Chronic Diseases in Canada

Volume 23, number 1, 2002

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Chronic Diseases in Canada

a publication of the Population and Public Health Branch, Health Canada

Chronic Diseases in Canada (CDIC) is a quarterly scientific journal focusing on current evidence relevant to the control and prevention of chronic (i.e. non-communicable) diseases and injuries in Canada. The journal publishes a unique blend of peer-reviewed feature articles by authors from the public and private sectors that may include research from such fields as epidemiology, public/community health, biostatistics, behavioural sciences and health services. Authors retain responsibility for the content of their articles; the opinions expressed are not necessarily those of the CDIC Editorial Committee or of Health Canada.

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Indexed in *Index Medicus*/MEDLINE, PAIS (Public Affairs Information Service) and EMBASE, the Excerpta Medica database.

This publication is also available online at http://www.hc-sc.gc.ca/pphb-dgspsp/publicat/cdic-mcc/index.html

Published by authority of the Minister of Health

ISSN 0228-8699

Aussi disponible en français sous le titre *Maladies chroniques au Canada*

Evaluation of a risk factor survey with three assessment methods

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Abstract

This paper describes the evaluation of questions on a cancer risk factor survey using three different methods: dataset response patterns, qualitative feedback, and questionnaire appraisal. These methods addressed the survey data, procedures and questions. The three methods identified similar issues, but also made unique contributions. Dataset response patterns showed missing and out-of-range data, an order effect, and mixed coding. Qualitative feedback revealed lack of clarity, sensitive topics, technical or undefined terms, failure to hear all response options, overlapping response options (as perceived by respondents), coding problems and recall difficulties. Questionnaire appraisal showed technical or undefined terms, complex syntax, hidden definitions, and ambiguous wording. The survey assessment methods described here can improve data quality, especially when limited time and resources preclude in-depth questionnaire development.

Key words: *data collection; health surveys; population surveillance; questionnaires*

Introduction

This paper describes the evaluation of cancer risk factor survey questions through the application of three different assessment methods to the data, process and questions from a pilot survey. We report each method's unique contribution, and areas where the different approaches converge, in identifying areas for improved data collection or caution in interpreting responses.

Newell and co-authors, in reviewing the accuracy of self-reported cancer-related health behaviours, suggest strategies that include ensuring that respondents fully understand questions, phrasing questions to minimize social desirability bias, encouraging exact rather than rounded-off answers for continuous variables, and ensuring that questions have clear, exhaustive, mutually exclusive response options.[1] Investigators collecting and using survey data need mechanisms to assess attempts to implement these strategies.

Rapid risk factor surveillance systems offer opportunities for ongoing evaluation and ideally offer some flexibility in introducing changes to questions. A pilot test of such a system, carried out in Durham Region, Ontario, provided an opportunity for the assessment described here. The pilot was a collaboration between Health Canada, the Ontario Ministry of Health and Longterm Care, Durham Region Public Health Unit, and Cancer Care Ontario (CCO).

Materials and Methods

The Durham Region pilot survey

The pilot survey was designed to test collaboration among the sponsoring agencies, including the process of formulating, adding and changing survey content, and to determine whether survey data could be generated quickly in a useful format. Actual survey results and quality evaluation were secondary aspects. Interviews were held in five monthly waves of approximately 200 each in June through October 1999, resulting in 1,047 completed interviews with Durham Region residents aged 18 through 89. Of the eligible individuals contacted, 69% completed the interview. The Institute for Social Research (ISR) at York University, Toronto, was contracted to conduct the survey using Computer Assisted Telephone Interviewing (CATI). The members of the content development group, three epidemiologists and two survey methodologists, represented Durham Region Health Department, CCO, Health Canada and ISR. Content was limited to approximately 80 questions for a target average interview length of 20 minutes.

Cancer risk factor questions

Content areas of particular interest to a provincial cancer agency were addressed in 45 questions about 1) sun-related behaviour; 2) screening for breast, cervical, colorectal and prostate cancer; 3) diet; 4) physical activity; 5) tobacco consumption. The Appendix shows these in their final (fifth survey wave) form.

Questions on sun-related behaviour were adapted from those developed for surveys at the 1998 Canadian National Workshop on Measurement of Sun-Related Behaviours.[2] Slight changes in this group's wording were made by our content group's survey methodologists, based on their knowledge and experience of telephone surveys.

Questions on screening for breast, cervical, colorectal and prostate cancer all used the same format about 1) ever being tested, 2) time since last test and 3) reason for last test. Breast and cervical cancer questions were from the National Population Health

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Survey; the content group developed prostate and colorectal cancer screening questions. The reference period of two years since last test reflected breast and cervical screening guidelines. Mammogram questions were asked of women aged 35 and older, and colorectal screening questions of respondents 40 and older. Pap test questions were restricted to women who reported not having had a hysterectomy. Questions on prostate-specific antigen (PSA) tests had no age restriction because some men in the pretest reported PSA testing in their 30s. Questions about reasons for cancer detection tests were expanded from the US Behavioral Risk Factor Surveillance System (BRFSS)[3] question about Pap smears (routine examination or to check a current or previous problem) to include a third response option to distinguish between concern about symptoms and follow-up of a medically diagnosed problem.

To address diet, we incorporated a set of BRFSS questions on fruit and vegetable consumption.

Physical activity questions were adapted from a set proposed for the BRFSS by the Centers for Disease Control and Prevention's Physical Activity and Health Branch.

Tobacco consumption questions were those used in the BRFSS (1999)[3] with minor changes to reflect Canadian experience and to capture quit attempts.

Evaluation methods

We evaluated the 45 questions on cancer risk factors using 1) analysis of traditional dataset descriptors and response patterns; 2) qualitative feedback from interview monitoring, interviewer debriefing, and direct questions to respondents; 3) questionnaire appraisal with a checklist, modified from a published questionnaire coding system, to describe and assess potential problems related to comprehension or response generation.[4]

Dataset descriptors and response patterns

Data characteristics alone can yield substantial information on question quality. For instance, a substantial number of refusals to answer a particular question may indicate a sensitive topic that could be dropped or the need to reword the question; unexpected answers may mean that a question is being misunderstood. Response patterns used to assess the quality of this set of questions were appropriate adherence to skip patterns, the proportion of refusals or "don't know" responses, the range of responses, and ease of analysis. An apparent order effect in days of vigorous and moderate activity was tested with a chi-squared statistic on three degrees of freedom.

Qualitative feedback

Qualitative analysis of text compiled from three activities (interview monitoring, interviewer debriefing and respondent feedback) revealed themes in question and interview attributes that indicated potential problems with the survey data. ISR's equipment enables switching among interviews undetected by interviewers and respondents. Four pilot survey investigators monitored interviews by telephone and computer on separate evenings during wave three. Three investigators debriefed interviewers and supervisors together after completion of all five waves. Respondent feedback was sought with two questions at the end of the interview in the two final waves, which included 412 respondents. Interviewers first asked whether any questions had been confusing or difficult to understand and, if yes, which questions. Cancer risk factor questions were difficult in four instances: three people had difficulty with the physical activity questions, and one said "the food questions" were confusing. Interviewers then asked all 412 respondents whether there were questions they understood but still found difficult to answer. One respondent reported trouble in answering the physical activity question, four the fruit and vegetable questions and one the reason for a Pap test.

Questionnaire appraisal

Lessler and colleagues have developed a scheme for coding questions, response options and instructions to characterize the mental burden involved in responding to a questionnaire.[4,5] Its purpose is to identify features that may affect question comprehension and interpretation, response accuracy and willingness to respond. In adapting their scheme we excluded items relating to attitude rather than behaviour, and items that we felt would require a cognitive interview. (Cognitive interviews use various techniques for investigating the mental information processing necessary to respond to questions.)

We then fine-tuned the coding scheme by independently coding three questions, comparing results, and achieving consensus on coding definitions and on elements inappropriate for our risk factor questionnaire. One author (JF) then coded all the questions using the resulting refined scheme.

Results

Dataset descriptors and response patterns

Skip patterns were appropriate, with minor exceptions. A few males were asked female cancer screening questions because interviewers asked respondents all questions when they could not determine sex from a person's voice. (If still in doubt, interviewers asked directly whether respondents were male or female at the end of the interview.)

None of the questions evaluated had more than 1.5% refusals. Ten questions had more than 10% "don't know" responses; all were to questions requiring detailed recall about time or frequency, such as hours spent in the sun, time since screening, or frequency of fruit and vegetable consumption.

Responses sometimes did not match questions as asked. For example, four respondents reported an answer of less than 10 minutes to the question "On days when you do moderate activity for *at least* 10 minutes at a time, how much total time do you spend doing these activities?". Others seemed unlikely or extreme (more than eight hours of vigorous physical activity a day, smoking 90 cigarettes a day, PSA testing 24 years ago).

FIGURE 1
Days per week respondents reported engaging in vigorous or moderate physical activity, according to order of defining activity levels (Durham Region pilot risk factor survey, Ontario, 1999)

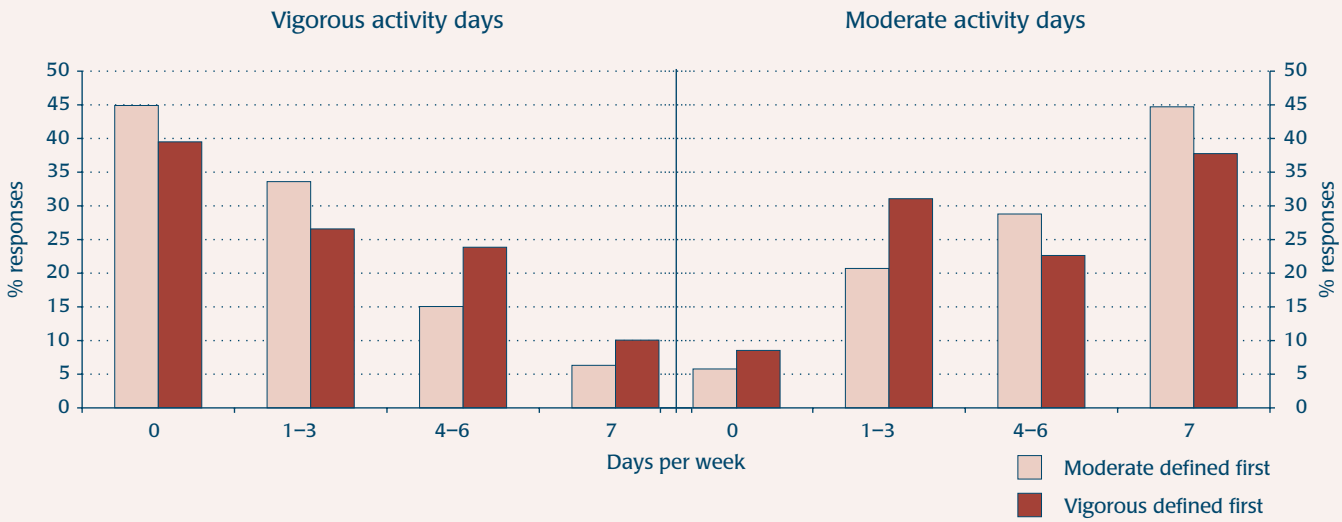

Figure 1 shows an order effect in the distribution of the number of days per week respondents reported that they engaged in vigorous or moderate activity. Although the preamble to the physical activity questions told respondents they would be asked about participation in vigorous and moderate activity, "vigorous" and "moderate" were not defined until each question was read. In waves two and three respondents were asked first about the number of days per week they engaged in vigorous activity, and then about moderate activity. The order was reversed for waves four and five. The first definition heard by respondents may have become a reference point for answering the second question. As a group, respondents reported engaging in vigorous activity on more days when the reference point (the first definition) was vigorous activity and on fewer days when the reference point was moderate activity ($p = 0.003$). Similarly, they reported engaging in moderate activity on more days when the reference point was moderate activity and on fewer days when the first definition was vigorous activity ($p = 0.001$).

Analytic difficulty arose from combined response options for three types of questions. Response coding options for physical activity were a mixture of continuous and categorical: <enter the number of MINUTES> or <more than 8 hours>. Average time spent exercising cannot be calculated because of the <more than 8 hours> categorical response unless either these respondents are removed from the calculation, or some assumption is made regarding the distribution of these values. Similarly, the question about time in the sun mixed numeric and text response fields. Interviewers were instructed to code responses as answered either in minutes, hours or a combination of the two. While responses in minutes or whole hours were recorded as numeric values, combination responses ("an hour and a half", for example), were recorded as text, which then had to be converted to numeric form (1.5 hours) and manually entered into the numeric field for combining with numeric responses. Partial answers to fruit and vegetable consumption resulted in missing or excluded information when daily, weekly or monthly consumption was reported but quantity could not be recalled: 14.6% of respondents were unable to estimate amounts for their daily, weekly or monthly consumption of at least one fruit and vegetable category. Analysis attempts also revealed missing "zero" values when some responses were contingent on answers to preceding questions. For instance, when respondents said "no" to the question asking whether they engaged in moderate or vigorous activity for at least 10 minutes at a time, the CATI system was programmed to skip the following question asking how many days they engaged in such activity, but was not programmed to enter "0" for physical activity days. Compensating for this oversight required some vigilance before data analysis reflected actual reporting.

Qualitative feedback

Four major themes emerged: stylistic problems, sensitive questions, question clarity and response validity.

Stylistic issues were noted during interview monitoring and interviewer debriefing. Investigators monitoring interviews were concerned that some interviewers' monotone and rapid pace might interfere with question comprehension or lead to respondent frustration, although they did not detect any such frustration. Both interviewers and investigators felt the need for more transitional statements, particularly before such sensitive topics as tobacco use and (for interviewers) colorectal and women's cancer screening. In addition, investigators heard inaccurate or incomplete explanations from interviewers in response to questions about the purpose of

the survey, how results would be used, and the reasons for randomly selecting respondents.

Topics noted as sensitive during interview monitoring and interviewer debriefing were not necessarily mentioned in respondent feedback. Both interviewers and investigators observed respondent defensiveness about weight and tobacco consumption. Interviewers also felt the colorectal screening question was sensitive. Respondents, on the other hand, were more likely to report that the survey questions about income and education were too personal; only one mentioned weight as uncomfortably personal, and none reported discomfort with cancer screening questions.

Problems with question clarity were noted from all three qualitative sources. Both interviewers and investigators felt that some questions were open to misinterpretation. In some cases this was related to either undefined or unfamiliar terms. Interviewers reported, for instance, that many respondents apparently thought PSA was a routine blood test, and gave a potentially invalid "yes" response; investigators noted that definition was an issue for some fruit and vegetable questions (some respondents had trouble understanding "green salad", for instance). One respondent reported that definitions of moderate and vigorous physical activity were not clear enough to distinguish them. In other cases, question intent was unclear. For instance, interviewers felt that the sun avoidance question might need clarifying if meant to capture moving purposely "out of the sunlight" as opposed to "out of the heat", and that "clothing with long sleeves" would better capture covering-up behaviour than asking specifically about a "shirt".

Interview monitoring identified questions to which respondents offered answers before all response options had been read or terms defined. Questions incorporating lists of responses (reasons for cancer screening tests, for example) needed rewording to signal clearly that a list was coming; definitions of "moderate" and "vigorous" in the physical activity questions needed to be placed so that respondents heard them before offering an answer.

Difficulties in interpreting some response categories could result in misclassification. As a reason for screening tests, interviewers described some respondents answering "concerned that I might have a problem", yet saying that this was routine screening. Only one respondent singled out a screening question as difficult to answer; she had a Pap smear "because I was having my tubes tied" and didn't see how this fit the offered response options.

Investigators noted that interviewers had difficulty appropriately coding some responses to questions about time in the sun and fruit and vegetable consumption. Some interviews showed the necessity of providing coding instructions, for instance, about what counts as "fruit" or "fruit juice" when questions have been used from another survey. (One respondent in a monitored interview asked whether apple juice counted in response to a question from the BRFSS about "fruit juices such as orange, grapefruit, or tomato").

Respondents reported trouble in answering some questions because behavioural details were difficult to report correctly (time exercising, vegetable consumption) or because the question did not ask for a response about a specific time period (vegetable consumption).

Questionnaire appraisal

Table 1 summarizes the results of applying the questionnaire coding scheme to the 45 cancer risk factor questions. The scheme scores the questions themselves, memory/judgement tasks required to answer the questions, and the responses.

Most questions asked about past rather than current behaviour. The frequency of carry-over and embedded reference periods reflects several question series asking for increasingly detailed information; for instance, "Have you ever had a mammogram?", then "Did you have your mammogram in the last 2 years?", and then "How many years/months ago was that?". Undefined reference periods were in questions about current behaviour. Ill-defined reference periods occurred in the physical activity questions. Between a quarter and a third of questions used technical terms, many undefined, or ambiguous wording, and/or complex syntax. Technical terms were usually screening test names; complex syntax was largely needed to clarify the wording of "moderate" and "vigorous" physical activity and the reference time and type of day for being outside on a sunny day.

