

Alcohol-attributable burden of disease and injury in Canada, 2004

Kevin D. Shield · Tara Kehoe · Ben Taylor ·
Jayadeep Patra · Jürgen Rehm

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Abstract

Objective This analysis aimed to estimate the burden of disease and injury caused and prevented by alcohol in 2004 for Canadians aged 0–69 years and compare the effects of different magnitudes of adjustment of survey data on these estimates.

Methods Alcohol indicators were obtained from the Canadian Alcohol and Drug Use Monitoring Survey 2008 and were corrected to 80% coverage using adult per capita recorded and unrecorded consumption. Risk relations were taken from meta-analyses. Estimates of burden of disease and injury were obtained from the World Health Organization. **Results** In 2004, 4,721 (95% CI 1,432–8,150) deaths and 274,663 (95% CI 201,397–352,432) disability-adjusted life years lost (DALYs) of Canadians 0–69 years of age were attributable to alcohol. This represented 7.1% (95% CI 2.1–12.2%) of all deaths and 9.3% (95% CI 6.8–11.9%) of DALYs for this age range. The sensitivity analysis showed that the outcome estimates varied substantially based on the adjusted coverage rate.

Conclusion More attention to burden of disease and injury statistics is required to accurately characterize alcohol-related harms. This burden is preventable and could be reduced by implementation of more effective policies.

Keywords Alcohol · Canada · Burden of disease · Mortality · Morbidity · Disability

Introduction

Alcohol consumption is a substantial risk factor for mortality and morbidity in Canada (Patra et al. 2007; Rehm et al. 2006; Single et al. 1999) and globally (Rehm et al. 2009a, b), with more than 30 International Classification of Diseases version 10 (ICD-10) three-digit codes containing alcohol in their name and more than 200 ICD-10 three-digit codes where alcohol is a component cause (Rehm et al. 2010a; World Health Organization 2007). To address the substantial burden of disease and injury attributable to alcohol at the World Health Organization's (WHO) 63rd World Health Assembly, a global strategy to reduce the harmful use of alcohol was agreed upon. This agreement calls for member states to collect and disseminate data on alcohol consumption and alcohol-related harms (World Health Organization 2010). Currently, alcohol consumption is estimated to be the eighth leading cause of mortality, responsible for 3.8% of all mortality globally, and the third leading cause of burden of disease as measured in disability-adjusted life years lost (DALYs), responsible for 4.5% of all DALYs globally (World Health Organization 2009). This relative proportional difference of 18% between mortality on the one hand and mortality and disability on the other hand arises from alcohol-attributable mental health disorders, in particular from alcohol use

K. D. Shield (✉) · T. Kehoe · B. Taylor · J. Patra · J. Rehm
Centre for Addiction and Mental Health, 33 Russell Street,
Toronto, ON M5S 2S1, Canada
e-mail: Kevin.shield@utoronto.ca

K. D. Shield · B. Taylor · J. Rehm
Dalla Lana School of Public Health, University of Toronto,
Toronto, Canada

T. Kehoe
Department of Statistics, University of Toronto,
Toronto, Canada

J. Rehm
Institute for Clinical Psychology and Psychotherapy,
Technische Universität Dresden, Dresden, Germany

disorders, that are more associated with long-term non-fatal consequences when compared to other chronic diseases such as heart disease or cancer (Rehm et al. 2010a). Thus, it is important for Canada to monitor not only alcohol-attributable mortality-based outcomes but also the non-fatal consequences of alcohol consumption measured through indicators such as DALYs (Beaglehole and Bonita 2009; Casswell and Thamarangsi 2009; Gilmore 2009). DALY is a gap measure of the burden of disease combining years of life lost due to premature mortality with years of life lost due to disability and is not only used in research by the Global Burden of Disease (GBD) study but also as the standard measure to characterize population health by the WHO and the World Bank.

To estimate alcohol-attributable harms such as mortality and DALYs, data on average daily alcohol consumption, drinking patterns, risk relations, mortality, and burden of disease are needed. These data are then used in combination to calculate the alcohol-attributable fraction (AAF), defined as the proportion of the disease in the population that would disappear if alcohol consumption in a population were zero (Eide and Heuch 2001). However, exposure data on alcohol consumption from population surveys when compared to per capita consumption typically underestimate alcohol consumption by 30–70% (Rehm et al. 2007). This undercoverage of alcohol consumption is due to non-response to the alcohol survey, the construct validity of alcohol consumption measures, response bias, and the populations excluded from the survey. Thus, using unadjusted alcohol consumption data from surveys such as the Canadian Alcohol and Drug Use Monitoring Survey (CADUMS) 2008, where a coverage rate of 27% was observed when compared to overall per capita consumption in Canada as measured from taxation, sales, and producers' reports, will lead to a gross underestimation of alcohol-related harms (Shield and Rehm 2009). To adjust for this undercoverage, recent methods developed by Rehm et al. (2010c) allow for the adjustment of survey data by modeling alcohol consumption.

