Chemico-Biological Interactions 331 (2020) 109280

Contents lists available at ScienceDirect

Chemico-Biological Interactions

journal homepage: www.elsevier.com/locate/chembioint

Alcohol consumption. A leading risk factor for cancer

Jürgen Rehm [a,b,c,d,e,f,g], Kevin D. Shield [a,b,c], Elisabete Weiderpass [h,*]

[a] WHO Collaboration Centre, Centre for Addiction and Mental Health, 33 Russell Street, Toronto, ON, M5S 2S1, Canada
[b] Institute for Mental Health Policy Research, Centre for Addiction and Mental Health, 33 Russell Street, Toronto, ON, M5S 2S1, Canada
[c] Dalla Lana School of Public Health, University of Toronto, 155 College Street, 6th floor, Toronto, ON, M5T 3M7, Canada
[d] Institute of Medical Science, University of Toronto, Faculty of Medicine, Medical Sciences Building, 1 King's College Circle, Room 2374, Toronto, ON, M5S 1A8, Canada
[e] Campbell Family Mental Health Research Institute, Centre for Addiction and Mental Health, 33 Russell Street, Toronto, ON, M5S 2S1, Canada
[f] Department of Psychiatry, University of Toronto, 250 College Street, 8th floor, Toronto, ON, M5T 1R8, Canada
[g] Institute of Clinical Psychology and Psychotherapy & Center of Clinical Epidemiology and Longitudinal Studies (CELOS), Technische Universität Dresden, Chemnitzer Str. 46, 01187, Dresden, Germany
[h] International Agency for Research on Cancer, 150 Cours Albert Thomas, 69372, CEDEX 8, Lyon, France

ARTICLE INFO

Keywords:
Alcohol consumption
Cancer burden
Risk factor
Prevention

ABSTRACT

In 2016, alcohol consumption was one of the leading risk factors for cancer development and cancer death globally, causing an estimated 376 200 cancer deaths, representing 4.2% of all cancer deaths, and 10.3 million cancer disability-adjusted life years lost, representing 4.2% of all cancer disability-adjusted life years lost. The impact of alcohol consumption on cancer in 2016 varied by age group; the proportion of cancer deaths attributable to alcohol consumption ranged from 13.9% of cancer deaths among people aged 30–34 years to 2.7% of cancer deaths among people aged 80–84 years. The burden of cancers caused by alcohol consumption might be decreased through (i) individual-level and societal-level interventions that reduce alcohol consumption, and (ii) measures that target those risk factors that interact with alcohol consumption to increase the risk of cancer or that directly affect the risk of alcohol-related cancers.

1. Alcohol consumption as a risk factor for cancer

The IARC Monographs [1] and the Continuous Update Project of the World Cancer Research Fund (WCRF)/American Institute for Cancer Research (AICR) [2] have attributed the highest level of causal evidence to the association between consumption of alcoholic beverages and the development of cancer. IARC classified alcohol consumption as carcinogenic to humans (Group 1), and the WCRF/AICR Continuous Update Project concluded that there is convincing evidence that consumption of alcoholic beverages increases cancer risk.

Alcoholic beverages contain numerous carcinogenic compounds, but the majority of the risk relationship between alcohol consumption and the development of cancer is due to ethanol [3]. Although carcinogenesis due to alcohol is far from being fully understood, the main pathophysiological carcinogenic mechanisms of ethanol that have been postulated include its metabolism into the carcinogenic metabolite acetaldehyde, its inhibition of the one-carbon metabolism pathway and DNA methylation (especially among people with a low dietary intake of folate), and its effect on increasing serum levels of endogenous estrogens [2]. Ethanol has also been hypothesized to increase the risk of cancer through the production of reactive oxygen species and polar metabolites, through the conversion of pro-carcinogens in the metabolic pathway of ethanol, by lipid peroxidation, by the production of prostaglandins, by altering the insulin-like growth factor 1 pathway, and by acting as a solvent for cellular penetration of environmental carcinogens (e.g. tobacco) [2]. The biological pathways involved, and the relative contributions of these pathways to carcinogenesis, differ by cancer site.

On the basis of the evidence from epidemiological studies in humans, studies in experimental animals, and mechanistic data, the IARC Monographs and the WCRF/AICR Continuous Update Project have reported that alcohol consumption causes cancers of the oral cavity, oropharynx, hypopharynx, oesophagus (squamous cell carcinoma), colon, rectum, liver and intrahepatic bile duct, larynx, and female breast (both premenopausal and postmenopausal as evaluated by IARC [1]; postmenopausal only as evaluated by the WCRF/AICR Continuous Update Project [2]). For all of these sites, there are dose–response

* Corresponding author.
E-mail address: Director@iarc.fr (E. Weiderpass).

https://doi.org/10.1016/j.cbi.2020.109280
Received 20 May 2019; Accepted 29 September 2020
Available online 1 October 2020
0009-2797/

relationships, with almost linear gradients of relative risks and no apparent lower risk threshold [4,5]. The risk relationships depend mainly on the level of lifetime exposure to alcohol [5,6]. However, for female breast cancer, in addition to the dose–response relationship between level of exposure and cancer incidence, patterns of alcohol consumption, especially episodic heavy drinking, may play an important role [7].

The risk relationships have been shown to differ by population. For example, Mendelian randomization studies have found genetic variations that affect the metabolism of acetaldehyde in humans. In particular, people with at least one copy of the aldehyde dehydrogenase *ALDH2*2* allele (with the Glu487Lys polymorphism), a variant that is prevalent in eastern Asian populations, have a higher risk of cancers of the upper aerodigestive tract and of colorectal cancer [8]. Variations in the alcohol dehydrogenase 1B (*ADH1B*) and 1C (*ADH1C*), cytochrome P450 2E1, and methylenetetrahydrofolate reductase (*MTHFR*) genes are also hypothesized to modify the relationship between alcohol consumption and the development of cancer [9,10].

