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Commentary

Monitoring Tobacco Use in Canada: The Need for a Surveillance Strategy

Roberta Ferrence and Thomas Stephens

Abstract

Smoking behaviour has been monitored nationally through population surveys for 35 years in Canada, but these surveys have not been as consistent or rigorous as the magnitude of the smoking problem demands. Inconsistent methods and irregular survey intervals are just two of the characteristics that have made it difficult to know exactly how smoking is changing. Further, an absence of routine data on tobacco control policies (other than the price of cigarettes) has hampered understanding of the determinants of changing prevalence. The advent of two survey series—Canadian Tobacco Use Monitoring Survey (CTUMS) and Canadian Community Health Survey (CCHS)—promises to change this situation for the better. We suggest that both are critical elements of a national smoking surveillance system and that, with a commitment to CTUMS in particular, Health Canada could set a new international standard for surveillance.

Key words: *Canada; monitoring; smoking; surveillance*

Introduction

There should be no need to detail the devastation caused by tobacco in Canada. Estimates of health and economic costs totalling $9.56 billion in 1992 dollars[1] are conservative. Even before the 1994 federal and provincial tax cuts on tobacco, smoking-related costs were double the tax revenue produced by tobacco.[2]

Although there is considerable knowledge about strategies that can reduce this damage [3] and many such strategies are in place to varying degrees across Canada, routine surveillance and evaluation of their impact are generally absent. Unlike the United States and the province of Ontario, Canada has never implemented a surveillance system for systematically collecting data on various aspects of smoking behaviour and the determinants of smoking. Although Canada has 35 years of experience in surveying adult smoking at the national level, inconsistent methods and irregular frequency of surveys have provided less useful information than the magnitude of the problem deserves. This inconsistency has led to great difficulty in estimating the impact of policy changes, such as the major tax cut introduced with little advance notice in five Canadian provinces in February 1994,[4] almost three years after the previous national survey of smoking behaviour. The resulting apparent confusion about the effects of government policies, especially taxation, has been exploited routinely by the tobacco industry, most recently in May of this year.[5]

Health Canada's Commitment

Like the health consequences of smoking and their attendant economic costs, the need for national surveillance appears to be well understood and accepted, at least in principle. Almost a decade ago, the first *Directional Paper of the National Program to Reduce Tobacco Use in Canada*[6] identified research and knowledge development as a strategic direction, including "ongoing surveys of tobacco use . . . to aid planning at the regional level." Data gaps, including a critical absence of baseline information needed for many national goals, were again recognized when the National Strategy was updated in 1993.[7] Yet many of these gaps remain today.

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TABLE 1
Repeated national surveys of tobacco use, Canada, 1965–1999

Year(s)	Survey(s)	Data collection	Design (cross-sectional unless specified)	Sample coverage
1965, 1970, 1974, 1975, 1977, 1981, 1983, 1986	Labour Force Survey supplements	Personal interview, considerable proxy data for ages 15–19 until 1980s	Collection in December; multiple persons per household	All persons aged 15+ in 19,000–30,000 households
1978/79 (intended to be ongoing; cut after one year)	Canada Health Survey	Drop-off self-completed questionnaire	July 1978–March 1979; multiple persons per household	21,000 persons aged 15+
1985, 1990 (1996/97)	Health Promotion Survey	Telephone interview in 1985, 1990; personal interview in 1996/97	June 1985; June 1990; part of NPHS in 1996/97; one person per household	1985: 11,000 persons aged 15+; 1990: 14,000 persons aged 15+; 1996/97: ages 12+
1994/95, 1996/97, 1998/99	National Population Health Survey	Personal interview	Longitudinal, with periodic cross-sectional (XS) supplements	1994/95: 17,000 persons aged 12+; 1996/97: large XS sample in Ontario
1994/95 (4 cycles)	SOSIC (Survey of Smoking in Canada)	Telephone interview	Longitudinal, quarterly collection starting spring 1994	16,000 persons aged 15+, fewer over subsequent cycles
1985, 1991, 1995	GSS (General Social Survey)	Telephone interview (personal for ages 65+ in 1985)	September–October 1985; January–December 1990	11,000–12,000 persons aged 15+

Note: For further detail on these and other health surveys, including frequency, topic areas and response rate, see the recent review by Kendall et al. (Reference 10).

Health Canada's report *Tobacco Control: A Blueprint to Protect the Health of Canadians*[8] added a potentially valuable element to the discussion by proposing to expand the requirements for the tobacco industry to report sales, product constituents and other proprietary data. However, although the industry has the longest running consistent time series on tobacco use in Canada, these data are still not available for tobacco control efforts.[a]

Most recently, the National Strategy to Reduce Tobacco Use in Canada has again recognized the need to "monitor knowledge, attitudes and behaviour about smoking and second hand smoke among children, youth and adults on an ongoing basis," and to "monitor and evaluate intervention activities and outcomes on an ongoing basis."[9]

This official recognition of the need for surveillance has been matched by much survey activity at the national level (Table 1), but without a systematic approach.[10] The interval for most data series is well over two years, much too long to be able to detect changes let alone attribute them to policy developments. For example, there were no definitive national surveys in 1992 and 1993, which, as already noted, has resulted in much speculation as to what was happening to smoking rates during this period.

There have been other important single surveys besides those cited in Table 1, notably the 1994 Youth Smoking Survey,[11] which covered ages 10–19. These have produced very useful data, but their infrequency diminishes their value. Moreover, as Table 1 shows, it is relatively rare to have data for the critical years before age 15, a hold-over from the early days when tobacco surveillance was an add-on to a survey of labour force activity.

Since the mid-1990s, there have also been several studies of tobacco control policies, including national surveys of the following.

Retailer compliance (AC Nielson 1995, 1996, 1997, 1998)[12]

Smoking restrictions in municipalities in 1991 and 1995 (Health Canada, 1992, 1995)[13,14]

Smoking restrictions in other public settings (Goss Gilroy, 1995)[15]

Attitudes and knowledge regarding smoking restrictions in homes (EKOS, 1995)[16]

School-based smoking prevention programs (Health Canada, 1994)[17]

[a] Even if such data were available, they would not replace the need for federal government surveillance. Although consistent and regularly collected, industry data do not cover youth and are based on quota sampling, which has several limitations.

TABLE 2
Features of two national surveys of tobacco use and related indicators

Feature	CTUMS[21]	CCHS
TOPICAL COVERAGE		
Tobacco-related	- extensive: behaviours	- basic behaviours
Other topics	- demographics only	- extensive (omnibus health survey)
Determined by	- Health Canada	- Statistics Canada, extensive consultation process
Additional tobacco topics	- highly feasible	- unlikely at the national level
SAMPLE		
Overall size	- 20,000 (10,000 aged 15–24)	- 130,000, and 30,000 alternating years
Age coverage	- 15+ years	- 12+ years
Provincial reliability	- very good overall, good for youth due to oversampling	- excellent for larger years; good in off years overall, fair for youth
TIMELINESS		
Introducing new topics	- approximately 3 months	- approximately 16–18 months
Data available	- 2–3 months after collection	- unknown, but 3–6 months promised by Statistics Canada
Publishing results	- additional 3 months	- additional 3-6 months
Total "turnaround" time	- 8–9 months	- 2–2.5 years +

Although not intended for monitoring tobacco control, an important addition to this list is Statistics Canada's quarterly survey of the price of cigarettes.[18]

All of these studies have provided useful data regarding tobacco control policy. However, apart from the monitoring of prices, only one survey (retailer compliance) has so far produced more than two data points.

Besides the importance of being able to assess policy and whether it is being implemented as intended (e.g. retailer compliance), these studies can offer important insights into the relative contributions of particular measures to tobacco control when linked to data on smoking behaviour from population surveys.[19]

Current Surveillance Tools

More recently, population surveillance appears to have improved markedly. Beginning in 1999, the Canadian Tobacco Use Monitoring Survey (CTUMS) was put in place to monitor tobacco use in Canada.[20] It is only the second adult survey in Canada to be devoted to tobacco use, and its topical coverage is broad: use of cigarettes and alternative forms of tobacco, age of initiation, access to cigarettes, cessation (including reasons and incentives), use of cessation aids, readiness to quit, environmental tobacco smoke (ETS) exposure, restrictions on smoking at home, attitudes toward tobacco control policies, beliefs about "light" cigarettes and awareness of tobacco-industry sponsorship activity. Monthly data collection allows for more precise assessment of specific changes and a larger number of data points for more powerful analyses.

Starting in September 2000, Statistics Canada will inaugurate the Canadian Community Health Survey (CCHS). As part of the core survey content this omnibus health survey will include type of smoker, amount smoked, cessation, age of initiation, use of other tobacco products, workplace restrictions and ETS exposure. The CCHS will be an important addition to the surveillance arsenal, but what it will provide in the way of consistency and robustness will probably be offset by its lack of flexibility and timeliness. This is where CTUMS fits in. When we compare the two surveys on a number of parameters, each shows important strengths for tobacco surveillance (Table 2).

The CCHS is designed to provide basic planning data to 130 local area health units on a wide range of topics. Its strength will be its large sample size (every second year) and geographic coverage. With updated small area data from other sources (e.g. retailer compliance or municipal bylaw coverage), there is very strong potential not only for monitoring but also for assessing the impact of certain tobacco control policies on the fundamentals of tobacco use. However, the reliability of youth data in the smaller sample years will be less than that of CTUMS. Another weakness of the CCHS is timeliness. The gap between introducing a new topic and the time when the results are available will be well over two years, judging by performance to date with the National Population Health Survey and the sheer size of this new survey. In contrast, CTUMS has completed one cycle and has shown that it can produce data on new topics in well under a year. This advantage stems from its more focused

content and in-house management of the survey. Further, there are few limitations on the kinds of tobacco content that can be included.[20]

Components of a National Surveillance System

Given the importance of monitoring not only tobacco use but also tobacco policy "inputs,"[21] we suggest that an effective national surveillance system should have the following components.

An ongoing population survey to provide small area data on basic tobacco use indicators (i.e. CCHS every second year, starting in 2000/01)

An ongoing program of surveys and special studies to provide small area data on tobacco policy inputs, to be linked with the CCHS data, for assessing the impact of such policies as taxation, clean air bylaws and public education

An ongoing population survey to provide reliable provincial data on emerging issues in tobacco control as well as basic tobacco use, with the flexibility to target specified groups such as youth, women of child-bearing age, heavy smokers, etc. (i.e. CTUMS, preferably every year or, at a minimum, every second year after 1999)

The elements of such a surveillance system are known and could be put in place readily. However, there is apparently still no long-term commitment to support CTUMS, nor is there any evident plan for routinely assessing policy inputs. Continued failure to develop this or an equivalent system—with a commitment to consistency over future years—will seriously hamper tobacco control policy and tobacco research in this country. Adoption of such a plan, given the existence of the CCHS and CTUMS as complementary elements, could simultaneously place Canada in a position of international leadership on tobacco surveillance and build a constructive partnership between Health Canada and its provincial counterparts.

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Predictors of Smoking Cessation in an Incentive-based Community Intervention

Namrata Bains, William Pickett, Brian Laundry and Darlene Mecredy

Abstract

The Quit and Win Challenge, an incentive-based intervention, was implemented in two counties in Eastern Ontario to encourage adult smokers to quit smoking. Participants (n = 231) were compared with adult smokers selected at random (n = 385) from a larger, four-county area. Baseline characteristics were assessed by telephone interview, including socio-demographic and smoking-related factors. Follow-up interviews were also conducted by telephone. Initial and follow-up response rates were high (over 84%) in both groups. Compared with the random survey group, Quit and Win participants tended to be younger, more educated, employed and heavier smokers, with fewer friends or co-workers who smoked. After one year, 19.5% of them reported that they were smoke-free, whereas less than 1% of the random group had achieved cessation. This translates into an impact rate of 0.17%, affecting 1 in 588 adult smokers. With the exception of the smokers' baseline "stage of change," none of the socio-demographic or smoking factors was predictive of cessation. We conclude that this intervention achieved only limited success and attracted certain sectors of the community disproportionately, i.e. smokers who were highly motivated to quit. We argue that increased access to proven cessation therapies would improve the impact of such interventions.

Key words: *community; contest; descriptive epidemiologic study; smoking cessation*

Introduction

In Canada, over 41,000 deaths are attributed to tobacco use annually, and cigarette smoking remains the leading cause of preventable illness and premature death.[1] Causal associations have been established between cigarette smoking and many diseases, including respiratory and coronary heart disease, and lung and other cancers.[2–7] Population-based smoking cessation programs have the potential to play an important role in the prevention of these diseases.

Guidelines for the provision of public health programs and services require that boards of health in Ontario ensure the availability of smoking cessation programs in the communities that they serve.[8] In accordance with these guidelines, health units in Eastern Ontario are involved in a variety of cessation activities.[9] In the past, this included the implementation of the incentive-based Quit & Win Challenge within the general adult population. This program was a variation of an intervention developed in Minnesota,[10] in which smokers pledged to quit smoking in exchange for the chance to win prizes. Other tobacco control activities offered by the health units included the distribution of educational materials packaged in a folder called the Quit Kit.

Quit and Win contests and Quit Kits are examples of "minimal intervention strategies" for smoking cessation.[11] They have been developed and implemented because up to 90% of smokers who quit can be expected to do so on their own, rather than through participation in an organized smoking cessation program.[11,12] From the perspective of public health, minimal intervention strategies are quite important because, in general, limited public resources are available for more individualized smoking cessation programs in Canada.[6,9,10]

We had the opportunity to recruit and then follow a sample of adult smokers from Eastern Ontario who had been exposed to these specific smoking cessation interventions. At the same time, a reference population

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of adult smokers was selected by a random telephone survey from the same geographic area. Initial investigation of this cohort involved an analysis of progression through the stages of change[13–15] within these groups of smokers.[16] We then conducted a descriptive epidemiologic study with the following goals.

To describe adults who had been exposed to the smoking cessation intervention, including their baseline socio-demographic characteristics as well as a variety of salient indicators of smoking behaviours

To compare the intervention group of smokers with the sample of adult smokers selected by random telephone survey, according to the above baseline characteristics/indicators

Within the intervention group, to quantify associations between these characteristics/indicators and the achievement of smoking cessation one year after the intervention

Methods

Setting

The study was conducted between 1995 and 1996 within four counties in Eastern Ontario having a combined population of approximately 306,000. About two thirds of the population resided in an urban area, and the first language of 92% of residents was English. The median household income in 1991 was about $44,000, and the overall rate of unemployment was 8.6%.[17]

The Minimal Intervention

The Quit and Win Challenge was an incentive-based program that enrolled adult smokers who pledged to quit smoking for a designated period of time. In exchange, they were entered into a lottery with a cash prize of $1,000 and secondary prizes of lesser values. The initiative was promoted through the local print and radio media, as well as through the distribution of leaflets. Contest entry forms included a description of official rules and were available in local newspapers and at health unit locations.

A contest winner, who was required to be smoke-free in the month leading up to the prize ceremony, was selected by random draw approximately three months after the contest was initiated. As described in the contest rules, the winner was asked to provide the name of a "buddy" to be contacted to verify his or her smoke-free status. Those who enrolled in the contest were also given the educational Quit Kit, which contained a letter of encouragement, information on cessation methods, a list of local cessation programs, helpful tips on maintaining a smoke-free status and a refrigerator magnet with the telephone number of a health unit information line.

Overview of Study Design

The two groups of adult smokers were identified, contacted by telephone and recruited for study. The intervention group consisted of 231 Quit & Win Challenge participants from two of the four Eastern Ontario counties (Frontenac, Lennox & Addington). Smokers selected by random telephone survey (n = 385) came from these regions as well as two neighbouring counties (Hastings, Prince Edward). Baseline telephone interviews were conducted in order to document socio-demographic characteristics and a variety of smoking indicators. Follow-up telephone interviews were conducted after one year in order to re-examine smoking behaviours. All indicators of smoking behaviours were self-reported, and there was no opportunity for biochemical validation of these self-reports.

Descriptive and etiologic analyses were used to describe the intervention group at baseline, quantify associations between baseline characteristics and smoking cessation after one year and compare the intervention group with the smokers selected at random.

