Cancer Association of South Africa (CANSA)

Fact Sheet and Position Statement on Alcohol Consumption and Cancer Risk

Introduction

The International Agency for Research on Cancer (IARC) is part of the World Health Organization (WHO). Its major goal is to identify causes of cancer. The most widely used system for classifying carcinogens comes from the IARC.

In the past 30 years, the IARC has evaluated the cancer-causing potential of more than 900 likely substances, placing them into one of the following groups:

[Picture Credit: Alcohol]

- Group 1: Carcinogenic to humans
- Group 2A: Probably carcinogenic to humans
- Group 2B: Possibly carcinogenic to humans
- Group 3: Unclassifiable as to carcinogenicity in humans
- Group 4: Probably not carcinogenic to humans

Alcohol was declared a carcinogen (Group 1) in 1988 by IARC. Surprisingly, most people are not aware of alcohol's cancer risk. The IARC reports that no amount of alcohol is safe to drink. Alcohol is clearly identified as causative for cancers of the mouth, pharynx, larynx, oesophagus, colon, rectum, liver, and female breast. IARC re-confirmed that alcohol is a Group 1 carcinogen in 2007 and again in 2009.

Even 'light drinking' has been linked to causing cancers of the mouth, oesophagus and breast. The more one drinks the greater the cancer risk. There is no risk-free level of alcohol consumption. There is always some risk, and the risk increases in accordance with the level of consumption.

Alcohol is blamed for 1 in every 30 cancer deaths worldwide. It will not be surprising if, in the near future, alcoholic beverages are required to have warning labels explaining risk of cancer, hypertension, stroke, atrial fibrillation and cardiomyopathy.
(IARC Working Group; Bagnardi, *et al*. 2013; American Cancer Society; Cancer Research UK).

IARC Definitions

The International Agency for Research on Cancer (IARC) uses the following definitions to classify cancer causing substances:

Group 1

The agent is carcinogenic to humans. This category is used when there is sufficient evidence of carcinogenicity in humans. Exceptionally, an agent may be placed in this category when evidence of carcinogenicity in humans is less than sufficient but there is sufficient evidence of carcinogenicity in experimental animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

Group 2

This category includes agents for which, at one extreme, the degree of evidence of carcinogenicity in humans is almost sufficient, as well as those for which, at the other extreme, there are no human data but for which there is evidence of carcinogenicity in experimental animals. Agents are assigned to either Group 2A (probably carcinogenic to humans) or Group 2B (possibly carcinogenic to humans) on the basis of epidemiological and experimental evidence of carcinogenicity and mechanistic and other relevant data. The terms probably carcinogenic and possibly carcinogenic have no quantitative significance and are used simply as descriptors of different levels of evidence of human carcinogenicity, with probably carcinogenic signifying a higher level of evidence than possibly carcinogenic.

Group 2A

The agent is probably carcinogenic to humans. This category is used when there is limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. In some cases, an agent may be classified in this category when there is inadequate evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals and strong evidence that the carcinogenesis is mediated by a mechanism that also operates in humans. Exceptionally, an agent may be classified in this category solely on the basis of limited evidence of carcinogenicity in humans. An agent may be assigned to this category if it clearly belongs, based on mechanistic considerations, to a class of agents for which one or more members have been classified in Group 1 or Group 2A.

Group 2B

The agent is possibly carcinogenic to humans. This category is used for agents for which there is limited evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals. It may also be used when there is inadequate evidence of carcinogenicity in humans but there is sufficient evidence of carcinogenicity in experimental animals. In some instances, an agent for which there is inadequate evidence of carcinogenicity in humans and less than sufficient evidence of carcinogenicity in experimental animals together with supporting evidence from mechanistic and other relevant data may be placed in this group. An agent may be classified in this category solely on the basis of strong evidence from mechanistic and other relevant data.

Group 3

The agent is not classifiable as to its carcinogenicity to humans. This category is used most commonly for agents for which the evidence of carcinogenicity is inadequate in humans and

inadequate or limited in experimental animals. Exceptionally, agents for which the evidence of carcinogenicity is inadequate in humans but sufficient in experimental animals may be placed in this category when there is strong evidence that the mechanism of carcinogenicity in experimental animals does not operate in humans. Agents that do not fall into any other group are also placed in this category. An evaluation in Group 3 is not a determination of non-carcinogenicity or overall safety. It often means that further research is needed, especially when exposures are widespread or the cancer data are consistent with differing interpretations.

Group 4
The agent is probably not carcinogenic to humans. This category is used for agents for which there is evidence suggesting lack of carcinogenicity in humans and in experimental animals. In some instances, agents for which there is inadequate evidence of carcinogenicity in humans but evidence suggesting lack of carcinogenicity in experimental animals, consistently and strongly supported by a broad range of mechanistic and other relevant data, may be classified in this group.
(IARC Press Release No 196, 2 November 2009).