The WCRF/AICR Continuous Update Project concluded that there is probable evidence that alcohol consumption is associated with the risk of non-cardia stomach cancer, and limited–suggestive evidence that alcohol consumption is associated with the risk of cancers of the lung, pancreas, and skin (basal cell carcinoma and malignant melanoma) [2]. However, alcohol consumption is associated with other risk factors, including diet and smoking, and therefore confounding may explanation these associations. Furthermore, there are inconsistent epidemiological findings for a relationship between alcohol consumption and the development of cancers of the gall bladder and prostate [4]. In addition, there is no evidence that alcohol consumption affects breast cancer survival or recurrence [2].

The WCRF/AICR Continuous Update Project concluded that there is probable evidence that alcohol consumption is associated with a decreased risk of kidney cancer; this may be due to improved insulin sensitivity, improved blood lipid profiles, and higher adiponectin levels among people with light and moderate alcohol consumption [2]. Resveratrol (a substance found in red wine) has received attention for its hypothesized anticarcinogenic properties; however, based on the empirical evidence, for every cancer case that the resveratrol in wine prevents, 100 000 cancer cases are caused by ethanol [5]. Inconsistent inverse associations between alcohol consumption and the development of thyroid cancer, Hodgkin lymphoma, and non-Hodgkin lymphoma also have been found in epidemiological studies, but there is currently not sufficient evidence to determine the causality of these relationships [1,2].

As a result of its effects on the propensity to engage in unprotected sex and its weakening of the immune system, alcohol also may indirectly increase the risk of infection with sexually transmitted viruses that potentially cause cancer (including Kaposi sarcoma-associated herpesvirus and human papillomavirus) and of HIV-1; the immunosuppression caused by HIV-1 is thought to increase the carcinogenic effect of other infectious agents [5]. However, more research is needed to further establish and quantify any indirect effect of alcohol on an increased risk of cancers caused by infectious diseases.

2. The global cancer burden due to alcohol

In 2016, alcohol consumption caused an estimated 3.0 million deaths from all causes worldwide, representing 5.3% of all deaths [11]. A large proportion of the health burden caused by alcohol consumption stems from cancer. In 2016, alcohol caused an estimated 376 200 (95% uncertainty interval, 324 900–439 700) cancer deaths, representing 4.2% (95% uncertainty interval, 3.6–4.9%) of all cancer deaths, and an age-standardized rate (ASR) of 4.8 deaths (95% confidence interval, 4.2–5.7) per 100 000 people (Table 1). Here, the term "alcohol--attributable cancers" is used to refer to cancers caused by alcohol. The proportion of alcohol-attributable cancers is thus defined by the proportion of cancers that would not have occurred if there had been no alcohol exposure (for definitions of causality, see [12]; for alcohol-attributable fractions, see [13]).

ICD-10, International Statistical Classification of Diseases and Related Health Problems, 10th revision.

Of the 245 million disability-adjusted life years (DALYs) lost in 2016 due to cancer, 10.3 million (95% uncertainty interval, 8.7 million–12.0 million) were due to alcohol consumption, representing 4.2% (95% uncertainty interval, 3.6–4.9%) of all cancer DALYs lost (Table 2). The majority (97.7%) of these alcohol-attributable cancer DALYs lost were due to years of life lost because of premature death resulting from high cancer fatality rates.

DALYs, disability-adjusted life years; ICD-10, International Statistical Classification of Diseases and Related Health Problems, 10th revision.

In 2016, cancers of the colorectum, liver, and oesophagus were the largest contributors to the alcohol-attributable cancer burden, responsible for 23.9%, 22.3%, and 19.3%, respectively, of all alcohol-attributable cancer deaths.

Among all cancers types, alcohol consumption had the largest impact on cancers of the upper aerodigestive tract. Alcohol was responsible for 26.4% of all cancers of the lip and oral cavity, 30.5% of all other

Table 1
Alcohol-attributable cancer deaths in 2016, by sex and cancer site.

Outcome and cancer site	ICD-10 code	Number of alcohol-attributable deaths/1000 (95% uncertainty interval)			Percentage of deaths attributable to alcohol consumption (95% uncertainty interval)			Percentage of the total alcohol-attributable cancer deaths
		Men	Women	Both sexes	Men	Women	Both sexes	
Cancer	C00–97	297.6 (246.9–346.1)	78.6 (66–115.4)	376.2 (324.9–439.7)	5.8 (4.8–6.8)	2.0 (1.7–3.0)	4.2 (3.6–4.9)	100.0
Lip and oral cavity	C00–08	38.9 (30.4–46.0)	5.2 (3.8–7.3)	44.0 (35.3–52.3)	34.7 (27.1–41.0)	9.4 (7.0–13.3)	26.4 (21.2–31.4)	11.7
Other pharynx	C09–10, C12–14	31.7 (24.9–37.7)	2.1 (1.5–3.0)	33.8 (27.0–39.9)	35.3 (27.8–42.1)	9.9 (7.3–14.2)	30.5 (24.4–36.1)	9.0
Oesophagus	C15	66.9 (51.6–79.7)	5.8 (3.9–8.9)	72.7 (56.8–87.2)	21.7 (16.7–25.8)	4.8 (3.2–7.4)	16.9 (13.2–20.3)	19.3
Colorectum	C18–21	75.9 (61.5–89.6)	13.8 (6.6–25.2)	89.8 (73.1–107.4)	17.6 (14.3–20.7)	3.8 (1.8–6.9)	11.3 (9.2–13.5)	23.9
Liver	C22	65.1 (31.5–102.5)	18.9 (9.5–34.4)	84.0 (49.8–125.3)	11.1 (5.4–17.5)	7.8 (3.9–14.1)	10.1 (6.0–15.1)	22.3
Larynx	C32	19.1 (14.8–23.1)	0.8 (0.6–1.0)	19.9 (15.6–24.0)	23.7 (18.4–28.6)	6.7 (5.2–9.2)	21.6 (16.9–26.1)	8.5
Breast	C50	–	32.0 (26.8–51.1)	32.0 (26.8–51.1)	–	5.5 (4.6–8.8)	5.5 (4.6–8.7)	5.3
All causes	A00–Z99	2307.3 (1929.7–2720.1)	681.0 (536.4–990.7)	2988.3 (2596.8–3523.9)	7.7 (6.4–9.0)	2.6 (2.0–3.8)	5.3 (4.6–6.2)	–

Table 2
Alcohol-attributable cancer disability-adjusted life-years lost in 2016, by sex and cancer site.