This paper aimed to:

1. estimate and discuss the burden of disease and injury for 2004 for Canadians 0–69 years of age and
2. explore the assumptions of such estimates by quantifying and discussing different magnitudes of adjustment of survey data for Canada through a sensitivity analysis.

Methods

Exposure estimates

Alcohol consumption was calculated from the CADUMS 2008. This survey was utilized since it is the largest survey on alcohol-specific measures closest to 2004 in Canada; it

has been conducted since 2008, and it is planned to be conducted annually in the future. This allows for estimates on consumption and alcohol-related harms derived from this study to be compared to future estimates. The exact methods of the CADUMS 2008 are described elsewhere (Health Canada 2008, 2009). Briefly, the CADUMS 2008 was a random digit dialing telephone survey that contacted 43,328 households over an 8-month period (of whom 16,674 individuals responded). Of these survey respondents, 16,640 provided a valid age and sex and were included in the survey. A posteriori weighting of the CADUMS 2008 survey participants was performed by age, sex, and region by triangulating the survey information with the 2006 Canadian census. Of the weighted survey participants, 15,531 individuals provided an alcohol consumption estimate and a specific age, resulting in an overall participation rate of 36.5%.

Drinking status was defined in the CADUMS 2008 as “current drinkers,” “former drinkers” (individuals who consumed at least one drink in their lifetime, but no alcohol in the last year), and “lifetime abstainers” (individuals who had never consumed alcohol). Alcohol intake in the CADUMS 2008 was measured in terms of standard drinks consumed in the 7 days prior to the survey; a standard drink in Canada was defined as 13.6 g of pure alcohol (Canadian Centre on Substance Abuse 2004; Health Canada 2009). Based on the volume of alcohol consumed according to the CADUMS 2008, we calculated the number of drinking occasions per week, the grams of alcohol per occasion, and the average grams of alcohol consumed per day.

Recorded alcohol consumption per adult for Canada was based on government records. Unrecorded alcohol consumption was calculated based on Macdonald et al.'s (1999) study indicating that 19.5% of total alcohol consumption is unrecorded. When compared to the Canadian adult per capita recorded alcohol consumption in 2008, the CADUMS exhibited a coverage rate of 27% of recorded and unrecorded per capita consumption. Based on expert opinion, data concerning grams of alcohol consumed per day were upshifted by multiplying the mean intake by 80% of the inverse of the coverage rate for recorded and unrecorded consumption since we assumed that part of the 20% of the estimated per capita consumption estimate of alcohol was not actually consumed (due to wastage or spillage) and to account for undercoverage observed in the studies used to calculate the relative risks (RRs).

The prevalence of alcohol consumed by women while pregnant (13.3%) was estimated from a systematic review of the literature (including Canadian surveys) performed by the Public Health Agency of Canada (PHAC 2007). Drinking estimates for pregnant women were calculated

weighting consumption estimates by the prevalence of births among women of different age groups.

Modeling alcohol consumption

Using 1,001 average daily alcohol consumption distributions from over 66 different countries, Rehm et al. (2010c) have shown that alcohol consumption in most cases is best modeled through a gamma distribution. Before modeling alcohol consumption with a gamma distribution we checked that the distribution of alcohol consumption in the CADUMS 2008 followed a gamma distribution.

By regressing over 500 means (μ) and standard deviations (σ), Rehm et al. (2010c) found that the standard deviation of a distribution of alcohol consumption could be expressed as a function of the mean as follows:

$$\sigma_{\text{men}} = 1.171\mu_{\text{men}} \quad (1)$$

$$\sigma_{\text{women}} = 1.258\mu_{\text{women}} \quad (2)$$

We used the standard deviations to calculate the shape and scale parameters for the upshifted group means, which were then used to characterize the upshifted gamma distribution.

Outcome categories and estimates

We used both incidence- (mortality) and time-based measures (DALYs) of public health in our analysis. Mortality and morbidity categories were defined by the 2005 GBD study (<http://www.globalburden.org/gbdops.html>). The WHO provided measures, including mortality, potential years of life lost (PYLLs), and years lived with disability (YLDs) for 2004, the most recent year available. DALYs were calculated using age-weighting and time-discounting methodology, as outlined in the World Health Report 2004 (World Health Organization 2009). Methods and data used to estimate mortality in the GBD project are described elsewhere (Institute for Health Metrics and Evaluation 2010; Mathers et al. 2003). Population estimates for 2004 were based on the latest revisions by the United Nations Population Division (2007). Age groups used in the analysis were 0–14, 15–29, 30–44, 45–59, and 60–69 years. Analysis of the burden of disease for people 70+ was not performed because cause of death and the RR functions were not considered accurate for these individuals.