Most memory retrieval and judgement tasks involved remembering an episode or set of episodes that included a blend of common habits, distinct habits, rare events and time estimates. Most questions required qualitative judgement, reflecting the large number of yes/no and categorical response options, whereas fewer questions required estimation of the actual number of times something happened or how long ago. Questions about cancer screening were coded as sensitive in this scheme because of the physically personal nature of breast, cervical, colorectal and PSA screening.

One major problem with response options was hidden definitions, information provided only if respondents requested clarification. Although most were for fruits and vegetables (respondents asked, for example, "Are potato chips vegetables?", "Does the fruit in a Pop Tart count?"), there were others throughout the questionnaire. The other response problem was the inclusion of ambiguous or vague terms, mainly in sun behaviour questions ("rarely" and "often") and in reasons for screening tests ("*routine* screening", "*ongoing* problem", "*concerned* about a problem").

Multimodal analysis

All three methods (dataset response patterns, qualitative feedback, and questionnaire appraisal) pointed to potential problems with response validity, respondent reluctance, and recall difficulty (Table 2). By validity we mean the extent to which responses were directed to the intent of a question and were correctly captured by interviewers. While potential problems were identified, usually through contributions unique to each method, there was convergence on the broad areas of sensitive topics (although respondents singled out different topics than interviewers, monitors and questionnaire coding), undefined technical terms,

TABLE 1
Cancer risk factor question coding: percentage (%)[a] of characteristics and potential problems

Questions	Memory/Judgement Tasks	Responses
Reference Set	**Retrieval Task**	**Response Description**
31% Current characteristic/behaviour	47% Remember episode	34% Yes/No
69% Past characteristic/behaviour	49% Remember set of episode	30% Qualitative: category
Potential Problems	4% Remember general information	2% Qualitative: ordinal
Terminology:	0% Remember previous answer	0% Qualitative: open
27% Technical term	**Memory Content**	0% Quantitative: count
24% Undefined technical term	7% General self knowledge	23% Quantitative: complex
29% Ambiguous or vague	91% Specific behaviour (or try)	20% Duration
Structure:	Class of behaviour	7% Time point
16% Hidden question	35% Common habit	0% Age
31% Complex syntax	16% Distinctive habit	**Potential Problems**
0% Several questions	40% Rare	*Instruction:*
0% Several definitions	47% Low volume	0% Hidden instructions
2% Unclear goal	16% High volume	20% Hidden definitions
0% Q/A mismatch	20% Time point/interval	*Terminology:*
9% Violates conventional conversation	**Type of Judgement Process**	9% Technical terms
Reference Period	20% Estimate total	7% Undefined terms
18% Lifetime	58% Determine +/- occurrence	25% Ambiguous/vague terms
9% 12 months	9% Determine date/onset	**Response Structure**
16% 30 days	0% Determine age	7% Boundary problems
0% Today	20% Estimate duration	9% Overlapping categories
33% Tied to behaviour/previous question	9% Estimate average	9% Missing categories
27% Undefined: e.g. currently	13% Complex estimation	
Potential Problems	**Information Integration**	
0% Unanchored boundary	0% Count	
0% Non-fixed boundaries	60% Qualitative judgement	
13% Ill-defined periods	40% Quantitative judgement	
20% Undefined period	**Potential Problems**	
16% Embedded period	*Information Evaluation:*	
29% Carry-over reference period	38% Sensitive (general)	
	0% Socially undesirable	

[a] The number of questions coded as having the specified characteristics or potential problems divided by the total number of coded questions (45).

Note: Because characteristics are not mutually exclusive, the % within a category may sum to greater than 100%

question clarity and hard-to-remember information. In our evaluation, only examination of the dataset revealed analytic difficulties associated with the responses as entered.

Discussion

Without critical assessment of survey data and the methods used to collect them, health agencies risk basing policy decisions on inaccurate information. Users of survey data know that self-reports are, to varying degrees, the result of imperfect recall, biased reporting[6] and misclassified responses. Within these limitations,

TABLE 2
Potential problems addressed by three assessment methods

Potential problems	Assessment method		
	Response patterns	Qualitative feedback	Questionnaire coding
Validity (clarity, response options)	Responses ■ out of range ■ unlikely ■ extreme Order effect	Undefined/unfamiliar terms Intent unclear Response options ■ unheard ■ overlapping ■ not exhaustive Response coding problems	Technical/undefined terms Vague wording Complex syntax Hidden definitions
Respondent reluctance	Refusals	Sensitive questions Tone and pace Transitions Survey explanations	Sensitive questions
Recall difficulty	"Don't know"	Hard to remember Prefer specified time period	Reference periods ill defined
Analytic difficulty	Mixed responses ■ categorical + continuous ■ numeric + text ■ frequency, no quantity		

Newell and co-authors emphasize the scope for improved data collection on health-related behaviours.[1] Each of the assessment methods we describe can offer insight into the data or opportunities for improvement. These methods identified problems not only in aspects of this pilot survey, but also in individual questions adopted from other surveys. In addressing the problems raised, investigators must often weigh the pros and cons of opposite approaches. In ongoing surveys, the benefits of changes may not outweigh the benefits of data comparability.

Dataset descriptors and response patterns are traditional evaluation tools; skip patterns, refusals, question-response mismatches and extreme responses indicate areas for cautious interpretation of data or, in ongoing surveys, for programming changes and interviewer instructions. Many refusals or "don't know" responses may identify sensitive or misunderstood questions. A high proportion of "don't know" responses or evidence of an order effect (if different orders have been tried) raises an alert about the validity of all responses to those questions. Responses outside the expected range show items for which programming to restrict allowable CATI entries or to prompt interviewers to repeat a question may improve data quality. The analytic difficulties of mixed continuous and categorical, or numeric and text, responses should be avoided unless theoretical reasons exist for including them. (There may, for instance, be arguments for grouping numeric responses above a certain threshold for some behaviours.) Similarly, although allowing respondents to select their own units of reporting presented analytic problems in our pilot survey, this must be weighed against the benefits of giving respondents the freedom to provide information at a level they feel is most accurate.

Qualitative feedback can point to possible areas for change. In this survey, interview monitoring identified areas where data quality could be improved through question rewording, additional interviewer training, or more comprehensive coding instructions. Monitoring can reveal particular wording requirements of a telephone interview, especially for investigators more familiar with self-administered or face-to-face questionnaires. In this ongoing pilot survey, for instance, we altered wording so that respondents would wait to hear a list of response options. Debriefing interviewers provides the experience of a wider range of interviews than investigators can monitor. Interviewers are especially aware of the usefulness of transitional statements to alert respondents that a personal question is coming, suggest that no personal judgements will be made, or generally "soften" the approach. For this pilot survey, interviewers requested definitions, described a response option problem, and noted areas where transitional statements would be helpful.

Despite the rich detail on potential problems that interviewer and respondent feedback provided, such information may need careful assessment before it prompts changes. Interviewer discomfort may be less informative than refusal or quit rates

in identifying topics sensitive enough to warrant changes in wording, transitions, or placement. In this survey, while only a small proportion of our wave four and five interviewees responded to the request for feedback, they did provide qualitative detail on the high proportion of "don't know" responses for activity time and fruit and vegetable consumption. Whether or not a questionnaire change is justified when comparatively few respondents are willing to lengthen the interview to provide negative feedback is a matter best decided in the context of the project as a whole. Different decisions may be made depending on, for instance, the importance of data comparability across survey waves or different surveys, or whether an ongoing survey is at an early or later phase.

Our questionnaire appraisal was exploratory and carried out after the pilot survey had been conducted. A more appropriate use would be to identify areas for change before field testing. We adapted another group's published scheme for application to a health behaviour interview. Our adaptation may need further revision for application to other questionnaires. As application of the codes necessarily involves individual judgement, another group intending to use the scheme will need to agree internally on item definitions (what constitutes a "technical term", for instance). More fundamentally, the published scheme that we adapted depends on the validity of the underlying models of the cognitive processes involved in question response.[4]

Intuitively, however, some form of checklist seems appropriate for indicating potential problems prior to any pretest in the field. Shorter lists have been published.[7,8] A coding scheme or checklist could be expanded to include aspects identified in this pilot survey through response pattern analysis (mixed categorical and continuous responses, for instance) or qualitative feedback (such as announcing a list of response options). An advantage of the list we used is that it aids choices in wording by quantifying different aspects of respondent burden. Analysis could show, for example, that a high proportion of questions required complex estimation on the part of respondents. These might be memory retrieval tasks unavoidable when reports of preventive health behaviours are required. In such a case those designing the survey might want to make other changes (dropping some questions, for instance) to compensate for this aspect of respondent burden. This is best done in the context of the survey as a whole, rather than trying to establish acceptable levels for respondent burden or potential problems. As with the other methods described here, changes must be weighed against new problems they might introduce or advantages that would be lost. Although complex syntax increases burden, for instance, it may be required in order to clarify questions and provide definitions. Another example is a reference period tied to a previous question; while this can reduce topic sensitivity by minimizing repeats of sensitive wording, extensive use could lead to response fatigue.

Ideally, survey questions are developed using focus groups, cognitive interviews and pretesting, or at least some of these, in the population targeted for the survey. Additional methods of assessing survey quality exist. Questionnaire responses can be compared, for instance, with food records or 24-hour food recall, pedometers or other physical activity monitors, mammogram reports in medical records, or responses to related questions within a survey. When limited time and resources preclude in-depth development and assessment, the combination of methods described here offers useful insights. They are equally important for questions adopted from other surveys, as differences in populations, questionnaire administration and question order can alter validity. Rapid risk factor surveys lend themselves especially well to these data quality measures: data are available quickly for quality assessment, and flexibility in altering question wording or transitions is likely to be a key principle. Such surveys may provide suitable frameworks for investigating order effects like the one revealed for physical activity in our Durham pilot survey, or for using more than one question for the same issue and assessing inter-item correlation. Again, insights gained from similar experiments in wording or order must be weighed against data comparability.

A multimodal approach like the one described here can confirm observations where the findings of different methods converge.[9] More important, when resources are scarce the use of these different methods can compensate for aspects missed by any single method and thus reveal a broader range of potential problems.

Acknowledgements

We thank Dr Bernard Choi and Dr Philippa Holowaty for their helpful comments on an earlier draft of this paper. David Northrup and his staff at the Institute for Social Research, York University, collaborated in this work by inviting investigators to monitor interviews, providing insightful feedback from interviewers, and suggesting the addition of questions for respondent feedback. Dr. Holowaty, Mr. Northrup and (originally) Dr. Margaret de Groh of Health Canada, with two of the authors (BT and LDM), worked as the content development group for the pilot survey.

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APPENDIX
Cancer Risk Factor Questions

Sun-related Behaviour Questions			
S1.	Now some questions about being outdoors. First we want to ask you about sunburns. By sunburn we mean any reddening or discomfort of your skin that lasts longer than 12 hours after exposure to the sun or other UV sources, such as tanning beds or sunlamps. During the last year, including the months of June, July and August last summer, as well as winter vacations, has any part of your body been sunburned?	<1> yes <5> no	<8> don't know <9> refused
S2.	Did ANY of the sunburns you had in the last year cause your skin to peel?	<1> yes <5> no	<8> don't know <9> refused
S3.	Did ANY of the sunburns you had in the last year cause blistering where the skin is pushed up and there is water underneath?	<1> yes <5> no	<8> don't know <9> refused
S4.	For the next question we want to ask you about spending time outside when it is MOSTLY sunny. Think about the last sunny day WHEN YOU WERE NOT AT WORK, such as a weekend day. About how much time did you spend outside, BETWEEN 11 AM AND 4 PM? *Interviewer: if R wants clarification, by mostly sunny we mean when it is sunny about 75%, or more, of the time.*	<0> R volunteers that they always avoid being outside on sunny days <1> R answers in minutes <3> R answers in hours <7> R volunteers that they avoid being outside during those hours <8> don't know <9> refused	
S5.	Thinking about the last month, during the time you spent outside while it was sunny, how often did you get out of the sun and move to an area that was in the shade, would you say: always, often, sometimes, rarely, or never?	<1> always <2> often <3> sometimes <4> rarely	<5> never <8> don't know <9> refused
S6.	And in the last month when you were outside and it was sunny, how often did you wear a hat that shaded your EARS and NECK, as well as your face: would you say always, often, sometimes, rarely, or never?	<1> always <2> often <3> sometimes <4> rarely	<5> never <8> don't know <9> refused
S7.	Wear a shirt with long sleeves? In the last month while you were outside and it was sunny, did you do this always, often, sometimes, rarely, or never? *Interviewer: If appropriate, this includes any type of apparel that covers the arms; i.e., jackets, sweat shirts etc.*	<1> always <2> often <3> sometimes <4> rarely	<5> never <8> don't know <9> refused
S8.	How often did you wear *If female then "long skirt", if male then "long pants"* in the last month while you were outside and it was sunny, did you do this always, often, sometimes, rarely, or never?	<1> always <2> often <3> sometimes <4> rarely	<5> never <8> don't know <9> refused
S9.	Use sunscreen? In the last month while you were outside and it was sunny, did you do this always, often, sometimes, rarely, or never?	<1> always <2> often <3> sometimes <4> rarely	<5> never <8> don't know <9> refused

APPENDIX *(continued)*
Cancer Risk Factor Questions

Sun-related Behaviour Questions *(continued)*		
S10. Wear sunglasses while you were OUTSIDE? In the last month while you were outside and it was sunny, did you do this always, often, sometimes, rarely, or never?	<1> always <2> often <3> sometimes <4> rarely	<5> never <8> don't know <9> refused
Women's Health		
Next we want to talk with you about women's health issues.		
WH1. Have you ever had a mammogram, that is, a breast x-ray?	<1> yes <5> no	<8> don't know <9> refused
WH2. Did you have your last mammogram within the last two years?	<1> yes <5> no	<8> don't know <9> refused
WH3. Can you tell me how many months/years ago that was? *Combination of months and years – unit dependent on the answer to WH2*	<0> less than one month ago <1–70> enter exact number of months/years <98> don't know <99> refused	
WH4. For which of the following three reasons did you have your last mammogram: ONE: as part of a regular check-up, or routine screening; TWO: because of an ongoing or past breast problem; or THREE: because you were concerned that you might have a problem? *Interviewer: if required: a breast problem is when a women has been diagnosed with breast cancer or some other breast problem in the past, a concern is when a women has noticed something that she wants to have checked out.*	<1> regular check-up/routine screening <2> ongoing or past breast problem <3> concern about possible problem <8> don't know <9> refused	
WH5. Have you had a hysterectomy?	<1> yes (includes partial hysterectomy) <5> no <8> don't know <9> refused	
Cervical cancer screening questions		
CC1. Have you ever had a Pap smear test? *Interviewer, if required: A Pap test is done during an internal examination. Cells are taken from a woman's cervix; that is, from the opening of her uterus (womb). It is done to look for any cancer cells or any cells that might change into cancer cells.*	<1> yes <5> no	<8> don't know <9> refused
CC2. Did you have your last Pap smear test within the last two years?	<1> yes <5> no	<8> don't know <9> refused
CC3. Can you tell me how many months/years ago that was? *Combination of months and years – unit dependent on the answer to question CC2*	<0> less than one month ago <1–70> enter exact number of months/years <98> don't know <99> refused	

APPENDIX *(continued)*
Cancer Risk Factor Questions

Cervical cancer screening questions *(continued)*		
CC4.	For which of the following three reasons did you have your last Pap smear test done: ONE: as part of a regular check-up, or routine visit to a doctor or clinic; TWO: because of an ongoing or past problem; or THREE: because you were concerned that you might have a problem?	<1> regular check-up/routine visit <2> ongoing or past problem <3> concern about possible problem <8> don't know <9> refused
Colorectal Cancer Screening		
CO1.	A test for blood in your stool is where you have a bowel movement and use a stick to smear a small sample of it on a special card. Have you ever had this test?	<1> yes <8> don't know <5> no <9> refused
CO2.	Did you have this test within the last two years?	<1> yes <8> don't know <5> no <9> refused
CO3.	Can you tell me how many months/years ago that was? *Combination of months and years – unit dependent on the answer to CO2*	<|> less than one month ago <1–70> enter exact number of months/years <98> don't know <99> refused
CO4.	For which of the following three reasons did you have a test for blood in your stool: ONE: as part of a regular check-up, or routine screening; TWO: because of an ongoing or past bowel problem; or THREE: because you were concerned that you might have a problem?	<1> regular check-up/routine screening <2> ongoing or past bowel problem <3> concern about possible problem <8> don't know <9> refused
Next we want to ask you about PSA testing, a PSA test is a blood test that a doctor orders to check for prostate cancer.		
P1.	Have you ever had a PSA test?	<1> yes <8> don't know <5> no <9> refused
P2.	Did you have your last PSA test within the last two years?	<1> yes <8> don't know <5> no <9> refused
P3.	Can you tell me how many months/years ago that was? *Combination of months and years – unit dependent on the answer to P2*	<0> less than one month ago <1–70> enter exact number of months/years <98> don't know <99> refused
P4.	For which of the following three reasons did you have your last PSA test: ONE: as part of a regular check-up, or routine screening; TWO: because of an ongoing or past prostate problem; or THREE: because you were concerned that you might have a problem?	<1> regular check-up/routine screening <2> ongoing or past prostate problem <3> concern about possible problem <8> don't know <9> refused

