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Chronic Diseases in Canada
Population and Public Health Branch
Health Canada, 130 Colonnade Road
Address Locator: 6501G
Ottawa, Ontario   K1A 0K9

Fax: (613) 952-7009
E-mail: cdic-mcc@hc-sc.gc.ca

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The Medical Care Cost of Childhood and Adolescent Cancer in Manitoba, 1990–1995

Wei Luo, Rachel Lane, Kent Stobart, Howard Morrison, Dena Schanzer, Ronald Barr and Mark Greenberg

Abstract

The main purpose of this study is to estimate the medical care costs of childhood and adolescent cancer in Manitoba, and to determine the elements that influence these costs. Retrospective chart reviews were done to obtain all the information. A total of 118 childhood (age 0–14 years) and 41 adolescent (age 15–19 years) cancer patients were included. For childhood cancer, in-patient hospitalizations accounted for 59% of the total cost, followed by bone marrow transplant (BMT) (9%), medications (8%), laboratory investigations (7%) and physician fees (7%). For adolescent cancer, in-patient hospitalization accounted for 37% of the total cost, followed by BMT (25%), physicians' fees (11%), medications (9%) and laboratory investigations (7%). Overall, the average cost for the first, second and third year following diagnosis was $50,902 (median 35,708), $13,939 (4,127) and $6,769 (2,565) respectively for childhood cancer patients, and $57,354 (24,192), $16,888 (3,267) and $3,436 (3,267) respectively for adolescent cancer patients. Further work involving long-term data linkage of medical charts with hospital and clinic financial billing codes is needed to provide more accurate estimates of the costs of childhood and adolescent cancer care.

Key Words: *adolescent cancer; childhood cancer; cost*

Introduction

Cancer is the most common fatal disease of childhood and adolescence; only accidents kill more Canadian children and adolescents aged 1–19 years.[1] Recent improvements in the prognosis for children with cancer, due to advances in treatment,[2] has focused attention on the burden of therapy on the children and their families,[3] and the long-term effects of the disease and its management.[4–5] Costs to the health care system have been less well studied.

Although reports on childhood cancer often include adolescents, teenagers have a mix of cancers which differ from those of both adults and children. The embryonal cancers found in young children (e.g., neuroblastoma, retinoblastoma, and hepatoblastoma) are almost unheard of among those aged 15–19 years, while the epithelial carcinomas of adults (e.g., lung, breast, colon) are equally rare. Patterns of care also differ, as adolescent cancer patients may be treated either in pediatric oncology centres using pediatric protocols or adult oncology centres using either pediatric or adult protocols. Adolescent patients are much less likely than children with cancer to be enrolled in clinical trials.[6]

To date, there have been no published estimates of the direct medical care costs of childhood and adolescent cancer in Canada. Information on the cost of treating childhood and adolescent cancer patients may be useful to health care planners to direct and plan priorities of childhood cancer control programs and to make the most effective use of limited medical care budgets.

Only a handful of studies have been conducted to estimate the medical care cost of childhood and adolescent cancer. In 1983, Lansky et al.[7] reported medical costs for childhood cancer patients in the US that ranged from $100 (histiocytosis X) to $1800 (lymphoma) per month. In-patient hospital charges were the most expensive component (mean cost $400 per month), followed by pediatricians' fees ($100 per month). The estimation of medical costs was based on a randomly chosen four-week period in the years 1978 to 1980 and the sample size (N= 64) was small.

In 1985, also in the US, Bloom et al.[8] estimated that the mean cost of care for children with cancer was $29,708 per patient-year, based on a followup time of six months. The authors concluded that direct medical expenditures were about two thirds of the total costs.

Birenbaum and Clarke-Steffen[9] conducted a study (N= 19 families) to describe the health care costs for childhood cancer patients in the terminal phase of illness in the US in 1987. Direct health care costs, defined as expenses billed for the provision of health care services for the dying child from the time of admission to the study until death, accounted for $8,456, or 76% of the total costs. The median costs of out-patient and in-patient services were $949 (range: $55–$2,138) and $6,877 (range: $0–$79,027) respectively.

Unlike the US, which relies upon a mix of privately and publicly funded health care, Canada has a publicly funded health care system which provides universal access to hospitalization and physician care. How-

Author References

Wei Luo, Rachel Lane, Howard Morrison, Dena Schanzer, Cancer Division, Population and Public Health Branch, Health Canada, Ottawa, Ontario
Dr. Kent Stobart , CancerCare Manitoba, Winnipeg, Manitoba
Dr. Ronald Barr, McMaster University, Hamilton, Ontario
Dr. Mark Greenberg, Hospital for Sick Children, Toronto, Ontario
Correspondence: Wei Luo, Cancer Division, Population and Public Health Branch, Health Canada, Tunney's Pasture, PL 0601C1 Ottawa, Ontario, Canada K1A 0L2; Fax: (613) 941-1732; E-mail: Wei_Luo@hc-sc.gc.ca

ever, the nature of childhood cancer care in Canada and the US is similar; in both countries, treating institutions are almost always members of the two major childhood cancer cooperative study groups (Pediatric Oncology Group and Children's Cancer Group), that have merged recently to form the Children's Oncology Group.

The main purposes of this population-based study are to estimate the medical care costs of childhood and adolescent cancer in Canada, and to determine which elements influence these costs from the perspective of health care providers. The study was undertaken in Winnipeg, the capital city of the province of Manitoba, as all children with cancer in the province are referred to the Children's Hospital in Winnipeg. All of the costs included in this study refer to 1992 Canadian dollars.

Materials and methods

Medical care components

Information on all newly diagnosed cases of cancer (excluding skin carcinomas) in children and adolescents aged 0–19 years between January 1, 1990 and December 31, 1992 was obtained retrospectively from the population-based Manitoba Cancer Registry. These cancer cases were followed for three years from diagnosis, up to December 31, 1995. The diagnoses of childhood (age 0–14 years) and adolescent (age 15–19 years) cancer were grouped using the International Classification of Childhood Cancer[10] which is based on the 2nd edition of the *International Classification of Disease for Oncology*[11] morphology and topography codes.

Approximately 85% of the population of Manitoba lives within 100 kilometres of Winnipeg. As a result, childhood cancer care in Manitoba is centralized; all childhood cancers in the province are treated at the Children's Hospital in Winnipeg. Adolescent cancer patients are treated either in the Children's Hospital or in an adult setting at the Health Sciences Centre, also in Winnipeg. At the time of this study, the use of outreach centres in Manitoba was minimal.

Medical care utilization information was collected in five settings: the Children's Hospital in Winnipeg, the Health Sciences Centre in Winnipeg, the CancerCare Manitoba clinic, and the emergency room (ER) of the Children's Hospital and the Health Science Centre. All medical care information was abstracted retrospectively from the patients' medical charts using a data abstraction form developed for this purpose by a research assistant at the Manitoba cancer registry.

Medical care information was collected for the following components: length of hospital stay; medications administered (antibiotics, chemotherapy and other supportive pharmacological treatment); laboratory tests performed; physician visits by type of visit (clinic, hospital, emergency room, consultation or administration of chemotherapy); diagnostic tests (radiology and other diagnostic tests); diagnostic and operative surgeries; blood products administered and radiation treatments; and the type of visit: in-patient, outpatient or emergency room. Bone marrow transplantation was costed separately.

Information on any visits to an outreach centre or followup visits to medical professionals in private practice was not collected in the study.

Unit costs

We based our study on costs in 1992, which was roughly the midpoint of the study period. A cost was assigned to each component abstracted from the patient's chart, along with the date when the cost was incurred. Total costs were obtained by summing the individual costs. To avoid double counting, each cost component was estimated independently.

Bone marrow transplant

The average cost of a bone marrow transplant (BMT) was estimated by the department of pediatric BMT in the Children's Hospital at $108,990. This cost included comprehensive care for the time of transplant and for a 100 day period thereafter. To avoid double counting, no medical care utilization data were collected on the questionnaire during the BMT period. As a result, medical care costs are included within the comprehensive care costs component of total BMT costs.

Laboratory investigations

The costs per 100 workload units, which included salaries, supplies and overhead for each type of laboratory test, were obtained from Westman Laboratory Services (operated by Manitoba Health),[12] while the workload units for each specific test were obtained from the Canadian Institute for Health Information.[13] Workload units are defined as the number of minutes of direct labour time required to perform a specific test or procedure. The cost of a specific test was calculated by multiplying the cost per workload unit by the number of workload units utilized. The hospital pays for laboratory tests based on workloads, so unit costs were not available and had to be estimated.

Medications and blood products

The costs of chemotherapeutic agents, antibiotics, and other supportive pharmacological treatment were obtained from the Health Sciences Centre Drug Formulary, 1991–1992.[14] The price of blood products was provided by the Blood Services Centre[15] in 1994 and 1995 dollars. These were converted to 1992 dollars using the pharmaceutical component of the Industrial Product Price Index.[16]

Physician fees

There were five categories of physician payments in this study: consulting physician fees, hospital in-patient fees, ER physician fees, physician fees for chemotherapy, and sessional physician fees.

Consulting physician fees were obtained from the billable fee-for-service charges (e.g., surgeons, radiologists).[17] Hospital in-patient physician fees per patient day were calculated based on the concomitant care fees, (i.e., hospital physician fees varied by days of hospitalization).[17] ER physician fees for each visit to ER were based on the fee per ER visit.[17] Physician fees for the administration of chemotherapy were obtained from the Manitoba Health Services Insurance Plan.[17] The CancerCare Manitoba clinic has full-time pediatric oncologists on staff to deliver outpatient care; this care was billed as sessional physician fees, which were based on the frequency of clinic visits during the study period.

Diagnostic tests, surgeries, radiation therapy

The unit costs of these procedures were obtained from the Manitoba Health Services Insurance Plan.[17]

In-patient hospital care

Hospital in-patient charges were based on the Children's Hospital per diem rate, which included the cost of room and board, nursing, intensive care, radiology tests, laboratory investigations, drugs, use of the operating rooms and any additional treatment. From these charges we were able to separate out the costs of radiology tests, laboratory investigations, drugs and use of operating rooms. Because we were unable to determine the specific in-patient hospital care costs of room and board, nursing, intensive care and additional treatment associated with treating children with cancer, and because these costs for children with cancer may have differed from the costs of other hospitalized children, we conducted sensitivity analyses based on adding or subtracting 30% of the reported costs.

Results

A total of 118 childhood cancer patients (57 boys and 61 girls) and 41 adolescent cancer patients (23 boys and 18 girls) were included in the study with a mean age at diagnosis of 6.7 and 17.0 years respectively. Table 1 presents the distribution of cancers in childhood and adolescence by diagnosis. The four most common childhood cancers were central nervous system (CNS) tumours (N= 33), leukemias (N= 30), lymphomas (N= 14) and sympathetic nervous system (SNS) tumours (N= 13), which accounted for more than 70% of all childhood cancer cases. In comparison, lymphomas (N= 12), CNS tumours (N= 7), soft-tissue sarcomas (N= 7) and leukemias (N= 6) were the four most common cancers among adolescents, accounting for 78% of all cases. At the end of three years of follow-up, 91 childhood cancer patients and 31 adolescent cancer patients were alive, corresponding to a three-year survival rate of 77% and 76% respectively.

TABLE 1

Distribution of childhood and adolescent cancers cases by cancer type, Manitoba, 1990–1992

Cancer type (ICCC Code)	Number of cases	
	Childhood (% of total)	Adolescent (% of total)
LEUKEMIA (I)	30 (25.4)	6 (14.6)
Acute lymphoblastic leukemia (Ia)	30	3
Acute non-lymphoblastic leukemia (Ib)	3	1
Chronic myeloid leukemia (Ic)	0	2
LYMPHOMA (II)	14 (11.9)	12 (29.3)
Hodgkin's disease (IIa)	8	7
Non-Hodgkin's lymphoma (including Burkitt's lymphoma) (IIb)	6	5
CENTRAL NERVOUS SYSTEM (III)	33 (28.0)	7 (17.1)
Ependymoma (IIIa)	5	0
Astrocytoma (IIIb)	11	5
Primitive neuroectodermal tumour (IIIc)	9	0
Other gliomas (IIId)	3	1
Miscellaneous specified intracranial and intraspinal neoplasms (IIIe)	5	1
SYMPATHETIC NERVOUS SYSTEM (IV)	13 (11.0)	0 (0.0)
Neuroblastoma (IVa)	12	0
Other sympathetic nervous system tumours (IVb)	1	0
RETINOBLASTOMA (V)	2 (1.7)	0 (0.0)
RENAL (VI)	5 (4.2)	2 (4.9)
Wilms' tumour (VIa)	5	0
Renal carcinoma (VIb)	0	2
HEPATIC (VII)	1 (0.9)	0 (0.0)
Hepatoblastoma (VIIa)	0	0
BONE (VIII)	4 (3.4)	4 (9.8)
Osteosarcoma (VIIIa)	3	2
Ewing's sarcoma (VIIIc)	1	2
SOFT-TISSUE SARCOMAS (IX)	9 (7.6)	7 (17.1)
Rhabdomyosarcoma (IXa)	3	5
Fibrosarcoma, neurofibrosarcoma (IXb)	4	2
Other specified soft tissue sarcomas (IXd)	1	0
Unspecified soft tissue sarcomas (IXe)	1	0
GERM CELL (X)	1 (0.9)	2 (4.9)
Other and unspecified non-gonadal germ cell tumours (Xb)	1	0
Gonadal germ cell tumours (Xc)	0	2
CARCINOMA (XI)	6 (5.1)	1 (2.4)
Thyroid carcinoma (XIb)	1	0
Malignant melanoma (XId)	3	0
Other and unspecified carcinomas (XIf)	2	1
TOTAL	118 (100.0)	41 (100.0)

FIGURE 1
Distribution of total medical care costs of childhood and adolescent cancer, Manitoba, 1990–1995

Age 0–14 years
Total cost = $8,412,448

Age 15–19 years
Total cost = $3,053,149
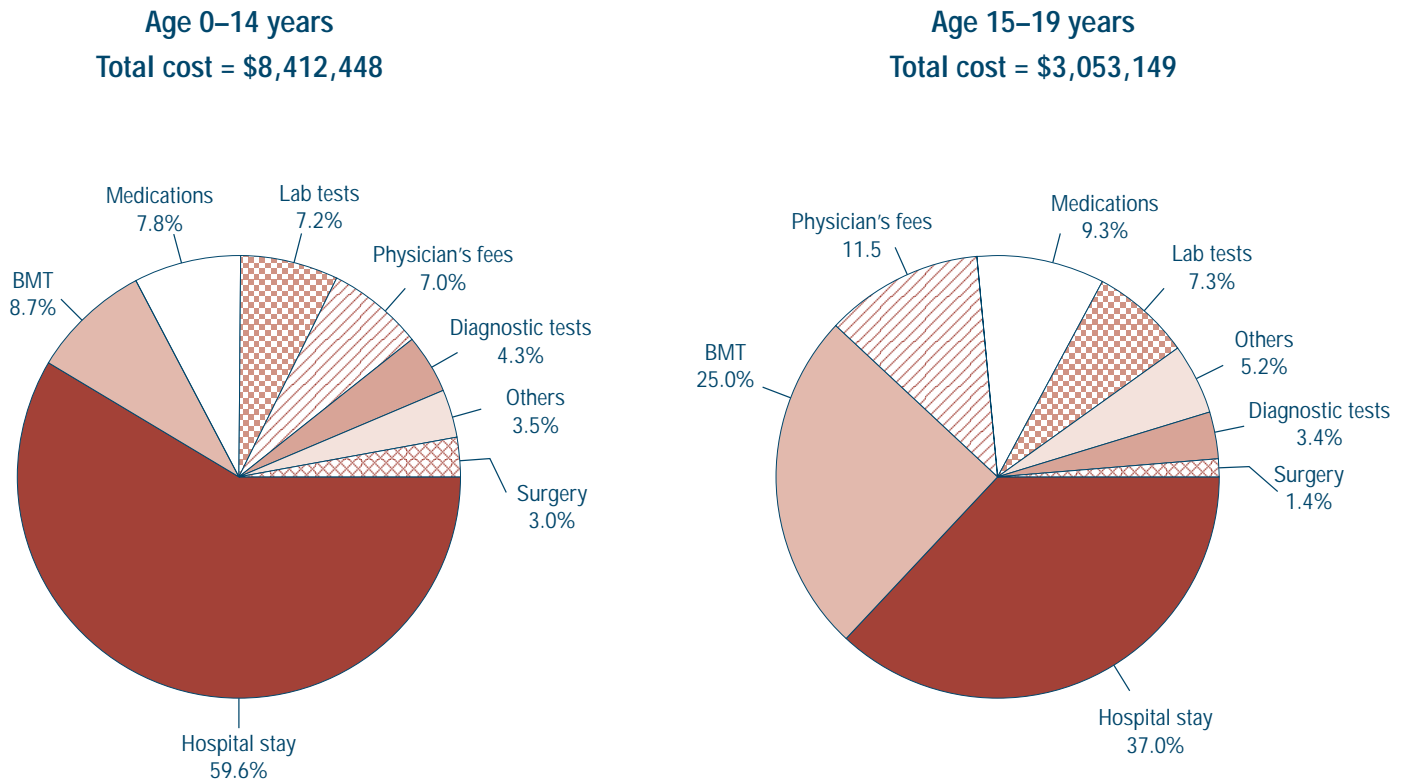
Diagnostic tests: radiology and other diagnostic tests.

Medications: chemotherapeutic agents, antibiotics and others.

Surgery: including both diagnostic and operative.

Others: blood products and radiation therapy.

For childhood cancer, in-patient hospitalizations accounted for 59% ($4.4 million) of the total cost, followed by BMT (9%, $653,000), medications (8%, $587,000), laboratory investigations (7%, $544,000) and physician fees (7%, $530,000) (Figure 1). Adolescent cancer in-patient hospitalizations accounted for 37% ($1.1 million) of the total cost, followed by BMT (25%, $762,930), physician fees (11%, $350,402), medications (9%, $283,682) and laboratory investigations (7%, $221,572) (Figure 1). Thirteen cancer patients (six childhood, seven adolescent) received BMTs during the study period; all but four were undertaken to treat leukemia. Sensitivity analyses indicated that the in-patient hospitalization cost for childhood cancer could have been as high as $5.7 million or as low as $3.1 million based on adding or subtracting 30% of the total in-patient hospitalization cost, re-

spectively. For cancer in adolescents, the in-patient hospitalization cost could have been as high as $1.4 million or as low as $0.8 million based on adding or subtracting 30% of total adolescent in-patient hospitalization costs respectively.

