

# Preventable cancer cases and deaths attributable to alcohol consumption in Korea from 2015 to 2030

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## Graphical Abstract

### Preventable cancer cases and deaths attributable to alcohol consumption in Korea from 2015 to 2030



#### Objectives



To estimate the fractions of cancer incidence and mortality attributable to alcohol consumption

#### Methods



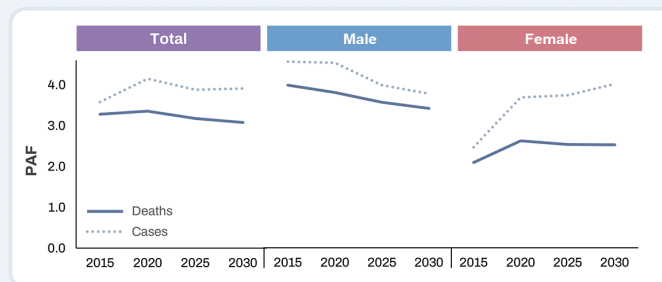
The population-attributable fraction (PAF) of cancer was estimated using a cohort study-based meta-analysis of relative risk (RR), national prevalence rates of alcohol consumption from 2000 to 2015, and national cancer statistics from 2015 to 2030, with a latency of 15 years.

#### Results



**Cases**  
7,727 (3.58%)

**Deaths**  
2,511 (3.28%)



#### Conclusions

This study highlights the impact of alcohol consumption on cancer in Korea, emphasizing the need for sex-specific regulation to address sex differences.

## Key Message:

In 2015, alcohol consumption was responsible for 3.58% of all cancer cases and 3.28% of cancer deaths in Korea, with a more significant impact on males (4.58% of new cases) than females (2.08%). Projections indicate a decrease in alcohol-related cancer cases among males but a sharp increase in females by 2030. These findings highlight the need for sex-specific public health measures to address the growing impact of alcohol on cancer, particularly the increasing trend in female cases.

## ORIGINAL ARTICLE

# Preventable cancer cases and deaths attributable to alcohol consumption in Korea from 2015 to 2030

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**OBJECTIVES:** Alcohol consumption is causally linked to several cancers, and major health organizations classify it as a carcinogen. This study assessed the impact of alcohol consumption on cancer incidence and mortality in Korea in 2015 and 2020, projected trends up to 2030, and compared results based on different criteria.

**METHODS:** The relative risk of cancer associated with alcohol consumption in Korea was determined through a meta-analysis of alcohol-related relative risks for specific cancers, using primary data from the Korean Cohort Study within the Korean Cohort Consortium. The population-attributable fraction (PAF) was calculated using Levin's formula, incorporating drinking prevalence and the number of cancer cases and deaths, with a 15-year latency period assumed.

**RESULTS:** In Korea, the PAF for alcohol consumption, based on ever/never drinking criteria, was higher than that calculated using other criteria, except for the PAF based on past and current/never drinking criteria. Alcohol consumption contributed to 3.58% of all cancer cases and 3.28% of cancer deaths in 2015. It accounted for 4.58% of new cancer cases in male and 2.08% in female, with a higher contribution to incidence than mortality (4.00 and 2.25% of cancer deaths in male and female, respectively). Projections indicate that alcohol-related cancer PAF will decrease by 17.2% in male but increase by 70.2% in female by 2030.

**CONCLUSIONS:** This study highlights the impact of alcohol consumption on cancer in Korea, emphasizing the need for sex-specific regulations to address sex differences.

**KEY WORDS:** Alcohol drinking, Population-attributable fraction, Epidemiology, Korea, Neoplasms

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## INTRODUCTION

The International Agency for Research on Cancer (IARC) has classified alcohol and its byproduct, acetaldehyde, as Group 1 carcinogens for cancers of the oral cavity, pharynx, larynx, esophagus, colorectum, liver, and breast [1,2]. Additionally, the World Cancer Research Fund/American Institute for Cancer Research has classified alcohol consumption as having a convincing grade of evidence for risks of mouth, pharynx, and larynx (MPL) cancer (2018), esophageal squamous cell carcinoma (2016), colorectal cancer (2017), liver cancer (2015), and postmenopausal breast cancer (2017), and as having a probable grade for gastric (2016) and premenopausal breast cancer (2017) [3] (Supplementary Material 1). However, lung (2017) and pancreatic cancer (2012), as well as malignant melanoma and basal cell carcinoma of the skin (2017), were classified as having limited evidence, while kidney cancer (2015) was classified as having a probable grade of reducing risk [3].

For cancers other than breast cancer, specific thresholds have been suggested for alcohol consumption, beyond which cancer risk increases. For colorectal cancer, a minimum threshold of 30 g/day has been proposed, while thresholds of 45 g/day have been suggested for liver, gastric, and pancreatic cancers [4]. However, the relationship between lower alcohol doses and cancer risk remains controversial [4]. The association between light-to-moderate alcohol consumption and cancer risk is inconsistent and uncertain, partly due to information bias, including underreporting [5-7]. A meta-analysis of 572 studies found that alcohol-related cancer risk follows a non-threshold positive (linear or non-linear) dose-response relationship [8]. Consequently, the World Health Organization (WHO) has stated that even minimal alcohol consumption can contribute to cancer risk [9]. The IARC supports this view, suggesting that the relationship between alcohol consumption and cancer should be considered a 'no minimum threshold' scenario, where any exposure increases cancer risk, and recommends calculating the attributable fraction accordingly [4].

The biological mechanisms by which alcohol induces carcinogenesis are not fully understood, but it is known that alcohol causes permanent damage to cellular DNA. Specifically, acetaldehyde,

a metabolite of alcohol, interferes with DNA repair processes and suppresses DNA repair mechanisms [4]. Genetic mutations resulting from alcohol consumption are directly linked to cancer incidence [2,10]. Alcohol also indirectly influences the regulation of the estrogen pathway, increasing the risk of endocrine-related cancers such as breast cancer, and contributes to carcinogenesis by causing nutritional deficiencies [11].

In Korea, the monthly alcohol drinking rate has been steadily increasing among female while decreasing among male [12]. These changes in drinking patterns can affect the population-attributable fraction (PAF) of cancer attributable to alcohol consumption. The National Cancer Center of Korea previously calculated the contribution of alcohol consumption to cancer incidence and mortality in 2009, based on risk factors from 1990 [13,14]. Given recent shifts in drinking prevalence, it is essential to reassess changes in PAF.