Eligibility Criteria/Recruitment

Subjects in the intervention group met each of the following criteria: 1) residents of Eastern Ontario; 2) aged 18 or older; 3) daily smokers, consuming a minimum average of 10 cigarettes per day; and 4) entered the Quit and Win contest in January 1995. Upon entry they filled in a ballot with identifying information. All members of this group were subsequently contacted by telephone and, if they consented, recruited to the study.

Members of the random smoker group had the following characteristics: 1) residents of Eastern Ontario; 2) aged 18 or older; and 3) daily smokers, consuming a minimum average of 10 cigarettes per day. The group was identified by direct telephone contact in January and February 1995 using a random selection process (available from the corresponding author upon request), and those who met the study criteria were asked to participate.

Variables Assessed at Baseline

In both groups, socio-demographic characteristics and smoking indicators were examined at baseline. Variables assessed included known predictors of smoking cessation in adult populations: age and sex,[12,18–21] smoking history (duration, frequency, previous quit attempts),[2,18–23] socio-economic status (education, employment status and occupation),[12,24,25] other smoking variables (the presence or absence of other smokers in the household, whether friends and co-workers smoked)[18–20,24] and intention to quit smoking, as indicated by one of the stages of change.[13–15]

The baseline questionnaire was developed from questions in existing surveys. Questions about smoking history and current smoking patterns were based on the Ontario Health Survey[26] and a current review paper.[27] Socio-demographic variables were based on questions suggested by Dillman.[28] Occupations were coded using the Statistics Canada Census coding manual[29] and were classified according to the Pineo-Porter classification of occupational status.[30] Intention to quit smoking was

measured using the transtheoretical (Stages of Change) model developed by Prochaska and DiClemente.[15]

Follow-up

Follow-up telephone interviews after one year were used to assess continuous abstinence from cigarette smoking in the six months prior to interview. This very rigid outcome was chosen rather than a point prevalence measure because the program organizers had stated, a priori, that the intervention was developed to help daily smokers to quit smoking completely.

Data Collection

The telephone surveys were designed using the principles outlined by Dillman.[28] The baseline and follow-up surveys were pilot tested with a convenience sample of peers and revised on the basis of their feedback. Four interviewers collected data from each of the two groups and at follow-up. All variables were pre-coded. Responses were entered into a computerized database manager and then checked for accuracy.

Statistical Analysis

Response rates (number of completed interviews/best estimate of smokers eligible for interview) were calculated for each group at baseline and at follow-up. Descriptive statistics (frequencies, chi-squared tests, *t*-tests) were used to describe each group and to compare the two groups at baseline by age and sex, socio-economic indicators, prior smoking history and other smoking variables. Logistic regression analyses were conducted to estimate the strength and statistical significance of associations between baseline factors and smoking cessation after one year. Bivariate odds ratios and associated 95% confidence intervals were produced. Multiple logistic regression was then used to refine the odds ratio estimates while simultaneously adjusting for the influence of other variables. Etiologic analyses were limited to members of the intervention (Quit and Win) group. Statistical analyses were conducted using SAS[31] and EGRET.[32]

Results

Response

Response rates at baseline were high in both the intervention (97.6%, $n = 231$) and random survey (92.8%, $n = 385$) groups of adult smokers. Of those recruited at baseline, 86.5% ($n = 200$) of the intervention group were re-contacted successfully after one year using a follow-up telephone call, and 84.4% ($n = 325$) of the random survey group were re-contacted.

Baseline Data

Members of the intervention group were predominantly female and, compared with the random survey group, were younger, more highly educated, more likely to be employed and more likely to be working as a semi-professional or professional (Table 1). In the intervention group, 73% began smoking as teenagers, and 77% had smoked for more than 10 years; they smoked more often but had been smoking for fewer years than subjects in the comparison group. During the previous year, 42% of the intervention group had made at least one quit attempt, one third (35%) lived in a household with at least one other smoker and 57% reported that at least half of their friends also smoked; however, they had fewer friends or co-workers who smoked than did the random survey group and more often worked in a smoke-free environment.

In order to be eligible to win the Quit and Win contest, respondents had to be smoke-free in the month before its conclusion (March 1995). As a result, a very high proportion (87%) were actively trying to quit at the time of the baseline interview. Thus they were more likely to be in the action or preparation stages of change.

Factors Associated with Cessation (Intervention Group)

Bivariate analyses were carried out to examine baseline variables and their association with smoking cessation after one year. These analyses were limited to members of the intervention group, since only 1% of the random survey group ($n = 4$) had achieved cessation after one year. One in five (19.5%) of the 200 smokers who were re-contacted after one year had quit smoking. However, there was no evidence of strong or statistically significant associations between socio-demographic factors and cessation, nor was there evidence of strong associations between baseline smoking indicators and cessation. The one exception to this was "motivation to quit," as measured by stage of change. Those in the action stage at baseline were six times more likely to have quit than those in all other stages combined, although this finding was of borderline significance. Multiple logistic regression analyses confirmed these basic findings (Table 2) and, as a result, no models were produced that included more than one explanatory variable.

Discussion

When compared with the random sample of adult smokers, the composition of the group of smokers enrolled in the Quit and Win intervention was different with respect to several salient characteristics. Consistent with the findings of Cummings et al.,[33] contestants were on average younger, heavier smokers, better educated and more likely to be employed than non-participants. There was also a significant difference between the two groups with respect to a smoke-free workplace, although this difference is largely due to the inclusion of the "not applicable" responses as a category. This category included subjects who either had no workplace (i.e. were retired, homemakers or unemployed) or had an atypical workplace (e.g. were bus drivers). When the analysis was conducted only for subjects who were employed or were students (66% of subjects), no difference was found between the two groups with respect to this variable.

Curiously enough, a small number of subjects in the intervention group were not in the action stage of the

TABLE 1
Baseline characteristics of groups of smokers with and without an incentive-based intervention

Characteristic		Intervention group (%)	Random survey group (%)	*p* value
Sex:	Female	59.3	54.0	0.2
	Male	40.7	46.0	
Age (years):	18–29	26.4	16.1	0.001
	30–39	35.5	25.5	
	40–49	23.4	22.1	
	50–59	10.8	16.6	
	60+	3.9	20.3	
Education:	Less than high school	1.8	11.2	0.001
	Some high school	13.2	22.9	
	Completed high school	29.8	34.0	
	Some college/university	16.7	14.0	
	Completed college	38.6	18.2	
Employment status:	Employed	74.9	53.0	0.001
	Homemaker	5.6	12.0	
	Retired	3.5	18.4	
	Unemployed	7.8	12.2	
	Other	8.2	4.4	
Occupation level:	Unskilled worker	17.3	28.1	0.001
	Semi-skilled worker	16.0	21.6	
	Skilled worker/supervisor	10.4	16.6	
	Semi-professional/professional	17.3	12.7	
	Occupation not known	39.0	21.0	
Cigarettes (daily number):	*mean (standard deviation)*	*23.4 (9.2)*	*20.8 (8.2)*	*<0.001*[a]
	10–24	38.5	54.6	0.001
	25–50	57.1	43.1	
	>50	4.3	2.3	
Age of starting smoking:	*mean (SD)*	*17.4 (5.2)*	*16.9 (4.6)*	*0.21*
	<15	24.7	26.5	0.48
	15–18	48.5	50.9	
	19–25	19.1	17.7	
	>25	7.8	4.9	
Years of smoking:	*mean (SD)*	*19.9 (11.1)*	*20.0 (14.7)*	*<0.001*[a]
	< 5	7.8	6.5	0.001
	5–10	15.2	7.5	
	11–20	38.5	20.3	
	21–30	23.8	24.7	
	>30	14.7	41.0	
Quit attempts in past year:	None	58.4	68.6	0.04
	1	25.1	19.0	
	2+	16.5	12.5	
Other smokers in household:	None	64.9	67.5	0.059
	1	25.5	27.8	
	2 +	9.5	4.7	
Friends who smoke:	Less than half	42.6	33.3	0.02
	Half or more	57.4	66.8	
Smoke-free workplace:	Yes	53.9	34.8	0.001
	No	29.1	20.8	
	Not applicable	17.0	44.4	
Co-workers who smoke:	Less than half	50.7	27.3	0.001
	Half or more	32.5	27.0	
	Not applicable	16.9	45.7	
Stage of change:	Action	86.8	2.3	0.001
	Preparation	7.1	6.2	
	Contemplation	5.7	41.6	
	Precontemplation	0.5	49.9	

[a] These *p* values are associated with Student's t-test. All others are based on chi-squared tests.

TABLE 2
Bivariate analysis of baseline factors and their association with smoking cessation after one year, Quit and Win intervention group

Baseline factor		Achieved cessation (*n* = 39)	Did not achieve cessation (*n* = 161)	Odds ratio (95% confidence interval)
Sex:	Male	15	60	1.0
	Female	24	101	0.95 (0.4–2.1)
Age (years):	18–29	12	39	1.0
	30–39	16	55	1.0 (0.4–2.5)
	40–49	8	41	0.6 (0.2–1.9)
	50+	3	26	0.4 (0.1–1.6)
Education:	Some high school or less	5	23	1.0
	Completed high school	8	50	0.7 (0.2–3.0)
	Some college/university	11	21	2.4 (0.6–9.7)
	Completed college	14	66	1.0 (0.3–3.5)
Employment status:	Employed	30	122	1.0
	Student	4	11	1.5 (0.3–5.4)
	Other (homemaker, retired)	3	16	0.8 (0.1–2.9)
	Unemployed	2	12	0.7 (0.1–3.3)
Occupation level:	Semi-professional/professional	11	38	1.0
	Skilled worker/supervisor	4	22	0.6 (0.2–2.5)
	Semi-skilled worker	4	35	0.4 (0.1–1.5)
	Unskilled worker	12	23	1.8 (0.6–5.3)
	Occupation not given	8	43	0.6 (0.2–2.0)
Cigarettes (daily number):	10–24	14	67	1.0
	25+	25	94	1.3 (0.6–2.8)
Age of starting smoking:	<15	14	36	1.0
	15–18	14	80	0.5 (0.2–1.1)
	19–25	7	33	0.6 (0.2–1.7)
	>25	4	12	0.9 (0.2–3.6)
Years of smoking:	<5	4	9	1.0
	5–10	9	22	0.9 (0.2–4.7)
	11–20	16	63	0.6 (0.1–2.5)
	21–30	7	41	0.4 (0.1–2.0)
	>30	3	26	0.3 (0.4–1.8)
Quit attempts in past year:	None	26	91	1.0
	1	6	47	0.5 (0.2–1.3)
	2+	7	23	1.1 (0.4–3.0)
Other smokers in household:	None	28	107	1.0
	1	9	40	0.9 (0.3–2.1)
	2+	2	14	0.6 (0.1–2.8)
Friends who smoke:	Less than half	16	71	1.0
	Half or more	23	89	1.2 (0.5–2.5)
Smoke-free workplace:	No	15	44	1.0
	Yes	18	89	0.6 (0.3–1.4)
	Not applicable	5	28	0.5 (0.2–1.8)
Co-workers who smoke:	Less than half	18	85	1.0
	Half or more	14	48	1.4 (0.6–3.2)
	Not applicable	5	28	0.8 (0.3–2.7)
Stage of change:	Action	37	138	6.1 (0.9–261.0)
	All other stages	1	23	1.0

Stages of Change model. Baseline interviews with subjects were conducted after the contest deadline. Thus it is possible that some subjects entered the contest with the intention of quitting, but were somehow discouraged during the short time period between contest entry and the baseline interview.

One interpretation of the findings is that systematic differences exist between smokers who choose to participate in these interventions and smokers who do not. For example, our study showed that the intervention did not reach older and retired people or entice them to enter, nor did it well represent smokers with lower socio-economic status. This may indicate different levels of motivation to quit smoking, or it may reflect the methods employed to advertise the contest and the varying exposures that different social groups had to the advertising. The contest was advertised through direct contact with

workplaces, in newspapers and on specific radio stations. People employed outside the home and those in certain occupations may have had more exposure to these messages and greater opportunity to sign up for the contest because of their physical proximity to health unit locations.

One-year follow-up data available from the intervention group of smokers showed that, even in this highly motivated group, only about 20% were abstinent; moreover, this is likely an overestimate. The smoking status of individuals was based on self-reported information obtained during a telephone call, and biochemical validation of these reports was not feasible. Further, participants were aware that they were providing information to a research study being conducted under the auspices of a public health unit. We expect that this may have led to some false reports of smoking cessation because of the need of some people to provide a socially acceptable response, particularly as they had declared their intention to quit smoking to a health agency in a very public format.

Evaluations of contests similar to this particular Quit and Win program, which have also used sustained cessation as an end point, have reported quit rates ranging from 13% to 37%, with a mean value of 23% for community settings.[34] The 20% rate of cessation achieved in Eastern Ontario is consistent with these results. The best available estimate of adult smokers in the four counties who were eligible for contest entry is 28,900,[26] and 239 of these people entered the Quit and Win program. This participation rate of 0.83% is slightly lower than those reported for contests held in other communities.[34] When the participation rate is combined with the cessation rate into a single measure of impact,[34,35] the program was successful for 0.17% of the smoking population. Expressed in another manner, if we assume a causal relation between entry into the contest and sustained cessation, we can extrapolate that 1 in every 588 smokers in the community was led to quit because of the contest. We consider this rate of impact to be quite low in practice. This type of information is useful as a benchmark that can assist in setting priorities for future community-based tobacco control initiatives.

One of the most striking observations in the present study was that strong and statistically significant associations were not identified between baseline variables and one-year smoking cessation within the Quit and Win intervention group. This was true of a number of categories of potential predictors, including socio-demographic factors (age group, sex, education, employment), smoking history (duration, consumption) and the presence of smokers in various social environments (household, friends, co-workers). Arguably, the one exception to this trend was that a person's motivation to quit, as indicated by categorization according to the Stages of Change model,[13–15] showed a strong (albeit not statistically significant) association with one-year cessation.

Although these findings are corroborated by some existing literature,[23,33,36,37] they are not consistent with other investigations. For example, in a number of studies[18,20,21,38] those in higher age groups were more likely to quit successfully. Other demographic factors, such as sex (men being more likely to quit) and marital status (married or common-law partnerships as a positive factor) have also emerged as possible predictors.[25,38] Cigarette consumption and number of previous quit attempts are also associated statistically with cessation. Subjects who made fewer previous quit attempts were more likely to quit in some investigations.[20,21,38] As well, success rates have been better among heavy consumers of cigarettes than among moderate ones,[20,21] although converse results[39] have been reported. The presence of social support from friends, family or co-workers also appears to contribute to cessation.[20,21]

The results of this study have implications for prevention. First, public health and other community-based agencies must recognize that Quit and Win interventions can achieve only limited success in reaching and affecting certain sectors of society. This is of particular concern because the groups with lower rates of participation (older people, those of lower socio-economic status) are especially vulnerable to chronic disease and mortality, in general, and smoking-related illness, in particular.[40–42] Quit and Win interventions represent one of many different options for communities embarking upon smoking cessation campaigns. Unless special efforts are made to target the particularly vulnerable sub-sectors of the adult smoking population in these contests, the latter are unlikely to respond to such efforts. Alternatively, such groups may be better served by other population-based approaches, such as price increases (which are beyond local control in Ontario) or promotion of more individualized support combined with subsidized antidepressant (bupropion) treatments[43] and/or nicotine replacement therapies.[44]

Our data suggest that once smokers have enrolled in a Quit and Win intervention, no other factors appear to influence the success of the cessation effort, but the person's underlying motivation to quit may be an important factor. This supports the need to provide general rather than targeted advice and support to these people in their efforts. The 20% success rate achieved in our study is quite typical and demonstrates the difficulty that even highly motivated smokers face. The consistency of cessation rates across different socio-demographic strata reinforces the idea that social and behavioural support offered in home and work situations may play some role in cessation, but is often insufficient, alone, to achieve cessation. Despite this fact, it is also important to recognize that Quit and Win participants achieved cessation rates that were as much as 20 times higher than those observed in general populations of smokers. It is highly probable that this could be further increased if proven therapies and professional support were made available at little or no cost to these highly motivated individuals. Without these, one can expect to have only a marginal influence on general smoking rates using incentive-based initiatives alone.