Carcinogenicity of Alcohol
Alcoholic beverages are carcinogenic to humans (Group 1). Ethanol in alcoholic beverages is carcinogenic to humans (Group 1). The latter evaluation [of ethanol] is based on by the International Agency for Research on Cancer (IARC):
(i) the epidemiological evidence, which showed little indication that the carcinogenic effects depend on the type of alcoholic beverage
(ii) the sufficient evidence that ethanol causes cancer in experimental animals; and
(iii) the mechanistic evidence in humans who are deficient in aldehyde dehydrogenase that acetaldehyde derived from the metabolism of ethanol in alcoholic beverages contributes to the causation of malignant oesophageal tumours.

Alcohol use is a cause of cancer. Any level of alcohol consumption increases the risk of developing an alcohol-related cancer; the level of risk increases in line with the level of consumption.

There is strong evidence that alcohol causes cancer at seven sites [in the body], and probably others. The evidence supports "a causal association of alcohol consumption" with cancer in the oropharynx (a part of the throat), the larynx, the oesophagus, the liver, the colon, the rectum and the female breast.

There is also growing evidence suggesting a strong link between alcohol and other cancers, such as prostate, pancreatic and melanoma. However, that evidence is not enough at this point to allow researchers to conclude that there is cause-and-effect relationship for these cancers, according to the article.

Moreover, for each of the seven cancers that are directly linked, previous studies have found that there is a "dose-response relationship," meaning that the more alcohol a person drinks, the more likely the person is to develop those cancers.

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Lin, H.Y., Fisher, P., Harris, D. & Tseng, T.S. 2019.

BACKGROUND: Alcohol intake is a leading modifiable cause related to cancer-specific deaths. Various alcohol intake patterns have shown to impact cancer progression differently, however, many studies only evaluated simplified patterns (such as heavy *vs*. non-heavy drinking) of alcohol intake for cancer survivors. The objective of this study is to provide population-based prevalence of the complex alcohol drinking patterns for cancer survivors, and compare it with that of non-cancer individuals. Additionally, we evaluated the impact of cancer related factors (binary, alcohol-related cancer type, and length of cancer history) to the alcohol intake patterns adjusted for the selected factors.

METHODS: A total of 193,197 individuals, including 16,504 cancer survivors, with age ≥18 years old in the 2012-2017 National Health Interview Survey (NHIS) were included in this study. The population-based prevalence of alcohol patterns was estimated. To evaluate cancer related factors associated with the alcohol intake patterns, we applied multinomial logistic models with the appropriate sampling weights and adjusted the selected demographic factors and smoking status.

RESULTS: There were 62.1% of cancer survivors and 66.0% of non-cancer individuals who were current alcohol drinkers in the past year. The prevalence of heavy drinking was identical for 5.2% of cancer and non-cancer individuals. For frequent binge drinking, cancer survivors tended to have less frequent binge than non-cancer individuals (2.8% *vs*. 4.9%). After adjusting for the selected demographic factors and smoking status, the cancer survivors were less likely to have the intermediate level of alcohol intake (light/moderate or occasional binge drinking) compared with non-cancer individuals, but no difference for the excessive alcohol intake (heavy or frequent binge drinking) was observed for those with and without cancer. As for cancer type, those with non-alcohol related cancer tended to be a current drinker compared with those with alcohol-related cancer. Compared with cancer survivors with a short cancer history (2-4 years), survivors with a cancer history of 5-9 years were more likely to be current drinkers after adjusting for the selected factors. Cancer status, alcohol-related cancer type and length of cancer history had no impact on excessive alcohol intake.

CONCLUSIONS: In summary, cancer survivors have similar excessive alcohol drinking patterns but were less likely to have the intermediate level of alcohol intake compared to non-cancer individuals. Alcohol intake may enhance cancer progression, interfere with cancer treatments and increase cancer-related mortality. To improve cancer survivors' health, custom alcohol interventions and cessation programs should be conducted to minimize alcohol intake for cancer survivors, especially for excessive alcohol drinkers.

Andersson, T.M., Engholm, G., Pukkala, E., Stenbeck, M., Tryggvadottir, L., Storm, H. & Weiderpass, E. 2018.

BACKGROUND: Alcohol consumption is an important and preventable cause of cancer. The aim of this study was to quantify the proportion of the cancer burden in the Nordic countries linked to alcohol and estimate the potential for cancer prevention by changes in alcohol consumption.

METHODS: Using the Prevent macro-simulation model, the number of cancer cases in the Nordic countries over a 30-year period (2016-2045) was modelled for six sites, under different scenarios of changing alcohol consumption, and compared to the projected number of cases if constant alcohol consumption prevailed. The studied sites were colorectal, post-menopausal breast, oral cavity and pharynx, liver, larynx as well as oesophageal squamous cell carcinoma. The alcohol consumption was based on the categories of non-drinkers/occasional drinkers, light drinkers (<=12.5 g alcohol per day), moderate drinkers (>12.5 and ≤ 50 g/day) and heavy drinkers (>50 g/day).

