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RESEARCH ARTICLE

Alcohol Consumption and Cigarette Smoking as Major, Modifiable Determinants of Site-Specific Cancer Burden in Adults Aged ≥50 Years: A Narrative Review

DR.R.GOVINDARAJAN [1]*, DR.M.BAGAVAD GEETHA [2] ,DR.G.JESINTHA [3]
[1]Professor & Head, Department of Pathology, Shri Sathya Sai Medical College & Research Institute, (Sri Balaji Vidyapeeth University (DU), Puducherry), Nellikuppam Post, Chengalpattu District, Tamil Nadu ,India
[2]Professor, Department of Physiology, Shri Sathya Sai Medical College & Research Institute, (Sri Balaji Vidyapeeth University (DU), Puducherry), Nellikuppam Post, Chengalpattu District, Tamil Nadu ,India
[3]Registered Medical Practitioner, Alandur, Chennai, Tamil Nadu, India

*Corresponding Author
DR.R.GOVINDARAJAN

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Abstract: ***Background:*** Alcohol and tobacco are established carcinogens whose cumulative exposure becomes clinically significant after 50 years of age. ***Objective:*** To summarize the most reliable evidence on site-specific cancer risks from alcohol consumption and cigarette smoking among adults ≥50 years, emphasizing dose–response and prevention potential. ***Methods:*** Narrative synthesis of authoritative evaluations and meta-analyses (IARC, WCRF, NCI, CDC, and major cohort studies). ***Results:*** Alcohol is causally linked to cancers of the oral cavity, pharynx, larynx, oesophagus (squamous cell carcinoma), liver, colorectum, and female breast. Dose–response analyses show ~7% higher risk per 10 g ethanol/day for breast and colorectal cancer. Smoking remains the dominant preventable carcinogen, responsible for large excesses in lifetime cancer risk and global cancer burden. ***Conclusions:*** After midlife, reducing alcohol and eliminating smoking provide meaningful absolute reductions in cancer risk.

Keywords: Alcohol, tobacco, carcinogenesis, prevention

INTRODUCTION

Alcoholic beverages are recognized as Group-1 human carcinogens with strong evidence for cancers of the oral cavity, pharynx, larynx, oesophagus (squamous cell), liver, colorectum, and female breast [1–3]. Quantitatively, pooled analyses reveal a steady rise in cancer risk with increasing intake, about 7 % per 10 g ethanol/day for breast and colorectal cancer [4,5]. Cigarette smoking remains the leading preventable cause of cancer worldwide, implicated in at least sixteen malignancies, including lung, head-and-neck, oesophagus, bladder, kidney, pancreas, liver, cervix, colon–rectum, and acute myeloid leukemia [6]. By late adulthood, cumulative exposure yields large absolute differences: in cohort data, the lifetime risk of any cancer to age 80 is substantially higher in current smokers than in never-smokers [7]. This review consolidates such site-specific magnitudes and dose–response patterns to inform prevention for adults aged ≥ 50 years.

MATERIAL AND METHODS

Study Design and Scope

This narrative review synthesized current high-level evidence examining the relationship between alcohol consumption, cigarette smoking, and site-specific cancer risk among adults aged 50 years and above. The study was conducted from April 2024 to March 2025. The objective was to identify consistent dose–response patterns, typical relative risk magnitudes, and potential windows for risk reduction following cessation. The review focused on common solid tumors causally linked to these exposures, including cancers of the oral cavity, pharynx, larynx, oesophagus (squamous cell carcinoma), liver (hepatocellular carcinoma), colorectum, breast, and lung.

Search Strategy and Data Sources

Published literature was searched across PubMed, Scopus, Web of Science, and Google Scholar using combinations of the terms *alcohol*, *smoking*, *cancer risk*, *dose-response*, *relative risk*, *meta-analysis*, *hepatocellular carcinoma*, *oesophageal carcinoma*, and *breast cancer*. Only studies published in English between 2010 and 2025 were considered. Authoritative epidemiological assessments from recognized bodies such as the International Agency for Research on Cancer (IARC), World Cancer Research Fund (WCRF), National Cancer Institute (NCI), Centers for Disease Control and Prevention (CDC), and the Global Burden of Disease (GBD) project were prioritized to ensure reliability and comparability.

Grey literature and non-peer-reviewed materials were excluded. When multiple meta-analyses existed for the same cancer site, the most recent or methodologically robust estimate was selected.

Eligibility Criteria

Studies were eligible if they:

1. Reported quantitative associations (relative risk, odds ratio, or hazard ratio) between alcohol or tobacco exposure and site-specific cancer incidence or mortality.