In this study, using a 15-year latency period, we calculated the PAF of cancer incidence and deaths attributable to alcohol consumption in 2015 and 2020, based on data from the Korean population in 2000 and 2005. The theoretical minimum risk exposure level for non-alcohol drinking was used for these calculations.

## MATERIALS AND METHODS

### Definition of alcohol drinking exposure and estimation of exposure prevalence

The safest level of alcohol intake concerning cancer risk was defined as no alcohol consumption, with daily alcohol intake set to zero. The amount of pure alcohol consumed, if any, was calculated for those who reported drinking. Data on alcohol drinking rates and the average amount of alcohol consumed by drinkers in the Korean population were obtained from the Korea National Health and Nutrition Examination Survey (KNHANES) [12]. Exposure prevalence and average consumption rates were calculated for adults aged 20 years and older, stratified by sex. The alcohol consumption rate in 2000 was estimated by standardizing the ever-drinking rates among adults aged 20 and older from the 1998 KNHANES data to the mid-year population in 2000. Past and current drinking rates were combined to determine the ever-drink-

ing rate. Data from the 2001 KNHANES were excluded due to differences in survey format and past drinking rate estimates compared to other years. The average daily alcohol intake (g/day) among current drinkers was calculated based on the average consumption frequency per month and the average alcohol intake per occasion. The survey included questions about drinking 4 or more times per week starting in 2007. To assess the median frequency of alcohol consumption (4 times/wk), data from 1998, 2001, and 2005 were evaluated. The 2001 data were excluded because they were entirely categorical. The median value for female in 2005 was higher than in other years, which contradicted previous reports. Therefore, the 1998 median value was used as an alternative.

### Estimation of relative risks for cancers

Cancers classified as Group 1 by the IARC in relation to alcohol and acetaldehyde, as well as those with strong evidence (convincing/probable grade) for alcohol consumption by the World Cancer Research Fund, were selected as alcohol-related cancers. These included cancers of the MPL (International Classification of Diseases, 10th revision [ICD-10] codes C00-C14, C32), esophagus (ICD-10 code C15), stomach (ICD-10 code C16), liver (ICD-10 code C22), colorectum (ICD-10 codes C18-C20), and breast (ICD-10 code C50). Due to the low frequency of MPL cancers, their risks were combined into the MPL category (ICD-10 codes C00-C14, C32) for analysis. For esophageal and liver cancers, risks were calculated for all esophageal cancers (ICD-10 code C15) and all liver cancers (ICD-10 code C22), as cohort studies on squamous cell carcinoma of the esophagus and hepatocellular carcinoma of the liver were rare.

Relative risks (RRs) were estimated using cohort studies from the Korean Cohort Consortium [15]. After analyzing individual cohort studies, a meta-analysis was conducted to calculate cancer risk in Koreans. Cancer risk was adjusted for age (continuous), sex, smoking status, body mass index (continuous), and regular exercise or physical activity status using a multivariable Cox model. For sensitivity analysis, a meta-analysis was performed using a systematic literature review of Asian and global cohort studies on the association between alcohol consumption (past and current vs. never drinking; drinking 10 g/day) and cancer risk.

### Calculation of population-attributable fraction (PAF) and prediction of PAF values in 2025 and 2030

Cancer incidence and mortality rates, as well as the number of cancer cases and deaths, were based on cancer registration statistics from the National Cancer Center and death data from Statistics Korea, focusing on adults aged 20 and older [16,17].

The PAF for specific cancers related to alcohol consumption, compared to non-alcohol consumption, was calculated using Levin's formula. The 95% confidence intervals for PAF were estimated using Monte Carlo methods [18-21]. For sensitivity analysis, equation (1) was used to calculate the attributable fraction of cancer for g/day of alcohol consumption, and equation (2) was used to calculate the attributable fraction of cancer by category

(light, moderate, and heavy alcohol consumption). The attributable fraction for all cancers was calculated directly without determining number of attributable cancer cases or deaths (ACs) for specific cancers.

$$PAF = \frac{P_e(e^{\beta \cdot dose} - 1)}{P_e(e^{\beta \cdot dose} - 1) + 1} \quad (1)$$

$$PAF = \frac{\sum_{i=1}^n P_{e,i}(RR_i - 1)}{\sum_{i=1}^n P_{e,i}(RR_i - 1) + 1} \quad (2)$$

The PAF of cancer was calculated by (1) determining the AC of specific cancer incidences or deaths attributable to alcohol consumption and summing these ACs to calculate the total contribution of alcohol consumption to cancer incidence or death, and (2) dividing this sum by the total number of cancer incidences or deaths to derive the PAF of cancer due to alcohol consumption [22].

For sensitivity analysis, the amount of alcohol consumed by current drinkers was categorized [15,23-27] (Supplementary Material 2). Four methods were used: First, a systematic literature review of Asian and global cohort studies was conducted to assess cancer risk associated with every 10 g increase in alcohol consumption, followed by meta-analysis. Then, a meta-analysis was performed for current drinkers, calculating cancer risk per average consumption in 3 or 4 categories based on alcohol intake, and determining the PAF of cancer, including the risk for past drinkers. Third, cancer risk was calculated for non-drinkers, past drinkers, and current drinkers, regardless of alcohol volume, and the PAF of cancer was derived. Fourth, the risks of individual cancers and overall cancer due to past and current drinking were analyzed using original data from Korean cohort studies.

Predictions for cancer incidence and mortality in 2025 and 2030 were based on annual age-specific and sex-specific cancer incidence and mortality rates [16,17], using the average annual percent change from a joinpoint regression model [28,29]. Detailed methods and results for predicting cancer incidence and mortality by 2030 have been described in a previous publication [30].

### Ethics statement

This study was approved by the Institutional Review Board of Seoul National University Hospital (IRB No. C-1911-188-1084).

## RESULTS

In 2000, the following drinking rates were observed: 84.7% of male and 56.4% of female were current drinkers, while 6.0% of male and 3.6% of female were past drinkers. Lifetime non-drinking rates were 9.4% for male and 40.0% for female. The median alcohol intake differed significantly between sexes, with male consuming 18.0 g/day and female consuming 3.4 g/day. Among male, the current drinking rate increased from 2000 to 2010, followed by a slight decline after 2010. For female, the drinking rate rose steadily from 2000 to 2010, with a sharp increase in current drinking from 2000 to 2005, followed by fluctuations of slight decreases and increases (Supplementary Material 3).

