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The burden of cancer attributable to modifiable risk factors in Canada: Methods overview

Darren R. Brenner[a,b,*], Christine M. Friedenreich[a,b], Yibing Ruan[b], Abbey E. Poirier[b], Stephen D. Walter[c], Will D. King[d], Eduardo L. Franco[e,f], Paul A. Demers[g], Paul J. Villeneuve[h], Xin Grevers[b], Robert Nuttall[i], Leah M. Smith[j], Karena D. Volesky[e,f], Dylan E. O'Sullivan[d], Prithwish De[k], on behalf of the ComPARe Study Team[1]

[a] Department of Oncology and Community Health Sciences, Cumming School of Medicine, University of Calgary, Calgary, Alberta, Canada
[b] Department of Cancer Epidemiology and Prevention Research, CancerControl Alberta, Alberta Health Services, Calgary, Alberta, Canada
[c] Department of Health Research Methods, Evidence, and Impact, McMaster University, Hamilton, Ontario, Canada
[d] Department of Public Health Sciences, Queen's University, Kingston, Ontario, Canada
[e] Department of Epidemiology, Biostatistics and Occupational Health, McGill University, Montreal, Quebec, Canada
[f] Gerald Bronfman Department of Oncology, Division of Cancer Epidemiology, McGill University, Montréal, Québec, Canada
[g] Occupational Cancer Research Centre, Cancer Care Ontario, Toronto, Ontario, Canada
[h] Department of Health Sciences, Carleton University, Ottawa, Ontario, Canada
[i] Health System Performance Branch, Health Quality Ontario (formerly Canadian Cancer Society), Toronto, Ontario, Canada
[j] Canadian Cancer Society, Toronto, Ontario, Canada
[k] Cancer Care Ontario, Toronto, Ontario, Canada

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ABSTRACT

Up-to-date estimates of current and projected future cancer burden attributable to various exposures are essential for planning and implementing cancer prevention initiatives. The Canadian Population Attributable Risk of Cancer (ComPARe) study was conducted to: i) estimate the number and proportion of cancers diagnosed among adults in Canada in 2015 that are attributable to modifiable risk factors and ii) project the future avoidable cancers by 2042 under various intervention targets. We estimated the population attributable risk (with 95% confidence intervals) and the potential impact fraction of cancers associated with selected lifestyle, environmental, and infectious factors. Exposure-specific sensitivity analyses were also completed where appropriate. Several exposures of interest included active and passive smoking, obesity and abdominal adiposity, leisure-time physical inactivity, sedentary behaviour, alcohol consumption, insufficient fruit and vegetable intake, red and processed meat consumption, air pollution (PM2.5, NO_2), indoor radon gas, ultraviolet radiation (UVR), hepatitis B and C virus, *Helicobacter pylori*, Epstein-Barr virus, human papillomavirus, human herpesvirus type 8 and human T-cell lymphotropic virus type 1. We used the 2015 cancer incidence data for 35 cancer sites from the Canadian Cancer Registry and projected cancer incidence to 2042 using historical data from 1983 to 2012. Here, we provide an overview of the data sources and methods used in estimating the current and future cancer burden in Canada. Specific methodologic details for each exposure are included in the individual articles included as part of this special issue.

* Corresponding author at: Department of Cancer Epidemiology and Prevention Research, CancerControl Alberta, Alberta Health Services, Holy Cross Centre – Room 513C, Box ACB, 2210-2nd St. SW, Calgary, AB T2S 3C3, Canada.
E-mail address: Darren.Brenner@ucalgary.ca (D.R. Brenner).

[1] Additional members of the ComPARe Study Team: Elizabeth Holmes, Canadian Cancer Society, Toronto, Ontario, Canada; Zeinab El-Masri, Cancer Care Ontario, Toronto, Ontario, Canada; Mariam El-Zein, Gerald Bronfman Department of Oncology, Division of Cancer Epidemiology, McGill University, Montréal, Québec, Canada; Tasha Narain, Department of Public Health Sciences, Queen's University, Kingston, Ontario, Canada; Priyanka Gogna, Department of Public Health Sciences, Queen's University, Kingston, Ontario, Canada.