Risk relations

The sources for the RR functions by GBD code and graphs showing select examples of RR curves are provided in the appendix. An outline of the causal relationship between alcohol and these GBD code categories is described in

detail elsewhere (Rehm et al. 2010a). Alcohol-attributable harms were with one exception (TB) calculated using RRs from meta-analyses which reported a continuous RR function obtained using fractional polynomial regression.

Calculating the alcohol-attributable fractions

The AAFs for each cause of death and morbidity were calculated by sex and age, taking into account the distribution of alcohol consumption and the prevalence of different drinking statuses (“current drinkers,” “former drinkers,” and “lifetime abstainers”) as follows:

$$\text{AAF} = \frac{P_{\text{abs}} + P_{\text{form}}\text{RR}_{\text{form}} + \int_{0+}^{150} P(x)\text{RR}(x)dx - 1}{P_{\text{abs}} + P_{\text{form}}\text{RR}_{\text{form}} + \int_{0+}^{150} P(x)\text{RR}(x)dx} \quad (3)$$

where P_{abs} represents the proportion of “lifetime abstainers,” P_{form} the proportion of “former drinkers,” and $P(x)$ the probability distribution function of drinkers. RR_{form} represents the RR for “former drinkers,” and $\text{RR}(x)$ the RR function for a given alcohol consumption in grams per day. A cap at an exposure of 150 g of pure alcohol was used as a conservative measure as very few individuals consume more than 12 standard drinks on a daily basis for an extended period of time.

Risk estimates for injuries were calculated by combining the calculated AAFs for average alcohol intake and alcohol intake on binge drinking occasions according to Taylor et al. (2008a, b). The grams of alcohol consumed on average were modeled by a gamma distribution, with the AAFs calculated using Eq. 3 adjusting the RR used for time at risk (see Eq. 5). We calculated the AAFs for injuries attributable to binge drinking as follows:

AAF

$$= \frac{P_{\text{abs+former}} + P_{\text{current(Non-Binge)}} + P_{\text{current(Binge)}}\text{RR}(x) - 1}{P_{\text{abs+former}} + P_{\text{current(Non-Binge)}} + P_{\text{current(Binge)}}\text{RR}(x)} \quad (4)$$

where $P_{\text{abs+former}}$ is the proportion of lifetime abstainers and former drinkers, $P_{\text{current(Binge)}}$ is the prevalence of current drinkers who engage in binge drinking, and $P_{\text{current(Non-Binge)}}$ is the prevalence of current drinkers who do not engage in binge drinking, respectively. $\text{RR}_{\text{binge}}(x)$ represents the risk ratio for binge drinkers given a binge amount of alcohol consumed corrected for both time at risk and number of drinking occasions.

To account for the number of drinking occasions and time at risk, the RR(x) for injuries was calculated as follows:

$$\text{RR}(x) = P_{\text{day at risk}} \times P_{\text{days at risk}} \times (\text{RR}_{\text{Crude}}(x) - 1) + 1 \quad (5)$$

where $P_{\text{day at risk}}$ (calculated based on a consumption amount of x) and $P_{\text{days at risk}}$ (for average consumption, this was set at 1) represent the proportion of a given day which

a person drinks and is at risk, and the percentage of days the person undertakes drinking (binge or average), respectively.

Globally, morbidity and mortality can only be grouped into broad categories; the 2005 GBD study uses 161 categories (Institute for Health Metrics and Evaluation 2010). Since these categories are broader than the ICD-10 codes, some specific AAFs for causes of mortality and morbidity were calculated by multiplying the proportion of the number of deaths for ICD-10-specific causes for the GBD categories (as obtained by Statistics Canada for 2004) by the ICD-10-specific AAFs (Statistics Canada 2010).

Construction of confidence intervals

95% confidence intervals (CIs) for AAFs were calculated using methodology by Rehm et al. (2010b) by taking a Monte Carlo approach. For each of the 10,000 simulations, estimates were generated for the prevalence of “former drinkers” and “lifetime abstainers,” from which the prevalence of “current drinkers” was determined. For all disease categories (except injuries), a generated population mean intake in grams per day was generated and then upshifted. The kappa (shape) parameter of the gamma distribution was then computed. The variance of the kappa parameter takes into account the variance in the relationship between the mean and the standard deviation as described by Rehm et al. (2010c). The gamma (scale) parameter of the gamma distribution was then calculated based on the upshifted mean and the kappa parameter. For injuries, the number of drinking occasions per week and the mean grams of alcohol consumed per occasion were generated. For each disease category, the regression coefficients for the RR were computed, and the resulting 10,000 AAF estimates were used to

calculate both the variance of the AAFs and the corresponding 95% CIs for each disease category.