2. Described exposure categories precisely (e.g., grams of ethanol/day, pack-years, current vs never smokers).
3. Included adult populations (≥18 years) but with stratified or interpretable data for participants aged 50 years and above.
4. Provided sufficient detail to extract baseline and exposure-specific risks.
   Case reports, animal experiments, and editorials were excluded. When population-based registries or cohort studies overlapped geographically or temporally, the larger or more recent dataset was retained.

Data Extraction and Synthesis

Data were manually extracted into a structured template capturing: author, publication year, study design, population size, exposure category, cancer site, and summary risk estimate (RR/OR/HR) with corresponding confidence intervals. For multi-level dose categories, incremental relative risks were standardized per 10–12 g ethanol/day for alcohol and per smoking status for tobacco (current, former, never).

No statistical meta-analysis or re-pooled estimate was performed. Instead, representative point estimates from authoritative sources were tabulated and visualized as bar and donut charts. These figures display the magnitude of risk differences without modification of the original data.

Outcome Measures

Primary outcomes were:

- **Site-specific relative risk or odds ratio** comparing exposed versus unexposed adults.
- **Dose–response gradient**, expressed per 10 g ethanol/day for alcohol.
- **Lifetime cancer risk by age 80**, stratified by smoking status.
- **Disability-adjusted life-years (DALYs)** attributable to smoking-related cancers.
  Secondary outcomes included relative risk decline following cessation or abstinence, where data were available.

RESULTS AND OBSERVATIONS:

1. Alcohol and cancer: causal sites and magnitudes

Across high-quality evaluations, alcohol is **causally linked** to cancers of the oral cavity, pharynx, larynx, oesophagus (squamous cell carcinoma – ESCC), liver (hepatocellular carcinoma – HCC), colorectum, and female breast.

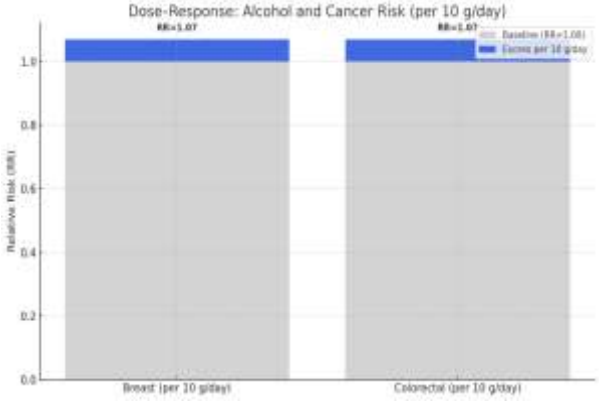
Table 1 below summarizes the recognized causal sites and their approximate magnitudes of effect.

Table 1. Alcoholic beverages (Group-1) and cancer sites with indicative magnitudes

Cancer site	Evidence class	Typical magnitude (illustrative)
Oral cavity, pharynx, larynx	Causal	Elevated risk with increasing intake
Oesophagus (SCC)	Causal	Any vs none ≈ 2.3-fold increase
Liver (HCC)	Causal	≈ 10 % higher per ~12 g/day; greater with heavy intake
Female breast	Causal	≈ 7 % higher per 10 g/day
Colorectum	Causal	≈ 7 % higher per 10 g/day

Figure 1 below illustrates the dose–response relationship, showing baseline versus excess risk for breast and colorectal cancers.

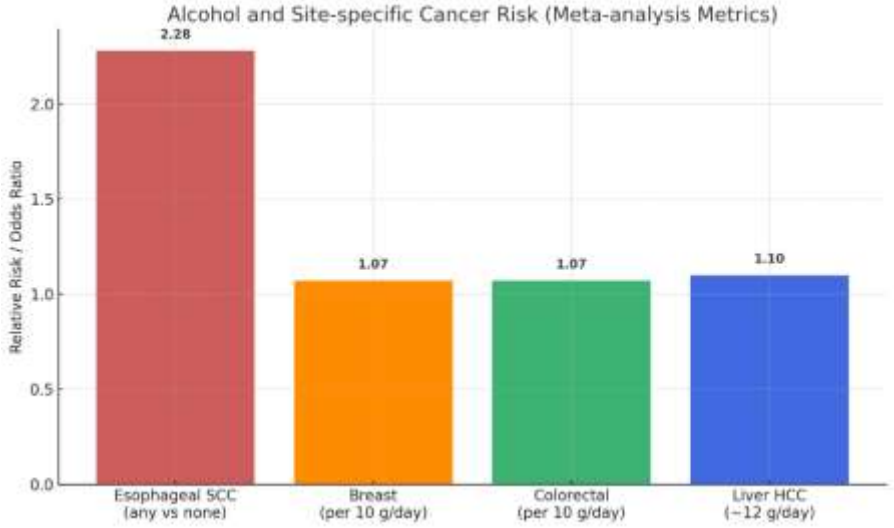
Figure 1: "Alcohol Dose–Response, Stacked Bar"



Alcohol dose–response: excess risk per 10 g/day for breast and colorectal cancers
(Data adapted from Bagnardi V et al., *Ann Oncol* 2015;26(8):1523–35 [4].)