The limitations of this descriptive study must be emphasized. Our study population consisted of selected adult smokers in a four-county area of Eastern Ontario. The findings may not be generalizable to larger communities or those with a different demographic structure. Further, they may not represent the potential for Quit and Win interventions that are conducted on a larger scale or in settings where cigarette prices are more prohibitive or cessation therapies more readily available to the average consumer. Our sample of random smokers may have been biased toward subpopulations that are more likely to be at home, and the disparities between them and the intervention group may have been exaggerated because of this. Finally, our study did not assess rigorously the role of social support and related factors, which are more difficult to quantify than the basic measures that were assessed. We therefore were unable to account for these factors as predictors of cessation, and this is an acknowledged limitation of our study.

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School-based Smoking Prevention: Economic Costs Versus Benefits

Thomas Stephens, Murray J Kaiserman, Douglas J McCall and Carol Sutherland-Brown

Abstract

The objective of this study was to conduct a cost–benefit analysis to compare the costs of developing and delivering an effective school-based smoking prevention program with the savings to be expected from reducing the prevalence of smoking in the Canadian population over time. A smoking prevention program that meets published criteria for effectiveness, implemented nationally in Canada, would cost $67 per student (1996 dollars). Assuming such a program would reduce smoking by 6% initially and 4% indefinitely, lifetime savings on health care would be $3,400 per person and on productivity, almost $14,000. The benefit–cost ratio would be 15.4 and the net savings $619 million annually. Sensitivity analyses reveal that considerable economic benefits could accrue from an effective smoking prevention program under a wide range of conditions.

Key words: *Canada; economic analysis; prevention; school; smoking*

Introduction

Background

The prevalence of smoking has declined impressively since the 1960s, but there are still more than six million Canadians who smoke.[1] Moreover, teen smoking in Canada increased after 1990[2] and has not declined since in concert with general population trends.[1] Among current smokers aged 15–17, 35% had had their first cigarette by the age of 12, and almost 80% had tried smoking by the age of 14.[3]

Unfortunately, effective smoking prevention has been the exception rather than the rule in Canadian schools.[4] Recent cutbacks in education budgets have jeopardized many school programs, including smoking prevention, while at the same time reduced health budgets have made it more imperative than ever to identify causes, especially preventable causes, of excess health care costs. It is clear that smoking is one of these causes, and it would therefore be useful to know how much money, if any, could be saved by effective smoking prevention programs in schools.

Study Objective

The objective of this study was to conduct a cost–benefit analysis of school-based smoking prevention programs in order to examine the potential payoff of effective programs. Although the ultimate rationale for preventing smoking is not economic, but human, an economic analysis may lend weight to arguments in favour of prevention and thus lead to improved health and enhanced quality of life.

Methods

General Approach

The general strategy adopted was to calculate a benefit–cost ratio for smoking prevention programs. This form of cost–benefit analysis takes a societal perspective and quantifies the potential effects on all parties involved.[5] Since the ratio expresses both costs and benefits in the same (dollar) terms, the following items need to be documented.

The costs of developing and delivering a prevention program

The effect of the program in terms of reduced prevalence

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The direct and indirect benefits of fewer smokers in the population

The approach here is similar to the one used in a recent study from the US Centers for Disease Control and Prevention (CDC)/Battelle Institute.[6] For cases in which variables such as costs and effects might be open to debate, we state our assumptions and use conservative values for the "base case" in order to produce a ratio that is defensible. Sensitivity analyses show the range of benefit–cost ratios that result from assumptions different from those in the base case. All dollar figures are expressed in 1996 terms, but, since the result of interest is a ratio of benefits to costs, the year is ultimately of little relevance.

Cost of Smoking Prevention

The components of program cost, expressed on a per-person basis, are these.

Program development, including evaluation, revision and dissemination to schools

Program delivery in schools, particularly teacher time and associated expenses

Program development costs

To obtain the cost of program development, we looked for prevention programs designed for teacher use (as distinct from volunteer- or peer-led programs) that would meet the criteria for effectiveness described by Glynn.[7] We selected the combination of two Canadian programs, *Peer Assisted Learning (PAL)*[8] and *Improving the Odds.*[9] Federal health department files were consulted to document the costs of staff salaries and consultants' fees for initial program development and later evaluation.[10] The total in round numbers was $1 million. To calculate this on a per-student basis, we assumed a three-year lifespan and nationwide implementation. An estimated cohort of 1,167,000 children across Canada (based on census estimates of three cohorts of 12-year-olds) would thus be exposed to the program before it became obsolete, for a *per-student development cost* of $0.86.

Program delivery costs

A national survey of smoking prevention programs[4] revealed that only a minority of Canadian schoolchildren are exposed to programs meeting the criteria for effectiveness.[7] Among other shortcomings, existing programs tend to have too few sessions.

Glynn describes a total of 10 sessions over four years (grades 6–9) as a minimum.[7] To cost such a program, we assumed that the 10 sessions were equally spread over the four years; elementary school sessions (grades 6–8) were 30 minutes long, and secondary school sessions (grade 9) were 45 minutes. For a program that clearly exceeds this minimum, we used a Nova Scotia curriculum with a total of 18 sessions in grades 4 through 7.[11] With regard to classroom activities, the teacher time per smoking prevention course was thus 5.6 hours for the minimum 10 sessions and 9.0 hours for the exemplary program.

Some time for initial teacher training must also be considered. We assumed that the teachers would be trained with videos or print materials, so the main cost would be their time. Glynn[7] suggested half a day as a minimum and one full day as preferable. We thus added 3.5 hours and 7.0 hours to the above times for classroom contact, for the following totals.

Minimum teacher time: 9.1 hours (half-day training + 10 classroom sessions)

Preferred teacher time: 16.0 hours (full-day training + 18 classroom sessions)

In 1995/96, the total cost of elementary and secondary education was $7.29 *per pupil per hour* of instruction.[12] This is a comprehensive figure that includes operating costs outside the classroom, debt service and capital expenditures for schools. Using the values of 9.1 hours and 16.0 hours, *per-pupil costs for program delivery* are thus $66.34 for the minimum 10 sessions and $116.64 for the preferred 18 sessions.

Total program costs, including development and evaluation, teacher training and classroom delivery, thus work out as follows.

Minimum exposure: $67.20

Preferred exposure: $117.50

For the base case in our cost–benefit analysis, we used a per-student cost of $67.00.

Annual costs are required to provide an estimate of net savings. For this purpose, we amortized the program development costs over three years and added to this the annual program delivery costs. The latter are based on the per-pupil hourly cost of $7.29 for 1,167,000 pupils, each of whom is exposed annually to 2.275 hours of instruction, including teacher training time (a total of 9.1 hours over four years), for the base case. Total annual costs for a national smoking prevention program in Canada would thus be $19.7 million.

Program Effect

The calculation of a benefit–cost ratio requires a value for the difference in prevalence of smoking in groups receiving prevention programs and those receiving none. The use of such an effect size makes it unnecessary to specify the actual prevalence of smoking prior to the introduction of the prevention program, and thus allows for the application of the results to any population. The CDC/Battelle study[6] adopted as its base case an initial effect size of 6%, based on an extensive review of the literature, decaying by 20% over four years, to an enduring 4.8%. We have assumed for our base case a decay of 33% to an enduring 4% after four years.

Potential Savings

The economic benefits of a population with fewer smokers are in the form of direct and indirect savings. Direct savings arise from the reduced need to provide health care to smokers; the indirect savings come from

the greater productivity of non-smokers by virtue of their reduced number of sick days and longer working life. In the present study, values for these savings were obtained by a cost-of-illness approach.

Direct benefits (reduced health care costs)

Previously calculated smoking-attributable costs for physician care, hospital use and medications, by sex, for 1991[13] were adjusted for inflation to 1996 dollars. Annual costs per smoker were obtained by dividing the total cost by the number of "ever smokers" (current and former smokers). *Lifetime per-smoker* costs were estimated using present-value calculations of the annual costs at four discount (inflation) rates over life expectancies of 78 years for men and 82 years for women.[14]

Indirect benefits (less absenteeism)

In a similar fashion, smoking-attributable absenteeism costs, by sex, were calculated using present-value analysis. Costs were calculated for a working career with retirement at age 65 for both sexes. Annual per-smoker absenteeism costs attributable to smoking were determined by dividing the total annual cost of such work loss by the number of ever smokers in the work force. Data by sex were available for both 1991[13] and 1994.[15] Since all of the other data were for 1991, absentee data for that year were used in this analysis.

Indirect benefits (less premature death)

Income lost due to premature death attributable to smoking was also calculated using present-value analysis. We assumed that no smoking-attributable deaths occur before age 45 and that individuals normally retire at age 65. As a result, the indirect smoking-attributable cost of premature death reflected total lost income from age 45 to 65. Indirect costs per year were calculated from the average industrial wage adjusted to 1996 dollars.

Sensitivity analyses

Several of the parameters that go into calculating the benefit–cost ratio require assumptions, as already described, and sensitivity analysis was conducted to test how the benefit–cost ratio changed as these assumptions changed. The following parameters were tested through sensitivity analyses: inflation rates of 3%, 5% and 8% in addition to the base case of 4%; program effect sizes of 4% declining to 2% and 6% declining to 1% in addition to the base case of 6% declining to 4%; and higher program development costs of $117 per student for the preferred 18 sessions as well as the base case of $67 per student for 10 sessions.

Lost income is usually treated as a cost that must be borne indefinitely by the family of the prematurely dead worker. Some argue that this is not a realistic cost and that the true cost to society is limited to the approximately three months that it takes to replace a deceased worker. We have tested the result of using this reduced "friction cost" method in the sensitivity analyses.

Results

Table 1 summarizes the annual costs in 1996 dollars of smoking-attributable disease and early death for all smokers (first double column). The resulting health care bill (direct costs) totalled $2.4 billion, most of which was due to the excess hospital care required by smokers. Another $13.6 billion (indirect costs) was due to lost productivity through sick days and early death, the latter being by far the more important factor. Deaths before age 65 due to smoking accounted for about 88% of the indirect costs for men and 66% of these costs for women. The total cost of smoking was $16 billion annually for both sexes.

Over a lifetime, the cost to the Canadian economy was almost $20,000 for every man and almost $15,000 for every woman who had ever been a smoker (Table 1, second double column). On average for both sexes, each adult who had ever smoked created health expenditures of $3,400 and indirect costs of almost $14,000. The total annual costs were substantial (first double column)

TABLE 1
Costs attributable to smoking-related disease (1996 dollars), Canada

	1. Annual cost, all smokers ($000,000)		2. Lifetime cost per smoker ($)		3. Potential annual savings ($000,000)	
Type of cost	Males	Females	Males	Females	Males	Females
DIRECT	1,570	843	4,161	2,629	63	34
- MD use	59	48	161	147	2	2
- hospital visits	1,495	775	3,952	2,416	60	31
- medications	16	20	48	65	1	1
INDIRECT	11,194	2,368	15,548	12,009	448	95
- sick days	1,345	868	5,177	5,110	54	35
- early death	9,849	1,500	10,371	6,899	394	60
TOTAL	12,764	3,211	19,710	14,638	511	128

Note: Columns may not add due to rounding.

because of the number of current and former smokers—7.4 million men and 6.4 million women in 1996/97.[2]

Table 1 also shows the savings that could arise from preventing smoking with a national school-based smoking prevention program of modest success—our base case of 6% declining to 4% (third double column). For men and women respectively, reduced smoking would result in savings of over half a billion and over one quarter of a billion dollars annually. The total potential savings could amount to $639 million annually, of which almost $100 million would be saved on health care alone. The *net savings* (net present value) of smoking prevention after paying for program delivery would be sizable — $619 million for the base case.

The benefit–cost ratio of smoking prevention was obtained by dividing the per-person savings (Table 1) by the per-student cost of $67 and adjusting for the effect of the program. The resulting ratio of benefits to costs was 17.7 for males and 13.1 for females for the base case (Table 2). In other words, a school-based program of smoking prevention of modest success could produce an overall return of $15.40 for every $1.00 spent.

This overall benefit–cost ratio of 15.4 is reduced when inflation is higher, the program effect is reduced or the costs of the prevention program are increased (Table 2). However, in each of these cases, the benefit–cost ratio is still well above 1.0, that is, there is economic benefit for all of these scenarios.

Indeed, the returns on prevention are positive even when *only health costs* are considered as benefits or when lost productivity due to early death is assumed to last only three months. Even in the extreme case of a relatively expensive program of 18 sessions that achieves only a minimal effect of a 1% decline in smoking, $2.00 would be returned for every dollar invested in prevention.

Discussion

These results show that, under a wide range of conditions and assumptions, smoking prevention programs in schools can produce a substantial economic benefit. Indeed, the returns described here are conservative, since they omit several costs that are difficult to estimate: disease-related costs from environmental tobacco smoke (ETS), property damage costs from ETS, the cost of creating separately ventilated public smoking areas, increased life insurance costs for smokers, the cost of deaths before age 45 and work lost during smoking breaks away from the workplace. Some of these costs have been estimated in a study of the working population.[15] In 1995 dollars, the annual cost per smoking employee was estimated at $75 for life insurance, $85 for a smoking area and $2,175 for decreased productivity due to cigarette breaks. These costs were not included in the present study, as the methods for estimating them are not yet widely accepted, but they do serve to indicate that the present estimates of the economic toll of smoking are conservative.

Moreover, the method we used to estimate the direct and indirect savings is also conservative. For smokers of all ages we used the *average* costs of their health care, work absence and early death, and applied these averages to the appropriate number of smoker-years. When we calculate these costs on an *age-specific* basis, as some would prefer, the total (i.e. the potential saving) is substantially higher—about 60% higher for women and 80% higher for men. This is because the income foregone by early death occurs during workers' peak earning years. We used the more conservative averaging approach because the alternative puts too much emphasis on the indirect costs.

Further, the values used for program *costs* in this study are very inclusive, covering all operating costs inside and outside the classroom, debt service and even capital costs. The per-student cost of $67 used in our base case is thus considerablly higher than the US value of $48.[6] As illustrated by the sensitivity analyses, the benefit–cost ratio is very sensitive to the cost value used.

The benefits of prevention are understated in this study for another reason: the cost-of-illness approach

TABLE 2
Benefit–cost ratios for smoking prevention under various assumptions

Assumptions	Males	Females	TOTAL
Base case: 6% program effect declining to 4% after 4 years, 4% inflation, $67/student cost	17.7	13.1	15.4
Same as base case, but $117/student cost	10.1	7.5	8.8
Same as base case, but 3% inflation	23.4	17.6	20.5
Same as base case, but 5% inflation	13.4	10.0	11.7
Same as base case, but 8% inflation	6.8	5.3	6.0
Same as base case, but program effect declines to 1%	9.0	6.7	7.8
Same as base case, but effect declines from 4% to 2%	4.1	3.1	3.6
Same as base case, but lost income is limited to 3 months	8.5	7.0	7.7
Same as base case, but health (direct) costs only	3.5	2.3	2.9
Worst case: 18 sessions to achieve program effect of 1%	2.3	1.8	2.0

emphasizes the individual's productive potential and omits any consideration of pain, suffering or reduced quality of life. It thus produces a *lower-bound* estimate of the benefits of prevention.[5] Despite these difficulties, the cost-of-illness approach is the most satisfactory for this type of analysis at the present time, because reliable data are available for estimating the costs averted.