Sensitivity analysis

To analyze the effect of using different corrected estimates of alcohol consumed on the estimated burden of disease and injury attributable to alcohol, the mean alcohol consumption by age and sex was corrected by multiplying each age and sex group mean by 0.4, 0.6, 0.8, 0.9, and 1.0 times the inverse of the estimated undercoverage of the CA-DUMS 2008.

All statistics were performed using R version 2.10.1.

Results

Table 1 gives the prevalence of “current drinkers,” “former drinkers,” and “lifetime abstainers,” and Table 2 provides an overview of the estimated volume of alcohol consumed before and after upshifting the estimates by age and sex. As expected in North America, binge drinking and average alcohol consumption per day were highest in individuals aged 15–29 years, particularly among males in that age group.

Table 3 presents the net number of deaths attributable to alcohol in Canada in 2004 according to GBD code. Overall, 7.1% (95% CI 2.1–12.2%) of all deaths in Canada in 2004 among individuals aged 0–69 years were attributable to alcohol, 6.4% for women and 7.5% for men. This represents 16.3 deaths (11.5 for women and 21.0 for men) per 100,000. The top three causes of death attributable to alcohol were malignant neoplasms (1,452 deaths; 95% CI 1,143–1,761), digestive diseases (1,112 deaths; 95% CI 905–1,318), and

Table 1 Prevalence of alcohol consumption in Canada in 2008 by age and sex

Gender	Age group	Current drinkers		Former drinkers		Lifetime abstainers	
		Percent	95% CI	Percent	95% CI	Percent	95% CI
Women	15–29	75.8	71.0–80.6	8.2	5.2–11.2	16.0	11.8–20.2
	30–44	78.9	75.7–82.2	12.6	10.0–15.1	8.5	6.1–10.9
	45–59	77.7	74.8–80.6	15.0	12.6–17.5	7.3	5.4–9.2
	60–69	69.9	65.6–74.2	17.7	14.2–21.2	12.4	9.3–15.5
	70–79	60.1	54.3–66.0	21.7	16.9–26.6	18.1	13.6–22.7
	80+	51.3	43.2–59.4	17.9	12.1–23.7	30.9	23.4–38.4
Men	15–29	85.0	81.2–88.7	4.3	2.3–6.3	10.7	7.5–14.0
	30–44	85.6	82.0–89.2	9.3	6.2–12.3	5.2	2.9–7.4
	45–59	81.7	78.6–84.9	13.8	11.0–16.7	4.4	2.8–6.1
	60–69	75.3	70.0–80.5	19.7	14.9–24.6	5.0	2.6–7.5
	70–79	69.9	62.1–77.7	20.9	13.9–28.0	9.2	4.4–14.0
	80+	71.3	60.0–82.7	18.9	9.2–28.6	9.8	2.6–17.0

Table 2 Alcohol consumption estimates for Canada in 2008

Gender	Age group	Raw estimates (current drinkers)		Corrected estimates (current drinkers)		Number of drinking occasions per week	
		Mean (g/day)	95% CI	Mean (g/day)	95% CI	Mean number	95% CI
Women	15–29	9.0	3.7–14.4	27.9	23.6–32.2	0.8	0.6–1.0
	30–44	3.6	3.1–4.1	11.2	10.5–11.9	0.8	0.7–0.9
	45–59	4.9	4.2–5.6	15.2	14.1–16.3	1.1	0.1–1.2
	60–69	4.6	3.8–5.5	14.3	13.0–15.6	1.0	0.8–1.2
	70–79	4.4	3.1–5.6	13.5	11.8–15.1	0.9	0.7–1.2
	80+	4.1	2.5–5.6	12.6	10.8–14.3	0.9	0.5–1.2
Men	15–29	12.8	10.1–15.6	39.7	36.0–43.4	1.1	0.9–1.3
	30–44	9.7	8.1–11.4	30.1	27.8–32.5	1.4	1.2–1.5
	45–59	11.7	9.3–14.2	36.3	33.9–38.8	1.7	1.5–1.8
	60–69	11.1	8.2–13.9	34.3	29.9–38.7	1.7	1.4–2.0
	70–79	9.9	7.0–12.7	30.6	25.9–35.2	1.7	1.2–2.1
	80+	5.9	3.2–8.6	18.2	15.0–21.3	1.4	0.8–2.0