In male, significant associations were found between ever drinking and the incidence of cancers of the MPL, esophagus, stomach, and liver, as well as deaths from esophageal, stomach, and liver cancers. In female, significant associations were only observed between ever drinking and deaths from MPL, stomach, and liver cancers. When examining the association between past and current alcohol consumption and cancer risk, both past and current drinking were linked to increased incidence and mortality from individual cancer types in both sexes. In male, significant associations were observed between past drinking and the incidence of esophageal, stomach, and liver cancers, as well as deaths from liver cancer. In female, significant associations were only seen between past drinking and the incidence and mortality from liver cancer (Supplementary Materials 4-6). The risk of cancer incidence and mortality associated with alcohol consumption increased with every 10 g increment of pure alcohol. This trend was consistent in both Asian and global cohort studies (Supplementary Material 7). Additionally, analyses based on the European Medicines Agency (EMA) [23], WHO [24], and Korean Alcohol Practice Guidelines [27] also demonstrated an increased risk of cancer incidence and mortality (Supplementary Materials 8 and 9).

Alcohol consumption accounted for 3.40% and 3.86% of incident cancer cases among Korean adults aged 20 or older in 2015 and 2020, respectively, and contributed to a slightly lower percentage of cancer deaths (3.34 and 3.43%, respectively). The PAF for cancer incidence due to drinking was higher in male (2015: 4.58%; 2020: 4.55%) than in female (2015: 2.08%; 2020: 3.09%). Similarly, the PAF for alcohol-related cancer deaths was slightly higher than that for cancer incidence (male in 2015 and 2020: 4.00 and 3.82%, respectively; female in 2015 and 2020: 2.25 and 2.80%, respectively) (Table 1, Supplementary Materials 10-13).

In 2015, among 7,323 cancer cases attributed to alcohol consumption, 1,276 were stomach cancer cases (1,233 male and 43 female). Of these, 6.26% of stomach cancer cases in male and 0.45% in female were linked to alcohol consumption. Among the 2,556 cancer deaths attributed to alcohol consumption in the same year, 756 were due to liver cancer (632 male and 124 female). In these cases, 7.55% of liver cancer deaths in male and 4.22% in female were attributed to alcohol consumption (Table 1).

When comparing the PAF of cancer using the RR for every 10 g/day increase in alcohol consumption in Asian and global populations, the PAF values were generally higher in the global

**Table 1.** PAF<sup>1</sup> and AC by alcohol consumption in 2015 and 2020, Korea

Variables (ICD-10 code)	Cancer incidence				Cancer mortality			
	2015		2020		2015		2020	
	PAF (%)	AC (n)	PAF (%)	AC (n)	PAF (%)	AC (n)	PAF (%)	AC (n)
<b>Total</b>								
MPL (C00-C14, C32)	12.85	574	13.66	716	9.21	139	10.04	161
Esophagus (C15)	46.77	1,143	47.74	1,312	44.42	680	44.88	702
Stomach (C16)	4.34	1,276	4.58	1,221	5.29	451	6.54	491
Colorectal (C18-C20)	6.46	1,751	6.92	2,444	4.25	353	4.61	409
Liver (C22)	5.63	895	6.04	914	6.69	756	7.32	773
Breast (C50, C67)	8.75	1,684	2.05	2,901	7.55	177	10.15	276
All cancers	3.40	7,323	3.86	9,508	3.34	2,556	3.43	2,812
<b>Male</b>								
MPL (C00-C14, C32)	14.70	513	15.28	614	9.69	116	10.10	129
Esophagus (C15)	49.68	1,105	50.81	1,245	47.89	671	49.02	688
Stomach (C16)	6.26	1,233	6.53	1,167	3.12	172	3.26	157
Colorectal (C18-C20)	9.67	1,557	10.07	2,091	6.60	310	6.88	346
Liver (C22)	6.77	800	7.06	786	7.55	632	7.87	615
All cancers	4.58	5,208	4.55	5,903	4.00	1,901	3.82	1,935
<b>Female</b>								
MPL (C00-C14, C32)	6.18	61	8.35	102	7.44	23	10.00	32
Esophagus (C15)	17.54	38	22.72	67	6.60	9	8.90	14
Stomach (C16)	0.45	43	0.62	54	9.24	279	12.34	334
Colorectal (C18-C20)	1.77	194	2.42	353	1.18	43	1.63	63
Liver (C22)	2.34	95	3.21	128	4.22	124	5.75	158
Breast (C50, C67)	8.75	1,684	11.70	2,901	7.55	177	10.15	276
All cancers	2.08	2,115	3.09	3,605	2.25	655	2.80	877

PAF, population-attributable fraction; AC, attributable cancer cases or deaths; ICD-10, International Classification of Diseases, 10th revision; MPL, mouth, pharynx, and larynx.

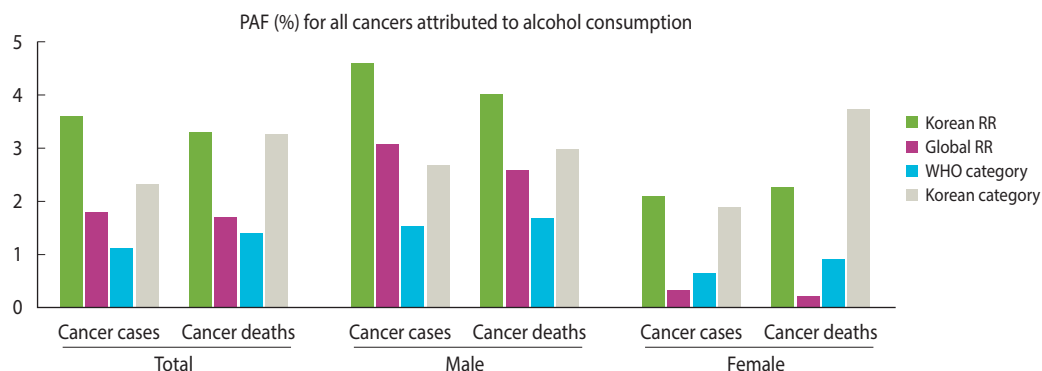
<sup>1</sup>Alcohol drinking was classified as ever drinking and never drinking (reference).



population than in the Asian population. The only exception was cancer mortality among female, where the trend was reversed. When classified according to the WHO, EMA, and Korean guidelines, the PAF values from the WHO and EMA were similar. How-

ever, the values based on the Korean guidelines showed the highest PAF (Figure 1 and Table 2, Supplementary Material 14).