Outcome and cancer site	ICD-10 code	Number of alcohol-attributable DALYs lost/100 000 (95% uncertainty interval)			Percentage of DALYs lost attributable to alcohol consumption (95% uncertainty interval)			Percentage of the total alcohol-attributable cancer DALYs lost
		Men	Women	Both sexes	Men	Women	Both sexes	
Cancer	C00–97	81.6 (67.0–95.9)	21.1 (18.0–31.4)	102.6 (87.3–120.0)	5.9 (4.9–7.0)	2.0 (1.7–2.9)	4.2 (3.6–4.9)	100.0
Lip and oral cavity	C00–08	12.2 (9.2–14.7)	1.4 (1.0–2.0)	13.6 (10.6–16.5)	33.2 (25.0–40.0)	8.6 (6.3–12.2)	25.7 (19.9–31.0)	13.3
Other pharynx	C09–10, C12–14	9.7 (7.6–11.6)	0.6 (0.4–0.9)	10.3 (8.2–12.3)	35.5 (27.6–42.5)	9.3 (6.8–13.4)	30.6 (24.1–36.5)	10.1
Oesophagus	C15	17.7 (13.8–20.9)	1.4 (0.9–2.1)	19.0 (15.0–22.6)	22.1 (17.2–26.1)	4.7 (3.2–7.2)	17.5 (13.8–20.7)	18.5
Colorectum	C18–21	18.0 (14.4–21.4)	3.2 (1.6–5.8)	21.2 (17.2–25.3)	16.7 (13.4–19.9)	3.8 (1.9–6.9)	11.1 (9.0–13.2)	20.6
Liver	C22	18.6 (8.9–29.7)	4.5 (2.3–8.2)	23.1 (13.2–35.0)	10.7 (5.1–17.0)	7.2 (3.7–13.1)	9.7 (5.6–14.8)	22.5
Larynx	C32	5.4 (4.2–6.5)	0.2 (0.2–0.3)	5.6 (4.4–6.7)	23.8 (18.5–28.8)	6.7 (5.2–9.0)	21.8 (17.0–26.3)	9.6
Breast	C50	–	9.9 (8.2–16.4)	9.9 (8.2–16.4)	–	5.2 (4.4–8.7)	5.2 (4.3–8.6)	5.4
All causes	A00–Z99	1065.4 (903.2–1240.8)	261.0 (234.4–331.5)	1326.4 (1164.1–1539.8)	7.6 (6.5–8.9)	2.2 (1.9–2.7)	5.1 (4.5–5.9)	–

pharyngeal cancers (excluding nasopharyngeal cancers), 21.6% of all laryngeal cancers, and 16.9% of all oesophageal cancers. These findings reflect the stronger associations – i.e. the higher gradients of the dose–response curves – between levels of alcohol consumption and cancers of the upper aerodigestive tract compared with cancers of the colorectum, liver, and breast [11].

Like with cancer deaths, in 2016 the largest contributors to the alcohol-attributable cancer DALYs lost were cancers of the liver, colorectum, and oesophagus, responsible for 22.5%, 20.6%, and 18.5%, respectively, of all alcohol-attributable cancer DALYs lost.

Similarly, alcohol consumption had the largest contributory impact on DALYs lost due to cancers of the upper aerodigestive tract. Alcohol was responsible for 25.7% of all lip and oral cavity cancer DALYs lost, 30.6% of all other pharyngeal cancer DALYs lost (excluding nasopharyngeal cancers), 21.8% of all laryngeal cancer DALYs lost, and 17.5% of all oesophageal cancer DALYs lost.

Based on different consumption levels by age [14], the impact of alcohol consumption on cancer in 2016 varied by age group (Fig. 1); the proportion of cancer deaths attributable to alcohol consumption ranged from 13.9% of cancer deaths among people aged 30–34 years to 2.7% of cancer deaths among people aged 80–84 years. At younger ages, cancers of the liver, breast, and colorectum were the leading contributors to the alcohol-attributable cancer burden, responsible for 32.2%, 19.4%, and 18.4%, respectively, of all alcohol-attributable cancer deaths among people aged 30–34 years. At older ages, cancers of the colorectum, liver, and oesophagus were the leading contributors to the alcohol-attributable cancer burden, responsible for 39.1%, 20.1%, and 14.9%, respectively, of all alcohol-attributable cancer deaths among people aged 80 years and older. The impact of alcohol on cancer deaths and DALYs lost among people aged 29 years and younger is unknown, because data are lacking and the etiology of these cancers is complex; however, the proportion of alcohol-attributable cancers among this age group is hypothesized to be relatively small [15].

In 2016, there were large variations between countries and geographical regions in the ASRs of alcohol-attributable cancer deaths (Fig. 2) and cancer DALYs lost (Fig. 3). Based on the regions as defined by the Institute for Health Metrics and Evaluation's Global Burden of Disease study, the burden of alcohol-attributable cancers was lowest in North Africa and the Middle East (ASRs of 0.8 cancer deaths and 24.2 cancer DALYs lost per 100 000 people) and highest in eastern Europe (ASRs of 12.0 cancer deaths and 360.4 cancer DALYs lost per 100 000 people).

Similarly, the proportion of alcohol-attributable cancer deaths and cancer DALYs lost also varied between countries and regions. The proportions were lowest in North Africa and the Middle East (0.8% of cancer deaths and 0.8% of cancer DALYs lost) and highest in eastern Europe (8.1% of cancer deaths and 8.6% of cancer DALYs lost).

The burden of cancer by site also varied across geographical regions (Fig. 4). In particular, alcohol-attributable cancers of the colorectum were prominent in southern Latin America, high-income North America, high-income Asia Pacific, Australasia, and central, eastern, and western Europe; all of these regions have countries with high or very high levels of the Human Development Index (HDI).

