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# Distributions of alcohol use and alcohol-caused death and disability in Canada: Defining alcohol harm density functions and new perspectives on the prevention paradox

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## Abstract

**Aims:** The aims of this study were to examine the distribution of alcohol use and to define ‘harm density functions’ representing distributions of alcohol-caused health harm in Canada, by sex, towards better understanding which groups of drinkers experience the highest aggregate harms.

**Design:** This was an epidemiological modeling study using survey and administrative data on alcohol exposure, death and disability and risk relationships from epidemiological meta-analyses.

**Setting:** This work took place in Canada, 2019.

**Participants:** Canadians aged 15 years or older participated.

**Measurements:** Measures included modeled life-time mean daily alcohol use in grams of pure alcohol (ethanol) per day, alcohol-caused deaths and alcohol-caused disability-adjusted life-years.

**Findings:** As a life-time average, more than half of Canadians aged 15+ (62.8% females, 46.9% males) use fewer than 10 g of pure alcohol per day (g/day). By volume, the top 10% of the population consume 45.9% of the total ethanol among males and 47.1% of the total ethanol among females. The remaining 90% of the population experience a slim majority of alcohol-caused deaths (males 55.3%, females 46.9%). Alcohol harm density functions compose the size of the using population and the risk experienced at each volume level to show that the population-level harm experienced is highest for males at 25 g/day and females at 13 g/day.

**Conclusions:** Almost 50% of alcohol use in Canada is concentrated among the highest 10% of drinkers, but more than half of the alcohol-caused deaths in Canada in 2019 were experienced by the bottom 90% of the population by average volume, providing evidence for the prevention paradox. New alcohol harm density functions provide insight into the aggregate health harm experienced across the mean alcohol use spectrum and may therefore be used to help determine where alcohol policies should be targeted for highest efficacy.

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## KEYWORDS

Alcohol, alcohol policy, alcohol prevention paradox, alcohol use, alcohol-attributable mortality, Canada

## INTRODUCTION

Alcohol use is responsible for a substantial global burden of death and disability, causing nearly 3.0 million deaths and 132.6 million disability-adjusted life-years in 2016, each exceeding 5.0% of the world-wide total [1]. Alcohol ranked seventh among leading risk factors for premature death and disability across all ages [1]. In Canada, recorded alcohol use per capita (APC) among those 15 or older was 8.9 l ethanol per year (l/year) in 2016 [2]; this is 40% higher than the global average of 6.4 l/year [2]. Canadian APC is projected to rise further to 10.9 l/year by 2030, and prevalence of use is also projected to increase [3]. Such increases would place upward pressure on already substantial alcohol-caused (AC) harms and costs in Canada. Alcohol was causally responsible for more than 18 000 deaths and CAD\$16.6 billion in social costs in 2017 [4].

Understanding which drinking populations experience the balance of this harm is key, as two broad policy avenues exist towards mitigating AC harms. Population-level approaches target alcohol use and harm in the population as a whole, while individual-level approaches target those highest-volume alcohol consumers at high risk of AC harm at a certain point in time [5]. This tension between population and individual approaches is heightened as alcohol industry actors frame the large burden of AC harms 'in terms of a small minority of problem drinkers versus the moderate majority... (p. 1579)' [6]. Against this are study findings describing an alcohol prevention paradox, wherein those not in this 'small minority' altogether experience the majority of alcohol-caused harms, due to the larger population size [7–11].

Central to the debate is defining this 'minority' of heavy drinkers and the measurement of harm caused by alcohol: studies differ in these decisions. For an overview of studies, see Rossow & Romelso [7, p. 85], but two definitions present themselves in the alcohol prevention paradox literature. The majority tradition in alcohol prevention paradox studies has been to define the 'high-use' group as the highest-using 10% of the population, while the other 90% of the population constituted 'the rest'. For example, a study using linked Finnish survey data and registry data on hospital admissions and deaths found that the lowest-using 90% of men/women experienced approximately 70%/64% of AC hospital stays and 64%/60% of AC deaths [11]. In Brazil, Sweden and Norway, the majority of AC social and dependence problems were experienced by non-high-risk consumers, when the high-risk group was defined as those in the top 10%, by sex, of the chronic alcohol use distribution [7, 8]. Another potential definition is to study whether people using alcohol are above or within chronic alcohol use guidelines. In Canada, a study found that the 12% of the population using above Canadian chronic guidelines experienced 62% of AC deaths [12]. In Switzerland, those using below chronic alcohol use guidelines experienced far more AC problems collectively (87%) than those guidelines above (13%) [13].