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1. Introduction

Prevention is essential in reducing the long-term population burden of cancer. Evidence on the role of lifestyle, environmental and occupational factors in cancer etiology and the burden of cancer associated with these exposures is necessary for decision-makers and public health practitioners to identify priority focus areas in prevention. Comprehensive studies on the burden of disease such as the Global Burden of Disease Study (Fitzmaurice et al., 2015) bring a focus on public health management of chronic diseases which, in turn, affects how much attention is given to cancer. Quantifying the impact of cancer, especially in relation to other chronic conditions, helps evaluate the profile of this disease and allows decision-makers to allocate the right balance of resources to address initiatives aimed at decreasing the societal burden of cancer. Canadian data from international studies such as the Global Burden of Disease (Forouzanfar et al., 2015) show that among the top 15 risk factors for injury and disease, the highest cancer burden (as measured by daily adjusted life-years [DALYs]) can be attributed to tobacco smoking, diet, physical inactivity, obesity, alcohol, occupation and pollution from ambient particulate matter. While the Global Burden of Disease study identified priority exposures for chronic disease, it did not include population attributable risks (PARs) for cancer incidence or DALYs for specific cancers, nor did it incorporate the most detailed and up-to-date exposure information. This information is particularly important for planning as new cancer cases in Canada are projected to rise to over 277,000 in 2032, imposing a large burden on the Canadian healthcare system and economy, as well as having important implications for the quality of life of those affected (Xie et al., 2015).

Many cancers are caused by risk factors related to lifestyle, environment, infectious agents, or occupational exposures which can be controlled or even eliminated (Parkin et al., 2011a; Renehan et al., 2010; Schutze et al., 2011; Whiteman et al., 2015; Whiteman and Wilson, 2016). Previous studies have used PAR methods to estimate the cancer burden attributed to a single risk factor in Canada (Brenner, 2014; Chen et al., 2012; Zakaria and Shaw, 2017) or, in some cases, multiple risk factors, for some Canadian provinces (Cancer Care Ontario, 2014, 2015; Grundy et al., 2017). PAR, also referred to as Population Attributable Fraction, requires a measure of risk and the prevalence of an exposure of interest. Efforts to quantify the cancer burden attributable to various modifiable risk factors have been recently published for the United Kingdom (Parkin et al., 2011a), United States (Colditz and Wei, 2012; Schottenfeld et al., 2013), Australia (Whiteman et al., 2015), France (Shield et al., 2018a; Shield et al., 2018b; Shield et al., 2018c), and Brazil (Azevedo et al., 2016). Limitations of previously published PAR estimates include the lack of modelling of the joint effects of risk factors on cancer and, in particular, the ability to estimate future potential to prevent cancer, which are both addressed in the current series of articles. The need for country-specific estimates are further complicated by the fact that PAR estimates cannot be extrapolated to other jurisdictions because of differences in populations' age and sex distributions (which are related to exposure risk), prevalence of risk factors, definition of cancer types and risk exposures, availability and precise measurement of exposure prevalence, and how timely those exposure measures are.

The articles in this special issue report on the findings from the Canadian Population Attributable Risk of Cancer (ComPARe) study, which provides comprehensive estimates for Canada and its provinces and addresses several of the methodologic issues previously mentioned. In addition to deriving PAR estimates, we also calculated the potential impact fraction (PIF), which measures the proportion of disease incidence that could be prevented under a hypothetical intervention scenario.

The ComPARe study also included an integrated knowledge translation (iKT) component, through which key stakeholders (knowledge users) were involved in elements of the study design as well as in the strategic dissemination of the findings. For the PAR estimates to be useful for cancer prevention, a clear vision for the uptake and implementation of the findings is needed, which was supported by knowledge users participating in this research from its inception. Details of the knowledge translation approach in ComPARe are described in this special issue (El-Masri et al., 2019).