Some economists might object that our calculations do not account for *savings* on pensions due to early death, but such "savings" are illusory, since pensions are typically paid to survivors even if the worker dies early. Nor have we taken account of the economic contribution of cigarette manufacturing and retailing in the form of excise and income taxes. Other analyses, however, show that the societal costs of smoking far outweigh these public revenues.[16,17]

Tables 1 and 2 reveal substantial male/female differences in both direct and indirect costs attributable to smoking. This is due to men's longer length of hospital stay (since they tend to be sicker than women smokers), the greater labour force participation of men aged 45 and older, the higher earnings of these men and their tendency to die of smoking-related causes younger than women do. This sex difference is likely to change with time as male and female rates for smoking prevalence and incomes converge. Present indications are that convergence in costs will arise as much from increased smoking-related disease among women as from decreased disease among men.[2]

These tables also show that indirect costs due to lost productivity are far higher than even the considerable direct costs due to health care. Lost productivity arising from worker illness and early death is a genuine loss to the economy, and it needs to be included to give a full picture of the cost of smoking. The cost-of-illness approach in this study is similar to that adopted by other researchers as diverse as the Conference Board of Canada,[15] the US Centers for Disease Control and Prevention[6] and the Canadian Centre on Substance Abuse,[16] with similar results. Although cost–benefit analysis takes a societal perspective and attempts to include most relevant outcomes,[5] the calculation of the indirect costs is sometimes controversial. For this reason, we tested the effect on the benefit–cost ratio of limiting the cost of early death to three months rather than the balance of the smoker's expected work life. The result is still a substantial $7.70 returned for every $1 spent on prevention.

There are few published studies comparable to the present one. The CDC/Battelle Institute analysis[6] that provided the model for the present study is the most similar, but its results were based on parameters slightly different from ours. Using an inflation rate of 5% and a long-term program effect of 4.8%, they calculated a benefit–cost ratio of 18.5. Our result of 15.4 compares reasonably well, given the sensitivity of the ratio to inflation and the fact that the US program delivery costs were less inclusive than those in our study while their health care system is more expensive. The returns for prevention programs in both these analyses are rather larger than those reported for a cessation program for pregnant women, which ranged from $3.31 to more than $6.00 for each dollar invested.[18]

It is instructive to note that smoking prevention programs also provide far higher economic returns than either drug education or sex education, according to the CDC/Battelle Institute study.[6] This is consistent with a cost-effectiveness analysis whose conclusion was that smoking prevention would save lives more efficiently than most other lifestyle interventions, such as weight loss or cholesterol lowering.[19] Only immunization for measles, mumps and rubella appears to have a benefit–cost ratio (14.0) comparable with that of smoking prevention.[20]

The results of the sensitivity analyses in Table 2 show that positive economic returns from smoking prevention could be expected across a wide range of conditions. Even when a more extensive (and expensive) program achieves only a modest effect of 1%, the future savings are substantial. But the fact remains that effective programs do not currently exist in Canada on a sufficiently widespread basis that even such modest returns could reasonably be expected. Thus, the savings indicated here must be regarded as only *potential* benefits until such time as effective smoking prevention programs are implemented nationally. Effective programs are definitely achievable.

School-based intervention appears to work best when accompanied by coordinated community action.[21] Among other measures, this should include a complementary mass media campaign,[22] smoke-free schools,[23] accessible smoking cessation services[24] and peer-led extracurricular programs[25] together with widespread smoke-free bylaws and high prices for cigarettes.[26] Such multi-faceted approaches will cost more but may well have a greater effect than the modest 4% in our base case. At the same time, better efficiency may result if prevention is focused on high-risk schools.[27] Current programs expose all students regardless of their level of risk, and they are not very effective programs.[4]

In conclusion, this study reveals that very substantial economic returns could be expected from an effective program of smoking prevention in Canadian schools. Other benefits such as enhanced quality of life have not been considered here, but they are at least as important and the gain in this regard would likely also be impressive. However, such prevention programs are not currently implemented widely enough to expect such benefits. This analysis provides an argument in favour of more widespread implementation.

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Performance of the Composite International Diagnostic Interview Short Form for Major Depression in a Community Sample

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Abstract

Recently, short-form versions of structured psychiatric diagnostic interviews have been developed for epidemiologic and survey research. These short forms can reduce research costs in large-scale studies; however, their accuracy is likely to be less than that of a full diagnostic interview. We evaluated the positive and negative predictive values of a short-form interview derived from the Composite International Diagnostic Interview (CIDI). Samples of subjects who scored both positively (n = 277) and negatively (n = 136) on the CIDI Short Form for Major Depression (CIDI-SFMD) were administered the full depressive disorders section of the CIDI. Almost all subjects who were negative on the short form were similarly classified as not having major depression by the CIDI. Approximately 25% of subjects had false positive results; these subjects tended to be older and less educated than true positives. Approximately 75% of subjects scoring five or more on the CIDI-SFMD had major depression according to the full CIDI, and a proportion of the remainder had less severe depressive syndromes. Some CIDI-SFMD positive subjects may have had depressive symptoms attributable to organic or other etiologies excluded under the definition of major depression.

Key words: *depressive disorders; measurement instruments; mental disorders; statistical and numerical data*

Introduction

The Composite International Diagnostic Interview (CIDI) is a fully structured diagnostic interview designed for administration by non-clinicians. The latest version of this instrument (version 2.1) can generate psychiatric diagnoses according to the definitions in the fourth edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) or the tenth revision of the World Health Organization's *International Classification of Diseases* (ICD-10). Recently, a variety of short forms for the CIDI have been developed. One of these, the CIDI Short Form for Major Depression (CIDI-SFMD), has been used in survey research as an indicator of major depression.[1,2] In this study, the performance of the CIDI-SFMD in a community sample was evaluated in relation to the results of a subsequent application of the depressive disorders section of the full CIDI.

A second objective of the study was to describe the characteristics of subjects with discordant results on the CIDI-SFMD and the full CIDI. One characteristic of potential importance is education, since more highly educated people might provide more accurate descriptions of their depressive symptoms. Other potentially relevant characteristics are those related to physical illness, alcohol and drug exposures. The full CIDI includes attribution-oriented questions, which discount symptoms as contributing to a diagnosis of major depression if they are reported to be due to physical illness, alcohol or drug exposure. The short form does not do this. Therefore, more false positive results might be expected in subjects who had medical conditions or had consumed alcoholic beverages or drugs.

Methods

Data collection took place between February 1, 1998, and July 1, 1999. The sample was selected by random digit dialling. The target population consisted of adults aged 18 or more who were residents of Calgary households with a telephone. Telephone numbers were randomly

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generated using an application of the Mitofsky-Waksberg method[3] with a modification attributed to Waksberg that was described in a review by Potthoff.[4]

The procedure began with the random selection of a single three-digit prefix from among those in use in Calgary during the study interval. Next, a four-digit suffix was generated randomly. These seven-digit telephone numbers were called, and if a residential household was reached, a set of 10 additional numbers were called within the cluster of 100 numbers identified by the first five digits of the seven-digit number. Multiple attempts were made to contact each household: at least six for each number generated, including two during daytime hours, two during evening hours and two on weekends. When a residential household was reached, one member of the household was selected according to which household resident had most recently had a birthday.

Once the subjects had been selected for inclusion in the study, they were interviewed over the telephone using the CIDI-SFMD. The questions contained in this instrument derive originally from the CIDI,[5,6] although many of the questions were modified during the development of the short form.[7] Demographic data were also collected. Interviews were tightly scripted, including the use of standardized introductory statements, transitional statements and a series of "fallback" statements for responding to subject queries.

The sample size for the survey was 2,542 people, a subset of whom were included in the validation study presented here. Those subjects scoring positively (score 5) on the CIDI-SFMD and a random sample (initially 5%, subsequently increased to 10%) of those with negative results (score <5) were selected for the validation study. The proportion of the total sample expected to be positive on the CIDI-SFMD was approximately 5%; therefore, the selection of all CIDI-SFMD positive and 5% of negative subjects was intended to produce two approximately equal groups. However, the proportion of positive results was higher than expected, and despite the increase to a 10% random sample during the data collection, subjects with positive results predominated in the sample. Since the objective of the validation study was to explore the performance of the CIDI-SFMD in relation to the full depressive disorders section of the CIDI rather than to generate estimates applicable at the general population level, the data were not weighted to better approximate the general population.

Each selected subject who consented to be recontacted was telephoned as soon as it was convenient for them in the few weeks following initial contact and was administered the full depressive disorders section of the CIDI, version 2.1. The automated version of the instrument was used (CIDI-Auto). The proportion of CIDI-SFMD positive subjects with major depression according to the full CIDI was regarded as an estimate of the positive predictive value of the instrument, and the proportion of CIDI-SFMD negative subjects without major depression according to the CIDI provided an estimate of the negative predictive value.

Confidence intervals for the positive and negative predictive values of the CIDI-SFMD were calculated using exact methods based on the binomial distribution. In order to evaluate the impact of other variables on the level of association between the two tests, the probability of false positive status among those subjects who were positive on the CIDI-SFMD was evaluated in relation to other variables using Fisher's exact test (FET) for nominal variables with two categories, RxC χ^2 tests for nominal variables with more than two categories or Kruskal-Wallis tests comparing the median values for ordinal and non-normally but approximately symmetrically distributed continuous variables.

Results

A total of 521 subjects were invited to participate in the validation study: 361 with positive results and 160 with negative results on the CIDI-SFMD. Of the CIDI-SFMD positive subjects, 277 (76.7%) agreed to participate in the validation study, as did 136 (85.0%) of the CIDI-SFMD negative subjects. The demographic features of these subjects are presented in Table 1. Women were overrepresented in the sample, even in the CIDI-SFMD negative group, possibly because of the use of last birthday method,[8] but the female preponderance was most marked in the CIDI-SFMD positive group, as expected, since major depression has a higher prevalence in women.

Overall, 208 of the 277 CIDI-SFMD positive subjects who were administered the full CIDI were identified by that instrument as having major depression, for a positive predictive value of 75.1% (95% CI = 69.7–79.8%). Of the 136 CIDI-SFMD negative subjects administered the CIDI, 133 were found not to have major depression, for a negative predictive value of 97.8% (95% CI = 94.1–99.3%).

The proportion of false positive results did not differ with respect to sex of subject (FET, $p = 1.0$) or marital status ($\chi^2 = 4.38$, df = 4, $p = 0.36$). An examination of the impact of level of education using an omnibus chi-squared test suggested a trend toward statistical significance ($\chi^2 = 9.98$, df = 5, $p = 0.08$), so additional exploratory analysis was conducted. When subjects with high-school education or less, or with trade certification were compared with subjects with at least some post-secondary education (partial university, university degree or diploma, or advanced degree), the former group had a 29.1% false positive rate compared with 12.7% in the latter group (FET, $p = 0.0064$). In the sample as a whole, false positive subjects had a median age of 41 years, significantly greater than the median age (35 years) of the true positives (Kruskal-Wallis H = 6.168, df = 1, $p = 0.01$).

Subjects who reported having one long-term medical condition or more were not significantly more likely to have false positive results on the CIDI-SFMD: 27.4% versus 22.5% in the subjects without such conditions

TABLE 1
Demographic features of the CIDI-SFMD validation study sample

Demographic variables		CIDI-SFMD positive (*n* = 277)		CIDI-SFMD negative (*n* = 136)		Refusals[a] (*n* = 108)	
		n	%	*n*	%	*n*	%
Sex:	Male	76	27.4	51	37.5	31	28.7
	Female	201	72.6	85	62.5	77	71.3
Age:	18–29	86	31.0	28	20.6	40	37.4
	30–39	73	26.4	33	24.3	27	25.2
	40–49	66	23.8	36	26.5	23	21.5
	50–59	34	12.3	20	14.7	9	8.4
	60+	18	6.5	19	14.0	8	7.5
Marital status:	Married	92	33.2	73	53.7	41	38.0
	Never married	99	35.7	39	28.7	48	44.4
	Separated	16	5.8	4	2.9	4	3.7
	Divorced	53	19.1	14	10.3	13	12.0
	Widowed	17	6.1	6	4.4	2	1.9
Education:	Less than high-school graduation	36	13.0	8	5.9	13	12.0
	High-school graduation	77	27.8	33	24.3	25	23.1
	Secondary certificate or diploma	118	42.6	52	38.2	41	38.0
	University degree/diploma	40	14.4	31	22.8	28	25.9
	Advanced degree (>Bachelor)	6	2.2	12	8.8	1	0.9

[a] Subjects invited to participate in the validation study, but who declined participation. Missing data on age for one subject.

(FET, $p = 0.41$). Most (82.6%) of the subjects reported some consumption of alcohol in the previous year. Subjects reporting consumption of alcohol were expected to have a higher false positive rate because of alcohol-induced depressive symptoms, which, in the CIDI, are discounted sometimes as symptoms of major depression. However, contrary to expectation, subjects reporting any drinking had a lower false positive rate (21.7%) than subjects who reported no drinking in the previous year (39.2%), a statistically significant difference (FET, $p = 0.01$). CIDI-SFMD positive subjects were no more likely to be false positives if they reported alcohol consumption more than once per week (24.1%) than if they reported it less than once per week (25.1%). Similarly, subjects reporting that they had consumed five or more drinks on their occasion of maximal drinking in the previous year were no more likely to be false positives than the remaining subjects (22.7% versus 26.8% respectively, FET, $p = 0.49$). Of the 277 subjects scoring in the positive range on the CIDI-SFMD, 10.8% reported the use of street drugs in the preceding month. Primarily, these subjects reported the use of cannabis (23/30), with the remainder reporting the use of hallucinogens or cocaine. The rate of false positives among drug users was 23.3%, closely resembling that of non-drug users, 25.1%.

In order to better explain the occurrence of false positive results in the relevant 69 subjects, two additional analyses were conducted. First, the individual item responses on the full CIDI depressive disorders section were examined. Next, each CIDI interview was rescored using ICD-10 diagnostic criteria rather than DSM-IV criteria.

Eleven (15.9%) of the false positive subjects responded differently to the two initial symptom items covering depressed mood and loss of interest. At least one of these symptoms must be present for a diagnosis of major depression. The wording of the relevant questions is similar in the two instruments, and so these differences appeared to reflect unreliable subject responses. Another 9 (13.0%) subjects answered affirmatively to at least one of the questions on both of the instruments, but in the case of the CIDI the presence of these key symptoms was attributed to an organic etiology, resulting in their being coded as not present and termination of the CIDI interview at that point. Questions exploring possible organic etiologies are not included in the short form.

Twelve subjects in the false positive category (17.4%) were assigned another depressive diagnosis by the CIDI (either dysthymia, an ICD-10-defined depressive episode

or both), suggesting that the short form was capturing a broader range of depressive morbidity than that subsumed under the major depression category. Finally, 37 subjects (53.6%) were affirmative on the initial depressed mood and loss-of-interest questions for each instrument, but failed to meet severity or clinical significance criteria contained in the CIDI. The CIDI contains numerous questions evaluating the severity and clinical significance of symptoms, for example, whether symptoms such as psychomotor agitation were noticed by other people, whether symptoms followed the death of a loved one or whether they interfered substantially with activities. None of these probes is contained in the short form.

There were only three false negative subjects, each of whom endorsed four of the required five symptoms on the CIDI-SFMD. Hence, they would have scored in the range that was regarded as positive in this study had they reported one additional symptom. Here, the occurrence of false negative results was easily explicable because the full CIDI probes several symptoms in much greater detail than does the short form. For example, in the full CIDI there are questions about insomnia and hypersomnia, whereas a single sleep-related question on the short form refers specifically to initial insomnia. Also, the full CIDI covers symptoms such as agitation and libido that are not covered in the short form.

Conclusions

In this study, the CIDI Short Form for Major Depression was evaluated in relation to the full depressive disorders section of the CIDI interview. The short form was scored categorically using the cut-point of five symptoms, which in previous studies has been related to a 90% positive predictive value.[7] It is also a logical cut-point for the instrument and one associated with the property of face validity, since the DSM-IV requires the presence of five symptom-based criteria for major depression in order for the diagnosis to be assigned.

One methodologic weakness of the study was that the order of administration of the instruments was not randomized. Hence, the outcome of the second interview (the full CIDI depressive disorders section) could be related to that of the first. Respondents might be motivated to make their responses consistent with one another, such that those subjects scoring in the positive range on the CIDI-SFMD might be motivated to provide more affirmative answers in the subsequent CIDI interview. An effect of this nature would tend to exaggerate both the positive and negative predictive value of the CIDI-SFMD. On the other hand, interview fatigue could lead to reduced symptom reporting upon reinterview,[9] thereby exaggerating the number of apparent false positives. However, the extent of such effects cannot be determined using the data collected in this study.