The average number of hospitalization days differed dramatically by diagnosis for both childhood and adolescent cancers (Table 2). Among the more common childhood cancers (those listed in Table 2), the average number of days was highest for SNS tumours, and lowest for lymphomas. The average number of days was highest for leukemias, and lowest for CNS tumours for adolescent cancers. Hospitalizations were concentrated in the first year after diagnosis (data not shown).

Overall, the average costs for the first, second and third years following diagnosis

were $50,902, $13,939 and $6,769 respectively for childhood cancers (Table 3). In comparison, the average costs for the first, second and third years following diagnosis were $57,354, $16,888 and $3,437 respectively for the adolescent cancers (Table 4). However, these costs varied by diagnosis and by vital status at the end of the study period. Among the four most common childhood cancer sites, SNS tumours had the highest average cost ($86,715) for the first year after diagnosis followed by leukemias ($59,595), CNS tumours ($42,859) and lymphomas ($35,834). The average costs of treating leukemias, lymphomas and CNS tumours dropped by 67%, 39% and 71% respectively between the first and second years, while for all other cancer sites (except kidney, for which costs declined 55%), there was more than a 90% decline between these time periods.

TABLE 2
Average number of days of hospitalization by diagnosis, Manitoba, childhood and adolescent cancer cases diagnosed 1990–1992

Type of neoplasm	Childhood cancer				Adolescent cancer			
	# of cases	Mean # of days	Median	SD[§]	# of cases	Mean # of days	Median	SD[§]
Leukemia	30	53.3	42.5	42.9	6	96.5	102.0	38.4
Lymphoma	14	37.9	32.5	28.6	12	46.5	11.5	63.0
CNS	33	47.9	44.6	46.9	7	11.4	8.0	10.2
SNS	13	82.4	96.0	54.8	0	–	–	–
Malignant bone	0	–	–	–	4	63.5	57.3	46.0
Soft-tissue sarcomas	9	30.6	39.3	21.6	7	67.1	92.0	49.8
Wilms' tumour	5	61.2	38.0	70.1	0	–	–	–
Others	14[†]	44.9[†]	32.0	42.0	5[‡]	17.9[‡]	7.0	27.7
Total	118	63.2	51.2	57.8	41	49.5	25.5	51.5

[§] standard deviation

[†] retinoblastoma; liver; bone and germ cell tumours; and carcinomas

[‡] renal and germ cell tumours; and carcinomas

TABLE 3
Average cost per patient by year since diagnosis and cancer types, Manitoba aged 0–14 years, diagnosed 1990–1992

Diagnosis	Year 1			Year 2			Year 3			Total		
	N	Cost ($)	Median	N	Cost ($)	Median	N	Cost ($)	Median	N	Cost ($)	Median
Leukemia	30	59,595	46,004.8	27	19,795	5,623.6	25	5,641	3,656.4	30	85,031	55,171.9
Lymphoma	14	35,834	29,803.4	13	21,752	3,753.1	13	4,962	1,692.8	14	62,547	43,442.5
CNS	33	42,859	29,541.6	23	12,619	2,325.1	21	15,359	2,553.8	33	70,836	44,527.2
SNS	13	86,715	96,216.5	9	6,650	4,625.2	9	1,287	495.8	13	94,652	99,400.2
Wilms'	5	43,962	33,161.3	5	19,975	8,934.5	5	6,173	975.4	5	70,110	46,214.6
Bone	4	65,698	64,882.4	4	6,360	6,166.5	3	851	850.4	4	72,909	71,646.3
Soft-tissue sarcoma	9	33,794	20,383.1	8	1,370	930.2	7	1,642	991.6	9	36,807	22,640.9
Carcinomas	6	12,738	3,048.4	5	1,643	1,639.4	5	923	897.1	6	15,300	6,172.0
Other*	4	84,986	73,004.1	4	2,426	2,101.3	4	1,069	1,032.3	4	88,481	75,745.6
Total	118	50,902	35,708.4	98	13,939	4,127.2	92	6,769	2,656.6	118	71,610	56,252.2

* including retinoblastoma, liver and germ cell tumours

Among the four most common adolescent cancer sites, leukemias had the highest average cost ($165,800) for the first year following diagnosis, reflecting the use of BMT, followed by soft-tissue sarcomas ($73,355), lymphomas ($36,092) and CNS tumours ($16,365). The average cost of treating lymphomas, CNS tumours and leukemias dropped by 30%, 95% and 89% respectively between the first and second years, while for all other cancer sites except bone tumour, for which costs declined 53%,

there was more than an 85% decline between these time periods.

For both childhood and adolescent cancer patients, the average medical costs in the first year after diagnosis for patients who survived were lower than for those who did not (Figure 2). This was particularly true of childhood leukemia cases; those who died had medical costs ($89,707) which were almost double those of the cases who survived ($52,066). For adolescent soft-tissue sarcomas, those who died had medical

costs ($103,804) that were triple those of the cases who survived ($32,757). For childhood cancer cases, no real differences were observed for lymphomas ($36,586 versus $35,708) and SNS tumours ($88,316 versus $86,182), while cases with CNS tumours who survived actually cost more than those who died ($44,913 versus $39,435). The average cost of treating childhood bone and soft-tissue sarcoma patients who died was more than double that of those who survived, as were the treatment costs of adolescent CNS tumours, bone tumours

TABLE 4
Average cost per patient by year since diagnosis, and cancer types, Manitoba Aged 15–19 years, diagnosed 1990–1992

Diagnosis	Year 1			Year 2			Year 3			Total		
	N	Cost ($)	Median	N	Cost ($)	Median	N	Cost ($)	Median	N	Cost ($)	Median
Leukemia	6	165,800	192,895	5	22,281	24,235	5	7,857	4,863	6	195,938	220,123
Lymphoma	12	36,092	20,977	12	25,258	22,263	12	4,463	1,800	12	65,813	27,871
CNS	7	16,365	12,315	6	956	1,220	6	862	950	7	18,203	14,313
Bone	4	52,502	51,806	3	32,808	37,655	2	2,649	2,649	4	87,960	85,114
Soft-tissue sarcoma	7	73,355	77,798	5	13,885	17,356	3	1,818	1,655	7	89,058	100,057
Other#	5	17,115	8,794	4	720	872	4	301	352	5	18,136	8,794
Total	41	57,354	24,192	35	16,888	3,267	32	3,437	3,267	41	77,679	38,820

including renal and germ cell tumours, and carcinomas

FIGURE 2
The average cost of the first year after diagnosis by vital status and cancer site for childhood and adolescent cancer
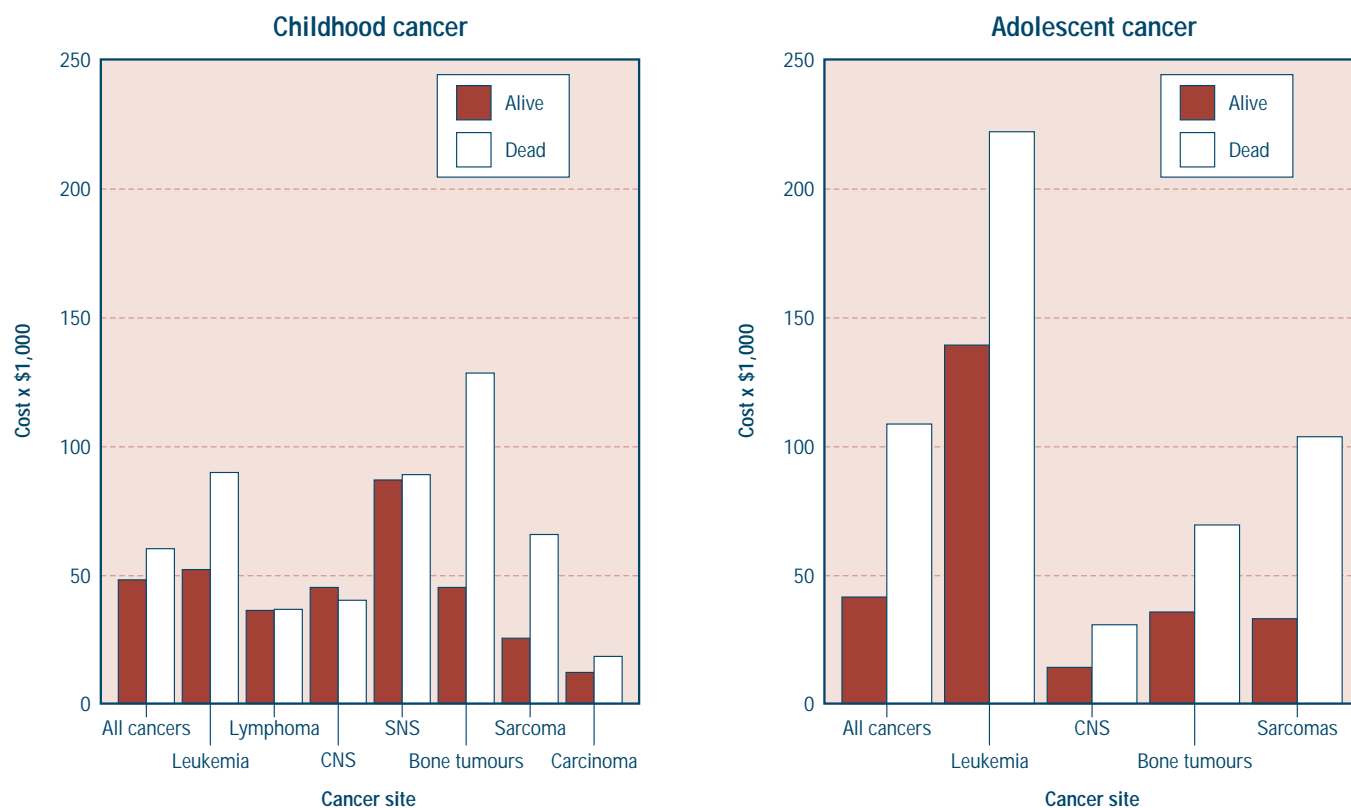
and leukemia patients. Higher costs for cancer patients who did not survive reflect more hospitalization days than those who survived.

Discussion

To reduce the possibility of selection bias, which may affect hospital-based studies, this study used population-based cancer registry data to identify all children and adolescents aged 0–19 years diagnosed with malignant neoplasms in Manitoba. Three years of incident cases provided enough data to allow costs to be categorized by cancer type, while three years of followup provided a good overview of the various components of care.

In our study, the most expensive component was in-patient care, which accounted for 59% and 37% of the total costs for childhood and adolescent cancer respectively. The other expensive components were: BMT, medications, laboratory investigations and physicians' fees for both childhood and adolescent cancers. These rela-

tive proportions are similar to those noted by Bloom et al.[8]

We estimated the costs for five childhood cancer patients for whom information was unavailable in the medical charts, based on treatment procedures received for patients with similar diagnoses. It is likely that this resulted in a minor underestimation of costs, as only major treatment components were costed (e.g., surgical procedures).

Similarly, 30% of laboratory costs had to be estimated because there was insufficient information on the medical charts to determine which specific tests were undertaken. Expenditures for these tests were imputed from the mean cost of tests for the same diagnostic category (e.g., renal, microbiological) for both childhood and adolescent cancer patients. This may also mean that costs were underestimated.

Likewise, while hospital care was assigned from a global hospital figure, cases of cancer in childhood and adolescence have a higher-than-average relative intensity weighting (a measure of the complexity of the care required and so the nursing hours involved and so the cost), and this may have also contributed to an underestimation of the generalizable average costs. For this reason, we conducted sensitivity analyses by adding and subtracting 30% of the reported hospital care. The usefulness of such sensitivity analyses may be appreciated from a earlier Canadian report of an economic evaluation of allogeneic BMT.[18]

We did not include the costs of outreach centres (rural areas) and private health professionals. Those costs should be minimal as almost all childhood and adolescent cancer cases were treated in the Children's Hospital or the Health Sciences Centre in Winnipeg.

The average medical cost of patients who survived to the end of three years was less than that of patients who did not survive, especially for childhood leukemia and adolescent soft-tissue sarcoma patients. Much of the difference occurred because patients who died had more days of hospitalization than patients who survived. Successful treatment not only saves lives, but reduces the cost of treating childhood and adolescent cancer patients over the initial three years.

Among the four most common types of childhood cancers, costs were highest in the first year for SNS tumours (almost all of which were neuroblastomas), followed by leukemias, CNS tumours and lymphomas. The high cost of treating neuroblastomas reflects the greater number of days of hospitalization for these patients. This finding differs from those of Lansky et al.[4] and Bloom et al.;[8] the leading average costs in these studies being for lymphomas and malignant bone tumours respectively. However, the present study represented the costs in the initial three years after diagnosis while the Lansky study focused on children with cancer treated as outpatients. In the study by Bloom et al., hospital visits were used to identify participants rather than using a registry of all children with malignant diseases. This could have reduced the probability of including low-cost patients who return for visits less frequently than once every six months.

In contrast, among the four most common types of adolescent cancers, costs were highest in the first year after diagnosis for leukemias, followed by soft-tissue sarcomas, lymphomas and CNS tumours. The high cost of treating leukemia reflects the greater number of days of hospitalization and relatively high use of BMT for these patients.

The present study only estimated the medical care cost of childhood and adolescent cancers for a three-year period after diagnosis. We were not able to estimate long-term increased health care costs in long-term survivors, or to estimate the additional costs which some survivors of childhood and adolescent cancer may require for special education or home care services, or the non-medical costs borne by the families of these children and adolescents.[3,19,20]

Long term data linkage of medical charts with hospital and clinic financial billing codes would provide more accurate and valuable information for estimating the costs of childhood cancer care.

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Work and life stressors and psychological distress in the Canadian working population: A structural equation modelling approach to analysis of the 1994 National Population Health Survey

Donald C Cole, Selahadin Ibrahim, Harry S Shannon, Fran E Scott and John Eyles

Abstract

Work stressors are increasingly recognized as potentially important determinants of mental health status. We examined such relationships using a structural equation modelling approach with data on adult, working Canadians who participated in the first wave of the National Population Health Survey (NPHS). Work stressors formed a composite construct with paths from psychological demands, decision latitude, work social support and job insecurity, each measured through a reduced version of the Job Content Questionnaire. Life stressors also formed a composite construct composed of chronic stressors and recent life events. Psychological distress was the outcome, mediated by the latent effect constructs of mastery and self-esteem. Work stressors had consistently positive total effects on distress (sum of standardized path coefficients from 0.004 to 0.153 across gender-occupation strata), with all of these effects mediated through reduced self-esteem and mastery (work stressors to these mediators: -0.188 to -0.413). Life stressors had larger positive total effects on distress (0.462 to 0.536), with the majority of these effects direct.

***Key Words**: Canada; cross-sectional; health surveys; mental health; models-statistical; occupational health; psychology-industrial; stress-psychological*

Introduction

Problem and research question

"Work stress" has been increasingly recognized both as a health outcome and as an important health determinant by the public health community. Analysis of the 1985 US National Health Interview Survey, for example, produced estimates of 11 million workers experiencing health-endangering levels of mental stress at work.[1] Although it is generally accepted that work stressors can adversely affect mental health,[2] some argue that life stressors are far more important than work stressors as determinants of psychological well-being.[3] Fortunately, the majority of researchers include both of these key domains in their research: work/job/intra-organizational stressors and non-work/family/life/extra-organizational stressors.[4–8] Psychological mediator variables such as self-esteem and mastery between stressors and health outcomes have been recognized as important to the understanding of stressor-health outcome relationships.[4,7] Yet the relative importance of each stressor domain and the extent of mediation by psychological variables is contested among health practitioners, workplace parties and policy makers. Hence, our research question was:

> "Among working Canadians, what relative contribution do work stressors and life stressors make to explaining variation in levels of distress, both directly and indirectly through self-esteem and mastery as mediating variables?"

We were further interested in assessing the extent to which stressor-distress relationships varied between genders[7,9] and across occupational strata.[10] We drew on the results of previous analyses of the NPHS 1994/5 data,[11–16] relevant scientific literature and current debates among workplace health practitioners to hypothesize a structural model linking the main constructs of interest. Our analysis was facilitated through the use of structural equation modelling (SEM) techniques that permit simultaneous estimation of both measurement and structural components.[17] Comparisons of model estimates by gender-occupational strata provided results to explore the extent to which gender-occupational contexts modulate the relationships of interest.