The ComPARe study had two aims: (1) to estimate PAR of cancers in Canada in 2015 that were attributable to selected modifiable lifestyle, environmental, and infectious exposures; and (2) to project the future attributable cancer burden in Canada up to 2042 and estimate the PIF under various evidence-based interventions and possible exposure reduction targets. The PARs and PIFs provide population-specific, quantitative estimates of the prevention impact of different risk factors on cancer and serve as valuable information that can be used to inform policy, allocate resources, and prioritize prevention activities, now and in the future.

The objective of this paper is to provide an overview of the methods used in common throughout the articles included in this special issue, and the methodologic considerations for individual risk factors. Details of the methodologic framework have been previously published (Brenner et al., 2018). Although occupational exposures are important to consider, they are being considered in a separate project (Occupational Cancer Research Centre, 2017) and are not covered here.

2. Methods

2.1. Risk factor selection and risk estimates

We reviewed the International Agency for Research on Cancer's (IARC) Monographs and the World Cancer Research Fund's (WCRF)/American Institute of Cancer Research (AICR) Continuous Update Project reports to identify modifiable lifestyle, environmental and infectious risk factors with known, probable, convincing or sufficient evidence of an association with cancer. Measures of exposure-cancer associations (relative risks or odds ratios) were abstracted. Additional exposure-cancer associations, not reviewed by IARC or WCRF/AICR but suggested by recent studies, were identified through our own reviews of recently published, high-quality systematic reviews and meta-analyses published up to August 2017 from which risk estimates were also abstracted into standardized templates. We excluded exposures for which the evidence was inconsistent, as characterized by high levels of heterogeneity in the literature or disagreement between meta-analyses published within a short time frame. All risk estimates were adjusted for confounding factors. When a summary risk estimate for an exposure of interest was not available such as for air pollution, radon, ultraviolet radiation (UVR) risk behaviours (indoor tanning, sunburn, and sunbathing), leisure-time sedentary behaviour, Epstein-Barr virus, *Helicobacter pylori*, and human papillomavirus (HPV) – we conducted our own meta-analysis of published studies on a specific cancer, the details of which are provided in the individual articles in this series.

2.2. Exposure prevalence

We estimated the prevalence of lifestyle and environmental exposures among Canadians mainly using population-based national health surveys, including the Canadian Community Health Survey (CCHS) (Statistic Canada, 2018), the Canadian Health Measures Survey (CHMS) (Statistics Canada, 2018) and the Second National Sun Survey (NSS2) (Marrett et al., 2010) (Table 1). Wherever the data were available, the prevalence was estimated by sex, 5-year age groups, and at both national and provincial level. For most exposures, we also identified the theoretical minimal exposure level for reducing the risk of cancer (Table 1). For estimation of PAR we attempted to identify data with a plausible latency period based on a compromise between exposure periods as reported in the literature and data availability. For most exposures, we adopted a latency of 12–15 years, i.e., we assumed

Table 1
Risk factors, theoretical optimum exposure level, sources of prevalence data, and associated cancers.