Both the consumption of alcohol and the burden of cancer increase as countries develop [11,16]. In 2016, the ASRs of the alcohol-attributable cancer burden were highest for countries with very high HDI (7.3 cancer deaths and 203.8 cancer DALYs lost per 100 000 people) and lowest for countries with medium HDI (2.5 cancer deaths and 78.8 cancer DALYs lost per 100 000 people) (Fig. 5). The site-specific alcohol-attributable cancer burden also varied by HDI. The largest contributors to the ASRs of alcohol-attributable cancer deaths were colorectal cancer in countries with very high HDI, liver cancer in countries with low HDI and countries with high HDI, and cancers of the lip and oral cavity in countries with medium HDI.

The alcohol-attributable cancer deaths and cancer DALYs lost discussed above include only cancer sites for which sufficient causal evidence exists, as determined by the IARC Monographs, and do not include cancer sites for which there was insufficient evidence of carcinogenicity in humans [1]. However, an analysis conducted for France in 2015 found that the proportion of cancer incidence due to alcohol increased from 7.9% when limited to cancers for which sufficient causal evidence exists to 8.4% when including cancers for which at least limited evidence of a causal association exists [17].

Country- and region-specific analyses of the relative contributions of risk factors to the cancer burden in the USA [18], France [15], the United Kingdom [19], Australia [20], and the Nordic countries (Denmark, Finland, Iceland, Norway, Sweden, the Faroe Islands, and Greenland) [21] have shown that alcohol is a leading risk factor for cancer development and cancer death. In some analyses and countries, alcohol is the second most important risk factor for cancer development and cancer death after tobacco, for example in an analysis of nine behavioural and environmental risk factors for the Global Burden of Disease 2000 study [22] and in an analysis of 13 risk factors for France in 2015 [15].

Trends in the cancer burden due to alcohol from 2010 to 2016.

Trends in the alcohol-attributable cancer burden depend on changes

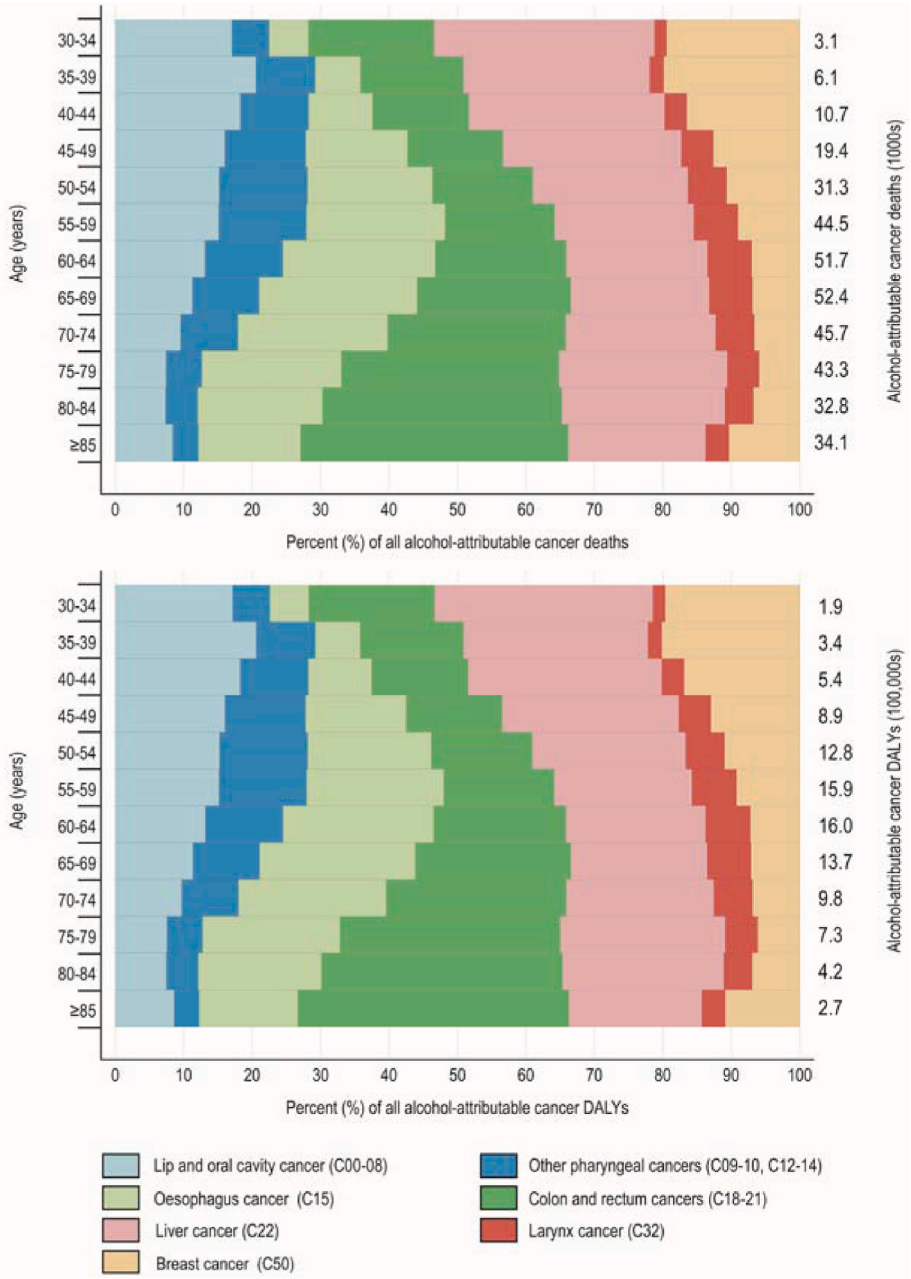

Fig. 1. Alcohol-attributable cancer deaths (top) and alcohol-attributable cancer disability-adjusted life years (DALYs) lost (bottom) in 2016, by age group [11].

in alcohol consumption as well as in cancer incidence, treatment, and mortality. As a result of population growth and ageing and the economic development of countries, the total number of cancer deaths worldwide increased from 8.1 million in 2010 to 9.0 million in 2016 [11]. However, the ASR of cancer mortality decreased by 6.0% (from 122.4 per 100 000 in 2010 to 115.0 per 100 000 in 2016), less than the 9.0% decrease in the ASR of overall mortality (from 791.3 per 100 000 in 2010 to 720.1 per 100 000 in 2016).