Previous prevention paradox studies [7–10, 14] have largely leaned upon self-reported social, financial and intoxication-related measures of AC harm and self-reported alcohol use; although useful, this method of measurement is susceptible to bias and may result in under-reporting [15, 16]. Lastly, previous studies built categorical variables representing measures of high-risk use; however, the risk of alcohol health harms exist along a continuum [1].

Accordingly, the current study aims to address the shortcomings discussed above through characterizing, by sex, the distribution of alcohol use and the distribution of alcohol-caused death and disability in Canada throughout the life-course. The highest-consuming 10% definition will be used to study the prevention paradox; further, this study defines a novel concept, alcohol harm density functions, which depict how AC harm is distributed across the drinking population.

## METHODS

### Study design and methodological overview

This epidemiological modeling study used a life-time risk methodology [17] to estimate the risk of death and disability attributable to alcohol in the Canadian population, by sex and by chronic mean alcohol use per day. This method estimates life-time risks by integrating three main data sources: (i) exposure to alcohol use, (ii) disease- and injury-specific dose-response relative risk functions and (iii) population, mortality and disability data, as categorized by disease/injury, age group and sex.

These foundational analyses closely followed the life-course analyses completed for Canada's Guidance on Alcohol and Health (CGAH) [18]. Methods for the life-time risk analyses were comprehensively detailed for the CGAH study [19] and are summarized below. As CGAH required sex-specific analyses and alcohol use and related relative risks are known to be differential by sex, all modeling was stratified by sex. The current study is not a component of the CGAH study, and all results presented herein are novel. For this current study, the research questions and analytical plan were not pre-registered on a publicly available platform; the findings may therefore be considered exploratory.

### Data sources

Alcohol exposure information was from three sources. The relative mean daily alcohol use in each sex-age group and the prevalence of current drinkers, former drinkers, life-time non-drinkers and past-month binge drinking was obtained from the 2016 Canadian Tobacco, Alcohol and Drugs Survey [20]. Recorded alcohol use per capita information was taken from official Statistics Canada sales information for

2019 [21]. An estimate of unrecorded use in Canada was from the World Health Organization's 2018 Global Status Report on Alcohol and Health [2].

Relative risk (RR) function sources have been published previously [19, 22]. For conditions wholly attributable to alcohol, absolute risk functions were calculated as detailed elsewhere [23].

Population estimates by age and sex, population metrics of age at time of death by sex and number of deaths by disease/injury cause were obtained through Statistics Canada's public databases [24–26]. Years of life lost due to disability were obtained from the Institute for Health Metrics and Evaluation's 2018 Global Burden of Disease study [27]. Population, death and disability data were obtained for 2017, 2018 and 2019 and 3-year averages were computed.

All data were available stratified by sex. The four pieces of prevalence information stated in the Data sources section (above) were aggregated into the following age groups: 15–24, 25–34, 35–44, 45–54, 55–64, 65–74 and  $\geq 75$  years of age. Mortality and disability data were available in 5-year age groups, but were aggregated to match the above. Relative risk estimates were available by sex only.

All data used for this study were secondary data, acquired from publicly available data systems. Accordingly, an ethics application was not required for this study.

### Summary of life-time alcohol use methodology

A gamma distribution methodology was applied to the alcohol exposure data described above; this resulted in continuous prevalence distributions of mean daily alcohol use for each sex and age group [28]. The continuous prevalence distributions were assumed to hold throughout the life-course; that is, that they represent the life-time average consumption (in g/day) for each member of the population.

### Summary of life-time risk methodology

The life-time methodologies used for this study have been published previously [17, 19], and were operationalized using the above data sources. Distribution of alcohol use for each sex–age group were modeled using a gamma distribution methodology, which integrates the data collected on alcohol exposure. First, total APC was found using recorded alcohol sales and adding an estimate of unrecorded alcohol. Total APC was then matched to the under-reporting present in epidemiological alcohol studies from which relative risk functions were estimated [29], with the result then apportioned to each sex–age group based upon the relative mean use in each of these groups from the CTADS survey. Next, this APC in each sex–age group were fitted to the gamma distribution using the prevalence of current drinkers, former drinkers and past-

month binge drinking [28]. These distributions were integrated against the identified condition-specific dose–response RR functions to compute alcohol-attributable fractions (AAFs) [28]. For cancers, a lag time of 10 years was used based on approximate latency periods

for breast, colorectal, oral cavity, esophageal, pharyngeal, laryngeal and liver cancers [30]. In this summary mortality methods are described, but years lost due to disability were calculated analogously. The number of AA deaths were computed by multiplying AAFs against the counts of deaths for each age group, sex and disease/injury. The risk of death for life-time abstainers from alcohol from a given disease/injury was computed as the death rate multiplied by the fraction of non-attributable deaths (i.e.  $1 - \text{AAF}$ ); see also formulae 7 and 8 in Shield *et al.* [19, p. 15].

