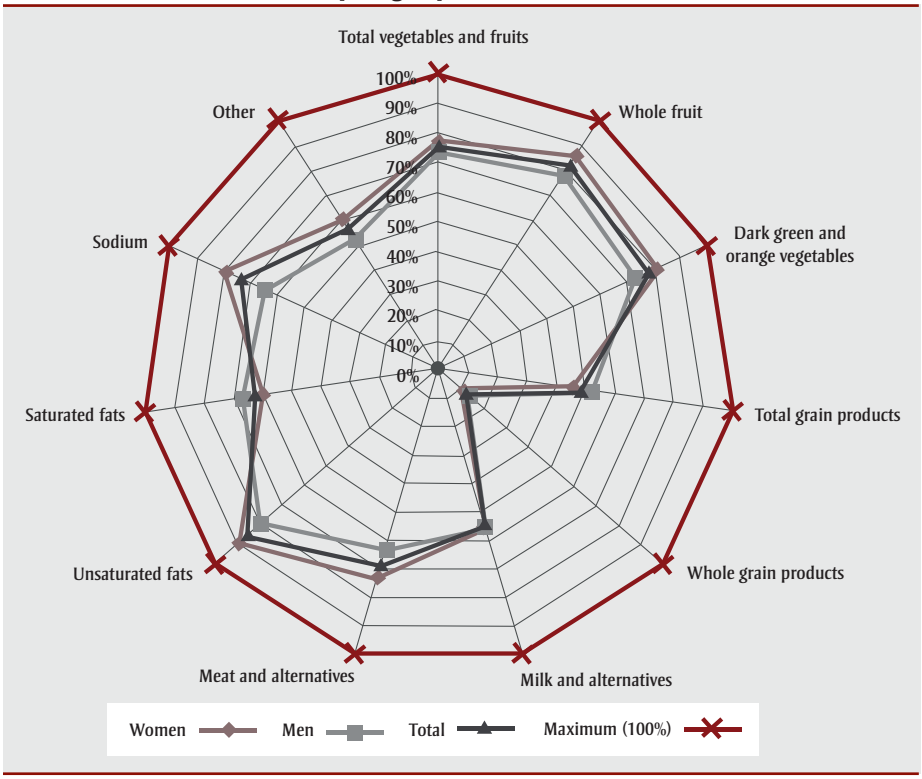
the highest scoring components were unsaturated fat, whole fruits, dark green and orange vegetables, and total fruits and vegetables.

Discussion

We describe methods for creating food group equivalents (CFG serving variables) for the C-DHQ II nutrient database estimation of the C-HEI. Algorithms were developed to create the food group equivalent variables and applied to the most commonly consumed foods from the Canadian Community Health Survey food and recipes file which were linked to food questions on the C-DHQ II. This approach was an efficient and robust strategy since the C-DHQ II food category nutrient profiles were weighted most heavily on foods that contributed substantially to Canadian diets[15] and hence were foods that were most representative of C-DHQ II food categories. The mean C-HEI in the participants from the Pathways to Health study was comparable, albeit somewhat higher, than values previously reported for the Canadian population.[8] Consistent with findings from previous Canadian data[8] we found differences in the C-HEI by sex, smoking status, and education.

The range and normal distribution of the mean daily food group equivalent intakes observed suggest that the method applied yielded reasonable estimates of intake given their similarities to previous reports in the Canadian population.[8] The intakes of the number of total fruits and vegetables,

FIGURE 3
Radar graph of average daily C-HEI component scores for total sample and by sex, comparing to perfect C-HEI score



whole fruits, dark green and orange vegetables, and meat servings were slightly left skewed which is expected given that North American diets tend to be high in animal protein[27,28] and over-reporting of fruit and vegetable consumption is common due to social desirability.[29] Consistent with previously reported Canadian dietary patterns, women reported a higher median number of fruit and vegetable servings per day compared with men[30,31], while men reported a higher median number of meat and alternatives servings per day.[30] Intake of grains and whole grains as measured by mean daily CFG equivalent intake was low. A potential explanation for low total grain and whole grains intakes may be the increase in non-celiac gluten sensitivity[32] and a trend toward the adoption of gluten-free and low-carbohydrate diets[33–35] which can limit the intake of grain products. Finally, the food group equivalents intake distributions were similar to what has been previously observed in Alberta.[20]

The mean daily C-HEI score (64.4) was somewhat higher than what has been previously observed nationally (58.8)[8] and in Alberta (men: 51; women: 56).[20] The differences between the C-HEI scores reported in this study population and those reported by others likely reflect differences in the study sample designs and data collection strategies. Nevertheless, the differences in C-HEI scores across levels of socio-demographic characteristics in our study were consistent with findings from another Canadian study that reported differences in C-HEI by sex, smoking status, and education level.[8] For the C-HEI component scores, with the exception of total vegetables and fruits, meat and alternatives, and whole grains, all possible minimum and maximum values were observed, demonstrating that the primary food method allowed a full spectrum of component scores to be obtained.

Limitations

Underreporting is known to exist with most methods of dietary assessment[36,37] and is expected to have played a role in our study.[38] Food frequency questionnaires have been observed to be associated with substantial energy intake underreporting when compared with the objective measure doubly labelled water[38–40]; and hence underreporting is also expected with the C-DHQ II. Underreporting of the C-DHQ II likely led to the underestimation of overall energy intake. However, evidence suggests there is also differential misreporting by food type on food frequency questionnaires.[41,42] For example, vegetables and fruits tend to be overestimated while sugars, sweets, jams and some grain products are underestimated.[42] Additionally, social desirability response bias is plausible given that those who chose to respond to the questionnaire may have underreported food groups labeled as 'unhealthy' in popular media (e.g. grain products). Hence, dietary misreporting may lead to the under- or over-estimation of C-HEI adequacy and moderation components, and total C-HEI scores. Although the average daily food group equivalents and C-HEI estimates in the current study are generally consistent with existing CFG food group servings[30] and C-HEI distributions[8], some estimates may be low (particularly grain and whole grain estimates). This could be an artefact of not being able to identify the primary food given the lack of name brands and the difficulty of estimating the grain component of mixed foods or using only the most commonly consumed foods reported in the Canadian Community Health Survey and linked to food questions on the C-DHQ II.

The cut-off values for confirming sufficient representativeness of the primary foods (< 5% difference) and the difference in compositions of mixed foods using the US MPED and FPEDs (~20%) were determined by the authors. If the cut-offs were too liberal or conservative, it could have resulted in misclassification of the C-DHQ II food group into the C-HEI variables.

Conclusion

The addition of food group equivalents to the C-DHQ II nutrient database allows researchers to compare average daily food group intakes to the healthy eating recommendations of the CFG. Further, the derivation of the C-HEI allows Canadian researchers to examine the relation between diet quality and chronic disease risk using a questionnaire designed specifically for Canadian populations.

Acknowledgements

This research was supported by funds to the Canadian Research Data Centre Network (CRDCN) from the Social Sciences and Humanities Research Council (SSHRC), the Canadian Institutes of Health Research

(CIHR), the Canadian Foundation for Innovation (CFI), and Statistics Canada.

Stipend support to MM funded by the CIHR Training Grant in Population Intervention for Chronic Disease Prevention: A Pan-Canadian Program (Grant #53893); CIHR Canada Graduate Student–Masters Award; and the William H Davies Medical Research Scholarship. GRM is supported by a CIHR New Investigator award. AK was supported by the Fonds de recherche du Québec-Santé (FRQS) Research Scholar Program and the CIHR New Investigator program.VH received a postdoctoral fellowship from the CIHR and Lung Cancer Canada to conduct this work and is currently supported by the Cancer Research Society, FRQS and Ministère de l'Économie, de la Science et de l'Innovation du Québec (MESI).

Conflicts of interest

The authors have no conflicts of interest to disclose.

Authors' contributions and statement

MM/IC conceived the methodologies and algorithms used to create Canada's Food Guide variables in the Canadian Diet History Questionnaire II nutrient database. MM led the Canada's Food Guide variable creation. IM/IR/AK/VH reviewed the algorithms and methodologies. VH modified the SAS code for deriving the Canadian Healthy Eating Index from Canadian Diet History Questionnaire II data. IM/IR conducted quality checks of the newly created variables. GRM advised on the analysis. MM led the writing, drafting, and editing of the manuscript. All authors contributed to the interpretation of findings and writing of the manuscript. All authors read and approved the final manuscript.

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada. Although the research and analysis are based on data from Statistics Canada, the opinions expressed do not represent the views of Statistics Canada.

References

1. Alkerwi A. Diet quality concept. Nutrition. 2014;30(6):613–8. doi: 10.1016/j.nut.2013.10.001.
2. Anic GM, Park Y, Subar AF, Schap TE, Reedy J. Index-based dietary patterns and risk of lung cancer in the NIH–AARP diet and health study. Eur J Clin Nutr. 2016;70(1):123-9. doi: 10.1038/ejcn.2015.122.
3. Reedy J, Mitrou PN, Krebs-Smith SM, Wirfält E, Flood A, Kipnis V, et al. Index-based dietary patterns and risk of colorectal cancer the NIH-AARP diet and health study. Am J Epidemiol. 2008;168(1):38–48. doi: 10.1093/aje/kwn097.
4. Bosire C, Stampfer MJ, Subar AF, Park Y, Kirkpatrick SI, Chiuve SE, et al. Index-based dietary patterns and the risk of prostate cancer in the NIH-AARP diet and health study. Am J Epidemiol. 2013;177(6):504–13. doi: 10.3945/an.115.009746.
5. Chiuve SE, Fung TT, Rimm EB, Hu FB, McCullough M, Wang M, et al. Alternative dietary indices both strongly predict risk of chronic disease. J Nutr. Am Soc Nutrition. 2012;142(6):1009–18. doi: 10.3945/jn.111.157222.
6. Liese AD, Krebs-Smith SM, Subar AF, George SM, Harmon BE, Neuhouser ML, et al. The Dietary Patterns Methods Project: synthesis of findings across cohorts and relevance to dietary guidance. J Nutr. 2015;145(3):393–402.
7. Krebs-Smith SM, Subar AF, Reedy J. Examining dietary patterns in relation to chronic disease: mMatching measures and methods to questions of interest. Circulation. 2015;132:790–3. doi:10.1161/CIRCULATIONAHA.115.018010.
8. Garriguet D. Diet quality in Canada. Health Rep. 2009;20(3):41–52.
9. Kennedy ET, Ohls J, Carlson S, Fleming K. The healthy eating index: design and applications. J Am Diet Assoc. 1995;95(10):1103–8. doi: 10.1016/S0002-8223(95)00300-2.
10. Dubois L, Girard M, Bergeron N. The choice of a diet quality indicator to evaluate the nutritional health of populations. Public Health Nutr. 2000;3(3):357–65.
11. Glanville NT, McIntyre L. Diet quality of Atlantic families headed by single mothers. Can J Diet Pract Res. 2006;67(1):28–35. doi: 10.3148/67.1.2006.28.
12. Shatenstein B, Nadon S, Godin C, Ferland G. Diet Quality of Montreal-Area Adults Needs Improvement: Estimates from a Self-Administered Food Frequency Questionnaire Furnishing a Dietary Indicator Score. J Am Diet Assoc. 2005;105(8):1251–60.
13. Woodruff SJ, Hanning RM, McGoldrick K, Brown KS. Healthy Eating Index-C is positively associated with family dinner frequency among students in grades 6–8 from Southern Ontario, Canada. Eur J Clin Nutr. 2010;64(5):454–60. doi: 10.1038/ejcn.2010.14.
14. Health Canada. Eating Well with Canada's Food Guide [Internet]. 2007 [cited 2013 Oct 23]. Available from: http://www.hc-sc.gc.ca/fn-an/food-guide-aliment/order-commander/index-eng.php
15. Office of the Nutrition and Promotion Health Products and Food Branch - Health Canada. Canadian Community Health Survey Cycle 2.2, Nutrition (2004); a guide to accessing and interpreting the data. 2006.
16. Diet History Questionnaire II. Canadian DHQ II (C-DHQ II). C-DHQ II Forms and Diet Calc Files. 2015. Available from: https://www.canadiandhqii.com/survey_resources.html
17. Csizmadi I, Boucher B, Lo Siou G, Massarrelli I, Rondeau I, Koushik A, et al. Using national dietary intake data to evaluate and adapt the US Diet History Questionnaire: the stepwise tailoring of an FFQ for Canadian populations. Public Health Nutr. 2016;19(18):3247-3255. doi: 10.1017/S1368980016001506.
18. Lo Siou G, Csizmadi I, Boucher BA, et al. The comparative reliability and feasibility of the past-year Canadian Diet History Questionnaire II: comparison of the paper and web versions. Nutrients. 2017;9(2):E133. doi: 10.3390/nu9020133.
19. Awadalla P, Boileau C, Payette Y, Idaghdour Y, Goulet J-P, Knoppers B, et al. Cohort profile of the CARTaGENE study: Quebec's population-based biobank for public health and personalized genomics. Int J Epidemiol. 2013;42(5):1285–99. doi: 10.1093/ije/dys160.

