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*Commentary*

# *The Brave New World—What Can We Realistically Expect to Achieve Through Cancer Control Early in the New Millenium?*

*Anthony B Miller*


## Abstract

*Cancer control requires strategic planning, and thus knowledge about the trends of incidence and mortality associated with cancer as well as future projections, in order that appropriate decisions on priorities can be made. Cancer prevention requires tobacco control and dietary modification. Screening should utilize only effective strategies. The trends in Canada are expected to be mainly favourable by the year 2020, apart from rising trends in non-Hodgkin's lymphoma. Tobacco control policies in Canada are having an impact, while dietary modification is probably having an impact on colorectal cancer incidence. Screening for cancer of the cervix has achieved maximum impact with present levels of compliance, but as yet there is no evidence of an impact of breast screening. Priority for the future will have to be placed on prevention, especially on encouraging young adults to quit smoking and on dietary modification starting at young ages, and care should be taken with cost-effective application of screening.*


***Key words:*** *cancer; control; prevention; projections; screening*

## Introduction

Cancer control comprises five components: prevention, early detection and screening, treatment, rehabilitation and palliative care. The World Health Organization (WHO) has developed the concept of national cancer control programs with the goals of preventing future cancers, diagnosing cancers early, providing curative therapy when available, ensuring freedom from suffering and reaching all members of the population.[1]

National cancer control programs are intended to be based on strategic planning, involving a realistic assessment of need within each country, the resources available and the priorities for action. To facilitate such planning, a situation analysis should be done—that is, an assessment of the current incidence and mortality of cancer within the country, and of past and future trends. Estimating future trends is difficult and is usually based on a model that is dependent on what has gone on before. In the annual *Canadian Cancer Statistics* published by the National Cancer Institute of Canada, a useful feature has been the projections, although they extend for only a few years. However, for adequate cancer control planning, projections that last longer than three to five years are required, particularly to help us take stock and decide whether we can expect to see favourable or unfavourable trends in the future, and therefore to determine what our priorities should be as we undertake strategic planning.

In this paper, I first review available cancer control strategies for prevention and screening. Then I present the results of an exercise in which I have extended projections of cancer incidence and mortality in Canada through to the year 2020, in the hope that this will not only engender a productive debate but also facilitate more thought on what has been achieved in cancer


***Author References***

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control over the last 25 years in Canada, and what may be achieved over the next 20.

The opinions expressed in this paper are often personal, based on my own assessment of the evidence. Thus, when a statement that is not referenced is made on the interpretation of a trend, the reader should assume that this is my own view. If supporting references are available and relevant, however, I cite them, although I have not provided references for many well-recognized facts, which may be found elsewhere.[2]

## Prevention in Cancer Control

Population attributable risks help in establishing priorities. The population attributable risk for tobacco-induced cancers is of the order of 30% in Western populations, but over the world as a whole it is closer to 20%; for dietary-associated cancers it is perhaps 30%; for cancers associated with infection, about 15%; and for occupational and environmental carcinogens, about 3–9%, depending on the prevalence and intensity of exposure in that particular population.[2] So, if we could apply all the knowledge we have now, we could prevent over half the cancers in the world.

Some years ago I estimated that if, in Canada, the effects of smoking could be eliminated, approximately 29% of cancer deaths might be prevented, and 22% of cancer cases. If dietary modification away from a diet of high fat, high energy and low plant content was successful, the effect would be roughly a 20–24% reduction. Breast cancer screening might prevent 25% of breast cancer deaths. Screening for cancer of the cervix should prevent 60% of cases and deaths from the disease.[3]

However, these actions will take time. Tobacco control will take at least 30 years. It has taken more than 30 years to build up the tobacco epidemic, and it is probably going to take a great deal longer than 30 years for control strategies to have a major impact.

Dietary modification, depending on the time of life when it takes effect, might take as little as 10 years or as long as 60 for any results to be apparent. A hint comes from the impact on cancer incidence of migration to high-risk areas: for colorectal cancer the change is fairly rapid, but for gastric and breast cancers the changes are much slower.

Hepatitis B vaccination can be expected to prevent liver cancer in high-risk countries with a high prevalence of infection. However, infants have to be vaccinated, and a major impact on liver cancer incidence cannot be expected for about 40 years, although there are other, more immediate non-cancer-related benefits from vaccination. Similarly, even if a vaccine for human papilloma virus (HPV) became available soon, it would take at least 30 years from vaccination before there was a clear effect on invasive cancer of the cervix. In contrast, screening and/or treatment, if effective, can have a rapid impact.

A serious problem in obtaining support for cancer prevention is that the future patient is unknown and under-represented. Cancer will develop in a third of the population, but for the majority we have no idea who will be affected, and so we do not know who the people are who should be pressing for the necessary actions to prevent their future cancers.

Tobacco control has to be the highest priority for cancer prevention. In Western populations, dietary modification involves encouraging people to eat more plant-based foods, whereas in countries where adequate amounts of plant-based foods are eaten, it is important to prevent people from changing over to high consumption of animal-based foods. Known carcinogens must be avoided, and this is particularly critical for many developing countries, as there is a tendency to import new industry and the relevant carcinogens. Hepatitis B vaccination has been available for many years, and yet in countries where children need vaccination probably only about 1% have been vaccinated. Health promotion programs therefore have to be reinforced, and we have to learn how to impart information in such a way that people will act to preserve their own health.

## Screening in Cancer Control

When screening is proposed as a part of cancer control, only strategies known to be effective should be used in the general population. Effective programs can be run only on the basis of an educated target group. There have been many programs, particularly in developing countries, where the right educational background has not been provided to the public, for whom the concept of screening and prevention of death from cancer is foreign to their culture; thus, screening fails to reach those at greatest risk.

Programs have to be based on the natural history of the cancer. Part of the problem is that the natural history of the detectable precancerous phase may not be known until screening has been performed. Screening should be performed at the right ages and the right frequency. Screening for cancer of the cervix is not yet performed at the right frequency in Canada, so it is more expensive than it should be. Screening should always be conducted with attention to high quality, with adequate facilities and preferably in an organized setting. Fortunately, the Canadian breast cancer screening programs were set up in an organized way; the cervical cancer screening programs were not.

For breast cancer screening, there is continuing controversy over whether mammography screening is effective in women under the age of 50. However, it is now clear that in those for whom screening begins in their 40s there is no reduction in breast cancer deaths while they are in their 40s.[4–6] Two studies have suggested the effectiveness of breast self-examination in this age group.[7,8] These results, combined with evidence on the lack of benefit from adding mammography to breast physical examination and the teaching of breast

self-examination[9] as well as the early benefit found from screening women over the age of 50 in the first breast cancer screening trial,[10] suggest that the major benefit from screening derives from the earlier detection of relatively advanced, not early, disease.[11]

Even the best of the breast cancer screening trials comparing screening with no action did not reduce breast cancer deaths among women aged 50–69 by more than 30%.[4] There is a possibility that in the period since the trials were completed showing benefit from screening versus no screening to the years when modern treatments became available, i.e. adjuvant chemotherapy and tamoxifen, the component that benefited from screening and early detection is now cured by modern treatment. So improved treatment could remove the opportunity to observe a benefit from screening.

Turning to cervical cancer screening, there is now better evidence on the advantages of conservative management of low-grade lesions.[12] In this study, the records of the largest cytology laboratory in Toronto were used, and women who had a cytologic label of dysplasia, whatever the grade, were identified. The records were subsequently linked to the Ontario Cancer Registry. About 11% of the cases of mild dysplasia were found to progress to moderate dysplasia or worse in the following two years, and about 20%, in five years; for progression to severe dysplasia or worse, the figures were only 2% after two years and 6% after five. Among cases of moderate dysplasia, only 16% progressed to severe dysplasia or worse in two years, and 25%, in five. So the proportion of women with mild or moderate dysplasia in whom there was progressive disease is relatively small. The majority of women with mild or moderate dysplasia showed regression to normal within five years.

The Bethesda classification puts the moderate and severe dysplasias in one category: high-grade lesions (HSIL).[13] This is unfortunate, in that the line should have been drawn between moderate and severe dysplasia, and it results in a large number of women being overtreated at unnecessary cost. When young women are screened many will be found to have HPV effects, of which the large majority will regress untreated. To refer all women categorized as LSIL (mild dysplasia and HPV effects) is to risk substantial overtreatment; but even in the case of HSIL, many are also being overtreated, because the majority of the moderate dysplasias regress.

There is now good evidence that screening for colorectal cancer with the fecal occult blood test will reduce colorectal cancer mortality by about 20%.[14–17] Whether the same can be achieved by flexible sigmoidoscopy is still under investigation. However, other cancer control strategies may be having an impact on colorectal cancer. So to initiate screening now, when incidence and mortality from the disease is already on the downturn, is to risk a great deal of expenditure for a small impact.

Evaluating new screening technologies in the future is not likely to be easy, as the rapid dissemination of information and the pressures induced by commercial interests may appear to preclude the use of the preferred evaluation method: the large-scale, randomized screening trial. This is precisely the situation that is beginning to emerge over spiral computed tomography scanning for lung cancer, in which enthusiastic early reports[18] are fuelling demand from high-risk subjects, especially in the US. The window of opportunity for evaluating this approach may be very small.

This situation is similar to the confusing one that still exists over screening for prostate cancer using prostate-specific antigen (PSA) testing. Interpreting the rapid changes in prostate cancer incidence and mortality that have occurred is not easy, although it seems clear that there are many artifacts that could be responsible for the changes in mortality.[19–21] Only randomized trials of screening, however, will determine the precise causes of the changes and whether screening is effective. Fortunately, two large trials have nearly completed their intake.[22]

## Past Trends in Cancer Incidence and Mortality

Comparison of trends in both the incidence and mortality of the major cancer sites in Canada and other countries is informative. Incidence data are derived from the successive volumes of *Cancer Incidence in Five Continents* (published by the International Agency for Research on Cancer), and mortality data are from the WHO cancer mortality database. Canadian national data are available only from 1969, although for some provincial registries data were included from the first volume of *Cancer Incidence in Five Continents*, which relates to the period 1960–1962. In general, I include data from the United States, and sometimes from the United Kingdom and Japan in these comparisons, but if there are remarkable changes occurring in other countries that appear to facilitate interpretation of the trends (e.g. some of the Mediterranean countries in the case of diet-associated cancers), data from these are added also.