Formulation of the structural model

Organizational psychologists have long argued that a variety of factors are likely relevant to psychological well-being.[18] Work stressors, including lack of social support, have been associated with a variety of adverse health outcomes, including depression and distress.[19] Occupational health researchers have often used measures of depressive symptoms or psychological distress as indicators of job strain.[20] A wide variety of life stressors are also seen as contributing to adverse mental health in general and depression in particular.[21,22]

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Donald C Cole, Selahadin Ibrahim, Institute for Work & Health *and* Department of Public Health Sciences, Univeristy of Toronto, Toronto, Ontario
Harry S Shannon, Institute for Work & Health *and* Program in Occupational Health and Environmental Medicine, McMaster University, Hamilton, Ontario
Fran E Scott, John Eyles, Department of Clinical Epidemiology and Biostatistics *and* Institute of Environment and Health, McMaster University, Hamilton, Ontario
Correspondence: Dr Donald C Cole, Senior Scientist, Institute for Work & Health, 481 University Ave, Ste 800, Toronto, Ontario, Canada M5G 2E9;
Fax: (416) 927-4167; E-mail: dcole@iwh.on.ca

FIGURE 1
Hypothesized model (for all strata)
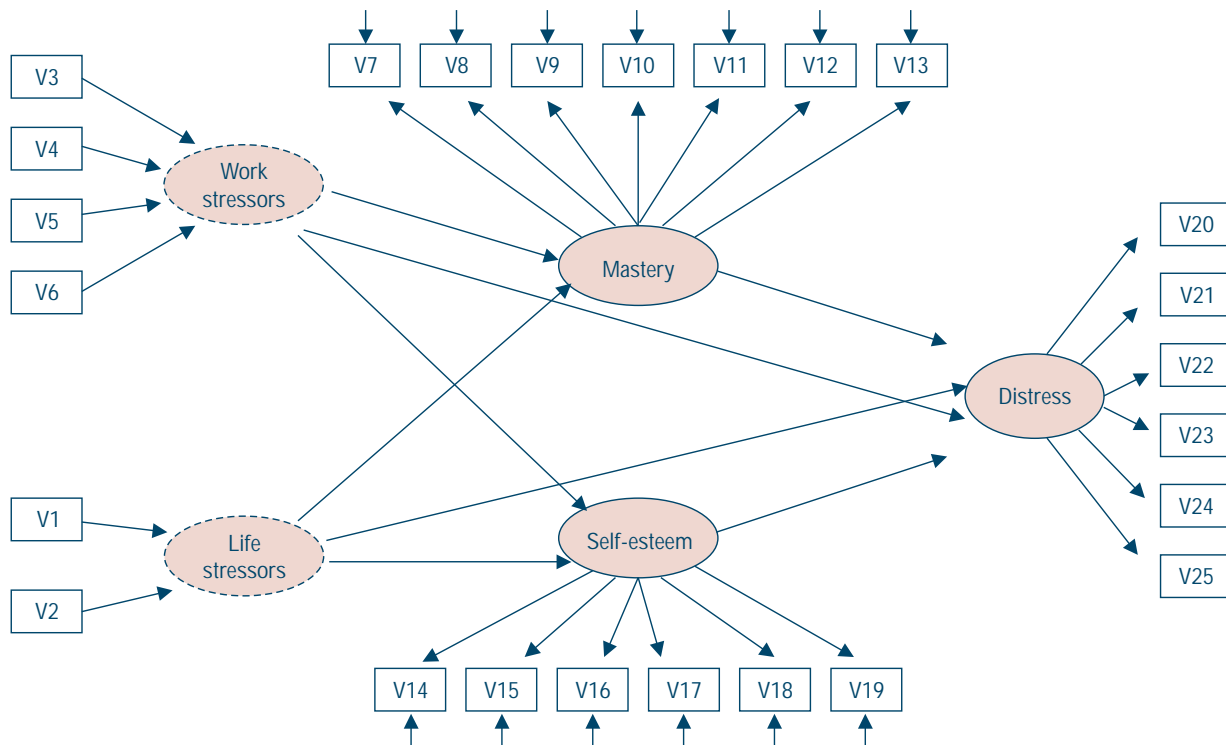
V1= chronic stress, V2= recent events, V3= psychological demands, V4= lack of control, V5= lack of support, V6= job insecurity, V7= little control, V8= unable to solve some problems, V9= unable to change things, V10= feeling helpless, V11= feel pushed around, V12= future depends on you, V13= you can do anything, V14= have good qualities, V15= equal to others, V16= do things as well as others, V17= positive attitude towards self, V18= satisfied with self, V19= feel as a failure, V20= so sad nothing cheers you up, V21= feel nervous, V22= feel restless, V23= feel hopeless, V24= feel worthless, V25=everything is an effort

These two broad domains, work and life stressors, are therefore postulated to be important determinants of distress among a working population, with higher levels of stressors being associated with higher levels of distress (see direct paths in Figure 1.)

A variety of psychological variables may mediate the relationship between stressors and distress i.e., be impacted by work stressors and in turn affect distress.[23] Mediation is in contrast to moderation ("interaction" or "effect modification" in epidemiological terms), where the psychological variable affects the strength of a stressor-strain relationship, but is not itself affected by stressors e.g., self-esteem in Jex and Elacqua, 1999.[24] As a mediator, self-esteem can be influenced by work factors, as eloquently argued by Locke and colleagues, 1996:[25] "We consider the realm of work to be crucial to self-esteem, because it is through

work that people master reality and sustain their existence ... thus work which one chooses as a value and which is done rationally, in full mental focus, is a source of self-esteem". Closely related is mastery, which declines as role strains reduce the extent to which people feel that they are able to manage their lives.[26] Hence, we postulated that paths would go from self-esteem and mastery to distress (indirect paths in Figure 1). The extent of mediation, from minimal, where most stressor effects would be direct to distress, to substantial, where the majority of stressor effects would be indirect through the mediators to distress, was left open.

Gender/occupational differentiation

The effects of work stressors on distress may also be shaped by social contexts.[10,27]

"Work contexts" and "family contexts" have been described as background factors for psychological distress.[6] Sociodemographic factors have also been depicted as independent predictors of depression in the vulnerability model elaborated by Phelan and colleagues.[4]

Studies relevant to gender comparisons present contrasting findings. Some reports show similar relationships between stressors and distress for both genders.[28,29] But some demonstrate gender differences. For example, women's distress levels showed reductions by social integration/resources for social support[7] and work stressors had a stronger effect on distress among men.[6] Highly routine jobs were more strongly associated with increased distress among women[30] and women were more affected by stressful personal events than men.[31]

Fewer studies inform occupational strata comparisons, as many regression analyses control for occupational grade/level and cannot examine differences in relationships within a strata (e.g., higher demands being confounded with higher employment grade in Stansfeld et al., 1997).[32] Several studies have shown greater life stressors among those of lower occupational categories and income (e.g., Stephens et al., 1999).[33] Joint examination of gender/occupational strata is rare. Comparisons are made difficult by the differential gender distributions across occupational grades and problems associated with the unmeasured factors varying across such strata.[34,35] Fortunately, the NPHS provides relatively large, representative samples of both working women and men, allowing us to create separate gender/occupational strata.

Secondary data

Population

The Canadian National Population Health Survey recruited a large, nation-wide sample of randomly selected participants.[36,37] The 1994–1995 cycle was the first wave. A complex, stratified, multi-staged design identified approximately 20,000 households, excluding people living on Indian reserves, at military bases, in institutions, and in some remote areas of Ontario and Quebec. In each household, one adult, chosen at random, was asked more detailed questions. Response rates were 88.7% at the household level and 96.1% at the individual level within households.

We selected all participants in the public use data set (Statistics Canada, 1995)[38] who answered the detailed questions, were aged 18 to 64 years old inclusive, were currently in paid employment, and responded to an abbreviated version of the Job Content Questionnaire (JCQ) (see below): in total, 4230 adult working men and 4043 adult working women. Responders to the JCQ (88%) were comparable in age and income distributions to the NPHS working population of interest. However, women respondents were less likely to be working part-time (71.5% respondents versus 78.6% of non-respondents, p= 0.02) and respondent men were less likely to be blue collar (48.7% respondents versus 54.7% non-

respondents, p= 0.006), college/university graduates (38.5% versus 45.5%, p= 0.003) and married/common law (70.5% versus 78.0%, p= 0.0008).

Respondents were asked if they had worked for pay or profit in the past 12 months, with up to six jobs recorded. The main job was classified according to the sixteen-point Pineo occupational prestige classification.[39] These were grouped into Lower Pineo (skilled, semi-skilled & unskilled) and Upper Pineo (supervisor, semi/professional & management) for the purposes of occupational stratification.

Measures by domain

Work stressors

Respondents completed a version of the JCQ[40] with respect to their current main job. The JCQ included questions on work psychological demands, decision latitude (control), physical exertion, job insecurity, and social support at work. Although the larger pool of items from the JCQ has undergone extensive validity testing,[41] only 12 items were included in the abbreviated NPHS version. Responses were based on a five-point scale (0= strongly agree; 4= strongly disagree), modified from four-point standard versions. Psychological demands were assessed by asking how hectic the job is and the degree of freedom from conflicting demands made by others. Decision latitude or "control" was measured through two dimensions: skill discretion (learning new things, level of skill and "doing things over and over") and decision authority (freedom to decide how to do the job and how much say a worker has about what happens in the job). The assessment of work social support asked about exposure to hostility or conflict from co-workers, supervisor help in getting the job done and co-workers' help in getting the job done. Job security and physical effort were each tapped with a single item. For our purposes, both decision latitude and work support were reverse scaled to connote "lack of" each as a work stressor, with higher scores connoting higher values for that stressor (lack of control and lack of work support, respectively).

Life stressors

Measures in this domain covered both particular events and chronic strains, as advocated by other stress researchers.[42] The chronic stress index[43] included 18 questions about ongoing concerns with children, spouse, housework and family – found in developmental work to have lasted on average more than five years. The index was adjusted for marital status and children in the home. The measure of recent life events[44–46] consisted of 10 questions requiring yes/no responses about the respondent or someone in his or her family in the last 12 months. Recent life events included major financial crisis, change of job, demotion, cut in pay and increase in arguments. The recent life events score was adjusted to take into account social roles (e.g., marital status, presence of children).

Psychological mediators

Self-esteem was tapped with six questions from the classic Rosenberg (1965)[47] scale. Respondents answered on a five-point scale from "strongly disagree" to "strongly agree" and responses were summed to a maximum of 24, higher scores indicating stronger self-esteem. Mastery, reflecting the extent to which individuals believe that their life chances are under their control,[48] were measured with seven questions on a five-point scale, yielding scores ranging from 0 to 28, with higher scores reflecting superior mastery. Cronbach's α for self-esteem and mastery were both > 0.65 and similar across gender/occupational strata.

Mental health outcome

From the relatively wide range of mental health indicators available in the NPHS,[33] we chose the distress measure based on the short form of the Composite International Diagnostic Interview (CIDI) with six questions. Cronbach's α for distress was also > 0.65 and similar across gender/occupational strata.

Analytical approach

Formulation of measurement models

A variety of approaches have been taken to modelling with JCQ scales: as independent

continuous variables[49] or di/trichotomized scores,[32] job strain as demand/control differences,[50] ratio[15] and median-based dichotomized interactions,[31,51] and iso-strain as interactions with work social support,[9,52] among others. Creating a composite latent construct[53] of work stressors[54] leaves open the nature of the relationships between the various scales. Conceiving of individual work stressor scales as formative, rather than reflective in the classical measurement sense, reduces concerns about the distinct nature of the work stressors e.g., job insecurity compared to other scales in the JCQ,[41] and low consistency associated with limited numbers of component items e.g., psychological demands in the NPHS version.[14] The different timing and nature of the chronic stressor and recent life events indices[55] also supported construction of a composite construct, where each index is formative of life stressors. In contrast, where items reflect one underlying construct and demonstrate relatively high measures of internal consistency as a group of items,[56] such as items making up self-esteem, mastery and distress, formulation as latent effect constructs was deemed most appropriate.[17]

Model estimation and testing

Structural equation modelling was performed using EQS version 5.7b.[57] Since EQS does not accommodate survey weights, potentially biasing standard errors,[58] we used SAS version 6.12[59] to calculate covariance matrices of the variables using survey weights provided by Statistics Canada.[38] The covariance matrices for each stratum were used as input to EQS.

Each of the four gender-occupational stratum models was estimated separately, and paths within strata were compared using standardized estimates.[6,60] Maximum likelihood estimation was used. Covariances among residuals of the endogenous variables were initially fixed at zero.

As the chi-square is highly sensitive to sample size and distributional assumptions,[61] five other measures of the overall goodness of fit were used.[62] The goodness of fit index (GFI) and the adjusted goodness of fit index (AGFI) were chosen for their low sensitivity to methods of estimation.[63] The

TABLE 1

Socio-demographic characteristics of working adult population used in analyses, by gender/occupation strata*

Variable	Women		Men	
	Lower Pineo‡ (n=2,438) (%)	Upper Pineo‡ (n=1,466) (%)	Lower Pineo (n=2,572) (%)	Upper Pineo (n=1,496) (%)
Age				
18–34	46.1	37.6	46.6	29.9
35–44	27.9	33.5	27.1	34.7
45–54	17.6	22.0	17.9	26.2
55–64	8.4	6.9	8.4	9.2
Marital status				
Never married	24.0	16.8	28.0	16.2
Married/common law	66.0	71.1	65.9	77.9
Widowed/separated/divorced	10.0	12.1	6.1	5.9
Education				
Less than secondary	18.3	3.1	25.4	6.8
Secondary complete	21.9	9.7	20.7	12.2
Some college/university	30.8	25.6	28.6	22.3
College/university complete	28.9	61.6	25.2	58.7
Missing	0.1	0.0	0.1	0.0
Household income				
Lower income	12.8	5.4	10.1	5.9
Lower middle income	29.9	16.5	28.6	16.4
Upper middle income	39.7	44.2	41.6	42.4
Higher income	13.3	31.9	15.4	30.5
Missing	4.3	2.0	4.3	4.8

* Survey weights that add to the sample size were used in these calculations.

‡ Lower Pineo comprised skilled, semi-skilled & unskilled
   Upper Pineo comprised supervisor, semi/professional & management

root mean square error of approximation (RMSEA) and the comparative fit index (CFI) are least sensitive to sample size[63] and Bollen's IFI is the least biased due to non-normality of variables.[64] The GFI, AGFI, CFI, and IFI range in value from 0 to 1, with a value of greater than .9 indicating a good fit. RMSEA values range upwards from 0, a perfect fit, through to .05, a good fit, up to .08, a fair fit, and > .1, a not acceptable fit.[65]

The Lagrange Multiplier (LM) test[57] was used to suggest the addition of potentially significant paths and the deletion of insignificant ones (p> 0.05). Correlations of some errors between sub-scales of the items of latent variables were suggested by the LM

test and so item errors were allowed to co-vary freely. The strength of the associations represented by the standardized estimates of the paths were judged according to Cohen's (1992)[66] criteria for multiple analysis of variance i.e., small= 0.02, medium= 0.15 and large= 0.35. Proportions of effects and ratios of standardized path coefficients were calculated using a spreadsheet to facilitate relevant comparative statements.

Results

The sociodemographic characteristics of the population are set out in Table 1. Greater proportions of workers with lower education and income and proportionately more

of the youngest workers can be seen among lower Pineo groups.

Variations in distributions of model variables across the four gender/occupation strata are apparent in Table 2. Covariance matrices of the analysis variables were too large for reproduction here (25×25 for each stratum) but are available from the authors upon request.

Adjustments to the hypothesized model were required to improve the fit of the stratum-specific models (Table 3). In addition to item correlations, LM tests suggested the addition of a path from self-esteem to mastery. All final models had a fair fit according to all the measures used, with less than 1% of the standardized residual having a value of greater than 0.2 in absolute value.

In the composite measurement models, work stressors most consistently reflected large contributions from lack of control (all paths above 0.5) and job insecurity (paths from 0.264 to 0.545). Intriguingly, psychological demands actually had negative relationships with work stressors except for lower Pineo men, though all were small in magnitude. Strengths of paths varied across gender/occupation strata, particularly for lack of social support: 0.039 among upper Pineo women to 0.630 among lower Pineo women, with intermediate values for men.

TABLE 2
Predictor, mediator and outcome variables, by gender/occupation strata*

Variables (range)	Women		Men	
	Lower Pineo Mean (sd)‡	Upper Pineo Mean (sd)	Lower Pineo Mean (sd)	Upper Pineo Mean (sd)
Predictors				
Work stressors				
Psychological demands (0–8)	4.48 (1.78)	5.26 (1.77)	4.31 (1.77)	5.04 (1.81)
Lack of decision latitude (0–20)	9.17 (3.25)	6.01 (2.74)	8.03 (3.34)	5.34 (2.75)
Lack of work support (0–12)	3.98 (2.12)	4.05 (2.11)	3.97 (2.05)	4.07 (2.08)
Job insecurity (0–4)	1.40 (1.13)	1.40 (1.22)	1.38 (1.15)	1.26 (1.15)
Life stressors				
Chronic stressors (0–14)	3.50 (2.60)	3.12 (2.40)	3.11 (2.46)	2.67 (2.16)
Recent events (0–8)	0.72 (1.07)	0.66 (0.99)	0.65 (1.02)	0.55 (0.89)
Mediators				
Self-esteem score (0–24)	19.96 (3.01)	20.77 (2.80)	20.25 (2.78)	20.89 (2.67)
Mastery score (0–28)	19.49 (4.08)	20.72 (4.16)	19.90 (3.98)	21.12 (3.82)
Outcome				
Distress (0–24)	3.62 (3.29)	3.12 (2.89)	3.09 (3.04)	2.68 (2.52)

* Survey weights that add to the sample size were used to calculate all means and standard deviations.

‡ sd = standard deviation

Chronic stress was more important than recent life events for life stressors but both were of similar magnitudes across strata.

With respect to structural paths, both work and life stressors had significant positive total path coefficients to distress in all four stratum-specific models (see table 4). Work stressor total effects were consistently smaller (minimal to medium using Cohen's criterion) than life stressor total effects (all

TABLE 3
Model Goodness of Fit indices*

	χ^2 (df)	CFI	GFI	AGFI	IFI	RMSEA (90% CI)
Women						
Lower Pineo group (n = 2,438)						
Hypothesized model	3,825 (254)	.799	.872	.837	.799	.076 (.074, .078)
Final model	1,927 (246)	.905	.942	.923	.905	.053 (.051, .055)
Upper Pineo group (n = 1,466)						
Hypothesized model	2,387 (254)	.815	.877	.842	.815	.076 (.073, .078)
Final model	1,071 (243)	.928	.945	.926	.928	.048 (.045, .051)
Men						
Lower Pineo group (n = 2,572)						
Hypothesized model	3,602 (254)	.816	.887	.855	.816	.072 (.070, .074)
Final model	1,894 (247)	.909	.945	.927	.910	.051 (.049, .053)
Upper Pineo group (n = 1,496)						
Hypothesized model	2,558 (254)	.784	.868	.831	.785	.078 (.075, .081)
Final model	1,285 (243)	.902	.934	.911	.903	.054 (.051, .056)

* χ^2 = Chi-square; df = degrees of freedom; CFI = Comparative Fit Index; GFI = Goodness of Fit Index; AGFI = Adjusted Goodness of Fit Index; IFI = Bollen's index; RMSEA= Root Mean Square Error of Approximation; CI = Confidence interval

TABLE 4
Standardized path coefficients for composite stressor measurement models, by gender/occupation strata

Path	Women		Men	
	Lower Pineo	Upper Pineo	Lower Pineo	Upper Pineo
To work stressors				
Psychological demands	−.178	−.099	.015	−.060
Lack of control	.539	.909	.631	.640
Job insecurity	.322	.264	.545	.396
Lack of work social support	.630	.039	.307	.419
To life stressors				
Chronic stress	.905	.940	.868	.825
Recent life events	.210	.155	.288	.359

large). Ratios of work to life stressor total effects ranged from 0.01 for upper Pineo women to 0.33 for upper Pineo men, with lower Pineo women (0.20) and men (0.23) intermediate.

With respect to mediators, work and life stressor effects were consistently negative or absent, with effects on mastery consistently of greater magnitude than those on self-esteem. All of the effects of work stressors on distress were indirect i.e., mediated through mastery and self-esteem. In con-

trast, the majority of the effects of life stressors on distress were direct: indirect/total ratios from 0.54 among lower Pineo women to 0.93 among upper Pineo men, with lower Pineo men (0.60) and upper Pineo women (0.70) intermediate.