RESULTS: About 83,000 cancer cases could be avoided in the Nordic countries in a 30-year period if alcohol consumption was entirely eliminated, which is 5.5% of the expected number of cases for the six alcohol-related cancer types. With a 50% reduction in the proportion with moderate alcohol consumption by year 2025, 21,500 cancer cases could be avoided. The number of avoidable cases was highest for post-menopausal breast and colorectal cancer, but the percentage was highest for oesophageal squamous cell carcinoma.

CONCLUSION: The results from this study can be used to understand the potential impact and significance of primary prevention programmes targeted towards reducing the alcohol consumption in the Nordic countries.

Alcohol Converted to Acetaldehyde in the Body

In the human, alcohol (ethanol) is converted into a toxic chemical called acetaldehyde. Acetaldehyde was previously classified as possibly carcinogenic to humans (Group 2B) because there was inadequate evidence in humans for the carcinogenicity of acetaldehyde, although there was sufficient evidence in experimental animals for the carcinogenicity of acetaldehyde (IARC Monograph 71, 1999).

However, there is now evidence that acetaldehyde can cause cancer by damaging DNA and stopping the cells from repairing the damage. The International Agency for Research on Cancer has also classified acetaldehyde formed as a result of drinking alcohol as being a cause of cancer, along with alcohol itself.

Nearly 2 billion adults worldwide are estimated to consume alcoholic beverages regularly, with an average daily consumption of 13g of ethanol (about one drink). Alcohol consumption has already been shown to cause cancers of the oral cavity, pharynx, larynx, oesophagus, colorectum, liver and female breast; there is now also some evidence for cancer of the pancreas.

The relative risk of breast cancer increases with increasing alcohol intake by about 10% per 10g/day.

Higher risk for East-Asian populations linked to alcohol metabolism. Alcohol consumption results in exposure to acetaldehyde, present in the beverage itself and also formed when the body breaks down alcohol. Alcohol is metabolised to acetaldehyde, (which is a genotoxic chemical), then this acetaldehyde is further metabolised to acetate (a harmless chemical) by enzymes known as aldehyde dehydrogenases (ALDH).

A large proportion of people of east-Asian origin worldwide (up to 30% in some populations) has an inactive enzyme (known as ALDH2*2) that has only about 10% residual enzymatic activity. Carriers of the inactive enzyme are extremely slow to metabolise acetaldehyde, as a result, they experience higher internal levels of acetaldehyde and have much higher risks of oesophageal cancer and cancers of the head and neck compared with individuals with the active enzyme.

The IARC Working Group concluded that acetaldehyde associated with alcohol consumption is carcinogenic to humans (Group 1) and confirmed the classification in Group 1 of alcohol consumption and of ethanol in alcoholic beverages. (IARC Press Release No 196, 2 November 2009; Wold Cancer Research Fund International).

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Energy Drinks and Alcohol Consumption
Energy drinks are popular beverages that typically include high levels of caffeine and other ingredients such as taurine, or caffeine-containing herbs, such as guarana. While energy drinks are often consumed alone, they are also frequently used as mixers for alcoholic beverages.

Marczinski, C.A. & Fillmore, M.T. 2014.
Energy drinks are popular beverages that typically include high levels of caffeine and other ingredients such as taurine, or caffeine-containing herbs, such as guarana. While energy drinks are often consumed alone, they are also frequently used as mixers for alcoholic beverages. This review summarizes what is known about the scope of use of alcohol mixed with energy drinks, the risks associated with such mixtures, and the objective laboratory data examining how the effects of their consumption differ from consuming alcohol alone. The weight of the evidence reveals that consuming alcohol mixed with energy drinks is riskier than consuming alcohol alone and constitutes a public health concern. Consumption of these mixed beverages is frequent, especially in young and underage drinkers, and compared with alcohol alone, their use is associated with elevated rates of binge drinking, impaired driving, risky sexual behavior, and risk of alcohol dependence. Laboratory research (human and animal) has demonstrated that consuming alcohol mixed with energy drinks leads to altered subjective states including decreased perceived intoxication, enhanced stimulation, and increased desire to drink/increased drinking compared to consuming alcohol alone. Possible underlying mechanisms explaining these observations are highlighted in this review.

Alcohol and Cancer Risk
Drinking alcohol increases the risk of mouth and throat cancer (larynx and pharynx), oesophageal cancer, bowel cancer (colon and rectum), liver cancer and female breast cancer. It is not just heavy drinking – even small amounts of alcohol increases cancer risk, but the more one drinks, the greater the risk. One's risk of cancer is the same for all types of alcohol including beer, wine and spirits, and there is no evidence that alcohol helps protect one from any type of cancer.