Table 3 Mortality attributable to alcohol for Canadians aged 0–69 years in 2004

Category	0–14 years		15–29 years		30–44 years		45–59 years		60–69 years		Total		Total
	F	M	F	M	F	M	F	M	F	M	F	M	
All causes	5	6	102	474	164	489	751	1,209	636	885	1,658	3,063	4,721
Communicable, maternal, perinatal, and nutritional conditions													
Infectious and parasitic diseases	0	0	0	0	0	1	0	2	1	3	2	6	8
Respiratory infections	0	0	1	1	4	5	14	15	14	17	34	38	72
Conditions arising during the perinatal period	5	6	0	0	0	0	0	0	0	0	5	6	11
Noncommunicable diseases													
Malignant neoplasms	0	0	5	3	66	31	385	283	322	356	778	674	1,452
Diabetes mellitus	0	0	4	0	−2	−1	12	17	6	9	21	25	46
Neuropsychiatric conditions	0	0	7	19	18	81	58	179	34	122	117	400	518
Cardiovascular diseases	0	0	1	1	7	−18	97	59	108	−72	213	−30	183
Digestive diseases	0	0	3	4	58	63	168	360	142	313	371	741	1,112
Injuries													
Intentional injuries	0	0	59	308	5	185	8	172	6	93	78	757	835
Unintentional injuries	0	0	22	139	6	142	8	121	2	43	38	446	484

intentional injuries (835 deaths; 95% CI 463–1,343). The leading specific causes of death were liver cirrhosis (1,061 deaths; 95% CI 862–1,261) and motor vehicle accidents (546 deaths; 95% CI 316–845). Among men, ischemic heart disease (264 deaths prevented; 95% CI −1,082 to 1,610) was the leading specific cause of death prevented by alcohol consumption; however, alcohol was attributed with 150 deaths from ischemic heart disease in women (95% CI −41 to 340).

Table 4 presents the number of alcohol-attributable DALYs in Canada for 2004 according to GBD code. Overall, 9.3% (95% CI 6.8–11.9%) of all DALYs were attributable to alcohol in Canada in 2004, 5.7% for women and 12.5% for men. This represents 948 DALYs (562 for

women and 1,328 for men) per 100,000. In total, 274,663 (95% CI 201,397–352,432) DALYs were attributable to alcohol, with 193,932 (95% CI 143,501–249,685) DALYs for men and 80,731 (95% CI 57,895–102,747) DALYs for women. Specifically, neuropsychiatric conditions (180,282 DALYs; 95% CI 179,341–181,223) were responsible for the largest proportion of the DALYs, while unintentional injuries were responsible for the second largest proportion (27,899 DALYs; 95% CI 14,840–44,944). The largest specific cause of DALYs was alcohol use disorders, which contributed 176,350 DALYs (men 130,973, women 45,377).

Table 5 outlines the deaths and DALYs expected in Canada when using survey data that are uncorrected and

Table 4 DALYs attributable to alcohol for Canadians aged 0–69 years in 2004

Category	0–14 years		15–29 years		30–44 years		45–59 years		60–69 years		Total		Total
	F	M	F	M	F	M	F	M	F	M	F	M	
All causes	884	1,438	32,896	94,627	17,233	52,964	21,890	34,474	7,827	10,429	80,731	193,932	274,663
Communicable, maternal, perinatal, and nutritional conditions													
Infectious and parasitic diseases	0	0	15	14	13	32	7	48	13	32	48	125	173
Respiratory infections	0	0	37	53	121	118	255	254	142	154	555	579	1,134
Conditions arising during the perinatal period	269	305	0	0	0	0	0	0	0	0	269	305	574
Noncommunicable diseases													
Malignant neoplasms	0	0	226	122	2,625	792	8,243	4,726	3,853	3,558	14,947	9,198	24,146
Diabetes mellitus	0	0	636	–4	–425	–111	1,538	993	161	148	1,910	1,025	2,935
Neuropsychiatric conditions	615	1,133	27,440	74,808	11,947	40,676	5,933	14,663	778	2,291	46,712	133,570	180,282
Cardiovascular diseases	0	0	32	81	241	–378	2,044	1,851	1,097	–282	3,414	1,272	4,686
Digestive diseases	0	0	216	221	2,330	2,116	3,508	6,417	1,665	3,099	7,719	11,853	19,572
Injuries													
Intentional injuries	0	0	3,359	13,629	208	5,865	222	3,501	96	1,018	3,885	24,014	27,899
Unintentional injuries	0	0	936	5,702	174	3,855	140	2,022	22	411	1,272	11,990	13,262

Table 5 Estimated deaths and DALYs attributable to alcohol by adjusting the alcohol consumption data from the CADUMS with per capita consumption of alcohol data