20. Solbak NM, Xu JY, Vena JE, Csizmadi I, Whelan HK, Robson PJ. Diet quality is associated with reduced incidence of cancer and self-reported chronic disease: observations from Alberta's Tomorrow Project. Prev Med. 2017;101:178-187. doi: 10.1016/j.ypmed.2017.06.009.

21. McInerney M, Csizmadi I, Friedenreich CM, Nettle-Aguirre A, Sandalack BA, Potestio M, et al. Associations between neighbourhood food environment, neighbourhood socioeconomic status, and diet quality. BMC Public Health. 2016;16(1):984-999. doi: 10.1186/s12889-016-3631-7.

22. McInerney, M. Associations between the Neighbourhood Food Environment, Neighbourhood Socioeconomic Status and Diet Quality in Canadian Adults. Calgary, Alberta: University of Calgary; 2016. Available from: http://hdl.handle.net/11023/2848.

23. Elvidge Munene L-A, Dumais L, Esslinger K, Jones-Mclean E, Mansfield E, Verreault M-F, et al. A surveillance tool to assess diets according to Eating Well with Canada's Food Guide. Health Rep. 2015;26(11):12–20. doi: 10.1186/s12889-016-3631-7.

24. Health Canada. The Development and Use of a Surveillance Tool: The Classification of Foods in the Canadian Nutrient File According to Eating Well with Canada's Food Guide. 2014. Available from: http://publications.gc.ca/site/archivee-archived.html?url=http://publications.gc.ca/collections/collection_2014/sc-hc/H164-158-2-2014-eng.pdf

25. Bowman S, Clemens J, Friday J, Thoerig R, Moshfegh A. Food Patterns Equivalents Database 2011-12: Methodology and User Guide. Beltsville. 2014. Available from: https://www.ars.usda.gov/ARSUserFiles/80400530/pdf/fped/FPED_1112.pdf

26. Health Canada. Canadian Nutrient File (CNF) 2001b. 2001. Available from: https://www.canada.ca/en/health-canada/services/food-nutrition/healthy-eating/nutrient-data/canadian-nutrient-file-2015-download-files.html

27. Statistics Canada. Nutrition: Findings from the Canadian Community Health Survey (82-620-M). 2007. Available from: http://www.statcan.gc.ca/pub/82-620-m/2006002/4053669-eng.htm

28. Daniel CR, Cross AJ, Koebnick C, Sinha R. Trends in meat consumption in the United States. Public Health Nutr. 2011;14(4):575–83. doi: 10.1017/S1368980010002077.

29. Miller TM, Abdel-Maksoud MF, Crane LA, Marcus AC, Byers TE. Effects of social approval bias on self-reported fruit and vegetable consumption: a randomized controlled trial. Nutr J. 2008;7(18). doi: 10.1186/1475-2891-7-18.

30. Garriguet D. Canadians' eating habits. Health Rep. 2007;18(2):17–32.

31. Pérez CE. Fruit and vegetable consumption. Health Rep. 2002;13(3):23–31.

32. DiGiacomo D V, Tennyson CA, Green PH, Demmer RT. Prevalence of gluten-free diet adherence among individuals without celiac disease in the USA: results from the Continuous National Health and Nutrition Examination Survey 2009-2010. Scand J Gastroenterol. 2013;48(8):921–5. doi: 10.3109/00365521.2013.809598.

33. Volland A. Gluten-free diet: a cure for some, a fad for most. U.S. news and world report. 2008;145(12):66-68.

34. Cadenhead K, Sweeny M. Gluten elimination diets: facts for patients on this food fad. BCMJ. 2013;55:161.

35. Moore LR. "But we're not hypochondriacs": The changing shape of gluten-free dieting and the contested illness experience. Soc Sci Med. 2014;105:76–83. doi: 10.1016/j.socscimed.2014.01.009.

36. Grandjean AC. Dietary intake data collection: challenges and limitations. Nutr Rev. 2012;70 Suppl 2:S101–4. doi: 10.1111/j.1753-4887.2012.00545.x.

37. Shim J-S, Oh K, Kim HC. Dietary assessment methods in epidemiologic studies. Epidemiol Health. 2014;36:e2014009. doi: 10.4178/epih/e2014009.

38. Subar AF, Kipnis V, Troiano RP, Midthune D, Schoeller DA, Bingham S, et al. Using intake biomarkers to evaluate the extent of dietary misreporting in a large sample of adults: the OPEN study. Am J Epidemiol. 2003;158(1):1–13.

39. Prentice RL, Mossavar-Rahmani Y, Huang Y, Van Horn L, Beresford SAA, Caan B, et al. Evaluation and comparison of food records, recalls, and frequencies for energy and protein assessment by using recovery biomarkers. Am J Epidemiol. 2011;174(5):597-603.

40. Freedman LS, Commins JM, Moler JE, Arab L, Baer DJ, Kipnis V, et al. Pooled results from 5 validation studies of dietary self-report instruments using recovery biomarkers for energy and protein intake. Am J Epidemiol. 2014;180(2)172-88. doi: 10.1093/aje/kwu116.

41. Streppel MT, de Vries JHM, Meijboom S, Beekman M, de Craen AJM, Slagboom PE, et al. Relative validity of the food frequency questionnaire used to assess dietary intake in the Leiden Longevity Study. Nutr J. 2013;12(1):1–8. doi: 10.1186/1475-2891-12-75.

42. Marks GC, Hughes MC, van der Pols JC. Relative validity of food intake estimates using a food frequency questionnaire is associated with sex, age, and other personal characteristics. J Nutr. 2006;136(2):459–65.

https://doi.org/10.24095/hpcdp.38.3.04

The HANS KAI Project: a community-based approach to improving health and well-being through peer support

Alexandra Henteleff, MEd; Helena Wall, MEd

This original mixed methods research article has been peer reviewed.

Abstract

Introduction: HANS KAI is a unique health promotion intervention to improve participants' health by focussing on interrelated chronic disease prevention behaviours through peer support and strengthening of social support networks. The study objective was to determine the effectiveness of HANS KAI in an urban Canadian setting.

Methods: We used a mixed methods intervention research design that involved multiple sites from November 2010 to April 2015. Data was obtained from participant surveys as well as in-person interviews at zero, 6, 12 and 24 months. Participants met in groups at least once a month during the research period, to self-monitor health indicators, prepare and share a healthy snack, participate in a physical activity, set a healthy lifestyle goal (optional) and socialize.

Results: There were statistically significant mental health improvements from pre- to post-program, and 66% of the participants described specific behaviour changes as a result of HANS KAI participation. Additional positive health impacts included peer support; acquiring specific health knowledge; inspiration, motivation or accountability; the empowering effect of monitoring one's own health indicators; overcoming social isolation and knowing how to better access services.

Conclusion: The need to identify innovative ways to address chronic disease prevention and management has been the driver for implementing and evaluating HANS KAI. While further research will be required to validate the present findings, it appears that HANS KAI may be an effective approach to create environments that empower community members to support each other while promoting healthy lifestyle choices and detecting early changes in health status.

Keywords: *health promotion, chronic disease, prevention, social support, peer group, self-help groups, peer support*

Highlights

- Innovative methods are needed to address chronic disease prevention and management in socioeconomically challenged communities.
- HANS KAI harnesses peer support to empower community members to support each other while promoting healthy lifestyle choices that address interrelated chronic disease prevention behaviours and detecting early health changes.
- HANS KAI is unique as it focusses on health where most peer support interventions are disease specific.
- Participation in HANS KAI resulted in statistically significant improvement in mental health scores.
- Participants also reported decreased social isolation, healthy behaviour change, increased knowledge of and access to services, and empowerment from self-monitoring personal health indicators.

Introduction

This paper reports on an innovative community-based health promotion intervention offered by a Manitoba-based health care co-operative that explored the relationship between peer support and perceptions of wellness. Peer support was defined as "a system of giving and receiving help founded on key principles of respect, shared responsibility, and mutual agreement of what is helpful."[1,p.137] It is about understanding another's situation empathically through shared experience. As trust in their relationship builds, people are able to respectfully challenge each other, try out new behaviours with one another and move beyond previously held self-concepts. This is referred to as mutual empowerment.[1]

Peer support has been used to help individuals adjust to life-transitioning changes, such as the birth of a child, significant losses or long-term disabilities / chronic diseases, and in health promotion initiatives, including support for health behaviour changes.[2-4] Ford et al. stated that peer support programs "are emerging as highly effective and empowering ways for people to manage health issues"[5,p.5507] in a socially supportive context. We found no documented examples in the literature of Canadian community-based initiatives using a peer support model to create social networks to support personal wellness that did not primarily have a disease-specific focus.

Intervention

HANS KAI is a unique health promotion intervention to improve the health of participants through peer support and strengthening of social support networks. HANS KAI is modelled after a *han*, a style

Author reference:

Community Affiliated Nurse Researcher, University of Manitoba, Winnipeg, Manitoba, Canada

Correspondence: Alexandra Henteleff, Community Affiliated Nurse Researcher, University of Manitoba, 1469 Wellington Cres South, Winnipeg, MB R3N 0B6; Tel: 204-793-8759; Email: ahenteleff@gmail.com

of health management started by Japanese health cooperatives in the mid-1950s to encourage people to take responsibility for their own health and to promote preventive medicine.[6] In Japanese, *han* means "group" and *kai* means "assembly" or "meeting." The han group approach was intended to take the burden off primary care, and encourage peer support and personal empowerment.[6] HANS KAI groups (HANS groups) typically consist of about 10 members matched on characteristics such as age, community area and availability. Integral to this intervention is the idea that people who spend time together and monitor their health will live longer, healthier lives.[7]

Social learning theory[8] and empowerment theory[9] form the theoretical foundation for the HANS KAI intervention. Social learning theory explains human behaviour as a continuous interaction between cognitive, behavioural and environmental influences: people learn through observing others' behaviour and attitudes as well as the outcomes of the behaviour.[7] Like Bandura's social learning theory,[8] empowerment includes the concept of self-efficacy, where perceptions of competence, personal control and positive self-image support individuals to think positively about their ability to effect change and have mastery over issues.[9] The empowerment approach "redefines the professional's role … to one of collaborator … [where] participants have an active role in the change process."[9,p.44-45]

A review of the literature did not produce any research on the effectiveness of peer support groups to improve the health of participants without a specific disease focus. We undertook this research project to implement and evaluate the effectiveness of HANS KAI in an urban Canadian setting. In our program design, peers in HANS groups were fellow group members. Participants would attend a "Health School" at enrolment to receive baseline knowledge about a variety of health topics as well as training on how to take their own health measures and work independently as a group. Participants then would meet in small groups at least once a month for about two hours to learn about health topics from each other and from health care providers, participate in physical exercise, make and share a nutritious snack, monitor and record their health measures in logbooks, and have time to socialize.