The excess risk of death for people who consume alcohol from a given disease/injury was computed as the death risk for life-time abstainers multiplied by the excess risk from alcohol use (i.e.  $RR - 1$ ) for each level of mean daily alcohol use. The modeling assumed that there was no risk of alcohol-caused death for a person aged 0–14 years, that each person began using alcohol at age 15 years and continued using alcohol until their death. The total risk of death due to an AA condition, at a given level of mean daily alcohol use, was computed as the sum of cause-specific risks over all causes.

Life-time risk of AA death/disability for a given level of mean daily alcohol use was then computed in 1 g/day increments by iteration over each age from 15 to 94; see formulae 13 to 17 [19]. The proportion of the population surviving at the end of each 1-year age grouping was estimated based on sex and mean daily alcohol use. The total life-time risk per 1000 people of an alcohol-caused death was then found by multiplying the proportion of the alive population by the AA mortality risk in each 1-year age grouping and then summing this product for each sex and level of mean daily alcohol use.

This resulted in the life-time risk of death and life-time risk of years lost to disability by sex for each 1 g/day increment of mean daily alcohol use. The life-time risk of years of life lost due to alcohol per 1000 people, by sex and mean daily use, was estimated by multiplying the proportion of the alive population at the end each 1-year age by the age-specific AA death risk and summing this product. For each level of mean daily use and sex, the life-time risk of lost disability-adjusted life-years (DALYs) was then calculated by adding the life-time risk of years of life lost and the life-time risk of years lost to disability.

## Measurements

### Distribution of life-time mean daily alcohol use

The gamma distribution prevalence was aggregated into mean use categories for the presentation of results. Some categories were of fixed consumption width (e.g. 1 g/day, 10 g/day) and some categories were aggregated into population deciles.

### Distributions of alcohol-caused death and disability

The rates of alcohol-attributable death and DALYs were calculated for each 1-g increment of average daily use up to 150 g. Again in 1-g

increments, the distribution of AA death and DALYs was found by multiplying these rates against the proportions of the population at each use level. The result was normalized to produce a discrete probability density distribution and this was summed to assign proportions of AA death and DALYs to larger daily use categories [e.g. in 10-g/day or standard drinks (SD)/day intervals]. The values attributed to each category are thus the proportion of the total AA death and DALYs to have been incurred by members of each use category.

### Estimation of uncertainty

The model was simulated 1000 times in these steps. First, independent draws were sampled from the respective error distributions of the following lowest level model parameters: prevalence of current use, former use, past-month drinking, relative mean alcohol use in each sex-age group and each of the relative risk functions. The model simulation was then followed through, which was a deterministic process, and repeated over a further 999 times. For each necessary estimate, the 50th percentile was used to report the median estimate and the 2.5th and 97.5th percentiles were used to produce the uncertainty intervals. All analyses were performed in R version 3.6.1 [31].

## RESULTS

### The distribution of life-time alcohol use

When the population is divided using 10 g/day increments of life-time use, use distributions are differential by sex. As a daily average throughout their drinking life-time (15+), most Canadians use fewer than 10 g/day of pure alcohol (ethanol) throughout their life-time. From Table 1, 46.9% of males and 62.8% of females meet this criterion. More than half of females (51.5%) drink between 0.1 and 10.0 g/day. A small proportion of the population maintain a high mean intake level when considering their entire life-course: for males, 9.9% use more than 60 g/day throughout their life-time, while for females this percentage is 1.6%. Among the 10 g/day groups, the highest percentage of pure alcohol is used by males drinking between 20.1 and 30.0 g/day (11.9% of alcohol used) and by females drinking 10.1–20.0 g/day (23.4% of alcohol used). The 2.6% of males in the highest-using group in Table 1, which is a 50 g/day increment between 100.1 and 150.0 g/day, consume 14.0% of the total ethanol.

Males used approximately two-thirds (65.6%) and females one-third (34.4%) of the total ethanol consumed in Canada in 2019 (result not shown). Table 2 shows the concentration of alcohol use when dividing the drinking population into alcohol use groups defined by volume deciles. Alcohol use is highly concentrated among the highest-using populations throughout their life-times. Among males, 19.1% of alcohol is used by the highest 2.5% of consumers by volume, 30.4% by the highest 5% and 45.9% by the highest 10%. Use is slightly more

concentrated among females, as 21.8% is used by the highest 2.5%, 32.5% by the highest 5% and 47.1% by the highest 10%. Those drinking more than 52.8 g/day (four or fewer Canadian SD/day) define the highest-using 10% of males and those drinking more than 26.0 g/day (two or fewer SD/day) define the highest-using 10% of females. The bottom 50% of people using alcohol by volume account for only 8.9% of total alcohol use among males and 9.5% among females. Supporting information, Tables S1 and S2, show information similar to Tables 1 and 2, but use Canadian SD to define alcohol use groups and decile cut-points.