Compared to 2009, both the number of cancer incidences and deaths attributable to alcohol consumption and the PAF of cancer



**Figure 1.** Comparison of cancer population-attributable fraction (PAF) attributed to alcohol consumption when using different relative risks (RRs). WHO, World Health Organization.

**Table 2.** Comparison of population-attributable fraction (%) using different relative risks for cancer attributable to alcohol consumption

Variables	Cancer incidence							Cancer mortality						
	Asia 10 g/day	Global 10 g/day	WHO <sup>1</sup>	Guideline EMA <sup>2</sup>	Korean <sup>3</sup>	Ever vs. Never	Past and current vs. Never	Asia 10 g/day	Global 10 g/day	WHO <sup>1</sup>	Guideline EMA <sup>2</sup>	Korean <sup>3</sup>	Ever vs. Never	Past and current vs. Never
<b>Total</b>														
MPL	8.80	11.64	3.18	3.18	5.11	12.85	7.59	5.10	11.79	3.91	3.91	7.35	9.21	6.49
Esophagus	16.90	11.13	9.45	9.37	19.48	46.77	38.42	10.12	11.10	1.18	1.11	9.54	44.42	39.91
Stomach	1.65	1.88	1.47	1.47	3.25	4.34	6.14	1.61	1.82	1.62	1.61	3.16	5.29	5.24
Colorectal	3.32	5.95	0.96	0.96	2.36	6.46	7.11	3.71	5.69	1.17	1.17	5.86	4.25	9.60
Liver	4.04	4.10	5.60	5.59	8.47	5.63	8.32	2.65	2.72	6.20	6.19	8.90	6.69	11.71
Breast	2.12	1.24	2.13	2.12	6.81	8.75	12.46	2.14	0.51	2.18	2.14	19.80	7.55	26.73
All cancers	1.50	1.79	1.10	1.09	2.30	3.40	4.05	1.34	1.69	1.39	1.38	3.24	3.34	5.09
<b>Male</b>														
MPL	11.22	14.05	3.47	3.46	5.74	14.70	6.28	6.22	14.05	4.25	4.23	7.84	9.69	6.99
Esophagus	18.27	11.85	9.78	9.73	19.21	49.68	39.29	10.83	11.85	1.18	1.14	8.97	47.89	40.67
Stomach	2.33	2.73	2.19	2.18	4.10	6.26	8.94	2.33	2.73	1.88	1.87	3.50	3.12	7.28
Colorectal	5.49	9.85	1.57	1.56	3.44	9.67	8.56	5.49	9.85	1.41	1.40	4.33	6.60	12.60
Liver	5.21	5.21	5.87	5.85	8.86	6.77	9.31	3.35	3.35	6.72	6.70	9.40	7.55	13.93
All cancers	2.42	3.07	1.51	1.50	2.67	4.58	4.69	1.88	2.58	1.68	1.68	2.95	4.00	5.92
<b>Female</b>														
MPL	0.18	3.05	2.10	2.10	2.91	6.18	12.21	0.65	3.05	2.58	2.57	5.63	7.44	4.50
Esophagus	3.19	3.45	6.10	6.05	22.24	17.54	29.73	2.62	3.45	0.71	0.67	15.02	6.60	31.26
Stomach	0.28	0.17	0.03	0.02	1.53	0.45	0.44	0.29	0.17	1.13	1.13	2.51	9.24	1.54
Colorectal	0.15	0.25	0.06	0.06	0.78	1.77	4.98	1.38	0.25	0.87	0.85	7.85	1.18	5.69
Liver	0.65	0.88	4.81	4.80	7.37	2.34	5.43	0.65	0.88	4.71	4.70	7.45	4.22	5.34
Breast	2.12	1.24	2.13	2.12	6.81	8.75	12.46	2.12	0.51	2.19	2.14	19.82	7.55	26.73
All cancers	0.48	0.35	0.64	0.64	1.89	2.08	3.34	0.46	0.22	0.90	0.90	3.70	2.25	3.74

MPL, mouth, pharynx, and larynx; WHO, World Health Organization; EMA, European Medicine Agency.

<sup>1</sup>The category was classified using the WHO guideline for alcohol drinking (low, intermediate, and high alcohol drinking)+past drinking.

<sup>2</sup>The category was classified using the EMA guideline for the risk of alcohol drinking (low risk, intermediate risk, high risk, and very high risk)+past drinking.

<sup>3</sup>The category was classified using the Korean guideline for alcohol drinking (light, moderate, and heavy alcohol drinking)+past drinking.

incidence and mortality increased in 2015 (incidence: 1.77% in 2009 and 3.40% in 2015; mortality: 1.82% in 2009 and 3.34% in 2015). Alcohol consumption was responsible for 3.40% of cancer incidences and 3.34% of cancer-related deaths in the Korean population in 2015. It accounted for 4.58% of cancer incidences in male and 2.47% in female, while contributing to 4.00% of cancer-related deaths in male and 2.25% in female. The proportion of alcohol consumption contributing to cancer incidence was higher in 2015 than in 2009. Compared to 2009, the PAFs for esophageal, liver, and breast cancers increased in both incidence and mortality (Supplementary Material 15).

The PAF of cancer due to alcohol consumption is projected to decline continuously in male from 2015 to 2030 (4.58% in 2015 and 3.80% in 2030). Similarly, the PAF for cancer-related deaths in male is expected to decrease steadily (4.00% in 2015 and 3.52% in 2030). In contrast, the PAF for both cancer incidence and mortality due to alcohol consumption in female is predicted to rise continuously from 2015 to 2030 (incidence PAF: 2.08% in 2015 and 3.54% in 2030; mortality PAF: 2.25% in 2015 and 2.69% in 2030) (Table 3 and Figures 2 and 3).

DISCUSSION

In 2015, alcohol consumption contributed to 3.4% of cancer incidence among Koreans (4.6% for male and 2.1% for female)

and accounted for 3.3% of cancer deaths (4.0% for male and 2.3% for female). Notably, alcohol consumption was responsible for approximately half of esophageal cancer cases (49.7%) and deaths (47.9%) in male, underscoring its significant role in esophageal cancer among this group.