Risk factor	Exposure level for cancer risk reduction	Sources of exposure prevalence data (year of collection or publication)	Associated cancers
Active smoking	None	CCHS cycle 3.1 (2003)	Lung, oral cavity, larynx, esophagus, stomach, liver, pancreas, colon and rectum, kidney, bladder, ureter, ovary, cervix, breast, acute myeloid leukemia
Passive smoking	None	CCHS cycle 3.1 (2003)	Lung, colon and rectum, cervix, breast
Excess weight (as measured by body mass index)	≤25 kg/m^2	CCHS cycle 3.1 (2003)	Esophagus (ADC), stomach (cardia), liver, pancreas, colon, rectum, kidney, gallbladder, multiple myeloma, thyroid, breast (post-menopausal), ovary, endometrium, prostate (advanced)
High waist circumference/Waist-to-hip ratio	Men: < 94 cm; < 0.9 Women: < 80 cm; < 0.8	CHMS cycle 1 (2007–2009) and cycle 2 (2009–2011)	Colon and rectum, kidney, pancreas, thyroid, breast (post-menopausal), endometrium, prostate (advanced)
Lack of physical activity	≥3.0 kcal/kg/day	CCHS cycle 3.1 (2003)	Breast (post-menopausal), lung, endometrium, colon, rectum, kidney, bladder, esophagus (ADC), stomach, liver, small intestine, myeloid leukemia, multiple myeloma, head and neck, non-Hodgkin lymphoma
Sedentary behaviour	< 3 h/day	CCHS cycle 3.1 (2003)	Breast, colon and rectum, endometrium, ovary
Alcohol	None	CCHS cycle 3.1 (2003)	Breast, colon and rectum, oral cavity, larynx, liver, esophagus (SCC), pancreas, stomach
Low fruit	≥4 servings (400 g) per day	CCHS cycle 3.1 (2003)	Pancreas, bladder, lung, colon and rectum, breast, stomach, esophagus (SCC)
Low vegetable	≥4 servings (320 g) per day	CCHS cycle 3.1 (2003)	Pancreas, bladder, lung, colon and rectum, head and neck, ovary, liver, esophagus (ADC)
Low dietary calcium	≥1000 mg per day	CHMS cycle 1 (2007–2009) and cycle 2 (2009–2011)	Colon and rectum
Low serum 25-hydroxy vitamin D	> 50 nmol/L	CHMS cycle 1 (2007–2009) and cycle 2 (2009–2011)	Colon and rectum, breast, lung, bladder, kidney
Red and processed meat	None	CHMS cycle 1 (2007–2009) and cycle 2 (2009–2011)	Colon and rectum, pancreas, breast, stomach (non-cardia for processed meat), esophagus (SCC, processed meat only)
Ultraviolet radiation	1920 birth cohort	National Cancer Incidence Reporting System (melanoma incidence 1971–1991) and Canadian Cancer Registry (melanoma incidence 1992–2015)	Melanoma
Ultraviolet radiation risk behaviours (indoor tanning, sunburn, sunbathing)	None	NSS2 (2006)	Melanoma
$PM_{2.5}$	Zero	Hystad et al., 2011[a]	Lung
Radon	Zero	Health Canada (Cross-Sectional Survey of Radon Concentrations in Homes-2012)	Lung
Hepatitis B virus	None	CHMS cycle 1 (2007–2009) and cycle 2 (2009–2011) and NHANES (2007–2009) and (2009–2010)	Hepatocellular carcinoma
Hepatitis C virus	None	Trubnikov, Yan, and Archibald 2014[b] and Remis, 2009[c]	Hepatocellular carcinoma, non-Hodgkin lymphoma
Helicobacter pylori	None	NHANES data re-weighted to Canadian age, sex, and ethnic distribution	Mucosa-associated lymphoid tissue gastric lymphoma, stomach (non-cardia)
Epstein-Barr virus	None	Individual studies were meta-analyzed for each cancer	Burkitt lymphoma, Hodgkin lymphoma Extranodal natural killer T-cell lymphoma- nasal type, nasopharynx
Human papillomavirus	None	Individual studies were meta-analyzed for each cancer	Anus, cervix, penis, vagina, vulva, oropharynx, oral cavity, larynx
Human herpesvirus, type 8	None	None[d]	Kaposi sarcoma, primary effusion lymphoma
Human T-cell lymphotropic virus, type 1	None	None[d]	Adult T-cell leukemia/lymphoma

Abbreviations: ADC = adenocarcinoma, CCHS = Canadian Community Health Survey, CHMS = Canadian Health Measures Survey, NHANES = National Health and Nutrition Examination Survey, SCC = squamous cell carcinoma.

[a] (Hystad et al., 2011).

[b] (Trubnikov et al., 2014).

[c] (Remis, 2009).

[d] As the infections are necessary causes or part of the diagnostic criteria for the cancers they are associated with, no prevalence data were required, and 100% of the associated cancers are attributable to the infection.

that the cancer incidence in 2015 was due to the exposure prevalence in 2000–2003.

For several infections, Epstein-Barr virus, HPV, human herpesvirus type 8 (also called Kaposi sarcoma virus), and human T-cell lymphotropic virus type 1, we estimated the prevalence of the infection within the cancer tissue (i.e. among cancer cases).. To estimate the Canadian population prevalence of chronic hepatitis B and C viruses and *Helicobacter pylori*, we used a combination of prevalence figures from modelling studies, the CHMS, and the National Health and Nutrition Examination Survey (NHANES) in the USA. For the latter, exposure prevalence was reweighted to represent the age, sex, and ethnicity distribution of the Canadian population.