Another important methodologic issue is that of statistical power. For example, although indices of alcohol drinking (frequency of drinking and maximum consumption on any one occasion) were not associated with false positive status, the numbers of subjects in the frequent and heavy categories of use were small, such that some of the negative results may represent Type II errors. Another methodologic concern is the issue of work-up bias. Since administration of the full CIDI was dependent upon the outcome of the CIDI-SFMD, this form of bias precluded the direct calculation of sensitivity and specificity from the data collected here, although such bias would not systematically distort estimates of positive and negative predictive value.[10] In theory, it is possible to estimate sensitivity and specificity from predictive values and prevalence.[10] However, in the current study, the level of precision achieved in the estimate of negative predictive value precluded reliable estimation using these methods.

The higher positive predictive value (90%) reported for the five symptom cut-point by the developers[7] of the short form may be an overestimate. Since the predictive value of the short form appears to have been estimated from the same data set that was used to develop the instrument, lower predictive values in independent samples might have been expected. This is especially true when it is considered that the items included in the short form were specifically chosen to maximize the predictive values in that particular sample. The short form should be studied in additional independent samples in order to further refine the available data concerning its validity. The addition of questions directly addressing issues of clinical significance and expanding on the coverage of key symptoms might improve the concordance of the CIDI-SFMD with the CIDI.

The short form was found to have excellent negative predictive value and a positive predictive value of approximately 75%. The false positive results were largely understandable in terms of the nature of the instruments evaluated. Since each instrument keys in on two obligatory symptoms—depressed mood and loss of interest—inconsistencies in the answers to these critical questions explained some of the differences. Other discrepancies could be attributed to areas not explored by the short form: the clinical significance of symptoms and their relationship to organic causes. The CIDI assigned a diagnosis of other depressive disorders to some false positive subjects, suggesting that the short form picks up a broader range of depressive disorders than the major depression criteria as applied by the CIDI. These findings should be kept in mind when interpreting data from the CIDI short form. Not all subjects with high scores on this instrument have major depression; some may have symptoms that are due to physical conditions, and some may have more mild forms of depressive disorder or bereavement.

The lack of an association observed in this study between false positive status and both self-reported physical condition and crude indices of alcohol consumption suggests that simple correction of prevalence for such variables as medical condition and drinking status will not be feasible in surveys in which the CIDI-SFMD has

been used. An exploration of the severity, impact on functioning and potential etiology of each symptom would be required to eliminate these bases for lack of agreement, and this would amount to a re-elaboration of the full diagnostic interview.

Proper interpretation of the CIDI-SFMD involves recognizing that this instrument identifies a depressive syndrome that is closely related to, but not identical with, DSM-IV-defined major depression. Since treatment needs and the public health impact of depression are not related exclusively to the prevalence of any specific depressive syndrome but also to such factors as the duration of episodes and the extent of disability and distress, it is important that CIDI-SFMD-based prevalence estimates be interpreted with reference to these additional variables. In Canada, the inclusion of the CIDI-SFMD in the National Population Health Survey provides a suitably rich context for its meaningful interpretation.

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Health-adjusted Life Expectancy at the Local Level in Ontario

Douglas G Manuel, Vivek Goel, J Ivan Williams and Paul Corey

Abstract

Health expectancy measures are becoming a common method of combining information on mortality and health-related quality of life into one summary population health measure. However, health expectancy measures are infrequently measured at the local level, despite a shift toward health service planning to that level. Using a modified Sullivan method, we calculated health-adjusted life expectancy (HALE) for the 42 public health units in Ontario using life tables that were derived from mortality and population data for 1988–1992 and the Health Utilities Index from the 1990 Ontario Health Survey. There were large variations among health units in HALE at age 15 for both men (range: 51.3–58.2 years) and women (range: 56.6–62.9 years). Generally, rural and northern areas had the lowest HALE. Local differences in male HALE were greater than for life expectancy (7.1 versus 6.0 years). Despite a relatively large health survey (45,583 respondents, range: 729–1,746 per health unit), few HALE differences deviated significantly from the Ontario mean, raising concerns about the feasibility of estimating local health expectancy measures with adequate precision. Nevertheless, the wider local differences and different geographic distribution of local HALE compared with mortality measures, along with the additional benefit of being able to model the complex interaction of mortality and morbidity, suggest that HALE may be a useful population health measure.

Key words: *demography; demography/methods; health expectancy; health status; health status indicators/standards; life expectancy; Ontario/epidemiology*

Introduction

One of the most striking recent changes in health care delivery in Canada and other countries has been to allocate the responsibility for planning to the regional or local level.[a] Concurrently, there has been a growing interest in population health outcome measures, in particular summary measures that include health-related quality of life (HRQOL) and not merely mortality or disease. Health expectancy is a potentially useful measure that combines data on HRQOL with mortality data in the form of life tables; however, to date, local health expectancy measures have been infrequently reported. The main barrier to their development and dissemination is the lack of availability of life tables and local area surveys of health status with sufficient statistical power to yield significant findings.

Health expectancy describes a family of indices that combine mortality (life expectancy) with different measures of health-related quality of life.[1–3] In this way, health expectancy more closely reflects current definitions of health than do indicators of morbidity or mortality alone. Like other life table measures, health expectancy builds on the principles of a stationary population to model the effects of changing patterns of health.[4] Among its various applications, health expectancy is one of the few population indicators, when measured over time, that can assess whether there is a reduction or expansion of morbidity.

Health-adjusted life expectancy (HALE) is a particular type of health expectancy measure. It incorporates explicit weights to combine discrete health states into a single indicator of the expectation of equivalent years of

[a] In the context of this work, the local areas are the 42 health units in Ontario with a population ranging from 40,600 to 739,900 and a median of 385,900; total Ontario population was 10,341,200 in 1990.

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good health. Other popular health expectancy measures that use dichotomous weights include disability-free life expectancy and healthy life expectancy (e.g. using self-rated health). HALE is particularly attractive to health economists since it can be appropriately compared with other health status measures, such as life expectancy or disease-specific utility measures. Also, since HALE uses polychotomous weights, it is sensitive to changes in the severity of disability within a population.

For an illustration of the benefit of combining mortality and morbidity using health expectancy measures, consider the following hypothetical situation. Assume there are two communities that have identical mean HRQOL and life expectancy in 1998 (and therefore the same health expectancy). In 1999, there is a large outbreak of influenza in one community, and the frail people—those with a very low HRQOL—die. The other community is spared influenza. After the outbreak the cross-sectional HRQOL would be higher in the community with the outbreak, but the life expectancy for 1999 would be lower. Health expectancy will be lower in the outbreak community, and the difference between communities will be less than the difference between life expectancy. In this example, an acute illness has opposite effects on the community's HRQOL and life expectancy. A health expectancy measure is the most appropriate indicator to measure the population effect of the influenza outbreak.

There are many other additional health indicators and planning applications that are readily available once the basic life table is created for a local planning area and combined with an HRQOL measure. These include the impact of eliminating individual diseases using expected years of life lost from simple abridged life tables or healthy years of life lost;[5,6] the latter method is particularly useful for modelling the shifting burden of disease from acute to chronic conditions.[4] In a similar way, it is possible to estimate the contribution of the different HRQOL domains (ambulation, pain, cognition, etc.) or socio-economic factors on HALE.[7,8]

Most of the existing health expectancy estimates are for nations,[9] and most of the regional estimates are calculated for relatively large populations of 2–30 million[10–13] (although there are exceptions[14,15]). Most of these analyses have estimated disability-free life expectancy or healthy life expectancy (for derivations of HALE for larger populations see the studies by Wolfson[7] and others[12,16]).

It is possible to calculate a utility-based HALE at the local level in Ontario because of the 1990 Ontario Health Survey (OHS), the first province-wide health survey to include health status attributes needed to derive the Health Utilities Index (HUI).[17] This utility-based health status measure is included in the ongoing National Population Health Survey and Canadian Community Health Surveys. In order to calculate HALE, life tables for the local areas are also required. Although life tables have historically been calculated for small populations in many countries, the practice has been less common in many areas of Canada,[18] at least until very recently.[19] Most local planning areas can derive local life tables using established methods[20] and vital statistics, if there is accurate geographic coding.

This paper describes the derivation of HALE for the 42 health units in Ontario in 1990 using the Health Utilities Index[17] and vital statistics mortality data.

Materials and Methods

Data Sources

Data on health status were obtained from the 1990 OHS (described fully elsewhere[21,22]). Briefly, 61,239 subjects were selected through a stratified, multi-level cluster sampling method with the Ontario health units as the primary sampling unit. The target population included all residents living in private dwellings in Ontario. Residents of First Nations reserves and long-term care institutions, foreign service personnel and residents of remote areas were excluded.

There were two stages of the survey, the first involving an interview-completed questionnaire. For this stage, one individual responded on behalf of all members of the household (response rate: 87%). The second stage consisted of a self-completed questionnaire that was given to all members of the household over the age of 12 (response rate: 77%, effective sample size 729–1,746 per health unit). The questions pertaining to the HUI attributes were contained in the self-completed questionnaire. Response rates were higher in rural areas, among women and in the older population.

Mortality files from the Ontario Registrar-General/ Statistics Canada for 1988–1992 were used to calculate the age- and sex-specific death rates for each health unit. In total, 358,490 Ontario residents died during the study period (range for health units: 1,980–29,671). Postcensal population estimates for 1990 by sex and age group were obtained from Statistics Canada.

Health Utilities Index

The HUI is a multi-attribute health status classification system that estimates a summary value of individual health between 0 ("dead") and 1 ("perfect health") based on preference scores for different health states.[23] Each respondent answers questions pertaining to eight attributes of functional health (vision, hearing, speech, mobility, emotional state, thinking and memory, dexterity, and level of pain and discomfort). Each attribute has from four to six possible responses representing a range from an unrestricted to a highly disabled state (see Appendix).

The preference score for each attribute and the subsequent multi-attribute preference function were derived from an earlier (Mark II) version of the HUI that elicited responses from approximately 200 parents of school-aged children in a local municipality.[17] Preference weights were estimated using standard gamble and visual analog methods.[23] Mark II weights were applied to the Mark III questions included in our study using a provisional conversion scoring system.[24] The most important difference

between the two versions is that the Mark II version groups hearing, speech and vision into a single "sensation" attribute whereas the Mark III version treats them separately.

All individual and summary HUI estimates from the 1990 OHS were weighted according to the likelihood that the respondent would be selected from the provincial population. These weights were inversely proportional to the probability of being selected for the survey. Summary weighted HUI estimates for health units were age- and sex-standardized to the 1990 population of Ontario, using the direct method.

Life Tables

Chiang's method[20,25] was used to generate abridged life tables for each health unit by sex and 19 standard age groups (<1,1–4, 5–9,10–14 . . . 85+ years) for 1990 based on age-specific mortality rates for 1988–1992 and the 1990 mid-interval population estimates.

Health-adjusted Life Expectancy

HALE at age 15 by sex and health unit was calculated using a modified version of Sullivan's method.[26] From the 1990 OHS, the age- and sex-specific weighted HUI was estimated for the 42 health units by five-year age groups, from age 15 to 85 and over. For each age group, "life-years lived" (L_x in traditional life table nomenclature, where x is the age interval) from the corresponding life table was multiplied by the mean HUI estimate to create "health-adjusted years of life lived" ($L'_x = L_x \times \overline{\text{HUI}}_x$). Then, health-adjusted years of life lived were summed and divided by the total number of persons surviving at age 15, to provide HALE at age 15. Health-adjusted life expectancy lost is an estimate of the amount of life in an "ill" health state and is calculated by subtracting HALE from life expectancy.

Statistical Methods

To estimate 95% confidence intervals, variance for life expectancy was calculated by a method described by Chiang.[25] Variance for HALE was calculated by a method described by Bebbington,[27] which considers only the error of the health status measure (i.e. HUI), despite statistical error in the conditional probability of death in a life table. This assumption is reasonable since the statistical variance of HUI introduces much greater error than the probability of death, despite the small populations used in the present study. Standard errors took into consideration both design effect and sample weights. The large number of health units (42) meant that each health unit comprised a small proportion of the Ontario population, and therefore statistical significance was estimated by comparing each health unit with the Ontario mean. All analyses were performed using SAS (Statistical Analysis System) software, version 6.12.

Results

Of the 46,583 OHS respondents aged 15 or older, 45,583 (98.3%) gave valid responses to the questions used to derive the HUI. The mean HUI for Ontario was 0.92 (males: 0.92, females: 0.91) with a range among health units of 0.89 to 0.93. The high score for mean HUI and the small difference between health units reflect the large proportion of the population in a state of "high health," especially those at younger ages. When only those in a high health state are considered (HUI > 0.95, indicating perfect health or those with perfect health but wearing corrective vision glasses or hearing aids), a larger difference was observed among health units (68% of Ontarians' HUI was greater than 0.95, health unit range: 57–75%).

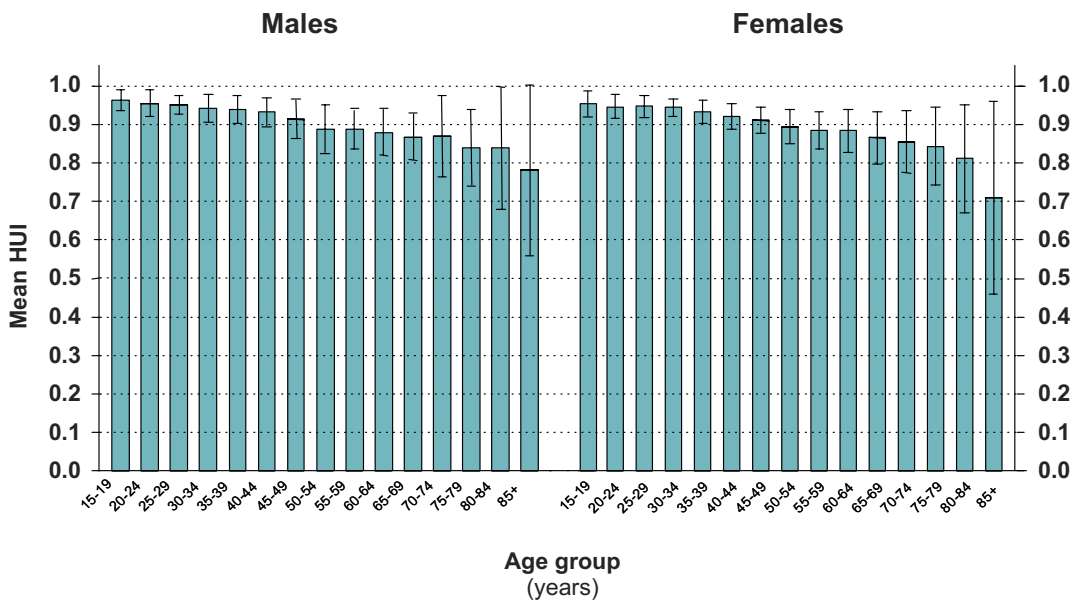
Figure 1 shows the mean age-specific HUI for Ontario and the 95% confidence intervals (CI) for the health unit estimates. Generally, health units with a high mean HUI in one age group had a high HUI in all age groups, an important consideration since the derivation of health expectancy combines the age-specific HUI scores with the age interval of life-years lived.

The life expectancy at birth in Ontario for 1988–1992 was 74.8 (95% CI = ±0.1) years among males and 80.9 years among females; at age 15 it was 60.7 and 66.6 years (95% CI = ±0.1 years) respectively. The mean HALE among males at age 15 in Ontario for the same period was 55.2 years and among females, 59.8 years (95% CI: ±1.43). The HALE sex difference (4.6 years) was less than the sex difference seen in years of life expectancy (5.9 years). This longer period of ill health (1.3 years) reflects a lower HRQOL (as measured by the HUI) among females at all age groups over age 15, but especially those at older ages.

Figure 2 shows the range of HALE across the 42 health units. Among males, the range of HALE was 51.3–58.2 years at age 15; among females, 56.6–62.9 years. The life expectancy range among males was 57.2–63.2; among females, 62.6–69.0 years. There were few health units that had a statistically significant difference in HALE (at $p < 0.05$) from the provincial mean (10 of 42 health units for males, 4 for females; range in 95% CI = 1.79–3.21 years) compared with life expectancy (37 of 42 for males, 33 for females; range in 95% CI = 0.23–0.93 years). HALE lost from ill health varied from 4.2 to 6.9 years among males, and from 6.0 to 8.4 years among females. HALE lost was moderately and negatively correlated with life expectancy (Pearson's unweighted correlation = –0.35).