To reduce the risk of cancer, one should limit one's intake of alcohol or, better still, avoid it all together.

According to a recent study by Burton and Sheron (2018):
"The conclusions of the study are clear and unambiguous: alcohol is a colossal global health issue and small reductions in health-related harms at low levels of alcohol intake are outweighed by the increased risk of other health-related harms, including cancer. There is strong support here for the guideline published by the3 Chief Medical Officer of the UK who found that there is 'no safe level of alcohol consumption'."

Alcohol and Oral and Pharyngeal Cancer
According to the IARC Working Group, a large body of evidence from epidemiological studies of different design, and conducted in different populations, consistently shows that consumption of alcoholic beverages is associated with a higher risk for both oral and pharyngeal cancer, and that the risk increases with increasing amounts of alcohol consumed.

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Although tobacco use has been proven to increase the risk of oral cancer, people who use both alcohol and tobacco are at an especially high risk of contracting the disease. Scientists now believe that these substances synergistically interact, increasing each other's harmful effects.

There is now considered to be no safe limit for alcohol intake. Studies have shown that risk of mouth cancer increases with greater alcohol intake (in particular when associated with the use of tobacco).
(Ogden, 2018)

Kawakita, D. & Matsuo, K. 2017.
"In this article, we reviewed the association between alcohol drinking and head and neck cancer (HNC) and its subsites, using the available literature. Alcohol drinking is an established risk factor for HNC, and this association may be stronger among cancers of the oropharynx and hypopharynx than the oral cavity or larynx. In addition, higher alcohol consumption over a shorter period was more harmful than fewer alcohol consumption over a longer period, and the most frequently consumed alcoholic beverages in a population is likely to be associated with the highest risk of HNC in that population. The risk of HNC after ≥ 20 years of alcohol cessation appear to be similar to the risk among never drinkers. The interaction between genetic polymorphisms related to alcohol metabolism and alcohol drinking on the risk of HNC has been noted, and the prevalence of these genetic polymorphisms in each population should be of concern. Finally, the association between alcohol drinking and the survival of individuals with HNC remains unclear, and mortality due to competing causes should be considered in future research to evaluate this association."

Alcohol and Cancer of the Larynx
Studies of different design conducted in Asia, Europe, North America and South America have shown a consistent association between the consumption of alcoholic beverages and the risk for laryngeal cancer. This association increases with increasing amounts of alcoholic beverages consumed and, compared with non-drinkers, regular consumption of about 50g of alcohol per day is associated with an approximately twofold increase in risk. These associations were observed for various types of alcoholic beverages (IARC).

Drinking alcohol, especially spirits, over a long period of time increases a person's risk of getting laryngeal cancer. The risk is much higher for people who are both smokers and heavy drinkers

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to alcohol metabolism and alcohol drinking on the risk of HNC has been noted, and the prevalence of these genetic polymorphisms in each population should be of concern. Finally, the association between alcohol drinking and the survival of individuals with HNC remains unclear, and mortality due to competing causes should be considered in future research to evaluate this association."

Alcohol and Cancer of the Oesophagus
More than 50 prospective and case–control studies from most regions of the world found a consistent association between the risk for oesophageal cancer (squamous cell carcinoma) and the consumption of alcoholic beverages.

The risk increases with increasing amounts of alcoholic beverage consumed and, compared with non-drinkers, regular consumption of about 50g alcohol per day is associated with an approximately twofold increase in risk.

The increased risk for oesophageal cancer was consistently observed for a range of different types of alcoholic beverage. However, the association, if any, is weak for adenocarcinoma of the oesophagus. (IARC).

A strong association exists between alcohol use and cancers of the oesophagus, pharynx, and mouth, with links to alcohol as a cause of liver, breast, and colorectal cancers.

Oesophageal cancer is associated with a number of risk factors:
• Alcoholic beverages (and acetaldehyde associated with their consumption)
• Betel quid (with and without tobacco)
• Smokeless tobacco
• Tobacco smoking
• X-radiation, gamma-radiation
• Obesity

Matejcic, M., Gunther, M.J. & Ferrari, P. 2017.
"Alcohol is a major risk factor for oesophageal squamous cell carcinoma (OSCC), the most prevalent histological subtype of oesophageal cancer (OC) worldwide. The metabolism of alcohol is regulated by specific enzymes whose activity and expression is influenced by genetic polymorphisms. We conducted a systematic review of current epidemiological evidence of the relationship between alcohol intake and OC risk, including the role of tobacco smoking and functional polymorphisms of alcohol dehydrogenases (ADHs) and aldehyde dehydrogenases (ALDHs). Potential biological mechanisms underlying oesophageal carcinogenesis are also discussed. Frequency and intensity of alcohol intake have been consistently associated with an increased risk of OSCC in regions with low and high incidence of the disease. The highest risk was reported among tobacco smokers, whereas the association between alcohol and OSCC risk was weak in the absence of tobacco use. The ADH1B, ADH1C and ALDH2 gene polymorphisms influence the risk of OSCC through modulation of acetaldehyde metabolism and propensity to alcohol intake. These functional variants may be suitable proxies of alcohol exposure for use in Mendelian randomization studies if complemented by reported alcohol intake data. Recent epidemiological and experimental studies investigating the role of alcohol consumption in OC development have implicated the microbiome as

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a new promising avenue for research, which entail novel potential mechanisms of alcohol-related oesophageal carcinogenesis. Microbial communities associated with alcohol consumption might be used as biomarkers to raise the potential of intervening among susceptible individuals."