Discussion

Our analyses show associations of work stressors with levels of distress among the broad Canadian working population. Our results are consistent with literature show-

ing relationships between work stressors in general, and job demands and lack of control in particular, and mental health outcomes.[31,51] Work stressor-distress relationships were almost entirely mediated by mastery and self-esteem. From an analytical perspective, these results support the inclusion of such mediator variables on the paths between work stressors as predictors and distress as an outcome rather than examining multiple independent associations between potential exposures, mediators and outcomes in a less-than-clear structure, resulting in the observation of "trivial" relationships.[67] From an applications perspective, our findings suggest that human resource interventions that either reduce work stressors, bolster self-esteem or increase mastery at work may have similar effects on distress.

The overall similarities in valence of structural paths across genders support the idea that the characteristics and dynamics of work stressors and mental health are similar for women and men.[28,29] The majority of structural paths were similar at different levels of occupational prestige, in keeping with other work stress literature.[31] Our construction of work stressors as a composite variable demonstrated variation in

TABLE 5
Standardized direct, indirect and total effects for model structural paths, by gender/occupation strata*

Paths	Women						Men					
	Lower Pineo			Upper Pineo			Lower Pineo			Upper Pineo		
	Direct	Indirect	Total*	Direct	Indirect	Total*	Direct	Indirect	Total*	Direct	Indirect	Total*
Work stressors to												
Mastery	−.144	−.056	−.200	−.156	−.108	−.264	−.180	−.076	−.257	−.236	−.177	−.413
Self-esteem	−.188	—	−.188	−.242	—	−.242	−.243	—	−.243	−.338	—	−.338
Distress	—	.100	.100	−.123	.127	.004	—	.122	.122	—	.153	.153
Life stressors to												
Mastery	−.453	−.021	−.474	−.258	−.101	−.359	−.408	—	−.408	−.221	—	−.221
Self-esteem	—	−.070	−.070	−.227	—	−.227	—	—	—	—	—	—
Distress	.274	.236	.510	.361	.153	.514	.323	.193	.536	.430	.032	.462
Mastery to												
Distress	−.499	—	−.499	−.304	—	−.304	−.474	—	−.474	−.147	—	−.147
Self-esteem to												
Mastery	.298	—	.298	.446	—	.446	.315	—	.315	.523	—	.523
Distress	—	—	—	−.194	−.135	−.330	—	−.149	−.149	−.274	−.077	−.351

*   total = direct + indirect

—— indicates insignificant path p > 0.05

contribution by different measures across the different strata. Paths from lack of control were the greatest in magnitude, consistent with the important role of control or decision latitude in predicting health impacts associated with hierarchies at work.[68] Yet they varied in magnitude particularly between women's occupational strata, indicative of variations in meaning or conditions noted by the scale developers.[41] Job insecurity was the next most consistently important measure, in keeping with the strong health impact of labour market vulnerability.[69] Intriguingly, lack of work social support was the most important for lower Pineo women and of larger magnitude for this stratum than any other. The incongruous negative valence for psychological demands in three strata, may indicate the extent to which the reduced set of JCQ items inadequately represent the contribution of specific work stressors to the overall composite latent construct.

Unfortunately, imprecise measurement in the measures contributing to work stressors may have reduced structural path coefficients from work stressors to mediators and distress as well. The tradeoffs inherent in bargaining for inclusion of work stressor content in national surveys (fewer items versus nothing) are only too apparent. Without some items, analyses such as those presented here would not be possible, but with too few items the impact of work stressors may be underestimated relative to life stressors. Such underestimation was exacerbated by other measurement challenges. Financial stressors associated with changes at work and job promotion were both potential recent life events contributing to life stressors. Further, the life stressor domain represented more of the life course while comparative cumulative work stressor information over a working life was not available. Work histories with imputed scores from job-exposure matrices based on aggregated data, as have been used in cohort studies, would make this possible.[70]

National Population Health Survey data were limited in other ways. Measures of work-family interaction or conflict[71] were absent, despite demonstration of their effects on mental health[72] and recognition of their growing importance.[73] Alternative formulations of relationships between stressors and mental health may also challenge our analyses. Some models interpret stressor valuation as consequent to levels of psychological symptoms[74] or to core evaluations, such as self-esteem, as determinants of life and job satisfaction.[3] Without reference to corroborative data on stressor evaluations by others than the worker her/himself,[75,76] such alternative formulations are difficult to counter directly. Conceptually helpful are theoretical approaches that reinforce the extent to which self-esteem or other core evaluations can be assaulted by stressors and are thus outcomes rather than predictors.[26] Hence, contributions of our analyses to the understanding of cause must be tempered,[67] as the data set does not provide the precise measures, independently assessed, with clear temporal relationships (i.e., not cross-sectional) necessary to construct causal diagrams based on classic epidemiological contrasts.[77]

Future research on work stressors and mental health must continue to struggle with measurement constructs and to work with modelling techniques that permit incorporation of measurement as well as structural relationships, as advocated by Hurrell and colleagues (1998).[20] In the meantime, promotion of "good" work organization[78] or "healthy workplaces"[79] as ways of reducing levels of work stressors is one of the applications of health research. Our analyses here provide support to mental health practitioners, workplace parties and policy makers taking actions on reducing work stressors, drawing on work stress intervention literature[80–82] and to existing health promotion campaigns to change determinants of health.[83]

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Small area comparisons of health: applications for policy makers and challenges for researchers

Paul J Veugelers and Shane Hornibrook

Abstract

It is a challenge to researchers to present their results in a way that serves the needs of health policy makers. Small area maps of life expectancy provide an insightful presentation. In this study, we pursued small area comparisons on a scale that is smaller than is currently available on a province-wide basis. We visualized Nova Scotia's provincial variation in health and identified the Cape Breton Regional Municipality and Halifax's disadvantaged "North End" neighbourhood as areas with major health concerns. The observed health differences are only partially explained by socioeconomic factors such as income and unemployment. The study also demonstrated the feasibility of small area comparisons at the level of census consolidated subdivisions and neighbourhoods. There are various methodological challenges for researchers, however: allocation procedures such as the postal-code-conversion-file may introduce substantial error; the application of appropriate spatial smoothing procedures is crucial to the interpretation of regional variation in health; and the migration of frail individuals to nursing homes affects the geographic variation in health.

Key Words: Bayesian methods; chronic diseases; health inequalities; health policy; life expectancy; mapping of disease, multilevel analysis; socioeconomic factors; spatial statistics

Introduction

Health inequality is a major public health issue.[1] Health inequalities occur in all kinds of geographically and socio-economically determined subgroups.[2,3] Their identification is crucial to health policy makers for planning and prioritizing prevention and intervention activities. The Government of Nova Scotia addressed health inequalities in its policy plan, stating: "fairness to regions and to people" and "where you live must no longer determine what you can be".[4] The principle of fairness extends to equal rights to good health among regions. To make this fairness a reality, provincial health inequalities should be monitored and reported in a way that is meaningful to policy makers.

Health policy makers are not necessarily trained as epidemiologists or statisticians and may not have a thorough understanding of the results reported by researchers. Researchers are challenged to present their results in ways that serve the needs of health policy makers.[5,6] Various researchers are using geographic maps in which disease and mortality differences are visualized through colour patterns as a means of presenting their research results.[7–9] It would further ease interpretations if these colour patterns translate into quantities that are easy to comprehend: for example, if they translate into life expectancy rather than into standardized mortality rates or ratios.

Traditionally, geographic comparisons of health have used countries, states and provinces as their geographic units.[10] These comparisons may fail to disclose the health concerns of smaller areas and are therefore not meaningful to health policy makers who govern such small areas.[11,12] It is for this reason that in past decades increasing numbers of studies have been investigating the health inequalities of smaller geographic units.[8,10–12] These small area studies have benefited from improvements in computers, geographic information systems, and statistical methods,[8,10,13,14] but remain hampered due to concerns about the accuracy of the population information of small areas.[8,15] In addition, ecological bias resulting from selective migration is a larger concern in small area studies since intra-regional migration affects small area comparisons to a larger extent than inter-regional comparisons.[16] For example, nursing homes host relatively frail individuals with a life expectancy shorter than that of their age-equivalents who are able to live independently. Small area studies will therefore identify geographies with nursing homes as geographies with health concerns. Alternatively, this ecological bias will not affect interprovincial comparisons since all provinces provide nursing homes for their elder residents.

In this study, we try to present vital statistics in a format that is meaningful to policy makers by presenting small area maps of life expectancy. We pursue small area comparisons on a scale that is smaller than that currently available on a province-wide basis. We investigate the importance of socioeconomic factors and selective migration to nursing homes. In addition, we provide details of considerations and choices of data resources, functional geographic units, sources of error and statistical methods. These details are crucial to other small area applications that we have planned and will be helpful to researchers who

Author References

Paul J Veugelers and *Shane Hornibrook*, Department of Community Health and Epidemiology, Faculty of Medicine, Dalhousie Univeristy
Correspondence: Paul J Veugelers, Department of Community Health and Epidemiology, Dalhousie University, 5849 University Avenue, Halifax, Nova Scotia, Canada B3H 4H7; Fax: (902) 494-1597; E-mail: paul.veugelers@dal.ca

want to pursue small area comparisons elsewhere in Canada.

Methods

Geographies

Nova Scotia comprises nine District Health Authorities, 18 counties, 52 census consolidated subdivisions, 110 census subdivisions, 1511 federal enumeration areas and 18,864 postal codes. Census subdivisions comprise cities, towns, villages, municipal districts and subdivisions of counties.[17] A census consolidated subdivision is a grouping of census subdivisions where the smaller, more urban census subdivisions (towns, villages, etc.) are combined with the surrounding, larger, more rural census subdivision (municipal districts and subdivisions of counties).[17] A census consolidated subdivision is a functional grouping: rural residents frequently have mailboxes in nearby communities with different postal codes, causing researchers to introduce misclassification when using geographic units smaller than census consolidated subdivisions.[18] In addition, studies have reported that manual coding of place of residence inaccurately overcounts cities and undercounts their peripheral areas.[19] For the above reasons we use "census consolidated subdivision" as our unit of comparison in nonurban geographies. The two urban areas, Metropolitan Halifax and Cape Breton Regional Municipality, are subdivided into geographies not exceeding 50,000 residents. Metropolitan Halifax is subdivided by grouping enumeration areas into 11 neighbourhoods, and the Cape Breton Regional Municipality is subdivided by grouping enumeration areas into four geographies largely divided by natural borders (lakes and rivers). This brings the number of customized geographies to a total of 64 with populations ranging from approximately 2,500 to 41,000 (see Appendix). The population of Nova Scotia is approximately 940,000.

Life expectancy and health deficiencies

We present estimates of life expectancy for each of the 64 geographies. Life expectancy is an easy-to-comprehend measure of local health status and thus preferable to health policy makers. We used Chiang's standard period life table methods to calculate life expectancy at birth and standard errors resulting from sampling error (SE_{SE}).[20] When calculating the combined life expectancy of females and males, we summed the radix and combined female and male deaths as a means to provide more stable estimates in geographies with small populations.[21] All calculations are based upon 20 age categories (less than one year, one to four years, 17 consecutive five-year categories, and 90 years and older). Health deficiencies are defined as the difference between local life expectancy and that of the provincial average. This is broken down into cause-specific components, cardiovascular diseases (ICD 9: 390 to 459), cancer (ICD 9: 140 to 208), lung cancer (ICD 9: 162 and 163), colorectal cancer (ICD 9: 153 and 154), breast cancer (ICD 9: 174) and respiratory diseases (ICD 9: 460 to 519). These calculations are based upon cause-eliminated life table methods as described in detail elsewhere.[22,23]

Population estimates

For the purpose of calculating life expectancy we need accurate population and mortality estimates for each of the 64 geographies. We considered three sources of population information: age and gender specific population counts for census years 1986, 1991, and 1996; Statistics Canada's online statistical database (CANSIM)[24] for 1986, 1991, and 1996; and population counts from the Nova Scotia Medical Services Insurance registration file at midyears that are available to us for 1996, 1997, 1998, and 1999. In 1996, for the province as a whole, the population estimates on the basis of the Medical Services Insurance registration file were 0.29% higher than on the basis of CANSIM and 2.73% higher than on the basis of the census. We adjusted the census population estimates in 1986 and 1991 for the underestimation observed in 1996. We estimated the population sizes for each age and gender subgroup in the years 1990 and 1992 to 1995 by applying cubic splines to the adjusted estimates for 1986 and 1991 and to the Medical Services Insurance counts for 1996 to 1999.[22]

The postal code conversion file is a software instrument issued by Statistics Canada that allows researchers to allocate enumeration areas on the basis of postal codes.[18,25] In situations where a postal code constitutes more than one enumeration area, the postal code conversion file will select enumeration areas using a randomization procedure that considers the population size of the enumeration areas. As part of this study we investigated the variation in allocating postal coded places of residence to each of the 64 customized areas and the extent to which this affects the estimates of life expectancy. To do so, we repeated the allocation procedures 10 times and calculated standard errors of the 10 life expectancy estimates for each of the 64 geographies. Small standard errors are indicative of reproducible allocation procedures.

Mortality estimates

We obtained annual cause-specific mortality from Statistics Canada for the years 1995 to 1999, specified by age and gender. The available geographic information included postal code and geographic specifications by census subdivisions or county. Both postal code and geographic specifications introduce error in the allocation of mortality to geographic units. Postal codes require the use of the postal code conversion file (discussed above) and geographic specifications may cross boundaries or be missing and require random allocation. As part of the current study we assessed the extent of error introduced by allocating mortality when using postal codes, geographic specifications or the combination of both. We repeated the allocation procedures 10 times and subsequently calculated the standard error of the 10 repetitions. Small standard errors are indicative of reproducible allocation procedures.

Institutionalized population

Residents of nursing homes are relatively frail and have shorter life expectancies than their age-equivalents living independently. Migration of frail individuals from geographies without nursing homes to geographies with nursing homes will in-

crease the life expectancy in the former and reduce the life expectancies in the latter. Small-area studies that aim to identify local health status and its determinants are hampered by this selective migration of frail individuals.[16] Deaths in nursing homes are identifiable through their institutional postal codes. We retrieved the postal codes of the prior residential addressed for approximately 80% of these deaths. To illustrate the importance of the selective migration of frail individuals we compared life expectancy estimated using nursing home addresses with life expectancy estimated using previous residential addresses.

Determinants of Local Health Status

Area level measures of socioeconomic status, average household income and unemployment rate are investigated with respect to their association with local life expectancy. This information is taken from the 1996 Canada Census.

Statistical Methods

We estimated life expectancy and health deficiencies over the calendar period of 1995 to 1999 for each of the 64 geographies. These estimates fluctuate more than expected on the basis of sampling error (overdispersion) as a result of the sparseness of (cause specific) mortality and varying population sizes of the 64 geographies.[8,13] If overdispersion is ignored it creates the impression of spurious geographic variation and consequent instable estimates of associations with covariates such as areal socioeconomic characteristics.[8] Bayesian hierarchical or multilevel models have been suggested as appropriate methods to analyze such small area data.[8] We considered a multilevel model whereby information of the 64 geographies and their direct neighboring geographies are pooled (level 1) resulting in robust estimates of the geography-specific life expectancy (level 2). This model also allows us to incorporate the various sources of standard error described above. We will refer to the empirical Bayes estimates generated by this model as "spatially smoothed estimates" in the remainder of this manuscript.

Using multilevel approaches, we further considered geographies (level 1) within regions (level 2) to generate empirical Bayes (spatially smoothed) estimates of regional variation. The four regions considered include: non-metropolitan mainland, metropolitan Halifax, non-metropolitan Cape Breton Island, and Cape Breton Regional Municipality (see appendix). With this multilevel model we also analyzed the association of socioeconomic characteristics, income and unemployment rate to life expectancy. The analyses were conducted by using HLM5 and S-PLUS 2000.

Results

Health deficiencies, defined as the differences between local life expectancy and the provincial average, are visualized in Figure 1 through colour patterns with red, indicative of reduced life expectancy and blue, indicative of prolonged life expectancy. Various geographies have substantially reduced, dark red, or substantially prolonged life expectancy, dark blue (Figure 1 top panel). Life expectancies are also listed in the appendix along with the population size, numbers of deaths and various sources of standard error. Sampling error (SE_{SE}) is determined by the number of residents and deaths in each age and gender subgroup. Geographies with large populations have generally small SE_{SE} (appendix). Standard error resulting from the allocation procedures in estimating the population sizes, SE_{POP}, is negligible relative to SE_{SE}. The appendix lists three estimates of standard error corresponding with three means of allocating mortality: SE_{M1}, if only geographic specifications are considered, is substantial relative to SE_{SE} in urban areas. SE_{M2} considers only postal code information and is substantial particularly in rural areas. SE_{M3} uses both geographic and postal code information and is generally less than SE_{M1} and SE_{M2}, but for a few geographies still substantial relative to SE_{SE}.

Both SE_{SE} and SE_{M3} were considered in the multilevel model generating the spatially smoothed estimates that are listed in the appendix and depicted in the bottom panel of Figure 1. These smoothed estimates allow better judgment of the geographic distribution: Cape Breton Island has reduced

life expectancy and within the Halifax Regional Municipality considerable variation persists. The differences between the crude and spatially smoothed estimates are frequently large in geographies with small populations (Figure 2 – top panel). The differences of estimates with and without adjustment for selective migration of nursing home residents is of a smaller magnitude and not as clearly related to the population size of the geography (Figure 2 – bottom panel).

Health deficiencies resulting from cardiovascular and cancer mortality are displayed in Figure 3 and reveal distinct geographic patterns.

Table 1 presents the univariate association of income and unemployment with life expectancy. An increment in income of $10,000 is associated with an increase of 0.956 years in life expectancy, whereas an increment in unemployment of 10% is associated with a reduction of 0.862 years in life expectancy. Multilevel regression (spatially smoothed) estimates an increment in income of $10,000 to be associated with an increase of 0.617 years in life expectancy and the association with unemployment not to be statistically significant. Table 2 presents the extent of regional variation: Life expectancy in the Cape Breton Regional Municipality is estimated to be 1.46 less than in the non-metropolitan mainland, and 1.57 (in Table 2: 1.46+ 0.11) less than in the Halifax Regional Municipality. These differences are statistically significant and only partly explained by differences in income or unemployment (Table 2). Table 3 shows the regional variation in life expectancy broken down in disease specific components. Due to cardiovascular mortality, life expectancy of female residents of Cape Breton Regional Municipality is 0.36 years less than that in the non-metropolitan mainland; for male residents it is 0.74 years. The estimates are higher for cardiovascular disease than for cancer.