For oesophageal SCC, even light drinking confers measurable elevation, while for HCC, risk begins to climb around one drink/day and rises steeply with heavier intake. Combined site-specific estimates for ESCC, breast, colorectum, and HCC are presented visually in **Figure 2**.

Figure 2: "Alcohol and Site-Specific Cancer Risk, Bar Chart"



Alcohol and site-specific cancer risk metrics (bar): oesophageal SCC any vs none; breast and colorectum per 10 g/day; hepatocellular carcinoma per ~12 g/day
(Data combined from Bagnardi V et al. [4], Middleton DRS et al. [9], and Chuang SC et al. [10].)

2. Dose–response increments for alcohol

To demonstrate quantitative trends, **Table 2** compiles the standardized increments used in the figures.

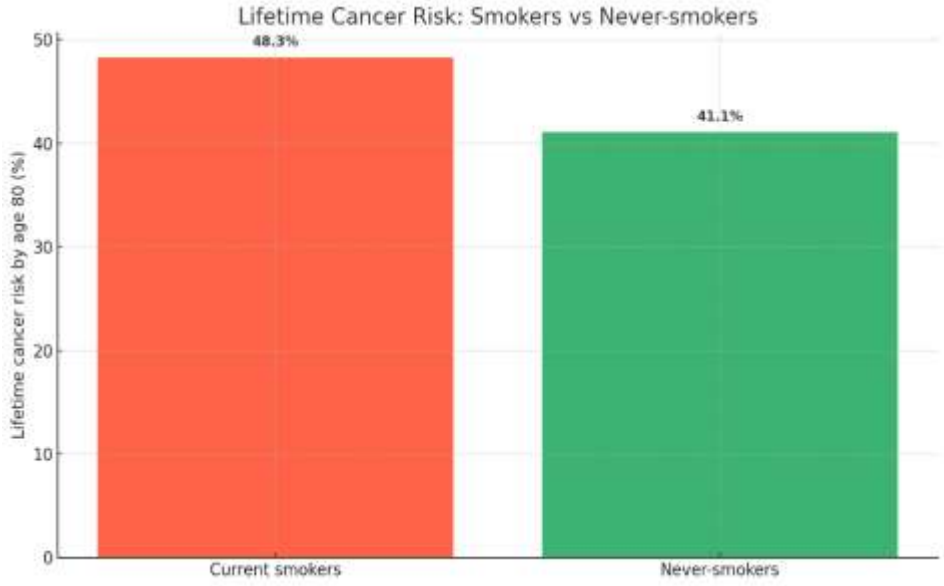
Table 2. Dose–response parameters for alcohol-related cancers

Cancer site	Dose metric	Increment	Relative risk applied in the figure
Breast	Per 10 g ethanol/day	+10 g/day	RR ≈ 1.07
Colorectum	Per 10 g ethanol/day	+10 g/day	RR ≈ 1.07
Liver (HCC)	Per ~12 g/day (~1 drink)	+12 g/day	RR ≈ 1.10

3. Smoking and cancer: lifetime risk and burden

In a population-based cohort, the lifetime risk of any cancer by age 80 was markedly higher among current smokers compared with never-smokers. This absolute difference is illustrated in **Figure 3**.

Figure 3: "Lifetime Cancer Risk, Bar Chart"

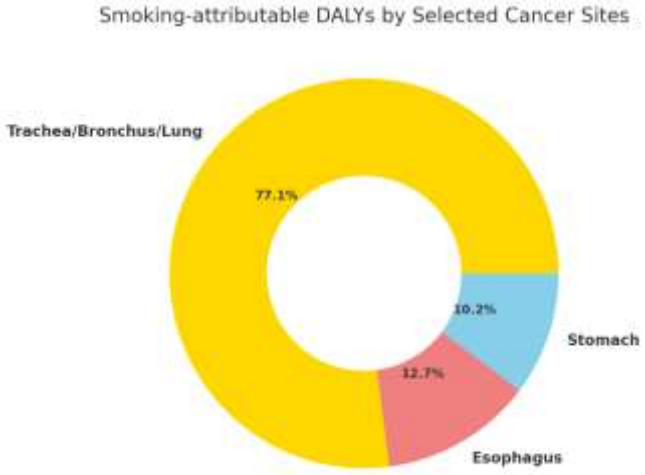


Lifetime risk of any cancer by age 80 among current vs never-smokers.