Asombang, A.W., Chishinga, N., Nkhoma, A., Chipaila, J., Nsokolo, B., Manda-Mapalo, M., Montiero, J.F.G., Banda, L., Dua, K.S. 2019.
BACKGROUND: Esophageal cancer (EC) is associated with a poor prognosis, particularly so in Africa where an alarmingly high mortality to incidence ratio prevails for this disease.
AIM: To provide further understanding of EC in the context of the unique cultural and genetic diversity, and socio-economic challenges faced on the African continent.
METHODS: We performed a systematic review of studies from Africa to obtain data on epidemiology, risk factors, management and outcomes of EC. A non-systematic review was used to obtain incidence data from the International Agency for Research on Cancer, and the Cancer in Sub-Saharan reports. We searched EMBASE, PubMed, Web of Science, and Cochrane Central from inception to March 2019 and reviewed the list of articles retrieved. Random effects meta-analyses were used to assess heterogeneity between studies and to obtain odds ratio (OR) of the associations between EC and risk factors; and incidence rate ratios for EC between sexes with their respective 95% confidence intervals (CI).
RESULTS: The incidence of EC is higher in males than females, except in North Africa where it is similar for both sexes. The highest age-standardized rate is from Malawi (30.3 and 19.4 cases/year/100000 population for males and females, respectively) followed by Kenya (28.7 cases/year/100000 population for both sexes). The incidence of EC rises sharply after the age of 40 years and reaches a peak at 75 years old. Meta-analysis shows a strong association with tobacco (OR 3.15, 95%CI: 2.83-3.50). There was significant heterogeneity between studies on alcohol consumption (OR 2.28, 95%CI: 1.94-2.65) and on low socioeconomic status (OR 139, 95%CI: 1.25-1.54) as risk factors, but these could also contribute to increasing the incidence of EC. The best treatment outcomes were with esophagectomy with survival rates of 76.6% at 3 years, and chemo-radiotherapy with an overall combined survival time of 267.50 d.
CONCLUSION: Africa has high incidence and mortality rates of EC, with preventable and non-modifiable risk factors. Men in this setting are at increased risk due to their higher prevalence of tobacco and alcohol consumption. Management requires a multidisciplinary approach, and survival is significantly improved in the setting of esophagectomy and chemoradiation therapy.

Oze, I., Charvat, H., Matsuo, K., Ito, H., Tamakoshi, A., Nagata, C., Wada, K., Sugawara, Y., Sawada, N., Yamaji, T., Naito, M., Tanaka, K., Shimazu, T., Mizoue, T., Tsugane, S. & Inoue, M. 2019.
"Cigarette smoking and alcohol drinking are two major risk factors for esophageal cancer. Not all, but several of case-control studies have indicated interaction between the two factors; however, no prospective study has validated this phenomenon to date. Therefore, the interaction between smoking and alcohol drinking is still open-ended question. To answer this, we conducted a pooled analysis using large-scale population-based cohort studies in Japan. Male subjects from eight cohort studies were included. Cigarette smoking and alcohol drinking were both categorized categorically (never/ever), and in the three consumption levels of pack years and ethanol consumption/day. Effects of smoking and drinking in each study were estimated by Cox regression models. The study-specific results were combined through meta-analysis to obtain summary effects of hazard ratios (HRs) and measures of interactions at both additive and multiplicative scales. Population attributable fractions (PAFs) from smoking and drinking were obtained using distributions of exposures and fully adjusted HRs. In 162 826 male subjects, 954 esophageal cancer incidences were identified. HRs of

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ever smoking, ever drinking, and their combination were 2.92 (1.59-5.36), 2.73 (1.78-4.18), and 8.86 (4.82-16.30), respectively. Interaction between cigarette smoking and alcohol drinking was significantly positive on the additive scale, but not significant on the multiplicative scale. The joint effect of smoking and drinking in three levels of evaluation showed a similar significant super-additive interaction. PAFs from smoking, drinking, and their combination were 55.4%, 61.2%, and 81.4%, respectively. Cigarette smoking and alcohol drinking had a significant positive additive interaction for esophageal cancer risk."

Alcohol and Cancer of the Mouth

Although tobacco use has been proven to increase the risk of oral cancer, people who use both alcohol and tobacco are at an especially high risk of contracting the disease. Scientists now believe that these substances synergistically interact, increasing each other's harmful effects.