Discussion

This study demonstrates the geographic variation of health within Nova Scotia. Geographies with major health concerns include Cape Breton Regional Municipality and the disadvantaged "North End" neigh-

FIGURE 1
Life expectancy in Nova Scotia

Observed health deficiencies

Spatially smoothed health deficiencies

More than 1.5 years below provincial average
0 to 1.5 years below provincial average
0 to 1.5 years above provincial average
More than 1.5 years above provincial average

50   0   50   100

N

kilometers
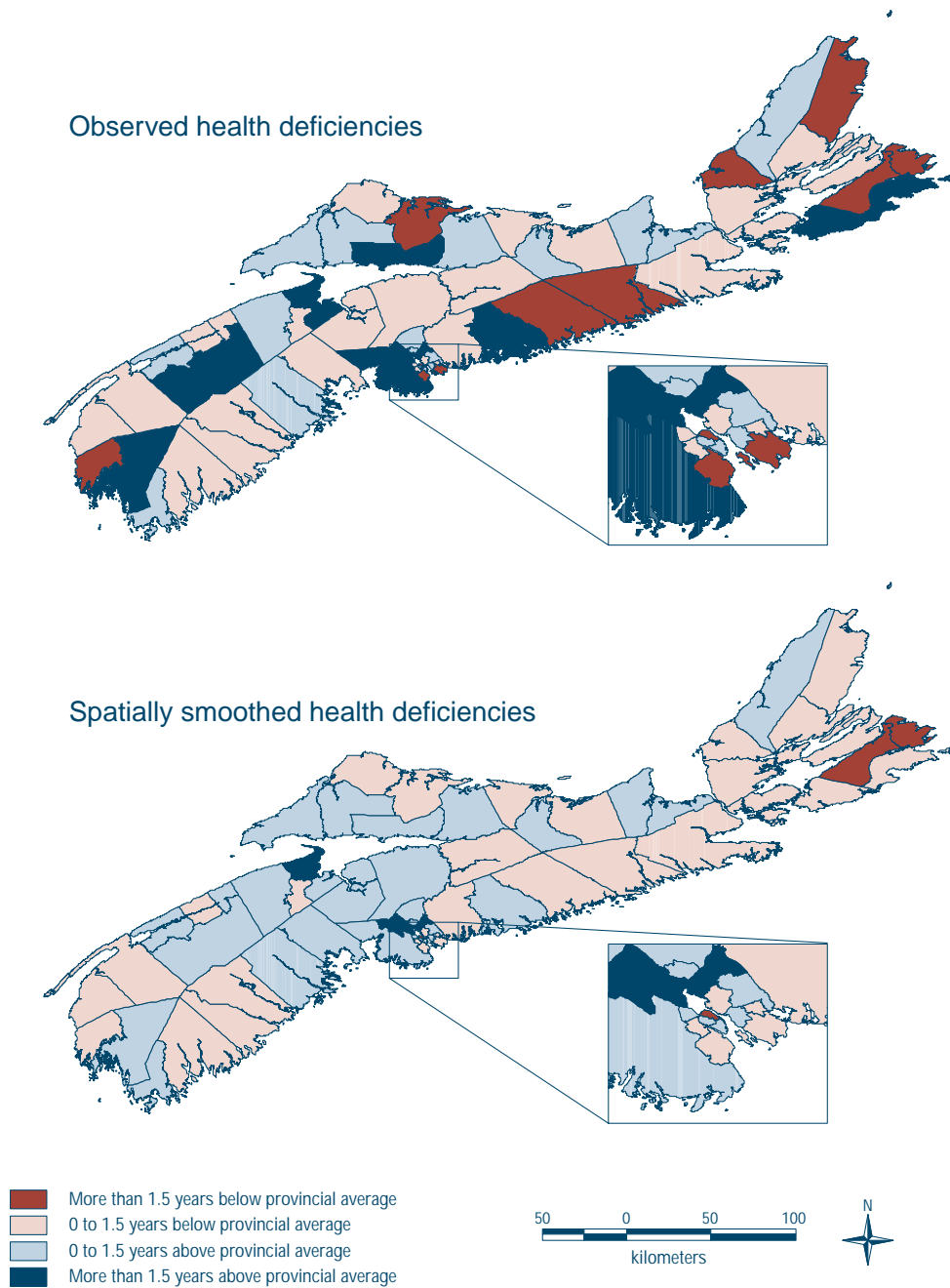
bourhood within metropolitan Halifax. Although socioeconomic factors are important determinants of health, they are only partially responsible for the observed variation in health. This study also demonstrates the feasibility of small area comparisons of health and the importance of appropriate allocation procedures, statistical methods and selective migration.

We presented small area maps visualizing geographic patterns in health and identifying geographies and regions of concern. Life expectancies in some geographies are reduced by more than 1.5 years relative to the provincial average which, in turn, is approximately one year less than the Canadian average.[26] These differences are substantial: health in these geographies lag 10

to 15 years behind that of Canadians, if one considers the national increase in life expectancy of approximately two years per decade.[22]

The health concerns in Cape Breton County have previously been addressed in ecological studies.[16,22,23,27,28] Suggested underlying factors include life style choices such as smoking and obesity, participation in

FIGURE 2
Top panel: Differences between crude and spatially smoothed health deficiencies by population size
Bottom panel: Differences between health deficiencies with and without adjustment
of selective migration by nursing home residents
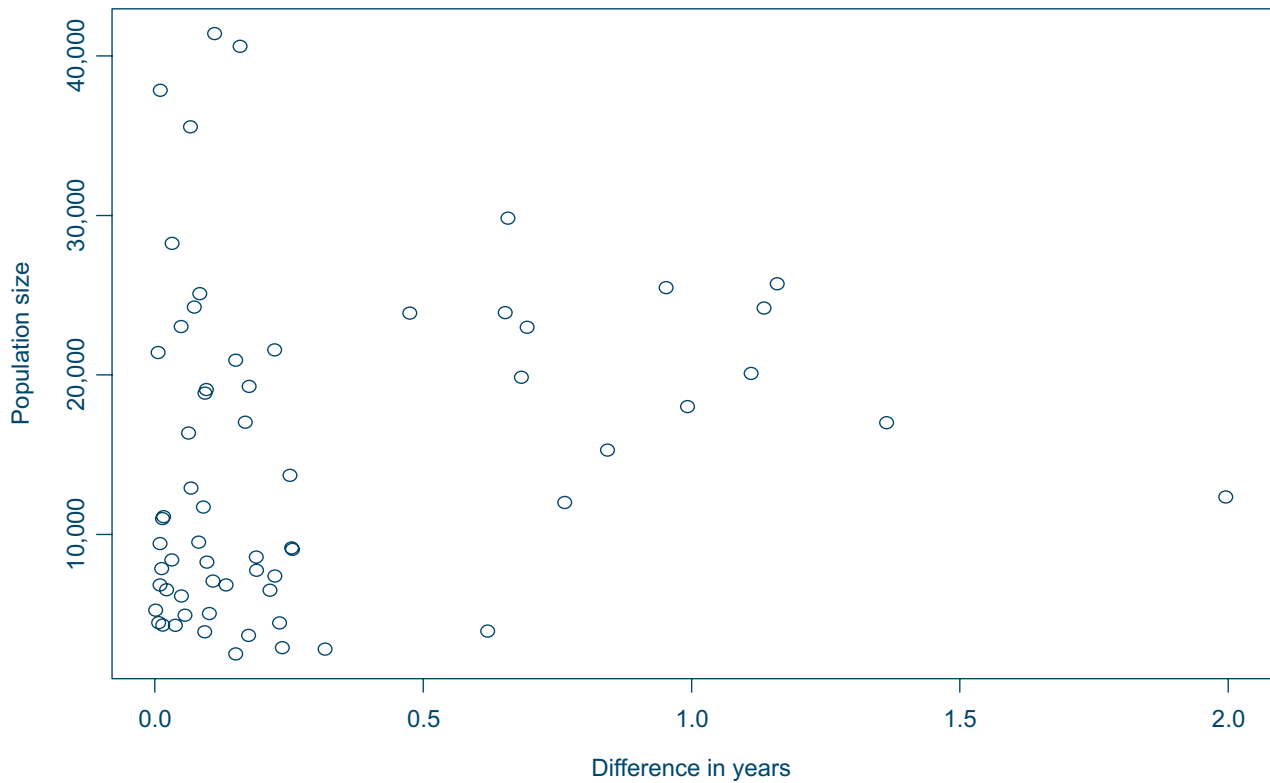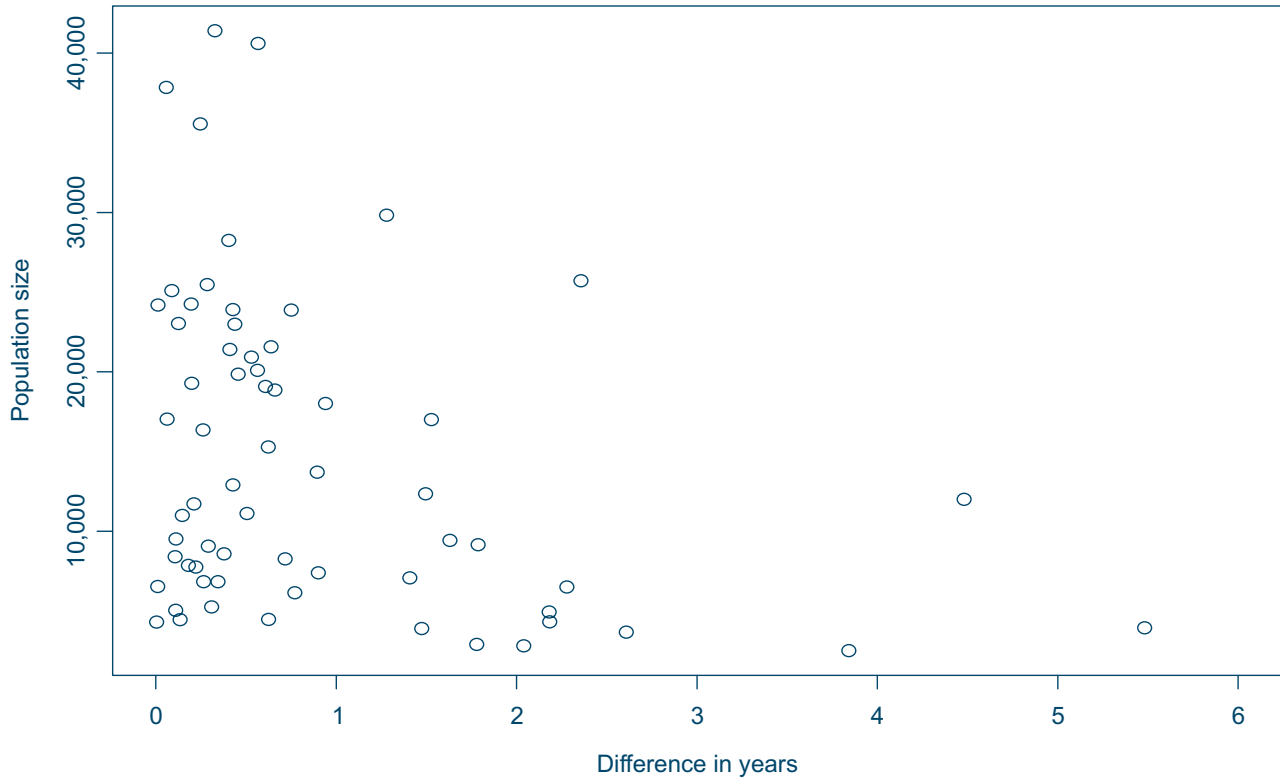
FIGURE 3
Health deficiencies in Nova Scotia

Health deficiencies resulting from
cardiovascular mortality among females

Health deficiencies resulting from
cancer mortality among females

- More than 0.5 years below provincial average
- 0 to 0.5 years below provincial average
- 0 to 0.5 years above provincial average
- More than 0.5 years above provincial average

50   0   50   100

kilometers

N
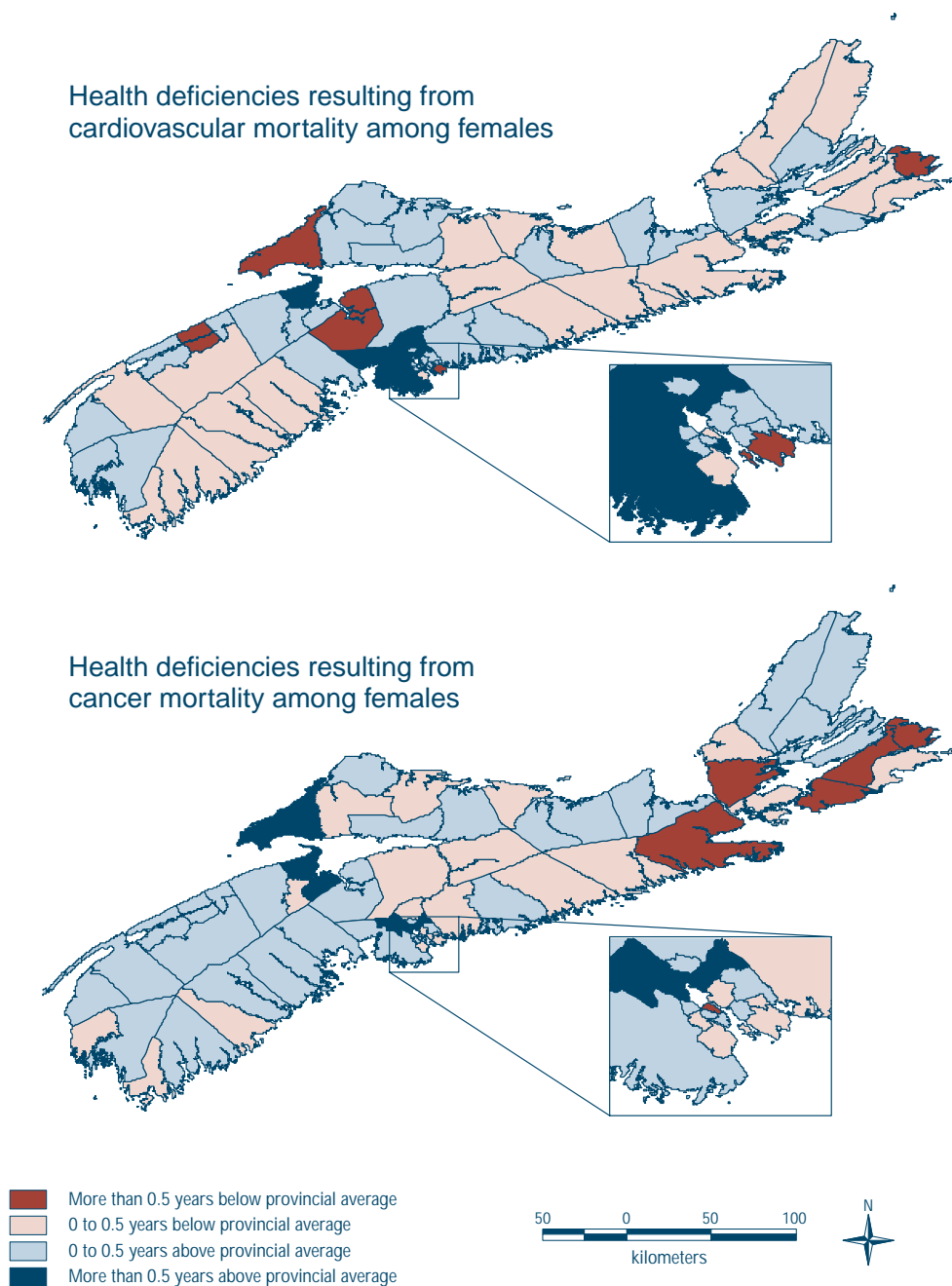
cont'd

screening programs, environmental conditions and socioeconomic factors.[22,23,29–31] This study provides additional understanding by demonstrating that regional differences are only in part explained by income or unemployment. Health differences elsewhere in the province have not been evaluated previously. This study provides a province-wide evaluation and reveals that Cape Breton County is not the only area with major health concerns. The "North End" neighbourhood in Halifax exhibits health concerns of similar magnitude in contrast to the wealth and health of other neighbourhoods.