The ASRs of alcohol-attributable mortality decreased less than overall cancer mortality rates in general (by 4.8%, from 5.1 deaths per 100 000 in 2010 to 4.8 deaths per 100 000 in 2016), resulting in an increase of 1.5% in the proportion of cancer deaths attributable to alcohol consumption (from 4.1% in 2010 to 4.2% in 2016). Thus, the relative impact of alcohol on cancer mortality increased slightly from 2010 to 2016.

Trends in the ASRs of alcohol-attributable cancer mortality and in the proportion of cancers attributable to alcohol consumption showed heterogeneous patterns by cancer site. In particular, the ASR of mortality due to cancers of the lip and oral cavity was the only ASR to increase (from 2.1 deaths per 100 000 in 2010 to 2.2 deaths per 100 000 in 2016), and the ASR of mortality due to oesophageal cancer decreased the most (from 6.2 deaths per 100 000 in 2010 to 5.5 deaths per 100 000 in 2016).

In the long term, increases in the economic wealth of countries are likely to lead to further increases in life expectancies, resulting in higher incidence of and mortality from cancer and a concomitant higher relative importance of cancer as a cause of death (http://www.healthdata.org/results/country-profiles), as well as to higher per capita alcohol consumption [11,14]. Furthermore, because the median latency between mean alcohol consumption and the diagnosis of cancer is 10 years [23], it is expected that alcohol-attributable cancer mortality will continue to increase in the countries that have had the most pronounced increases in alcohol consumption over the past few years. Examples of such countries are China and India, countries in which life expectancies

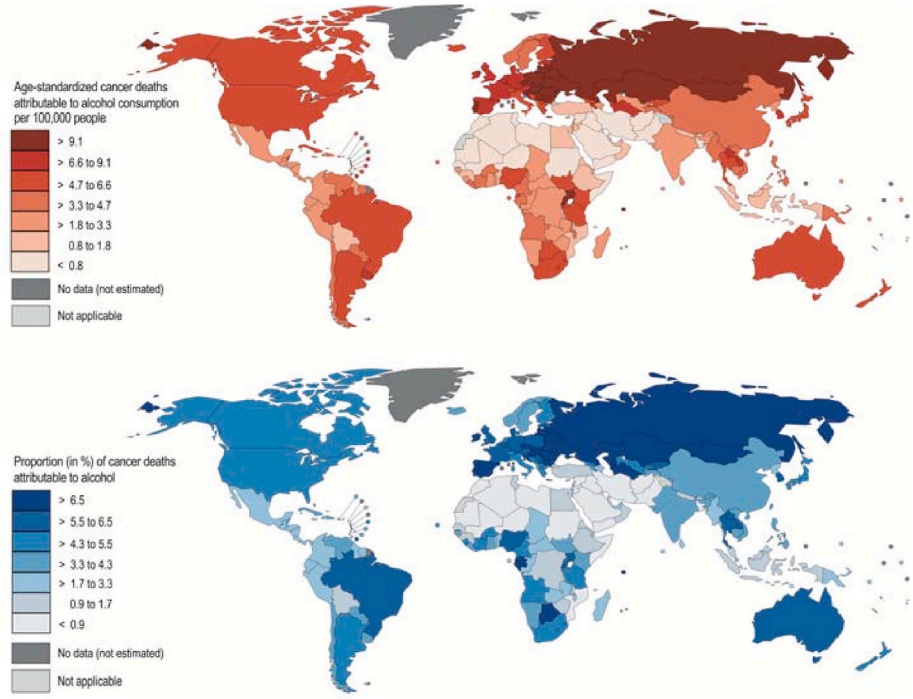

Fig. 2. Global burden of cancer deaths caused by alcohol consumption in 2016: (top) age-standardized cancer deaths attributable to alcohol consumption per 100 000 people; (bottom) percentage of cancer deaths attributable to alcohol [11].

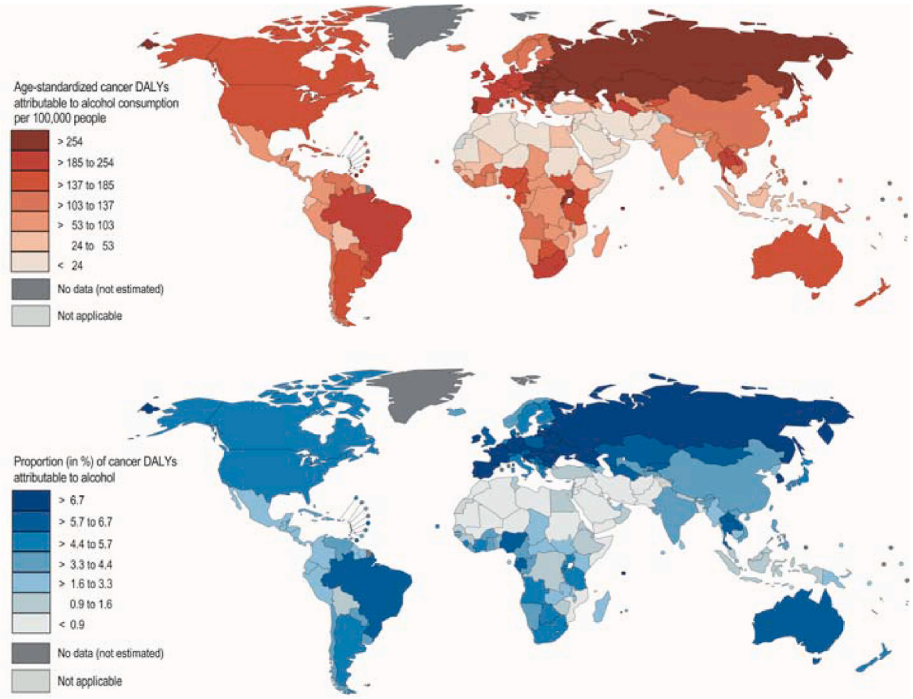

Fig. 3. Global burden of cancer disability-adjusted life years (DALYs) lost caused by alcohol consumption in 2016: (top) age-standardized cancer DALYs lost attributable to alcohol consumption per 100 000 people; (bottom) percentage of cancer DALYs lost attributable to alcohol [11].

have also increased [11]; http://www.healthdata.org/results/country-profiles). Accordingly, whereas in high-income countries alcohol consumption, cancer mortality rates, and alcohol-attributable cancer mortality rates have declined, and may continue to decline, the overall global burden of alcohol-attributable cancers is not expected to decrease, and may increase in the long term.