Rogha, M., Berjis, N., Laievardi, S.M., Alamdaran, M. & Hashemi, S.M. 2019.
BACKGROUND: Tongue cancer is the most common malignancy of the mouth. In recent decades, reported tongue cancer incidence and mortality rates have increased all over the world while survival has not improved that sometimes is related to mutation, especially in gene P53 (such R249, R248 mutations). Hence, this study aimed to identification of R249 mutation in P53 gene of tumor tissue in tongue cancer.
METHODS: In a cross-sectional study, 48 patients with squamous cell carcinoma (SCC) of tongue were selected, and mutation of R249 was investigated in sample of tumors. In addition, demographic data and medical history of patients were determined and registered in a collected data form. Finally added data were entered to computer and analyzed by SPSS software.
RESULTS: Polymerase-chain reaction test done on tissue samples from cancer patients showed that in a studied sample of 48 patients, 4 of them (8.3%) had R249 mutation. After selecting the codon 249 as a hotspot in oral cancer, forward and reverse primers for amplification of exon 7 were obtained from the articles.
CONCLUSIONS: Considering the findings of our study, R249 mutation in P53 gene in patients with SCC is relatively high. Age and alcohol consumption were factors affecting incidence of the mutation. It is necessary to take an early treatment with a single lesion of tongue to prevent severe disease and prevent disease in patient's family with screening test and prevent cancer in future with gene therapy.

Fonseca, A.L.A., Prosdocimi, F., Bianco, B., Perez, M.M., Fonseca, F.L.A., & da Costa Agular Alves, B. 2019.
"Among the types of cancers that may occur in the oral cavity, squamous cell carcinomas (SCC) of the mouth have a higher incidence and are associated with increased rates of morbidity and mortality. Among steps from the beginning to the progression of the tumour, DNA Repair System is highlighted. The present study aims to conduct a systematic review of the literature on the expression of the repair genes hMSH2 and hMSH6 in patients with SCC in the mouth and oropharyngeal region. The search was performed in databases such as PubMed, Lilacs, and Scielo and included articles published in English from 1999 until 2015. The search in the above-mentioned databases initially yielded 15 scientific articles related to the proposed objective. After a detailed analysis of each of them, only 8 were included in the present review, precisely because they met the inclusion criteria determined in the method. All the reviewed works were unanimous in recognizing

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the veracity and complexity of the Genomic Repair System, also called Mismatch Repair System, confirming the participation of repair gene proteins (such as hMSH2 and hMSH6) in patients with oral cancer and even of lesions that are susceptible to malignization. SIGNIFICANCE OF THE STUDY: Worldwide, there are an estimated 300 thousand new cases of oral cancer per year. Studies have shown a greater risk in individuals who are smokers and alcohol consumers in developing mouth cancer. Many steps are observed from the beginning to the progression of the tumour, highlighted among them is the moment in which genetic, and epigenetic alterations will interfere in the functioning of the DNA Repair System. This work presents a survey of current knowledge about the involvement of repair genes, especially those of the MutSα system, in the development and progression of oral cancer."

Alcohol and Cancer of the Liver

Studies provide firm evidence that the consumption of alcoholic beverages is an independent risk factor for primary liver cancer. Various types of alcoholic beverage consumed do not have substantially different effects on liver cancer.
(IARC).

Primary liver cancer (hepatocellular carcinoma) tends to occur in livers damaged by birth defects, alcohol abuse, or chronic infection with diseases such as hepatitis B and C, haemochromatosis (a hereditary disease associated with too much iron in the liver), and cirrhosis.

More than half of all people diagnosed with primary liver cancer have cirrhosis - a scarring condition of the liver commonly caused by alcohol abuse. Hepatitis B and C and haemochromatosis can cause permanent damage and liver failure. Liver cancer may also be linked to obesity and fatty liver disease.

Alcohol and Female Breast Cancer

More than 100 epidemiological studies conducted in all regions of the world have evaluated the association between the consumption of alcoholic beverages and female breast cancer, and have consistently found an increased risk with increasing intake.

The effects of duration or cessation of consumption of alcoholic beverages on the risk for breast cancer are uncertain.
(IARC).

Each increment in one drink per day was associated with 10% increased risk of Androgen Receptor-positive and Oestrogen Receptor-positive breast cancer, respectively. (Wang, *et al.*, 2015).

Research consistently shows that drinking alcoholic beverages - beer, wine, and spirits - increases a woman's risk of hormone-receptor-positive breast cancer. Alcohol can increase levels of oestrogen and other hormones associated with hormone-receptor-positive breast cancer. Alcohol also may increase breast cancer risk by damaging DNA in cells.

Compared to women who do not drink alcohol at all, women who have three alcoholic drinks per week have a 15% higher risk of breast cancer. Experts estimate that the risk of breast cancer goes up another 10% for each additional drink women regularly have each day.