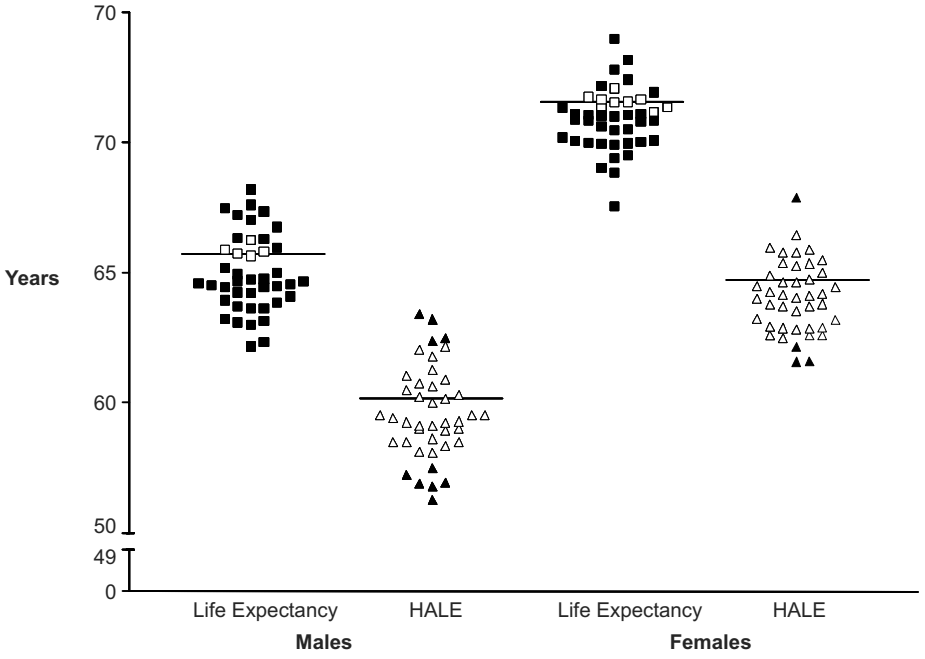
The geographic pattern for HALE for males is shown in Figure 3; the female distribution is similar and not shown. Generally, northern and rural health units had lower HALE than their urban counterparts. The urban/rural difference in HALE is more noticeable than that seen with life expectancy (data not shown). Males in the City of Toronto were the notable exception, with the lowest life expectancy and second lowest HALE. Females in the City of Toronto fared somewhat better, with both life and HALE ranking in the lowest and second lowest quintile.

FIGURE 1
Health Utilities Index (HUI) by age group and sex, Ontario, 1990[a]



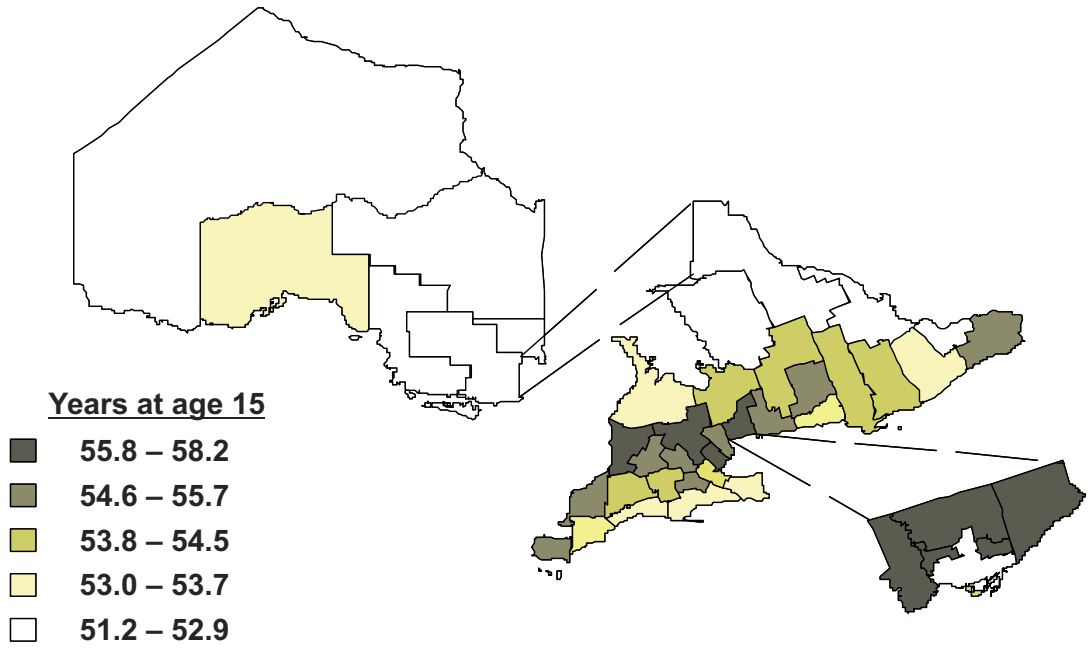
[a] Each bar represents the mean and 95% confidence interval of the HUI for the combined population of the 42 health units in Ontario.

FIGURE 2
Life expectancy and health-adjusted life expectancy (HALE) at age 15 by health unit and sex, Ontario, 1990[a]



[a] Each point represents the life expectancy or health-adjusted life expectancy for one health unit. The horizontal bar represents the Ontario mean. HALE and life expectancy estimates that are significantly different ($p < 0.05$) from the Ontario mean are shaded in black.

FIGURE 3
Health-adjusted life expectancy by quintile for males aged 15, Ontario, 1990



Discussion

In this paper, health status at the local level in Ontario was estimated by combining mortality with a utility-based health status index. The results highlight two applications of HALE and other health expectancy measures. First, there is interest in using health expectancy as a summary population-based measure of health. Clearly, HALE measures health differently from other mortality-based indicators and, in some ways, it more closely reflects current concepts of health. Although there is a growing interest in the geographic comparison of health-related quality of life indicators, our theoretic example of the impact of an influenza outbreak demonstrates the difficulty in assessing population health status among only the survivors. This leaves us with the need to combine mortality and morbidity together in a combined indicator, of which health expectancy is increasingly becoming one of the most practical choices for developed countries.[2]

One advantage of a utility-based measure of health expectancy such as HALE, incorporating the HUI, is the ability to value the equivalent influences of mortality and health-related quality of life together to create a combined perspective of health.

Since HALE captures a relatively broad perspective of health, even small HALE differences have important public health significance. In this light, the wide difference in HALE at the local level (7.1 years at age 15 among males, 6.3 years among females) suggests that there is a large and important disparity in health between local areas. As with mortality indicators there is a strong north/south, urban/rural gradient in health; however, the larger male difference in HALE compared with life expectancy (7.1 versus 6.0 years) indicates larger health differences than seen with mortality indicators alone. HALE at the local level indicates that the magnitude of health differences among males may be even larger than previously estimated using other indicators.

Sex differences were smaller for HALE than for life expectancy (4.6 versus 5.9 years). Compared with males, women had a longer life expectancy but lived a smaller proportion of their life in a healthy state.

The second application of health expectancy measures is the ability to model health effects that cannot be assessed using measures of mortality or morbidity individually. The message that male residents in northern and rural health units not only have a shorter life but also experience a smaller proportion of their life in good health is not captured by any other single population health indicator.

For health modelling, health expectancy's largest potential is probably seen with further breakdown of disease, health status states and the transitions between these states in different populations and over time. For example, northern health units have a higher prevalence of acute diseases, such as childhood infectious diseases and unintentional injuries, resulting in a higher mortality at younger ages. However, there is also the concern that individuals and communities in these areas have fewer resources, and those non-fatal acute events are more likely to result in chronic disability. If this were the actual situation there would be a greater difference in

local HALE compared with life expectancy, as seen in our estimates.

This study does have some limitations to consider. The analysis highlights the difficulty of relying on population-based surveys to assess health expectancy at the local level. Despite the large sample and large absolute differences in HALE, there is likely insufficient power to detect statistically significant differences between many local areas in Ontario.

This raises a concern regarding the benefit of estimating health status with a utility-based measure at the local level in large and costly surveys such as the OHS. One possible solution would be to have a limited number of questions on health status (not necessarily the HUI) included in the Canadian census. Questions on disability were included in the 1991 and 1996 censuses, and these could be used to estimate disability-free life expectancy. Another alternative is to first estimate the statistical power before performing analysis at the local level, and to proceed only if the power is adequate. The results presented here may be used to gauge the power for future analysis, but the estimation of life table variances is a complex problem, and no power estimation methods are currently available.

Patients living in institutions were not surveyed in the OHS. Estimates would be lower if the highly disabled population in institutions were included, even though institutionalized elderly people are a small proportion of the total population.[16] The effect is important when comparing the difference in HALE between men and women, since a greater proportion of women aged 65 and over are in institutions. Differences in HALE among health units would likely be slightly larger if institutionalized patients were included, since there is a strong positive correlation between the rank of HALE and the age-standardized institutionalized bed census data (data not shown).

Health expectancy uniquely models complex morbidity and mortality interactions that are increasingly important for health planners and are otherwise extremely difficult to observe. Health policy directed at improving or reducing local disparities in health expectancy may be different from policies based on current indicators. Whether similar large disparities in local health expectancy exist in other areas is not known. However, local differences are seen in other health indicators, such as infant mortality and self-perceived health status, in other provinces in Canada, and they likely exist for health expectancy.

Local life tables as well as health status data are required. Although the former are usually readily available, health status data are not routinely collected at this level of aggregation. The justification for collection of such expensive data is based on the need for local planning and the additional benefit over using provincial level data. Nevertheless, there are several methodologic statistical issues for local estimates that require further evaluation.

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APPENDIX
The Health Utilities Index Mark III attributes and levels

Attribute	Level	Description
Vision	1	Able to see well enough to read ordinary newsprint and recognize a friend on the other side of the street, without glasses or contact lenses
	2	Able to see well enough to read ordinary newsprint and recognize a friend on the other side of the street, but with glasses or contact lenses
	3	Able to read ordinary newsprint with or without glasses but unable to recognize a friend on the other side of the street, even with glasses or contact lenses
	4	Able to recognize a friend on the other side of the street with or without glasses but unable to read ordinary newsprint, even with glasses or contact lenses
	5	Unable to read ordinary newsprint and unable to recognize a friend on the other side of the street, even with glasses or contact lenses
	6	Unable to see at all
Hearing	1	Able to hear what is said in a group conversation with at least 3 other people, without a hearing aid
	2	Able to hear what is said in a conversation with 1 other person in a quiet room without a hearing aid, but requires a hearing aid to hear what is said in a group conversation with at least 3 other people
	3	Able to hear what is said in a conversation with 1 other person in a quiet room with a hearing aid, and able to hear what is said in a group conversation with at least 3 other people, with a hearing aid
	4	Able to hear what is said in a conversation with 1 other person in a quiet room without a hearing aid, but unable to hear what is said in a group conversation with at least 3 other people even with a hearing aid
	5	Able to hear what is said in a conversation with 1 other person in a quiet room with a hearing aid, but unable to hear what is said in a group conversation with at least 3 other people even with a hearing aid
	6	Unable to hear at all
Speech	1	Able to be understood completely when speaking with strangers or people who know me well
	2	Able to be understood partially when speaking with strangers but able to be understood completely when speaking with people who know me well
	3	Able to be understood partially when speaking with strangers or people who know me well
	4	Unable to be understood partially when speaking with strangers but able to be understood partially by people who know me well
	5	Unable to be understood when speaking with other people (or unable to speak at all)
Ambulation	1	Able to walk around the neighbourhood without difficulty, and without walking equipment
	2	Able to walk around the neighbourhood with difficulty, but does not require walking equipment or the help of another person
	3	Able to walk around the neighbourhood with walking equipment, but without the help of another person
	4	Able to walk around the neighbourhood with walking equipment, and requires a wheelchair to get around the neighbourhood
	5	Unable to walk alone, even with walking equipment. Able to walk short distances with the help of another person, and requires a wheelchair to get around the neighbourhood
	6	Cannot walk at all

APPENDIX *(continued)*
The Health Utilities Index Mark III attributes and levels

Attribute	Level	Description
Dexterity	1	Full use of 2 hands and 10 fingers
	2	Limitations in the use of hands or fingers, but does not require special tools or help of another person
	3	Limitations in the use of hands or fingers, is independent with use of special tools and does not require the help of another person
	4	Limitations in the use of hands or fingers, requires the help of another person for some tasks (not independent even with the use of special tools)
	5	Limitations in use of hands or fingers, requires the help of another person for most tasks (not independent even with use of special tools)
	6	Limitations in use of hands or fingers, requires the help of another person for all tasks (not independent even with use of special tools)
Emotion	1	Happy and interested in life
	2	Somewhat happy
	3	Somewhat unhappy
	4	Very unhappy
	5	So unhappy that life is not worthwhile
Cognition	1	Able to remember most things, think clearly and solve day-to-day problems
	2	Able to remember most things, but has a little difficulty when trying to think and solve day-to-day problems
	3	Somewhat forgetful, but able to think clearly and solve day-to-day problems
	4	Somewhat forgetful, and has a little difficulty when trying to think or solve day-to-day problems
	5	Very forgetful, and has great difficulty when trying to think or solve day-to-day problems
	6	Unable to remember anything at all, and unable to think or solve day-to-day problems
Pain	1	Free of pain and discomfort
	2	Mild to moderate pain that prevents no activities
	3	Moderate pain that prevents a few activities
	4	Moderate to severe pain that prevents some activities
	5	Severe pain that prevents most activities

Ontario Familial Colon Cancer Registry: Methods and First-year Response Rates

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Abstract

The Ontario Familial Colon Cancer Registry (OFCCR) is a novel registry that collects family history information, epidemiologic data, blood samples and tumour specimens from a population-based sample of colorectal cancer patients and their families. Families are classified as either high familial risk, intermediate familial/other risk or low (sporadic) risk for colorectal cancer. Obtaining high response rates in genetic family studies is especially challenging because of both the time commitment required and issues of confidentiality. The first-year response rate was 61%, resulting in 1,395 participating probands. In an attempt to assess potential response bias, we compared participants with non-participants. The age and sex of participants did not differ from non-participating probands; however, cases in rural areas were somewhat more likely to participate. To date, 57% of 1,587 relatives participated; females were more likely to participate, and relatives of low familial risk were least likely to participate. The OFCCR is an excellent resource that will facilitate the study of genetic and environmental factors associated with colorectal cancer.

Key words: *colorectal neoplasms; genetic family studies; hereditary nonpolyposis colorectal cancer; methodology; response rates*

Introduction

The Ontario Familial Colon Cancer Registry (OFCCR) is one of six international sites participating in the Co-operative Familial Registry for Colorectal Studies, established by the US National Cancer Institute. Since its inception in 1997, the OFCCR has been collecting detailed family history information, epidemiologic data, blood samples and tumour tissue from a population-based sample of colorectal cancer patients (probands) and their families. The OFCCR was designed to facilitate future colorectal cancer investigations in such areas as genetic epidemiology, gene discovery, primary prevention, psychosocial research, screening and treatment.

Families in the OFCCR are classified as high familial risk, intermediate familial/other risk or low (sporadic) risk on the basis of their family history of cancer as well as other information (see Table 1). Of particular interest are families with hereditary nonpolyposis colorectal cancer (HNPCC), a condition in which individuals are at high risk for colorectal as well as certain other cancers.[1,2] Because this condition accounts for 2–3% of all colorectal cancer cases,[3,4] only large investigations such as the OFCCR can provide adequate numbers of families for study. Within such families, the recent identification of the DNA mismatch repair (MMR) genes responsible for HNPCC has made it possible to identify carriers of these gene mutations.