APPENDIX *(continued)*
Cancer Risk Factor Questions

Fruits and Vegetables		
These next questions are about the foods you usually eat or drink. Please tell me how often you eat or drink each of the following foods, for example, twice a week, three times a month, and so on. Include all foods you eat at home and away from home.		
FV1. How often do you drink fruit juices such as orange, grapefruit, or tomato? *If R asks, frozen juice from concentrate as well as fresh juice is included but juice drinks that are only partly made of juice are not included.*	<1> per day <2> per week <3> per month <4> per year	<5> never <8> don't know <9> refused
FV2. Not counting juice, how often do you eat fruit? *Interviewer: if asked: this includes frozen and canned fruit, as well as fresh fruit and fruit used in cooking when the fruit is the major component of the food, such as a pie, but not when fruit is a small component of the food such as a muffin.*	<1> per day <2> per week <3> per month <4> per year	<5> never <8> don't know <9> refused
FV3. How often do you eat green salad?	<1> per day <2> per week <3> per month <4> per year	<5> never <8> don't know <9> refused
FV4. NOT including french fries, fried potatoes, or potato chips, how often do you eat potatoes? *Interviewer: if asked, this does not include yams or sweet potatoes.*	<1> per day <2> per week <3> per month <4> per year	<5> never <8> don't know <9> refused
FV5. How often do you eat carrots? *Interviewer: if asked, this includes frozen as well as fresh carrots.*	<1> per day <2> per week <3> per month <4> per year	<5> never <8> don't know <9> refused
FV6. Not counting carrots, potatoes, or salad, how many servings of vegetables do you usually eat?	<1> per day <2> per week <3> per month <4> per year	<5> never <8> don't know <9> refused
Physical Activity		
Now some questions about physical activities or exercise that you do during your normal activities, including your time working, doing chores, and in your leisure time. I'll ask you first about moderate activities and then about vigorous activities.		
PA1. In a usual week, do you do moderate activities for at least 10 minutes at a time, such as brisk walking, bicycling on flat ground, vacuuming, gardening, or anything else that causes some increase in breathing or makes your heart beat somewhat faster?	<1> yes <5> no	<8> don't know <9> refused
PA2. How many days a week, on average, do you do these moderate activities for at least 10 minutes at a time?	<0> None <1–7> Enter number of days <8> Don't know <9> Refused	

APPENDIX *(continued)*
Cancer Risk Factor Questions

Physical Activity *(continued)*		
PA3.	On days when you do moderate activities for at least 10 minutes at a time, how much total time do you spend doing these activities? INTERVIEWER: Enter EXACT number of MINUTES here please. DO NOT ROUND!!	<0> never <5–480> Enter number of MINUTES <481> more than 8 hours <999> refused <998> Don't know
PA4.	In a usual week, do you do vigorous activities for at least 10 minutes at a time, such as running, aerobics, bicycling on hills, heavy yard work, or anything else that causes large increases in breathing or makes your heart beat much faster?	<1> yes <8> don't know <5> no <9> refused
PA5.	How many days a week, on average, do you do these vigorous activities for at least 10 minutes at a time?	<0> None <1–7> Enter number of days <8> Don't know <9> Refused
PA6.	On days when you do vigorous activities for at least 10 minutes at a time, how much total time do you spend doing these activities? Interviewer: Enter EXACT number of MINUTES here please. DO NOT ROUND!!	<0> never <5–480> Enter number of MINUTES <481> more than 8 hours <999> refused <998> Don't know
Tobacco (Cigarette) Use by Respondent		
T1.	Have you smoked at least 100 cigarettes in your entire life?	<1> yes <8> don't know <5> no <9> refused
T2.	Currently do you smoke cigarettes everyday, some days, or not at all?	<1> everyday <3> some days (occasionally/sometimes) <5> not at all <8> don't know <9> refused
T3.	Did you ever smoke cigarettes on a daily basis?	<1> yes <8> don't know <5> no <9> refused
T4.	[# only asked of daily smokers] On average, about how many cigarettes a day do you now smoke? *Interviewer: 1 large pack = 25 cigarettes; 1 small pack = 20 cigarettes*	<1–90> enter exact number of cigarettes <98> don't know <99> refused
T5.	[# only asked of occasional smokers] On average, when you smoked during the last 30 days, about how many cigarettes did you smoke a day?	<0> less than one whole cigarette <1–90> enter exact number of cigarettes <98> don't know <99> refused
T6.	During the last 12 months, have you quit smoking for 1 day or longer?	<1> yes <8> don't know <5> no <9> refused

Environmental tobacco smoke and deaths from coronary heart disease in Canada

Margaret de Groh and Howard I Morrison

Abstract

A series of recent meta-analyses have concluded that non-smokers who live with smokers face an elevated risk of coronary heart disease (CHD). In this study, we estimated the number of CHD deaths among non-smokers attributable to environmental tobacco smoke (ETS) exposure in their homes. Population-attributable risk estimates suggest that in 1997 over 800 Canadians died of CHD caused by passive exposure to ETS. This figure is likely an underestimation of the total number of CHD deaths attributable to ETS, since our study did not estimate the number of deaths among non-smokers caused by ETS exposure in the workplace. However, this partial picture can still help to highlight the burden of disease resulting from this pervasive involuntary environmental exposure.

Key words: *coronary heart disease; environmental tobacco smoke; mortality; population attributable risk; smoking*

Introduction

There is increasingly compelling evidence that exposure to environmental tobacco smoke (ETS) is associated with an increased risk of coronary heart disease (CHD) in non-smokers. [3,7,9,13,14,20] Three recent meta-analyses [5,10,17] have reported significantly increased risks of similar magnitude for coronary heart disease among non-smokers exposed to ETS in their homes. Studies examining the exposure of non-smokers to ETS in the workplace have also reported significantly elevated risks of CHD compared to unexposed non-smokers.[5]

Much of the experimental work exploring the relationship between CHD and ETS has focused on acute effects.[2,4,20] Experimental studies demonstrate that acute exposure to ETS enhances platelet aggregation.[7,10] ETS exposure can injure the endothelial layer of blood vessels, which could contribute to the initiation or progression of atherogenesis; [7] ETS exposure may also increase the risk of developing atherosclerosis through the promotion of plaque development.[3] A focus on the acute effects of ETS exposure seems appropriate, given the fall in CHD risk that occurs when active smoking stops. The US Surgeon General's report on the health benefits of smoking cessation concluded that the risk of CHD among former smokers is cut in half after just one year of cessation.[16] Former smokers show some residual long-term elevated risk that declines over time.[4,16] It seems reasonable to assume that similar risk declines in non-smokers would occur when exposure to ETS ceases. There is support for this interpretation from prospective cohort studies, which have found a higher elevated risk of CHD among non-smokers living with current smokers than among non-smokers living with former smokers.[5,14]

Coronary Heart Disease and ETS Exposure in Canada

Although the number of CHD deaths resulting from active smoking have been estimated for Canada, [8,11] similar estimates for ETS are lacking. Estimating the number of deaths associated with passive exposure to ETS is more complex, since there are multiple settings of potential exposure, including the home, workplace and other public places. However, generating estimates based on what we do know about the exposure level in specific environments can highlight the burden of disease resulting from this type of involuntary exposure.

Methods

The number of CHD (ICD-9 rubrics 410–414) deaths among those aged 25 years or older by Canadian province in 1997 were obtained from Statistics Canada. Estimates of the prevalence of household exposure to passive cigarette smoking by province in 1999 were obtained from the Canadian Tobacco Use Monitoring Survey (CTUMS).[6] This is an ongoing, cross-sectional survey that collects information from a representative sample of provincial residents on a range of tobacco control issues, including individual smoking status and ETS exposure in the home. Individuals were considered to be exposed to ETS if they were non-smokers (never smokers or those who had quit at least three years previously) and lived in a household in which smoking occurred every day or almost every day inside the home.

The incidence density ratio (relative risk) associated with exposure to environmental tobacco smoke was estimated from two recent meta-analyses. Thun and colleagues[17] noted relative risks of 1.24 for males and 1.23 for females exposed to passive smoking, while He and colleagues[5] estimated a relative risk of 1.25. Both of these meta-analyses examined more than 18 prospective cohort and case control studies. These individual studies, which included US, European and Asian studies using slightly different operational definitions of ETS home

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exposure, nevertheless reported very similar elevated CHD risk estimates for non-smoking men and women. For this study, we used an estimate of 1.24, which is at the lower end of the range of risk estimates produced by these studies. Because relative risks in the study by Thun et al[17] were almost identical for males and females, and to increase the precision of our estimates, we calculated passive smoking attributable deaths for both sexes combined.

Population-attributable risk (also known as attributable fraction)[18] was estimated for each province using the following formula:

$$PAR = p(IDR - 1)/p(IDR - 1) + 1$$

where p is the proportion of the total population with the exposure (i.e., non-smokers regularly exposed to ETS in their homes) and IDR is the incidence density ratio estimated from the two cohort studies summarized above (IDR = 1.24). Passive smoking-attributable CHD mortality was calculated as the product of the passive smoking-attributable fraction and the number of CHD deaths.

Results

Prevalence of ETS Exposure

Table 1 summarizes the prevalence of active smoking and passive ETS exposure of non-smokers in the home in the 1999 Canadian population, age 25+. The table also includes a group of recent quitters (i.e., within the past three years). In 1999, an estimated 8% (approximate CI: ± 1%) of the Canadian population aged 25+ were non-smokers regularly exposed to ETS in their homes. The percentage within each province who were both non-smokers and were exposed to ETS in their homes ranges from a low of 3% in British Columbia to a high of 12% in Newfoundland and Quebec. On average, the degree of variability around these provincial estimates of ETS exposure was about ± 1.9%.

The national and provincial estimates of ETS exposure in the home combine the results for men and women. Preliminary analysis indicated that, although the prevalence of active smoking among men aged 25+ was somewhat higher than for women in 1999 (26% and 22%, respectively), the percentages of non-smoking men and women exposed to ETS in their homes were quite similar. Seven percent of all men and 8% of all women in Canada aged 25+ were non-smokers regularly exposed to ETS in their homes.

Estimated CHD Deaths Attributable to ETS

Population-attributable risk estimates suggest that in 1997 over 800 Canadians died from CHD as a result of involuntary tobacco smoke exposure in their homes (Table 2). A disproportionate number of men and women in Quebec and Newfoundland were estimated to have died from CHD resulting from passive smoking, reflecting the high prevalence of active and passive smoking in these provinces. The number of estimated deaths per 100,000 population was significantly lower in British Columbia, reflecting the very low prevalence of household exposure to second-hand cigarette smoke in the province. If the prevalence of exposure to environmental tobacco smoke

TABLE 1
Smoking prevalence and exposure to environmental tobacco smoke among non-smokers[1] males and females, age 25+, for Canada and the provinces

Province	Sample Size (CTUMS, 1999)	Non-smokers of Interest[1]		Transition "Non-smokers"[2] (%)	Current Smokers (%)
		Exposed to ETS in the Home (%)	Not Exposed to ETS in the Home (%)		
Newfoundland	1,229	12	56	4	27
PEI	1,123	10	61	5	24
Nova Scotia	1,105	9	59	4*	28
New Brunswick	1,003	8	61	5	25
Quebec	1,032	12	55	4*	29
Ontario	952	7	68	3*	22
Manitoba	1,113	7	68	3*	22
Saskatchewan	1,148	8	65	2*	24
Alberta	1,137	7	65	4*	24
British Columbia	1,039	3	73	5*	19
Canada	**10,881**	**8**	**64**	**4**	**24**

[1] Never smokers and former smokers who have quit for at least 3 years, combined.
[2] Individuals who have quit smoking within the past 3 years.
* Moderate sampling variability, interpret with caution.
Percentages may not sum to 100% due to rounding.

TABLE 2
Deaths from CHD attributable to exposure to household environmental tobacco smoke, males and females age 25+, Canada and Provinces, 1997

	Passive Smoking Prevalence	Number of CHD Deaths	Number of CHD Deaths Attributable to Passive Smoking	Rate of CHD Deaths Attributable to Passive Smoking per 100,000 Person-years
Newfoundland	0.12	1,095	31	8.66
PEI	0.10	198	5	5.64
Nova Scotia	0.09	1,597	34	5.40
New Brunswick	0.08	1,146	22	4.36
Quebec	0.12	11,221	302	6.10
Ontario	0.07	16,750	267	4.48
Manitoba	0.07	1,988	34	4.65
Saskatchewan	0.08	1,751	33	5.19
Alberta	0.06	3,653	56	2.99
British Columbia	0.03	4,988	36	1.30
Canada	**0.08**	**44,421**	**803**	**3.98**

across Canada could be lowered to the levels observed in British Columbia, there would be an estimated 480 fewer CHD deaths each year.

Discussion

In 1997, over 800 Canadian non-smokers were estimated to have died of coronary heart disease as a result of exposure to second-hand smoke in their homes. Our estimation is a good example of how the application of population attributable risk estimates can contribute to understanding the impact of an exposure in the population. In the current case, we are dealing with a modest increase in risk with exposure (e.g., RR of about 1.24), but an environmental exposure within the population that is quite large (e.g., 8% of the adult population aged 25+).

It is likely that we have underestimated the overall number of ETS-related deaths because only exposure in the home was considered; deaths attributable to workplace exposure were not included. Such workplace exposure may be substantial, given that a single smoker may expose multiple individuals within the workplace. In 1996–1997, 32% of men and 19% of women (aged 25+) who smoked every day worked in workplaces with no restrictions on smoking.

Our analysis is also likely to underestimate attributable deaths because we used ETS exposure data derived from a 1999 survey, rather than for 1997. Active smoking among older adults (age 25+) declined from 1996–1997 to 1999 (from 28% to 24%), and it is reasonable to assume that passive smoking prevalence also declined.[6,12] Since risks associated with CHD can fall quite dramatically after just one year in former active smokers,[16] we also chose a relatively conservative definition of non-smoker, which included never smokers plus former smokers who had to have quit for at least three years. Our estimate also does not include any increased risk to current smokers that may result from their exposure to environmental tobacco smoke.

Our estimate of the number of CHD deaths attributable to ETS were derived from a single estimated relative risk. A more accurate estimate would have resulted from the use of age-specific relative risks; unfortunately, no satisfactory age-specific relative risks for CHD and ETS exposure among non-smokers are available. The limited evidence that does exist, however, suggests that this would result in only modest changes in the estimated number of CHD deaths attributable to ETS.[19]

Finally, although there is general acceptance that ETS exposure increases the risk of CHD in non-smokers, the magnitude of the effect is greater than might be anticipated based on the risk observed with active smoking. This has led a few to suggest that existing studies overestimate the relative risk associated with passive smoking and CHD,[1,15] which would, in turn, overestimate the number of deaths among non-smokers attributable to residential passive smoking. However, review of a range of experimental and clinical studies suggests that the impact of tobacco smoke on the heart is primarily acute and that the biological mechanisms involved in platelet aggregation, for example, are similar for both active and passive smoking. There is also evidence of a nonlinear dose-response relationship across passive and active smoking. Passive smoking produces elevated risks for CHD that are similar to low-level active smoking (e.g., about one cigarette per day). Glantz and Parmley[3] have

suggested that the reason active smoking does not produce higher (i.e., linear) dose-response results is because the effects of cigarette smoke on the heart may reach a saturation point, making a monotonic dose-response effect unlikely. Therefore, concerns about an overestimation of a passive smoking risk estimate is less tenable in light of the highly consistent relative risks across cohort studies, evidence for a plausible dose-response relationship, and evidence in support of biological plausibility.[3,7,13,19,20]

In Canada, the rates of home exposure to ETS among non-smokers varies dramatically by province. The high proportion of non-smokers in Quebec exposed to ETS as compared to the low proportion in British Columbia reflects both the active smoking prevalence differences in these two provinces and provincial differences as to whether smokers face household smoking restrictions. Eighty-eight percent of smokers in Quebec lived in a household where someone smoked every day or almost every day inside the home, compared to 59% of smokers in British Columbia.[6] If the prevalence of exposure to environmental tobacco smoke in Canada could be lowered to the levels observed in British Columbia, there would be an estimated 480 fewer coronary heart disease deaths each year in Canada.

Highlighting the number of CHD deaths caused by passive smoking in homes can have important implications for the development of public health programs and awareness campaigns. In particular, such information clearly underscores the importance of promoting smoke-free homes across Canada. Often, these programs and awareness campaigns focus on reducing ETS exposure in homes with children. These results indicate that promoting smoke-free homes can have a positive health impact on both children and adults.

Tobacco smoke is a potentially deadly environmental exposure. To provide some perspective on the degree of complacency with which ETS is often treated, Steenland[13] pointed out that, within workplaces, environmental exposure limits for specific toxins are often set to contain the number of excess deaths resulting from exposure to the toxin. These environmental limits are usually in the range of one death in 10^5 or one in 10^6. As reported in Table 2, excess CHD deaths attributed to passive smoking are about 4 in 10^5 for Canada as a whole, a rate that far exceeds what is acceptable for other toxic exposures.