The contribution of alcohol consumption to cancer in Korea in 2015 was moderate compared to reports from Western countries during the same period (Canada: 1.8%, United Kingdom: 3.3%, and France: 8.0%) [31-38]. It was lower than the 5.6% contribution to cancer mortality attributed to alcohol consumption reported in the United States in 2014 [39]. In China, alcohol consumption accounted for 3.1% of cancer-related deaths in 2013 [40], a decrease from 4.4% in 2005 [41]. Similarly, in the United Kingdom, the contribution of alcohol consumption to cancer incidence declined from 4.0% in 2010 to 3.3% in 2015 (Figure 4) [42,43].

In France in 2015, alcohol consumption was categorized according to WHO guidelines: for male, < 40 g/day, 40 g/day to < 60 g/day, and ≥ 60 g/day were defined as light, moderate, and heavy drinking, respectively, while for female, < 20 g/day, 20 g/day to < 40 g/day, and ≥ 40 g/day were used [34]. In Canada, slightly different definitions were applied: light, moderate, and heavy drinking were defined as 0 g/day < to ≤ 12.5 g/day, 12.5 g/day < to ≤ 50 g/day, and > 50.0 g/day, respectively [44].

A key strength of our study is its comprehensive sensitivity analyses, which explored various potential scenarios. Most previous

Table 3. Changes in the PAF and AC caused by alcohol consumption in Korea

Variables		Cancer incidence			Cancer mortality		
		2015	2030	Changed % <sup>1</sup>	2015	2030	Changed % <sup>1</sup>
Total population							
MPL	PAF (%)	12.85	13.77	7.2	9.21	10.34	12.3
	AC (n)	574	916	59.6	139	181	30.2
	n	4,466	6,652	48.9	1,510	1,750	15.9
Esophagus	PAF (%)	46.77	47.95	2.5	44.42	45.58	2.6
	AC (n)	1,143	1,464	28.1	680	671	-1.3
	n	2,444	3,053	24.9	1,531	1,472	-3.9
Stomach	PAF (%)	4.34	4.68	7.8	5.29	6.73	27.2
	AC (n)	1,276	1,325	3.8	451	398	-11.8
	n	29,386	28,329	-3.6	8,525	5,912	-30.7
Colorectal	PAF (%)	6.46	7.21	11.6	4.25	4.79	12.7
	AC (n)	1,751	3,126	78.5	353	590	67.1
	n	27,120	43,375	59.9	8,298	12,323	48.5
Liver	PAF (%)	5.63	6.11	8.5	6.69	7.45	11.4
	AC (n)	895	910	1.7	756	762	0.8
	n	15,896	14,897	-6.3	11,307	10,223	-9.6
Breast	PAF (%)	8.75	12.38	41.5	7.55	10.76	42.5
	AC (n)	1,684	5,433	222.6	177	394	122.6
	n	19,258	43,890	127.9	2,338	3,663	56.7
Alcohol related cancers <sup>2</sup>	PAF (%)	3.40	3.67	7.9	3.34	3.20	-4.2
	AC (n)	7,323	13,174	79.9	2,556	2,996	17.2
All cancer (n)		215,570	358,627	66.4	76,621	93,690	22.3

(Continued to the next page)

**Table 3.** Continued

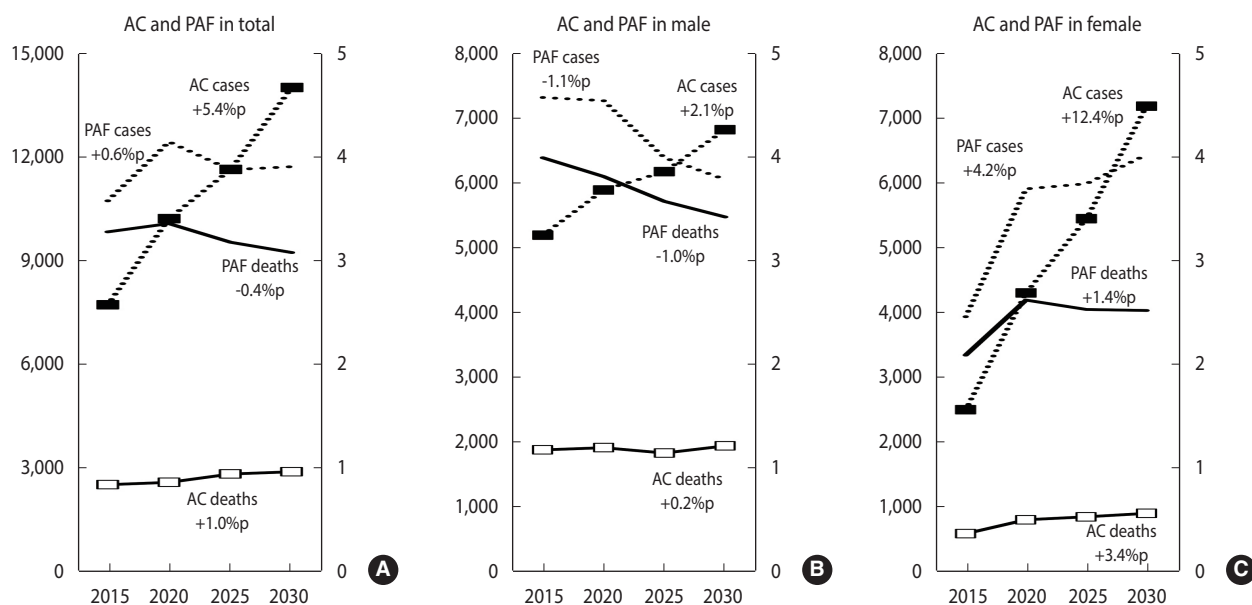
Variables		Cancer incidence			Cancer mortality		
		2015	2030	Changed % <sup>1</sup>	2015	2030	Changed % <sup>1</sup>
Male							
MPL	PAF (%)	14.7	15.48	5.3	9.69	10.24	5.7
	AC (n)	513	764	48.9	116	140	20.7
	n	3,487	4,938	41.6	1,200	1,367	13.9
Esophagus	PAF (%)	49.68	51.21	3.1	47.89	49.41	3.2
	AC (n)	1,105	1,377	24.6	671	658	-1.9
	n	2,225	2,689	20.9	1,401	1,331	-5.0
Stomach	PAF (%)	6.26	6.63	5.9	3.12	3.31	6.1
	AC (n)	1,233	1,264	2.5	172	127	-26.2
	n	19,689	19,065	-3.2	5,506	3,832	-30.4
Colorectal	PAF (%)	9.67	10.21	5.6	6.60	6.98	5.8
	AC (n)	1,557	2,685	72.4	310	500	61.3
	n	16,103	26,287	63.2	4,698	7,163	52.5
Liver	PAF (%)	6.77	7.16	5.8	7.55	7.98	5.7
	AC (n)	800	768	-4.0	632	587	-7.1
	n	11,831	10,723	-9.4	8,380	7,354	-12.2
Alcohol related cancers <sup>2</sup>	PAF (%)	4.58	3.80	-17.0	4.00	3.52	-12.0
	AC (n)	5,208	6,858	31.7	1,901	2,012	5.8
All cancer (n)		113,739	180,250	58.5	47,544	57,141	20.2
Female							
MPL	PAF (%)	6.18	8.85	43.2	7.44	10.60	42.5
	AC (n)	61	152	149.2	23	41	78.3
	n	979	1,714	75.1	310	383	23.5
Esophagus	PAF (%)	17.54	23.87	36.1	6.60	9.44	43.0
	AC (n)	38	87	128.9	9	13	44.4
	n	219	364	66.2	130	141	8.5
Stomach	PAF (%)	0.45	0.66	46.7	9.24	13.05	41.2
	AC (n)	43	61	41.9	279	271	-2.9
	n	9,697	9,264	-4.5	3,019	2,080	-31.1
Colorectal	PAF (%)	1.77	2.58	45.8	1.18	1.74	47.5
	AC (n)	194	441	127.3	43	90	109.3
	n	11,017	17,088	55.1	3,600	5,160	43.3
Liver	PAF (%)	2.34	3.41	45.7	4.22	6.10	44.5
	AC (n)	95	142	49.5	124	175	41.1
	n	4,065	4,174	2.7	2,927	2,869	-2.0
Breast	PAF (%)	8.75	12.38	41.5	7.55	10.75	42.4
	AC (n)	1,684	5,433	222.6	177	394	122.6
	n	19,258	43,890	127.9	2,338	3,663	56.7
Alcohol related cancers <sup>2</sup>	PAF (%)	2.08	3.54	70.2	2.25	2.69	19.6
	AC (n)	2,115	6,316	198.6	655	984	50.2
All cancer (n)		101,831	178,377	75.2	29,077	36,549	25.7