A high response rate is important in order to ensure that the families in the OFCCR are representative of the population from which they were selected. However, obtaining high response rates in genetic family studies of colorectal cancer is especially challenging because of the

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TABLE 1
Criteria used to classify probands in the OFCCR

High familial risk/HNPCC[a] (Amsterdam criteria[16])

1. At least three relatives with colorectal cancer, one a first-degree relative to the other two, *and*
2. At least two successive generations affected with colorectal cancer, *and*
3. Colorectal cancer diagnosed under 50 years of age in at least one affected member, *and*
4. No familial adenomatous polyposis (FAP)

Intermediate familial/other risk (*familial* [#1–3], *other* (pathologic [#5–11], other [#4, 12]))

1. Proband has two relatives with any of the HNPCC cancers[b] *and* two of the three are first-degree relatives, *or*
2. Any family member with an HNPCC cancer[b] ≤35 years of age, *or*
3. Proband <50 and relative with colon cancer <50 (first- or second-degree relative only), *or*
4. Proband ≤35 years of age, *or*
5. Proband with multiple primary colon cancers, *or*
6. Proband with other primary HNPCC cancer(s)[b], *or*
7. Proband has multiple polyps, *or*
8. Peutz-Jeghers or hamartomatous polyp, *or*
9. Juvenile polyp, *or*
10. Inflammatory bowel disease, *or*
11. Unusual colorectal cancer histologies[c], *or*
12. Proband is Ashkenazi Jewish

Low (sporadic) risk

1. All other colorectal cancer cases (probands) not classified as high or intermediate risk

[a] HNPCC: hereditary nonpolyposis colorectal cancer

[b] Colorectal, endometrial, gastric, small bowel, gastroesophageal, liver, pancreas, biliary tract, ovarian, kidney, ureter, brain, lymphoma

[c] Carcinosarcoma, adenosquamous, spindle cell, metaplastic, choriocarcinoma, signet ring, undifferentiated, trophoblastic differentiation, small cell neuroendocrine carcinoma

time commitment required to complete the many phases of data collection, issues of confidentiality[5] and the high mortality rate among the cancer cases.[6]

The purpose of this paper is threefold: to outline the design and methods of the OFCCR; to report the response rates for the first year of proband identification, stratified by subject characteristics to assess potential response bias; and to describe the basic characteristics of families enrolled in this registry during the first year of recruitment.

Methods of the OFCCR

Recruitment of Colorectal Cancer Cases/Families

The population-based Ontario Cancer Registry is used to identify living, incident colorectal cancer cases (probands) aged 20–74 who were diagnosed between July 1, 1997, and June 30, 2000. Physicians identified from pathology reports are asked to permit contact with their patients and to provide each patient's address, telephone number and vital status. Once a physician gives consent, his/her patient is mailed a package containing an introductory letter, a brochure describing the various phases of the OFCCR, a family history questionnaire and a return postage-paid envelope. A reminder postcard is sent two weeks after this mailing, and non-responders are followed up with a telephone call approximately eight weeks after the initial mailing. Proxies are not currently sought for deceased patients. On average, there is an eight-month lag between colorectal cancer diagnosis and patient contact (by mail) by the OFCCR (the main reason for this being the six-month average delay between diagnosis and case identification by the OFCCR).

The family history questionnaire requests information from the proband on all first-degree relatives, including name, birth date, vital status, date and age of death, cancers diagnosed and age at diagnosis as well as details of any cancers in second- and third-degree relatives. Pedigrees are constructed on the basis of this information. The proband is then classified as belonging to either a high familial risk family (satisfying HNPCC Amsterdam criteria [see Table 1]), an intermediate familial/other risk family (see Table 1) or a low (sporadic) risk family. All high risk and intermediate/other risk cases and a 25% random sample of the sporadic cases are selected to participate in additional phases of this familial registry (see sections below).

Genetic counsellors and trained research assistants telephone all selected cases and obtain or clarify information reported on the family history questionnaire regarding all first-degree relatives. In addition, the callers expand the family pedigree by obtaining information for all second-degree relatives on surname, given name,

cancer diagnosis, vital status, current age or age at death. To assist in the selection of relatives to be invited to participate in the OFCCR, one, both or neither side of the proband's family is designated "at risk" according to the proband's pedigree (i.e. possibly having inherited a genetic mutation associated with colorectal cancer).

Additional Phases of the OFCCR

Participating probands are asked to (1) complete a self-administered mailed epidemiologic questionnaire (providing information on bowel screening, medical conditions, medication use, diet, reproductive factors, physical activity, sociodemographic factors and anthropometric measures) and a food frequency questionnaire; (2) provide a blood sample for possible future genetic analysis; and (3) give permission to obtain, for molecular analysis, their paraffin blocks of tumour tissue that are stored in pathology departments.

In addition, all probands classified as high and intermediate risk and a 25% sample of the selected sporadic cases are asked to give permission for their first-degree relatives to be contacted as well as any living "at risk" relatives with an HNPCC-related cancer and their first-degree relatives. If permission to contact these relatives is granted, relatives are mailed a letter inviting them to participate in the OFCCR. Only those relatives who consent are sent an epidemiologic risk factor and diet questionnaire and are asked to provide a blood sample.

Genetic Counselling

All probands and their relatives are offered genetic counselling before they provide a blood sample. Based on a review of family history, this counselling includes an assessment of risk and provision of colorectal cancer screening recommendations. To fulfill the requirements of informed consent, counsellors also educate the proband about the process of genetic testing, outlining its risks and benefits. If the proband understands and agrees to all aspects of genetic testing, a blood sample is drawn for the OFCCR.

Verification of Reported Cancers

Following written permission and additional funding, the OFCCR will verify the pathology findings and date of diagnosis of all reported colon and extra-colonic HNPCC cancers in all "at risk" relatives using pathology reports obtained from either the Ontario Cancer Registry, other cancer registries outside Ontario, hospitals or clinics. The OFCCR will also attempt to verify cancer diagnoses reported among relatives living in other countries by writing to the relevant registry or hospital (subject-reported) to request a copy of the pathology report.

Molecular Genetic Analysis

Colorectal tumour tissue from all probands classified as high risk (HNPCC) and intermediate familial/other risk is reviewed by a pathologist, processed and stored in the Biospecimen Repository of the Ontario Cancer Genetics Network (OCGN) at Mount Sinai Hospital, Toronto. Microsatellite instability (MSI), a molecular marker of MMR gene deficiency, is defined as the presence of altered/additional bands in the PCR (polymerase chain reaction) amplified product of tumour DNA in comparison with the matched normal DNA samples obtained from the adjacent normal colon. Tumours are designated as MSI high (MSI-H), low (MSI-L) and microsatellite stable (MSS), according to recently published international guidelines.[7] Only probands who are found to have MSI-H tumours are actually tested for MMR gene mutations (described below).[8] In addition, before MMR mutation testing, immunohistochemical studies are done to help identify which MMR gene may be mutated.[9]

Blood samples obtained from MSI-H probands are used as a source of nucleic acids for MMR mutational analysis in the OCGN molecular laboratories. The coding region of certain MMR genes, including MLH1/MSH2, is amplified by PCR and screened for mutations by various molecular techniques. Following the identification of probands carrying an MMR gene mutation, all relatives on the "at risk" side of the proband's pedigree are offered genetic counselling for predictive testing, and those who agree are tested for the specific underlying MMR gene mutation.

Population Controls

Population controls are identified using Infodirect, a service of Bell Canada that provides a list of residential telephone numbers in Ontario. Randomly selected households from this list are telephoned to obtain a census of household members (age, sex). If there is more than one eligible household member (matched by sex and five-year age group with OFCCR case distribution), then one person is randomly selected and asked to participate. The family history and epidemiologic questionnaires are mailed to the consenting subject. A random sample of the controls will be asked for a blood sample and for consent to contact their relatives regarding participation in the OFCCR. Analyses of response rates in population controls is beyond the scope of this paper.

Descriptive Data Analyses

This paper reports response rates for cases of colorectal cancer diagnosed within the first year of the OFCCR (July 1, 1997, to June 30, 1998) and for their families.

Response rates were calculated for three phases of the OFCCR: (1) physician consent, (2) proband participation (i.e. completing the family history questionnaire) and (3) participation of relatives. To assess response bias, the distributions of several subject characteristics were compared for participants and non-participants using Pearson chi-squared statistics.

The distribution of familial risk (high [HNPCC], intermediate/other, low [sporadic]) was determined. The calculated mean and the actual number of first-degree relatives reported by probands were compared across familial classification and age group using analysis of variance statistics (ANOVA).

Results

Consent to approach patients was obtained from physicians for 93% (2,613) of the eligible colorectal cancer patients diagnosed between July 1, 1997, and June 30, 1998, who had a physician contact (Table 2). Of these 2,613 patients, contact was made with 2,289, of whom 61% participated by returning their family history questionnaire (Table 3). If the "unable to contact" patients are retained in the denominator, the response rate was 59%. Approximately two thirds of the colorectal cancer patients had colon cancer (data not shown). Non-participants did not differ markedly from participants with respect to age group and sex. However, cases living in a rural area were slightly more likely to participate. Participation rates did not differ between patients with early stage colon cancer and those with advanced (metastatic) disease.

These response rates were also calculated with "unable to contact" and deceased probands included in the denominator. The findings were no different for age, sex and residency (although the response rate was slightly lower); however, advanced cancer cases were less likely to be participants ($p = 0.02$) [data not shown]. This is because a higher proportion of advanced cancer patients were deceased, and proxies were not sought.

On the basis of reported family history and other information (see Table 1), 2% of cases were classified as high (HNPCC) familial risk, 32% as intermediate familial/other risk and 66% as low (sporadic) risk (Table 4). To date, the 535 probands selected to continue with the OFCCR reported having 4,284 first-degree relatives, of whom approximately 66% were alive (Table 5). Probands had an average of 8 (±4) first-degree relatives, and there was no significant association between the number of first-degree relatives and the familial risk status of the proband or the proportion alive. However, as might be expected, the number of first-degree relatives increased with proband age, and the youngest probands had the largest proportion of living relatives (78% for probands aged 55 years versus 59% for probands aged 66).

To date, 1,587 living relatives have been invited to participate, and 57% have agreed to do so (Table 6). Females were more likely to participate than males, and non-familial (sporadic) cases were least likely to participate. Rural/non-rural region and degree of relative were not significantly associated with family member participation.

Discussion

The OFCCR is the first population-based family colorectal cancer registry to be developed within Canada. Since this is a novel undertaking, there are no published reports with which to compare the OFCCR. However, our response rate was similar to, though slightly lower than, that of the companion Ontario Familial Breast Cancer Registry.[10] Obtaining high response rates in familial cancer

TABLE 2
Physician response (consent)

Total colorectal cancer cases	3,290
Unable to contact physician	– 187
Non-eligible patients (e.g. deceased)[a]	– 294
Eligible cases with a physician contacted	2,809
Physician consent provided	**2,613 (93%)**

[a] 267 cases were deceased (8% of total cases).

TABLE 3
Characteristics of responders and non-responders to the family history questionnaire (FHQ)

	Responders *n*[a] (%)	Non-responders *n*[b] (%)	*p* value[b]
Total number mailed FHQ[c]	1,395 (61)	894 (39)	N/A
Age group (years)			
55	317 (23)	209 (23)	
56–65	437 (31)	279 (31)	$p = 0.9$
66	641 (46)	406 (45)	
Sex			
Male	834 (60)	506 (57)	$p = 0.2$
Female	561 (40)	382 (43)	
Region[d]			
Rural	285 (20)	150 (17)	$p = 0.03$
Non-rural	1,110 (80)	744 (83)	
Tumour stage[e,f]			
I–III	588 (86)	338 (85)	$p = 0.8$
IV (metastatic)	93 (14)	61 (15)	

[a] Numbers may not add to total due to missing values.
[b] Pearson chi-squared *p* value reported.
[c] Note: 56 "unable to contact", 161 deceased (proxies not currently sought) and 107 ineligible cases were excluded.
[d] Rural/non-rural regions were defined using the patient's postal code.
[e] Only colon cancer cases staged (It was not possible to stage 400 patients because the necessary medical records were not available.)
[f] Determined using the tumour/nodal/metastasis (TMN) system (based on combined clinical and operative/pathological information available within 4 months of diagnosis)

TABLE 4
Distribution of participating colorectal cancer cases (probands) by familial risk[a]

Familial risk status[a]	*n*	(%)
High (HNPCC)	27	(2)
Intermediate/other	444	(32)
Low (sporadic)	924	(66)

[a] Defined in Table 1

TABLE 5
Family size (first-degree relatives) by familial risk status[a] and age group

	Proband (case) *n*	First-degree relatives		
		Mean (±SD)	Total	Alive (%)
Total sample	535	8 (±4)	4,284	2,821 (66)
Familial risk status[a,NS]				
High	24	9 (±5)	213	142 (67)
Intermediate/other	360	8 (±3)	2,880	1,897 (66)
Low (sporadic)	151	8 (±3)	1,191	782 (66)
Age group*				
55	131	7 (±3)	918	719 (78)
56–65	159	8 (±3)	1,257	862 (69)
>65	245	9 (±4)	2,109	1,240 (59)

[a] Defined in Table 1
[NS] No statistically significant difference between groups (ANOVA, $p = 0.43$)
* Statistically significant difference between groups (ANOVA, $p < 0.01$)

TABLE 6
Characteristics of relatives who participate/do not participate in additional phases of the OFCCR

	Participants *n* (%)	Non-participants *n* (%)	*p* value[a]
Total number of relatives invited	904 (57)	683 (43)	N/A
Relation to proband			
First degree	631 (70)	451 (66)	
Second degree	123 (14)	109 (16)	$p = 0.25$
third degree	150 (17)	123 (18)	
Sex			
Male	383 (42)	364 (53)	p 0.01
Female	521 (58)	319 (47)	
Familial risk status[b]			
High	91 (10)	86 (13)	
Intermediate/other	714 (79)	487 (71)	p 0.01
Low (sporadic)	99 (11)	110 (16)	
Region[c]			
Rural	159 (18)	132 (19)	$p = 0.38$
Non-rural	745 (82)	551 (81)	

[a] Pearson chi-squared *p* value reported
[b] Defined in Table 1
[c] Rural/non-rural regions were defined using postal codes.

research is especially challenging because of the potential for patients' concerns regarding confidentiality of sensitive information and family members' well-being[5] as well as the time commitment required of participants.

Recent US population-based case-control studies evaluating gene–environment interactions have reported response rates from colorectal cancer patients of approximately 65%,[11,12] only slightly higher than those of the OFCCR. The only population-based colorectal cancer study conducted in Ontario in the past decade achieved a similar physician response rate but had a 17% higher colorectal cancer patient response rate than the OFCCR (L Marrett, Cancer Care Ontario, personal communication, 2000). The OFCCR patient response rate is likely lower than these rates because of additional study requirements and concerns regarding the involvement of family members. Future research is needed to identify methods of overcoming such barriers to participation.

Response bias arising from differences in characteristics between participants and non-participants is always a concern in epidemiologic studies when response rates are low, as it may lead to biased estimates of prevalence and association.[13] However, response bias may be less of a concern for some genetic linkage studies, because it has been reported that the ability to detect major genes may not be markedly affected by response bias.[14] Estimates of gene frequency or penetrance based on family registries could be biased if families carrying the gene participated differentially according to the prevalence of cancer in their family. In addition, epidemiologic and gene–environment studies could result in biased measures of association if participation in the registry were differential regarding both case status and exposure(s) of interest.[13] In the OFCCR, however, it seems unlikely that there would be major bias regarding age and sex, since the distribution of proband participants and non-participants was similar across these characteristics.

Family member participation rates differed slightly by familial risk status, suggesting that proband participation may be differential across familial risk groups. The importance of known differences between proband participants and non-participants, for example, in rural/

urban residency and survival rate (since proxies are not currently sought) as well as in other factors such as smoking, education or diet (which could not be assessed), will depend on the hypothesis under study. Each future study or analysis based on the registry will need to evaluate the likelihood of bias with regard to the specific hypothesis under study.