For environmental factors (e.g. air pollution, UVR, radon) for which the entire population is exposed, specific methods were used to estimate PAR, including the risk distribution approach (Murray et al., 2003), birth cohort comparison (Parkin et al., 2011b) and the Biological Effects of Ionizing Radiation VI risk model (Hunter et al., 2015). Details on each of these exposures are included in the individual manuscripts in

this supplement.

2.3. Cancer incidence

We obtained cancer incidence data for the year 2015 from the Canadian Cancer Registry (CCR) at Statistics Canada. Cancers in the CCR are coded using the International Classification of Diseases for Oncology, 3rd Edition (ICD-O-3). The 2015 incidence data were the most recent data available at the time of the study for all Canadian provinces and territories, except for the province of Quebec where data were available for the year 2010. Consequently, we imputed the 2015 cancer incidence in Quebec (by cancer type, sex and five-year age group) by fitting a Poisson regression on the incidence cancer rate in Canada (excluding Quebec) from 2008 to 2015. For infection-associated cancer sites with fewer than 500 cases in Canada in 2015, we averaged the last five years of Quebec's available incidence data (2005–2010) to estimate the number of cancer cases in Quebec in 2015. The annual incidence rate ratio was used to estimate the 2015 Quebec incidence rate and the number of cancer cases.

2.4. Estimation of attributable cancer cases

For risk factors with prevalence data at the population-level, PAR was calculated by Levin's equation (Levin, 1953):

$$\mathrm{PAR} = \frac{\sum_x \mathrm{P_x}(\mathrm{RR_x} - 1)}{1 + \sum_x \mathrm{P_x}(\mathrm{RR_x} - 1)}$$

where P_x is the prevalence within each category x and RR_x is the RR for each risk category relative to the reference category.

For risk factors with very high relative risks, or the risk factor is a necessary cause or part of the diagnostic criteria for its associated cancer, such as Epstein-Barr virus, HPV, human herpesvirus type 8 (also called Kaposi sarcoma virus), and human T-cell lymphotropic virus type 1, the prevalence of the exposure in cancer cases (Pc) can approximate the PAR (Miettinen, 1974):

$$PAR = Pc$$

We calculated the confidence intervals of the PAR estimates to address statistical uncertainty. For dichotomized risk factors, we used the equation derived by Walter (Walter, 2010), which can be used when the prevalence and RR come from different datasets, to calculate the variance of PAR and estimate the confidence interval:

$$var[\ln(1 - PAR)] = PAR^2\left[\frac{1}{P^2}varP + \left(\frac{RR}{RR - 1}\right)^2 var[lnRR] + \frac{2}{P}\left(\frac{RR}{RR - 1}\right)cov\left(P, lnRR\right)\right]$$

where *var* and *cov* are the variance and covariance, respectively. For risk factors with multiple categories, we applied the Monte-Carlo simulation method as previously described (Brenner et al., 2018). When tested on dichotomized risk factors, the formula above and the Monte-Carlo method produced very close confidence intervals.

2.5. Projection of future prevalence

We projected the future prevalence up to 2032 of active and passive smoking, body mass index, low physical activity, sedentary behaviour, alcohol consumption, red and processed meat intake, and low fruit and vegetable intake based on data from three cycles of the National Population Health Survey (1994–1998) (Statistic Canada, 2012) – the predecessor of the CCHS – and five cycles of CCHS (2000−2011). (Statistic Canada, 2018) Using data from 1993 to 2014, we projected the future average air pollution levels (particulate matter < 2.5 μm in diameter; $PM_{2.5}$) to 2036 using Canadian-wide historical satellite data at a 1000 by 1000 (m) resolution. Our projections were based on the assumption that the trend observed from the historical data will continue into the future without reversion (i.e., from decreasing to increasing, or vice versa). The selected projection method had to fulfill two conditions: i) the projected prevalence must be bound between 0 and 1, and ii) the sum of prevalence for each year must be equal to 1 for multi-leveled exposures. For exposures with limited availability of historical data, including UVR risk behaviours, radon, and hepatitis B virus and human papillomavirus, we assumed that the future prevalence will remain constant from the most recent year(s) of observed data onward.