(Data derived from Weber MF et al., JAMA Netw Open 2021;4(5):e2114305 [7].)

Smoking also contributes heavily to the global cancer burden. The largest smoking-attributable disability-adjusted life-years (DALYs) arise from trachea/bronchus/lung, oesophagus, and stomach cancers, depicted in **Figure 4** and summarized numerically in **Table 3**.

Figure 4: "Smoking-Attributable DALYs, Donut Chart"



Smoking-attributable DALYs by selected cancer sites (donut chart: trachea-bronchus-lung, oesophagus, stomach)
(Based on data according to Sharma R et al., eClinicalMedicine 2022;48:101424 [8].)

Table 3. Smoking-related cancer burden and lifetime risk

Outcome	Metric	Estimate	Context
Any cancer (to age 80)	Lifetime risk (%	Current smokers 48.3 vs never-smokers 41.1	Population-based cohor
Smoking-attributable burde	DALYs (millions)	Trachea/bronchus/lung 28.6; Oesophagus 4.7; Stomach 3.8	Global burden estimate

(Note: All tables' data are summarized or redrawn from the same corresponding references as the figures)

DISCUSSION

The compiled evidence consistently shows that alcohol is carcinogenic across multiple sites, and risk increases with both duration and quantity of consumption [8–10]. The ~7 % risk rise per 10 g/day for breast and colorectal cancers, visualized in Figure 1 and Table 2, is small individually but considerable at the population level. The sharp odds increase for oesophageal squamous cell carcinoma and the measurable rise in hepatocellular carcinoma even at low intake reinforce that no safe threshold of alcohol use exists [9,10]. Smoking remains the strongest preventable determinant of cancer burden in later life. The approximate 7-percentage-point excess lifetime cancer risk for current smokers versus never-smokers (Figure 3, Table 3) mirrors global findings where lung, oesophagus, and stomach cancers account for the largest DALYs (Figure 4) [8]. When alcohol and tobacco exposures co-occur, risks amplify multiplicatively, particularly for head-and-neck and laryngeal cancers [11,12].

Mechanistically, the carcinogenicity of ethanol is mediated through its first metabolite, acetaldehyde, which induces DNA adduct formation, point mutations, and impaired DNA repair, while chronic exposure promotes oxidative stress, folate depletion, and hormonal perturbation that contribute to breast and colorectal carcinogenesis [14,15]. Tobacco smoke introduces over seventy established carcinogens, including nitrosamines and polycyclic aromatic hydrocarbons, that directly damage epithelial DNA and suppress immune surveillance. Combined exposure intensifies field cancerization in aerodigestive mucosa, explaining the multiplicative rather than additive effect observed epidemiologically [16]. Such mechanistic complementarity underscores why dual abstinence delivers substantially greater preventive benefit than partial modification of one behavior.

In India, the convergence of high-prevalence tobacco use (particularly smokeless forms) and rapidly increasing alcohol consumption among middle-aged adults represents a distinctive public-health challenge. Data from the National Family Health Survey-5 show that nearly one in three adult men use tobacco and one in five consume alcohol regularly, patterns associated with rising incidences of oral, oesophageal, and liver cancers in state registries [17].

Strengthening dual-risk screening through the National Programme for Prevention and Control of Cancer,

Diabetes, Cardiovascular Diseases and Stroke (NPCDCS) could integrate alcohol and tobacco cessation within routine midlife health checks. Public-sector initiatives such as alcohol-free day campaigns, nicotine-replacement therapy access, and viral-hepatitis vaccination for hepatocellular carcinoma prevention offer feasible, low-cost interventions. Beyond behavioral approaches, targeted taxation and warning-label enforcement remain powerful levers to reduce exposure at the population scale [18,19].Risk reduction after cessation is substantial: alcohol-related cancer risk diminishes gradually after sustained abstinence, with faster declines for upper-aerodigestive cancers [8,9]. For smoking, excess lung-cancer risk drops steeply within 10–15 years of quitting, though never fully returns to baseline [13]. These temporal gradients reinforce that interventions in the fifth and sixth decades of life remain worthwhile. Overall, the convergence of molecular, epidemiologic, and programmatic evidence positions alcohol and tobacco reduction as central pillars of site-specific cancer prevention beyond age fifty.

CONCLUSION

After age 50, accumulated exposure to alcohol and tobacco significantly heightens the risk for several cancers. Even modest drinking reduction and complete tobacco cessation translate into measurable reductions in lifetime cancer risk. Integrating dual-risk counselling and cessation support into routine midlife health checks offers high preventive value.

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