	Unadjusted (27% PCC)		40% PCC		60% PCC		80% PCC		90% PCC		100% PCC		
	Number	Total %	Number	Total %	Number	Total %	Number	Total %	Number	Total %	Number	Total %	
Men													
Deaths	1,092	2.66	1,492	3.64	2,337	5.70	3,063	7.48	3,357	8.19	4,253	10.38	
DALYs	147,882	9.54	156,954	10.13	176,720	11.40	193,932	12.51	201,303	12.99	223,755	14.43	
Women													
Deaths	1,356	5.23	1,389	5.36	1,499	5.78	1,658	6.39	1,746	6.73	1,857	7.16	
DALYs	69,731	4.95	70,645	5.02	74,606	5.30	80,731	5.74	84,180	5.98	88,844	6.31	
Total													
Deaths	2,448	3.66	2,881	4.31	3,836	5.73	4,721	7.06	5,103	7.63	6,110	9.13	
DALYs	217,613	7.36	227,600	7.70	251,326	8.50	274,663	9.29	285,483	9.65	312,600	10.57	

PCC per capita consumption

corrected adjusting the mean daily alcohol intake by 40, 60, 80, 90, and 100% of recorded and unrecorded per capita consumption. In all cases, alcohol causes a net harm in terms of the deaths and DALYs for both men and women. If using unadjusted consumption data from the CADUMS 2008, we estimated that alcohol would be responsible for 3.66% (2.66% for men; 5.23% for women) of deaths and 7.36% (9.54% for men; 4.95% for women) of DALYs, compared to 7.06% (7.48% for men; 6.39% for women) of deaths and 9.29% (12.51% for men; 5.74% for women) of DALYs when using consumption data adjusted for 80% of per capita consumption. The smaller proportion of deaths attributable to alcohol in women as compared to men in the unadjusted model is due to a greater rate of premature mortality (under 70 years of age) in men as compared to women.

Overall, 31.6% more of total mortality and morbidity (as calculated by the DALYs) is attributable to alcohol than the numbers obtained for mortality alone when comparing the two proportions. This relationship, however, does not hold for both sexes; in females there is a 11.3% decrease when comparing mortality proportions to mortality and morbidity proportions, and in males there is a 67.4% increase in these comparative proportions. This gender difference can be attributed in part to alcohol use disorders and intentional injuries.

Discussion

In accordance with the WHO global strategy to reduce the harmful use of alcohol, we report here the mortality and

burden of disease and injury attributable to alcohol in Canada for 2004. As observed on a global scale, alcohol consumption in Canada also contributes to a higher percentage of the burden of disease (i.e. the combination of years of life lost due to premature mortality and disability) than to mortality alone in Canada; however, this relationship is only seen for men and not women when stratified by gender. The difference in the relationships by gender is primarily caused by the non-fatal consequences of alcohol in specific neuropsychiatric conditions (mental illnesses, especially alcohol dependence) and non-fatal injuries, which heavily contribute to disability being much more prevalent in men.

This analysis does have its limitations, such as the quality of data concerning health outcomes and the assumptions used in calculating DALY weights (see Murray et al. 2002; Rehm and Frick 2010). Additionally, exposure estimates for this paper were based on survey data from 2008 and per capita consumption data from 2004; this should not affect the alcohol-attributable mortality and morbidity estimates to any substantial degree as alcohol consumption in Canada remained relatively stable during the period 2004–2008 (Health Canada 2004, 2009). It should also be noted that patterns of drinking were only taken into consideration for injury and not for ischemic heart disease (on the relationship between patterns of drinking and ischemic heart disease see Murray et al. 2002) which implicitly assumes that the frequency of heavy drinking is the same in Canada as in the cohorts underlying the meta-analysis used to model this relationship (Roerecke and Rehm 2010, 2011). In general, our study also has limitations in terms of the RR functions used in the analysis. One limitation of our approach was the use of adjusted RRs in determining AAFs. The RR formulas we used were developed for risks only adjusted for age (see Flegal et al. 2006; Korn and Graubard 1999; Rockhill and Newman 1998). Two arguments can be made to justify the use of these formulas. First, in risk analyses, such as the Comparative Risk Assessment (CRA) for the GBD Studies (Ezzati et al. 2004), almost all of the underlying studies outlining the various risk factors report only adjusted risks. Relying on unadjusted risks would severely bias the estimated risk functions as only a small proportion of generally older studies could be included. Second, for alcohol in particular, most of the analyses show no marked differences after adjustment for the usual tested risk factors (see Rehm et al. 2010a and the meta-analyses cited therein). The need for adjustment to the RRs may change when other dimensions of alcohol consumption, such as irregular heavy drinking occasions, are considered (see above). As already indicated, for RR functions, such as for cardiovascular diseases, some of the non-linear nature may be caused by other dimensions of alcohol consumption (for example, irregular heavy drinking occasions in the case of