Setting

NorWest Co-op Community Health (NorWest) is committed to engaging its community in cooperative health and wellness with a vision of people taking control of their health. NorWest is situated in the Inkster community of Winnipeg, a socioeconomically challenged community where 20% of families live below the low-income cut-off.[10] The area lacks recreational facilities, and, in a 2008 report, 86% of residents felt that social support was key to changing behaviours related to eating and physical activity, and that it was the most important factor for joining healthy lifestyle programs.[11] NorWest decided that HANS KAI matched their vision, mission and values, and wanted to see if implementing HANS KAI was feasible and beneficial.

Research objectives

The purpose of this research study was to determine if structured, peer-led community groups could be successfully initiated within an urban Canadian context and have a positive impact on participant health and well-being. The objectives of the intervention were to:

1. create social and peer support networks to increase participants' ability to make healthier lifestyle choices to support personal wellness, empower participants to take action to improve their health and develop connections with others to reduce social isolation;
2. increase participants' awareness of the connection between personal wellness, healthy lifestyle choices, healthy weights and those factors within and outside of their control;
3. maintain or improve measurable health indicators; and
4. increase access to primary care services and other community programs/services.

Methods

Research design

We used a participatory community-based design, involving community members during each stage of the research. A community board sought an investigator through a solicitation of interest (SOI), to explore the impact of participation in a HANS group on a range of health outcomes. The community board reviewed the responses to the SOI, selected, then met with the research team and had input in the final design. NorWest staff had the primary role to support the implementation of HANS and work closely with the researchers during the research processes. Members in pre-existing community groups participated in piloting the HANS KAI model and provided invaluable feedback to guide the finalization of the research tools. Preliminary results were shared via presentations to the community board and the HANS groups, and at community events and the NorWest AGM in June 2017.

A mixed methods intervention research design, involving multiple sites, was used as its flexible form of inquiry captured multiple perspectives about, and promoted a more complete understanding of, the intervention experience.[12]

Quantitative pre-test, post-test data were obtained from participant surveys and data were entered into a purpose-built database. Qualitative findings were obtained through one-on-one interviews with participants. Data collection took place from November 2010 to April 2015. The survey and interviews were done at 0 (baseline), 6, 12 and 24 months. The study received written approval from the University of Manitoba's Education/Nursing Research Ethics Board, protocol #E2010:102.

Recruitment

A variety of recruitment techniques were used, including community or workplace presentations; mail-outs to NorWest clients; health care provider referrals; posters located in areas where people congregate (grocery stores, pharmacies, community centres and medical clinics); and in-person recruitment through HANS awareness presentations at community events and groups. Inclusion criteria were: resident of the Inkster or Seven Oaks community areas of Winnipeg, 18 years of age or over, able to speak and read English, and be in relatively good health, including individuals with chronic conditions as long as the condition was stable.

A targeted approach was used to enrol participants who were socially isolated and/or economically challenged such as seniors, new mothers and new Canadians. Individuals who wished to participate in HANS had to complete an application form and were then assigned into either

pre-existing or new groups, according to age, community area and availability.

Group participants attended a HANS KAI "Health School," which consisted of six 2-hour face-to-face sessions to help participants learn how to monitor their own health and work independently as a group. Sessions supported the concept that many factors influence health. Topics included health indicators, chronic disease, nutrition, physical activity, sleep, stress, general health, primary care through the years, medications, supplements, smoking, social supports and how to work effectively as a group. It was mandatory for participants to attend Health School to ensure all participants had the same information, knowledge and engagement.

HANS groups were designed to be participant led and consisted of 8 to 15 people, with support and guidance from NorWest health professionals as needed. Access issues were assessed during the research period and steps taken to mitigate economic challenges to participation, for example, by providing child care or free exercise sessions, and encouraging carpooling. The groups met at least once a month for 1.5- to 2-hour sessions. Some met more often. Each session had required components including: 1) monitor health indicators; 2) prepare a healthy snack; 3) participate in physical activity; 4) share action plan for the month (set a lifestyle goal) – *optional*; 5) exchange contact information with a "buddy" to do regular check-ins; and 6) socialize.

Thirteen groups were started over a three-year enrolment period. While some groups succeeded at meeting regularly throughout the research period, others only met briefly and were unable to achieve the group cohesiveness necessary to continue meeting. Nine groups (and their members, n = 77) were included in this research. Criteria for inclusion were groups that met regularly and participated in at least three of the data collection periods within the time frame of the research (0, 6, 12, 24 months). Seven groups participated in community venues and two were workplace groups. Participant attendance at in-person interviews varied between groups (Table 1).

NorWest staff suggested a variety of reasons for the dropouts, e.g. that the group was not a good fit for the individual, changing demographics, returning to work, workplace limitations, changing needs of individuals, time-stressed families, people getting what they needed from the participation and leaving, and people moving.

TABLE 1
HANS KAI participant attendance for in-person interviews (2010-2015)

	Group type	Enrolment	Participated in 3 or more data collection periods
Group 1	Community	5	5
Group 2	Community	7	3
Group 3	Community	12	4
Group 4	Community	12	7
Group 5	Workplace	9	8
Group 6	Community	5	5
Group 7	Community	7	6
Group 8	Workplace	10	8
Group 9	Community	10	7
Total		**77**	**53**

Data collection

Data collection was accomplished using a participant survey and individual face-to-face interviews. Participant logbooks tracked selected health indicators but were used inconsistently and did not provide sufficient quantitative data for analysis.

HANS KAI participant survey

A comprehensive questionnaire, adapting validated instruments from multiple sources,[13-19] was developed specifically for this study to measure the effects of HANS group participation on the following health-related topics:

- knowledge of diabetes and hypertension;
- nutrition, physical activity and sleep self-assessments;
- smoking status;
- mental health status;
- access to health care providers;
- awareness of community programs and services;
- understanding of how to improve overall health; and
- connectedness to people in the neighbourhood and community.

The project questionnaire, which was developed to maximize content validity and reliability, was edited and modified by a multidisciplinary expert group with experience and expertise in community mobilization and service delivery.

NorWest staff distributed and collected the completed surveys, consisting of baseline surveys when newly enrolled participants attended their first Health School session (Time 1), and follow-up surveys at 6, 12 and 24 months. Data from the completed surveys were entered into a database by NorWest support staff.

Individual interviews

The researchers interviewed all participants at baseline (Time 1), with follow-up interviews taking place at 6, 12 and 24 months. The baseline interview included two questions about participant perception of factors that had a positive or negative impact on health. The follow-up interviews included the two original questions plus questions intended to elicit feedback about the perceived impact of regular participation in a HANS group and ways to improve HANS KAI. The baseline interviews were documented by the interviewers on paper and then transcribed; however, all follow-up interviews were audio recorded and then transcribed for analysis. The interview questions and schedule are presented in Table 2.

Baseline interviews (Time 1) were scheduled at the first HANS group meeting of each group following group initiation. Although follow-up interviews were planned at 6 and 12 months after the study began, not all participants could be interviewed at all time intervals due to participant

TABLE 2
Individual interview questions for HANS KAI participants

Question	Baseline interviews[a]	Follow-up interviews[b]
1. In the last 6 months, what has helped or made it easier to stay healthy and feel good?	✓	✓
2. In the last 6 months, what has not helped or made it harder for you to stay healthy and feel good?	✓	✓
3. Since joining the HANS group, have you received support from a member of the group? If yes, describe.		✓
4. Since joining the HANS group, have you provided support to a member of the group? If yes, describe.		✓
5. How has being part of a HANS group helped you to improve your health or stay healthy?		✓
6. What did you like best about the HANS program?		✓
7. What didn't you like about the HANS program?		✓
8. What changes would make it better?		✓

[a] Baseline interviews (pre-intervention).
[b] Follow-up interviews at 6, 12 and 24 months.

attendance issues and the inability to arrange meeting times. Consequently, the researchers were unable to obtain interviews at all three time intervals for most participants; however, responses were tracked for 53 individuals who participated in a baseline interview (Time 1) and at least two follow-up interviews (Time 2 and Time 3) at either 6, 12 or 24 months.

Data analysis

Survey

A *t* test was the test statistic used to perform the pre/post analysis of the survey data with alpha (or *p*) set at .05. This analysis approach was modified from the intent-to-treat approach often used in clinical trials.[20] A *z* test was used to compare the demographic characteristics between the participants who completed the project and those who did not complete the project and were lost to follow-up.

Interviews

A thematic analysis of all interview responses was conducted. The principal investigators separately analyzed data transcripts of the first 48 baseline interviews and the first 24 follow-up interviews. They then generated initial codes to search for, define and name themes, the outcome of which was a coding template. A principal investigator and research assistant then independently reviewed and coded all transcripts; together they discussed their separate analyses and reached agreement on the interpretation of the data.

Results

Demographic data

Client demographics were obtained from 63 participants though not all participants answered every question. Participants were primarily female (60/63) with an age range from 20 to 72 years (Figure 1). Of those who answered the question, marital status was equally split between married/common law (30/63) and single/widowed/divorced/separated (30/63). Thirty-seven percent (23/63) had children living at home, 52% (33/63) had no children living at home, and half of the participants (28/63) lived alone. Thirty-five percent had community college/university education, and 41% were high school graduates. Almost one-third of the participants had lived in their community for at least 25 years (Figure 2), more than one-third were unemployed (Figure 3), and a similar number reported an annual household income of less than $40,000 (although 38% [24/63] of the participants did not answer the last question; Figure 4).

The *z* test that compared the demographic data, identified statistically significant differences between the participants who completed the project and those who did not complete the project and were lost to follow-up. Those lost to follow-up were less educated and had higher unemployment and lower household incomes.