### *Colorectal Cancer*

The incidence of colorectal cancer has been rising in many countries (Figure 1), but in Canada and New Zealand there has been a recent decline among females. Declines have also been noted in the US.[23]

With regard to mortality, however, there have been declines in many countries over a considerable period. One exception is Japan (Figure 2), where the increase in mortality reflects the underlying increases in incidence. Similar trends are occurring in Spain and Greece, and to a lesser extent in Italy. France, however, is showing a reduction in colorectal cancer mortality.

**FIGURE 1**
**Trends in colorectal cancer incidence, New Zealand, Canada, Denmark and Finland**

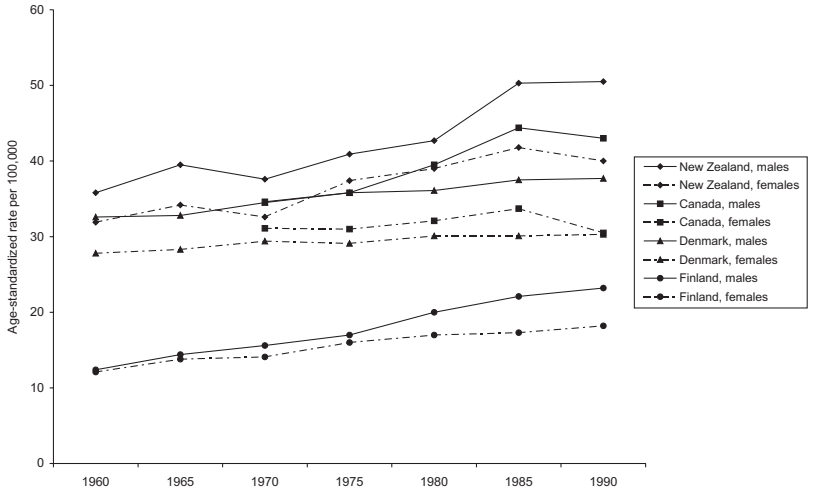



**FIGURE 2**
**Trends in colorectal cancer mortality, New Zealand, United Kingdom, Canada and Japan**

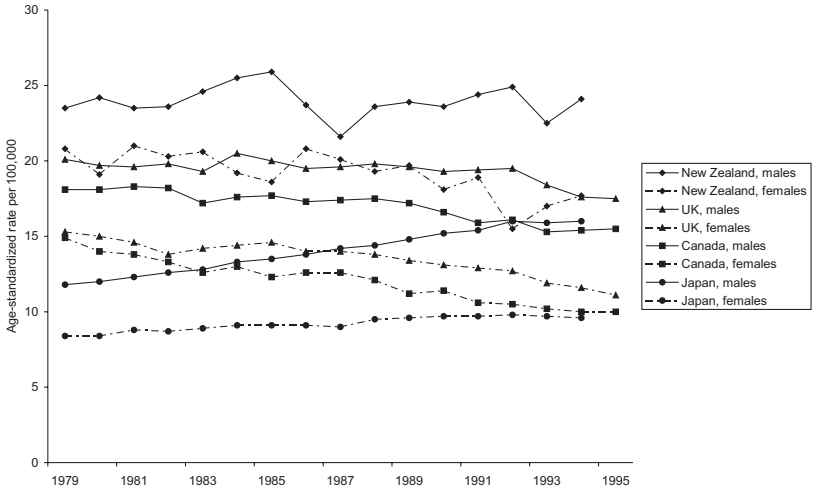



**FIGURE 3**
**Trends in colorectal cancer incidence and mortality, Canada**

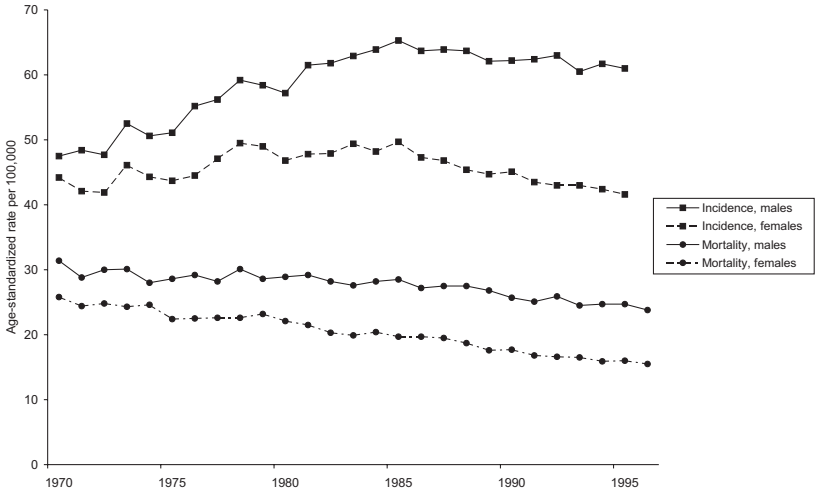


Figure 3 shows these trends together for Canada, based on data from *Canadian Cancer Statistics*. Decreases in both incidence and mortality can be attributed to the effects of primary prevention. The earlier rise in incidence in Canada could be artifactual, since there were improvements in cancer registration during this period, particularly in Quebec but also in some of the Atlantic provinces and probably in Alberta and Ontario. The other problem is that when cases (rather than people) are registered as having a disease in which multiple primary tumours are common, incidence will remain elevated as a result of duplicate or, in some instances, triplicate registrations.

Among the changes in diet that have occurred in the last two decades in Canada was one related to the standards of fat in beef. What was once regarded as of high quality, i.e. marbled beef with a lot of fat in it, came to be perceived as lower-quality beef; high-quality beef became lean beef. Subsequently, it is likely that many people made changes in their diet, initially induced by concern about heart disease, and then by the Canadian Cancer Society's campaign that began in the 1980s. Therefore, perhaps at least some, if not most, of the decline in both colorectal cancer mortality and incidence has been due to dietary modification.

**FIGURE 4**
**Trends in lung cancer mortality, United Kingdom, USA and Canada**

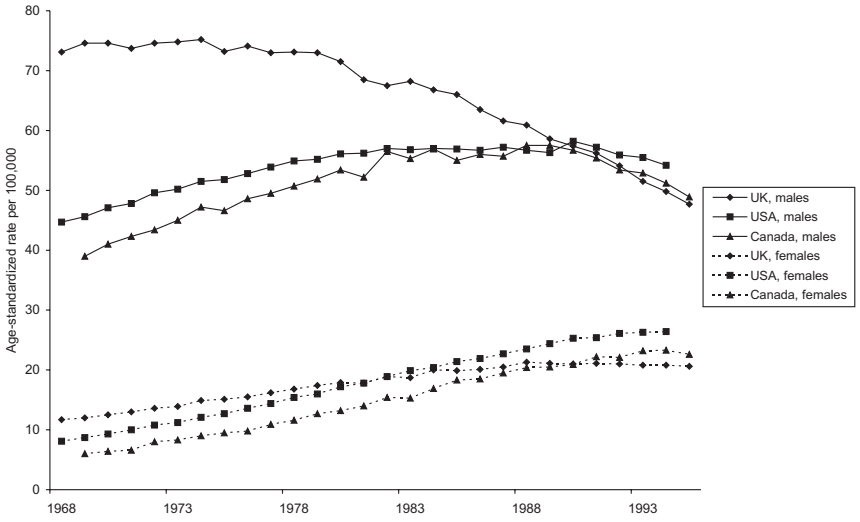
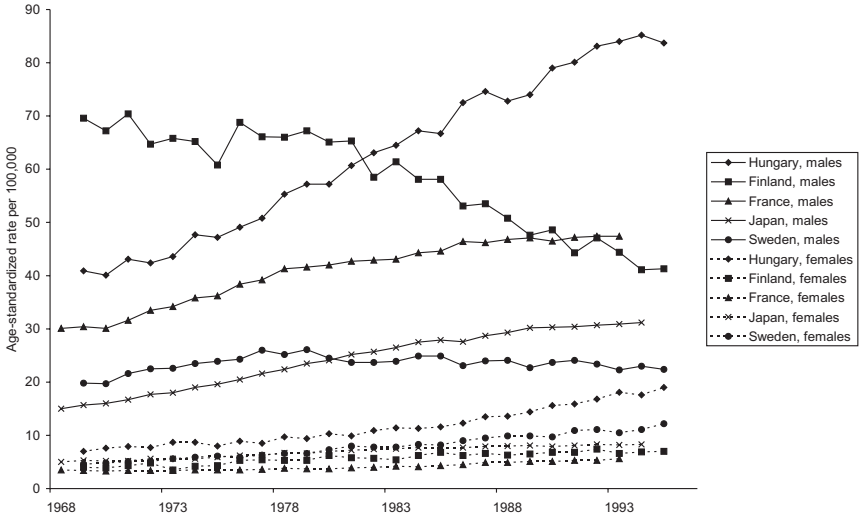



**FIGURE 5**
**Trends in lung cancer mortality, Hungary, Finland, France, Japan and Sweden**




### *Lung Cancer*

In the case of lung cancer we can restrict our attention to trends in mortality, as it so closely reflects incidence. Figure 4 contrasts the trends in lung cancer mortality in Canada, the UK and the US. Both in the US and Canada, there was a rise in lung cancer mortality among men and then a fall; neither reached the peak mortality in the UK, which occurred about 15 years earlier. Currently, however, lung cancer mortality among men in Canada is very similar to that in the UK, and lower than in the US. These trends in age-standardized rates are based on remarkable changes that have been largely cohort-related,[24] with the effect that rates among younger males have declined for several years, whereas those among older males have plateaued and declined only slightly.