ischemic diseases) (Puddey et al. 1999; Roerecke and Rehm 2010). In the future, it will not be sufficient to conduct additional epidemiological studies into the impact of average volume of alcohol consumption on the incidence of diseases (for an overview see Rehm et al. 2003; 2010a) if accurate estimates of the number of deaths and DALYs attributable to alcohol are desired. Instead, other relevant dimensions of alcohol consumption, which could play a role in confounding the average volume of alcohol consumption, should be included in the design of cohort studies and then should be statistically controlled for by using, for example, meta-regression techniques (Bagnardi et al. 2004). Undercoverage of alcohol is present in both the survey alcohol exposure data and the studies used in meta-analyses to calculate RR functions. The effects of these undercoverages are, however, different. Only undercoverage from measurement error (response bias and construct validity) affects the RR in observational studies. Undercoverage in exposure estimates from survey data comes from the same measurement error, non-response to the alcohol survey, and from populations excluded from the survey (i.e. from additional sources). Since we do not know the extent of undercoverage caused by measurement error in the studies that were used in the meta-analyses to correct the RR functions, it is impossible to know what coverage rate we should standardize survey data to; however, it is very likely that the undercoverage in epidemiological studies is less than in population surveys with rates in the 70–80% range, compared to the undercoverage observed in population surveys with rates between 30 and 70%. Thus, not adjusting alcohol consumption from survey data for undercoverage will lead to biased results. It is suggested by Rehm et al. (2010c) that if the coverage rate of the survey is between 70 and 80% no adjustment is needed; however, if the coverage rate is not within this range, survey data should be standardized to correct for undercoverage (or in some rare cases for overcoverage) of alcohol consumption. Adjustment of survey data for undercoverage (or for overcoverage) also standardizes the results from studies so that they are directly comparable (Rehm et al. 2010c).

In addition to leading to biased results, the heterogeneity in coverage rates which is observed for surveys across countries and time makes comparisons of unadjusted data from different surveys inaccurate. Thus, in order to compare results dependent on alcohol consumption from population surveys across countries and time, it is absolutely necessary to standardize alcohol consumption from surveys with per capita consumption no matter which coverage rate the data are standardized to.

For our analysis, we could only calculate the burden of disease attributable to alcohol where there exists a RR function for the disease or injury. Additionally, our analysis did not include all aspects of harm to others (such as

motor vehicle accidents and workplace injuries) which recently has been shown to constitute a large proportion of the burden of injury attributable to alcohol (Laslett et al. 2010).

This method for characterizing an upshifted gamma distribution, although correcting partially for undercoverage, also is limited as it may not fully correct for bias stemming from (1) non-response to the alcohol survey, (2) the construct validity of alcohol consumption measures, (3) response bias, and (4) populations excluded from the survey (Shield and Rehm 2009). Although Rehm and colleagues' method of upshifting alcohol consumption using per capita data and modeling alcohol consumption through a gamma distribution may not correct for all the biases that lead to undercoverage, this method does allow for the characterization of alcohol consumption in a population if it was measured by a survey with a coverage rate higher than 80%. This method is vastly superior to the previous method of adjusting for undercoverage which multiplied categorical estimates of consumption by the inverse of undercoverage without any knowledge of the effect that undercoverage had on these categorical estimates.

Despite these limitations, the estimates of burden of disease and mortality presented here are the best possible and most up-to-date for Canada. Overall, alcohol is an important contributor to both morbidity and mortality in Canada in individuals between the ages of 0 and 69 years. This burden of alcohol-attributable harm is unnecessary and preventable. Mortality and morbidity estimates presented in this study are based on multiple factors, such as drinking characteristics described by age and sex, RR functions which differ by sex and the mortality profiles of these groups. Although women drank less than men, they had a greater number of deaths from cardiovascular diseases (CVD) attributable to alcohol than did men, due to the steeper risk curve for women than for men (i.e. women experienced more harm than did men for the same average daily consumption) and a higher prevalence of former drinkers among women. Additionally, average consumption was higher for both men and women aged 45–59 years compared to men and women aged 30–44 and 60–69 years, leading to greater AAFs for people aged 45–59 years compared to people who were 30–44 and 60–69 years of age.

The estimates provided here are higher than those previously reported for Canada for the respective age groups (Patra et al. 2007); however, these differences are due to updated RR functions, the inclusion of new causes of mortality and morbidity (such as colon cancer, rectal can-