Pre/post program responses on health-related measures

The key finding for the pre/post analyses of the survey (Table 3) was that the only

FIGURE 1
Age of HANS participants

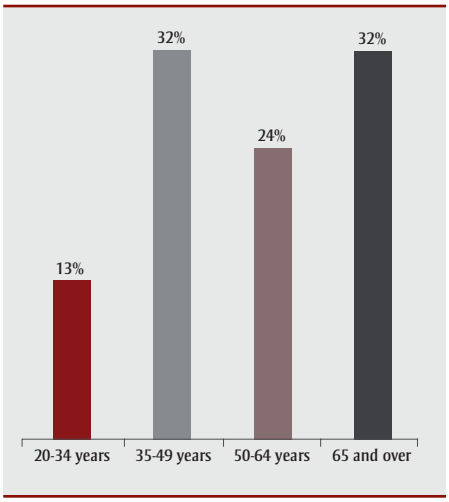


FIGURE 2
Years HANS participants have lived in the community

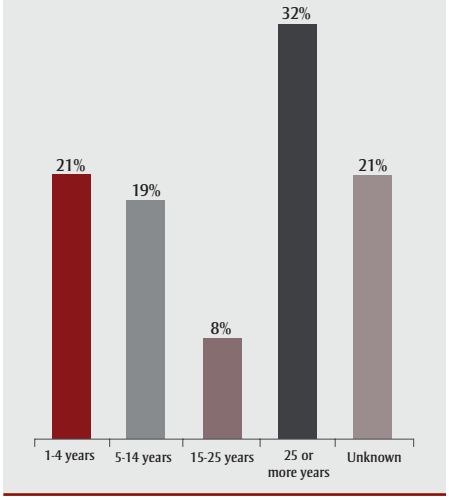


FIGURE 3
Employment status of HANS participants

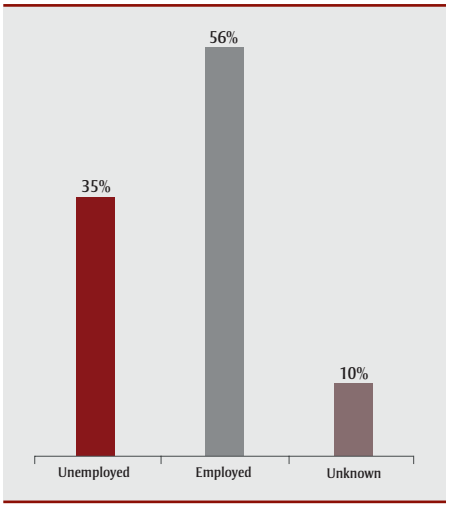
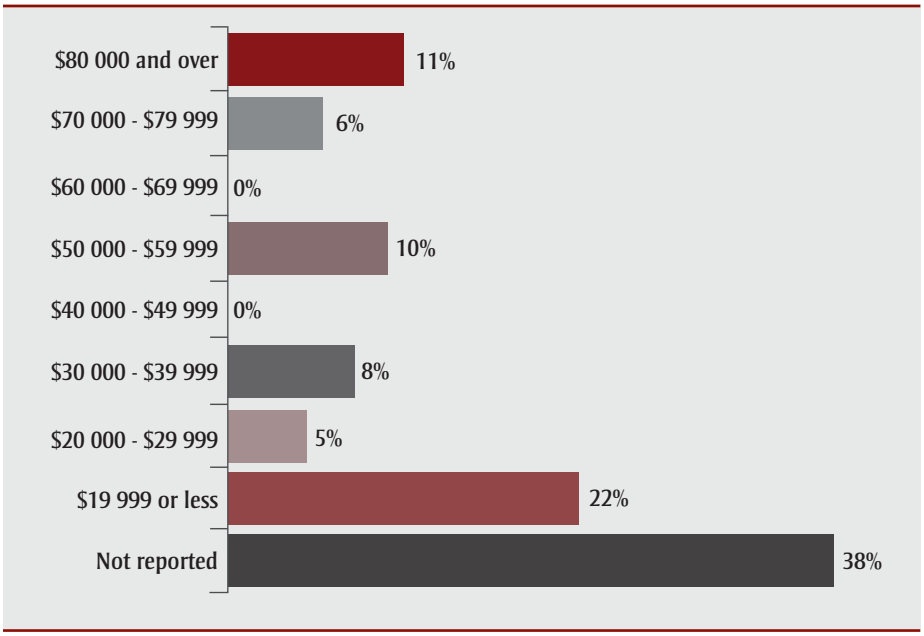


FIGURE 4
Annual household income of HANS participants



statistically significant improvements from pre- to post-program were in the Mental Health Continuum score. The Mental Health Continuum score assesses well-being and establishes results as "flourishing," "moderately mentally healthy" or "languishing."[18]

The other post-program health-related measures were not statistically significant.

Findings from individual interviews: factors affecting health

Participants were asked to identify factors that had a positive or negative impact on their health in the previous six months. The responses were consistent across data collection times, with the only change at 6, 12 and 24 months being increased identification of HANS group participation as a positive impact.

Positive factors affecting health

Seven factors were identified as having a positive effect on health. *Supportive relationships* with family and friends was the most frequently mentioned factor having a positive effect. This was consistent across all interview periods. Support from fellow HANS group members was also identified. *Physical activity*, including both organized activities and leisure activities, and *improved nutrition* were consistently identified across interview periods as improvinging one's health. *Community supports* were identified at the beginning (baseline/pre-intervention); however, at subsequent time periods, HANS group participation was the most frequent response regarding community supports. Participants also identified *good mental health* as having a positive impact on overall health, including being happy at work and experiencing less stress, and having a *positive attitude and beliefs*. More participants identified the positive impact of *access to services* over time and, in particular, services at NorWest.

Negative factors affecting health

Six factors were identified as having a negative impact on health. *Compromised mental health/stress* was most commonly cited as negatively affecting overall health. Participants decribed being overwhelmed and experiencing work, family or other stresses. However, by Time 3, fewer participants identified compromised mental health or stress as having a negative impact on their health. Many participants identified *poor nutrition* as having a negative impact, including social environments that contributed to increased eating, unhealthy food choices and challenges in maintaining weight or weight gain. However, by Time 3, fewer participants identified poor nutrition as negatively affecting their health. *Lack of physical activity*, *compromised health*, *lack of time/work–life balance* and *unsupportive relationships* were also consistently identified as negative impacts on health across all time periods. Table 4 groups the factors that had positive or negative health impacts into themes with representative quotes.

Findings from individual interviews: positive impact of HANS participation

Questions 3 to 6 were intended to identify if involvement in HANS had a positive impact on participants' health and what participants liked about HANS. The different themes that emerged from our analysis are presented pictorially with number of responses (Figure 5) and described more fully below.

Peer support

The most common responses about the benefits and impact of participation in a HANS group were related to peer support. A much repeated phrase was "we all support each other." Participants described listening to or supporting one another, sharing problems, providing support during a difficult life situation and feeling safe and unjudged. They also described their new friends and sense of community as a result of HANS group participation. One participant recalled how she provided support to another whose child was being bullied at school and helped her pursue it further and "reach a resolution." Some described giving or receiving rides to the group meetings or to medical appointments, or sharing resources that might be helpful. Others described reaching out to another outside the group meeting and keeping connected between meetings through regular phone contact, emails, texts, walks or coffee outings.

Learning/knowledge

Participants described how they received information about health-related topics and healthy choices from the Health School, guest speakers and each other. They identified the impact of the new skills they had learned, including chair exercises; measurements (e.g. blood sugar, blood pressure); Zumba; cooking; and meditation.

Reported behaviour change

Sixty-six percent of the participants were able to describe specific behaviour changes as a result of participating in the HANS group. Behaviour changes were primarily in the areas of nutrition and exercise, and in other areas such as stress and weight, blood sugar and/or blood pressure

TABLE 3
Analysis of HANS participants' pre/post-program responses on health-related measures

Health-related measure	Response	Data collection event		*p* (for column comparison)
		First survey (%)	Last survey (%)	
Diabetes knowledge score (n = 57)	Low score	19.3	7.0	> .05
	High score	80.7	93.0	> .05
Hypertension knowledge score (n = 58)	Low score	6.9	5.2	> .05
	High score	93.1	94.8	> .05
Nutrition score (n = 61)	Low nutrition risk	31.1	41.0	> .05
	Moderate nutrition risk	37.7	37.7	> .05
	High nutrition risk	31.1	21.3	> .05
Physical activity score (n = 61)	Very active	4.9	9.8	> .05
	Active	34.4	39.3	> .05
	Acceptable	23.0	21.3	> .05
	Inactive	13.1	13.1	> .05
	Sedentary	24.6	16.4	> .05
Sleep scale (n = 60)	Not a problem	70.0	68.3	> .05
	Problematic	30.0	31.7	> .05
Mental Health Continuum score (n = 60)	Flourishing	43.3	63.3	< .05
	Languishing	10.0	6.7	> .05
	Moderate	46.7	30.0	> .05
Have you ever smoked (n = 60)	No	55.0	58.3	> .05
	Yes	45.0	41.7	> .05
Do you smoke now (n = 60)	No	93.3	91.7	> .05
	Yes	6.7	8.3	> .05
Access to health care provider (n = 61)	Neutral	6.6	3.3	> .05
	Disagree	14.8	8.2	> .05
	Agree	78.7	88.5	> .05
Aware of community programs and services (n = 61)	Neutral	14.8	11.5	> .05
	Disagree	24.6	11.5	> .05
	Agree	60.7	77.0	> .05
Understand how to improve health (n = 61)	Neutral	6.6	3.3	> .05
	Disagree	16.4	6.6	> .05
	Agree	77.0	90.2	> .05
Connected to people in neighbourhood (n = 59)	Neutral	13.6	11.9	> .05
	Disagree	27.1	16.9	> .05
	Agree	59.3	71.2	> .05

management. Participants also described the positive aspects of goal setting and other changes. Additionally, participants described how learning from the HANS group had extended farther with positive impacts on other members of their families.

Inspiration/motivation/accountability

Participants described how participation in a HANS group has had an impact on their health through inspiration, motivation or accountability. Participants also described their desire to share the benefits of HANS group participation by encouraging others to join or sharing the information they had received in the group.

Monitoring indicators

Participants identified that taking responsibility for monitoring their own health indicators in their logbooks was empowering, reassuring and motivating. They appreciated being able to do their own monitoring, and these measures also spurred action to seek medical support if needed (e.g. "Because I discovered high blood pressure at HANS KAI I went to see my doctor and it is now under control").

Overcoming social isolation

Some participants identified that participation in a HANS group contributed to their sense of belonging and motivated them to "get out."

Access to services

Participants noted that their involvement in HANS provided a gateway to access

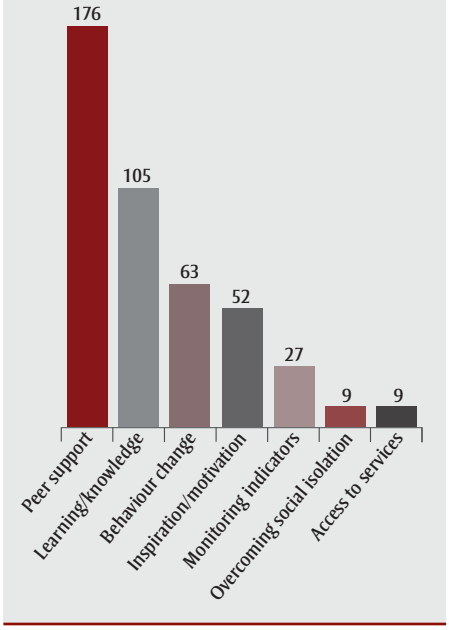
TABLE 4
Positive and negative factors affecting health with representative quotes from HANS participants

Factors impacting health		Quotes
Positive factors	Supportive relationships	"a very supportive family"; "my husband"; "my kids"; "my friends"; "HANS members have helped"; "[they] play a good role in it"; "love talking to them and some are good friends and we socialize outside HANS KAI so it's good to talk out things and share"
	Physical activity	"regular exercise"; "exercise class 3x per week"; "Hip Hop"; "Zumba"; "walking"; "playing with kids"; "yoga"
	Improved nutrition	"change in diet"; "eating healthy foods"; "eating better"
	Community supports	"meeting with other people"; "interacting with the [HANS] group helps me … [the] relationships we have built"
	Good mental health	"love my work"; "changes at my work"; "quit work"; "learning to cope with issues"; "less stress"
	Positive attitude and beliefs	"accountability to the group"; "motivated when [I'm] with others"; "self reliant"
	Access to services	[Access to]"doctor"; "dietician"; "psychologist"; "nurse practitioner"; "I have a team now"; "all kinds of [NorWest] specialists"
Negative factors	Compromised mental health / stress	"I feel overwhelmed"; "busy schedules"; "work responsibities and issues with … family and friends"; "lack of support … [at work]"; "I have a very stressful job"; "I work too many hours … have too many responsibilities"; "family life is always stressful"; "my … [son, daughter, husband, mom, children]"; "death in the family"
	Poor nutrition	"attending … gatherings"; "eating out"; "the holidays"; "bad eating habits"; "[we eat unhealthy] when I don't have time"; "being overweight";
	Lack of physical activity	"not enough time"; "don't make time [to exercise]"; "pain from injury"; "the weather"
	Compromised health	"heart condition"; "diabetes"; "arthritis"; "pain"
	Lack of time / work–life balance	"work demands"; "family demands"; "just been really busy … not much time for myself"
	Unsupportive relationships	"not having enough family support"; "family has made it harder"

services: services provided by NorWest, encouragement to see health care providers regularly and information about other services in the community.