2.6. Projection of cancer incidence

Using established methods for cancer projections, we projected the future cancer incidence in Canada for all relevant cancer sites, the details of which have been published elsewhere (Poirier et al., 2019a). Canadian cancer incidences were projected to 2042 and provincial cancer incidences were projected to 2038 based on 1983–2012 cancer incidence data and the available projected populations for modelling from Statistics Canada. Briefly, a data-driven decision algorithm to choose the most appropriate model for the projection was used. Models considered included: age-only, age-period (including common trend and age-specific trend), age-cohort and Nordpred (age-drift-period-cohort) (Moller, 2002). All projected results were evaluated for statistical goodness-of-fit using the Pearson's Chi-squared test as well as on face validity based on expert opinion.

2.7. Estimation of avoidable future cancer cases

Evidence-based intervention targets from the scientific literature were used for estimating PIF under varying scenarios. The appropriateness and relevance of these scenarios were further validated through consultation with key informants from the study's stakeholders. We also included “benchmark” targets for all lifestyle and environmental risk factors such as 10%, 25%, and 50% relative reductions in prevalence. These targets were specifically included to allow policy and decision makers to examine multiple alternative (best case) scenarios. Depending on whether the intervention targets were assumed to change the prevalence of exposure, the distribution of risk factor, or the relative risks, we used proportions shift, distribution shift, or RR shift methods to estimate PIF (Table 2) (Brenner et al., 2018). For PIF estimations, using proportions shift and distribution shift, we assumed a fixed 10-year latency period between the prevalence of exposure and cancer incidence for lifestyle factors, and shorter or longer periods for environmental risk factors, depending on the exposure in question. When using RR shift methods, we assumed that the change in RR occurred in a log-linear fashion over a 10-year period. A summary of the various assumptions used in the PIF analyses is included in Table 2.

2.8. Multiple risk factors and sensitivity analyses

A summary article in this supplement provides the combined PAR values and number of attributable cancer cases for all of the risk factors within an exposure category (e.g., for lifestyle risk factors) and for all exposure categories combined (i.e., lifestyle, environment, and infections). The following equation was used to estimate the combined PAR under the assumption that the prevalence of the risk factors are independent and the joint relative risks are multiplicative (Steenland and Armstrong, 2006):

$$\mathrm{PAR_{combined}} = 1 - \prod (1 - \mathrm{PAR_i})$$

However, we showed elsewhere that the magnitude of bias in the estimated combined PAR varied by prevalence and RR values and the degree to which the two assumptions are violated.(Poirier et al., 2019b)

Table 2
Potential Impact Fraction (PIF) modelling methods and the applicable risk factors.

Method	Formula	Risk factors	Assumptions
Proportions shift	$PIF = \frac{\sum_c P_c RR_c - \sum_c P_c^* RR_c}{\sum_c P_c RR_c}$	Active and passive smoking, physical inactivity, sedentary behaviour, alcohol, insufficient fruit and vegetable, insufficient fibre, insufficient dietary calcium, insufficient serum vitamin D, UVR risk behaviours, radon	Intervention changes the prevalence of the risk factor across population.
Distribution shift	$PIF = \frac{\int RR(x)P(x)dx - \int RR(x)P^*(x)dx}{\int RR(x)P(x)dx}$	Body mass index, air pollution	Intervention changes the prevalence of the risk factor across population. Relative risk is a function of the level of exposure.
Relative risk shift	$PIF = \frac{\sum_c P_c RR_c - \sum_c P_c RR_c^*}{\sum_c P_c RR_c}$	Waist circumference, waist-to-hip ratio, alcohol, insufficient fruit and vegetable, red and processed meat	The distribution of risk factor remains unchanged across population. The relative risk for each level of exposure decreases due to intervention.