cer, and tuberculosis), and the new methodologies of modeling alcohol consumption and of calculating the AAFs (see Rehm et al. 2010c) for differences in the methodology). However, from Patra et al.'s (2007) paper, we see an upward trend in the number of deaths in Canada attributable to alcohol consumption. Adjusting our results for 80% of adult per capita consumption for the Canadian population and using methodology outlined by Rehm et al. (2010c), we found that the number of deaths in Canada attributable to alcohol in 2004 (excluding the number of deaths prevented) would be 9,270 representing 4.1% of all deaths, compared to 8,103 representing 3.6% of all deaths in Canada in 2002 which was observed using methodology outlined by Rehm and colleagues (Patra et al. 2007; Rehm et al. 2006). Globally, in 2004, alcohol was responsible for 1.6% of all deaths in high-income countries, while in Canada in 2004 alcohol was responsible for 2.7% of all deaths, in both cases including the number of deaths prevented (World Health Organization 2009). Thus, Canada's burden of mortality attributable to alcohol may be higher than most other high-income countries. However, the differences in these mortality estimates may be due to the above-noted differences in methods used to calculate the estimates (see Rehm et al. 2010c) for an in-depth discussion of the differences between methods). Our study will be directly comparable to the 2005 CRA study and to future research outlining the burden of disease for global regions and individual countries.

When implementing effective public health strategies, both mortality and burden of disease estimates should be taken into consideration. Given Canada's epidemiological profile, policies should be implemented to reduce both Canadians' overall alcohol consumption and their average alcohol consumption per occasion (Chisholm et al. 2004). Effective alcohol policies, such as increases in taxation, decreases in the legal blood alcohol content while driving, and other interventions aimed at reducing harmful alcohol consumption, would reduce not only alcohol-related mortality and morbidity but also other alcohol-related social harms (Schmidt et al. 2010; Single et al. 1999; World Health Organization 2002).

Conflict of interest The authors declare they have no competing interests.

Appendix

See Table 6.

Table 6 Categories of alcohol-related disease and sources used for determining alcohol-attributable fractions

Condition	GBD code	Source for RRs
Communicable, maternal, perinatal, and nutritional conditions	I	
Infectious and parasitic diseases	I A	
Tuberculosis	I A 1	Lönnroth et al. (2008) (causal relationship; see Rehm et al. 2009a, b)
Respiratory infections	I B	
Lower respiratory infections	I B 1	Pneumonia RR obtained from Samokhvalov et al. (2010a, b)
Conditions arising during the perinatal period	I D	
Low birthweight	I D 1	Patra et al. (2011)
Noncommunicable diseases	II	
Malignant neoplasms	II A	
Mouth and oropharynx cancers	II A 1	Baan et al. (2007) (based on relative risks from Corrao et al. 2004)
Esophageal cancer	II A 2	Baan et al. (2007) (based on relative risks from Corrao et al. 2004)
Colon and rectal cancers	II A 4	Baan et al. (2007) (based on relative risks from Corrao et al. 2004)
Liver cancer	II A 5	Baan et al. (2007) (based on relative risks from Corrao et al. 2004)
Breast cancer	II A 9	Baan et al. (2007) (based on relative risks from Corrao et al. 2004)
Other malignant neoplasm	II A 17	Colon and rectal cancer RRs obtained from Baan et al. (2007) (based on relative risks from Corrao et al. 2004)
Diabetes mellitus	II C	Baliunas et al. (2009)
Neuropsychiatric conditions	II E	
Epilepsy	II E 4	Samokhvalov et al. (2010b)
Alcohol use disorders	II E 5	100% AAF per definition
Alzheimer's disease and other dementias	II E 6	Degeneration of the nervous system due to alcohol, 100% AAF per definition
Other neuropsychiatric conditions	II E 16	Alcohol polyneuropathy, 100% AAF per definition
Cardiovascular diseases	II G	
Hypertensive heart disease	II G 2	Taylor et al. (2010)
Ischemic heart disease	II G 3	Baan et al. (2007, 2011)
Cerebrovascular disease	II G 4	Ischemic and hemorrhagic stroke RRs obtained from Baan et al. (2007) and Patra et al. (2010)
Inflammatory heart disease	II G 5	Alcohol cardiomyopathy, 100% AAF per definition
Other cardiovascular diseases	II G 6	Cardiac arrhythmias RR obtained from Samokhvalov et al. (2010b)
Digestive diseases	II I	
Cirrhosis of the liver	II I 2	Rehm et al. (2010a, b, c, d)
Other digestive diseases	II I 4	Acute and chronic pancreatitis RR obtained from Irving et al. (2009); Chronic pancreatitis (alcohol-induced) and alcoholic gastritis, 100% AAF per definition
Injuries	III	
Intentional injuries	III A	
Road traffic accidents	III A 1	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Poisonings	III A 2	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Falls	III A 3	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Fires	III A 4	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Drownings	III A 5	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Other unintentional injuries	III A 6	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Intentional injuries	III B	

Table 6 continued

Condition	GBD code	Source for RRs
Self-inflicted injuries	III B 1	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Violence	III B 2	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods
Other intentional injuries	III B 4	Taylor et al. (2010) for relative risk, Rehm et al. (2008) and Taylor et al. (2008a, b) for AAF calculation methods

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