Table 5 summarizes the positive health impacts of HANS participation with representative quotes.

FIGURE 5
Benefits of HANS participation (# responses)



Findings from individual interviews: recommendations for improving HANS groups

Questions 7 and 8 were intended to gather participant feedback on how to improve HANS. Participants identified three main areas for improvement.

Meeting format

Participants described issues such as frequency and duration but did not agree on optimal frequency and duration. Some identified the need to have additional group members.

Leadership/structure/organization

HANS groups are participant led, but the most common feedback about improvement was for more leadership and structure. In most groups a leader emerged; however, not all participants shared the leadership role. Groups were destabilized when the leader was no longer available to lead, and groups became smaller due to attrition and changes in meeting dates and/or locations. Some groups were more successful than others in identifying the types of education sessions they wanted. Participants wanted more interaction with their NorWest contact person and suggested a number of program improvements such as regular meetings with planned agendas.

Access

Consistent attendance at HANS groups was a challenge for some participants. Participants identified busy schedules and competing responsibilities as well as issues such as transportation and meeting location. Workplace groups had particular challenges with the meeting times.

Table 6 summarizes the suggested improvements to HANS KAI with representative quotes from participants.

Discussion

The purpose of this study was to determine if structured peer-led community groups could be successfully initiated within an urban Canadian context and have a positive impact on the health of participants. A meta-analysis of 148 studies found that having supportive relationships was related to decreased mortality risks.[21] Berkman and Glass[22] identified that adults who are socially isolated have a two- to five-fold higher death rate than others. While peer support has been used in health promotion initiatives, it has been more widely used in disease-specific health promotion initiatives.[23-25]

TABLE 5
Positive health impacts of HANS particpation and representative quotes

Health impacts of HANS participation	
Peer support	**Sharing/listening/supporting:** "We share many of the same problems … it's a relief to know you are not alone.… My problems … seem so much more manageable.… If they can do this, I can … do this too"; "HANS KAI may have saved my life"; "I pick her up [to get to HANS]"; "She drove me to my doctor's appointment"; "I've been giving her cookbooks because she doesn't have any"
	Feeling safe and unjudged: "If someone brings out a problem we can talk about it … [with] no judgment … I feel so supported"; "If I need help, I'm not afraid to ask"; "Everyone has their own struggles and in [this] environment we have a safe place to talk about these things.…"
	Friendship/sense of community: "feel a part of the community"; "they are my friends now"; "we meet outside the group"; "[the calls and texts] just help to kinda keep track"
Learning/knowledge	**Specific health information:** "healthy eating … snacks … diet"; "managing blood sugars"; "reading labels"; "I … get information I still use"
	Impact of learning and new skills: "We get information and then get to practice it"; "bringing new things into my life—it's good"
Reported behaviour change	**Nutrition:** "I am making better food choices"; "now I eat more fruits and vegetables"; "I have lessened my salt and I buy lean meats"
	Exercise: "The HANS group motivated me to start walking"; "I started exercising"
	Stress: "It's helped me recognize what's causing it all [the stress]"; "I … [used] the stress release techniques I learned from HANS KAI"
	Management of weight/blood sugar/blood pressure: "I have lost weight"; "[since participating in HANS] I have started taking my medication regularly"; "My sugars are better"; "My doctor has reduced my … medications"; "[My doctor] noticed a significant [improvement] … from when I started the HANS program.…"
	Goal setting: "I am learning how to set reasonable goals"; "I am sticking to [my goals]"
	Impacts to others: "When you buy food … you have to look at the label.… [I am] also teaching my family members to [look at labels]"; "Makes me think [when] I prepare … lunch for my kids"
	Other changes: "I quit smoking"; "I am making better and healthier choices"; "I laugh more"
Inspiration/ motivation/ accountability	**Inspiration:** "When I see other people change towards the better or really trying hard to get out of their ruts … it inspires me"; "There is inspiration to try new things"
	Motivation: "They keep you doing things you don't want to do"; "motivation to stick to it"
	Accountability: "The group keeps me accountable"; "taking [health] into your own hands … [it is] a whole mind-set that's different"
	Sharing the benefits: "I encourage others to attend … 'You will learn something!'"; "I try to we invite [others when] we have special speakers.…"; "I share the information with my sister and mom … [like] exercising, eating health foods…"
Monitoring indicators	**Self-Monitoring:** "keeps me on top of stuff ... on my toes"; "I am monitoring … more regularly, … my blood sugars have become more consistent"; "don't have to … wait for the next appointment"
Overcoming social isolation	**Belonging:** "I feel like I belong"; "I am part of something"; "I don't have a lot of friends … it's nice to know someone is out there thinking about [and remembering] you"; "I have lived in this area for 30 years, but have only begun to feel a part of the community since HANS KAI"
	Getting out: "helped to … get me out"; "getting people out of their isolation"; "It forces us stay-at-home moms to get out … that's probably the best thing.…"
Access to services	**Community services:** "They [NorWest] tell us what is offered in the community"; "If I need information I [know who] I can call and it's good.…"
	NorWest Services: "I have a dietician [and] foot care … through NorWest … as a result of HANS KAI"; "The support is really good between NorWest and HANS KAI and [how they] reach out to the community is really something"; "NorWest provides a safety net"

Our findings suggest that there were benefits to participation in a HANS group, including peer support, learning/knowledge, behaviour change, inspiration/motivation/accountability, overcoming social isolation and increased access to primary care and other health-related services. These findings are congruent with previous research undertaken in the area of peer support groups and their impact on health. Peer-led approaches that contribute to community "belonging" had a positive effect on the "most prominent health behaviours (exercise, weight loss and improved diet)."[26,p.277] A systematic review of 25 randomized controlled trials assessing health-related behaviour change in older adults concluded that peer-based interventions contributed to positive health-related behaviour change such as increased physical activity, decreased smoking, increased condom use and increased completion of advance directives.[27] While most community-based peer support initiatives have focussed on a specific health behaviour, others were implemented to create social support or social networks to prevent social isolation.[2] In a 2015 study of a community-based program developed to create peer support networks, the major themes that emerged were creation of social networks, enhancement of well-being and provision of empowering services.[28] Peer support has also been found to increase access to primary care services, including health information, community programs and support services.[24,26,28-36] In addition, peer-based interventions have been reported to bring about shared achievement through doing, providing role models and sharing knowledge, which in turn brought about satisfaction, self-confidence and acceptance among group members.[24,36-41]

Where this research adds to the literature is in the area of support groups that address interrelated chronic disease prevention

TABLE 6
Participants' suggestions to improve HANS KAI and representative quotes

Improving HANS KAI	
Meeting format	**Frequency/duration:** "I wish we could meet more often"; "I think it should be longer"; "I would rather meet [just] once per month"
	Membership: "Our numbers are not as strong as we want it to be"; "If we could have a couple more members that would be nice"
Leadership/structure/ organization	**Leadership:** "Not having a lead person may work in Japan but I don't think it works here"; "With rotating leaders there is no one really [coordinating] it"; "We have ___ she keeps everything together. It would be hard if she wasn't [here]"; "We had … a person who took more charge [who] left and since then it pretty much fell apart"
	Structure: "We are getting different speakers which is great"; "I wish [the presenters were] better prepared"; "more topics"; "more group discussions"; "more exercise classes"; "return funding for snacks"
	Organization: "I would put a strong emphasis on … providing more structure and more support from the NorWest staff contact person; "sometimes they just fly in and fly out"; "[need] a regular check"
Access	**Access:** "finding the time"; "distance I have to come to go to it"; "I can't attend during the day"; "use handi transit … sometimes I don't have enough for extra tickets"; "busy schedules"; "it's … after work"

behaviours (healthy eating, regular physical activity, monitoring indicators and social support) within a model of peer support and their impact on health versus those that focus on a single chronic disease or condition and a related specific health behaviour.

Although the benefits of peer support are well documented in the literature, it cannot be said that the empirical evidence is unequivocal on this issue. Webel et al.[27] conducted a systematic review of the effectiveness of peer-based interventions for specific behaviour change and concluded that the evidence was mixed. Some interventions were effective (physical activity, smoking and condom use) while others were not (breastfeeding, medication adherence and women's health screening).

The findings of this study are similar. Quantitative results from the participant survey show that participation in a HANS group resulted in statistically significant improvement only in mental health scores and resulted in possible positive trends in other health-related measures. However, the qualitative results from thematic analysis of the in-person interviews identified that HANS group participation had a positive impact on participants' health primarily through peer support and through learning/increased knowledge. Additionally, 66% of the participants reported a behaviour change even though there was no statistically significant change identified in the quantitative data. This could be related to the way the behaviour change questions were presented in the survey or could indicate that open-ended qualitative methods may be a better way of eliciting behaviour change information. An unexpected finding was the absence of reported income-related stress (which the researchers had anticipated from the open-ended question "What has not helped or made it harder for you to stay healthy and feel good?") despite the proportion of participants being unemployed or in a lower-income group.

In summary, HANS KAI groups appear to have a significant positive impact on participants' mental health as identified in both the quantitative and the qualitative findings. Additionally, participants experienced increased support and connectedness, which may generate positive effects in some areas of health including participant-reported behaviour changes. It is less clear whether HANS KAI improves measurable indicators such as blood sugar, blood pressure, weight and waist circumference. More research is needed to identify if HANS participation has an impact on these measurable indicators over time.

Limitations

There were a number of limitations of this study. Our sample was recruited from one (Canadian) jurisdiction and was primarily female. Lack of male participation may be attributable to male hesitance to seek assistance for health issues, especially related to preventive interventions, but presents an opportunity to consider how to include more men in the intervention. These may limit the transferability of findings. In addition, our sample was not large; the findings are based on only groups that met regularly and on self-reported feedback, although the qualitative methodology that was used provided in-depth information that could compensate for this limitation. We were unable to compare the measurable indicators of blood sugar, blood pressure, weight and waist circumference over time due to incomplete logbooks that tracked these self-measured health indicators. Self-reported health behaviour changes (general and specific) may not capture true behaviour change and may be subject to recall or social desirability biases. Additionally, four groups that started were unable to continue for a variety of reasons (e.g. loss of interest, struggle to schedule meeting times). Participants missed meetings when interviews were scheduled, were unavailable or lost to follow-up, and there were challenges with regular participant attendance. Similar to this research, Gustavson et al. identified the challenge of high attrition rates in public health intervention research (30-70%), which may impact the generalizability of the findings.[42] Attempts were made to contact those who stopped participating; however, researchers were only able to conduct a few exit interviews.

There were statistical differences between the demographics of the research participants and those lost to follow-up, and the lack of data from dropouts may have affected the findings of the study. The peer support model used in this research was intentionally peer-led where all participants were peers and guidance and support was provided from NorWest health professionals. However, the most common feedback about improving HANS were requests for more leadership and more structure.

While peer support may provide knowledge, a sense of connection and improvements in self-care, there are other methods, both individual and group-related interventions, that may also work, and health professionals need to understand which might be the best match for a client.[43-44] Peer support groups to improve health may not be the right fit for or be effective for everyone, and the peer-led model with the perceived lack of supports may have contributed to the attrition rate.