### The distribution of life-time alcohol-caused harm

Looking again at Table 1, when the population is divided into alcohol use groups defined by 10 g/day increments, males using more than 100 g/day experience the greatest aggregate AC harm (13.4% of life-time AC deaths; 17.6% of life-time AC DALYs). After this highest-using group, which is a catch-all of the five highest 10 g/day groupings, the most AC harm is experienced by males using 20.1–30.0 g/day (12.5% of life-time AC deaths; 11.0% of life-time AC DALYs). Among females, those using 10.1–20.0 g/day experience the most AC harm (26.2% of life-time AC deaths and 25.1% of life-time AC DALYs).

Table 2 shows that AC health harms are concentrated among those using the most alcohol. The highest-drinking 2.5% of drinkers together experience 18.3% of life-time AC deaths and 23.2% of life-time AC DALYs among males, and 18.8% of life-time AC deaths and 19.5% of life-time AC DALYs among females. For both sexes, more than half of all AC deaths (55.3% for males; 53.1% for females) and approximately half of all AC DALYs (for males: 49.8%; for females: 53.7%) are experienced by the lowest drinking 90% of the drinking population.

### The prevention paradox and alcohol harm density functions

The prevention paradox will first be studied by using the definition of the highest-using 10% of the population versus the other 90%. The prevention paradox is substantiated, as the highest-using 10% of males experience fewer than half (44.7%) of AC deaths and half (50.2%) of AC DALYs. Among females, the highest-using 10% experience 46.9% of AC deaths and 46.3% of AC DALYs.

The core of the prevention paradox is illustrated in Figure 1. Panels (a) and (b) show the percentage of the population and the risk experienced (the life-time probability of experiencing an AC death) at each life-time alcohol use level (in 1.0 g/day increments). At low levels of chronic alcohol use the risk is low, but there are many people consuming at this level. As chronic use increases, risk increases but the number of consumers decreases. The aggregate harm experienced by the population integrates these two concepts. In Figure 1, panels (c) and (d) define the probability density functions of AC life-time

**TABLE 1** Percentages of the population aged 15+, alcohol use and alcohol-caused deaths and DALYs experienced, non-cumulative and cumulative, by alcohol use groups defined in 10 g/day increments, Canada 2019.

Males Life-time alcohol use	Group Range of life- time alcohol use (g/day)	Not cumulative				Cumulative			
		% Population	% Life-time alcohol used	% Life-time AC deaths experienced	% Life-time AC DALYs experienced	% Population	% Life-time alcohol used	% Life-time AC deaths experienced	% Life-time AC DALYs experienced
None	None	8.5	0.0	0.0	0.0	8.5	0.0	0.0	0.0
↓	Low	38.4	6.6	6.4	5.7	46.9	6.6	6.4	5.7
	10.1–20.0	16.6	11.0	11.3	9.8	63.5	17.6	17.6	15.4
	20.1–30.0	10.6	11.9	12.5	11.0	74.1	29.5	30.1	26.5
	30.1–40.0	7.2	11.5	11.9	10.8	81.3	41.0	42.0	37.3
	40.1–50.0	5.1	10.5	10.6	9.9	86.4	51.4	52.6	47.2
	50.1–60.0	3.7	9.2	9.3	9.0	90.1	60.6	61.9	56.2
	60.1–70.0	2.7	8.0	7.9	7.9	92.8	68.6	69.8	64.1
	70.1–80.0	2.0	6.8	6.6	6.9	94.8	75.4	76.5	71.1
	80.1–90.0	1.5	5.7	5.5	6.1	96.3	81.2	82.0	77.1
	90.1–100.0	1.1	4.8	4.6	5.3	97.4	86.0	86.6	82.4
High	100.1–150.0	2.6	14.0	13.4	17.6	100.0	100.0	100.0	100.0
Females Life-time alcohol use	Group Range of life-time alcohol use (g/day)	Not cumulative				Cumulative			
		% Population	% Life-time alcohol used	% Life-time AC deaths experienced	% Life-time AC DALYs experienced	% Population	% Life-time alcohol used	% Life-time AC deaths experienced	% Life-time AC DALYs experienced
None	None	11.3	0.0	0.0	0.0	11.3	0.0	0.0	0.0
↓	Low	51.5	17.2	12.9	15.3	62.8	17.2	12.9	15.3
	10.1–20.0	18.5	23.4	26.2	25.1	81.3	40.6	39.1	40.4
	20.1–30.0	8.9	19.2	22.1	20.9	90.2	59.8	61.3	61.4
	30.1–40.0	4.6	13.8	15.2	14.6	94.7	73.7	76.5	75.9
	40.1–50.0	2.4	9.4	9.5	9.3	97.1	83.1	86.0	85.2
	50.1–60.0	1.3	6.2	5.9	5.9	98.4	89.3	91.9	91.2
	60.1–70.0	0.7	4.0	3.5	3.6	99.1	93.3	95.3	94.7
	70.1–80.0	0.4	2.5	2.0	2.1	99.5	95.8	97.3	96.9
	80.1–90.0	0.2	1.6	1.2	1.3	99.7	97.5	98.5	98.2
	90.1–100.0	0.1	1.0	0.7	0.8	99.8	98.5	99.2	98.9
High	100.1–150.0	0.2	1.5	0.8	1.1	100.0	100.0	100.0	100.0