The cancer burden due to alcohol is preventable.

The current burden of cancers caused by alcohol consumption is large, and this burden is expected to increase in the future. Therefore, programmes designed to reduce alcohol consumption in the general population are an effective and cost-effective means of targeting and improving cancer control. The observed differences between countries and regions in alcohol-attributable fractions of cancer deaths and cancer DALYs lost provide an evidence base for how to reduce this burden

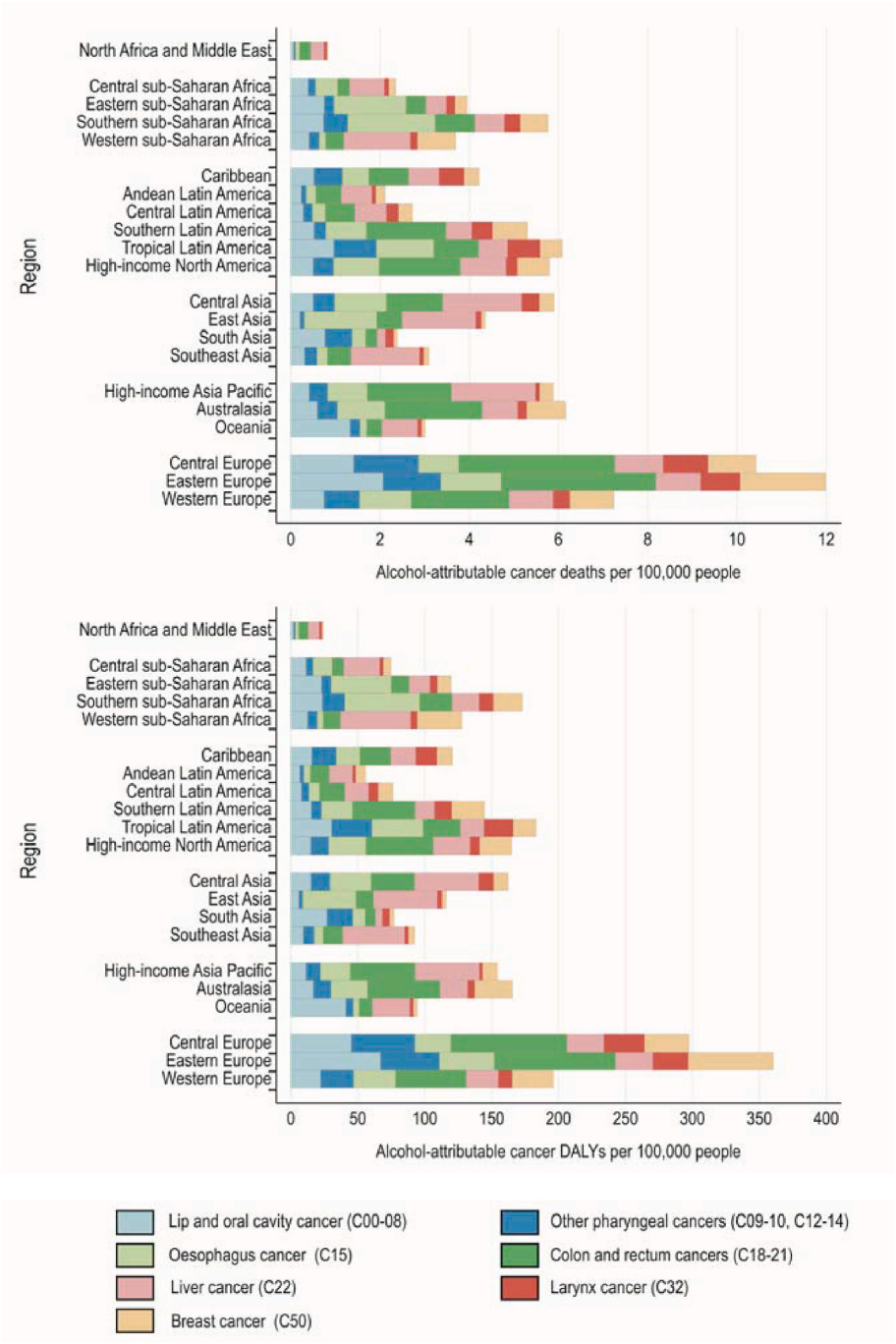

Fig. 4. Age-standardized alcohol-attributable cancer deaths per 100 000 people (top) and alcohol-attributable cancer disability-adjusted life years (DALYs) lost per 100 000 people (bottom) in 2016, by geographical region [11].

through individual-level and societal-level programmes that reduce alcohol consumption, such as the WHO intervention strategies known as alcohol policy "best buys", which include increasing excise taxation of alcoholic beverages, restricting access to retailed alcoholic beverages, and limiting advertising and promotion of alcoholic products [24]. Furthermore, the burden of alcohol-attributable cancers could be reduced through measures that target those risk factors that interact with alcohol consumption to increase the risk of cancer or that directly affect the risk of alcohol-related cancers, such as tobacco smoking. In addition, early recognition of the signs and symptoms of cancer, as well as prompt diagnosis of precancerous lesions and tumours, are in many cases vital to patient survival, and therefore screening for colorectal cancer and breast cancer may also reduce the burden of alcohol-attributable cancers [25]. Finally, despite the evidence of the causal relationship between alcohol consumption and the development of cancer, the majority of the general population is unaware of this causal link [26]. Warning labels can be used to raise awareness of the link between alcohol and cancer; however, the effectiveness of these labels to reduce alcohol consumption is currently unknown [11]. In addition, explaining the causal link between alcohol and cancer could be part of brief interventions by medical professionals in primary care, to reduce alcohol consumption [27].