Health promotion literature has identified that interventions focussed on lifestyle or behaviour change at the individual level may have limited long-term effects as "health behaviours are influenced by many competing factors: cultural pressures, health literacy, health inequalities, mental capacity, genetic predisposition and, in the case of smoking and alcohol, addiction to a substance."[45,p.1] At the same time, a 12- to 24-month intervention may not be long enough to know about the sustained health effects of HANS group participation.

Conclusion

As the focus of health care changes from treating disease to promoting health, the use of peer support is becoming more common not only in the discipline of health but also in behavioural science.[2] HANS KAI participation, embedded in a model of peer support, is intended to support health using a variety of health promotion interventions such as education, action, access to services and empowerment. This community-based research is driven by a need to identify innovative ways to address chronic disease prevention and management in a community challenged with interrelated factors (social determinants of health) such as lower education and income, social isolation and lack of access to health and recreation services.

This research used a participatory design between NorWest and the researchers, and included direction and feedback from a community board and community residents. The duration of this research project followed individuals over a period of up to 24 months. Participation in a HANS group is intended to be for the long term with participants and groups continuing well after the life of the research project. This may be an improvement over other chronic disease prevention interventions of prescribed length (6-12 weeks) or around a single chronic disease or behaviour change.

The findings of this research suggest that HANS KAI proved to be an effective intervention to realize statistically significant improvements in the area of mental health. The findings from the qualitative analysis also suggest that there were benefits to participation in a HANS group, including peer support, learning/knowledge, behaviour change, inspiration/motivation/accountability, overcoming social isolation and increased access to primary care and other health-related services.

While further research will be required to validate these findings, it appears that the HANS KAI approach, which goes beyond focussing on individual behaviour change and considers the importance of community, may be effective for creating environments that empower community members to support each other while promoting healthy lifestyle choices and detecting early changes in health status.

Acknowledgments

Funding was provided by the NorWest Co-op Community Health Centre. The authors would like to acknowledge the support of the NorWest staff and all the participants of the HANS KAI groups who participated in the research. We would also like to acknowledge the support and input provided by the NorWest HANS KAI organizers, Nancy Heinrichs (Executive Director), Michelle Kirkbride (Community Development Team Coordinator), Shannon Milks (Primary Care Coordinator) and Mike Sadlowski (Health Promotion Specialist); Dr. Benita Cohen (qualitative research advisor); Dr. Nawal Lutfiyya (data analyst – quantitative data); Dr. Lawrence Elliot (quantitative research advisor); Kristy Boyko, Kate Hodgson and Adebusola Adekoya (research assistance); and the Manitoba Centre for Nursing & Health Research.

Conflicts of interest

The authors have no conflicts of interest.

Authors' contributions and statement

AH and HW worked closely with the HANS KAI advisory group from the NorWest Co-op Community Health Centre to conceptualize, plan and conduct the research. Additional support was obtained from contributors as described in the acknowledgements. HW had a primary role in conceptualizing and executing the project plan, and AH had the primary role in data collection and analysis as well as drafting of the preliminary report. Both authors read and approved the final manuscript.

The content and views expressed in this article are those of the authors and do not necessarily reflect those of the Government of Canada.

References

1. Mead S, Hilton D, Curtis L. Peer support: a theoretical perspective. Psychiatr Rehabil J. 2001;25(2):134-41.

2. Dennis CL. Peer support within a health care context: a concept analysis. Int J Nurs Stud. 2003;40(3): 321-32.

3. Dennis CL. The process of developing and implementing a telephone-based peer support program for postpartum depression: evidence from two randomized controlled trials. Trials. 2014; 15(1):131. doi: 10.1186/1745-6215-15-131.

4. Thomas GN, Macfarlane DJ, Guo B, et al. Health promotion in older Chinese: a 12-month cluster randomized controlled trial of pedometry and "peer support". Med Sci Sports Exerc. 2012;44(6):1157-66. doi: 10.1249/MSS.0b013e318244314a.

5. Ford P, Clifford A, Gussy K, et al. A systematic review of peer-support programs for smoking cessation in disadvantaged groups. Int J Environ Res Public Health. 2013;10:5507-22. doi: 10.3390/ijerph10115507.

6. Panayotof-Schaan L. An overview of health co-operatives: a case study perspective using Canadian and international examples [Internet]. Victoria, BC: British Columbia Institute for Co-operative Studies; 2009 [cited 27 Mar 2017]. Available from: http://planh.ca/sites/default/files/panayotof-schaan_healthcooperatives.pdf

7. Girard JP, Restakis J. To Life! Japan's model of co-operative health care & what it means for Canada. Making Waves. 2008;19(1):5-8.

8. Bandura A. Social learning theory. Englewood Cliffs, NJ: Prentice Hall; 1977

9. Zimmerman MA. Empowerment theory. In: Rappaport J, Seidman E, editors. Handbook of community psychology. New York: Kluwer Academy / Plenum Publishers; 2000. p 43-63.

10. Winnipeg Regional Health Authority; George and Fay Yee Centre for Healthcare Innovation, Evaluation Platform. Inkster community area profile, 2015 [Internet]. Winnipeg (MB): Winnipeg Regional Health Authority; 2015 [cited 27 Mar 2017]. Available from: http://www.wrha.mb.ca/research/cha2014/files/Inkster.pdf

11. Kaplan and Associates. Inkster in action report. [Winnipeg (MB): Kaplan and Associates]; 2008.

12. Cresswell J, Klassen A, Clarke V, et al. Best practices for mixed methods research in the health sciences [Internet]. [Washington]: National Institutes of Health, Office of Behavioral and Social Sciences Research (US); 2011 [cited 27 Mar 2017]. Available from: https://www.nursing.virginia.edu/media/Best_Practices_for_Mixed_Methods_Research.pdf

13. Government of Manitoba, Manitoba in Motion. Physical activity self-assessment. Winnipeg (MB): Government of Manitoba; 2010.

14. Harland Gregoire MJ, Chow S, Spooner GR, et al. A screening instrument to identify adults with nutritional risks in a family medical practice. Can J Diet Pract Res. 1999;60(4)205-11.

15. Government of Manitoba, Tobacco Control & Cessation. Commit to quit self-assessment. Winnipeg (MB): Government of Manitoba; 2010.

16. Williams MV, Baker DW, Parker RM, et al. Relationship of functional health literacy to patients' knowledge of their chronic disease. A study of patients with hypertension and diabetes. Arch Intern Med. 1998;158:166-72. doi: 10.1001/archinte.158.2.166.

17. Hayes R, Stewart A. Sleep measures. In: Stewart A, Ware J, editors. Measuring functioning and well-being: the Medical Outcomes Study approach. Durham (NC): Duke University; 1992. p. 235-59.

18. Keyes C. Brief description of the mental health continuum short form (MHC-SF) [Internet]. Atlanta (GA): [Emory University]; 2009 [cited 27 Mar 2017]. Available from: https://www.aacu.org/sites/default/files/MHC-SFEnglish.pdf

19. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36): I. Conceptual framework and item selection. Med Care. 1992;30(6): 473-83.

20. Gupta SK. Intention-to-treat concept: a review. Perspect Clin Res. 2011;2(3): 109-12.doi: 10.4103/2229-3485.83221.

21. Holt-Lunstad J, Smith T, Layton JB. Social relationships and mortality risk: a meta-analytic review. PLoS Med. 2010;7(7):e1000316. doi: 10.1371/journal.pmed.1000316.

22. Berkman LF, Glass TA. Social integration, social networks, social support, and health. In: Berkman LF, Kawachi I, editors. Social epidemiology. New York: Oxford University Press, 2000. p. 137-73.

23. Bagnall AM, South J, Hulme C, et al. A systematic review of the effectiveness and cost-effectiveness of peer education and peer support in prisons. BMC Public Health. 2015;15:290. doi: 10.1186/s12889-015-1584-x.

24. Woolhouse S, Cooper E, Pickard A. "It gives me a sense of belonging": providing integrated health care and treatment to people with HCV engaged in a psycho-educational support group. Int J Drug Policy. 2013;24(6):550-7. doi: 10.1016/j.drugpo.2013.05.018.

25. Zhong X, Wang Z, Fisher EB, et al. Peer support for diabetes management in primary care and community settings in Anhui Province, China. Ann Fam Med. 2015;13 Suppl 1:S50-8. doi: 10.1370/afm.1799.

26. Hystad P, Carpiano RM. Sense of community-belonging and health-behaviour change in Canada. J Epidemiol Community Health. 2010;66(3):277-83. doi: 10.1136/jech.2009.103556.

27. Webel AR, Okonsky J, Trompeta J, et al. A systematic review of the effectiveness of peer-based interventions on health-related behaviors in adults. Am J Public Health. 2010;100(2):247-53. doi: 10.2105/AJPH.2008.149419.

28. Liamputtong P, Koh L, Wollersheim D, et al. Peer support groups, mobile phones and refugee women in Melbourne. Health Promot Int. 2016; 31(3):715-24. doi: 10.1093/heapro/dav015.

29. Aschbrenner KA, Naslund JA, Shevenell M, et al. Feasibility of behavioral weight loss treatment enhanced with peer support and mobile health technology for individuals with serious mental illness. Psychiatr Q. 2016;87(3): 401-15. doi: 10.1007/s11126-015-9395-x.

30. Coşkun A, Karakaya E. Supporting safe motherhood services in Diyarbakir: a community-based distribution project. Matern Child Health J. 2013; 17(6):977-88. doi: 10.1007/s10995-012-1102-z.

31. Crawford S, Bath N. Peer support models for people with a history of injecting drug use undertaking assessment and treatment for hepatitis C virus infection. Clin Infect Dis. 2013;57 Suppl 2:S75-9. doi: 10.1093/cid/cit297.

32. Hanson LC, Armstrong TD, Green MA, et al. Circles of care: development and initial evaluation of a peer support model for African Americans with advanced cancer. Health Educ Behav. 2013;40(5):536-43. doi: 10.1177/1090198112461252.

33. Hanson LC, Green MA, Hayes M, et al. Circles of care: implementation and evaluation of support teams for African Americans with cancer. Health Educ Behav. 2014;41(3):291-8. doi: 10.1177/1090198113512127.

34. Mason K, Dodd Z, Sockalingam S, et al. Beyond viral response: a prospective evaluation of a community-based, multi-disciplinary, peer-driven model of HCV treatment and support. Int J Drug Policy. 2015;26(10):1007-13. doi: 10.1016/j.drugpo.2015.04.012.

35. Rempel LA, Moore KC. Peer-led prenatal breast-feeding education: a viable alternative to nurse-led education. Midwifery. 2012;28(1):73-9. doi: 10.1016/j.midw.2010.11.005.

36. Swarbrick MA. Integrated care: wellness-oriented peer approaches: a key ingredient for integrated care. Psychiatr Serv. 2013;64(8):723-6. doi: 10.1176/appi.ps.201300144.

37. Ashing-Giwa K, Tapp C, Rosales M, et al. Peer-based models of supportive care: the impact of peer support groups in African American breast cancer survivors. Oncol Nurs Forum. 2012;39(6):585-91. doi: 10.1188/12 .ONF.585-591.

38. Coniglio FD, Hancock N, Ellis LA. Peer support within Clubhouse: a grounded theory study. Community Ment Health J. 2010;48(2):153-60. doi: 10.1007/s10597-010-9358-5.

39. Lu Q, You J, Man J, et al. Evaluating a culturally tailored peer-mentoring and education pilot intervention among Chinese breast cancer survivors using a mixed-methods approach. Oncol Nurs Forum. 2014;41(6):629-37. doi: 10.1188/14.ONF.629-637.