MPL, mouth, pharynx, and larynx; PAF, population-attributable fraction; AC, attributable cancer cases or deaths; IARC, International Agency for Research on Cancer.

<sup>1</sup>Cancers categorized as Group 1 in relation to alcohol and acetaldehyde by the IARC and cancers categorized as having strong evidence at the convincing/probable grade for alcohol consumption by the World Cancer Research Fund were selected as alcohol-related cancer in this study.

<sup>2</sup>Cancers categorized as Group 1 in relation to alcohol and acetaldehyde by the IARC and cancers categorized as having strong evidence at the convincing/probable grade for alcohol consumption by the World Cancer Research Fund were selected as alcohol-related cancer in this study.





**Figure 2.** Changing trends of population-attributable fraction (PAF) and attributable cancer cases and deaths (AC) in cancer attributed to alcohol consumption in Korea, 2015 to 2030 (A) total, (B) male, and (C) female. %p, percentage points.

studies applied latency periods of 10 years or 15 years, and our adoption of a 15-year latency period supports the validity of our approach. Additionally, our projection analysis extends to 2025 and 2030, providing valuable insights into future trends in alcohol-related PAFs. Notably, while the contribution of alcohol consumption to cancer incidence is projected to decrease among male by 2030 compared to 2015, it is expected to rise significantly among female, highlighting the need for sex-specific policy interventions.

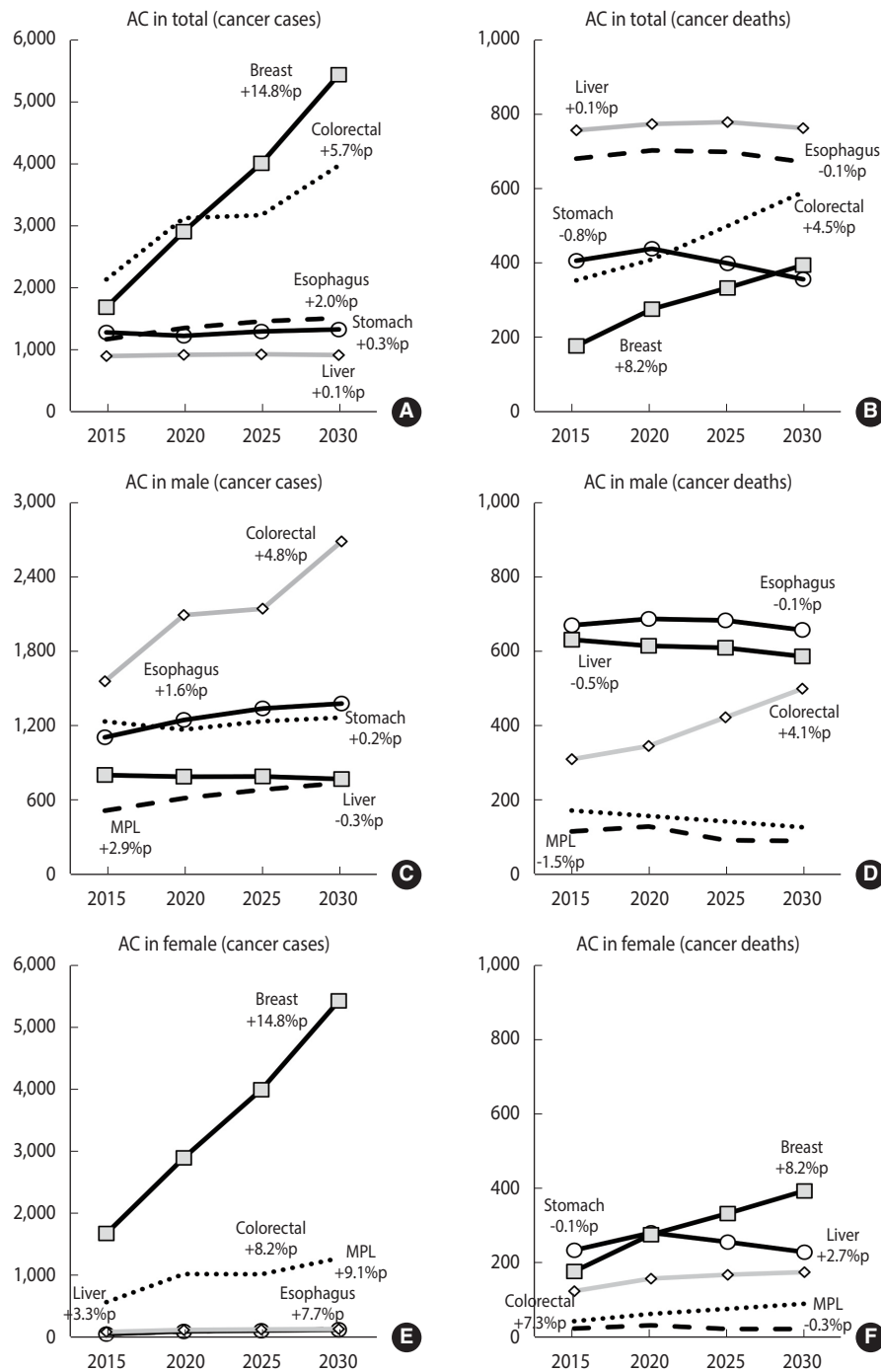
In Korea, alcohol consumption rates increased steadily for both male and female from 2000 to 2020 (Supplementary Material 3). Despite declining incidence and mortality rates for prevalent cancers, including gastric, liver, esophageal, and colorectal cancers, between 2009 and 2015, the total number of cases has risen [45,46]. This discrepancy may stem from the fact that cancer risk in 2009 was calculated using data from a limited number of cohorts or case-control studies, with insufficient data to calculate risks for female, leading to a focus on male. In contrast, our study leveraged multiple Korean cohorts to calculate cancer risks for female, significantly reducing the risk of underestimation. Furthermore, unlike the 2009 Korean study, which used a 20-year latency period and excluded stomach cancer, our study employed distinct methodological approaches. While the 2009 study primarily used alcohol consumption as a continuous variable, our main analysis utilized a binary variable, supplemented by sensitivity analyses. As a result, direct comparisons between the PAF values reported in the 2009 study and our findings from 2015 and 2020, which reflect an increased alcohol-related cancer burden, are challenging. When comparing our results with prior Korean or international studies, it is crucial to consider differences in included cancer types, exposure variables, and methodologies [13,14].