Declaration of competing interest

The authors declare that they have no known competing financial

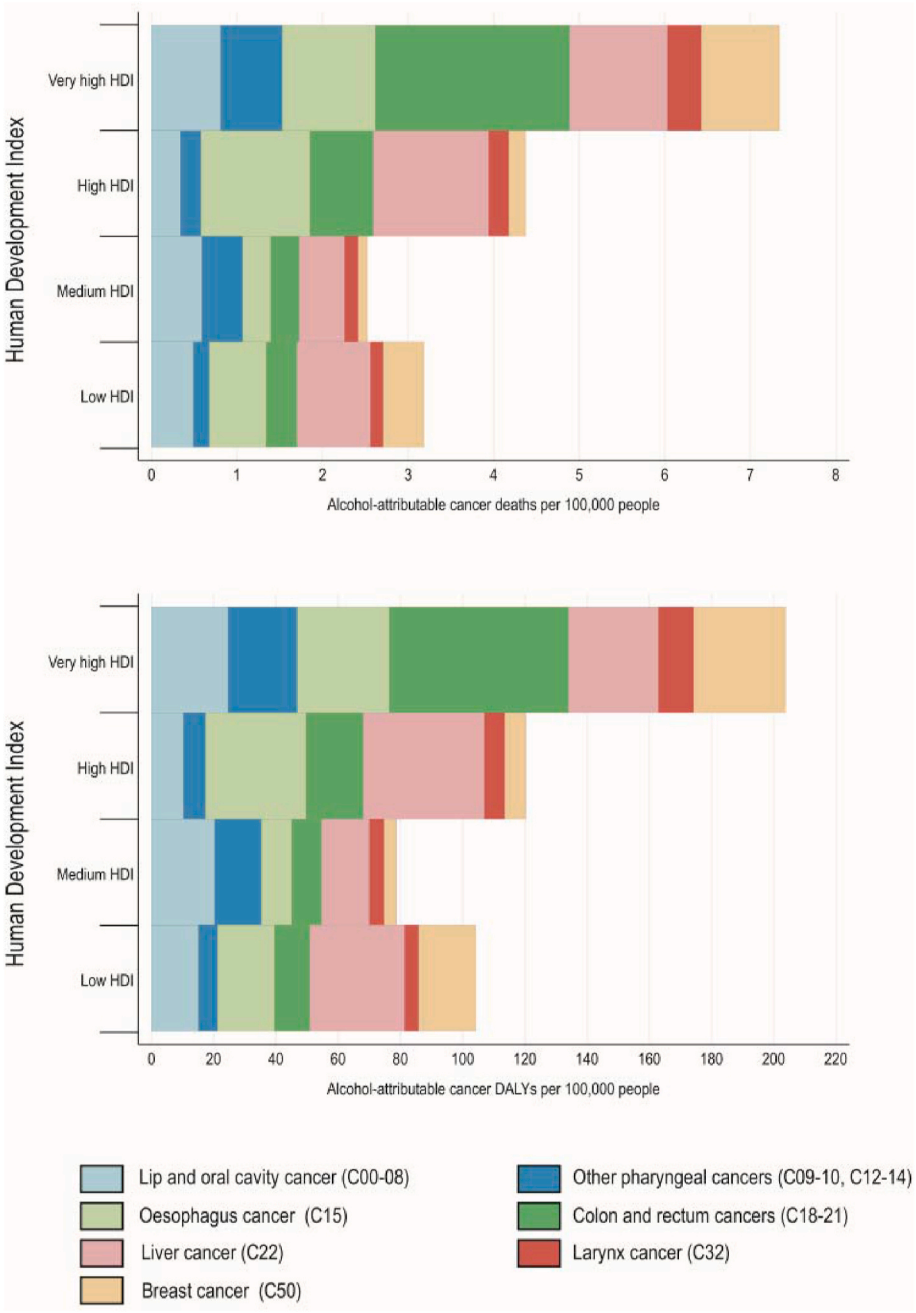

Fig. 5. Age-standardized alcohol-attributable cancer deaths per 100 000 people (top) and alcohol-attributable disability-adjusted life years (DALYs) lost per 100 000 people (bottom) in 2016, by level of Human Development Index (HDI) [11].

interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cbi.2020.109280.

References

[1] IARC, Personal Habits and Indoor Combustions, 2012. IARC Monogr Eva, Carcinog Risks Hum. 100E:1–575. PMID:23193840. Available from: http://publications.iarc.fr/122.

[2] WCRF/AICR, Diet, nutrition, physical activity and cancer: a global perspective, Continuous Update Project Expert Report 2018, World Cancer Research Fund/ American Institute for Cancer Research, 2018, https://www.wcrf.org/dietandcancer.

[3] T. Pflaum, T. Hausler, C. Baumung, S. Ackermann, T. Kuballa, J. Rehm, et al., Carcinogenic compounds in alcoholic beverages: an update, Arch. Toxicol. 90 (10) (2016) 2349–2367, https://doi.org/10.1007/s00204-016-1770-327353523.

[4] V. Bagnardi, M. Rota, E. Botteri, I. Tramacere, F. Islami, V. Fedirko, et al., Alcohol consumption and site-specific cancer risk: a comprehensive dose-response meta-analysis, Br. J. Canc. 112 (3) (2015) 580–593, https://doi.org/10.1038/bjc.2014.57925422909.

[5] J. Rehm, G.E. Gmel Sr., G. Gmel, O.S.M. Hasan, S. Imtiaz, S. Popova, et al., The relationship between different dimensions of alcohol use and the burden of disease – an update, Addiction 112 (6) (2017) 968–1001, https://doi.org/10.1111/add.1375728220587.

[6] M.M. Bergmann, J. Rehm, K. Klipstein-Grobusch, H. Boeing, M. Schütze, D. Drogan, et al., The association of pattern of lifetime alcohol use and cause of death in the European Prospective Investigation into Cancer and Nutrition (EPIC) study, Int. J. Epidemiol. 42 (6) (2013) 1772–1790, https://doi.org/10.1093/ije/dyt15424415611.