40. Raine P. Promoting breast-feeding in a deprived area: the influence of a peer support initiative. Health Soc Care Community. 2003;11(6):463-9. doi: 10.1046/j.1365-2524.2003.00449.x.

41. Repper J, Watson E. A year of peer support in Nottingham: lessons learned. Journal of Mental Health Training, Education and Practice. 2012;7(2):70-8. doi: 10.1108/17556221211236466.

42. Gustavson K, von Soest T, Karevold E, et al. Attrition and generalizability in longitudinal studies: findings from a 15-year population-based study and a Monte Carlo simulation study. BMC Public Health. 2012;12:918. doi: 10.1186/1471-2458-12-918.

43. Sav A, McMillan Sara, Kelly F, et al. Consumer health organisations for chronic conditions: why do some people access them and others don't? Prim Health Care Res Dev. 2014;15:1-12. doi: 10.1017/S1463423614000036.

44. Lawn S, Schoo A. Supporting self-management of chronic health conditions: common approaches. Patient Educ Couns. 2010;80(2):205-11. doi: 10.1016/j.pec.2009.10.006.

45. Thirlaway K, Davies L. Lifestyle behaviours and personal responsibility for health. eLS [Internet]. 2013 Sep [27 Mar 2017]. doi: 10.1002/9780470015902 .a0025159/abstract.

https://doi.org/10.24095/hpcdp.38.3.05

At-a-glance

Traumatic brain injury management in Canada: changing patterns of care

Deepa P. Rao, PhD; Steven McFaull, MSc; Wendy Thompson, MSc; Gayatri C. Jayaraman, PhD

Abstract

Introduction: With growing awareness about traumatic brain injuries (TBI), there is limited information about population level patterns of TBI care in Canada.

Methods: We examined data from the Canadian Community Health Survey (years 2004, 2009, and 2014) among all respondents ages 12 years and older. TBI management characteristics examined included access to care within 48 hours of injury, point of care, hospital admission, and follow-up.

Results: We observed that many Canadians sought care within 48 hours of their injury, with no changes over time. We found a significant decline in the proportion of Canadians opting to visit an emergency department ($p = 0.03$, all ages), and a significant increase in youth opting to visit a doctor's office ($p < 0.01$).

Conclusion: TBIs are an important and growing health concern in Canada. Care for such injuries appears to have shifted towards the use of health care professionals outside the hospital environment, including primary care doctors.

Keywords: *traumatic brain injury, concussion, surveillance*

Highlights

- Approximately 80% of Canadians reporting a traumatic brain injury (TBI) sought care within 48 hours of their injury.
- Examining trends over 10 years, there is a significant decline in the proportion of youth reporting a TBI who went to an emergency department for their care.
- Current data demonstrates that approximately 1 in 10 (interpret with caution) Canadians who report a TBI said they went to a doctor's office for treatment following injury.

Introduction

Ms. Rowan Stringer was a high school rugby player who died following brain injury due to a concussion. Subsequent to her death, a strong initiative to raise awareness and improve treatments for concussions ultimately lead to the Royal Assent of the Rowan's Law Advisory Committee Act in 2016.[1] At present, an ever-growing number of professionals across Canada are investing resources in preventing and addressing concussion and other traumatic brain injuries (TBI). A recent study reports that the incidence of TBIs is increasing in Canada: the annual percent change (APC) in TBI among those who reported any type of serious injury in the past 12 months was found to be 9.6% (95% CI: 8.2–11.0).[2]

This so-called 'invisible epidemic' of TBIs is challenged by difficulties in accurate and timely diagnosis.[3] Whether or not a diagnosis is made, some of those with a TBI are at risk of persistent post-concussion syndrome (PPCS; symptoms that persist over a period of weeks or months[4,5]). With young athletes there is a risk (albeit small) of second impact syndrome (a subsequent concussion before a previous one has resolved).[6] Early identification of a TBI event can be important given that the consequence of misdiagnosis or faulty management is the possibility of disability or even death.[7,8] With regard to where people seek care, the latest international sport-related concussion guidelines, as well as the Canadian guidelines, describe that when an individual is suspected to have a TBI they should be removed from play and assessed by a physician or licensed healthcare provider.[9,10] Where individuals choose to seek care appears to be changing: a recent study reported a shift in pediatric TBI care from emergency departments (ED) towards primary and speciality providers.[11,12]

The objective of the present descriptive analysis is to provide population level estimates related to TBI care: whether individuals reporting receiving care within 48 hours after their injury, where they went for treatment, whether they were admitted to hospital, and whether they were receiving ongoing care. Given the dearth in national-level information regarding TBIs in Canada, a secondary objective of this study is to use questions available from existing national surveys to examine trends in TBI management over time.

Methods

Data sources

The Canadian Community Health Survey[13] (CCHS) is a cross-sectional health survey

Author reference:

Public Health Agency of Canada, Ottawa, Ontario, Canada

Correspondence: Deepa P. Rao, Public Health Agency of Canada, 785 Carling Avenue, Office 707B1, Ottawa, ON K1A 0K9; Tel: 613-867-8303; Fax: 613-941-2057; Email: deepa.rao@canada.ca

of Canadians aged 12 years and older but not those living in nursing homes or long-term care facilities or on reserves, full-time members of the Canadian Armed Forces, or civilian residents of military bases. The survey was designed to derive estimates at the national and provincial levels and for 110 health regions in Canada. Self-reported data from the CCHS (years 2005, 2009 and 2014) were used to examine characteristics associated with TBI among the Canadian population aged 12 years (youth: ages 12 to 17 years, adults: ages 18 years and older).

Key variables

Cases of TBI were identified from those who reported an injury (all types of injury or ATI) "that occurred in the past 12 months and [that was] serious enough to limit [their] normal activities the day after the injury occurred," and who identified this primary ATI as a TBI. The TBI care characteristics examined in the present analysis, which were only analyzed among those individuals who self-reported a TBI, are: (i) access to care within 48 hours of injury, (ii) point of care, (iii) admission to the hospital overnight, and (iv) ongoing follow-up care. These were selected from the limited survey questions available regarding care received, and do not reflect any recommendations regarding TBI management.

The 'access to care within 48 hours of injury' characteristic was selected based on a corresponding survey question meant to collect information on the time taken to access care; "Did [you] receive any medical attention for the injury from a health professional in the 48 hours following the injury?" Individuals who responded yes were then asked where they went for their care, which was used to examine 'points of care' such as the ED or other settings (an outpatient clinic, chiropractor's office, community health center, hospital outpatient clinic, doctor's office, or where the TBI occurred). Among those who indicated treatment within 48 hours, 'hospitalization following injury', was examined through the survey question, "Were you admitted to a hospital overnight?" Finally, information regarding 'ongoing follow-up care, was captured at the time of data collection from a response to, "At the present time, are you getting follow-up care from a health professional because of this injury?"

Statistical analyses

We completed descriptive analyses using SAS Enterprise Guide version 5.1 (Cary, NC). Incidence and proportion estimates were weighted to reflect the Canadian household population and 95% confidence intervals were calculated using bootstrap re-sampling methods. Generalized logistic models were used to estimate annual percent change (APC) and significance was determined at $p < 0.05$.

Results

Roughly 4 in 5 individuals reporting a TBI sought medical care within 48 hours of their injury in 2014 (Table 1). While the majority of these individuals went to an ED, an analysis of the trend over time suggests this choice of point of care is significantly decreasing among youth (Table 1) (youth APC: −3.1%, 95% CI: −3.8 to −2.4, $p < 0.001$; adults APC: −2.6%, 95% CI: −5.4 to 0.3, $p = 0.08$; all ages APC: −2.7%, 95% CI: −5.1 to −0.2, $p = 0.03$). Although estimates from 2005 and 2009 were too rare to report, those from 2014 show that approximately 1 in 10 people with a TBI injury now seek care at a doctor's office (11.5%, 95% CI: 5.2–17.8, interpret with caution). Examining trends over time, there is a significantly increasing trend of people reporting that they visited a doctor's office among youth (youth APC: 0.3%, 95% CI: 0.2–0.3, $p < 0.001$; adults APC: 0.06%, 95% CI: −0.07 to 0.2, $p = 0.34$; all ages APC: 0.1%, 95% CI: 0.02–0.2, $p = 0.02$).

Data from 2005 shows that 21.9% (95% CI: 12.7–31.2, interpret with caution) of Canadians reporting a TBI were admitted overnight, compared with 13.4% (95% CI: 5.5–21.3, interpret with caution) in 2014. A higher proportion of people who were hospitalized, than not hospitalized, reported that they were being followed-up by a health professional at the time of data collection (Figure 1). Independent of hospitalization history, the most recent data shows that the majority of individuals who reported a TBI in the past year were not receiving follow-up care from a health professional at the time they participated in the survey (youth: 86.8%, 95% CI: 77.8–95.9; adults: 69.3%, 95% CI: 57.6–80.9; all ages: 73.5%, 95% CI: 64.2–82.7).

Discussion

Increasing rates of TBI have been reported both in Canada[2] and in the U.S.[14] This prompts the question: where are Canadians going for their TBI care and what follow-up are they receiving? Examining these questions, we observed significant changes in where many Canadians were opting to receive care, namely that a doctor's office was an important and emerging point of care and that there were declines in the proportion of youth opting to visit an ED. Our findings are similar to those observed in a recent study examining TBI care in a pediatric population in the U.S.[12,15] Although changes in locations of care do not likely bear the same insurance concerns here in Canada as they may in the U.S.,[15] the ease and accessibility to primary care may nevertheless play a role. We did not observe any changes with regard to whether people sought health care advice within 24 hours of their TBI. The recent increasing trend in TBI has been attributed to factors such as improved data capture of mild TBI cases.[16,17] A recent assessment of sport concussion and return-to-play guidelines,[18] however, suggests that, while research in this area is still preliminary, there is no evidence of an effect of these guidelines on TBI prognosis.[19] Future studies to examine factors associated with the increasing trend in TBIs, and to evaluate prognosis after TBI, should help to better inform evidence-based guidelines for TBI care.[19]

Strengths and limitations

National surveys rely on self-reported information and do not capture fatal cases. The former provides for the potential of respondent bias and for diminished validity due to retrospective self-reported injury recall.[20] The lack of fatal cases limited the generalizability of our findings to only non-fatal cases. In the examination of follow-up care, the phrasing of the question limited the ability to detect cases where an individual had received such care but which had been completed by the time of the survey, or where the individual had yet to begin follow-up care. This question did, however, permit detection of differences between key groups; i.e., between those who were hospitalized versus those who were not. While limitations to the internal and external validity may exist, the better data capture of cases

TABLE 1
Ten-year trends in self-reported treatment for traumatic brain injury among Canadians, CCHS, 2005, 2009 and 2014

		2005			2009			2014			Annual percent change	
		Population	Incidence (%)	95% CI	Population	Incidence (%)	95% CI	Population	Incidence (%)	95% CI	%	95% CI
Received care within 48 hours of injury	All	38 214	72.3	65.5–79.1	68 525	83.8	75.3–92.3	123 478	81.0	73.5–88.5	1.9%	−1.9 to 5.7
	Youth	11 596	68.7	56.1–81.4	13 127	70.6	51.3–89.9	27 447	75.2	63.3–87.1	2.2%	−1.8 to 6.2
	Adults	26 618	74.0	65.8–82.1	55 398	87.7	79.7–95.7	96 031	82.8	73.7–91.9	2.0%	−1.9 to 6.0
Treatment at ED	All	30 879	80.8	73.2–88.4	45 452	66.3	52.0–80.7	79 037	64.0	53.1–74.9	−2.7%	−5.1 to −0.2
	Youth	9 396	81.0	68.3–93.7	9 158	69.8	49.9–89.6	16 878	61.5	45.4–77.6	−3.1%	−3.8 to −2.4
	Adults	21 483	80.7	71.0–90.4	36 295	65.5	48.5–82.5	62 159	64.7	51.1–78.3	−2.6%	−5.4 to 0.3

Source: CCHS, 2005, 2009 and 2014.