Among women, mortality from lung cancer has been rising in both the US and Canada; the rates in Canada are a little lower than in the US. In the UK the rates were initially higher, but reached a peak about the middle of the 1980s and then fell, so that they are now below those in the US and Canada. There is a suggestion in both the US and Canada that rates may have peaked recently, but it is too early to be certain.

Figure 5 includes data from selected countries to illustrate some of the contrasting trends that are occurring. Finland shows similar reductions to the UK, but Hungary has now achieved the remarkable

distinction of having the highest reported lung cancer mortality in the world. France and Sweden seem to have avoided the major increases among males that occurred in North America and many other countries of Northern and Western Europe. Japan has overtaken Sweden in lung cancer rates among males but not females, and Hungary shows important increases among females.

### *Breast Cancer*

Most countries have been recording increases in the incidence of breast cancer. Canada is no exception to this trend (Figure 6). Much of this increase is likely to be the effect of early detection programs of various sorts, greater awareness among the public and professionals working in breast cancer and the introduction of mammography screening.

Canada has slightly higher breast cancer mortality rates than the US. For most of the period reviewed, rates have been stable, but recently there has been a fall (Figure 7). The UK displays a rise and then a fall. Some other countries have shown different trends, for example, increases in both Finland and Japan. There has been a tendency in the UK to claim that at least half of the decline has been due to the breast screening program. However, the time relationships of the declines do not suggest that they are related to screening. It is important to note that Sweden has not yet experienced anything like the decreases reported in the UK, Canada and the US, yet there were several major screening trials in Sweden, and Sweden was the first country to decide on organized breast cancer screening.

**FIGURE 6**
**Trends in breast cancer incidence, cancer registries of USA (Connecticut), Canada (Saskatchewan), Denmark, Finland and Japan (Miyagi)**

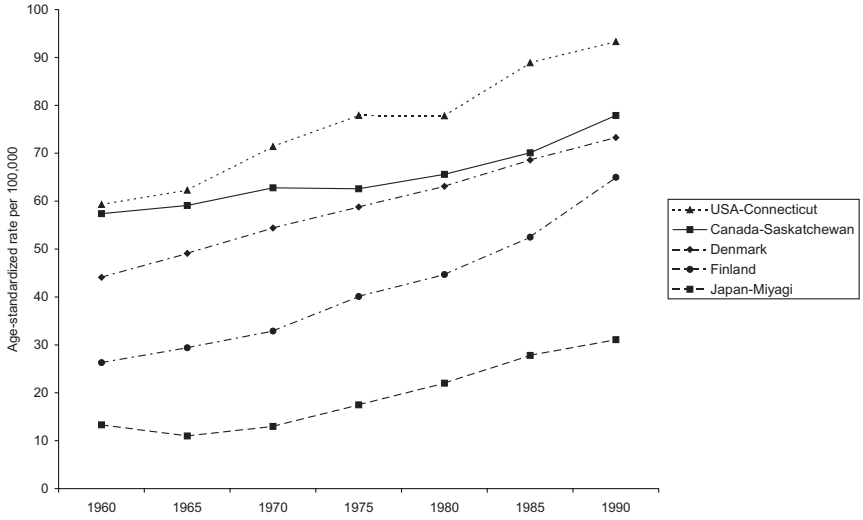
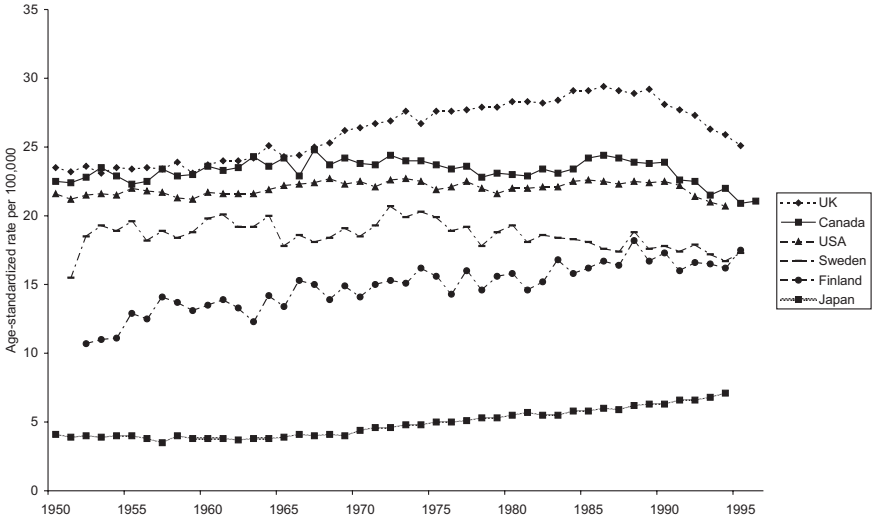



**FIGURE 7**
**Trends in breast cancer mortality, United Kingdom, Canada, USA, Sweden, Finland and Japan**




It takes nearly nine years for the impact of breast cancer screening on mortality in a population to be detectable, because most of the deaths that occur in the initial years after introduction of screening are among women whose breast cancers were diagnosed before the program was introduced. With the lack of a reduction in breast cancer mortality in Sweden, it seems unlikely that the reductions in the UK, the US and Canada are due to screening. Rather, they are probably due to the introduction of adjuvant chemotherapy for premenopausal women and tamoxifen for post-menopausal women. So we are probably seeing an impact of treatment on mortality, with the impact of screening, if any, yet to follow.

### *Cancer of the Cervix*

For some time, the incidence and mortality associated with cancer of the cervix has been falling in Canada. Much of the recent decline is attributable to screening, but possibly some of the earlier decline was due to improvements in early detection and treatment, as in Sweden.[25] The incidence of cervical cancer in Canada has fallen to a similar extent as in most of the Nordic countries, where major impacts of screening were observed except for Norway, which failed to introduce

organized programs (Figure 8). In Canada this resulted largely from annual screening, yet the impact of five-year screening in Finland and screening every three or four years in Sweden has been almost identical to our own. So cervical cancer screening has been successful in Canada, but at a cost— an opportunity cost.

Figure 9 depicts trends in cervical cancer mortality for selected countries. The UK, in spite of showing lower mortality than the US and Canada in the 1950s, had much slower downward trends, and only recently has this begun to accelerate. Much of the earlier fall in the UK was probably not due to screening.[26] The mortality trends in Denmark and Finland are as expected from the incidence trends in Figure 8. After a rise Japan shows a slow decline, and Israel's trend is stable, although there was a suggestion of a rise in the 1970s, which may have been aborted by the screening that was introduced.

## Future Canadian Trends in Cancer Incidence and Mortality

Figures 10–13 illustrate my projections of cancer incidence and mortality for Canada through to the year 2020. These have, in a few instances, been derived from a simple linear projection of recent trends, but for most, I have superimposed my understanding of recent changes and what I expect for the future. These are not based on any statistical model, but can be regarded as "educated guesses" on my part, with all the problems of validity that such guesses imply. Table 1 summarizes past trends and my projections.

Among females, I mainly anticipate stability or reductions in incidence of the major cancers (Figure 10). I do not foresee a continued increase in breast cancer incidence, since much of it in the past was artifactual. One might expect an increase a little later on in terms of the impact of the changes in fertility practices, but the early indications are that the baby boomers are not experiencing an increased breast cancer incidence in their younger years, which may be a result of improved diet in their early years or some other factor.

I anticipate an increase in female lung cancer incidence until about 2010, when it will probably start to drop. However, if the blip in 1995 turns out to be the beginning of a reduction in incidence, that projection will have been much too pessimistic.

I project that colorectal cancer incidence will continue to fall among females, but I think the reduction in cancer of the cervix has plateaued and will probably remain level because there has already been a major impact of screening on incidence in this country (Figure 8 and Table 1). In practice, the 60% reduction in incidence expected from the coverage with screening in place has already occurred, and to reduce it further, those at high risk outside the programs have to be screened. With regard to melanoma among females, I suspect the increase in incidence has largely ceased, but not so for non-Hodgkin's lymphoma, one of the major emerging problems.

**TABLE 1**
**Actual (1970–1996) and projected (1996–2020) changes in cancer incidence and mortality in Canada, by sex**

| Site | | 1970–1996 | 1996–2020 |
|---|---|---|---|
| **Stomach** | | | |
| Males: | Incidence | -43% | -8% |
| | Mortality | -59% | -9% |
| Females: | Incidence | -44% | -5% |
| | Mortality | -60% | -7% |
| **Colorectum** | | | |
| Males: | Incidence | +28% | -19% |
| | Mortality | -21% | -27% |
| Females: | Incidence | -6% | -30% |
| | Mortality | -38% | -41% |
| **Melanoma** | | | |
| Males: | Incidence | +206% | +12% |
| | Mortality | +108% | +8% |
| Females: | Incidence | +115% | +4% |
| | Mortality | +45% | +7% |
| **Lung** | | | |
| Males: | Incidence | +44% | -31% (-14%)[a] |
| | Mortality | +31% | -32% (-15%)[a] |
| Females: | Incidence | +344% | +22% |
| | Mortality | +270% | +23% |
| **Breast** | | | |
| Females: | Incidence | +27% | +2% |
| | Mortality | -8% | -18% |
| **Cervix** | | | |
| Females: | Incidence | -53% | -2% |
| | Mortality | -67% | -4% |
| **Body of uterus** | | | |
| Females: | Incidence | 0% | -12% |
| | Mortality | -32% | -12% |
| **Ovary** | | | |
| Females: | Incidence | -3% | 0% |
| | Mortality | -22% | -11% |
| **Prostate** | | | |
| Males: | Incidence | +123% | -3% |
| | Mortality | +19% | -5% |
| **Bladder** | | | |
| Males: | Incidence | +2% | -23% |
| | Mortality | -22% | -14% |
| **Non-Hodgkin's lymphoma** | | | |
| Males: | Incidence | +138% | +44% |
| | Mortality | +55% | -9% |
| Females: | Incidence | +113% | +29% |
| | Mortality | +49% | +12% |

[a] Pessimistic estimate

Figure 11 shows the projections for cancer mortality among females. As in the case of lung cancer incidence, unless the blip in 1995–1996 turns out to be the beginning of a fall (as in the UK), extending the previous increase would lead one to expect a continued increase to about 2010 before a decrease commences roughly five

years later. With regard to breast cancer, I predict a continued drop in mortality, initially from a continued treatment effect and then from the effect of screening. Colorectal cancer in females may not continue to fall to the extent shown, but at present there is no indication as to when the reduction in mortality will end.