Teen and tween girls aged 9 to 15 who drink three to five drinks a week have three times the risk of developing benign breast lumps. Certain categories of non-cancerous breast lumps are associated with a higher risk of breast cancer later in life.

While only a few studies have been done on drinking alcohol and the risk of recurrence, a 2009 study found that drinking even a few alcoholic beverages per week (three to four drinks) increased the risk of breast cancer coming back in women who had been diagnosed with early-stage disease.

The bottom line is that regularly drinking alcohol can harm one's health, even if one does not binge drink or get drunk. All types of alcohol count.

Liu, Y., Nguyen, N. & Colditz, G.A. 2015.
"Alcohol consumption by adult women is consistently associated with risk of breast cancer. Several questions regarding alcohol and breast cancer need to be addressed. Menarche to first pregnancy represents a window of time when breast tissue is particularly susceptible to carcinogens. Youth alcohol consumption is common in the USA, largely in the form of binge drinking and heavy drinking. Whether alcohol intake acts early in the process of breast tumorigenesis is unclear. This review aims to focus on the influences of timing and patterns of alcohol consumption and the effect of alcohol on intermediate risk markers. We also review possible mechanisms underlying the alcohol-breast cancer association."

Choi, Y.J., Myung, S.K. & Lee, J.H. 2018.
PURPOSE: The purpose of this study was to determine whether light alcohol drinking increases the risk of cancer by using a meta-analysis of cohort studies because the newly revised 2015 European Code against Cancer fourth edition on alcohol and cancer was based on critical flaws in the interpretation and citation of the previous meta-analyses.
MATERIALS AND METHODS: PubMed and EMBASE were searched in April, 2016. Two authors independently reviewed and selected cohort studies on the association between very light (≤ 0.5 drink/day), light (≤ 1 drink/day), or moderate drinking (1-2 drinks/day) and the risk of cancer incidence and mortality. A pooled relative risk with its 95% confidence interval was calculated by a random-effects meta-analysis. Main outcome measures were cancer incidence and mortality.
RESULTS: A total of 60 cohort studies from 135 articles were included in the final analysis. Very light drinking or light drinking was not associated with the incidence of most cancers except for female breast cancer in women and male colorectal cancer. Conversely, light drinking was associated with a decreased incidence of both female and male lung cancer significantly and both female and male thyroid cancer marginally significantly. Moderate drinking significantly increased the incidence of male colorectal cancer and female breast cancer, whereas it decreased the incidence of both female and male hematologic malignancy.
CONCLUSION: We found that very light or light alcohol drinking was not associated with the risk of most of the common cancers except for the mild increase in the incidence of breast cancer in women and colorectal cancer in men.

Alcohol and Male Breast Cancer

It has been estimated that alcohol drinking increases the risk of breast cancer in women by approximately 7% for each increment of 10g alcohol per day. However, the few studies conducted on breast cancer among men have failed to detect an association with quantitative measures of alcohol drinking, even if the alcohol intake is generally higher in men than in women. On the other hand, increased risks of male breast cancer were inconsistently reported in alcoholics or patients with liver cirrhosis. The researchers investigated the role of alcohol drinking in male breast cancer using data collected in a population-based case-control study on seven rare cancers, conducted in Denmark, France, Germany, Italy, and Sweden.

The cases were 74 histologically verified male breast cancer patients aged 35-70 years. The controls (n = 1 432) were selected from population registers, and frequency-matched to the cases by age group and geographic area. To check for consistency, a separate analysis was conducted using as controls the patients with a rare cancer other than male breast cancer recruited simultaneously in the European study (n = 519 men).

Based on population controls, the risk of developing breast cancer in men increased by 16% (95% CI: 7-26%) per 10g alcohol/day (p < 0.001). An odds ratio of 5.89 (95% CI: 2.21-15.69) was observed for alcohol intake greater than 90g per day, as compared with light consumers (< 15g per day). Similar associations were observed when other rare cancer patients were used as controls.

The researchers found that the relative risk of breast cancer in men is comparable to that in women for alcohol intakes below 60g per day. It continues to increase at high consumption levels not usually studied in women.
(Guénel, *et al.*, 2004).

Cancer and Pancreatic Cancer

Alcoholic beverages are a known carcinogen. Drinking alcohol has been identified as a cause of pancreatitis which is a risk factor for pancreatic cancer. Therefore, stopping consumption of alcohol can reduce your risk for pancreatitis, and your risk for pancreatic cancer.