Abbreviations: AC = alcohol-caused; DALYs = disability-adjusted life-years; g/day = grams ethanol per day.

death—from now termed ‘alcohol harm density functions’—for males and females, respectively. These represent the density of the aggregate harm experienced at each 1 g/day increment; that is, the total area under each function is 100%, integrating both the size of the population and the risk experienced by consumers at each volume level.



The harm density functions show that the aggregate harm experienced is highest for males at 25 g/day (fewer than two SD/day) and females at 13 g/day (less than one SD/day). Harm density is highly concentrated among females using between 5 and 30 g/day, as represented by the high, steep peak. Harm density among males is distributed more evenly across the full range of use, with the highest harm density occurring among males between 10 and 50 g/day.

## DISCUSSION

### The distribution of alcohol use

Almost 50% of alcohol use is concentrated in Canada among the highest 10% of drinkers. This is similar to Brazil [8] but less concentrated than in the United States, where this percentage is 55–61% [32]. Measured by sex, males use nearly-two thirds of all alcohol in Canada. Although striking, this is less than in the United States and Brazil, where males use more than three-quarters of all alcohol [8, 33]. Females may continue to close this gap in consumption, as the long-term trend shows increasing frequency

**TABLE 2** Alcohol use ranges, mean alcohol use and percentages of life-time alcohol use, life-time AC deaths experienced and life-time AC DALYs (non-cumulative and cumulative), by alcohol use groups as defined by deciles of the drinking population 15+, Canada 2019.

Males	Use Group Percentile of the drinking population	Alcohol use range in g/day	Mean alcohol use in g/day	Not cumulative			Cumulative		
				Life-time alcohol used	Life-time AC deaths experienced	Life-time AC DALYs experienced	Life-time alcohol used	Life-time AC deaths experienced	Life-time AC DALYs experienced
Low 	0–10th	> 0.0–1.3	0.5	0.3	0.1	0.2	0.3	0.1	0.2
	10th–20th	> 1.3–3.0	2.1	0.8	0.7	0.6	1.0	0.8	0.8
	20th–30th	> 3.0–5.4	4.1	1.5	1.5	1.3	2.6	2.3	2.2
	30th–40th	> 5.4–8.4	6.8	2.5	2.5	2.2	5.1	4.8	4.3
	40th–50th	> 8.4–12.3	10.2	3.8	3.9	3.4	8.9	8.7	7.7
	50th–60th	> 12.3–17.4	14.7	5.6	5.8	5.0	14.5	14.5	12.7
	60th–70th	> 17.4–24.3	20.6	8.2	8.3	7.3	22.7	22.7	20.0
	70th–80th	> 24.3–34.5	29.1	12.1	12.9	11.5	34.8	35.7	31.4
	80th–90th	> 34.5–52.8	42.7	19.3	19.7	18.3	54.1	55.3	49.8
	90th–95th	> 52.8–71.3	61.1	15.5	15.4	15.3	69.6	70.8	65.1
High	97.5th–100th	89.4+	111.4	19.1	18.3	23.2	100.0	100.0	100.0
Females	Use Group Percentile of the drinking population	Alcohol use range in g/day	Mean alcohol use in g/day	Not cumulative			Cumulative		
Alcohol use				Life-time alcohol used	Life-time AC deaths experienced	Life-time AC DALYs experienced	Life-time alcohol used	Life-time AC deaths experienced	Life-time AC DALYs experienced
Low 	0–10th	> 0.0–0.8	0.4	0.3	–0.7	–0.1	0.3	–0.7	–0.1
	10th–20th	> 0.8–1.8	1.3	0.9	–0.2	0.4	1.2	–0.9	0.3
	20th–30th	> 1.8–3.1	2.4	1.7	0.7	1.2	2.9	–0.2	1.5
	30th–40th	> 3.1–4.7	3.9	2.7	1.9	2.3	5.6	1.7	3.8
	40th–50th	> 4.7–6.6	5.6	3.9	3.5	3.7	9.5	5.2	7.5
	50th–60th	> 6.6–9.1	7.8	5.6	5.5	5.6	15.1	10.7	13.1
	60th–70th	> 9.1–12.5	10.7	8.0	8.6	8.4	23.1	19.4	21.6
	70th–80th	> 12.5–17.3	14.7	11.6	13.1	12.5	34.7	32.4	34.1
	80th–90th	> 17.3–26.0	21.2	18.2	20.7	19.6	52.9	53.1	53.7
	90th–95th	> 26.0–35.1	30.1	14.6	16.8	15.9	67.5	69.9	69.5
High	97.5th–100th	44.3+	60.4	21.8	18.8	19.5	100.0	100.0	100.0