Figure 12 presents projections of cancer incidence among males, and Figure 13, projections in mortality. For lung cancer I have projected continuing reductions in what I call my *optimistic* estimates, but reductions largely coming to an end by about 2010 in my *pessimistic* estimates. The latter scenario would require a reversal of the decline in smoking uptake and cessation of the effects of quitting in young adults, and hopefully both can be avoided. Prostate cancer incidence is already falling, probably from a saturation of the effect of PSA screening, but unless PSA screening ceases, incidence is likely to remain elevated and relatively stable. I am assuming relatively little decline in prostate cancer mortality, though that could turn out to be too pessimistic if screening has a larger effect than anticipated.

Similar to my projection for females, I predict a continued drop in male colorectal cancer and a continuing rise in non-Hodgkin's lymphoma, but to a higher level than in females. Better understanding the reasons for the growth in non-Hodgkin's lymphoma (possibly environmental factors[27]) and introducing corrective actions, if possible, are now major cancer control research priorities.

Both incidence and mortality for those cancers not included in my calculations will probably remain relatively stable. There will be some reductions, especially for the tobacco-related cancers. It is quite likely that the incidence of testis cancer will continue to rise, a situation for which we have no certain explanation, and that mesothelioma will continue to increase in both incidence and mortality.

**FIGURE 8**
**Trends in cervical cancer incidence, Denmark, Canada, Norway, Sweden and Finland**

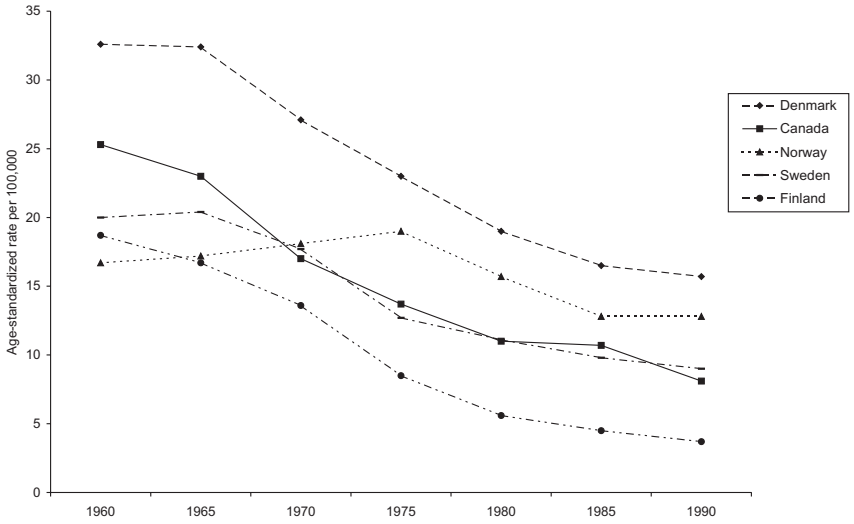
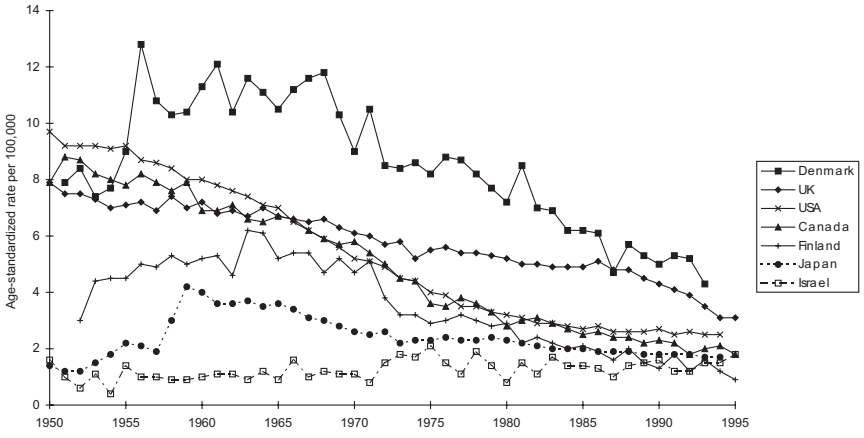



**FIGURE 9**
**Trends in cervical cancer mortality, Denmark, United Kingdom, USA, Canada, Finland, Japan and Israel**




## Discussion

There are clearly question marks over many of my projections. Will we continue to see a beneficial effect on tobacco-induced cancers? Will the current reversal of smoking rates in teenagers have a major adverse effect? Is the effect of diet as strong as I indicate? What will the effect of screening really be?

I am not too pessimistic about teenage smoking, in that it may be possible to influence behaviour after the teenage years in sufficient time to avoid major increases in cancer incidence and mortality. The studies on the effects of quitting smoking show that if people smoke only to the age of 30 or 35, there will be hardly any increase in their lung cancer risk. Quitting at the age of 40 will not have as strong an impact. If quitting does not occur until the age of 50, there is a major elevation in lifetime risk, since duration of smoking is so important.[2]