[7] Y. Liu, N. Nguyen, G.A. Colditz, Links between alcohol consumption and breast cancer: a look at the evidence, Womens Health (Lond) 11 (1) (2015) 65–77, https://doi.org/10.2217/WHE.14.6225581056.
[8] B.L. Pierce, P. Kraft, C. Zhang, Mendelian randomization studies of cancer risk: a literature review, Curr. Epidemiol. Rep. 5 (2) (2018) 184–196, https://doi.org/10.1007/s40471-018-0144-130034993.
[9] N. Druesne-Pecollo, B. Tehard, Y. Mallet, M. Gerber, T. Norat, S. Hercberg, et al., Alcohol and genetic polymorphisms: effect on risk of alcohol-related cancer, Lancet Oncol. 10 (2) (2009) 173–180, https://doi.org/10.1016/S1470-2045(09)70019-119185835.
[10] N.K. LoConte, A.M. Brewster, J.S. Kaur, J.K. Merrill, A.J. Alberg, Alcohol and cancer: a statement of the American society of clinical oncology, J. Clin. Oncol. 36 (1) (2018) 83–93, https://doi.org/10.1200/JCO.2017.76.115529112463.
[11] WHO, Global Status Report on Alcohol and Health 2018, World Health Organization, Geneva, Switzerland, 2018. Available from: https://www.who.int/substance_abuse/publications/global_alcohol_report/en/.
[12] K.J. Rothman, S. Greenland, T.L. Lash, Modern Epidemiology, third ed., Lippincott Williams & Wilkins, Philadelphia (PA), USA, 2008.
[13] K.D. Shield, D.M. Parkin, D.C. Whiteman, J. Rehm, V. Viallon, C.M. Micallef, et al., Population attributable and preventable fractions: cancer risk factor surveillance, and cancer policy projection, Curr. Epidemiol. Rep. 3 (3) (2016) 201–211, https://doi.org/10.1007/s40471-016-0085-527547696.
[14] J. Manthey, K.D. Shield, M. Rylett, O.S.M. Hasan, C. Probst, J. Rehm, Global Alcohol Exposure between 1990 and 2017 and Forecasts until 2030: a Modelling Study, 2019, https://doi.org/10.1016/S0140-6736(18)32744-2. Lancet. [Epub ahead of print].
[15] I. Soerjomataram, K. Shield, C. Marant-Micallef, J. Vignat, C. Hill, A. Rogel, et al., Cancers related to lifestyle and environmental factors in France in 2015, Eur. J. Canc. 105 (2018) 103, https://doi.org/10.1016/j.ejca.2018.09.00930445359.
[16] F. Bray, A. Jemal, N. Grey, J. Ferlay, D. Forman, Global cancer transitions according to the Human Development Index (2008-2030): a population-based study, Lancet Oncol. 13 (8) (2012) 790–801, https://doi.org/10.1016/S1470-2045(12)70211-522658655.
[17] K.D. Shield, C. Marant Micallef, C. Hill, M. Touvier, P. Arwidson, C. Bonaldi, et al., New cancer cases in France in 2015 attributable to different levels of alcohol consumption, Addiction 113 (2) (2018) 247–256, https://doi.org/10.1111/add.1400928833736.
[18] F. Islami, A. Goding Sauer, K.D. Miller, R.L. Siegel, S.A. Fedewa, E.J. Jacobs, et al., Proportion and number of cancer cases and deaths attributable to potentially modifiable risk factors in the United States, CA A Cancer J. Clin. 68 (1) (2018) 31–54, https://doi.org/10.3322/caac.2144029160902.
[19] D.M. Parkin, L. Boyd, L.C. Walker, 16. The fraction of cancer attributable to lifestyle and environmental factors in the UK in 2010, Br. J. Canc. 105 (Suppl 2) (2011) S77–S81, https://doi.org/10.1038/bjc.2011.48922158327.
[20] D.C. Whiteman, P.M. Webb, A.C. Green, R.E. Neale, L. Fritschi, C.J. Bain, et al., Cancers in Australia in 2010 attributable to modifiable factors: summary and conclusions, Aust. N. Z. J. Publ. Health 39 (5) (2015) 477–484, https://doi.org/10.1111/1753-6405.1247126437735.
[21] T.M. Andersson, G. Engholm, E. Pukkala, M. Stenbeck, L. Tryggvadottir, H. Storm, et al., Avoidable cancers in the Nordic countries – the impact of alcohol consumption, Eur. J. Canc. 103 (2018) 299–307, https://doi.org/10.1016/j.ejca.2018.03.02729739641.
[22] G. Danaei, S. Vander Hoorn, A.D. Lopez, C.J. Murray, M. Ezzati, Comparative Risk Assessment collaborating group (Cancers), Causes of cancer in the world: comparative risk assessment of nine behavioural and environmental risk factors, Lancet 366 (9499) (2005) 1784–1793, https://doi.org/10.1016/S0140-6736(05)67725-216298215.
[23] J. Holmes, P.S. Meier, A. Booth, Y. Guo, A. Brennan, The temporal relationship between per capita alcohol consumption and harm: a systematic review of time lag specifications in aggregate time series analyses, Drug Alcohol Depend. 123 (1–3) (2012) 7–14, https://doi.org/10.1016/j.drugalcdep.2011.12.00522197480.
[24] D. Chisholm, D. Moro, M. Bertram, C. Pretorius, G. Gmel, K. Shield, et al., Are the “best buys” for alcohol control still valid? An update on the comparative cost-effectiveness of alcohol control strategies at the global level, J. Stud. Alcohol Drugs 79 (4) (2018) 514–522, https://doi.org/10.15288/jsad.2018.79.51430079865.
[25] R.A. Smith, K.S. Andrews, D. Brooks, S.A. Fedewa, D. Manassaram-Baptiste, D. Saslow, et al., Cancer screening in the United States, 2018: a review of current American Cancer Society guidelines and current issues in cancer screening, CA A Cancer J. Clin. 68 (4) (2018) 297–316, https://doi.org/10.3322/caac.2144629846940.
[26] J.K. Scheideler, W.M.P. Klein, Awareness of the link between alcohol consumption and cancer across the world: a review, Canc. Epidemiol. Biomarkers Prev. 27 (4) (2018) 429–437, https://doi.org/10.1158/1055-9965.EPI-17-064529615419.
[27] T.F. Babor, J.C. Higgins-Biddle, Alcohol screening and brief intervention: dissemination strategies for medical practice and public health, Addiction 95 (5) (2000) 677–686, https://doi.org/10.1046/j.1360-0443.2000.9556773.x.