The endpoint of this analysis was mortality, the most extreme adverse outcome associated with exposure to ETS. Glantz and Parmley[3] have estimated that the occurrence of nonfatal myocardial infarction due to passive exposure to ETS is likely to be three times as high as deaths from CHD. These less extreme outcomes also contribute to the disease burden and health care costs associated with passive smoking and CHD.

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Estimating the population at risk for Ontario Workplace Safety and Insurance Board-covered injuries or diseases

Dianne Zakaria, James Robertson, Joy C MacDermid, Kathleen Hartford and John Koval

Abstract

Difficulty in quantifying the population at risk for a work-related injury or disease limits the usefulness of workers' compensation data for surveillance. This article presents a method of obtaining estimates of the Ontario Workplace Safety and Insurance Board (OWSIB)-covered workforce using the Canadian Labour Force Survey (LFS). The method involves extracting that class of worker most likely to be insured by the OWSIB and using actual hours worked to estimate full-time equivalents at risk. Compared to population at risk estimates readily available from published tables, the refined crude estimate was 26% lower and ranged from 15 to 79% lower depending on the age group. The percentage decrease from published estimates was generally greater for women compared to men, particularly in the 25 to 39 year age categories. Consequently, the method of deriving population at risk estimates should be considered when comparing rates across sexes, ages, industries or occupations.

Key words: *denominators, Labour Force Survey, Ontario Workplace Safety and Insurance Board coverage, population at risk, work-related injury or disease rates*

Introduction

Statement of Problem

A major limitation to using workers' compensation data for the surveillance of work-related injuries or diseases is the difficulty in generating denominators for the calculation of accurate rates.[1–4] In Ontario, the workers' compensation system is funded by premiums paid by employers. The premium is dependent on the nature of the business, the employer's health and safety record, and the size of the payroll, not the number of full-time equivalent workers to be insured.[5] For these reasons, an estimate of the insured population in Ontario is not readily available. In an attempt to produce an estimate, some researchers have relied on government census data, which overestimates the number of full-time equivalent workers at risk because the defined employed population includes full-time, part-time and temporary workers and workers not covered by workers' compensation.[3]

In Canada, it is estimated that 20 to 30% of the workforce is not covered by workers' compensation.[6] Furthermore, since women are more likely than men to hold part-time or temporary positions, a greater overestimation of the at-risk population is likely for women relative to men.[3] Other investigators[1,7,8] have used Statistics Canada's Labour Force Survey (LFS) data. For example, Ashbury[1] used LFS published estimates of the employed population of Ontario, which are overestimates because they include full- and part-time workers, unpaid family workers and workers not covered by the Ontario Workplace Safety and Insurance Board (OWSIB).

Brooker et al.[7] and Rael et al.[8] improved upon Ashbury's[1] method by using the LFS to obtain estimates for employed, paid workers. These estimates would exclude unincorporated business owners and unpaid family help, the former not likely to be insured and the latter definitely not insured by the OWSIB. Although the authors did not detail the mechanics of extracting this class of worker, it is clear that the actual hours worked by employed, paid workers was not utilized in quantifying the at-risk population. Consequently, an employed, part-time, paid worker would contribute the equivalent of an employed, full-time, paid worker to the population at risk estimate, producing an overestimate.

Rael[9] found injury rates calculated using employed, paid workers were equivalent to those calculated using employed, paid hours to derive the employed, paid workers. However, Rael's[9] population at risk estimates were for males aged 15 to 64 in the Ontario construction industry, a group not likely to contain many part-time, paid workers. Thus, this equivalency may not be consistent across sex, industry or occupation and suggests that a method that adjusts for actual hours worked by employed, paid workers would be more appropriate for most applications.

Relevant Background Information

Canadian Labour Force Survey

The Canadian LFS is a monthly household survey that utilizes a multilevel sampling strategy to collect labour market activity information on those 15 years of age or

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older. Since July 1995, the national sample size has been 52,350 households, of which 15,858 are in Ontario. Data is collected every month in the week following the reference week, which is defined as the entire calendar week, Sunday to Saturday, usually containing the 15th day of the month. Specifically excluded from the survey's coverage are residents of the Yukon and Northwest Territories, Aboriginal people living on reserves, full-time members of the Canadian Armed Forces and inmates of penal institutions. These groups together represent an exclusion of approximately 2% of the Canadian population aged 15 or over. [10,11] The LFS is a large, regular survey which, via its multistage sampling strategy and extensive quality control measures, is the most readily available valid and reliable source of information on the working age population of Canada.

Ontario Workplace Safety and Insurance Board Coverage

Although most businesses in Ontario that employ workers, including family members and sub-contractors, must register with the OWSIB within 10 days of hiring their first full- or part-time worker, registration is voluntary for banks, trust companies, insurance companies and other financial institutions; computer programmers; private healthcare practices, such as those of doctors and chiropractors; veterinary work; dentistry; law offices; trade unions; private daycare establishments; travel agencies; recreational and social clubs, such as golf or health clubs; educational and recreational camps; churches; theatres with live performances; broadcasting stations; motion picture productions; photographers; barbers, hair salons and shoeshine stands; taxidermy; and funeral directing and embalming. An employer not mandatorily covered is almost always permitted to apply for coverage, but the OWSIB may apply conditions. Sole proprietors, independent operators, partners and executive officers are not required to have personal coverage.[5] Consequently, the employed labour force is not an accurate estimate of the population at risk of an OWSIB-covered injury or disease.

Purpose of Present Research

The purpose of this research is twofold. First, to detail how the LFS can be used to improve the accuracy of the population at risk estimates needed to generate crude and specific rates of OWSIB-covered injuries or diseases. Second, to demonstrate the degree to which population at risk estimates vary depending on the method of derivation from the LFS.

Methods

Statistics Canada provides a public use microdata LFS file for those wishing to undertake their own analyses. The files for the 12 months in 1997 were obtained and data for the province of Ontario was extracted. First, the employed labour force was calculated. This is an estimate that has been used previously[1] due to its availability from regularly published Statistics Canada tables. It will be used as a baseline for comparison with more refined methods.

To estimate the employed labour force in Ontario for 1997, the frequency of "labour force status" values equal to "employed, at work" or "employed, absent from work" were calculated for each of the 12 months using the final weights provided. These 12 monthly estimates were then averaged to produce an annual estimate for the employed labour force. This employed labour force estimate includes the self-employed as well as employees; full-time as well as part-time employed; unpaid family workers; and the employed who were not at work during the reference week due to factors such as illness or disability, personal or family responsibilities, vacation, or labour dispute.[11] To produce a measure that would reflect the actual hours worked, an annual estimate of the employed, full-time equivalents was calculated. The actual hours worked per week at all jobs by the employed labour force was calculated for each of the 12 months using the "actual hours per week at all jobs" variable and the final weights provided. These 12 estimates were then averaged to produce an annual estimate of the actual hours worked per week at all jobs by the employed labour force. This annual estimate for the employed labour force was multiplied by 52 weeks and divided by 2,000 hours (assuming a 40 hour work week for 50 weeks out of the year) to produce an annual estimate of the employed, full-time equivalents for Ontario during 1997. This estimate includes the self-employed, who are not automatically covered by the OWSIB; unpaid family workers who are not covered by the OWSIB; and employees, that is, individuals who collect wages or salary and are usually covered by the OWSIB.

To remove the self-employed and unpaid family workers from the annual estimates of the employed labour force and employed, full-time equivalents, the above procedures were repeated after using the "class of worker, main job" variable to extract public and private employees from the employed. To examine the effect of sex and age on the variability of the estimates, sex and age-specific annual estimates of the employed labour force and employed, full-time equivalents were calculated using the "sex" and "age group" variables to extract the appropriate data.

Results

Table 1 presents age-specific employed and employed, full-time equivalent annual estimates for the labour force as a whole and the employee subgroup. After extracting employees from the employed labour force and using the "actual hours per week at all jobs" to calculate full-time equivalents, the crude annual estimate of the at-risk population insured by the OWSIB, 4,014,181 employee full-time equivalents, was 26% lower than the employed labour force value of 5,412,868 employed persons (Table 1) readily available from published tables. This difference ranged from a low of 15% in the 25 to 29 age group to a high of 79% in the 70 plus age group. The sex-specific data (Tables 2 and 3) demonstrated similar trends but the percentage difference between the employed labour force and employee, full-time equivalent estimate was always greater for women except in two age categories: 60 to 64 and 70 plus years, where the sex differences in the percentage change were minimal. The female to male percentage change ratio was greatest in the 25 to 39 year age categories, ranging from 1.93 to 2.44.

TABLE 1
Estimation of at-risk population insured by the Ontario Workplace Safety and Insurance Board in 1997

Age group	Employed Labour Force			Employees Only			
	Employed[a]	Employed FTE[b]	% Change[c]	Employed[d]	% Change	Employed FTE	% Change
15 to 19	281,764	133,691	53	250,480	11	122,770	56
20 to 24	490,424	402,948	18	462,649	6	377,200	23
25 to 29	651,357	611,920	6	591,844	9	551,140	15
30 to 34	784,679	756,802	4	674,229	14	635,518	19
35 to 39	790,942	760,535	4	662,994	16	624,858	21
40 to 44	742,323	723,747	3	598,090	19	566,625	24
45 to 49	626,576	604,860	3	503,059	20	469,854	25
50 to 54	500,814	474,007	5	389,830	22	353,134	29
55 to 59	294,267	273,253	7	218,049	26	195,654	34
60 to 64	160,516	139,407	13	109,089	32	91,897	43
65 to 69	55,500	42,536	23	25,856	53	18,603	66
70 plus	33,707	22,629	33	12,386	63	6,927	79
All	5,412,868	4,946,336	9	4,498,555	17	4,014,181	26

Note: All values have been rounded to the nearest whole number. FTE = Full-Time Equivalent.

[a] This employed estimate includes the self-employed as well as employees; full- and part-time employed; unpaid family workers, and the employed who were not at work during the reference week.

[b] A full-time equivalent is defined as 2,000 worked hours (50 weeks × 40 hours per week).

[c] Percentage change is always calculated relative to the first employed column as it represents the most commonly used estimate.

[d] This employed estimate includes employees only; the self-employed and unpaid family workers have been removed.

Discussion

This research supports concerns about the overestimation of the full-time equivalent workforce at risk for an OWSIB-covered injury or disease and the differential overestimation in women relative to men, which can occur with the use of data readily available in published tables.[1,3]

Limitations in Method of Estimation

Although the method presented above attempts to make estimates more accurate, these refined estimates have limitations. First, although most businesses in Ontario that employ workers must register within 10 days of hiring their first full- or part-time worker, registration is voluntary for some.[5] The extent to which these businesses voluntarily choose to insure themselves is not known. If the tendency is low, even the refined population at risk estimates will be excessive, particularly in certain industry or occupational groups. It was not possible to produce reasonable lower limits on the population at risk estimates specifically examined (Tables 1–3) by removing those businesses in Ontario for which registration is voluntary due to the crude method that the LFS uses to code industries and occupations. For example, veterinarians are classified in the LFS industry code "agriculture" which includes all agricultural and related services such as livestock farms; other animal specialty farms; field crops; horticultural specialities; combination farms; and services incidental to agriculture, where veterinary services would be classified.[12] Hence, attempting to remove veterinarians would remove many others not exempt from coverage.

Second, the variable "actual hours per week at all jobs" has a 99-hour limit. Consequently, those working greater than 99 hours per week would not have their additional hours included in the full-time equivalents at-risk estimate. Since the percentage of employees with "actual hours per week at all jobs" greater than or equal to 99 hours was 0.09 for 1997, this limit will be negligible. The final limitation is the error introduced by multiple job holders. Since the "class of worker" is based on the "main job" those employees who are self-employed outside of their main job would inappropriately have these additionally worked hours added to their employee hours worked. Conversely, those who have main jobs classified as self-employed or unpaid family work, but have secondary jobs as public or private employees, would not have their employee hours worked included in the employee, full-time equivalents at risk estimate. Since only 4.9% of the employed labour force in Ontario during 1997 were multiple job holders and the main job accounted for

TABLE 2
Estimation of at-risk male population insured by the Ontario Workplace Safety and Insurance Board in 1997

Age group	Employed Labour Force			Employees Only			
	Employed[a]	Employed FTE[b]	% Change[c]	Employed[d]	% Change	Employed FTE	% Change
15 to 19	146,038	78,075	47	132,584	9	72,269	51
20 to 24	257,074	227,532	11	240,536	6	211,198	18
25 to 29	348,276	359,724	3	309,383	11	315,532	9
30 to 34	427,589	462,385	8	358,240	16	375,497	12
35 to 39	433,169	464,882	7	353,832	18	369,325	15
40 to 44	399,902	428,637	7	309,458	23	319,183	20
45 to 49	331,759	354,283	7	253,892	23	260,672	21
50 to 54	283,550	294,616	4	207,606	27	205,662	27
55 to 59	174,524	178,175	2	123,794	29	121,399	30
60 to 64	100,628	95,816	5	60,787	40	56,307	44
65 to 69	36,555	30,508	17	15,694	57	12,379	66
70 plus	23,649	16,961	28	7,380	69	4,616	80
All	2,962,712	2,991,596	1	2,373,186	20	2,324,040	22

Note: All values have been rounded to the nearest whole number. FTE = Full-Time Equivalent.

[a] This employed estimate includes the self-employed as well as employees; full- and part-time employed; unpaid family workers, and the employed who were not at work during the reference week.

[b] A full-time equivalent is defined as 2,000 worked hours (50 weeks × 40 hours per week).

[c] Percentage change is always calculated relative to the first employed column as it represents the most commonly used estimate.

[d] This employed estimate includes employees only; the self-employed and unpaid family workers have been removed.

98.3% of the actual hours worked, it is likely that this error will have a negligible effect on subsequent calculated rates.

Limitations of the Canadian Labour Force Survey

The LFS assumption that the reference week is representative of the whole month has been challenged.[13] Webber[13] was concerned that annual estimates of worked hours may be biased if labour disputes or public holidays occurred disproportionately in the 12 reference weeks relative to the remaining 40 weeks in a year. For example, certain important public holidays, such as Thanksgiving, Good Friday, Easter Monday and Remembrance Day, can fall in the LFS reference week. Conversely, the mid-month location of the reference week precludes the remaining important public holidays from ever falling in that time period. For this reason, the LFS estimate will sometimes exceed the true weekly average of worked hours for the month, and sometimes will be less. To examine the combined effect of labour disputes and public holidays, Webber[13] used statistics published by Labour Canada on person-days lost through work stoppages by month, data from the Pay Research Bureau, and data from the LFS on the magnitude of hours lost due to holiday. The annual actual hours worked estimates, adjusted for labour dispute and holiday effects, ranged from 1.5% below to 1.5% above the unadjusted estimates. Webber[13] concluded that the unadjusted survey results could be preferred on the basis of ease of calculation and the small effect of adjustment relative to the errors inherent in the original data, but for data users interested in measures of year-to-year changes in aggregate annual actual hours worked, the adjustments have a substantial impact.

Summary and Conclusions

This research provides evidence that regularly published labour force survey estimates overestimate the population at risk for an OWSIB-covered injury or disease. The degree of overestimation was demonstrated to vary with sex and age and certainly varies across occupations and industries. A method was presented to obtain more accurate estimates of the at-risk population. This method extracts those employed individuals who are most likely to be insured by the OWSIB and uses the actual hours worked to estimate full-time equivalents at risk. Although there is no gold standard to establish the veracity of the derived estimates, certainly rate comparisons across age groups and sexes within industry and occupational groups would be more valid if discrepancies in the actual hours worked between the sexes and age categories were acknowledged in

TABLE 3
Estimation of at-risk female population insured by the Ontario Workplace Safety and Insurance Board in 1997

Age group	Employed Labour Force			Employees Only			
	Employed[a]	Employed FTE[b]	% Change[c]	Employed[d]	% Change	Employed FTE	% Change
15 to 19	135,726	55,616	59	117,896	13	50,502	63
20 to 24	233,350	175,415	25	222,113	5	166,002	29
25 to 29	303,081	252,196	17	282,461	7	235,607	22
30 to 34	357,090	294,418	18	315,989	12	260,021	27
35 to 39	357,773	295,652	17	309,162	14	255,533	29
40 to 44	342,422	295,110	14	288,632	16	247,442	28
45 to 49	294,817	250,577	15	249,167	15	209,182	29
50 to 54	217,264	179,391	17	182,224	16	147,472	32
55 to 59	119,743	95,078	21	94,254	21	74,255	38
60 to 64	59,888	43,590	27	48,302	19	35,590	41
65 to 69	18,946	12,028	37	10,162	46	6,223	67
70 plus	10,059	5,668	44	5,006	50	2,311	77
All	2,450,156	1,954,740	20	2,125,369	13	1,690,141	31

Note: All values have been rounded to the nearest whole number. FTE = Full-Time Equivalent.

[a] This employed estimate includes the self-employed as well as employees; full- and part-time employed; unpaid family workers, and the employed who were not at work during the reference week.

[b] A full-time equivalent is defined as 2,000 worked hours (50 weeks × 40 hours per week).

[c] Percentage change is always calculated relative to the first employed column as it represents the most commonly used estimate.