**FIGURE 10**
**Projections of trends in incidence of the major cancers in Canada, in females**

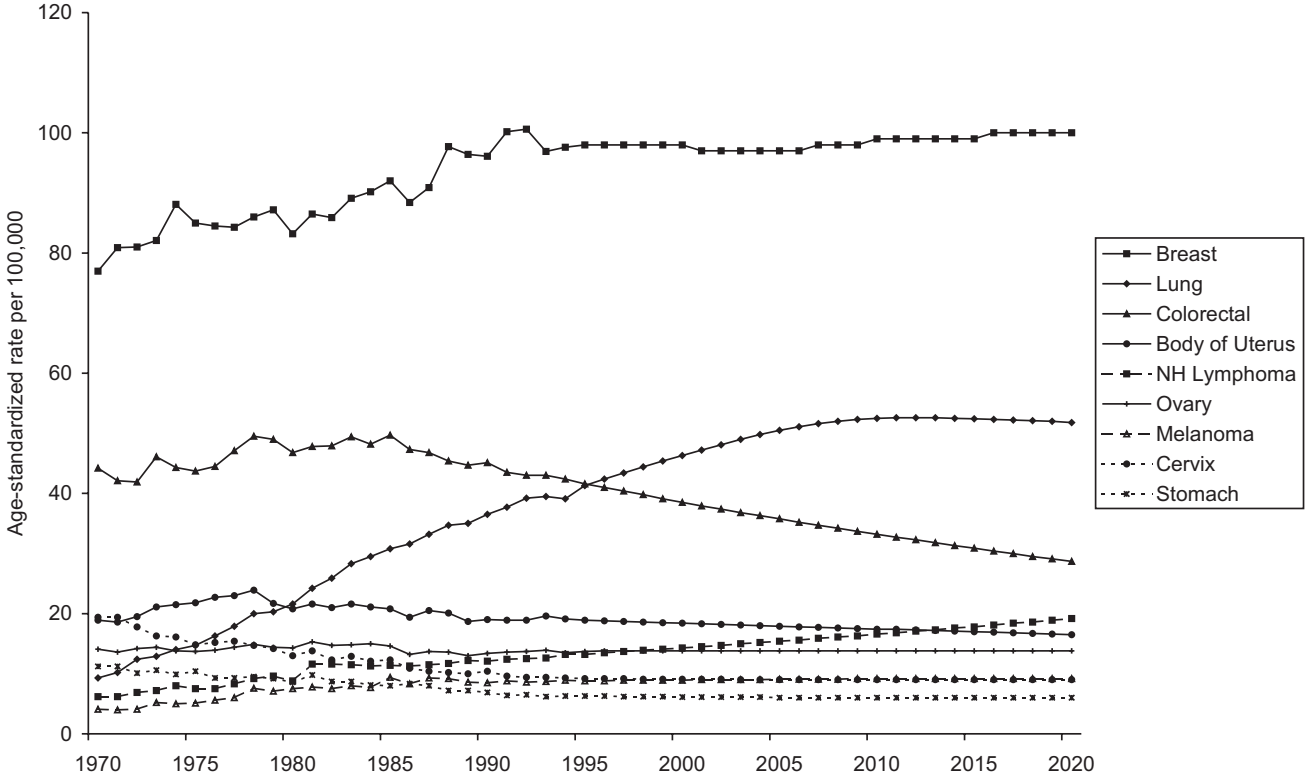



**FIGURE 11**
**Projections of trends in mortality from the major cancers in Canada, in females**

**FIGURE 12**
**Projections of trends in incidence of the major cancers in Canada, in males**

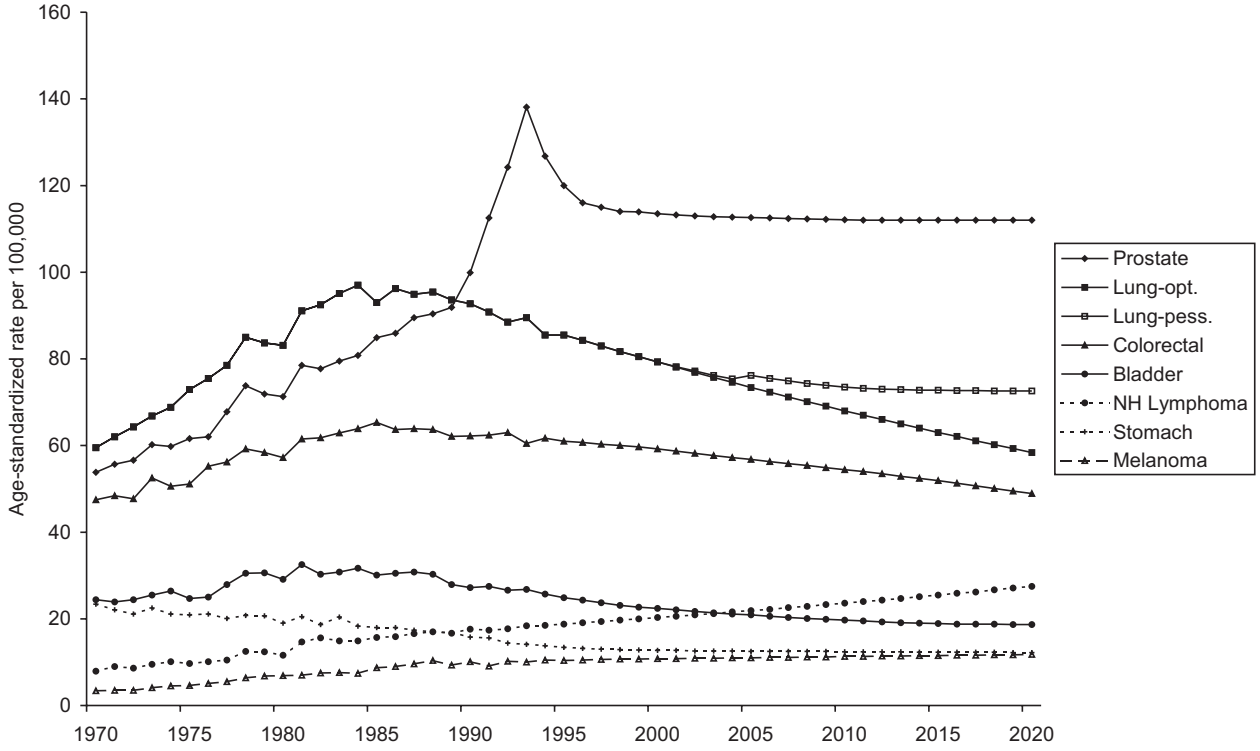



**FIGURE 13**
**Projections of trends in mortality from the major cancers in Canada, in males**

Thus there are dual challenges for tobacco control: prevention in uptake and acceleration in dealing with addiction, with a big challenge to ensure that young adults recognize the major benefits they will achieve if they stop smoking.

It is quite possible that the effects that nutrition has on some of the risk factors for breast cancer may result in a major future impact on breast cancer incidence. We know that Western-type nutrition has an impact on age of menarche and that age of menarche is related to breast cancer risk; this type of nutrition also has the effect of postponing age at menopause. The longer the ovaries function from menarche to menopause, the greater the breast cancer risk. Nutrition also has an impact on height. Height is clearly related to breast cancer risk, particularly in premenopausal women. Thus, the mechanism by which Western-type nutrition affects breast cancer is probably indirect, in that it has an impact on factors that themselves affect ovarian activity.

For colorectal cancer, the mechanism is probably completely different. Some of the evidence suggests that the impact of fibre or bulky foods is on the metabolism of carcinogens in the bowel. There may also be an indirect effect of fat through bile acids on colon mucosa, with necrosis of the superficial layers increasing the turnover of the cells in the base of the crypts, thus increasing the chance of errors occurring and cancers occurring. There is also some evidence that there is an effect mediated through insulin.

So, although we do not know enough about the mechanisms, we should not assume that they are identical for breast cancer and colorectal cancer. However, the expectation in terms of when changes in nutrition will show a beneficial effect are different: the effect on colorectal cancer incidence has probably already started; the effect on breast cancer incidence could be delayed, unless it is already being seen in the reduced risk among baby boomers.

Many are hoping for major advances from the human genome project. The proportion of cancers of our common sites caused by dominant genes of the BRCA1 and 2 type is likely to be low, however. For breast cancer, the proportion is probably about 5%, as it probably is for colorectal cancer, but for other cancers the proportion will be much less. Thus, in terms of these dominant genetic effects, there will not be a major population impact.

What seems to be increasingly likely is that genetic polymorphisms of various sorts will be identified that will begin to make it possible to get a better handle on an individual's susceptibility to various cancers. Whether that will then result in an improvement in our ability to control cancer generally is unclear. If a particular subgroup that is at increased risk of a specific cancer can be identified, it may be worthwhile to use certain drugs or other agents to prevent cancer in them, or to concentrate screening on them. That may make certain types of cancer control actions more cost-effective, but may not necessarily result in a greater impact in the population.

For screening, the great unknown with current tests is the extent to which the effects will be complementary to either prevention (for colorectal cancer) or treatment (for breast cancer). Only long-term monitoring of trends will reveal this. For prostate cancer, if the effect of early diagnosis is to improve the results of treatment of advanced disease (as is probably true for breast cancer), then it may be far more immediate than one might anticipate from the apparent long lead times gained by PSA screening and could become detectable very soon from downturns in prostate cancer mortality. For cancer of the cervix the challenge is, as it has been for over two decades, to reach those at risk and avoid overscreening those not at risk. With regard to lung cancer, there is increasing interest in helical CT scanning,[18] in part because of concern over the increasing proportion of cases of lung cancer occurring in North America in ex-smokers. However, large randomized trials are essential to decide whether that approach really works, and these will take some time to conduct.

In conclusion, therefore, there has been major success in Canada over the last 25 years in controlling stomach and cervical cancers, and in setting the stage for the eventual control of tobacco-associated cancers and probably some of the diet-associated cancers such as colorectal cancer. Apart from cervical cancer, this success comes from prevention, which deserves continuing high priority in terms of both research and application. We need to be cautious in our approach to screening, however. Success in cervical cancer screening was achieved at a greater cost than was probably necessary; the role of screening is uncertain for colorectal cancer, and as yet it is unknown for prostate cancer or lung cancer.

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*Short Report*

# *A Simple Method for Estimating Incidence from Prevalence*

*Gerry B Hill, William F Forbes and Jean Kozak*


## Abstract

*A two-state deterministic model is used to estimate the incidence of an irreversible disease from prevalence and mortality data. The method is simpler than those described previously. Diabetes and dementia are used as examples.*


***Key words:*** *dementia; diabetes; incidence; mortality; prevalence*

## Introduction

This paper deals with diseases that are non-communicable and irreversible. Most so-called "chronic diseases" are of this type. Even where treatment is available to reduce case fatality, such diseases are never cured. The age-specific prevalence of a disease can be measured in population surveys, either by interview or by examination, and the mortality of people with the disease can be obtained from following up the survey subjects or from cohort studies.

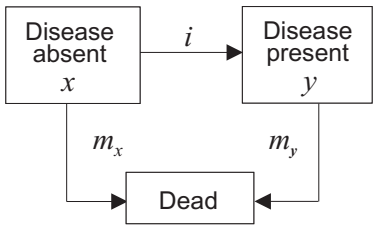
In the absence of a population-based disease registry, age-specific incidence is more difficult to estimate, since new cases are rare. However, in a stationary population with fixed incidence and mortality rates, the prevalence of a disease is a function of incidence and mortality, and if two elements are known the third can, in principle, be derived.

Several approaches to the problem have been suggested,[1–5] usually involving rather complex probabilistic models. We describe a simpler method, using a deterministic model, and illustrate its use with data on diabetes and dementia.

## Method

Figure 1 shows a two-state model of disease within a cohort. At a given age, *a, x(a)* is the number of people without the disease, *y(a)* is the number of people with the disease, *i(a)* is the incidence rate, and $m_x(a)$ and $m_y(a)$ are the mortality rates among those without and with the disease. (Note that *i* is the "true" incidence rate based on those without disease, not on the total population.)

**FIGURE 1**
**Cohort model of disease**




The prevalence of disease at the given age is then $p(a) = y(a)/\{x(a) + y(a)\}$. If $p'(a)$ is the slope of the age-specific prevalence curve, then it can be shown (see Appendix) that

(1) $i(a) = p'(a)/\{1 - p(a)\} + \{m_y(a) - m_x(a)\}p(a)$.

Also, if $m_y(a)/m_x(a) = r$, independent of *a*, and *m(a)* = the overall mortality rate, then

(2) $i(a) = p'(a)/\{1 - p(a)\} + (r - 1)p(a)m(a)/\{1 + (r - 1)p(a)\}$.

Since the estimates of age-specific prevalence are usually "noisy," it is necessary to smooth them, taking into account that *p(a)* must lie between 0 and 1 and, for most diseases, increases monotonically with age. A suitable smoothing function is the logistic $ln[p(a)/\{1 - p(a)\}] = c + b(a)$. Formula 1 (above) then simplifies to the following formula.

(3) $i(a) = \{b + m_y(a) - m_x(a)\}p(a)$

For diabetes, estimates of *p(a)* were taken from the 1994/95 National Population Health Survey,[6] the 1991 Canadian life table[7] was used to estimate *m(a)*, and the estimate of *r* was taken from US data.[8] For dementia, the Canadian Study of Health and Aging (CSHA)[9] provided estimates of *p(a)*, and $m_x(a)$ and $m_y(a)$ were derived from a follow-up of the CSHA subjects. The estimates of *i(a)* using Formula 3 were compared with provisional estimates of incidence from the CSHA (Canadian Study of Health and Aging Working Group, unpublished observations).