Our study has several limitations. Unlike many international

studies that derive RRs from large-scale, population-representative cohort studies, our study utilized meta-analyses of multiple representative cohorts, which may introduce overlap among study populations. Additionally, data for female were insufficient in some cases, necessitating the use of male-derived RRs, and for cancers with limited mortality data, RR values for incidence were substituted. These adjustments may have slightly reduced the accuracy of our results.

Although the contribution of alcohol to cancer in Korea is lower than in many Western countries, alcohol consumption rates remain high and are rising rapidly among female. Moreover, the monthly heavy drinking rate—defined as consuming 7 or more glasses for male and 5 or more glasses for female on a single occasion at least once a month in the past year—has slightly decreased for male (from 55.3% in 2005 to 50.8% in 2018) but remains above 50%. For female, this rate increased by 9%, from 17.2% in 2005 to 26.9% in 2018 [47]. While the contribution of alcohol to specific cancers is projected to continue rising, the overall PAF for all alcohol-related cancers is expected to decrease in both incidence and mortality across the total population. However, the difference in colorectal cancer incidence PAF is highest among female, excluding breast cancer, even though the number of colorectal cancer cases increased by only 55.1%. Furthermore, the incidence and mortality of breast cancer, which are expected to rise steadily, will significantly impact the contribution of alcohol to cancer after 2030.

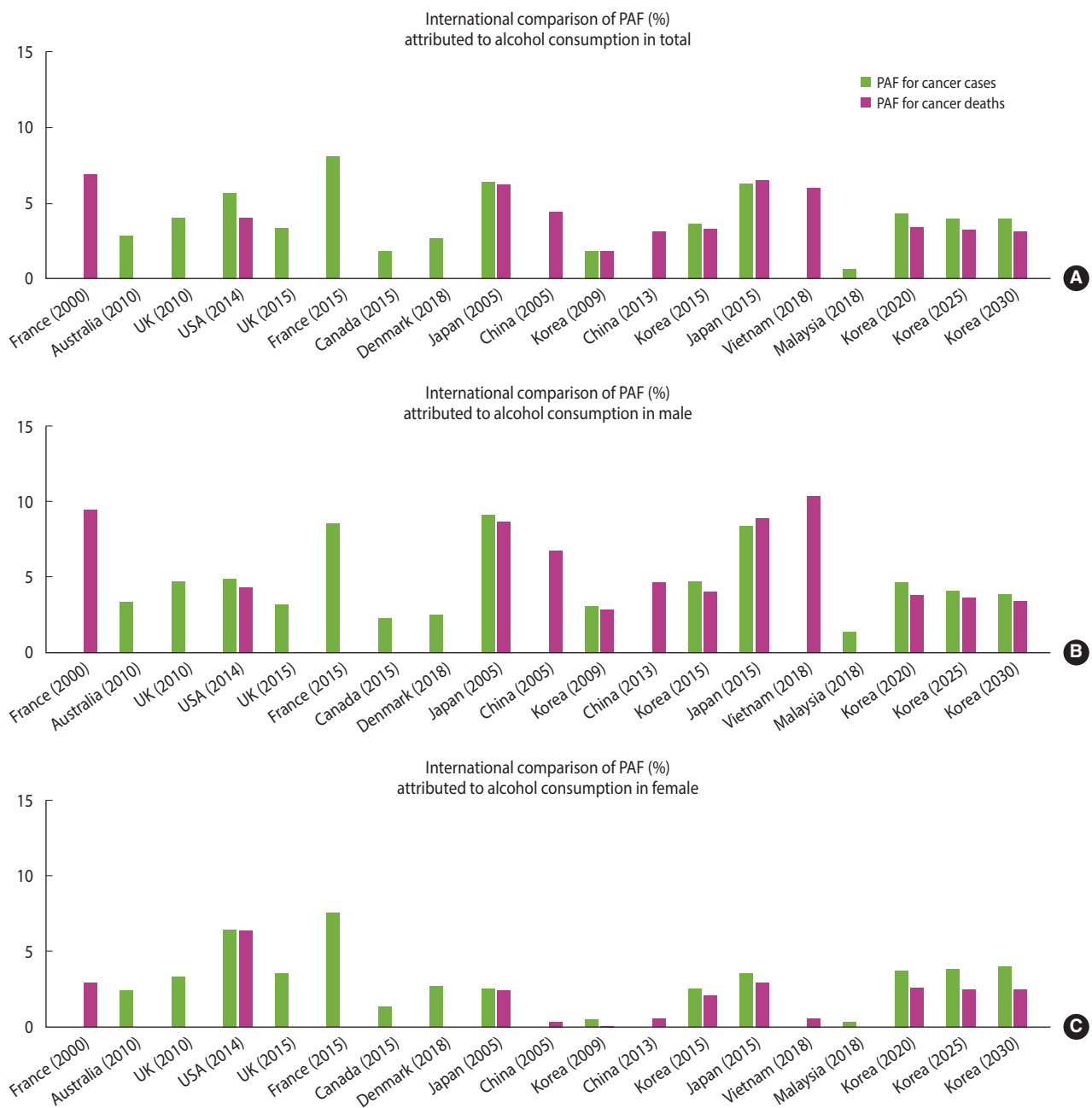
The IARC has stated that there is no minimum threshold for breast cancer risk associated with alcohol consumption, as even small amounts of alcohol increase the risk compared to non-drinkers [2]. Alcohol can elevate estrogen levels, thereby increasing the risk of breast cancer [48]. The liver, which is responsible