The aim of this study was to pursue small area comparisons at a scale that is smaller than what is currently available at a provincial level. We demonstrated the feasibility of mapping postal code data at the level of census-consolidated subdivisions in rural areas and neighbourhoods in urban areas. The various challenges we ex-

FIGURE 3 *(cont'd)*
Health deficiencies in Nova Scotia

Health deficiencies resulting from
cardiovascular mortality among males

Health deficiencies resulting from
cancer mortality among males

More than 0.5 years below provincial average
0 to 0.5 years below provincial average
0 to 0.5 years above provincial average
More than 0.5 years above provincial average

50   0   50   100   N

kilometers
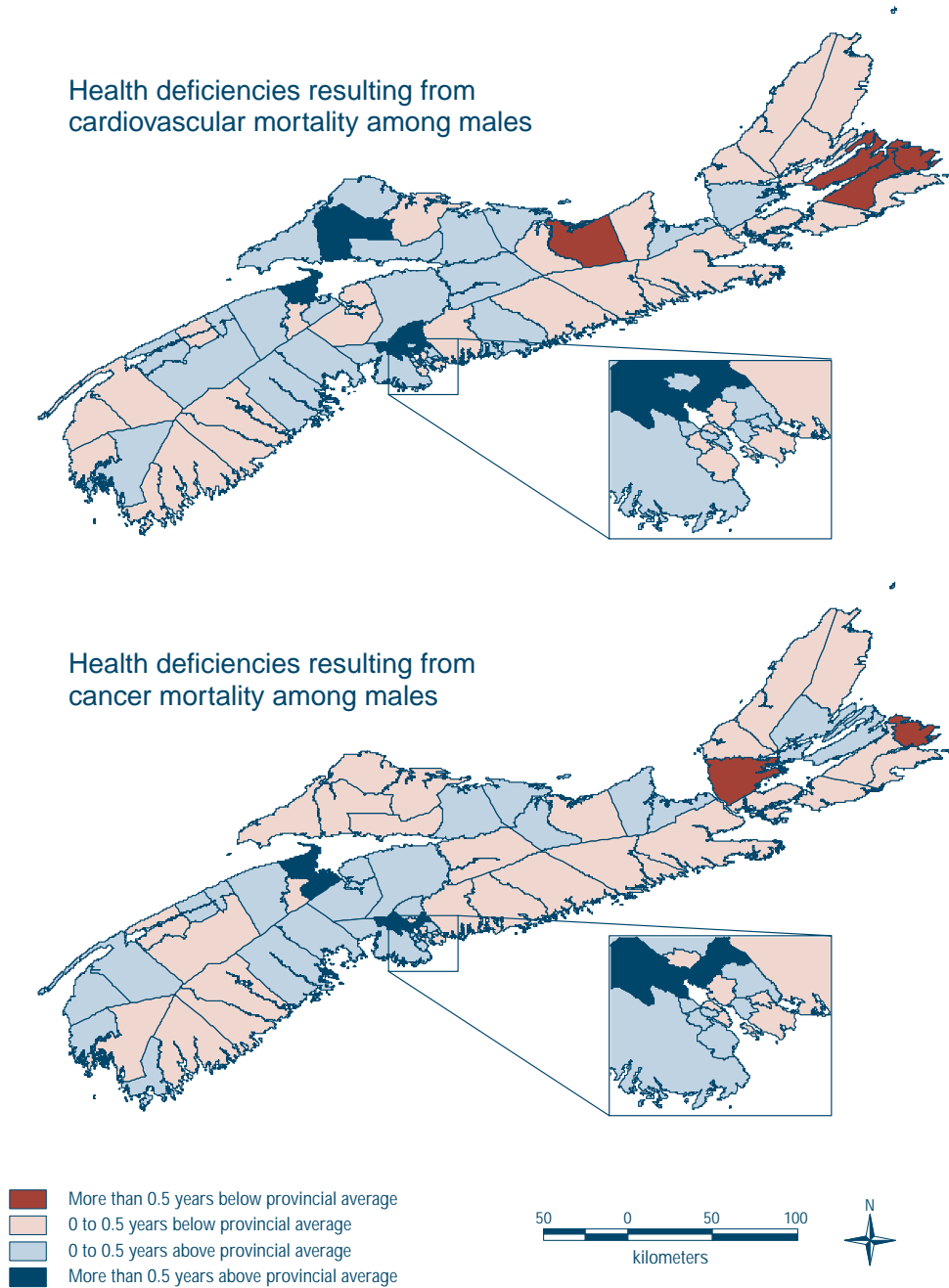
perienced in mapping at this small area level, including allocation procedures, statistical methods and selective migration, are discussed below:

Allocation procedures. The postal code conversion file has become a crucial instrument for the allocation of postal code information to geographic locations and has enabled the conduct of various re-

search.[3,18,32] Its appropriateness in small area studies has been evaluated here. For the allocation of large counts, such as for whole residential populations, the reproducibility of the postal code conversion file appeared excellent and was reflected in very small standard errors. However, if counts, such as mortality counts, are sparse, the reproducibility de-

creases. Investigators using the postal code conversion file should be aware of this additional source of error. In this study we demonstrated that this error is particularly present in rural areas, and decreases when considering additional geographic specifications. To reduce this error, investigators may alternatively want to repeat the allocations with the postal code conversion file

and consider the average value of the repeated allocations.

Statistical methods. We have presented maps of both crude and spatially smoothed estimates of life expectancy. Interpretation of these maps warrants caution: the crude estimates exhibit overdispersion and therefore create the impression of spurious geographic variation. The spatially smoothed estimates are a solution to overdispersion and are thus most appropriate for the judgment of geographic variation. All smoothing procedures, including those used in this study, are to some extent arbitrary.[8] We chose to consider the life expectancy of all neighbouring geographies in the smoothing procedures, although others investigators may have chosen differently.[13,14] These choices affect the magnitude of the smoothed estimates of life expectancy and should be considered when judging geographic variation. The importance of the choice of statistical methods was also reflected in the analyses of socioeconomic factors, where crude and spatially smoothed estimates differed considerably.

Selective migration of healthy or frail subgroups can affect local estimates of life expectancy and thus ecological comparisons, particularly small area comparisons.[16] In this study we demonstrated that in five of the 64 geographies (7.8%) selective migration to nursing homes altered local estimates of life expectancy by more than one year. Clearly, and in addition to established and causal risk factors, the presence of nursing homes should also be considered as a factor affecting estimates of life expectancy of small areas. Since the analysis of selective migration was based on the incomplete prior residential addresses of nursing home residents, the actual effect of selective migration is likely larger.

We have aimed to present health data in an easily comprehended manner and have chosen the format of provincial maps of life expectancy. They revealed various matters that are important to policy makers, such as the revelation that the Cape Breton Regional Municipality is not the only area with major health concerns. In addition, they revealed distinct geographic patterns in the underlying causes of death. In this respect, cardiovascular disease is demonstrated to be the single most important cause of death responsible for the health deficiencies in the Cape Breton Regional

TABLE 1
The relationship of household income and unemployment rate with life expectancy

	Life expectancy in years		
	Change	se	p
Crude observations:			
Household income (per $10,000 increment)	0.956	0.283	0.001
Unemployment rate (per 10% increment)	−0.862	0.350	0.017
Spatially smoothed estimates:			
Household income (per $10,000 increment)	0.617	0.160	0.000
Unemployment rate (per 10% increment)	−0.355	0.247	0.151

se: standard error

p: probability that the estimated change equals zero

TABLE 2
Regional variation in life expectancy within Nova Scotia

	Unadjusted	Income adjusted	Unemployment adjusted
Non-metropolitan mainland	reference	reference	reference
Metropolitan Halifax	+0.11	−0.51	−0.07
Non-metropolitan Cape Breton Island	−0.46	−0.52	−0.08
Cape Breton Regional Municipality	−1.46	−1.28	−1.11
p-value:	< 0.001	< 0.001	< 0.001

p: probability that the estimated regional differences equal zero

TABLE 3
Disease-specific components of regional differences in life expectancy (in years) relative to non-metropolitan mainland Nova Scotia

	Metropolitan Halifax		Non-metropolitan Cape Breton Island		Cape Breton Regional Municipality	
	Women	Men	Women	Men	Women	Men
Cardiovascular disease	0.18	0.14	−0.14	−0.24	−0.36	−0.74
Cancer (all sites combined)	−0.06	0.02	−0.26	−0.21	−0.36	−0.36
Lung	−0.06	0.07	−0.05	−0.08	−0.12	−0.13
Colorectal	0.01	−0.02	0.01	−0.03	0.01	−0.04
Breast	−0.10	0.00	−0.10	0.00	−0.08	0.00
Respiratory diseases	0.00	−0.05	0.00	−0.01	0.00	−0.14

Municipality, whereas past discussions have primarily focused on the high cancer rates in this region.[22,27,28] In addition to the well established relationship between wealth and health, policy makers are now informed that neither income nor unemployment explain the provincial health disparities and the health concerns of Cape Breton Regional Municipality. These are examples of how small area comparisons may contribute to decision processes of policy makers. More applications are to be expected from future small area comparisons of morbidity, health care utilization and other descriptive measures and determinants of health.

Acknowledgments

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No	Region	Geography	Population	Deaths	Life expectancy		Standard error				
					Crude	Smoothed	SE_{SE}	SE_{POP}	SE_{M1}	SE_{M2}	SE_{M3}
1	NMM	Barrington	9,061	67	79.08	78.79	0.63	0.03	0.05	0.39	0.04
2	NMM	Shelburne	8,268	90	77.32	78.04	0.61	0.01	0.03	0.14	0.03
3	NMM	Argyle	9,155	70	80.93	79.14	0.68	0.07	0.05	0.39	0.05
4	NMM	Yarmouth	19,082	200	77.00	77.61	0.46	0.04	0.01	0.19	0.01
5	NMM	Clare	9,513	104	78.55	78.44	0.65	0.03	0.01	0.21	0.01
6	NMM	Digby	11,708	123	78.09	78.30	0.50	0.02	0.01	0.30	0.01
7	NMM	Queens Subdivision A	6,534	66	78.42	78.40	0.74	0.05	0.03	0.46	0.04
8	NMM	Queens Subdivision B	6,136	73	77.34	78.11	0.66	0.08	0.03	0.43	0.04
9	NMM	Annapolis Subdivision D	2,895	24	80.55	78.77	1.15	0.28	0.10	1.24	0.08
10	NMM	Annapolis Subdivision A	7,755	92	78.88	78.66	0.57	0.06	0.03	0.30	0.03
11	NMM	Annapolis Subdivision B	5,042	72	78.33	78.44	0.63	0.05	0.05	0.37	0.08
12	NMM	Annapolis Subdivision C	6,833	66	78.23	78.49	0.78	0.06	0.05	0.47	0.06
13	NMM	Lunenburg	37,847	381	78.76	78.70	0.29	0.01	0.01	0.16	0.01
14	NMM	Chester	11,117	101	78.03	78.53	0.66	0.04	0.04	0.39	0.04
15	NMM	Kings Subdivision A	25,094	187	79.02	78.93	0.36	0.02	0.02	0.11	0.02
16	NMM	Kings Subdivision C	13,705	128	77.35	78.25	0.49	0.01	0.03	0.29	0.03
17	NMM	Kings Subdivision B	12,003	48	85.69	81.21	0.66	0.13	0.08	0.30	0.15
18	NMM	Kings Subdivision D	9,416	72	81.44	79.81	0.56	0.03	0.06	0.24	0.05
19	NMM	West Hants	19,282	188	78.33	78.52	0.38	0.02	0.03	0.19	0.04
20	NMM	East Hants	21,400	123	78.17	78.58	0.43	0.05	0.04	0.31	0.05
21	NMM	Halifax Subdivision E	20,926	111	77.86	78.39	0.42	0.05	0.03	0.38	0.02
22	NMM	Halifax Subdivision F	6,505	47	81.31	79.03	0.79	0.06	0.05	0.44	0.06
23	NMM	Halifax Subdivision G	4,316	50	75.55	77.74	1.07	0.07	0.00	0.42	0.03
24	MH	Sambro	29,830	121	81.06	79.78	0.42	0.05	0.11	0.22	0.07
25	MH	Upper Sackville	21,568	68	79.98	79.35	0.50	0.02	0.61	0.31	0.24
26	MH	Herring Cove	12,341	111	75.85	77.34	0.51	0.02	0.56	0.21	0.22
27	MH	Sackville	25,472	92	78.65	78.94	0.45	0.01	0.40	0.32	0.20
28	MH	Clayton Park	24,261	189	78.44	78.63	0.37	0.00	0.31	0.22	0.17
29	MH	Spryfield/Armdale	19,850	195	77.67	78.13	0.40	0.00	0.37	0.16	0.20
30	MH	Peninsula South End	20,097	168	79.30	78.74	0.48	0.01	0.37	0.23	0.19
31	MH	Peninsula West End	23,912	232	79.07	78.64	0.37	0.00	0.38	0.11	0.20
32	MH	Peninsula North End	17,011	242	75.24	76.77	0.45	0.00	0.44	0.19	0.09
33	MH	Bedford	25,719	87	82.92	80.56	0.40	0.03	0.38	0.12	0.20
34	MH	Crichton Park Albro Lake	23,882	169	77.49	78.24	0.43	0.00	0.34	0.17	0.10
35	MH	Southdale Regional Woodside	22,982	165	79.19	78.75	0.41	0.01	0.52	0.13	0.11
36	MH	Eastern Passage Cow Bay	18,015	96	76.44	77.38	0.42	0.01	0.37	0.21	0.12
37	MH	Portland Estates	24,200	88	78.37	78.38	0.39	0.00	0.40	0.19	0.16
38	MH	Woodlawn Montebello Forest Hills	15,292	64	79.62	79.00	0.48	0.05	0.52	0.23	0.19
39	NMM	Colchester Subdivision C	28,242	276	77.51	77.92	0.34	0.01	0.02	0.19	0.02
40	NMM	Colchester Subdivision B	18,864	127	79.75	79.09	0.43	0.03	0.03	0.22	0.04
41	NMM	Colchester Subdivision A	3,886	31	80.35	78.88	0.84	0.05	0.09	0.45	0.06
42	NMM	Cumberland Subdivision A	4,449	53	78.73	78.59	0.91	0.06	0.07	0.62	0.13
43	NMM	Cumberland Subdivision B	8,582	86	79.14	78.77	0.55	0.03	0.04	0.37	0.06
44	NMM	Cumberland Subdivision C	17,041	180	78.22	78.28	0.45	0.02	0.03	0.23	0.04
45	NMM	Cumberland Subdivision D	4,930	64	75.75	77.93	0.84	0.05	0.10	0.44	0.06
46	NMM	Pictou Subdivision A	10,997	111	78.22	78.37	0.54	0.01	0.05	0.40	0.02
47	NMM	Pictou Subdivision B	16,349	143	78.88	78.62	0.42	0.02	0.03	0.23	0.02
48	NMM	Pictou Subdivision C	23,039	228	78.15	78.28	0.36	0.01	0.03	0.16	0.01
49	NMM	St. Mary's	2,805	36	75.94	77.98	1.04	0.04	0.08	0.81	0.26
50	NMM	Guysborough	8,391	87	78.30	78.41	0.55	0.01	0.03	0.35	0.09

No	Region	Geography	Population	Deaths	Life expectancy		Standard error				
					Crude	Smoothed	SE_{SE}	SE_{POP}	SE_{M1}	SE_{M2}	SE_{M3}
51	NMM	Antigonish Subdivision A	12,905	110	79.29	78.86	0.49	0.02	0.01	0.20	0.01
52	NMM	Antigonish Subdivision B	7,383	41	79.85	78.95	0.84	0.04	0.04	0.55	0.04
53	NMCBI	Inverness Subdivision C	7,855	63	77.92	78.10	0.63	0.02	0.07	0.30	0.10
54	NMCBI	Inverness Subdivision B	7,065	72	76.19	77.60	0.77	0.06	0.06	0.33	0.07
55	NMCBI	Inverness Subdivision A	6,828	68	78.81	78.46	0.68	0.07	0.12	0.52	0.09
56	NMCBI	Richmond Subdivision B	4,292	42	78.38	78.38	0.91	0.00	0.04	0.38	0.06
57	NMCBI	Richmond Subdivision A	4,467	52	77.50	78.13	0.78	0.06	0.07	0.50	0.09
58	NMCBI	Richmond Subdivision C	2,504	22	82.14	78.30	1.18	0.16	0.15	0.47	0.17
59	CBRM	CBRM:Louisbourg Area	3,937	20	83.81	78.33	1.01	0.23	0.13	0.71	0.03
60	CBRM	CBRM:Sydney	40,602	489	75.77	76.34	0.27	0.01	0.08	0.13	0.05
61	CBRM	CBRM:North Sydney	35,559	261	78.27	78.02	0.34	0.02	0.02	0.25	0.02
62	CBRM	CBRM:Glace Bay	41,401	419	76.29	76.62	0.29	0.01	0.10	0.12	0.05
63	NMCBI	Victoria Subdivision B	5,243	51	77.89	78.20	0.80	0.10	0.05	0.62	0.05
64	NMCBI	Victoria Subdivision A	3,673	34	75.17	77.78	1.19	0.07	0.09	0.51	0.07

Region abbreviations: NMM, non metropolitan mainland; MH, metropolitan Halifax: NMCBI, non metropolitan Cape Breton island; CBRM, Cape Breton Regional Municipality.

Population: average population size in the 1995–1999 period estimated by the average of 10 repeated allocation procedures (see text).

Deaths: average annual number of deaths in the 1995–1999 period, estimated by the average of 10 repeated allocation procedures on the basis of both postal codes and geographic specifications (see text).

Life Expectancy: the crude and spatially smoothed estimates of life expectancy are calculated on the basis of deaths and population estimates described above.

Prevalence of PSA testing and effect of clinical indications on patterns of PSA testing in a population-based sample of Alberta men

S Elizabeth McGregor, Heather E Bryant, Rollin F Brant and Peter J Corbett

Abstract

An age-stratified population-based random digit dial (RDD) telephone survey determined awareness and prevalence of prostate-specific antigen (PSA) testing among Alberta men aged 40–74 years, and assessed the role of indications for PSA testing in explaining patterns of PSA testing. The sample of 1984 men (participation rate 65%) with no history of prostate cancer was divided into three age strata: 40–49, 50–59, and 60–74 years. Awareness of PSA tests was low, with fewer than half of the men indicating they had ever heard of PSA tests. The percentage of men who had ever had PSA testing was 4.5%, 13.1%, and 22.2% respectively, in the three age strata. PSA testing was strongly associated with having at least one clinical indication for PSA testing (prevalence 21.8%, 26.9%, and 42.2% respectively). PSA testing rates were very low among men who had no clinical indications for PSA testing, suggesting infrequent PSA screening prior to the survey. PSA testing patterns in this population-based sample were consistent with Alberta clinical practice guidelines.

Key words: *mass screening; prevalence; prostate cancer; PSA*

Introduction

The introduction of prostate-specific antigen (PSA) testing in the late 1980s has had a dramatic effect on the detection of prostate cancer. Prior to that time, Canadian age-standardized prostate cancer incidence rates were increasing slowly but steadily over a 25 year period.[1] However, starting in 1990, the year significant utilization of PSA tests began in Canada,[2] there was a rapid increase in prostate cancer incidence, peaking in 1993. The peak was followed by a decline in the age-standardized incidence, although rates remain higher than in the pre-PSA testing era. The early steady increases in incidence in Canada and elsewhere have been attributed to increased detection of incidental prostate cancers found during transurethral resection of the prostate (common in the treatment of benign prostatic disease) with the more recent large and transient increases attributed to the availability of PSA testing.[3–7]

Trends in age-standardized incidence rates of invasive prostate cancer in Alberta show the same pattern (Figure 1).

Declines in incidence after an initial rapid increase subsequent to the introduction of PSA testing have been observed in other areas with population-based cancer registries,[8–12] and in administrative data on PSA testing rates. Patterns in incidence correlate most strongly with patterns of first-time PSA testing[13] consistent with the hypothesis that PSA testing is detecting clinically unapparent cases from an existing prevalence pool.

The Canadian Task Force on the Periodic Health Exam,[14] systematic reviews by provincial health agencies,[15–17] the US Preventive Services Task Force,[18] and the American College of Physicians[19] have concluded that there is insufficient evidence to recommend routine PSA screening of asymptomatic men over 50 years of age. This conflicts with the American Cancer Society[20] and the American Urological Association (AUA)[21] which recommend annual PSA testing and digital rectal examination (DRE) for men aged 50 and over with at least a ten-year life expectancy, with screening beginning earlier for high-risk men. All recommendations include the need to inform men about the potential risks and benefits of screening.