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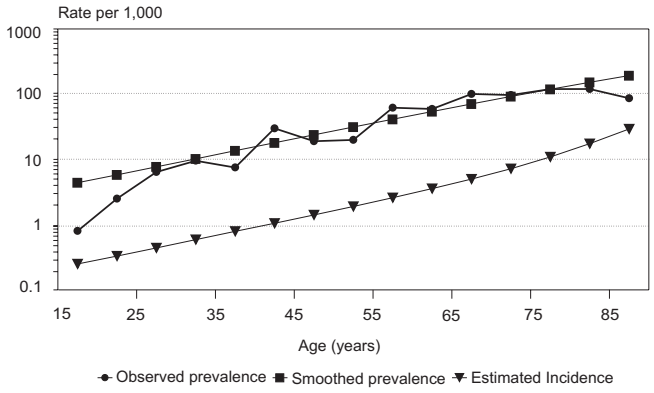
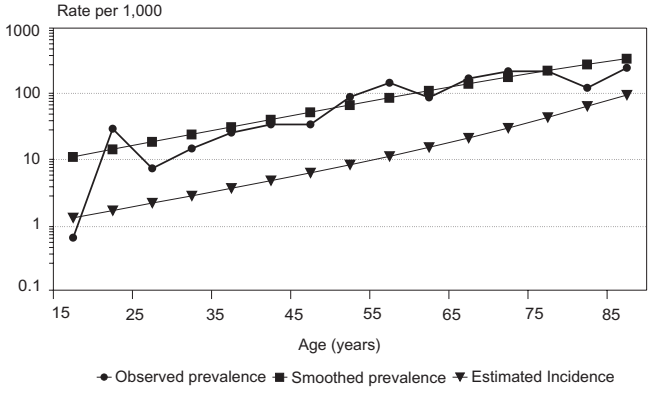
**FIGURE 2**
**Prevalence and incidence of diabetes in Canadian women**




Source: NPHS 1994 (Reference 6) and Kleinman et al., 1988 (Reference 8)

**FIGURE 3**
**Prevalence and incidence of diabetes in Canadian men**




Source: NPHS 1994 (Reference 6) and Kleinman et al., 1988 (Reference 8)

## Results

Figures 2 and 3 depict the observed prevalence of diabetes, the smoothed prevalence and the estimated incidence among Canadian women and men. The logistic function fits the observed prevalence reasonably well except at the extremes of the age range. Among women, the estimated incidence increases from 0.3 per 1,000 at ages 15–19 to 29 per 1,000 at ages 85–89. The corresponding estimates among men are 0.2 and 59 per 1,000.

Among elderly Canadian women and men, the observed incidence of dementia from the CSHA follow-up is compared with the incidence estimated from the initial prevalence and subsequent mortality (Figures 4 and 5). The two sets of estimates are similar, but the correspondence is better for women than for men.

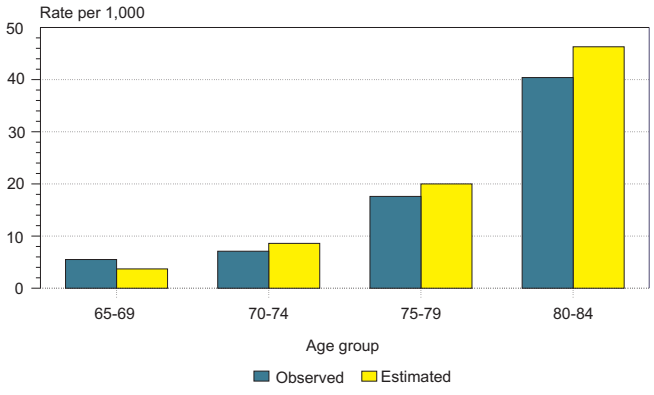
## Discussion

The method involves simple calculations, except for fitting the logistic function to the prevalence data, and programs for doing this are widely available. The use of the logistic is biologically plausible if we postulate, as in bioassays, that disease occurs as a result of a toxic dose that accumulates with age, and the tolerance of an individual (i.e. the lowest dose at which the disease occurs) is normally distributed. Then, conditional on survival, the functional relation between prevalence of disease and age would be that of the normal integral. The logistic is a good approximation to the normal integral and is easier to fit.

The incidence estimates for diabetes are consistent with the averages for developed countries,[10] but are somewhat lower than estimates for the United States.[11]

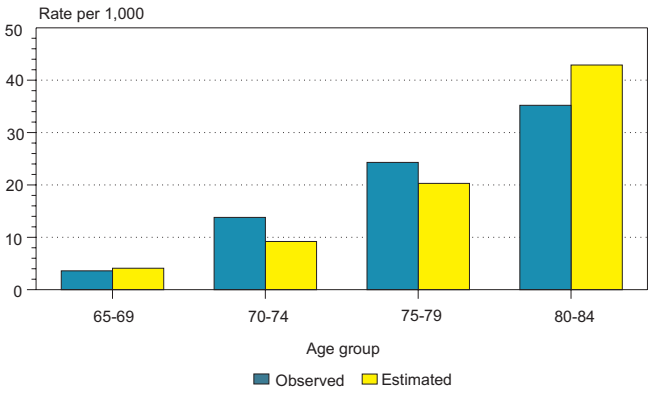
As mentioned, the model applies only to diseases that are irreversible, otherwise $x(a)$ would be replenished from $y(a)$. Thus, the method would be inappropriate for

**FIGURE 4**
**Incidence of dementia in Canadian women**




Source: Canadian Study of Health and Aging

**FIGURE 5**
**Incidence of dementia in Canadian men**




Source: Canadian Study of Health and Aging

some common chronic diseases (e.g. asthma, migraine, epilepsy) that tend to wane in middle age.

Even if the disease is irreversible, it is not necessarily the case that prevalence increases monotonically with age. In fact prevalence will decrease if $i(a) < \{m_y(a) - m_x(a)\}p(a)$. Thus the prevalence of the disease will decline with age if its incidence does not keep pace with the excess mortality associated with it. Examples would include congenital diseases and also some neurological diseases such as multiple sclerosis. In such circumstances the formulae for the estimation of incidence would still apply, but the use of the logistic function for smoothing would be inappropriate.

For some diseases, such as arthritis, and for some disabling conditions, such as deafness and blindness, there is no excess mortality. The formula for incidence then simplifies to $i(a) = p'(a)/\{1 - p(a)\}$.

At present we have direct estimates of the incidence of cancer derived from provincial cancer registries. In an ideal world we would have registries for all chronic diseases, but this would be very expensive. An alternative approach would be to link hospital discharge records and death records to form an electronic registry for each disease. This is feasible but is difficult to achieve at the national level because of confidentiality restrictions. Longitudinal follow-up of subjects in the National Population Health Survey may provide information on incidence as well as prevalence. In the interim, the approach suggested here may help to fill the gap.

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## APPENDIX

For convenience, we drop the notation indicating functional dependence on age.

(1) Differentiating $p$ with respect to age gives

$$p' = \{(x+y)y' - (x'+y')y\}/(x+y)^2 = (xy' - x'y)/(x+y)^2.$$

But from the model,

$$x' = -(i+m_x)x, \text{ and } y' = ix - m_y y.$$

So
$$\begin{aligned} p' &= \{ix^2 - m_y xy + (i + m_x)xy\}/(x + y)^2 \\ &= ix/(x + y) + (m_x - m_y)xy/(x + y)^2 \\ &= i(1 - p) + (m_x - m_y)p(1 - p). \end{aligned}$$

Hence $i = p'/(1 - p) + (m_y - m_x)p$.

Note that $p' >=< 0$ according as $i >=< (m_y - m_x)p$.

(2) By definition,

$$\begin{aligned} m &= (1 - p)m_x + pm_y \\ &= m_x\{(1 - p) + rp\} \\ &= m_x\{1 + (r - 1)p\}. \end{aligned}$$

Hence $(m_y - m_x)p = (r - 1)m_x p = (r - 1)pm/\{1 + (r - 1)p\}$. ■

# *Marital Status, Dementia and Institutional Residence Among Elderly Canadians: The Canadian Study of Health and Aging*

*Betsy Kristjansson, Barbara Helliwell, William F Forbes and Gerry B Hill*


## Abstract

*The association between marital status and mortality is well known; marital status has also been related to morbidity. In this paper, we examine the importance of marital status in relation to the presence or absence of dementia and to institutional residence, using data from the Canadian Study of Health and Aging. Three groups are compared: married, single and previously married. We show that the age-standardized prevalence of dementia and the proportions of elderly Canadians living in institutions with and without dementia are highest among single people and are also high for those who were previously married. These associations hold true for both women and men, but the relation between marital status and institutionalization is much stronger for men. Possible explanations and implications for the future care of the elderly are discussed.*


***Key words:*** *aging; Canada; dementia institutionalization; marital status*

## Introduction

The association between marriage and longevity has been recognized for at least a century.[1] Married people consistently have lower rates of mortality than single, widowed and divorced people of the same age and sex; these longevity benefits are greater for men than for women.[2–6]

A similar differential has been found for morbidity. Married people suffer fewer accidents and assaults,[7] have fewer acute and chronic conditions, fewer activity limitations, a lower probability of becoming disabled, less psychiatric morbidity, and lower physician and hospital utilization rates than those who are unmarried.[1,3,8–11] In general, widowed, divorced and separated people have the highest number of health problems, whereas people who remain single are only slightly more unhealthy than married people.[3,11] A person's sex confounds this relation: single men have more health problems than married men, but single women are no different than married women. Some studies have found that single women are healthier than married women.[3]

There is continuing debate as to whether this differential is due to the protective effect of marriage or to selection of healthy people into marriage and remarriage. Most researchers maintain that a combination of selection and causative factors are involved in producing this health differential.[2,3,9,12,13] Married people generally have more material resources, and the association between health and socio-economic status (SES) is well known. Marriage may also influence health through the provision of social support, which buffers the effects of stress. The presence of a caregiver may speed recovery after illness, reduce time in hospital and prevent admission to an institution.[2] Marriage also provides social control and regulation: married people indulge in fewer risk-taking behaviours, such as smoking and drinking.[2–4,13] Departures from the married state are extremely stressful.[2–4,9]

The greater health and longevity of married people are probably also due, in part, to the selection of healthy people into marriage and to the selection of unhealthy people out of marriage.[12] People who are seriously ill or disabled are less likely to marry because they are less


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desirable as partners;[3,4] chronic illness and disability may lead to marital dissolution.[1] Indirect selection through characteristics associated with health, such as SES, obesity and appearance, may also be at work.[12]

The relation between marital status and health is particularly important for Canada's elderly population because this group is at high risk for morbidity[14] and for cognitive and functional impairment, including dementia.[15,16] Dementia is a debilitating disease that impairs intellectual and functional capacity, and results in behaviour disturbance and personality change.[17] Because it is so debilitating, dementia is one of the most important reasons for institutional placement of older people.[18,19] Institutional care is often the only appropriate alternative for elderly people with severe cognitive and functional disabilities, but it is costly in both individual and societal terms.[20] As Canada's aging population increases, there will be growing pressure for institutional beds and increased interest in reducing or delaying admission to an institution.[14]

We know that institutionalization is largely due to severe declines in health, but it is also affected by a number of social factors, including SES and the availability of a caregiver. The study of social risk factors for institutionalization can help to identify people most at risk and suggest appropriate interventions. One risk factor may be the unmarried state; this is important because only 43% of elderly Canadian women and 77% of elderly Canadian men are married.[16]

Unmarried people are far more heavily represented in health care institutions than married people. In 1994 in Canada, fewer than 15% of the residents of health care institutions were married, whereas 60% of community residents in the same age group were married or living with a partner.[21] In the United States, single people have the highest rates of institutionalization and married people, the lowest; widowed, divorced and separated people are in between.[11] Multivariate analyses of the risks of institutionalization have generally shown an increased risk among the unmarried,[22,23] although results are inconsistent.[5,24] Many of these studies were limited to small clinical samples. As well, almost all of them dichotomized marital status, ignoring the distinction between single and previously married that has been so important in studies of other health states.