As expected from previously published studies,[3,4] only a small proportion of colorectal cancer cases were classified as being from a high familial risk (HNPCC) family. However, the large total number of cases participating in the OFCCR will ensure that there will be a substantial number of families of high/intermediate familial risk, and this will facilitate genetic linkage studies, penetrance studies and the investigation of gene–environment interactions. Furthermore, it is possible that the sample size of future studies may be increased through collaboration with the five other US and Australian registries participating in the Co-operative Familial Registry for Colorectal Studies. Thus, the OFCCR offers exciting opportunities for the study of genetic and environmental factors associated with colorectal cancer, as well as providing a resource for the development of chemoprevention trials, cohort studies and gene discovery projects.

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Book Review

The Burden of Disease Among the Global Poor: Current Situation, Future Trends, and Implications for Strategy

By Davidson R Gwatkin and Michel Guillot
Washington (DC): The World Bank, 2000;
vii + 44 pp; ISBN 0-8213-4619-9

This new World Bank publication commences with a discussion of the importance of information on the burden of disease that is specific to the poor. It takes the position that the recent identification of the rapidly increasing non-communicable disease (NCD) burden in the "global" population lacks "congruity" in that "the poor" (in all settings) are more liable to be affected by communicable diseases than are "the rich."

To address this deficiency, the authors compare disease burden among the 20% of the global population living in countries with the lowest per capita incomes, with similar estimates for the 20% living in countries with the highest per capita incomes. They conclude that 59% of deaths and disabilities "among the poorest 20%" are due to communicable diseases, whereas 85% among the world's "richest" are due to NCDs. Then they analyze the implications of disease reduction strategies during the 1990–2020 period for the world's poorest and richest 20%. They conclude that a fast decline in communicable diseases would decrease the poor–rich gap by 2020, but an accelerated rate of overall decline in NCDs would actually widen the gap. The authors close with sections dealing with interpretations and implications for strategy.

The work is said to have been conducted to help guide the World Bank's recent health policy, which gives highest priority to improvements in the health, nutrition and population status of the world's poor. While the authors acknowledge some conceptual and methodological problems, the fact that it comes out under the authority of the World Bank requires not only that we must pay attention to it, but also that we must subject it to critical standards of review.

The impetus for the publication was the study on the global burden of disease edited by Murray and Lopez, which concluded that by 1990 NCDs had overtaken communicable diseases as the leading cause of mortality worldwide (56% of all deaths, not including injuries which then accounted for 10%, the remaining 34% attributable to communicable diseases). By the year 2020, NCDs were projected to account for 73% of global mortality, with communicable diseases declining to 15%. This analysis of trends, incorporating the same DALYs methodology used in the publication under review, has been widely disseminated.[1]

In essence, Gwatkin and Guillot offer an aggregate comparison of the world's poorest countries with the world's richest countries. While the language throughout refers to "the poorest 20%" and "the richest 20%", the reader must remain alert to the fact that this refers not to people but to countries. The countries are not listed, but one assumes that we are comparing the likes of Rwanda, Haiti and Afghanistan with countries such as Japan, Switzerland and the United States.

Nowhere is the situation within countries actually examined, so that the approach must entail a major risk of ecological fallacy (defined in the dictionary of the International Epidemiological Association as "the bias that may occur because an association observed between variables on an aggregate level does not necessarily represent the association that exists at the individual level"[2]). This bias is compounded by an analysis (based on lower and upper quintiles) that effectively excludes 60% of the world's population (the second to fourth quintiles), most of whom live in developing countries.

The authors view the emergence of NCDs in less developed countries as a function primarily of population aging and of progress against communicable disease in infants and children. While these are indeed powerful forces, there is no recognition here of the power of globalization that is forcing rural-urban shifts and associated trends in determinants which, in turn, are driving epidemiologic transitions. The poor are less able to resist the negative consequences of these trends. In terms of solutions, there is no apparent recognition of life cycle, family centred or community-based approaches to prevention, nor of the potential to develop cost-effective approaches (including non-pharmacological) within integrated systems of primary health care.

In support of their interpretation of prevention and cost-effectiveness issues, the authors rely heavily on the

highly respected textbook *Disease Control Priorities in Developing Countries*, published for the World Bank in 1993.[3] The reviewer would refer to this also for certain purposes, but in terms of a fair comparison between communicable diseases as a whole versus NCDs as a whole, it is notable that the emphasis is on primary prevention for the former, with relatively more emphasis on secondary prevention and palliation for the latter. Therefore, it should not be surprising that an aggregate analysis ends up favouring communicable disease interventions generally over those for NCDs.

Arguably, addressing the root causes of poverty itself would do far more for the health of the global poor—whether or not they are now suffering from communicable or non-communicable diseases (and they are suffering from both)—than encouraging the view that to address the emergence of non-communicable diseases at this stage is not appropriate. Dividing the decision framework into communicable and NCDs is simplistic, and may even be a legacy of traditional medical thinking.

Instead, why does the World Bank not base its decisions on an assessment of disease burden, prevention effectiveness and cost-effectiveness of particular interventions, regardless of whether something is or is not communicable? Measures such as tobacco control, improved diet and physical fitness, education about care seeking and even promoting quality of care where service is already being provided are actually quite feasible in many developing countries. However, these approaches to NCDs are not well recognized in the document or its sources. For example, injury prevention is one of the most cost-effective opportunities for reducing disease burden across all income groups in all countries, and one in which the Bank has invested virtually nothing to date.

The role of the World Bank in international health has been increasing over the past decade, even eclipsing that of the World Health Organization in many respects, and the need for its participation in health policy development is undeniable. However, this publication, in which communicable diseases and NCDs are juxtaposed as if this is a dichotomous choice that has to be made in developing countries, is contentious and has little do with what is actually happening on the ground.

When people lose their health in an urban squatter community or in a poor rural village, should we differentiate their priority in terms of whether the condition is communicable or non-communicable? After all, within each category there are examples of interventions that are cost-effective and of others that are not. While the authors fill an important gap in the debate with this document, clearly it is a debate that must go on, as this analysis also lacks "congruity". Equally, the literature on prevention effectiveness generally needs to be more fully developed, taking into account a wider range of possible solutions.

In conclusion, this World Bank publication is an important contribution to the field of international health, not only because of its radical approach to analyzing the challenge of the "double burden" of disease that confronts developing countries, but also because it comes from an institution that is enormously influential on policy makers, especially those who are associated with international aid agencies. I would recommend that it become required reading in all graduate programs in the field of public health, in view of the inevitable debate that it should generate, not only in terms of its methodology but also in terms of the strengths and limitations of its analysis, interpretations and conclusions.

Overall rating:	Controversial
Strengths:	Reveals current World Bank thinking on the priority to be accorded to NCDs
Weaknesses:	Crude approach to analysis and subject to a range of inherent biases
Audience:	To be read critically by students, scholars and decision makers in international donor agencies

References

1. Murray CJL, Lopez AD, editors. *The global burden of disease — summary*. Cambridge (MA): Harvard School of Public Health (on behalf of the World Health Organization and the World Bank), 1996.
2. Last JM, editor. *A dictionary of epidemiology*. 3rd edition. New York: Oxford University Press (for the International Epidemiological Association), 1995.
3. Jamison DT, Mosley WH, Measham AR, Bobadilla JL, editors. *Disease control priorities in developing countries*. Oxford University Press (for the World Bank), 1993.

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New Resource

Health Effects of Interactions Between Tobacco Use and Exposure to Other Agents

Environmental Health Criteria, No 211
World Health Organization, 1999; xx + 149 pp
(English, with summaries in French and Spanish);
ISBN 92-4-157211-6; Order no 1160211

This book evaluates the findings of close to 600 studies aimed at determining whether the health risks associated with tobacco use are enhanced by co-exposure to numerous chemical, biological and physical agents commonly found in the workplace. Co-exposures in the domestic and general environment, which are especially important in newly industrializing countries, are also considered in this comprehensive review. Although all forms of tobacco use are covered, particular attention is given to risks arising from exposure to mainstream and side-stream smoke from cigarettes.

The book has four chapters. The first summarizes what is known about the health risks caused by tobacco use. A brief overview of the history of tobacco use is followed by a detailed explanation of the chemistry of processed tobacco and the many toxic compounds found in tobacco and in mainstream and side-stream smoke. The chapter also includes an overview of all documented acute and chronic adverse effects, including chronic obstructive lung disease, chronic bronchitis, small airways disease, emphysema, pulmonary fibrosis, many forms of cancer and effects on the cardiovascular system. The chapter concludes with a review of evidence demonstrating the health hazards of smokeless tobacco.

The second and most extensive chapter evaluates the evidence on health effects caused by interactions between tobacco smoke and asbestos, non-asbestos fibres, seven inorganic chemicals, five organic chemical agents (including ethanol), four physical agents and seven biological agents (including two widespread infectious agents). The chapter also explains the concept of interaction and how it can be measured, discusses vector effects, whereby cigarettes become contaminated with toxic chemicals in the workplace, and reviews data indicating that tobacco smoking can alter the metabolism of therapeutic drugs and other chemicals.

Chapter three considers whether adverse effects following co-exposure to tobacco smoke and other agents are separate effects or possible interactions. The evaluation draws on data from studies of coal mining, other mineral dusts, fibrous minerals, metals, pesticides and exposure in the rubber and petroleum industries.

The report found evidence for synergism in the production of adverse effects, including cancer, between tobacco smoking and exposure to asbestos, ethanol, silica and radiation. It also found evidence that tobacco smoking affects the health risks of exposure in coal mining, pesticide handling and in the rubber and petroleum industries. In addition, tobacco smoking can increase the risk of byssinosis produced by exposure to cotton dust and of nasal cancer caused by exposure to wood dusts.

On the basis of this evaluation, the final chapter concludes that all possible measures should be taken to eliminate tobacco use, particularly smoking. To avoid interaction with occupational exposure and to eliminate hazards arising from exposure to environmental tobacco smoke, smoking in the workplace should be prohibited. Moreover, since smoking can result in altered responses or adverse reactions to drugs and other treatments, appropriate dose adjustments and patient surveillance should be taken into consideration by clinicians.

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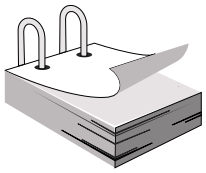

Calendar of Events

August 6–11, 2000 **Chicago, Illinois** **USA**	11th World Conference on Tobacco OR Health Hosts: American Cancer Society, American Medical Association and Robert Wood Johnson Foundation	11th World Conference on Tobacco OR Health c/o American Medical Association 515 North State Street Chicago, IL USA 60610 Attn: Anne Jenkins, Conference Manager Tel: (312) 464-9059 Fax: (312) 464-4111 E-mail: 11thwctoh@ama-assn.org <www.wctoh.org>
August 23–27, 2000 **Victoria, British Columbia**	ITCH 2000: "From Potential to Practice" International Conference on Information Technology in Community Health	ITCH 2000 c/o School of Health Information Science University of Victoria PO Box 3050, STN CSC Victoria, BC V8W 3P5 Tel: (250) 721-8576 Fax: (250) 472-4751 E-mail: itch@hsd.uvic.ca <www.itch.uvic.ca>
September 2–6, 2000 **Beijing, China**	"Challenges for Public Health at the Dawn of the 21st Century" 9th International Congress of the World Federation of Public Health Associations (WFPHA) Hosted by China Preventive Medicine Association and Chinese Academy of Preventive Medicine	WFPHA Secretariat c/o American Public Health Association Tel: (202) 777-2487 Fax: (202) 777-2534
October 11–14, 2000 **Vancouver, British Columbia**	"Suicide Prevention in Canada: Exploring Our Diverse Landscape" 11th Annual Conference of the Canadian Association for Suicide Prevention	Suicide Prevention Information & Resource Centre Tel: (604) 882-0740
October 22–25, 2000 **Ottawa, Ontario**	"Health for All in the Year 2000" Canadian Public Health Association 91st Annual Conference *and* Ontario Public Health Association 51st Annual Conference	CPHA Conference Services 400 – 1565 Carling Avenue Ottawa, Ontario K1Z 8R1 Tel: (613) 725-3769 Fax: (613) 725-9826 E-mail: conferences@cpha.ca <www.cpha.ca>
October 30–November 1, 2000 **Vancouver, British Columbia**	"The Preventive Dose — Is Your Community Getting Enough?" 13th Canadian Heart Health Network Meeting Sponsors: Health Canada, Heart and Stroke Foundation of Canada, BC Ministry of Health and Ministry Responsible for Seniors, Heart and Stroke Foundation of BC & Yukon *Abstract deadline: August 31, 2000*	Dr PJ Naylor Cardiovascular Disease Prevention Unit BC Ministry of Health 1520 Blanshard, 2nd Floor Victoria, BC V8W 3C8 <www.heart-health.org>

November 29–December 1, 2000 **Washington, DC** **USA**	"Living Healthier, Living Longer: The Will and the Way" 15th National Conference on Chronic Disease Prevention and Control Sponsors: Centers for Disease Control and Prevention (CDC), Association of State and Territorial Chronic Disease Program Directors (ASTCDPD) and Prevention Research Centers Program	Estella Lazenby The KEVRIC Company, Inc. Silver Spring Metro Plaza One 610 – 8401 Colesville Road Silver Spring, MD USA 20910 Tel: (301) 588-6000 <www.cdc.gov/nccdphp> <www.astcdpd.org>
May 13–18, 2001 **Toronto, Ontario**	9th International Women and Health Meeting York University Campus	Monica Riutort, Coordinator Canadian Planning Committee Tel: (416) 323-6249 Fax: (416) 323-7318 E-mail: monicari@web.net
June 13–16, 2001 **Toronto, Ontario**	Congress of Epidemiology 2001 Combined meeting of American College of Epidemiology, American Public Health Association (Epidemiology Section), Canadian Society for Epidemiology and Biostatistics and Society for Epidemiologic Research	<www.epi2001.org>
July 1–6, 2001 **Vancouver, British Columbia**	"Global Aging: Working Together in a Changing World" 17th Congress of the International Association of Gerontology *Abstract deadline: December 31, 2000*	Congress Secretariat Gerontology Research Centre Simon Fraser University 2800 – 515 West Hastings Street Vancouver, BC V6B 5K3 Tel: (604) 268-7972 Fax: (604) 291-5066 E-mail: iag_congress@sfu.ca <www.harbour.sfu.ca/iag>

Population Health Researcher – Cancer

Division of Epidemiology, Prevention and Screening
Alberta Cancer Board

The Alberta Cancer Board is the provincial agency responsible for the coordination of cancer prevention, early detection, treatment and supportive care, and it places a high value on research to underlie all of its activities. The Division of Epidemiology, Prevention and Screening includes the Scientific Research Group, the Alberta Cancer Registry (a population-based registry of all cancers in the province), the Provincial Breast Screening Program and several community prevention initiatives. Alberta provides a dynamic health research environment, and the Alberta Cancer Board has recently inaugurated fund-raising for a long-term cancer research endowment.

The Alberta Cancer Board invites applications for a full-time position in population health research in cancer for the Division of Epidemiology, Prevention and Screening. The Division conducts population-based research in cancer epidemiology, surveillance and modelling, in behavioural aspects of cancer prevention and screening, and in utilization of preventive and screening strategies. We are seeking scientists whose interests fall in one or more of the above areas, or complementary areas in cancer control research. This position offers an excellent opportunity to develop an independent research program within a multidisciplinary environment.

Applicants should have a PhD or MD with additional research training. These graduate degrees should be in appropriate fields of research. The selected candidate will receive core funding support, but will be encouraged to seek salary and grant support from external agencies such as the Alberta Health Foundation for Medical Research, the National Health Research and Development Program and/or the Medical Research Council of Canada. If successful in attaining external funding, additional benefits will be provided by the Alberta Cancer Board.

Collaboration will be encouraged with colleagues working in cancer etiology, prevention, early detection and surveillance, as well as with other scientists and clinicians at the Alberta Cancer Board and the Universities of Alberta and Calgary. Appropriate adjunct appointments within University departments will also be sought.

In accordance with Canadian immigration requirements, this advertisement is directed to Canadian citizens and landed immigrants; however, others are encouraged to apply to the address below.

Dr H Bryant, Director, Division of Epidemiology, Prevention and Screening
Alberta Cancer Board, 3330 Hospital Drive NW, Room 382, Calgary, Alberta T2N 4N1

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