[d] This employed estimate includes employees only; the self-employed and unpaid family workers have been removed.

the population at risk estimates. In conclusion, when utilizing the LFS to derive estimates of the population at risk over time, any changes in the methods employed by the LFS, the OWSIB policy on mandatory coverage, or the tendency towards voluntary registration should be acknowledged.

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Under-reporting of maternal mortality in Canada: A question of definition

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Abstract

In Canada, maternal mortality reporting is based on information contained on death certificates. To examine the extent to which maternal deaths are under-reported in Canada and whether this is likely to change under the 10th revision of the International Classification of Diseases (ICD), we linked live birth and stillbirth registrations to death registrations of women aged 10 to 50 for 1988 through 1992. We reviewed the death certificates of women found to have died while pregnant or within a year of the termination of pregnancy. The officially reported maternal mortality ratio for the study years was 3.7 deaths per 100,000 live births. Depending on whether we included deaths where the certifying physician did not list pregnancy as a contributing factor on the death certificate, revised ratios under ICD-9 ranged from 4.9 to 5.1 per 100,000 live births for deaths from direct obstetric causes and from 0.5 to 1.2 per 100,000 live births for deaths from indirect obstetric causes. Reflecting changes in classification criteria, revised ratios under ICD-10 were lower than those under ICD-9 for deaths from direct obstetric causes – ranging from 3.9 to 4.1 per 100,000 live births – and higher for deaths from indirect obstetric causes – ranging from 2.0 to 3.0 per 100,000 live births. Of deaths from direct obstetric causes, those from cerebrovascular disease were the most numerous and also the most likely to be underreported. Deaths from pulmonary embolism and indirect obstetric causes were the next most likely to be underreported. In a companion article we report an investigation on whether deaths from causes not directly related to pregnancy – such as injury, infectious disease and epilepsy – are more or less likely to occur among pregnant and recently pregnant women.

Key words: *definition of maternal mortality, maternal mortality, surveillance of maternal mortality*

Introduction

Less than 100 years ago in North America, childbirth was a leading cause of death among young women, second only to tuberculosis.[1] Over the course of the last century, maternal deaths have become rare events in Canada and other industrialized countries. However, maternal mortality continues to be a key health indicator around the world,[2] and maternal mortality ratios are still routinely compared, as shown in Figure 1. Low levels of maternal mortality in Canada reflect the general good health of our population, our universal access to medical care, and the status we accord to women and their health care needs.

Despite our good record, every maternal death is a cause for concern. Approximately 15 maternal deaths are reported in Canada each year. Those that occur in hospital are usually the subject of thorough investigation by review committees in the hospitals where the deaths occurred, but some deaths that could be "maternal" may not be included in the officially reported counts. Researchers in other countries, often using definitions of maternal death that are broader than the definition used by vital registrars, have suggested that up to twice as many maternal deaths occur as are reported.[3–7]

Classification of deaths as maternal is based on information contained on death certificates. Reasons for underreporting of maternal deaths include improper completion of death certificates and errors in coding the underlying cause of death. Death certification and classification by cause involve many steps (Appendix 1).

Before 2000, deaths in Canada were classified according to the 9th revision of the International Classification of Diseases (ICD-9);[8] since 2000, deaths have been classified according to the 10th revision (ICD-10).[9] Reflecting changes proposed over the years, the ICD-10 definition of maternal mortality is more comprehensive than its predecessors:[10,11] more causes of death are classified under maternal mortality, and two new categories have been added – "late maternal," which includes deaths that

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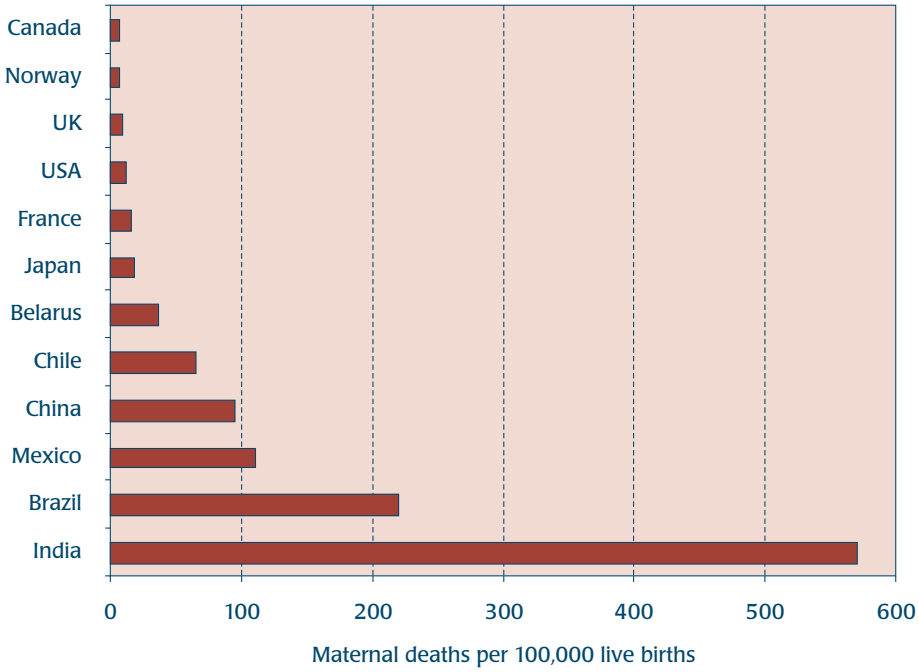
FIGURE 1
Maternal mortality ratios in selected countries (1999 estimates)[2]



occur beyond the traditionally defined 42-day postpartum period, and "pregnancy-related," which includes all deaths around the time of pregnancy regardless of cause. These changes and additions are described in more detail below and in Appendix 2.

The primary purpose of the investigation reported here was to determine whether maternal mortality is under-reported in Canada and to determine reasons for any omissions. Another purpose was to explore the effect of changes in the definition of maternal death under ICD-10 on the understanding and reporting of maternal mortality.

Methods

Ascertainment of previously unreported maternal deaths using record linkage

To identify deaths that occurred among pregnant or recently pregnant women, we linked live birth and stillbirth records to records of deaths among women of reproductive age that occurred within 365 days of a registered birth. Some deaths that occur around the time of pregnancy do not link to a birth registration, however. There will be no birth registration if the death occurred very early in pregnancy or the woman died undelivered. In other cases, a birth may not be registered or the birth registration may be missing. We therefore also requested the death certificates of women whose death had been classified as maternal (i.e., assigned an ICD-9 Chapter 11 code, see Appendix 1) but whose death records did not link to a birth. We were not able to identify women who died while pregnant or within 365 days of the termination of pregnancy if the death had not been classified as maternal and there was no birth registration.

To prepare for the linkage, we extracted to a separate file all records of deaths in the Canadian Mortality Data Base that occurred between January 1, 1988, and December 31, 1992, in females 10 to 50 years of age. We then extracted from the Canadian Birth Data Base all records of live births and stillbirths between January 1, 1987, and December 31, 1992. We excluded deaths from 1988 through 1990 and births from 1987 through 1990 in Newfoundland, due to under-counting of births in Newfoundland for those years.

To link the birth files to the death file, we used the mainframe version of the Generalized Record Linkage System (GRLS V1).[12] We generated an alternative entry if the mother's maiden surname field was different from the infant's. Surnames and maiden surnames were also assigned phonetic codes in case the names were misspelled. The mother's identifying information on the live birth/stillbirth file was compared with the decedent's identifying information on the death file. Identifying information included surname, maiden surname, given names or initials, date of birth, place of birth, marital status, place of event, and place of residence. We also assigned "weights" to each pair of records to reflect the probability that the computerized pairs of records represented the same person.[13] Pairs of records with weights above a pre-determined threshold were considered potentially good links. Computer printouts listing linkage identifiers and other items (e.g., street address, spouse's given names) were generated for all potentially good links and were manually reviewed. If we could not determine from the information on the computer printout that the decedent was the same person as the mother listed on the birth registration, additional sets of identifying information contained on the birth and death registration forms were compared. False-positive links identified in this way were removed.

Definition of maternal death under ICD-9 and ICD-10

Shown in Appendix 2 are the ICD-9 and ICD-10 definitions of maternal death and its two subcategories, termed "direct" and "indirect" *obstetric* death. ICD-9 Chapter 11 (Complications of Pregnancy, Childbirth and the Puerperium) comprises codes between 630.0 and 676.9. All deaths for which the underlying cause has been assigned a code in this range are designated "direct" obstetric deaths, with the exception of deaths assigned codes between 647.0 and 648.9. These codes are designated "indirect" obstetric deaths and include deaths from causes that would otherwise be coded to other ICD-9 chapters (see Appendix 3). Chapter 11 includes codes for conditions such as eclampsia, postpartum

hemorrhage and amniotic fluid embolism. These deaths are clearly obstetric deaths: they could only occur during pregnancy or around the time of childbirth. However, ICD-9 Chapter 11 also specifies that deaths from pulmonary embolism and cerebrovascular disorders – conditions that are not uniquely associated with pregnancy and childbirth – are classifiable as "direct" obstetric deaths if they occur during pregnancy or the postpartum period (within 42 days of the termination of pregnancy). More specifically, a death from pulmonary embolism is to be assigned a Chapter 11 code *only if* pregnancy has been listed as a contributing factor in Part 1 or Part 2 of the medical certificate of death, whereas *any* death from a cerebrovascular disorder during pregnancy or the postpartum period is to be classified as a direct obstetric death, regardless of whether the certifying physician listed pregnancy as a contributing factor.

Changes in the classification and designation of death as maternal death under ICD-10

Several changes in ICD-10 affect reporting of deaths as maternal deaths. First, the list of underlying causes of death included under the rubric "indirect" has been expanded to include all causes other than perinatal conditions, and injury and poisoning. Second, deaths from cerebrovascular disease during pregnancy or the postpartum period are classified as indirect rather than direct obstetric deaths.

Two new categories of "maternal mortality" are defined in ICD-10 (see Appendix 2). The first, "late maternal death," includes deaths from direct or indirect obstetric causes occurring more than 42 days but less than one year postpartum. A second, very broadly defined category, "pregnancy-related death," includes all deaths that occur during pregnancy or the postpartum period regardless of cause.[8]

Identification of maternal deaths

Two obstetricians (RK and RL) and a third medical expert reviewed the death certificates, but in cases where the underlying cause of death was given as cancer, injury or poisoning, they reviewed only those certificates on which there was a notation referring to pregnancy. Reviewers were informed of the number of days after the pregnancy outcome that each death occurred.

Provincial registrars provided revised certificates of death for 29 of 33 certificates marked "interim," indicating that an underlying cause had not been determined at the time the certificate was originally sent to Statistics Canada. A medical coder from Statistics Canada assigned a code to the underlying cause for 27 of these deaths. A specific underlying cause had not been determined for two deaths, although these deaths were known to have been caused by trauma. We included revised certificates in the review process if they qualified according to the above criteria.

To assess the reliability of judging relatedness to pregnancy using only death certificate information and knowledge of the timing of death in relation to pregnancy, two of the three reviewers independently made an initial assessment of each selected death certificate. Reviewers agreed in 92% of the cases as to whether the death certificates contained sufficient information to make a judgement; in 97% of cases judged to have sufficient information they further agreed as to whether the death was related to pregnancy.

Reviewers assigned corrected codes of underlying causes where they deemed appropriate. The record linkage and review of confidential death data were carried out at Statistics Canada.

Results

We identified 633 deaths that had occurred within 365 days of a pregnancy outcome. During the study years, 72 deaths had been assigned codes under ICD-9 Chapter 11 and reported as maternal deaths. We found that two of these had occurred more than 42 days postpartum and therefore were not classifiable as maternal deaths under ICD-9. Our capture strategy also missed three deaths that had been reported as maternal. For 11 of the 70 correctly reported maternal deaths, we found no corresponding birth registration.

The results are summarized separately for the two categories of reportable maternal death: direct and indirect obstetric deaths. Because the causes of death classified as direct and indirect obstetric death differ under ICD-9 and ICD-10, differences in case ascertainment under each of these two classification systems are given.

For interest, we also report the numbers of deaths that would be included in the two new ICD-10 categories "late maternal" and "pregnancy-related" death. Numbers of deaths in these categories, however, are not reported by the vital records system.

Identification of unreported direct obstetric deaths and reclassification of reported direct obstetric deaths

Shown in Table 1 are all direct obstetric deaths that occurred during the study years by cause and source of ascertainment. Of the 70 correctly reported maternal deaths, 66 were classifiable as direct obstetric deaths under ICD-9 and are shown in Table 1. Four of the 70 were classifiable as indirect obstetric deaths and are included in Table 3. Our review process netted 33 additional deaths that reviewers agreed should have been classified as direct obstetric deaths under ICD-9. Note that deaths from cerebrovascular disorders would be classified as indirect obstetric deaths under ICD-10.

Reviewers also judged that 16 deaths originally assigned a cause of death code in Chapter 11 had been miscoded. They therefore assigned a new code, but all newly assigned codes were within the range of codes designating obstetric deaths. This code re-assignment resulted in fewer deaths in some categories and more deaths in other categories. These category shifts are shown in column two of Table 1.

Table 2 summarizes possible reasons why the 33 newly ascertained direct obstetric deaths had not been originally so classified.

TABLE 1
Direct obstetric deaths by cause

Cause (ICD-9 code(s))	Reported		Newly ascertained	Total
	Original code assigned	Category changed		
Ectopic (630)	3		1	4
Spontaneous abortion (634)	1		1	2
Legally induced abortion (635)	1			1
Illegally induced abortion (636)	1			1
Antepartum hemorrhage (641)	4	–3		1
Hypertension complicating pregnancy (642)	14	–1, +1	2	16
Liver disorders in pregnancy (646.7)	0		1	1
Previous cesarean delivery (654.2)	1	–1		0
Rupture of the uterus (665.1)	2	+1		3
Postpartum hemorrhage (666)	8	+1	2	11
Anesthetic complications (668)	0	+2	1	3
Complications after cesarean section (669.4)	0	+1		1
Major puerperal infection (670)	1	+1	2	4
Venous complications (671)	7	–4		3
Amniotic fluid embolism (673.1)	9	+2	2	13
Other pulmonary embolisms (673.0, 673.2, 673.8)	2	+4	5	11
Cerebrovascular disorders (674)	3	+1	15	19
Postpartum cardiomyopathy (674.8)	1	+2		3
Other & unspecified (669.7, 669.8, 669.9, 674.9)	8	–7	1	2
Total direct obstetric deaths	66		33	99

Identification of unreported indirect obstetric deaths

Table 3 lists the deaths that reviewers judged to be indirect obstetric deaths if the death was eligible to be so classified under ICD-9 or ICD-10. Only four of the 70 correctly reported maternal deaths had been originally classified and reported as indirect obstetric deaths.

Identification of deaths under the newly defined ICD-10 category "late maternal" death and the newly defined concept "pregnancy-related" death

As stated above, two of the 72 deaths reported by the vital records system as maternal deaths occurred more than 42 days postpartum. These deaths are actually over-counts according to ICD-9 but would be included in the new ICD-10 category "late maternal" death. Our review process identified two additional late maternal deaths from direct obstetric causes and four from indirect obstetric causes.

The newly created category under ICD-10, "pregnancy-related death," included more deaths than the categories "direct" and "indirect" obstetric death. Of the total 633 deaths identified, 187 (29.5%) occurred (or were assumed to have occurred) during pregnancy or within 42 days of its outcome. All 187 would be included in this new ICD-10 category, including 34 deaths from injury or poisoning and 12 deaths from cancer (see Appendix 2).

The magnitude of under-reporting of maternal mortality under ICD-9 versus ICD-10

Table 4 shows ranges of maternal mortality ratios from causes classifiable as direct and indirect obstetric deaths and the differences in each under ICD-9 and ICD-10. Maximum values include all deaths that reviewers retrospectively judged to be obstetric deaths eligible under each ICD version. Minimum values exclude unreported deaths retrospectively judged to have been maternal deaths but for which the certifying physician had not listed pregnancy as a contributing factor in Part 1 or Part 2 of the medical certificate of death, a requirement for the death to be classified as maternal by medical coders.

TABLE 2
Reasons direct maternal deaths were not reported

Attributable to vital records system	Number of deaths
Death certificate interim at time of vital statistics report	6
Death correctly coded but not included in report (missed)	1
Underlying cause of death incorrectly coded by medical coder (cause of death as indicated on medical certificate clearly obstetric)	2
Subtotal	9
Attributable to unclear definition and classification principles or improper completion of the death certificate	
Death coded as accident or error occurring during medical care[a]	1
No indication of pregnancy on death certificate	
■ death caused by cerebrovascular disorder	5
■ death caused by other condition classifiable as direct obstetric death under ICD-9	2
No notation in Part 1 or Part 2 of the medical certificate that pregnancy was a contributing factor, but question on death certificate whether decedent pregnant within preceding 42 days answered "yes"	
■ death from pulmonary embolism (other than amniotic fluid embolism)[b]	4
■ death caused by other condition classifiable as direct obstetric death under ICD-9	2
Death caused by cerebrovascular disorder and pregnancy within previous 42 days clearly indicated on medical certificate of death	10
Subtotal	24
Total	**33**

[a] The underlying cause of death where death was the result of an accident or error in medical care is to be coded as an injury according to ICD-9 Rule 12,[7] although reviewers judged that this death, resulting from an error related to anaesthesia administered during childbirth, should be classified as a direct obstetric death and coded under anaesthesia complications in the pregnancy chapter (ICD-9 668).