Most studies on the risks of institutionalization in patients with dementia have concentrated on behavioural, functional and cognitive risk factors; few have considered marital status. Two that did include marital status came from registries in the United States.[18,19] In one, the risk of institutionalization of unmarried patients with Alzheimer's disease was 2.7 times higher than that of those who were married.[18] In the other study,[19] marital status was significant for men only.

In this article we use data from the 1991 Canadian Study of Health and Aging (CSHA) to examine the importance of marital status in relation to institutionalization of elderly Canadians with and without dementia; we also consider differences in the prevalence of dementia. Three different marital states are studied: married, never married and previously married.

## Methods

As mentioned, the data studied come from the CSHA. Between February 1991 and May 1992, the CSHA recruited a sample of Canadian residents aged 65 and over: 9,008 living in the community and 1,255 living in institutions. The primary objective of the study was to estimate the prevalence of dementia in Canada. People living in the community were screened for cognitive impairment. Those failing the screening test and all those in institutions were offered a clinical and psychometric examination to determine the presence or absence of dementia. A description of the methods used has been published.[15]

The study produced estimates of the numbers of elderly women and men with and without dementia in Canada in 1991, by age group (65–74, 75–84 or 85+) and type of residence (community or institution). Marital status was one of the demographic characteristics included in the study, and we have used this to estimate, for those married, never married and previously married, the prevalence of dementia and the proportions of the populations with and without dementia living in institutions. The married group included people who were in common-law relationships. Because the numbers of divorced or separated people were too small to allow precise estimates, we combined them with the widowed to form a group labelled "previously married."

The sample for the present analyses included the entire CSHA sample ($n$ = 10,263), with the exception of community residents who were found to be cognitively impaired on screening but refused clinical examination ($n$ = 508). People for whom marital status or age was missing ($n$ = 21) were also excluded. Thus, the total sample size for these analyses was 9,734; 8,496 were community residents and 1,238 were institutional residents.

To obtain age-standardized rates of dementia, the proportions by marital status, cognitive status, sex, age and residence were projected onto estimates of the corresponding 1991 Canadian population by cognitive status, sex, age and residence (estimated from CSHA-1). Age-standardized rates per 1,000 were calculated using the indirect method. This standardization was carried out separately for women and men, so that their rates are not completely comparable; however, the comparison of interest was between the rates for the married, never married and previously married, and the ratio of these rates. Although age-standardized rates were calculated, differences in dementia prevalence and institutionalization by marital status were in the same direction in each age group.

**TABLE 1**
**Prevalence of dementia[a] among Canadians aged 65 and over, by marital status and sex, 1991**

| | Married | Single | Previously married |
|---|---|---|---|
| WOMEN | *n* = 1,834 | *n* = 618 | *n* = 3,491 |
| Rate | 71 | 116 | 97 |
| Ratio to married | 1.0 | 1.6 | 1.4 |
| MEN | *n* = 2,753 | *n* = 207 | *n* = 831 |
| Rate | 52 | 120 | 73 |
| Ratio to married | 1.0 | 2.3 | 1.4 |

[a] Age-standardized rate per 1,000

**TABLE 2**
**Proportion of Canadians aged 65 and over with dementia[a] living in institutions, by marital status and sex, 1991**

| | Married | Single | Previously married |
|---|---|---|---|
| WOMEN | *n* = 104 | *n* = 103 | *n* = 567 |
| Rate | 397 | 551 | 570 |
| Ratio to married | 1.0 | 1.4 | 1.4 |
| MEN | *n* = 179 | *n* = 39 | *n* = 131 |
| Rate | 296 | 726 | 577 |
| Ratio to married | 1.0 | 2.5 | 1.9 |

[a] Age-standardized rate per 1,000

**TABLE 3**
**Proportion of Canadians aged 65 and over without dementia[a] living in institutions, by marital status and sex, 1991**

| | Married | Single | Previously married |
|---|---|---|---|
| WOMEN | *n* = 1,730 | *n* = 515 | *n* = 2,924 |
| Rate | 11 | 78 | 45 |
| Ratio to married | 1.0 | 6.8 | 4.1 |
| MEN | *n* = 2,574 | *n* = 168 | *n* = 700 |
| Rate | 10 | 137 | 45 |
| Ratio to married | 1.0 | 13.4 | 4.4 |

[a] Age-standardized rate per 1,000

## Results

Table 1 shows that the prevalence of dementia among the never married (i.e. single) was higher than among the married and previously married for both sexes. The prevalence of dementia among the previously married was between that of the never married and the married. These findings are true for both women and men, but the ratios of the rates for the unmarried to the married are slightly higher for men than for women.

Table 2 shows the proportions of subjects with dementia who were living in institutions. The institutionalization rate of married people who had dementia was higher among women than men; this was reversed for single and previously married people. Institutionalization ratios were higher among the unmarried than the married, particularly for men. Among the people with dementia, the proportion of single men in institutions was higher than that of previously married men.

Table 3 shows the proportions of subjects without dementia living in institutions. Although the rate of institutionalization was much lower among people without dementia, the pattern by marital status was qualitatively the same as that among subjects with dementia. The unmarried-to-married ratios were much higher, however. For example, the rate among single men was 13.4 times that among married men, and among single women it was 6.8 times higher than the rate among married women.

## Discussion

In the present study, the prevalence of dementia was highest among single men and women and was also elevated among previously married people when compared with married people. Women had higher rates in general, but the differentials in rate ratios were greatest for men. A few other studies have also found an increased prevalence or incidence of dementia among unmarried people.[25,26]

It seems unlikely that being unmarried directly increases the risk of dementia, but this relation could be due to factors that are related to both marital status and dementia risk. For example, married people exhibit fewer risk behaviours; they also have higher income and education levels. Those with higher education have lower mortality rates and hence lower rates of widowhood, as well as lower rates of divorce.[27] In some studies education has been shown to protect against Alzheimer's disease.[28] On the other hand, selection is plausible. Indirect selection may operate in young adulthood and middle age, since the presence of risk behaviours that may lead to dementia would reduce the likelihood of marriage. In the elderly, direct selection may occur because the presence of cognitive impairment or dementia would also impede marriage. Marriage rates are much higher among elderly men than elderly women,[29] which could partly explain the sex difference in the prevalence ratio.

The association between marital status and institutional residence is consistent with other studies.[5,21–24] Again, the selection hypothesis is plausible, but it seems unlikely that it could produce such a marked difference in the rates, and spousal support is a more likely explanation. It is reasonable to suppose that such support would be less effective in preventing institutionalization for those who have dementia, and that husbands would be less able to provide support than wives. The higher rates for the never married compared with the previously married could be due to the presence of adult children.

Longitudinal studies are needed to determine the relative importance of selection and support. If support plays a major role, then the implications for the future need for institutional care are enormous. Fortunately, the proportion of elderly Canadians who are married is currently increasing, as a result of the decrease in mortality rates and widowhood.[29] This may change as younger cohorts with higher rates of divorce enter the ranks of the elderly. If they are to remain in the community, then methods should be found to provide support for those who are unmarried.

## Acknowledgements

The data reported in this article were collected as part of the Canadian Study of Health and Aging. This was funded by the Seniors Independence Research Program, administered by the National Health Research and Development Program of Health Canada (Project No 6606-3954-MC[S]). The study was co-ordinated through the University of Ottawa and Health Canada's Laboratory Centre for Disease Control. The first author is supported by a doctoral fellowship from the Alzheimer Society and also by the Social Sciences and Humanities Research Council.

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## Book Review

# Social Determinants of Health

Edited by Michael Marmot and Richard G Wilkinson
Oxford: Oxford University Press, 1999;
xiii + 291 pp; ISBN 0-19-263069-5; $55.50

North American epidemiology has been justly criticized for its emphasis on risk factors (mainly relating to behaviour, physical environment, and now genetics) and specific diseases, to the relative exclusion of broader determinants and outcomes. This excellent compilation demonstrates both the possibilities and the importance of a broader focus.

The 10 sections of a 1998 booklet from the European Office of the World Health Organization (*Social Determinants of Health—The Solid Facts*) have been expanded into 10 chapters: social organization and stress, early life, life course and social gradient, unemployment, psychosocial environment at work, transport, social support and cohesion, food, poverty and social exclusion, and social patterning of health behaviours. The opening and closing chapters by the very distinguished editors comprise an excellent overview in themselves, while the intervening chapters fill in the details.

Nearly all of the authors are from the United Kingdom (the remainder are also European), most of them from University College, London, a decision that limits the choice of authors but enhances the unity of the message; on balance, the advantages probably outweigh the limitations. As expected in a British book, the writing is generally literate and clear, and it is refreshingly free of disciplinary jargon.

The book covers one of the key elements of a population health approach, and does it more accessibly than Amick et al. (*Society and Health*)[1] and more comprehensively than Evans et al. (*Why are Some People Healthy and Others Not?*).[2] Almost every chapter contains something of interest, for example, a very clear explanation of the role of the sympatho-adrenal and the hypothalamic-pituitary-adrenal pathways in acute and chronic stress, and lots of good material on social inequalities in health.