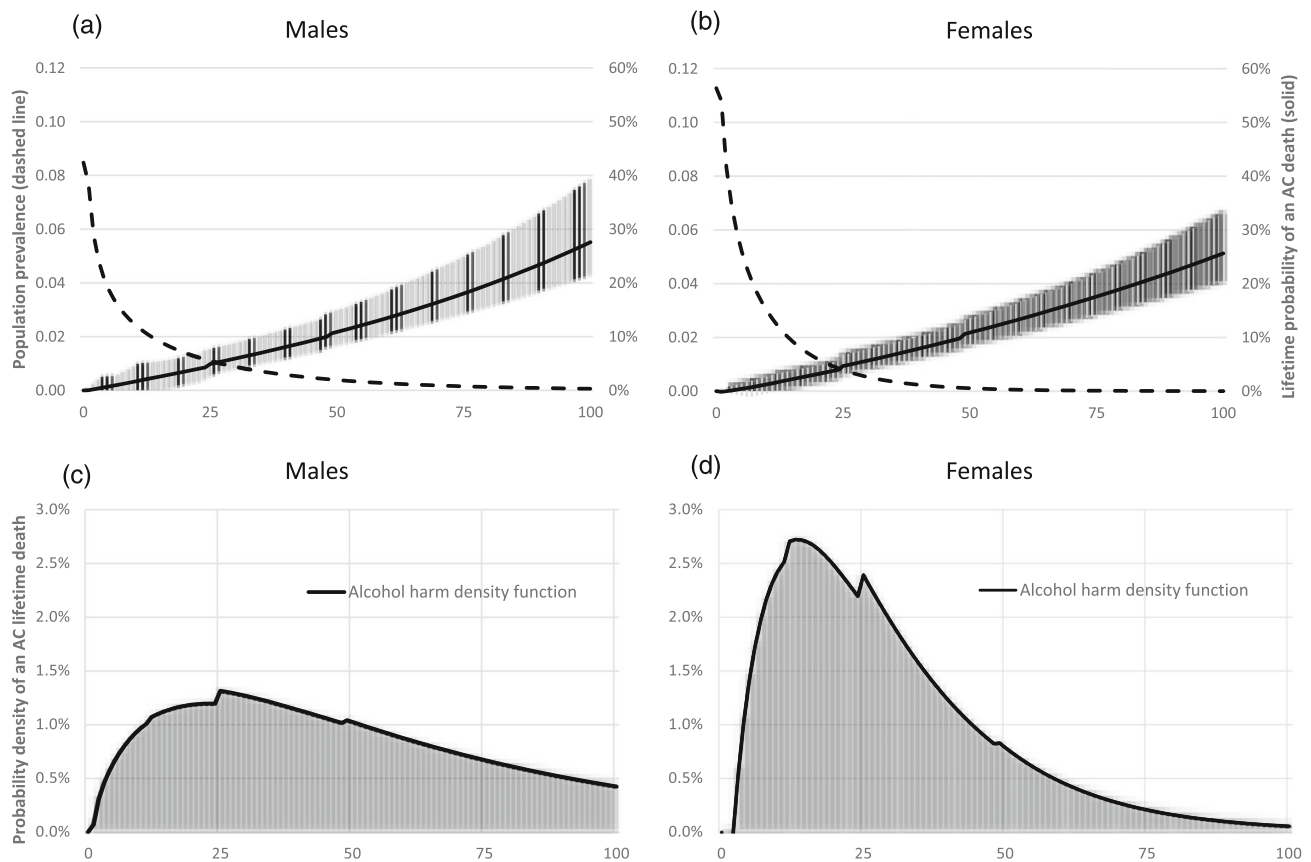
AC = alcohol-caused; DALYs = disability-adjusted life-years; g/day = grams ethanol per day.

and quantity of drinking among women—more so than men [34]. This will have critical policy implications, as the distribution of alcohol use is different among females than males, with the balance of alcohol use occurring at lower levels of mean life-time use.