P_c is the prevalence of exposure in category c; P_c^* is the prevalence of exposure in category c after intervention; $P(x)$ is the distribution of exposure in the form of a continuous variable; $P^*(x)$ is the distribution of exposure after intervention; $RR(x)$ is the relative risk as a function of the level of exposure; RR_c^* is the relative risk of exposure in category c after intervention.

In acknowledging this limitation, we conducted sensitivity analyses to address potential bias of the combined PAR under different scenarios of risk factor correlations and risk interactions.

Additional sensitivity analyses appropriate for individual risk factors are further described in each article in this supplement. In brief, these addressed the following issues: 1) prevalence data and RRs derived from different sources may result in different PAR estimates. For example, prevalence data on body mass index were collected from both CCHS and CHMS. A sensitivity analysis was carried out to compare the difference; 2) self-reported prevalence data may have been biased and adjustment would thus be needed. For example, we incorporated the studies that addressed the self-reporting bias in body mass index, alcohol consumption, and inadequate physical activity, and carried out the sensitivity analyses that estimated PAR after adjusting for the bias; and 3) missing data in population-based surveys may lead to an underestimation of the true prevalence. For example, various degrees of missing data were observed for body mass index, inadequate physical activity, and leisure-time sedentary behaviour. Sensitivity analyses were carried out under both missing-at-random and missing-not-at-random assumptions.

Ethics approval was granted for this project by the Health Research Ethics Board of Alberta - Cancer Committee (HREBA.CC-14-0220_REN4).

3. Discussion

Several overall limitations must be acknowledged when interpreting our results. First, exposure prevalence data from surveys were self-reported and are subject to measurement errors or exposure misclassification. For exposures such as *H. pylori* infection, the prevalence was extrapolated form the United States. Second, we attempted to obtain risk estimates that were adjusted for confounding factors, but it is possible that some of them may not be adequately adjusted for other important factors (e.g. we obtained risk estimates of physical inactivity adjusted for confounding factors such as body mass indexes and diet. However, sedentary behaviour as an important factor was not taken into account.). Third, we had to assume a latency period between the exposures and the onset of cancer, and this period may not reflect the true length of time for the natural history from exposure to disease diagnosis. Fourth, our projected burden of cancer based on assumptions regarding the projected future exposure prevalence and cancer incidence may not accurately predict future events. Finally, although Canada has a complete population-based cancer registry with over 95% case ascertainment in most provincial registries, the 2015 Quebec data were not available. These data had to be projected using 2010 data and numerous assumptions were applied.

While recognizing the above methodologic limitations, this study has several strengths: a comprehensive review of the extant epidemiologic literature on the associations between modifiable risk factors and cancer incidence; use of population-based national surveys that included province-level exposure estimates; use of a well-developed model for estimating future cancer incidence; and the estimation of both current and future burden under a series of counterfactual scenarios that were carefully vetted and reviewed by knowledge users who represented various organizations involved in chronic disease prevention and control. Our analyses also recognised emerging exposures such as sedentary behaviour and abdominal adiposity, which can help public health practitioners pre-empt the growing impact of these exposures on cancer. Most importantly, this study represents the first ever comprehensive assessment of current and future cancer burden attributable to a wide range of risk factors for Canada, including province-specific and sex-specific results by cancer site. More detailed discussions on specific methodologic aspects of this study pertaining to the cancer projections and modelling of multiple risk factors, as well as the overall methodologic framework for this study, are provided elsewhere (Brenner et al., 2018; Poirier et al., 2019a; Poirier et al., 2019b).

3.1. Conclusion

The ComPARe study is the first comprehensive evaluation of the attributable burden of cancer in Canada. We relied on widely-used methods in the epidemiologic research (Parkin, 2011), with several improvements. In particular, we extended the attributable risk estimation into the future and incorporated a range of intervention targets based on systematically reviewed evidence. This approach resulted in PIF estimates that support interventions which could be immediately feasible to ones that are aspirational goals for cancer control strategies in Canada. We also integrated a robust knowledge translation component from the onset of our study to allow for more directed planning and dissemination of our findings.

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Conflicts of interest

None declared.

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