The idea of life course pervades many of the chapters, explaining the importance of early childhood, while that of social exclusion is brought out very clearly in the chapter on poverty. Unfortunately, housing is addressed only as part of the chapters on social cohesion and poverty, and is not given a chapter of its own.

Most of the chapters are analytical in nature, looking for explanations of empirical evidence, whereas those on transport and food are strongly interventionist, urging changes in policy (and confirming many of my prejudices, which is always nice: "it is car driving that is unsafe, while cycling is much safer—the cause of an accident should not be attributed to the victim").

It is sobering to think of how many of the social determinants of health have been allowed or caused to deteriorate in Canada during the last 15 years or so. This book effectively demonstrates their importance, and it should give social epidemiology a shot in the arm.

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| **Overall rating:** | Excellent |
| **Strengths:** | Wide-ranging review of empirical evidence and discussion of its implications |
| **Weaknesses:** | Almost total lack of North American evidence (but then, we don't study social determinants much, do we?)<br>Too bad that medium-sized paperbacks now cost over $50 |
| **Audience:** | Epidemiologists, social scientists interested in health, public health policy makers |

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# New Resources

## Get On-line With Women's Health Experts

### Women's College and The Centre for Research in Women's Health launch Web site

On January 21, 2000, two internationally recognized leaders in women's health will launch a new Web site featuring the latest information on women's health.

The Centre for Research in Women's Health and the Women's College Campus, of Sunnybrook & Women's College Health Sciences Centre, will introduce <**www.womenshealthmatters.ca**> at the Women's Health Matters Forum & Expo. The site will provide consumers with the latest information, news and research findings on women's health, diseases and lifestyle.

This is the first Canadian Web site to focus on women's health issues and to offer reliable, evidence-based and up-to-date information on topics such as cancer, cardiovascular health and osteoporosis. All of the information published on the site will be provided, reviewed and approved by women's health experts from Women's College and The Centre for Research in Women's Health.

The Web site will also profile women's health experts from the two organizations, feature the latest developments in women's health research and provide an extensive directory of Canadian women's health resources.

Sunnybrook & Women's College Health Sciences Centre is an academic centre of excellence that, in partnership with its communities and fully affiliated with the University of Toronto, ensures a full range of high-quality, values-based, patient-centred services and is a leader in women's health.

The Centre for Research in Women's Health is a partnership of the Sunnybrook & Women's College Health Sciences Centre and the University of Toronto. Founded in 1995, it is commited to conducting and fostering women's health research, with a mandate to demonstrate "Leadership through Partnership."

*For further information*

Sheryl Mitchell
Director, Women's Health Partnerships
Tel: (416) 323-6321
E-mail: sheryl.mitchell@swchsc.on.ca

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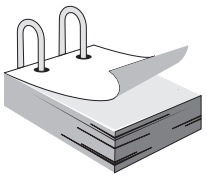

# Calendar of Events

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| **February 9–13, 2000**<br>**Washington, DC**<br>**USA** | "Addressing the Unequal Burden of Cancer"<br>7th Biennial Symposium on Minorities, the Medically Underserved and Cancer<br>Organized by the Intercultural Cancer Council | 7th Biennial Symposium<br>1720 Dryden, PMB-C<br>Houston, Texas<br>USA 77030<br>1-877-BIENNIAL (243-6642)<br>Tel: (713) 798-5383<br>Fax: (713) 798-3990<br>E-mail: symposium@bcm.tmc.edu<br><http://icc.bcm.tmc.edu/symposium> |
| **March 13–16, 2000**<br>**Quebec City, Quebec** | "Health and the Quality of Life: Our Municipalities in an Era of Globalization"<br>3rd Conference of Local Health Authorities of the Americas<br>Organized by *l'Institut national de santé publique du Québec* and the WHO Collaborating Centre for the Development of Healthy Cities and Villages | Secrétariat du 3e Congrès des responsables locaux de santé des Amériques<br>938, rue Saint-Maurice<br>Montréal (Québec) H3C 1L7<br>Tel: (514) 395-1808<br>Fax: (514) 395-1801<br>E-mail: 3econgres@opus3.com<br><http://www.msss.gouv.qc.ca/congres_quebec> |
| **April 17–20, 2000**<br>**New Orleans, Louisiana**<br>**USA** | CDC — Diabetes Translation Conference 2000<br>Centers for Disease Control and Prevention | Norma Loner<br>CDC/DDT<br>4770 Buford Highway NE, MS: K10<br>Atlanta, Georgia<br>USA 30341-3717<br>Tel: (770) 488-5376 |
| **April 18–20, 2000**<br>**New Orleans, Louisiana**<br>**USA** | "The Challenges of Cancer Surveillance in the New Millenium: Uniformity and Diversity"<br>North American Association of Central Cancer Registries (NAACCR) 2000 Annual Meeting | E-mail: pandre@lsumc.edu<br><www.naaccr.org> |
| **April 29–May 1, 2000**<br>**Victoria, British Columbia** | "Building Bridges: Creating an Integrated Approach to Women's Health"<br>Conference organized by the Health Association of BC and the Women's Health Bureau, BC Ministry of Health; other partners include Health Canada | Anne Speer<br>Women's Health Bureau<br>BC Ministry of Health<br>5-1, 1515 Blanshard St.<br>Victoria, BC V8W 3C8<br>Tel: (250) 952-2237<br>Fax: (250) 952-2799<br>E-mail: anne.speer@moh.hnet.bc.ca<br><www.health.gov.bc.ca/whb/bridges> |
| **May 7–10, 2000**<br>**Victoria, British Columbia** | "Science and Policy in Action"<br>First International Conference on Women, Heart Disease and Stroke<br>Heart and Stroke Foundation, American Heart Association, Health Canada and Centers for Disease Control and Prevention are providing early leadership | Taylor & Associates<br>18 – 5370 Canotek Road<br>Gloucester, Ontario K1J 9E8<br>Tel: (613) 747-0262<br>Fax: (613) 745-1846<br>E-mail: gtaylor@netrover.com |

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| **May 7–10, 2000**<br>**Ottawa, Ontario** | "Assessing Exposure to Disinfection By-products in Epidemiologic Studies: An International Workshop"<br>Sponsored by Health Canada and the US Environmental Protection Agency | Tye Arbuckle<br>Bureau of Reproductive and Child Health<br>Laboratory Centre for Disease Control<br>Health Canada, Tunney's Pasture<br>Address Locator: 0701D<br>Ottawa, Ontario K1A 0L2<br>E-mail: Tye_Arbuckle@hc-sc.gc.ca<br><http://www.hc-sc.gc.ca/hpb/lcdc/events/expo2000/index.html> |
| **May 16–19, 2000**<br>**Denver, Colorado**<br>**USA** | "Health Promotion Excellence in the New Century: Ascending New Heights"<br>18th National Conference on Health Promotion and Public Health Education and the 2000 SOPHE Midyear Scientific Meeting<br>Sponsors: Centers for Disease Control and Prevention, Association of State and Territorial Directors of Health Promotion and Public Health Education and Society for Public Health Education | <http://www.sophe.org><br><http://www.astdhpphe.org> |
| **May 28–30, 2000**<br>**Ottawa, Ontario** | "Charting the Course for Literacy and Health in the New Millennium"<br>First Canadian Conference on Literacy and Health<br>Organized by the Canadian Public Health Association's National Literacy and Health Program | CPHA Conference Department<br>400 – 1565 Carling Avenue<br>Ottawa, Ontario K1Z 8R1<br>Tel: (613) 725-3769<br>Fax: (613) 725-9826<br>E-mail: conferences@cpha.ca<br><www.nald.ca/nlhp.htm> |
| **June 11–13, 2000**<br>**Edmonton, Alberta** | "Statistics and Health 2000"<br>International conference organized by the Biostatistics Research Group (BRG), Statistics Centre, University of Alberta | KC Carrière (Program Committee Chair)<br>Tel: (780) 492-4230<br>Fax: (780) 492-6826<br>E-mail: BRG@stat.ualberta.ca<br><http://www.stat.ualberta.ca/~brg> |
| **June 14–18, 2000**<br>**Ottawa, Ontario** | "Beyond 2000: Healthy Tomorrows for Children and Youth"<br>Conference hosted by the Canadian Institute of Child Health, the Canadian Paediatric Society and the Canadian Academy of Child Psychiatry | Jackie Millette<br>Manager, Education Department<br>Canadian Paediatric Society<br>100 – 2204 Walkley Road<br>Ottawa, Ontario K1G 4G8<br>Tel: (613) 526-9397, ext 228<br>Fax: (613) 526-3332<br>E-mail: jackie@cps.ca<br><www.cps.ca/beyond2000> |
| **June 15–17, 2000**<br>**Seattle, Washington**<br>**USA** | 33rd Annual Meeting of the Society for Epidemiologic Research | Jacqueline C Brakey<br>Registration Co-ordinator, Conferences<br>University of Utah<br>Tel: (801) 581-5809<br>Fax: (801) 581-3165<br>E-mail: jbrakey@admin.dce.utah.edu<br><http://conferences.utah.edu> |

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| **August 23–27, 2000**<br>**Victoria, British Columbia** | ITCH 2000: "From Potential to Practice"<br>International Conference on Information Technology in Community Health | ITCH 2000<br>c/o School of Health Information Science<br>University of Victoria<br>PO Box 3050, STN CSC<br>Victoria, BC V8W 3P5<br>Tel: (250) 721-8576<br>Fax: (250) 472-4751<br>E-mail: itch@hsd.uvic.ca<br><http://itch.uvic.ca> |
| **October 22–25, 2000**<br>**Ottawa, Ontario** | "Health for All in the Year 2000"<br>Canadian Public Health Association<br>91st Annual Conference *and*<br>Ontario Public Health Association<br>51st Annual Conference | CPHA Conference Department<br>400 – 1565 Carling Avenue<br>Ottawa, Ontario K1Z 8R1<br>Tel: (613) 725-3769<br>Fax: (613) 725-9826<br>E-mail: conferences@cpha.ca<br><www.cpha.ca> |

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