We note that the majority of the Canadian population—more than 60% of females and more than 45% of males—use fewer than 10 g/day of alcohol, on average, throughout their life-time. This is key towards normalizing low levels of use; further, this is important, as alcohol consumers have a tendency to overestimate the use of others and to be influenced by this perceived higher consumption [35].

## The distribution of alcohol-caused health harm

Alcohol harm density functions provide a critical new lens through which to view the distribution of alcohol-caused harm (we suggest reviewing Figure 1). At a glance, it can be seen which levels of use contribute the greatest aggregate magnitude of harm by sex, and therefore where policies may be targeted to best reduce the overall alcohol-caused burden of disease. The female population experiences the greatest aggregate harm from drinking between approximately 10 and 30 g/day, which then tails off quickly. The male population experiences the greatest level of aggregate harm between 10 and 50 g/day. This then decreases slowly and steadily, leading males as a



**FIGURE 1** Population prevalence of alcohol use, lifetime probability of alcohol-caused death and alcohol harms density functions, by sex, Canada, 2019. All panels: The x-axis is the lifetime average grams ethanol per day; AC=alcohol-caused. (a) Among males, the population prevalence of alcohol use at each consumption level (left axis) and the risk function with 95% confidence intervals. The risk function is the lifetime probability of experiencing an alcohol-caused (AC) death (right axis). (b) Among females, the population prevalence of alcohol use at each consumption level (left axis) and the risk function with 95% confidence intervals. The risk function is the lifetime probability of experiencing an alcohol-caused (AC) death (right axis). (c) Among males, the probability density of alcohol-caused lifetime death at each consumption level, that is, the alcohol harm density function. As this is a density function, adding the probability at each 1 g interval will result in 100% of all deaths. (d) Among females, the probability density of alcohol-caused lifetime death at each consumption level, that is, the alcohol harm density function. As this is a density function, adding the probability at each 1 g interval will result in 100% of all deaths.

group to continue to collectively experience substantial harms well into heavy use levels of 100+ g/day.

For the same level of average chronic use, females experience a higher increased relative risk than males. However, males are more likely to experience death and disability from alcohol-related causes than females, especially in youth and middle age. Composed together into a measure of absolute risk—represented in Figure 1 by the probability of an AC death—this means that females and males at the same level of use have broadly similar chances of alcohol causing their death (Figure 1; risk functions), except for very high levels of use where males have a higher probability of death. Importantly, females who drink 10–20 g/day (< 0.7–1.5 SDs), representing 18.5% of females, experienced more than one-quarter of all female AC deaths and disability; this is the highest proportion of harm by 10 g/day grouping. This is cause for concern, as females in Canada have

increasing rates and changing patterns of drinking [34]. The recent trend of ‘mummy drinking sites’ and changing culture of alcohol use among women, and mothers especially, encouraging alcohol use as a panacea for the stresses of life, may in part be fueling this shift [36]. AC death and disability is concentrated in the larger population of females consuming less than 30 g/day (< 2.2 SD/day)—the 59.8% of females in this group collectively experience 61.3% of AC deaths (61.3%) and disability (61.4%).

Using 10 g/day grouping among males, AC health harms are distributed more evenly across the alcohol use spectrum than among females (as in Table 1). There is a wide peak from 10 to 50 g/day (0.7–3.7 SD/day) that includes 77.9% of the population experiencing 46.2% of AC deaths. Also, the 2.6% of highest-consuming males, who use between 100.1 and 150.0 g/day, together experience 13.4% of AC deaths and 17.6% of AC DALYs.

## Implications for the prevention paradox

More than half of the alcohol-caused deaths in Canada were experienced by the bottom 90% of the population by average volume, providing evidence for the prevention paradox.

For both males and females the absolute risk functions in Figure 1 were non-linear, although not steeply so. This aligns with theoretical considerations of the prevention paradox as laid out by Skog [37]; namely, that risk curves must be steeply non-linear (i.e. strongly convex) for the prevention paradox not to occur. The discontinuities in Figure 1, which occur at 25 and 49 g/day, occur as the risk function for ischemic stroke experiences a jump at these levels corresponding to 'more than two' and 'more than four' 12 g drinks per day; these are artifacts of the way that alcohol use groups are defined in the meta-analyses used to inform our risk functions.