Alberta has always had a very restrictive policy[22] on PSA testing. Reimbursement of PSA testing for screening purposes has never been authorized by the provincial health insurance plan. PSA testing in Alberta has been restricted to the monitoring of men who have been diagnosed and treated for prostate cancer; evaluation of patients with "symptoms of prostatism" or an abnormal DRE; and for men at higher risk of prostate cancer based on family history and/or ancestry. The guideline indicates prostatism is a symptom of both benign prostatic hyperplasia (BPH) and prostate cancer.

PSA testing is often used to rule out prostatic carcinoma among men presenting with lower urinary tract symptoms.[23] However, the prevalence of prostatic cancer among men presenting for prostate cancer

Author References

S Elizabeth McGregor, Heather E Bryant, Division of Epidemiology, Prevention & Screening, Alberta Cancer Board
Rollin F Brant, Department of Community Health Sciences, University of Calgary
Peter J Corbett, Department of Oncology, University of Calgary *and* Tom Baker Cancer Centre
Correspondence: S Elizabeth McGregor, Division of Epidemiology, Prevention & Screening, Alberta Cancer Board, 1331–29th Street NW, Calgary, Alberta, Canada T2N 4N2; Fax: (403) 270-8003; E-mail: elizabeth.mcgregor@cancerboard ab.ca

FIGURE 1
Invasive prostate cancer*, Alberta, 1973–1999

FIGURE 1
Invasive prostate cancer*, Alberta, 1973–1999
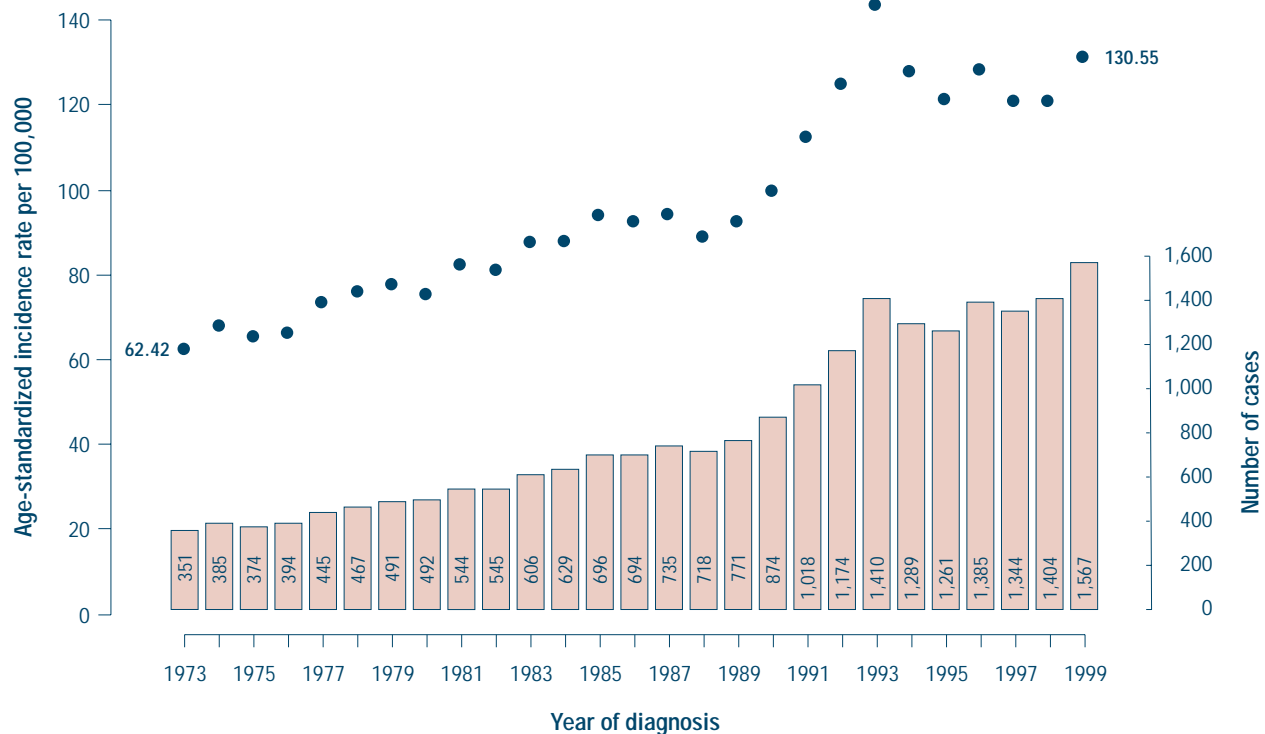
Standardized to 1991 Canada Revised Postcensal Population Estimate

* Alberta Cancer Registry

screening does not vary in men with or without urinary symptoms[24,26] and BPH is not etiologically related to prostate cancer.[25] Some health policies actually recommend against the use of PSA testing in men with uncomplicated urinary symptoms.[27] In this study, it was hypothesized that the presence of clinical indications for PSA testing including lower urinary tract symptoms would be an important predictor of PSA testing in Alberta because the PSA tests have never been approved for prostate cancer screening purposes.

The purpose of the current study was to determine the age-specific prevalence of PSA testing among Alberta men aged 40–74 years and to describe patterns of PSA testing among men with and without clinical indications for PSA testing.

Methods

An age-stratified population-based random digit dial (RDD) telephone survey was used to assess awareness of PSA testing and self-reported rates of PSA testing among Alberta men. Age strata were 40–49 years, 50–59 years, and 60–74 years. The two oldest age groups were over-sampled to allow for an expected higher prevalence of moderate to severe lower urinary tract symptoms, and hence ineligibility for screening.

The survey (available upon request) covered a variety of topics, and was introduced as concerning men's health to minimize bias due to men declining to participate specifically because of lack of interest in prostate cancer. The 36 questions ascertained information on health status, primary care practices, lower urinary tract symptoms, knowledge and awareness of prostate cancer, and demographics. Questions included awareness of the PSA test, discussion of the PSA test with a doctor, whether the respondent has had a PSA test, and the reason the test was performed. Only men who indicated they were aware of PSA tests were asked about

their use of them. Definitions of PSA tests were provided. Some questions were modified from a Canadian RDD telephone survey conducted in January 1995.[28] The AUA symptom index[29] was used to assess the severity of lower urinary tract symptoms over the previous month and classify men as having mild, moderate, or severe lower urinary tract symptoms. Indications for PSA testing were determined using criteria outlined in the Alberta clinical practice guideline (CPG)[22](Table 1).

The survey instrument was initially pilot tested on 41 male volunteers. Final revisions were made after a second pilot test on 43 RDD selected men.

The RDD computer-assisted telephone interview survey was conducted over a four-week period in November/December 1996. The short time frame for data collection reduced the chances of external influences (e.g., media reports) affecting survey responses. Data collection was contracted to

TABLE 1
Indications for PSA testing used to classify RDD survey respondents

Clinical Practice Guideline recommendation	Criteria from RDD telephone survey
Follow-up of a patient with:	
Prostate cancer[a]	"Yes" response to "Have you ever been told by a doctor that you had prostate cancer?"
Evaluation of a patient with:	
Symptoms of prostatism	AUA Symptom Score ≥8 (Moderate/severe lower urinary tract symptoms)
Benign prostatic hypertrophy (BPH)	"Yes" response to "Have you ever been told by a doctor you had an enlarged prostate (sometimes called BPH)?"
Abnormal digital rectal exam (DRE)	Men who indicated result of most recent DRE was abnormal.
Men at higher risk of cancer:	
First degree family history of prostate cancer diagnosed by age 70 years	Men who reported any first degree family history of prostate cancer
African-American men	African ancestry indicated in response to question on ethnic origin

[a] Note: men with a history of prostate cancer were excluded from this analysis.

the Population Research Laboratory (PRL), University of Alberta. The sampling frame consisted of a random sample of Alberta telephone numbers from the PRL working bank of residential telephone numbers. Standard protocols were used to determine eligibility, invite participation, and administer the telephone interview. The most recent birthday was used to randomly select the person to be interviewed if there was more than one eligible man in a household. Calls were made over three different time periods on all weekdays and Saturdays and two time periods on Sundays, with up to 12 attempts to reach each phone number.

Age-specific estimates of the prevalence of lower urinary tract symptoms and indications for PSA testing were calculated. Point estimates and 95% confidence intervals for PSA test awareness and prevalence of PSA testing were calculated for the three age groups. The prevalence of PSA testing among men with and without indications for PSA testing was described. Comparisons between the three age groups were made using chi-square tests for categorical variables and Wilcoxon rank sum tests[30] for ordinal variables. Exact tests were used for categorical variables when an expected

cell frequency was less than five. Logistic regression was used to test for differences in PSA testing rates by age group and indication for PSA testing. Subjects with missing values were excluded from the analysis and the number of missing cases is indicated in the descriptive tables. All analyses were conducted using SAS[31] and S-PLUS[32] statistical software.

Ethical approval for the study was obtained from the Research Ethics Committee of the Alberta Cancer Board and the Conjoint Medical Ethics Review Committee of the Faculty of Medicine, University of Calgary.

Results

There were 2,016 completed interviews (cooperation rate[33] 64.7%). The most common reason for non-response, after excluding ineligible telephone numbers, was household refusal (35.9%) followed by never answered (34.7%). Comparison with Alberta 1996 Census data indicated the RDD sample was reasonably representative of Alberta men of the same age groups (data not shown). Thirty-two men (1.6%) who reported they had prostate cancer were eliminated from further analysis.

Table 2 shows a description of the sample of 1,984 men without a history of prostate cancer. Among men who indicated they had a regular doctor, 96.2% indicated their doctor was a GP or family doctor. The percentage of men who indicated they went for regular checkups increased with age: 65.2%, 76.5%, and 81.0% of men aged 40–49, 50–59, and 60–74 years respectively.

Visits to a urologist and severity of urinary symptoms increased with age (Table 3). Men with a history of prostate problems were more likely to have discussed urinary symptoms with their doctor (66.2% of men with any prostate problem versus 14.5% of men without a prostate problem, age-adjusted OR = 11.2, 95% CI 8.6, 14.5) and to have visited a urologist (53.8% of men with any prostate problem versus 12.9% of men without a prostate problem, age-adjusted OR = 6.8, 95% CI 5.2, 8.8).

The majority of men in all three age groups were scored as having mild lower urinary tract symptoms.

As expected, older men were more likely to have at least one indication for PSA testing (Table 3). A history of BPH, moderate/severe lower urinary tract symptoms, and an abnormal finding on most recent DRE increased with age (all p < 0.001). First degree family history of prostate cancer was present in about 10% of men and did not vary by age group (p = 0.6). African ancestry was very infrequent in this sample (< 1% of men in all age groups). At least one indication for PSA testing was common, especially in men aged 60–74 years.

Men who had heard of prostate cancer (n= 1,930) were asked whether they had discussed it with their doctor. The percentage of men who had done so increased with age: 24.4%, 32.1%, and 43.6% of men aged 40–49, 50–59, and 60–74 years respectively (p< 0.001). The most frequent topics discussed were: general information (52.5%), detection of prostate cancer (47.2%) (only 8.5% specifically discussed PSA testing), and symptoms (32.6%). Treatment options and prognosis (5.7%) and other topics (8.5%) were far less frequently discussed. There were few differences by age group among the topics men discussed with their doctors. Age guide-

lines for screening were more often discussed in men aged 40–49 years (15.4% 40–49; 6.8% 50–59; 5.9% 60–74 years, p= 0.002).

Fewer than half the men had ever heard of PSA tests. Awareness increased with age with 26.7%, 40.0%, and 45.1% of men aged 40–49, 50–59, and 60–74 respectively having heard of the PSA test. Rates of PSA testing were low and strongly associated with increasing age with 4.5%, 13.1%, and 22.2% of men in the same three age groups reporting having had at least one PSA test. Most men (57.0%) had only one PSA test. The mean (median) time since last PSA test was 1.3 (0.9) years and ranged from just over one month to 12.4 years, with 98% reporting their most recent PSA test within the past five years. There was no difference in the median time since the last PSA test among the three age groups (Wilcoxon rank sum test, p= 0.5). Nine men could not recall the date of their last PSA test.

Physicians were the most common initial source of information for men who had been tested. This was particularly important for men aged 40–49 years where 74.1% indicated they first learned about PSA tests from their doctor, compared to 50.0% of men 50–59 years and 61.6% of men aged 60–74 years. Reading material such as newspapers and magazines were cited by 18.9% of men aged 50 and older as the next most important way they learned about PSA tests.

Table 4 shows PSA testing rates by whether or not at least one clinical indication for PSA testing was present. PSA testing in men with no indication for PSA testing was considered prostate cancer screening. The majority (63.8%) of PSA tests in these men were done at a routine checkup suggesting these tests were for screening purposes and 9.4% had specifically asked their doctor for a PSA test. Most men had been tested once (55.9%) or twice (15.8%) and this did not vary by age. The most common way these men first learned about PSA testing was from their physician (57.5%) followed by newspapers or magazines (15.0%). Few men aged 40–49 were aware of PSA tests and PSA testing was very infrequent.

Rates of PSA testing were higher in men with at least one clinical indication for PSA testing compared to men with no indications (Table 4). Awareness of PSA tests was similar in all three age groups but rates

of PSA testing increased with age. Like the men with no indication for PSA testing, 59.9% of men who had been tested first learned of the PSA test from their doctor. The majority of men reported having had one (57.8%), or two PSA tests (12.7%).

TABLE 2
Demographic characteristics, health status and primary care practices by age group (n = 1,984), % (n)

	Age group (years)		
	40–49 (n = 603)	50–59 (n = 702)	60–74 (n = 679)
Marital status			
Single	8.1 (49)	4.6 (32)	6.8 (46)
Married/common-law	83.9 (506)	85.9 (602)	81.2 (550)
Separated/divorced/widowed	8.0 (48)	9.6 (67)[a]	12.0 (81)[b]
Highest educational attainment			
Less than grade 9	2.0 (12)	8.2 (57)	16.0 (108)
Grades 9 to 13	30.2 (181)	37.6 (263)	48.6 (328)
Some/completed trade school/ college	36.0 (216)	22.3 (156)	15.7 (106)
Some/completed university	31.8 (191)[c]	31.9 (223)[c]	19.7 (133)[d]
Employment status			
Employed full time	91.0 (549)	82.0 (575)	29.3 (199)
Employed part time	1.3 (8)	3.3 (23)	6.3 (43)
Retired	0.7 (4)	9.4 (66)	61.9 (420)
Other	7.0 (42)	5.3 (37)[a]	2.5 (17)
Rating of health status compared to others of same age			
Excellent	22.4 (135)	23.1 (162)	16.2 (110)
Very good	38.1 (230)	36.2 (254)	34.9 (237)
Good	31.0 (187)	27.2 (191)	30.8 (209)
Fair/Poor	8.5 (51)	13.4 (94)[a]	18.1 (123)
Smoking habits			
Never smoker	34.5 (208)	26.5 (186)	29.0 (197)
Ex-smoker	33.5 (202)	46.2 (324)	52.7 (358)
Occasional smoker	2.8 (17)	3.0 (21)	1.0 (7)
Current smoker	29.2 (176)	24.4 (171)	17.2 (117)
Type of primary care provider			
Regular doctor	75.0 (452)	80.6 (566)	90.0 (611)
See a variety of doctors/attend Walk-in clinic	9.3 (56)	7.3 (51)	3.8 (26)
No regular doctor	15.8 (95)	12.1 (85)	6.2 (42)
Time since last checkup or physical exam			
Within the past year	39.1 (236)	48.3 (339)	58.8 (399)
1 to 5 years ago	38.6 (233)	34.1 (239)	29.6 (201)
More than 5 years ago	13.4 (81)	9.5 (67)	5.7 (39)
Can't recall	8.8 (53)	8.1 (57)	5.9 (40)

Missing information on: [a] = 1; [b] = 2; [c] = 3; [d] = 4 subjects.

The reason for their most recent PSA test was a problem their doctor wanted to check (32.4%), symptoms that were concerning them (16.9%), or a routine checkup (37.3%).

Logistic modeling showed the effect of indication for PSA testing (OR= 2.5, 95% CI 1.9, 3.3) was independent of the age effect (OR= 3.1, 95% CI 2.0, 4.9 for men aged 50–59; OR= 5.2, 95% CI 3.4, 8.1 for men aged 60–74).

Table 5 describes PSA testing practices among the subset of men who were aware of the PSA test. Less than half of these men had discussed PSA tests with their doctors. This pattern varied by age, with older men more likely to have discussed tests. PSA testing rates were higher in men with at least one clinical indication for PSA testing compared to men with no indications in all three age groups. Again, PSA testing was strongly associated with age. The percentage of men who reported having at least one PSA test increased with age for men both with and without an indication for PSA testing. Logistic modeling showed the effect of indication for PSA testing (OR= 2.1, 95% CI 1.5, 2.9) was independent of the age effect (OR= 2.6, 95% CI 1.6, 4.3 for men aged 50–59; OR= 4.6, 95% CI 2.9, 7.5 for men aged 60–74).

Discussion

This study found that in late 1996, six years after PSA tests had become available, awareness of PSA tests was quite low among Alberta men, as were rates of PSA testing. Patterns of self-reported PSA testing were consistent with trends in incidence data on invasive prostate cancer in Alberta over the same time period. There were few differences by age in the lifetime number of PSA tests and time since last PSA test, reflecting the relatively short time PSA testing had been available. The majority (57%) of men who had been tested reported having a single PSA test, consistent with the introduction of a new screening test and relatively little opportunity yet for serial screening.

Health care utilization records show similar patterns of PSA testing in three Canadian provinces. Large increases in the

number of PSA tests ordered have been reported in Saskatchewan (1990–1994)[16] and Ontario (1988–1996).[34] Utilization patterns in British Columbia showed the majority of PSA tests were ordered by general practitioners, and 76% of men tested had a single PSA test over the one-year study period, implying PSA testing was used for prostate cancer screening.[15] A linkage study using PSA test records from two laboratories, one private and one hospital based, in Ontario reported a mean (median) of 1.5 (1) PSA tests in men with no diagnosis of prostate cancer.[35] Similar to the current study, PSA testing rates were highest in men 50–70 years of age.