**Figure 3.** Changing trends of attributable cancer cases and deaths (AC) in specific cancer attributed to alcohol consumption in Korea, 2015 to 2030. Attributable cancer cases in (A) total, (C) male, and (E) female. Attributable cancer deaths in ((B) total, (D) male, and (F) female. MPL, mouth, pharynx, and larynx; %p, percentage points.

for deactivating estrogen, may fail to regulate estrogen levels effectively when its function is impaired by alcohol. Additionally, alcohol can reduce levels of adiponectin, a hormone involved in weight management and insulin sensitivity. Lower adiponectin levels can lead to insulin resistance, which further contributes to

elevated estrogen levels [49]. Estrogen promotes the growth of breast cells, and excessive estrogen can overstimulate cell division, potentially leading to breast cancer [50]. Among alcohol-related cancers in female, breast cancer accounts for 66.9% of cases and 29.0% of deaths. Therefore, it is critical to consider the relation-



**Figure 4.** International comparison of population-attributable fraction (PAF) attributed to alcohol consumption (A) total, (B) male, and (C) female.

ship between alcohol consumption, increased estrogen levels, and the incidence and mortality of breast cancer.

For postmenopausal breast cancer in Korea, some cohorts reported an  $RR < 1$  for alcohol-related cancer risk. These cohorts were excluded from the study due to difficulties in estimating the quantity and frequency of alcohol consumption per week. To accurately measure alcohol consumption, detailed information on frequency and dose is necessary, whether in existing or new cohorts. Re-estimating cancer risk based on the amount of alcohol

consumed is essential for future research.

This study included all cohort studies conducted by the Korean Cohort Consortium. By analyzing raw cohort data from national research institutes, we calculated cancer risks and conducted a meta-analysis to determine alcohol-related cancer risks in Korea. Quantitatively, we estimated the impact of alcohol consumption on cancer incidence and mortality among adults aged 20 and older. While the study confirmed results for less common cancers through a systematic literature review, statistical power limitations

led to a focus on more frequent cancers. By conducting a retrospective cohort study using the Korea National Health Insurance Service – National Health Information Database database, which includes data from 10 million individuals, we were able to analyze raw data and estimate cancer risks for even rare cancers using Korean data sources. Furthermore, the reliability of the results was increased through sensitivity analyses based on multiple scenarios.

However, this study did not address other major diseases caused by alcohol, the scale of alcohol-related accidents, or the effects of prenatal alcohol exposure on fetuses and newborns. Consequently, not all outcomes related to alcohol consumption were considered. For cancers with insufficient meta-analysis data in female, the PAF was calculated using RR values derived from male. This approach may lead to overestimation in some cases, as RR values for female are generally lower than those for male.

Despite projections of increasing alcohol-related cancer incidence and mortality, the alcohol-related PAF does not show a corresponding rise. This can be attributed to broader population dynamics and the influence of other risk factors. As the total cancer burden increases due to factors such as an aging population or the growing impact of other risk factors like smoking and obesity, the proportional contribution of alcohol consumption to the overall cancer burden may not increase as significantly. The PAF measures the attributable fraction relative to the total cancer burden, meaning that while the absolute number of alcohol-related cancers is expected to rise, the overall cancer burden may dilute the relative impact of alcohol, stabilizing or even reducing the PAF. This highlights the need for nuanced public health strategies that address not only the growing burden of alcohol-related cancers but also the broader context of changing risk profiles within the population.

In 2015, alcohol consumption accounted for 4.6% of cancer cases and 4.0% of cancer deaths in male, and 2.5% of cancer cases and 2.1% of cancer deaths in female. While the proportion of cancers caused by alcohol consumption is projected to decline in male, it is expected to increase in female until 2030. Alcohol consumption is a preventable risk factor that requires targeted national prevention policies, particularly for female.

## NOTES

### Supplementary materials

Supplementary materials are available at <https://doi.org/10.4178/epih.e2025009>.

### Conflict of interest

The authors have no conflicts of interest to declare for this study.

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The prevalence rates of risk factors were analyzed using data provided by the Korea National Institute of Health (KNIH), KDCA, and the Occupational Safety and Health Research Institute (OSHRI), Korea Occupational Safety and Health Agency (KOSHA), and the Korean Statistical Information Service (KOSIS).

The incidence and mortality rates of cancers were analyzed using data provided by the Cancer Registration Statistics, National Cancer Center of Korea (KNCC), and the Korean Statistical Information Service (KOSIS).

## Author contributions

Conceptualization: Sung S, Ko KP, Lee JE, Kim I, Park SK. Data curation: Sung S, Shin A, Jee SH, Kweon SS, Shin MH, Park SM, Ryu S, Yang SY, Choi SH, Kim J, Yi SW, Ko KP, Park SK. Formal analysis: Sung S, An J, Jung J, Lee HS. Funding acquisition: Park SK. Methodology: Sung S, Ko KP, Park SK. Project administration: Sung S, Park SK. Visualization: Sung S. Writing – original draft: Sung S. Writing – review & editing: Sung S, An J, Jung J, Lee HS, Moon S, Kim I, Lee JE, Shin A, Jee SH, Kweon SS, Shin MH, Park SM, Ryu S, Yang SY, Choi SH, Kim J, Yi SW, Choi YJ, Hong Y, Lee S, Lim W, Kim K, Park SH, Im JS, Seo HG, Ko KP, Park SK.

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