Abbreviations: CCHS, Canadian Community Health Survey; CI, confidence interval; ED, emergency department.

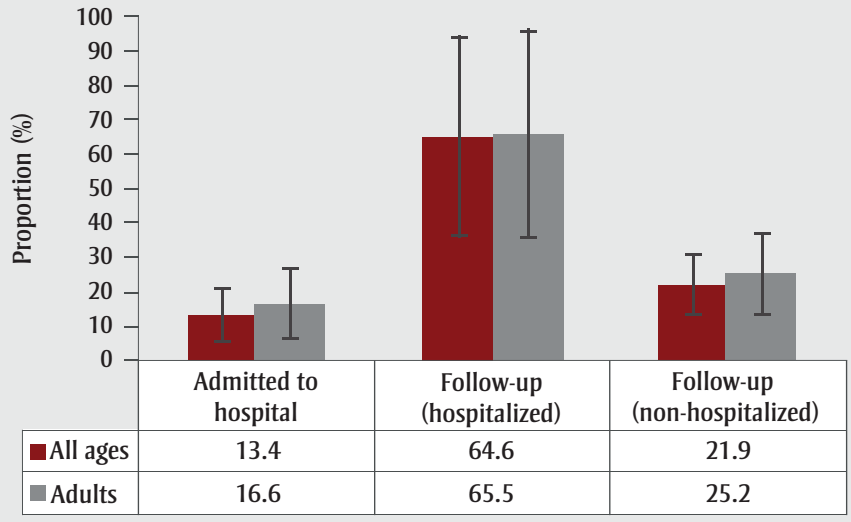
Notes: The youth category reflects ages 12 to 17 years, and the adults category reflects ages 18 years and above.
Estimates for traumatic brain injury are reported among all Canadians who reported any type of injury.
Estimates of those treated at the ED are calculated among those who reported a TBI and who reported receiving care within 48 hours of injury.
Estimates of annual percent change estimated from generalized logistic models.

outside of those diagnosed within an ED is a strength of the present study. Furthermore, since most TBIs are not fatal (only about 3% of TBI cases are[14]), our findings apply to the vast majority of TBIs in Canada.

Conclusion

There has been a significant increase in the incidence of reported TBIs over the past decade.[2] While these may have been influenced by a variety of factors, these trends call attention to how individuals manage their TBIs. Our observation of changes in where individuals reporting a TBI sought care is important, and the decreasing use of EDs among youth appears to be one such notable change in recent years. Building capacity among relevant professionals to identify TBIs would thus be beneficial given this changing landscape.

FIGURE 1
Self-reported estimates of follow-up care and hospitalization among Canadians reporting a traumatic brain injury, CCHS, 2014



Source: CCHS, 2014.

Abbreviation: CCHS, Canadian Community Health Survey.

Notes: Estimates for youth were too unstable to report. Estimates for adults reflect individuals ages 18 years and above.
Estimates for traumatic brain injury are reported among all Canadians who reported any type of injury.
Estimates of self-reported follow-up care (hospitalized) refer to respondents who reported they were currently receiving follow-up care and who said they received treatment within 48 hours and were hospitalized.
Estimates of self-reported follow-up care (non-hospitalized) refers to respondents who reported they were currently receiving follow-up care and who said they received treatment within 48 hours and who reported that they were not hospitalized.

Conflicts of interest

All authors declare no conflicts of interest.

Author contributions

All authors have read and approved of the content of this article. DPR was involved in data analysis, interpretation, and manuscript preparation. SM was involved in data interpretation as well as manuscript preparation and GJ and WT were involved in manuscript preparation.

References

1. MacLeod L. Bill 149: Rowan's Law Advisory Committee Act, 2016. Legislative Assembly of Ontario. 2016; Chapter Number: S.O. 2016 C.11.

2. Rao DP, McFaull S, Thompson W, et al. Trends in self-reported traumatic brain injury among Canadians, 2005–2014: a repeated cross-sectional analysis. CMAJ Open. 2017:5(2):E301-E307. doi: 10.9778/cmajo.20160115.

3. Feinstein A, Rapoport M. Mild traumatic brain injury: the silent epidemic. Can J Public Health. 2000;91: 325-6,332.

4. Tator CH. Concussions and their consequences: current diagnosis, management and prevention. CMAJ. 2013;185:975-979. doi: https://doi.org/10.1503/cmaj.120039.

5. Zemek R, Barrowman N, Freedman SB, et al. Pediatric Emergency Research Canada (PERC) Concussion Team. Clinical Risk Score for Persistent Postconcussion Symptoms Among Children With Acute Concussion in the ED. JAMA. 2016;315:1014-1025.

6. Bey T, Ostick B. Second impact syndrome. West J Emerg Med. 2009; 10:6-10.

7. McCrory P, Meeuwisse W, Aubry M, et al. Consensus statement on concussion in sport–the 4th International Conference on Concussion in Sport held in Zurich, November 2012. Clin J Sport Med. 2013;23:89-117. doi: 10.1136/bjsports-2013-092313.

8. Guskiewicz KM, Broglio SP. Sport-related concussion: on-field and sideline assessment. Phys Med Rehabil Clin N Am. 2011;22:603-17, vii.

9. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport-the 5th international conference on concussion in sport held in Berlin, October 2016. Br J Sports Med. 2017;51(11):838-847. doi: 10.1136/bjsports-2017-097699.

10. Parachute. Canadian Guideline on Concussion in Sport. 2017. Available from: http://www.parachutecanada.org/injury-topics/item/canadian-guideline-on-concussion-in-sport

11. Taylor AM, Nigrovic LE, Saillant ML, et al. Trends in Ambulatory Care for Children with Concussion and Minor Head Injury from Eastern Massachusetts between 2007 and 2013. J Pediatr. 2015;167:738-744. doi: 10.1016/j.jpeds.2015.05.036.

12. Zemek RL, Grool AM, Duque DR, et al. Annual and Seasonal Trends in Ambulatory Visits for Pediatric Concussion in Ontario between 2003 and 2013. J Pediatr. 2017;181:222-228.e2. doi: 10.1016/j.jpeds.2016.10.067.

13. Beland Y. Canadian community health survey–methodological overview. Health Rep. 2002;13:9-14.

14. Faul M, Xu L, Wald MM, et al. Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002–2006. 2010.

15. Arbogast KB, Curry AE, Pfeiffer MR, et al. Point of Health Care Entry for Youth With Concussion Within a Large Pediatric Care Network. JAMA Pediatr. 2016;170(7):e160294. doi: 10.1001/jamapediatrics.2016.0294.

16. Angus Reid Institute: Public Interest Research. One-in-five Canadians say they've suffered a concussion playing sports; most before turning 18. Available at: http://angusreid.org/concussion-in-sport/

17. Lincoln AE, Caswell SV, Almquist JL, et al. Trends in concussion incidence in high school sports: a prospective 11-year study. Am J Sports Med. 2011;39(5):958-63. doi: 10.1177/0363546510392326.

18. Parachute Canada. Return to play guidelines after a concussion. Available at: http://www.coach.ca/files/returntoplayguidelines.pdf.

19. Cancelliere C, Hincapie CA, Keightley M, et al. Systematic review of prognosis and return to play after sport concussion: results of the International Collaboration on Mild Traumatic Brain Injury Prognosis. Arch Phys Med Rehabil. 2014;95(3 Suppl): S210-29. doi: 10.1016/j.apmr.2013.06.035.

20. Gabbe BJ, Finch CF, Bennell KL, et al. How valid is a self reported 12 month sports injury history? Br J Sports Med. 2003;37:545-547. doi: 10.1136/bjsm.37.6.545.

https://doi.org/10.24095/hpcdp.38.3.06

With thanks to our 2017 peer reviewers

We are grateful to the following people for their significant contribution to *Health Promotion and Chronic Disease Prevention in Canada* as peer reviewers in 2017. Their expertise ensures the quality of our journal and promotes the sharing of new knowledge among peers in Canada and internationally.

Amanuel Abajobir
Alexander Allen
Laura Arbour
Christopher Ardern
Chris Barrington-Leigh
Marco Battaglia
William Bellew
Nicholas Birkett
Tara Boelsen-Robinson
Lisa Bourque Bearskin
Randy Boyes
Mayilee Canizares
Nick Cavill
Bernard Choi
Brydie Clarke
Adam Cole
Yang Cui
Jon Doan
Rachel Engler-Stringer
Gagan Fervaha
Shawn Fraser
Daniel Fuller
Didier Garriguet
Gary Goldfield
Samantha Gontijo-Guerra
Samantha Goodman
Linda Greene-Finestone
Grant Handrigan
Anthony Hanley
Rhona Hanning
Erin Hoare
Erin Hobin
Sheilah Hogg-Johnson
Wendy Hovdestad
Ilene Hyman
Brian Hyndman
Amanda Jones
Glenn Keays
Ryan David Kennedy
Alexandra Kent
Sharon Kirkpatrick
Primoz Kocbek
Vicki Kristman
Eric Lavigne
Justine Leavy
Yann Le Bodo
Eun-Young Lee
Isra Levy
Shiliang Liu
Emily Lowthian
Peter Macaulay
Zack Marshall
Lindsay McLaren
Amy Metcalfe
Stephanie Montesanti
Howard Morrison
Yeeli Mui
Patti-Jean Naylor
Cory Neudorf
Alvaro Osornio-Vargas
Marie-Claude Paquette
Alex Price
Stephanie Prince Ware
Kim Raine
Liam Remillard
Kim Robien
Kaley Roosen
Laura Rosella
Sarah Rotz
Richard Sadler
Travis Saunders
Mary Scourboutakos
Gabriel Shapiro
Shahin Shooshtari
Kelly Skinner
Anna Syrowatka
Valerie Tarasuk
Joan Tranmer
Cynthia Weijs
Ellen M. Wells
Russell Wilkins
Jinhui Zhao

Other PHAC publications

Researchers from the Public Health Agency of Canada also contribute to work published in other journals. Look for the following articles published in 2017:

Booth RA, Jiang Y, **Morrison H, Orpana H, Rogers Van Katwyk S**, Lemieux C. Ethnic dependent differences in diagnostic accuracy of glycated hemoglobin (HbA1c) in Canadian adults. Diabetes Res Clin Pract. 2017. doi: 10.1016/j.diabres.2017.11.035.

Chaput J-P, Colley RC, Aubert S, Carson V, Janssen I, **Roberts KC**, et al. Proportion of preschool-aged children meeting the Canadian 24-Hour Movement Guidelines and associations with adiposity: results from the Canadian Health Measures Survey. BMC Public Health. 2017;17. doi: 10.1186/s12889-017-4854-y.

Tremblay MS, Chaput J-P, Adamo KB, […] **Jaramillo Garcia A**, et al. Canadian 24-Hour Movement Guidelines for the early years (0-4 years): an integration of physical activity, sedentary behaviour, and sleep. BMC Public Health. 2017;17. doi: 10.1186/s12889-017-4859-6.

Willis C, **Greene J**, Riley B. Understanding and improving multi-sectoral partnerships for chronic disease prevention: blending conceptual and practical insights. Evid Policy. 2017;13(4):623-645. doi: 10.1332/174426417X15090122455415.