PSA testing rates were very low among men who had no clinical indications for PSA testing, suggesting infrequent screening with PSA testing in Alberta up to the time of the RDD survey. Both PSA test awareness and testing were strongly associated with increasing age. This may reflect the increase in prevalence of clinical indications for PSA testing, increasing index of suspicion by physicians, and the increased experience with, and perhaps salience of, prostate cancer as men age. Very little PSA testing, especially in the screening context, occurred in men aged 40–49 years.

TABLE 3
Lower urinary tract symptoms and indications for PSA testing by age group (n = 1,984), % (n)

	Age group (years)		
	40–49 (n = 603)	50–59 (n = 702)	60–74 (n = 679)
Ever bothered by urinary symptoms enough to discuss them with your doctor?			
Yes	15.5 (93)	21.1 (148)	28.5 (193)
No	84.5 (508)[b]	78.9 (554)	71.5 (484)[b]
Ever visited a urologist?			
Yes	11.6 (70)	16.7 (117)	27.0 (182)
No	88.4 (532)[a]	83.3 (584)[a]	73.0 (493)[c]
AUA symptom score			
None to mild	94.3 (561)	89.5 (623)	80.9 (538)
Moderate	5.2 (31)	9.3 (65)	17.3 (115)
Severe	0.5 (3)	1.2 (8)	1.8 (12)
Not scored due to missing information	8	6	14
Indications for PSA testing			
Evaluation of a patient with:			
Symptoms of prostatism			
(AUA Score ≥ 8)	5.7 (34)	10.5 (73)	19.1 (127)
Personal history of BPH	3.2 (19)	9.8 (69)	23.1 (157)
Abnormal DRE (most recent)	3.0 (18)	5.4 (38)	8.4 (57)
Men at higher risk of cancer:			
First degree family history	11.6 (70)	10.0 (70)	10.3 (70)
African-American ancestry	0.2 (1)	0.3 (2)	0.4 (3)
Number of indications for PSA testing (p < 0.001)			
None	78.2 (467)	73.0 (509)	57.7 (385)
At least one indication	21.8 (130)	27.0 (188)	42.3 (282)
Not assessed due to missing data for AUA Symptom Score	6	5	12

Missing information on: [a] = 1; [b] = 2 ; [c] = 4 subjects

Almost all studies have reported increased PSA testing rates with age.[28,36–39] The current findings are similar to those from a January 1995 RDD survey of Canadian men in which 6%, 13%, and 23% of men aged 40–49, 50–59, and 60–69 respectively reported ever having a PSA test.[28] Much higher PSA testing rates were found in a June 1996 mailed survey of Quebec City area men, where 21%, 29%, and 24% of men aged 50–59, 60–69 and 70 or older respectively had undergone a PSA test in the previous 12 months.[40] Men with prostate cancer were excluded from the analysis. These rates are considerably higher than rates in the current study even though the current study assessed lifetime PSA testing compared to PSA tests in the previous 12 months. The presence of a large prostate cancer screening study[41] in Quebec City may have increased awareness of PSA testing among both men, and health care providers, in the Quebec City area.

The higher rates of PSA testing reported for US men[36,42] may also be due to higher awareness of PSA tests among both men and physicians. A statewide survey of men aged 50 and over in New York State reported 58% were aware of PSA tests and 64% of them had had at least one PSA test.[43] Differences in methodology, timeframe, health care settings, and screening policy environments may also explain part of the variation in prevalence estimates.

In contrast to the findings of Mercer et al.[28] where physicians and the media played an equal role in PSA test awareness, in the current study physicians were more often cited as the way men first became aware of the PSA test. This difference may be due to the differences in media promotion of prostate cancer screening across provinces with different policies for PSA testing. The use of PSA testing primarily in men who have at least one indication for PSA testing when discussion of PSA tests likely arose in the investigation of a clinical problem, may also explain this.

PSA testing patterns based on self-report in this population-based sample appear to be consistent with the current Alberta clinical practice guideline and previous restrictions of PSA testing in Alberta. The importance of clinical indications for PSA testing

in explaining PSA testing rates is shown by the marked differences in PSA testing rate among men with and without an indication for PSA testing in all three age groups. The prevalence of clinical indications for PSA testing are sufficiently high in men

TABLE 4
PSA test awareness and testing by age group and indication, % (n)

	Age group (years)		
	40–49	**50–59**	**60–74**
Men with no clinical indications for PSA testing (n = 1,361)			
Ever discussed prostate cancer with their doctor (p < 0.001)			
Yes	20.3 (95)	27.9 (142)	36.1 (139)
No	79.7 (372)	72.1 (367)	63.9 (246)
Heard of PSA test? (p < 0.001)			
Yes	21.2 (99)	38.3 (195)	39.0 (150)
(95% CI)	(17.7, 25.1)	(34.2, 42.6)	(34.2, 43.9)
Had at least one PSA test (p < 0.001)			
	2.8 (13)	11.2 (57)	14.8 (57)
(95% CI)	(1.6, 4.7)	(8.7, 14.2)	(11.6, 18.7)
Men with at least one clinical indication for PSA testing (n=600)			
Ever discussed prostate cancer with their doctor (p = 0.009)			
Yes	36.9 (48)	41.0 (77)	51.4 (145)
No	63.1 (82)	59.0 (111)	48.6 (137)
Heard of PSA test? (p = 0.12)			
Yes	46.9 (61)	45.2 (85)	54.3 (153)
(95% CI)	(38.6, 55.5)	(38.3, 52.4)	(48.4, 60.0)
Had at least one PSA test (p < 0.001)			
	10.8 (14)	18.6 (35)	33.0 (93)
(95% CI)	(6.5, 17.3)	(13.7, 24.8)	(27.8, 38.7)

TABLE 5
PSA test practices among men in RDD sample who were aware of the PSA test by age group (n = 748), % (n)

	Age group (years)			
	40–49 **(n = 161)**	**50–59** **(n = 281)**	**60–74** **(n = 306)**	**Total** **(n = 748)**
Discuss PSA test with their doctor? (p < 0.001)				
Yes	23.6 (38)	39.2 (110)	52.6 (161)	41.3 (309)
No	76.4 (123)	60.8 (171)	47.4 (145)	58.7 (439)
Ever had a PSA test? (p < 0.001)				
Yes	16.8 (27)	32.7 (92)	49.4 (151)	36.1 (270)
(95% CI)	(11.8, 23.3)	(27.5, 38.4)	(43.8, 54.9)	(32.7, 39.6)
Ever had a PSA test by clinical indication among men aware of PSA tests (n = 743)				
At least one indication **(n = 299) (p < 0.001)**	23.0 (14)	41.2 (35)	60.8 (93)	47.5 (142)
(95% CI)	(14.2, 34.9)	(31.3, 51.8)	(52.9, 68.2)	(41.9, 53.2)
No indications present **(n = 444) (p < 0.001)**	13.1 (13)	29.2 (57)	38.0 (57)	28.6 (127)
(95% CI)	(7.8, 21.2)	(23.3, 36.0)	(30.6, 46.0)	(24.6, 33.0)

aged 50–74 years to have a marked effect on PSA testing rates. The use of PSA testing in men with clinical indications for PSA testing may explain the rapid increase in prostate cancer incidence in Alberta in the early 1990s.

Other surveys have not assessed the role of clinical indications in explaining patterns of PSA testing in a population-based sample. However, PSA testing rates were higher among men with BPH, moderate or severe lower urinary tract symptoms, and a family history of cancer in the Health Professionals Followup Study.[44] A cross-sectional survey of US primary care physician visits found PSA tests were much more frequently ordered in all age groups for men with lower urinary tract symptoms, a diagnosis of BPH, or attending for a general medical exam.[45]

Two studies of Australian men who had visited their doctors for troublesome lower urinary tract infections showed they were much more likely to have had a PSA test.[46,39] The strongest predictors of PSA testing among a sample of men aged 40–79 years in New South Wales, Australia were age, doctor's recommendation and presence of lower urinary tract symptoms.[47]

The lack of testing in some men with at least one clinical indication for PSA testing may be due to several reasons. Firstly, men may not seek care for symptoms. Not all men with lower urinary tract symptoms seek care.[48–50] Just over one third of British men with severe urinary symptoms and over one half (51.9%) of men with moderate urinary symptoms did not seek advice from their physicians.[51] Secondly, PSA testing may not have been offered to men who sought health care advice for their urinary symptoms. It is also possible that PSA testing was not available at the time they received a diagnosis of BPH. Finally, lack of testing may also be due to men declining a PSA test.

The prevalence of lower urinary tract symptoms in the Alberta sample is somewhat lower than that reported in an Olmsted County study,[52–53] a mailed survey of British men,[54] and a self-administered survey of a large consecutive sample of Australian men presenting for an office visit.[55] The higher prevalence of symptoms in the Olmsted County studies may be partly due to non-response bias resulting in a higher prevalence of urological disease among study participants.[56] Forty percent of the Australian sample were over 70 years of age, which may partly explain the higher estimate due to the increase in prevalence of lower urinary tract symptoms with age.

The lower prevalence of symptoms in the Alberta sample may also be due to the mode of questionnaire administration. Telephone interviews have been found to result in lower AUA symptom scores when compared to scores obtained from self-administered questionnaires.[57] A telephone survey of a random sample of Australian men reported the prevalence of moderate/severe symptoms of 4%, 11%, 14%, and 18% in men aged 40–49, 50–59, 60–69, and 70–79 years respectively.[47] These rates are similar to the prevalence rates found in the Alberta sample.

The current study is limited by the fact that the prevalence estimates are based on self-report. The validity of self-reported PSA testing has not been reported for Canadian men. A convenience sample of 276 male patients attending nine primary care offices in northwest Ohio found the sensitivity and specificity for PSA testing was 74% and 65% when compared to medical records.[58] Thus, 26% of men in the sample were unaware or failed to recall whether they had a PSA test. However, a larger proportion of men (35%) who had no record of a PSA test in the chart audit falsely reported they had been tested.

It is not clear that results from validation studies carried out in the US are generalizable to the Alberta setting. Social desirability bias may cause more US men to falsely report they have been screened in order to seem compliant with recommendations for prostate cancer screening that are more common in the US. A validation study has been carried out as part of this research and will be reported separately.

The classification of men as to whether they had a clinical indication for PSA testing in the current study was also based on self-report including a history of BPH. A comparison of self-report to medical records among controls in a prostate cancer case control study selected from members of a large health maintenance organization found that men tended to over-report genitourinary diseases that lack explicit diagnostic criteria, including BPH.[59] However, the authors concluded that self-report may provide a more complete estimate of BPH due to lack of completeness of medical records, high inter-physician variation, and inconsistent timeframes covered in the medical records.

The key strengths of the current study are the population-based sample, use of a valid and reliable measure of lower urinary tract symptoms, and a sufficiently large sample size to permit calculation of age-specific rates of PSA testing. Strong age effects were found, highlighting the usefulness of age-specific estimates. The sample size was sufficient to provide reasonably precise estimates of PSA testing in the subgroup of men with no indications for PSA testing who were considered eligible for screening. The cooperation rate of 65% compares favorably with response rates in other RDD surveys in general[60–61] and of men about prostate cancer in particular.[28,43,62] Comparison with census data indicated the RDD sample was representative of Alberta men in these three age groups with respect to demographic characteristics increasing the generalizability of the findings.

Current recommendations emphasize the importance of informing men about the potential harms and benefits of screening for prostate cancer and involving them in the decision-making about whether or not to undergo screening.[63–64] The potential for increased PSA testing as awareness of PSA tests increases is demonstrated by the higher rates of PSA testing in men of all age groups who are aware of PSA tests. The focus of education about prostate cancer screening should be to ensure that due consideration of potential benefits and harms is undertaken as awareness increases. Research on how both physicians and men respond to this potential demand is required.

It is concluded that screening for prostate cancer using the PSA test was infrequent in Alberta six years after PSA testing had become available and just after the release of

prostate cancer screening clinical practice guidelines. There is some evidence that a CPG recommending against prostate cancer screening in Saskatchewan reduced the use of PSA tests by family physicians.[34] These findings provide baseline data against which to evaluate changes in uptake of prostate cancer screening subsequent to the release of the CPGs and introduction of education and awareness programs about prostate cancer screening.

Acknowledgments

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Book Review
Territoire et vieillissement

In this slim volume, part of a specialized collection entitled "Médecine et société", Jean-Pierre Thouez of the Université de Montréal approaches the topic of aging from the perspective of social, and more specifically, health, geography. This field studies interactions between people and space: disability in older adult life has spatial implications because the possibilities for independent living in the community and social participation of a disabled senior depend upon the supports and/or barriers created by the physical and social environments. In this context, Thouez focuses on the implications of disability in older adult life for the financing and delivery of health and social services at the local, regional and national levels.

In Part 1, Thouez succinctly describes the key elements of aging as an individual experience and as a social phenomenon. He emphasizes the multidimensionality (bio-physical, cognitive and social) and heterogeneity of individual aging: biological make-up, personal choices, life events, occupation and culture determine the rate at which one ages. On the ambiguity of the meaning of "old age", Thouez quotes Henripin and Loriaux's incisive observation regarding the discrepancy between increasing healthy life expectancy and decreasing age of retirement: "Si la vieillesse biologique recule, celle de la vieillesse sociale progresse". The epidemiological transition from infectious to chronic disease prevalence resulting from an aging population is described, with a review of models classifying chronic diseases based on their direct or an indirect association with age.

Part 2 concentrates on the conceptualization and measurement of disability and its translation into health program and policy. Thouez provides an excellent critical summary of the evolution of the notion of disability and handicap and presents a very useful glossary of terms used in this area (e.g., independence, assistance, disability, activity limitation, impairment, handicap, assistance). Key indicators and instruments to measure disability are described and assessed, including the standard measures of Activities of Daily Living (ADL) and Instrumental Activities of Daily Living (IADL). Thouez shows how the concern to diagnose and serve individuals with varying needs and resources at a local level has led to the development of instruments like the Système de mesure de l'autonomie fonctionnelle (SMAF) in Quebec, whereas the need to identify broader population characteristics for regional and national resource allocation and program development leads to surveys like the Health and Activity Limitation Survey (HALS). He then describes some regional resource allocation approaches developed in Canada and France that include the size and needs of the older adult population as variables.

Territoire et vieillissement is a clear and concise introduction to health and social policy and program issues related to aging and disability. In addition to its probable intended use as a text for students in health and social services and public administration, it is a good reference for government health analysts. The text boxes of key concepts, theoretical models and measurement instruments are particularly helpful. Although it is a basic resource, the book does raise a few thorny policy debates that are the stuff of real-life decision-making, such as the issue of a needs-based or a rights-based approach in managing disability and the problem of unmet needs in resource allocation. Finally, not the least of its qualities is that it can be read easily in a day. ■

Overall Rating:
Good

Strengths:
Concise, clear introduction to individual and population aging and the social, health and policy consequences arising from disability. Good presentation of key concepts, theoretical models and measurement instruments.

Weaknesses:
None identified

Audience:
Health and social science and service students, service providers and planners.

Louise A Plouffe
Manager, Knowledge Development
Division of Aging and Seniors
Population and Public Health Branch
Health Canada
Tunney's Pasture
AL: 1908A1
K1A 1B4

Calendar of Events

October 24–26, 2002 **Halifax, Nova Scotia**	"Health Research in Rural and Remote Canada: Meeting Challenges and Creating Opportunities"	Tel.: (807) 343-2136

October 24–26, 2002
Montréal, Quebec
	"Aging and Society: Taking Charge of the Future"
	  Conference of the Canadian Association on
	    Gerontology
		Canadian Association on Gerontology
		100–824 Meath Street
		Ottawa, Ontario K1Z 6E8
		Tel.: (613) 728-9347
		Fax: (613) 728-8913
		E-mail: info@cagacg.ca
		< www.cagacg.ca>

November 3–5, 2002
Winnipeg, Manitoba
	"Injury Prevention Beyond 2002"
	  8th Annual Conference of the Canadian Coalition for
	    Agricultural Safety and Rural Health
		Canadian Coalition for Agricultural
		    Safety and Rural Health
		103 Hospital Drive, Box 76
		Saskatoon, Saskatchewan S7N OW8
		Tel.: (306) 966-8499
		Fax: (306) 966-8891

November 29–
    December 1, 2002
Toronto, Ontario
	"Social Determinants of Health Across the Life-Span"
	  A current accounting and policy implications
		The Centre for Social Justice
		489 College Street, Suite 303
		Toronto, Ontario M6G 1A5
		Tel.: (416) 927-0777
		Fax: (416) 927-7771
		Toll free: 1-888-803-8881
		E-mail: conference@socialjustice.org
		< www.socialjustice.org/conference>

December 1–4, 2002
Ottawa, Ontario
	"Science & Policy in Action"
	  The Third National Conference on Tobacco or Health
		Taylor & Associates
		18–5370 Canotek Road
		Gloucester, Ontario K1J 9E8
		Tel.: (613) 747-0262
		Fax: (613) 745-1846
		E-mail:
		    stmartin@taylorandassociates.ca
		< www.taylorandassociates.ca>

February 19–21, 2003
St. Louis, Missouri, USA
	"Gateway to Lifelong Health: The Community
	    Connection"
	  17th National Conference on Chronic Disease
	    Prevention and Control
		Department of Health and Human
		    Services
		Centers for Disease Control and
		    Prevention
		Mail Stop K-11
		Atlanta, GA 30341-3717 USA
		< www.cdc.gov/nccdphp/
		    conference>

May 12–16, 2003 **Vancouver, British Columbia**	"Child Health 2003" 3rd World Congress & Exposition	Venue West Conference Services Ltd. Tel.: (604) 681-5226 Fax: (604) 681-2503 E-mail: congress@venuewest.com

September 21–25, 2003
Orlando, Florida, USA
	5th International Symposium on the Role of Soy in
	    Preventing and Treating Chronic Disease
		American Oil Chemists' Society
		PO Box 3489
		Champaign IL 61826-3489 USA
		Tel.: (217) 359-2344
		Fax: (217) 351-8091
		E-mail: meetings@aocs.org
		Information: Mindy M. Cain at:
		    mindyc@aocs.org
		< www.aocs.org/meetings.soy03>

June 13–16, 2004
Milan, Italy
	"Technology, Bridging the Digital Divide – Strategies
	    for Global Heart Health"
		5th International Heart Health
		    Conference
		E-mail: sihh@g8cardio.org
		< www.g8cardio.org>

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Text: Double-spaced, 1 inch (25 mm) margins, 12 point font size.

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