## Limitations, strengths and future work

This study has limitations. We focused upon the health harms experienced by the alcohol user and did not measure harms to others, financial harms or social harms; this may modify the conclusions compared to other prevention paradox studies. Next, other studies have reported a second-order paradox, wherein non-heavy drinkers (in terms of average intake) together account for the majority of episodes of heavy use [38]. We studied average daily alcohol use, which includes heavy episodic drinking (HED) as a component, but did not study HED separately. However, conclusions regarding the second-order paradox have focused upon acute alcohol-caused problems, whereas the current study is largely focused upon chronic health effects. Relative risk functions used for risk modeling were from published meta-analyses constituting medical epidemiology studies wherein respondents, on average, underestimate their alcohol use. Although we corrected for this, imprecision remains [29]. We acknowledge significant limitations regarding using cross-sectional, survey-based alcohol exposure inputs towards methodologies designed to model alcohol use and related risks throughout the life-course, as there was no direct longitudinal measurement of alcohol use or mortality in each volume category during the life-course. Further, volume of use levels of any individual's life-time will be likely to vary substantially: in the methods used here, these levels are collapsed to the overall life-time mean daily alcohol use in grams/day.

This study benefits from strengths. For health harms we accessed administrative data regarding death and disability, as opposed to self-reports. For mean daily alcohol use, official sales data were used to reflect population use more accurately than self-reported use. A consideration is provided by the alcohol harms paradox, wherein for the same level of alcohol use those with lower socio-economic status (SES) experience higher rates of alcohol-caused harms compared to those with higher SES. It is unknown how this would mediate our conclusions regarding the prevention paradox; this an area for future work.

## Alcohol policy: roughly how

The framing of alcohol-caused health harms as occurring mainly within a small group of heavy drinkers is false. By most measures of 'heavy' use—for example, the top 10% of drinkers by volume—the remaining non-heavy consumers collectively experience the majority of alcohol-caused harms. While it is ethically essential to craft policy interventions for those who currently use alcohol at high levels and/or experience alcohol use disorders, as noted by Skog [38, p. 24], 'preventive polic[ies] targeted exclusively at this high-risk group may not have a substantial effect on' population health.

As suggested by the theory of the collectivity of drinking [39], policies targeting the total consumption of alcohol use in the population will reduce the number of heavy drinkers, as per capita mean use is associated with the prevalence of heavy use: a recent study reiterated this association [40]. Hence, although a comprehensive approach to reducing alcohol-caused harms will include measures directed towards chronic heavy drinkers and patterns of drinking, the current study suggests that population-level policy interventions aimed at reducing alcohol use at all volume levels, such as pricing policies [5] and limits to physical availability [41], may be best suited for the purpose of reducing the total alcohol-caused burden of disease.

## AUTHOR CONTRIBUTIONS

**Adam Sherk:** Conceptualization (lead); data curation (equal); formal analysis (supporting); funding acquisition (equal); investigation (lead); methodology (equal); project administration (equal); resources (equal); software (supporting); supervision (equal); validation (equal); visualization (equal); writing—original draft (equal); writing—review and editing (lead). **Samuel Churchill:** Conceptualization (equal); data curation (lead); formal analysis (lead); funding acquisition (supporting); investigation (supporting); methodology (equal); project administration (supporting); resources (supporting); software (lead); supervision (supporting); validation (equal); visualization (equal); writing—original draft (supporting); writing—review and editing (supporting). **Samantha Cukier:** Conceptualization (equal); methodology (supporting); visualization (supporting); writing—original draft (equal); writing—review and editing (supporting). **Sierra C. Grant:** Conceptualization (supporting); data curation (supporting); formal analysis (supporting); funding acquisition (supporting); investigation (supporting); methodology (supporting); project administration (equal); resources (supporting); software (supporting); supervision (supporting); validation (supporting); visualization (lead); writing—original draft (supporting); writing—review and editing (supporting). **Kevin Shield:** Conceptualization (supporting); data curation (supporting); formal analysis (equal); funding acquisition (supporting); investigation (equal); methodology (equal); project administration (supporting); resources (supporting); software (supporting); supervision (supporting); validation (supporting); visualization (supporting); writing—original draft (supporting); writing—review and editing (equal). **Tim Stockwell:** Conceptualization (supporting); data curation (supporting); formal analysis (supporting); funding acquisition (equal); investigation (supporting); methodology (supporting); project administration (equal); resources (supporting); software (supporting);

supervision (equal); validation (supporting); visualization (supporting); writing—original draft (supporting); writing—review and editing (supporting).

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## DECLARATION OF INTERESTS

None.

## DATA AVAILABILITY STATEMENT

All data used for this study was publicly available secondary data. Anyone wishing to replicate this study could do so without special access to added datasets.

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### SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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