[b] It is unclear under ICD-9 whether all deaths from obstetrical pulmonary embolisms other than amniotic fluid embolisms occurring during the postpartum period are to be classified as direct obstetric deaths, but our reviewers judged all to be direct obstetric deaths.

Discussion

Our main findings were a striking under-reporting of deaths from cerebrovascular disorders, pulmonary embolism and causes indirectly related to pregnancy. Approximately two-thirds of direct obstetric deaths that were not reported were associated with cerebrovascular disorders or pulmonary embolisms. In fact, cerebrovascular disorders became the most frequent category of obstetric death, as well as the most likely to be under-reported. Classification of deaths from this cause under ICD-9 is unclear.[4] Moreover, deaths from cerebrovascular disorders are to be classified as indirect obstetric deaths under ICD-10. If we do not include deaths from cerebrovascular disorders as direct obstetric deaths, then direct obstetric deaths were under-reported by approximately 20%, within the range of major classification errors reported for death certification in general.[14] In a companion article, we discuss our further findings with respect to whether deaths during pregnancy and the postpartum period from cerebrovascular disorders should be classified as direct obstetric deaths.

The category "indirect obstetric death" was introduced in ICD-9. Although this revision was published in 1975, few deaths are so classified, even when physicians clearly list pregnancy as a contributing factor on the death certificate. Given that few are reported and that the judgement on what constitutes an indirect obstetric death is subjective – in contrast to most direct obstetric deaths, which clearly would not have occurred had the woman not been pregnant – the utility of this category is questionable.

Deaths from cerebrovascular disorders may not have been captured as direct obstetric deaths because physicians who certify death certificates and medical coders may have been unaware that, under ICD-9, all deaths from this cause during pregnancy or the postpartum period were to be classified as direct obstetric deaths. Deaths from pulmonary embolism may not be classified as maternal deaths because physicians are not aware that in Part 1 or Part 2 of the medical certificate of death they must clearly state, if they so believe, that pregnancy was a contributing factor in the death.

We also found that deaths from direct obstetric causes tended to be somewhat misclassified within the major ICD-9 heading for pregnancy and childbirth. However, after reclassifying these deaths and including newly ascertained direct obstetric deaths, we found that the most frequent causes of direct obstetric death (other than deaths from cerebrovascular disorders) remained essentially the same – hypertensive disorders of pregnancy, amniotic fluid embolism, and postpartum hemorrhage. Deaths from other types of pulmonary embolism within 42 days of a pregnancy outcome ranked in the top four direct obstetric cause-of-death categories after our review, although only two such deaths had been reported previously for the study years. Deaths from this cause tended not to have been reported as maternal deaths or to have been misclassified under other subcategories of direct obstetric deaths.

The magnitude of under-reporting of maternal deaths depended in part on whether we included deaths retrospectively judged to have been maternal deaths even though the physician who completed the death certificate had not listed pregnancy as a contributing factor. In some cases, the physician may not have been aware that the woman had recently been pregnant. In other cases, however, a separate question on the death certificate as to whether the

TABLE 3
Deaths judged to be indirect obstetric deaths by underlying cause and eligibility for inclusion under ICD-9 and ICD-10

Underlying cause of death (ICD-9 code(s))	Reported	Not reported		Total
		Pregnancy listed as a contributing factor		
Eligible for inclusion under ICD-9 or ICD-10		Yes	No	
Infectious & parasitic (001–139)	–	2	6	8
Anemia (280–285)	–	1	–	1
Circulatory disease (390–398, 410–429, 435, 440–459, 648.6)	2	2	7	11
Congenital anomalies of circulatory system (745–747, 648.5)	2	1	–	3
Subtotal: eligible indirect under ICD-9 or ICD-10	4	6	13	23
Eligible for inclusion under ICD-10 only				
Cancer (140–208)	–	2	–	2
Other metabolic and immunity disorders (270–279)	–	1	–	1
Coagulation defects (286)	–	1		1
Epilepsy (345)	–	2	4	6
Respiratory diseases (460–519)	–	–	1	1
Diseases of the digestive system (520–579)	–	2	–	2
Systemic lupus (710.0)	–	1	1	2
Other specified (congenital) anomalies (759.8)	–	–	1	1
Total: indirect under ICD-10	4	15	20	39

TABLE 4
Number of obstetric deaths (n) and maternal mortality ratios (MMR)[a] under ICD-9 and ICD-10

	Reported and newly ascertained minimum and maximum under ICD-9 (n) MMR						Minimum and maximum under ICD-10 (n) MMR			
	Reported		Minimum[b]		Maximum[c]		Minimum[b]		Maximum[c]	
Direct	(68)[d]	3.5	(95)[e]	4.9	(99)[e]	5.1	(76)	3.9	(80)	4.1
Indirect	(4)	0.2	(10)	0.5	(23)	1.2	(39)[e]	2.0	(58)[e]	3.0
Total direct and indirect[e]	(72)[d]	3.7	(105)	5.4	(122)	6.3	(115)	5.9	(138)	7.1

[a] Deaths per 100,000 live births (calculations of maternal mortality ratios based on 1,948,540 live births during study years)

[b] Includes previously unreported deaths from pulmonary embolism (other than amniotic fluid embolism) and those judged indirect only if the certifying physician listed pregnancy as a contributing factor in Part 1 or Part 2 of the medical certificate of death

[c] Includes deaths judged obstetric regardless of whether the certifying physician listed pregnancy as a contributing factor in Part 1 or Part 2 of the medical certificate of death

[d] Over-reported by 2 deaths that occurred more than 42 days postpartum

[e] Includes reported (n = 4) and previously unreported (n = 15) deaths from cerebrovascular disorders.

decedent had been pregnant within the preceding 42 days had been appropriately answered "yes". In these latter cases, we could not determine whether the certifying physician omitted to list pregnancy as a contributing factor in Part 1 or Part 2 of the medical certificate of death because of neglect, or because he or she did not believe pregnancy to have been a contributing factor in the death.

Although a similar magnitude of under-reporting of maternal mortality has been documented in the United States and Europe[3–7] even before implementation of ICD-9,[15] previous studies have been inconsistent with respect to what constitutes

maternal death. Previous investigators have included as unreported many deaths not classifiable as obstetric under the ICD-9 definition, including deaths from injuries sustained in motor vehicle collisions, suicide, and deaths occurring more than 42 days postpartum.[4–6,16] Moreover, most investigators have categorized unreported deaths from cerebrovascular disorders as indirect rather than direct obstetric deaths, despite specification in the ICD-9 coding manual.[3,6,17–20]

Limitations and generalizability of the findings

Unless the underlying cause of death was initially classified as obstetric, we were not able to identify deaths among women who may have been pregnant at the time of death if the pregnancy did not result in a birth that was registered. This would apply to most women who died before 20 weeks' gestation from causes other than those directly related to pregnancy and to women who died outside of hospital and there was no attempt to deliver the fetus. Additionally, some births may not have been registered or the registration may have been missing. We found no birth registration corresponding to 11 of the 72 reported maternal deaths.

The problems we encountered with respect to death certificate completion and ambiguities of classification, including uncertainties about what constitutes maternal death, may be generalizable to other countries. These problems have been cited previously as a challenge not only to maternal death reporting[21] but also to classification and reporting of underlying causes of death generally, with recommendations for more attention to physician training at the postgraduate level in the completion of death certificates.[14]

Future surveillance of maternal mortality in Canada

The existing reporting system appears satisfactory for annual reporting of deaths from direct obstetric causes except for deaths from cerebrovascular disorders. The problems we detected – some misclassification and delays in replacing interim death certificates with the final version – may be correctable.

Ascertainment of late maternal deaths and most indirect obstetric deaths requires a labour-intensive process of record linkage and expert review of death certificates. Ellerbrock and colleagues have suggested that it is important to capture deaths of women who experience catastrophic events during childbirth but who die while on life support beyond 42 days postpartum.[11] We found only one such death, however. In total, only six deaths judged to be obstetric that occurred beyond 42 days postpartum were unreported. To find these six deaths required obtaining and partially or fully reviewing 446 death certificates in addition to the 187 death certificates for women who died within the 42-day postpartum period.

The other newly introduced category of maternal death under ICD-10, "pregnancy-related," requires record linkage but not expert review, but resulting rates or ratios would include an unknown number of deaths clearly ***unrelated*** to pregnancy.

For deaths from indirect causes, judgements of relatedness to pregnancy are necessarily subjective, both for physicians who certify death certificates and for reviewers making retrospective judgements. Unlike deaths from most direct obstetric causes, deaths from indirect causes may have been coincidental to the pregnancy. As already explained, the ICD-9 definition of indirect obstetric death includes deaths in only some cause-of-death categories, whereas under ICD-10 all cause-of-death categories other than injury and poisoning have been included. We could find no published description of the decision-making process that led to this more inclusive definition or to the introduction of the concept of indirect obstetric death under ICD-9. Demonstrating that death is more likely to occur from certain causes among pregnant or recently pregnant women would contribute to an evidence-based rationale for monitoring particular causes of death among pregnant and recently pregnant women. In a companion article, we explore whether pregnant or recently pregnant Canadian women were more or less likely to die of specific causes than women of the same age not known to have been pregnant during the same period.

In spite of low maternal mortality ratios in industrialized countries, surveillance of maternal mortality continues to be of interest. There is a need, however, to resolve current misunderstandings with respect to classification of deaths indirectly related to pregnancy. Until this is accomplished, comparisons of maternal mortality ratios among countries might be most appropriately limited to comparisons of direct obstetric deaths.

Acknowledgements

The authors wish to acknowledge the contribution of the provincial and territorial vital statistics registrars

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APPENDIX 1
Death Certification

Death certificates in Canada are legal documents standardized in accordance with World Health Organization guidelines. The last physician to attend the person who died, or, in some cases, a coroner or medical examiner, completes the death certificate. In some provinces, a trained medical coder assigns a code to the underlying cause of death; in other provinces these codes are assigned automatically by computer algorithm. The code is assigned according to information supplied by the physician, coroner, or medical examiner who completed the death certificate. The death certificate consists of two parts. Part one contains space for the underlying cause of death as well as for conditions arising as a consequence of this condition in order of causal sequence. Part two is reserved for conditions that contributed to the death but were not part of the causal sequence, such as smoking, use of alcohol, environmental exposures, as well as recent pregnancy if believed to have contributed to the death. Death registration forms in five provinces also contain a space or check box in which to indicate whether the death occurred during pregnancy or within 42 days (or 90 days in some provinces) thereafter. The underlying cause of death is assigned a disease-specific code under one of 17 ICD-9 major chapter headings. In Canada, automated coding systems are programmed to divert any death that may be a maternal death for manual coding. Maternal deaths are those that have been assigned a code under ICD-9 chapter 11: Complications of pregnancy, childbirth and the puerperium.

APPENDIX 2
Definition of maternal mortality – ICD-9 and ICD-10[7,8]

Under ICD-9 and ICD-10, maternal death is defined as:

> "the death of a woman while pregnant or within 42 days of the termination of the pregnancy, irrespective of the duration and the site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management but not from accidental or incidental causes."

Maternal deaths are considered to be either a) **direct obstetric deaths**, that is, deaths resulting from obstetric complications of the pregnant state (pregnancy, labour and puerperium); from interventions, omissions or incorrect treatment; or from a chain of events resulting from any of the above, or b) **indirect obstetric deaths**, that is, deaths resulting from previous existing disease or disease that developed during pregnancy, which was not due to direct obstetric causes but which was aggravated by the physiologic effects of pregnancy.

Differences in the coding and classification of maternal deaths between ICD-9 and ICD-10 include the following:

1. Deaths from cerebrovascular disorders during pregnancy or within 42 days of the termination of pregnancy are classified as direct obstetric deaths under ICD-9 but as indirect obstetric deaths under ICD-10.

2. Under ICD-9 the list of causes classifiable as indirect obstetric death is specific (see Appendix 3) and excludes deaths from causes such as cancer, respiratory disease, gastrointestinal disorders, etc. Under ICD-10, deaths from any cause other than perinatal conditions, injury and poisoning (and direct obstetric causes) are classifiable as indirect obstetric deaths if the underlying condition was believed to have been aggravated by pregnancy.

3. New under ICD-10 is a category termed "late maternal death," which includes deaths from direct or indirect causes that occur more than 42 days but less than a year following the termination of pregnancy.

4. Also new under ICD-10 is a category termed "pregnancy-related death," which includes all deaths that occur during pregnancy or within 42 days of the termination of pregnancy regardless of the cause or whether the certifying physician believed the underlying cause was aggravated by the pregnancy. This category therefore also includes all intentional and unintentional deaths from injury and poisoning.

APPENDIX 3
Ranges of codes specified as indirect obstetric deaths under ICD-9

ICD-9 pregnancy chapter code(s)[a]	Description	Codes or code ranges in other ICD-9 chapters
647	Syphilis	090–097
647.1	Gonorrhea	098
647.2	Other venereal diseases	099
647.3	Tuberculosis	010–018
647.4	Malaria	084
647.5	Rubella	056
647.6	Other viral diseases	050–079, except 056
647.8, 647.9	Other infectious and parasitic diseases (specified and unspecified)	none given
648	Diabetes mellitus	250
648.1	Thyroid dysfunction	240–246
648.2	Anemia	280–285
648.3	Drug dependence	304
648.4	Mental disorders	290–303, 305–316, 317–319
648.5	Congenital cardiovascular disorders	745–747
648.6	Other cardiovascular diseases	390–398, 410–429, 435, 440–459
648.7	Bone and joint disorders of the lower body	720–724, and 711–719, 725–738 if affecting lower limbs
648.8	Abnormal glucose tolerance	790.2
648.9	Other current conditions classifiable elsewhere – nutritional deficiencies	260–269

[a] "Includes the listed condition when complicating the pregnant state, aggravated by the pregnancy, or when a main reason for obstetric care."[8]

Cause-specific mortality during and after pregnancy and the definition of maternal death

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Abstract

As part of a study to determine whether maternal mortality in Canada is under-reported, we explored the validity of including deaths not directly related to pregnancy. We linked live birth and stillbirth registrations to death registrations of women of reproductive age from 1988 through 1992. We calculated standardized mortality ratios, by cause, from deaths in women known to have been pregnant and deaths in same-aged women not known to have been pregnant within the same time period. Women known to have been pregnant were approximately half as likely to die as would be expected in each of two six-month time periods: from 20 weeks gestation to 42 days postpartum (standardized mortality ratio [SMR] 0.4, 95% confidence interval [CI] 0.3–0.5), and from 42 days to 225 days postpartum (SMR 0.5, 95% CI 0.5–0.6). Furthermore, pregnant and recently pregnant women were not more likely to die from specific causes, with the exception of diseases of the arteries, arterioles, and capillaries (SMR 3.5, 95% CI 1.3–7.7) during pregnancy or within 42 days of pregnancy termination. The only other SMR that was > 1 was for death from cerebrovascular disorders during pregnancy and up to 42 days postpartum, although not significantly so (SMR 1.4, 95% CI 0.8–2.2). No other cause-specific SMRs were > 1. Moreover, recently pregnant women were found to be much less likely to commit suicide or to be the victims of homicide. We found no empirical justification for including deaths not directly related to pregnancy in reported counts of maternal deaths for most of the causal categories we considered.

Key words: *definition of maternal mortality, maternal mortality, surveillance of maternal mortality*

Introduction

Maternal mortality has been considered a key public health issue for many decades.[1] Comparisons of maternal mortality ratios over time and among countries provide a "report card" indicating trends and differences in the general level of health, the adequacy of medical care, and the economic and social status of women within a population.[2] An objective and consistent definition of this widely reported health indicator is therefore required. Before the ninth revision of the International Classification of Diseases (ICD-9) was adopted, deaths clearly the result of complications of pregnancy and childbirth, such as ruptured ectopic pregnancy, eclampsia, and postpartum hemorrhage, were classified as maternal deaths.

The publication of ICD-9, however, expanded the definition of maternal mortality to include deaths "indirectly" related to pregnancy.[3] Maternal death was defined as "the death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of the duration and the site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental or incidental causes." Separate definitions were given for two subcategories of maternal death, termed "direct obstetric deaths" and "indirect obstetric deaths." Direct obstetric deaths included deaths "resulting from obstetric complications of the pregnant state," while indirect obstetric deaths were defined as "deaths resulting from pre-existing disease or disease that developed during pregnancy … not due to direct obstetric causes, but exacerbated by the physiologic effects of pregnancy."

Although it is clear in most cases of direct obstetric death that death would not have occurred had the woman not been pregnant, determination of whether a death is indirectly related to pregnancy is necessarily subjective. The ICD definition of indirect obstetric death has become more inclusive with the publication of the tenth revision.[4] First, although some non-injury causes of death such as cancer were ineligible for inclusion as indirect obstetric deaths under ICD-9, the list of eligible categories was expanded in the tenth revision to include deaths from *any* cause that occurred during pregnancy or within 42 days of its termination, except for those that are injury-related.[4] Second, the 10th ICD revision added the category "late maternal deaths," defined as deaths from "direct or indirect obstetric causes more than 42 days but less than one year after [the] termination of pregnancy." The tenth revision also classifies deaths from cerebrovascular disorders that occur during pregnancy or within 42

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days of its termination as indirect rather than direct obstetric deaths.

Are pregnant and recently pregnant women at an increased risk of death from any or all causes? Some groups of researchers have attempted to answer this question, at least in part. Khlat and Ronsmans found higher rates of death from unintentional injuries among 15- to 19- year old women in Bangladesh during and shortly after pregnancy, although injury death rates among other age groups were not higher.[5] These researchers also found that rates of death from a global category of "other" causes not directly related to pregnancy were lower among pregnant and recently pregnant women over all groups.[5]

Dannenberg and colleagues found a higher than expected number of homicides among pregnant and recently pregnant women in New York City, although other injury death rates among these women were not higher.[6] Similarly, researchers in Tennessee reported that the adjusted death rate from all injuries was not higher among women who had delivered a live-born or stillborn infant in the previous year than among women who had not, although rates of homicide were somewhat higher among the women who had had a delivery (rate ratio 1.2, 95% confidence interval [CI] 0.75–1.92).[7] The same group of investigators also reported a somewhat higher rate of death from cardiovascular disorders among women who had delivered in the previous year (risk ratio 1.32, 95% CI 0.81–2.14) but significantly lower death rates among these women from cancer and a category that included all other non-injury and non-pregnancy related deaths.[7] Other investigators have reported lower risks of suicide during pregnancy[8] and within a year of childbirth.[9]

To our knowledge, no previous study has examined, across all major causal categories, whether pregnant women are more likely than non-pregnant women to die during pregnancy or the postpartum period from specific causes not directly related to pregnancy. Such information would help justify the inclusion of causes of death in the definition of maternal mortality other than causes obviously related to pregnancy. As part of a study to estimate maternal mortality in Canada, the primary results of which are reported in a preceding article (pages 23–30), we compared cause-specific death rates among women known to have died while pregnant or within a year after termination of pregnancy with rates among same-aged women not known to have been pregnant within the preceding year. In this paper, we report the methods and results of these comparisons.

Methods

We identified deaths among pregnant and recently pregnant women by linking live birth and stillbirth registrations to death registrations of women of reproductive age. The record linkage process and review of the identified death certificates by a panel of experts is described in the preceding article. It is likely that not all women who died during the first 20 weeks of pregnancy could be identified, because births in Canada are not registered if they occur before 20 weeks (although this varies somewhat by province), so that deaths that occurred during this period could not be linked to a birth registration. Eight deaths that occurred during this period for which the cause was classified as maternal (associated with ectopic pregnancy, and spontaneous and induced abortion), however, were captured (see Table 1 of the companion article, page 25). As well, we likely could not identify all women who died while pregnant or recently pregnant if the birth had not been registered for other reasons, but similarly we would have captured these deaths if they had been classified as maternal, regardless of the lack of a birth registration.

Deaths among women were included in the "obstetric" population if they occurred from the 20th week of pregnancy, a period of approximately 20 weeks (140 days) for the majority of women. To obtain a 1-year observation period for women in the obstetric population, we counted all deaths in the linked file that occurred between 20 weeks' gestation and delivery (approximately 140 days for the majority of women) or within 225 days of the termination of pregnancy (140 days + 225 days = 365 days). The total period of observation for the obstetric population was therefore 365 days, the same as for the non-obstetric population. If the follow-up period had not been limited to 225 days, this equivalency would not have been achieved without prolonging the period of observation in the non-obstetric population.

Using information available from the Health and Vital Statistics Data Section of Statistics Canada, we obtained the total numbers of deaths, by cause, for each of the five years under investigation among women within each of six age groups: 15–19, 20–24, 25–29, 30–34, 35–39, 40–44. Because of under-counting of births in Newfoundland between 1988 and 1990, deaths occurring in these years among the obstetric population in Newfoundland were excluded, as were deaths among women in the total Newfoundland population for these years.

We calculated the number of cause-specific deaths within each of the six age groups for the non-obstetric population – women not known to have been pregnant – by subtracting the number of deaths by cause among women in the obstetric population in each age category.

We obtained estimated five-year total person years of women in the age groups of interest for 1988 through 1992 from the Demography Division of Statistics Canada. We subtracted person years for women in each age group for Newfoundland for these years. To calculate the number of deaths that would be expected among the obstetric population from each cause in each age category, we first calculated the number of non-obstetric person years in each age category by subtracting the five-year total number of live births and stillbirths in each age category from the estimated five-year total person years of observation in each age category. (Because counts of total births within maternal age strata available from Statistics Canada include multiple births, we subtracted the reported number of twins, two-thirds of the reported number of triplets, three-quarters of the reported number of quadruplets, and four-fifths of the reported number of quintuplets within each maternal age category of live births and stillbirths.)

To calculate numbers of expected deaths separately for each of two six-month

periods of observation, we divided the number of total deaths, live births and stillbirths, and population person years in each cause-specific age stratum by two. Within each six-month period and cause-specific age stratum we calculated the number of deaths expected in this manner:

$$\text{Expected deaths} = \frac{½\,\text{td} - \text{od}}{½\,(\text{ppy} - \text{lbsb})} \times ½\,\text{lbsb}$$

Where:

td = total deaths in the age stratum

od = observed deaths for this cause among pregnant and recently pregnant women in the *six-month* period

ppy = total population years in the age stratum

lbsb = live births + stillbirths in the age stratum adjusted for multiple births

That is, we calculated a death rate among women not known to have been pregnant for each cause within each age category, and applied this rate to the number of women known to have been pregnant in the age category (lbsb). This gave us the number of deaths that would be expected for each cause if pregnant women in that age category died at the same rate from the same cause as non-pregnant women. For each cause and six-month period, we summed the numbers of observed and expected deaths over all age groups. We calculated cause-specific standardized mortality ratios (SMRs) for each period by dividing the number of observed deaths by the number expected and obtained exact 95% Poisson confidence limits for the SMRs using the strategy proposed by Sun et al.[10]

Results

Among the 1,939,471 women for whom a pregnancy or birth was known to have occurred during the study period, we identified 438 women who died either while pregnant or within 225 days of a registered live birth or stillbirth; 944 deaths would have been expected if death rates during pregnancy and within 225 days of its termination were the same as among non-pregnant women of the same age over a one-year period. The overall age-standardized mortality ratio was 0.4 (95% CI 0.3–0.5) for death from all causes during pregnancy or within 42 days of its termination. Similarly, the SMR was 0.5 (95% CI 0.5-0.6) for death from all causes for women between 43 and 225 days postpartum.

Displayed in Table 1 are the numbers of observed and expected deaths by causal category for each of the two observational periods. The appropriate causal categories include four deaths classified by medical coders under ICD-9 648 (indirect maternal deaths) as well as all deaths due to cerebrovascular disorders, whether these had been originally coded and reported as direct maternal deaths (i.e. ICD-9 code 674.0) or coded erroneously (see preceding article). Standardized mortality ratios (SMRs) and corresponding 95% CIs for main headings (in bold-faced type) and selected subcategories are displayed as well.

We found only one causal category in which deaths were more frequent among women known to have been pregnant than among women not known to have been pregnant. Deaths classified under diseases of arteries, arterioles and capillaries (ICD-9 codes 440–448) were found to be significantly more frequent than expected during the first six-month period (SMR = 3.5, 95% CI 1.3-7.7). Four of the six deaths in this category were due to thrombocytopenic purpura that occurred between six and 29 days postpartum, and the other two were due to aneurysms that occurred at 36 weeks' gestation. Deaths from cerebrovascular disorders were also somewhat higher than expected in the first six-month period (SMR = 1.4, 95% CI 0.8–2.2).

Discussion

We observed that deaths among women who were known to have been pregnant within each of two six-month periods were approximately half as likely as would be expected. Few previous studies have examined death rates among pregnant and recently pregnant women from non-obstetric causes. A Tennessee study reported similar findings, in that women who had delivered a live- or stillborn infant in the previous year were no more likely to die from any of the causes considered by the authors than were women who had not delivered.[7] Conversely, researchers in Bangladesh reported that overall death rates among pregnant and recently pregnant women were twice as high than among same-aged women not known to have been pregnant.[5] In the Bangladesh population, however, when deaths from direct obstetric causes were excluded, the non-injury death rate among pregnant and recently pregnant women aged over 20 was significantly lower than among women not known to have been pregnant. In our study, pregnant and recently pregnant women were less likely to die even when direct obstetric causes were included.

The differences in risk of death from direct obstetric causes between the Bangladesh population and our own highlights the benefits of our universal access to high-quality medical care during pregnancy and childbirth. The similar finding in these two populations – that pregnant and recently pregnant women are less likely to die from causes not directly related to pregnancy – means that some of the same underlying selective and protective effects discussed below are likely operating in both countries, regardless of the presumed differences in underlying population health status and access to medical care.

Considering specific cause-of-death categories, we found SMRs of < 1 for nearly all the ICD-9 cause-of-death categories we considered. The only cause-of-death category in which the number of deaths during pregnancy or within 42 days of its termination was significantly higher than expected was diseases of arteries, arterioles and capillaries.

We acknowledge the following methodologic limitations in our investigation. Record linkage can ascertain deaths occurring during pregnancy or a defined post-pregnancy period only if the death record links to a registered birth. Not all pregnancies result in a registered birth, and some birth registrations were found to be missing. Our strategy captured deaths among pregnant or postpartum women that did not link to a registered birth only if the underlying cause of death had been coded as a maternal death, that is, assigned an ICD-9 code between 630 and 676, which include deaths due to spontaneous or therapeutic abortion,

TABLE 1
Mortality among pregnant and recently pregnant women

Causes of death	Time Period							
	20 weeks' gestation to 42 days postpartum				43 days to 225 days postpartum			
Major ICD-9 headings and *selected sub-headings*	**Observed deaths**	**Expected deaths**	**SMR obs/exp**	**Exact 95% Poisson C.I.**	**Observed deaths**	**Expected deaths**	**SMR obs/exp**	**Exact 95% Poisson C.I.**
Infectious & parasitic	**8**	**12**	**0.7**	**0.3–1.31**	**4**	**12.6**	**0.3**	**0.1–0.8**
HIV	2	6.3	0.3	0.04–1.2	0	6.4	0	0–0.6
Endocrine/nutritional/ metabolic	**1**	**12.6**	**0.1**	**0.002–0.4**	**0**	**12.6**	**0**	**0–0.3**
Diabetes mellitus	0	5.4	0	0–0.7	0	5.4	0	0–0.7
Blood disease	**2**	**2.3**	**0.9**	**0.1–3.1**	**1**	**2.4**	**0.4**	**0.01–2.3**
Mental disorders	**0**	**5.4**	**0**	**0–0.7**	**4**	**5.1**	**0.8**	**0.2–2.0**
Alcoholic psychosis	0	3.4	0	0–1.1	3	3.2	0.9	0.2–2.7
Nervous system/sense organ disease	**8**	**16.9**	**0.5**	**0.2–0.9**	**3**	**17.5**	**0.2**	**0.04–0.5**
Epilepsy	6	6.6	0.9	0.3–2.0	3	7.1	0.4	0.1–1.2
Respiratory diseases	**1**	**15.2**	**0.1**	**0.002–0.4**	**8**	**14.5**	**0.6**	**0.2–1.1**
Circulatory disease	**33**	**38.4**	**0.9**	**0.6–1.2**	**36**	**36.7**	**1.0**	**0.7–1.4**
Chronic rheumatic heart disease	0	0.8	0	0–4.6	2	0.7	2.9	0.4–10.3
Ischemic heart disease	2	7.1	0.3	0.03–1.0	6	6.5	0.9	0.3–2.0
Cerebrovascular disease	19	13.8	1.4	0.8–2.2	9	14.1	0.6	0.3–1.2
Arteries, arterioles, & capillaries	6	1.7	3.5	1.3–7.7	3	1.9	1.6	0.3–4.6
Diseases of the digestive system	**2**	**11.4**	**0.2**	**0.02–0.6**	**3**	**11**	**0.3**	**0.05–0.8**
Diseases of the genitourinary system	**0**	**3.3**	**0**	**0–1.1.0**	**0**	**3.3**	**0**	**0–1.1.0**
Congenital anomalies of circulatory system	**3**	**3.5**	**0.9**	**0.2–2.5**	**3**	**3.5**	**0.9**	**0.2–2.5**
Diseases of the musculo-skeletal system and connective tissue	All systemic lupus (shown below)							
Systemic lupus	2	2.8	0.7	0.1–2.6	1	2.9	0.3	0.01–1.9
Cancer all sites	**11**	**110.4**	**0.1**	**0.05–0.2**	**41**	**108**	**0.4**	**0.3–0.5**
Cancer of the breast	1	26.3	0.04	0.001–0.2	9	25.7	0.4	0.2–0.7
Cancer of the cervix	0	9.8	0	0–0.4	5	9.3	0.6	0.2–1.3
Myeloid leukemia	2	4.6	0.4	0.05–1.6	3	4.5	0.7	0.1–2.0
All injuries	**34**	**217**	**0.2**	**0.1–0.2**	**141**	**207.9**	**0.7**	**0.6–0.8**
Motor vehicle accidents	22	85.5	0.3	0.2–0.4	52	83	0.6	0.5–0.8
Suicide	4	64.9	0.1	0.02–0.2	45	61.5	0.7	0.5–1.0
Homicide	2	20.9	0.1	0.01–0.4	16	19.6	0.8	0.5–1.3
All causes	187[a]	475	0.4	0.3–0.5	251[b]	469	0.5	0.5–0.6

[a] Includes 76 deaths from direct obstetric causes (other than cerebrovascular disorders), 8 of which occurred before 20 weeks gestation.

[b] Includes 4 deaths from direct obstetric causes.

or ectopic or molar pregnancy. Deaths incidental to a pregnancy in which no attempt was made to deliver the fetus, deaths among pregnant women in whom the pregnancy was not diagnosed, and deaths occurring during or shortly after a pregnancy that ended in an unreported stillbirth would have been misclassified as occurring in the non-obstetric population. This would be a serious misclassification given the purpose of this analysis, but because of the rarity of maternal deaths relative to other deaths among women, the impact on the SMR would be quite small. As not all paired birth-death registrations were manually reviewed, it is also possible that some links were undetected false-positives resulting in misclassification of these deaths as occurring in the obstetric population. This latter type of misclassification, however, would result in a further slight reduction in the true SMR.

We found that being pregnant or having had a child recently appears to protect Canadian women from death due to injury. Recently delivered women are likely to be at home with their babies in the early postpartum period, thus avoiding exposure to motor vehicle accidents, for example. We also observed that known pregnant and postpartum women were less likely to commit suicide or to be the victims of homicide. This finding is consistent with previous investigations that have shown a lower likelihood of suicide among pregnant and recently pregnant women.[8,9] Other investigators have found higher injury death rates among pregnant and recently pregnant women but only for some types of injury, most notably homicide, among specific subpopulations; total death rates from intentional and unintentional injuries in these earlier investigations have also generally been lower.[5–7]

Of the four suicide and two homicide deaths in the first six-month period, all occurred in the postpartum period; that is, we found no deaths that occurred during pregnancy from either of these causes (although we did find other injury-related deaths that had occurred while the woman was pregnant). We would not have captured deaths from suicide, homicide, or injury that may have occurred during pregnancy if no attempt had been made to deliver the fetus. In such cases there would have been no birth registration. Similarly, we would not have captured deaths from suicide, homicide or other injury that may have occurred very early in pregnancy, although the number of deaths from suicide or homicide could be presumed to be small given that we detected none during the latter stage of pregnancy and very few in the early postpartum period. We believe that deaths that occurred during the postpartum period were well captured by our record linkage methodology. The number of deaths from suicide increased substantially in the second six-month period but was still well below the expected number. Although postpartum depression clearly affects many women,[11] it apparently does not result in an increased incidence of suicide. It appears, conversely, that having a baby has a protective effect against suicide and, in Canada, against homicide as well.

For deaths due to causes other than injury or direct obstetric causes, there are many possible explanations for a lower SMR among pregnant or postpartum women. One of these is that women who become pregnant and sustain pregnancy are healthier than women who do not, a "healthy mother effect" analogous to the well-known healthy worker effect.[12] Furthermore, women contemplating pregnancy, those who are currently pregnant, and those caring for newborn infants may be more likely to avoid behaviours that may be harmful to themselves or their infants, whereas women who are seriously ill may be more likely to avoid becoming pregnant or to terminate their pregnancies. Additionally, pregnant women are nearly always under medical care in Canada; earlier detection and treatment of life-threatening illness is thus more likely. The care of women known to have underlying illness may also be better during pregnancy. Finally, the physiologic state of pregnancy itself appears to confer protection from some disorders. For example, although sometimes observed to worsen, serious conditions such as heart disease and asthma diagnosed before pregnancy sometimes improve over the course of pregnancy.[13,14]

If a given condition not directly related to pregnancy arises or worsens during pregnancy or the postpartum period and the woman dies, it cannot be said for certain that she would not have died had she not been pregnant. However, among the obstetric population, over a one-year period the number of deaths due to diseases of arteries, arterioles, and capillaries was higher than expected, as were deaths due to cerebrovascular disorders during pregnancy or within 42 days of its termination. This suggests that deaths in these categories should be counted as maternal deaths. Indeed, deaths due to cerebrovascular disorders during pregnancy and the puerperium have long been classifiable as direct maternal deaths,[15,16] although under ICD-10 they are classified under O99, "other maternal disorders classifiable elsewhere," along with other "indirect" causes.[4]

Although it was not clear that the deaths we observed due to thrompocytopenic purpura were directly related to pregnancy, it is possible that they were a manifestation of pre-eclampsia that was either undiagnosed or unknown to the person who completed the death certificate. Our results suggest that it would be reasonable to include deaths from diseases of the arteries, arterioles and capillaries in the category "direct obstetric death." It might also be more reasonable to classify deaths from cerebrovascular disorders as direct rather than indirect obstetric deaths. Our findings do not support the inclusion of deaths in any of the other causal categories we considered in counts of maternal deaths.

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16. World Heath Organization. *Manual of the international statistical classification of diseases, injuries, and causes of death, 8th revision*, Vol. 1. Geneva: WHO, 1967. ■

Departure of Principal Scientific Editor

We are sad to announce that Dr. Christina Mills, long-time Principal Scientific Editor of *Chronic Diseases in Canada*, will be leaving Health Canada to become Hallman Visiting Professor at the Centre for Behavioural Research and Program Evaluation (CBRPE), Faculty of Applied Health Sciences, University of Waterloo.

We would like to thank her for her many years of devotion to the development of CDIC and we wish her all the best in her new career.

Book Review

Quantitative Methods for the Evaluation of Cancer Screening

Edited by Stephen W Duffy, Catherine Hill and Jacques Estève

New York (US): Oxford University Press, 2001;
161 pp; ISBN 0-340-74125-2; $104.00(CDN)

Evaluation of cancer screening is a complex task with many pitfalls, even for the informed practitioner. This unique and much-needed volume not only systematically compiles diverse advanced quantitative methods normally scattered across the scientific literature, but also considers both their theory and practice in applied settings. It includes methods for evaluating cancer screening at various steps of program planning, development, implementation and monitoring. These approaches may be used to determine, first, whether screening works (or efficacy); second, whether to establish a program, often in a manner that reflects understanding of tumour biology (effectiveness); third, how to modify it commensurate with both biology and available resources (cost-effectiveness); and fourth, whether the program is having the desired impact (surveillance and evaluation).

Editors Steven Duffy, Catherine Hill and Jacques Estève have skillfully collated the contributions of the various authors based on material from a workshop held in Paris in 1997. The fourteen chapters, generally of very high quality, include study designs for measuring the efficacy and the effectiveness of cancer screening, advanced quantitative methods such as Markov chain models, use of routine data, and cost effectiveness. While most examples focus on breast and cervical cancer, some material is presented on colorectal cancer and childhood neuroblastoma.

Chapter 1 concisely summarizes approaches useful in evaluating cancer screening. These range from the rigorous examination of the criteria of Wilson and Jungner needed prior to implementing a screening program; through the role of the randomized controlled trial (RCT); then to the need for early indicators of program effectiveness, as mortality reductions may occur too far in the future to be useful. Concepts essential for an understanding of cancer screening techniques, including study results and lead time, are well illustrated with clear tables and figures. The summary of developments in the field over the past four decades provides a framework for study design in evaluation by emphasizing the importance of the RCT in establishing the efficacy of screening, then describing designs to evaluate programs already in place, including the uncontrolled cohort design and the retrospective case control study. The excellent overview of the important influences of effectiveness and costs on cancer screening evaluation given in Chapter 2 highlights the large amount of data required, including epidemiology in the absence of screening, demography, screening quality and policies, clinical practice and the costs involved.

Chapter 3 covers the issues of contamination and compliance in screening trials; the discussion on how to improve compliance in those invited for screening is relevant to both RCT design and to ongoing program operations. Statistical methods to adjust for contamination and non-compliance allow for an unbiased assessment of screening effectiveness yet still respect randomization, and will be useful to those assessing RCT results in the literature. Chapter 4 presents a more specialized analytic design to reduce selection bias in the absence of randomized controls by estimating the reduction in cancer mortality caused by screening at a younger age.

The next three chapters consider statistical models that evaluate various aspects of screening, based on improved understanding of the biology of the disease (in all cases with breast cancer as an example). The use of Markov chain models to estimate rates of disease progression, including estimates of sojourn time, or pre-clinical detection period, is described in depth in Chapter 5, and includes several pages of valuable SAS computer code. A statistical method that describes the theoretical relationship at the population level between the rate of clinically detectable metastases at diagnosis to the rates of occult metastases is presented in Chapter 6 with supporting data, figures and statistical appendices. Another Markov chain model, described briefly in Chapter 7, predicts the mortality reductions likely to accrue from different screening intervals by estimating the rates of development of preclinical breast cancer, progression from preclinical to clinical disease, and progression from clinical disease to death.

Practical considerations and the use of routine data for evaluation are discussed in Chapters 8 to 10. An excellent discussion of the use of the source data needed in evaluation, including cancer registry information, is accompanied by the many ways cancers can be categorized by detection mode. This is must reading for those analyzing interval cancers. The pitfalls of cohort and case-control design are discussed next, particularly as applied to screening exposure histories for cervical cancer. Finally, Hakolinen elegantly describes how to monitor the impact of screening using incidence and mortality data by utilizing methods that range from simple trends analysis or comparison of age-specific rates in different periods, to cancer projections, to multilevel analysis. Readers will gain many useful insights into confounding factors such as treatment advances on mortality and changes in diagnostic practice and risk factor prevalence on incidence.

The final four chapters look at the impact of screening on incidence and mortality for cervical, breast, and colorectal cancers, and childhood neuroblastoma. Walter analyzes enhanced data from the original Canadian case-control study on cervical cancer to calculate pre-clinical detection periods (PCDP), finding that the most important benefits accrue by detecting disease before it becomes invasive. The high sensitivity and long PCDP means that screening as seldom as once in 10 years is effective, and underscores the importance of recruiting women for the initial screen. Paci and colleagues next develop simple methods for estimating the extent of possible over-diagnosis of breast cancer and predicting mortality benefits from screening that will be useful as the program evolves to ensure that observed incidence and mortality counts are on track. A method to estimate the sensitivity of the fecal occult blood test (FOBT) for colorectal cancer that also accounts for the mean sojourn time allows for varying these estimates across the proximal and distal colon and the rectum. In the final chapter, Estève et al. develop models to assess screening strategies for neuroblastoma. Their analysis illustrates the importance of synthesizing biological, genetic, and epidemiologic knowledge into models; such wisdom would have been useful prior to implementing large-scale screening programs and efficacy trials and has contributed to the current consensus against screening or further trials.

The workshop nature of the original material used in the book limits its scope, as not all relevant topics are covered. The depth and breadth of coverage varies: while in most chapters the reader is guided to relevant literature that will fill any gaps, for some topics essential literature, such as the selection of control groups for case-control studies, is missing. A glossary of terms would help readers new to the field to understand that pre-clinical detection period and sojourn time are one and the same, for example. More importantly, while the book includes an excellent discussion of Markov type models together with many creative suggestions for their adaptation, there is almost no discussion of other modeling approaches used in the literature, such as simplified life-tables that can be based on spreadsheets, decision analysis approaches, or more comprehensive population-based micro and macro-simulation models. Finally, given the title of the book, readers may expect more information than is included on interim evaluation indicators essential to the ongoing monitoring of screening programs to ensure high quality service and cost-effectiveness.

Despite these caveats, this book will be an essential reference to serious evaluators of screening programs and policies. Applications are generally described in enough technical detail for a reader to try the approaches and supported by details of calculations, and, in some cases, computer code. This volume meets its aim to be accessible to a range of professionals in public health and disease control, as well to statisticians and epidemiologists. ■

Overall Rating:
Very good

Strengths:
This text provides an excellent discussion of a broad range of advanced quantitative topics relevant to the evaluation of many aspects of cancer screening and makes these methods far more accessible to potential users through clear descriptions using tables, graphs, worked examples, computer code and statistical appendices. It represents a timely and important contribution to ensuring the evidence base exists for the important societal goal of ensuring that health dollars spent on screening interventions are both effective and cost-effective at the population level.

Weaknesses:
The relatively broad scope inevitably means that not all relevant topics are included and leads to uneven coverage of some the individual topics. Readers will not find references more recent than 1999, and some gaps are evident.

Audience:
The book will be of most interest to biostatisticians and epidemiologists wanting to learn more about statistical methods, research designs and data requirements for evaluating cancer screening. Screening program managers and administrators will also benefit from an improved understanding of available methods and when and how to use them.

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Calendar of Events

February 27–March 1, 2002 **Atlanta, GA, USA**	"Cultivating Healthier Communities Through Research, Policy and Practice" 16th National Conference on Chronic Disease Prevention and Control Sponsors: Centers for Disease Control and Prevention Association of State and Territorial Chronic Disease Program Directors Prevention Research Centers Program	Terrye Hornsby Tel.: (301) 588-6000 x 270 Fax: (301) 588-2106 E-mail: thornsby@kevric.com <www.cdc.gov/nccdphp/conference>
March 21–23, 2002 **Toronto, Ontario**	"Programs, Progress and Promise" Ontario Tobacco Strategy Conference 2002	Justine Fields Conference Coordinator 2403–65 Broadway Avenue Toronto, Ontario M4P 1T9 Tel.: (416) 481-4660 Fax: (416) 488-1799 E-mail: justine@otsconference.com <otsconference.com>
April 7–10, 2002 **Victoria, British Columbia**	"Partnership Research for Health and Social Change" 6th National Health Promotion Conference Sponsors: Community Health Promotion Coalition University of Victoria Canadian Consortium for Health Promotion Research	Division of Continuing Studies University of Victoria PO Box 3030, STN CSC Victoria, British Columbia V8W 3N6 <www.hp2002.uvic.ca>
May 7–11, 2002 **Montréal, Quebec**	"ISSFAL 2002 – Dietary Fats and Health" 5th Congress of the International Society for the Study of Fatty Acids and Lipids	ISSFAL 2002 Secretariat c/o Golden Planners Inc. 301–126 York Street Ottawa, Ontario K1N 5T5 Tel.: (613) 241-9333 Fax: (613) 565-2173 E-mail: info@goldenplanners.com <www.issfal.org.uk>
May 26–31, 2002 **Vienna, Austria**	"Innovation and Prevention" XVIth World Congress on Safety and Health at Work	Allgemeine Unfallversicherungsanstalt Kongressbüro Adalbert-Stifter-Strasse 65 A-1200 Vienna, Austria Tel.: +43 1 33 111-537 Fax: +43 1 33111-469 E-mail: safety2002@auva.sozvers.at <www.safety2002.at>
June 5–7, 2002 **New Orleans, LA, USA**	"Strengthening America through Health Education and Health Promotion Alliances" 20th National Conference on Health Education and Health Promotion Sponsored by Centers for Disease Control and Prevention and the Association of State and Territorial Directors of Health Promotion and Public Health Education	Hyatt Regency New Orleans Professional and Scientific Associates Tel.: 1-800-772-8232 x 220 E-mail: HEHP2002@psava.com <www.astdhpphe.org/conf20/20confindex.htm>

June 6–11, 2002 **Washington, DC, USA**	"Healthy Ecosystems, Healthy People: Linkages between biodiversity, ecosystem health and human health" Presented by the International Society for Ecosystem Health in association with the Center for Applied Biodiversity Science at Conservation International	Healthy Ecosystems, Healthy People c/o International Society for Ecosystem Health Faculty of Medicine & Dentistry Health Sciences Addition, H121 The University of Western Ontario London Ontario N6A 5C1 Tel.: (519) 661-2111 x 86223 Fax: (519) 661-3797 E-mail: hehp@ecosystemhealth.com < www.ecosystemhealth.com/hehp >
June 11–13, 2002 **Toronto, Ontario**	NAACCR 2002 – "Achieving Equity in Cancer Control" The 2002 meeting of the North American Association of Central Cancer Registries Hosted by Cancer Care Ontario	Westin Harbour Castle Hotel Darlene Dale Cancer Care Ontario Tel.: (416) 217-1228 E-mail: Darlene.Dale@cancercare.on.ca < http://www.naaccr.org >
July 7–10, 2002 **Yellowknife, Northwest Territories**	"Our Environment, Our Health" 93rd Annual Conference of the Canadian Public Health Association Co-sponsored by the Northwest Territories/Nunavut Branch, CPHA	E-mail: conferences@cpha.ca < www.cpha.ca >
August 18–22, 2002 **Montréal, Quebec**	"Epidemiology and Modern Public Health" 16th World Congress of Epidemiology World Epidemiological Association *Call for abstracts deadline: March 15, 2002*	Events International Meeting Planners 759 Square Victoria, Suite 300 Montréal, Quebec H2Y 2J7 Tel.: (514) 286-0855 E-mail: iea2002@eventsintl.com < www.iea2002.com >

2001 Peer Reviewers

We are extremely grateful to the following people for their enormous contribution to *Chronic Diseases in Canada* as peer reviewers in 2001.

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Indexes for Volume 22, 2001

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No 3/4, 2001

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Volume 22 Author Index

Announcement

Chronic Diseases in Canada has changed, and not just in appearance.

From now on, the first issue in each volume will be released in January, with further issues to be published in April, July and October, in a regular quarterly schedule within the current calendar year.

Manuscripts can now be submitted by e-mail. Please see the revised Information for Authors on the inside back cover.

We look forward to receiving your comments on these new changes to CDIC.

CDIC: Information for Authors

Chronic Diseases in Canada (CDIC) is a peer-reviewed, quarterly scientific journal focusing on the prevention and control of non-communicable diseases and injuries in Canada. This may include research from such fields as epidemiology, public/community health, biostatistics, behavioural sciences and health services. CDIC endeavours to foster communication on chronic diseases and injuries among public health practitioners, epidemiologists and researchers, health policy planners and health educators. Submissions are selected based on scientific quality, public health relevance, clarity, conciseness and technical accuracy. Although CDIC is a Health Canada publication, contributions are welcomed from both the public and private sectors. Authors retain responsibility for the contents of their papers, and opinions expressed are not necessarily those of the CDIC Editorial Committee or of Health Canada.

Feature Articles

Regular Feature Articles: Maximum 4,000 words for main text body (excluding abstract, tables, figures, references) in the form of original research, surveillance reports, meta-analyses, methodological papers, literature reviews or commentaries.

Short Reports: Maximum 1,200 words (as above).

Status Reports: Describe ongoing national programs, studies or information systems at Health Canada (maximum 3,000 words).

Workshop/Conference Reports: Summarize workshops, etc. organized or sponsored by Health Canada (maximum 3,000 words).

Cross-country Forum: For authors outside of Health Canada to exchange information from research or surveillance findings, programs under development or program evaluations (maximum 3,000 words).

Additional Article Types

Letters to the Editor: Comments on articles recently published in CDIC will be considered for publication (maximum 500 words).

Book/Software Reviews: Usually solicited by the editors (500–1,300 words), but requests to review are welcomed.

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Submit manuscripts to the Editor-in-Chief, Chronic Diseases in Canada, Population and Public Health Branch, Health Canada, Tunney's Pasture, CDIC Address Locator: 0602C3, Ottawa, Ontario K1A 0L2, e-mail: cdic-mcc@hc-sc.gc.ca.

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Checklist for Submitting Manuscripts

Cover letter: Signed by all authors, stating that all have seen and approved the final manuscript and have met the authorship criteria of the Uniform Requirements and including a full statement regarding any prior or duplicate publication or submission for publication.

First title page: Concise title; full names of all authors and institutional affiliations; name, postal and e-mail addresses, telephone and fax numbers for corresponding author; separate word counts for abstract and text.

Second title page: Title only; start page numbering here as page 1.

Abstract: Unstructured (one paragraph, no headings), maximum 175 words (100 for short reports); include 3–8 key words (preferably from the Medical Subject Headings (MeSH) of Index Medicus).

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

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References: In "Vancouver style" (consult Uniform Requirements and a recent CDIC issue for examples); numbered in superscript (or within parentheses) in the order cited in text, tables and figures; listing up to 6 authors (first 3 and "et al." if more); without any automatic reference numbering feature used in word processing; any unpublished observations/data or personal communications used (discouraged) to be cited in the text in parentheses (authors responsible for obtaining written permission); authors are responsible for verifying accuracy of references.

Tables and Figures: Each on a separate page and in electronic file(s) separate from the text (not imported into the text body); as self-explanatory and succinct as possible; not duplicating the text, but illuminating and supplementing it; not too numerous; numbered in the order that they are mentioned in the text; explanatory material for tables in footnotes, identified by lower-case superscript letters in alphabetical order; figures limited to graphs or flow charts/templates (no photographs), with software used specified and titles/footnotes on a separate page.

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