Tsai, H.J. & Chang, J.S. 2019.
"Despite the advancement in medical knowledge that has improved the survival rate of many cancers, the survival rate of pancreatic cancer has remained dismal with a five-year survival rate of only 9%. The poor survival of pancreatic cancer emphasizes the urgent need to identify the causes or the risk factors of pancreatic cancer in order to establish effective preventive strategies. This review summarizes the current evidence regarding the environmental (non-genetic, including lifestyle, and clinical factors) risk factors of pancreatic cancer. Based on the current evidence, the established risk factors of pancreatic cancer are cigarette smoking, chronic diabetes, and obesity. Other strong risk factors include low consumption of fruits and vegetables, excess consumption of alcohol, poor oral hygiene, and the lack of allergy history. In the future, more studies are needed to identify additional risk factors of pancreatic cancer, especially the modifiable risk factors that could be included in a public health campaign to educate the public in order to reduce the incidence of pancreatic cancer."

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Alcohol and Colorectal Cancer

IARC looked at more than 50 prospective and case–control studies which reported on the association between consumption of alcoholic beverages and the risk for colon, rectal or colorectal cancer. There is no consistent evidence that the association of colorectal cancer with the consumption of alcoholic beverages is modified by gender or by tobacco smoking. The data on the effects of duration and cessation of consumption of alcoholic beverages on the risk for colorectal cancer are inadequate.
(IARC).

Alcohol consumption, amount and type of beverage, and drinking patterns at baseline were considered in examination of the effect of alcohol consumption on the risk of colon cancer. The consumption of one or more alcoholic beverages a day at baseline was associated with approximately a 70% greater risk of colon cancer [relative risk (RR)=1.69; 95% confidence interval (CI)=1.03, 2.79], with a strong positive dose-response relationship (P=0.04). This association appeared to be exclusively related to daily drinking of one or more drinks of liquor (RR=2.48; 95% CI=1.66, 4.53).

Overall, alcohol consumption was significantly associated with increased risk of colon cancer. The most important factor for colon cancer seems to be liquor consumption.
(Su & Arab, 2004).

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PURPOSE: The purpose of this study was to determine whether light alcohol drinking increases the risk of cancer by using a meta-analysis of cohort studies because the newly revised 2015 European Code against Cancer fourth edition on alcohol and cancer was based on critical flaws in the interpretation and citation of the previous meta-analyses.

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RESULTS: A total of 60 cohort studies from 135 articles were included in the final analysis. Very light drinking or light drinking was not associated with the incidence of most cancers except for female breast cancer in women and male colorectal cancer. Conversely, light drinking was associated with a decreased incidence of both female and male lung cancer significantly and both female and male thyroid cancer marginally significantly. Moderate drinking significantly increased the incidence of male colorectal cancer and female breast cancer, whereas it decreased the incidence of both female and male hematologic malignancy.

CONCLUSION: We found that very light or light alcohol drinking was not associated with the risk of most of the common cancers except for the mild increase in the incidence of breast cancer in women and colorectal cancer in men.

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Alcohol and Endometrial Cancer
Results suggest that only alcohol consumption equivalent to 2 or more drinks per day increases risk of endometrial cancer in postmenopausal women. Endometrial cancer is the most common gynaecological cancer in the United States and Europe.

Setiawan, V.W., Monroe, K.R., Goodman, M.T., Kolonel, L.N., Pike, M.C. & Henderson, B.E. 2008.
"The role of alcohol intake in the etiology of endometrial cancer is unclear. We examined the impact of alcohol intake on endometrial cancer risk among 41,574 postmenopausal African-American, Japanese-American, Latina, Native-Hawaiian and White women recruited to the prospective Multiethnic Cohort Study in 1993–1996. During an average of 8.3 years of follow-up, 324 incident invasive endometrial cancer cases were identified among these women. Data on alcohol intake and endometrial cancer risk factors were obtained from the baseline questionnaire. Relative risks (RRs) and 95% confidence intervals (CIs) for endometrial cancer associated with alcohol intake were estimated using log-linear (Cox) proportional hazard models stratified by age, year of recruitment, ethnicity and study center, and adjusted for several confounding factors. Increased alcohol consumption was associated with increased risk (p trend = 0.013). Compared to non-drinkers, women consuming ≥2 drinks/day had a multivariate RR of 2.01 (95% CI: 1.30, 3.11). There was no increase in risk associated with <1 drink/day (RR = 1.01; 95% CI: 0.77, 1.33) and 1 to <2 drinks/day (RR = 1.09; 95% CI: 0.62, 1.93). There was no clear effect modification by body mass index, postmenopausal hormone use, parity, oral contraceptive use or smoking status, though our power to detect such interactions was limited. Our results suggest that only alcohol consumption equivalent to 2 or more drinks per day increases risk of endometrial cancer in postmenopausal women."

Cancer and Prostate Cancer
Compared with non-drinkers, men who consumed at least 7 drinks per week during adolescence (ages 15–19) had 3 times the odds of being diagnosed with clinically significant prostate cancer.

Michael, J., Howard, L.E., Markt, S.C., De Hoedt, A., Baily, C. Mucci, L.A., Freedland, S.J. & Allott, E. 2018.
"Epidemiologic evidence for an association between alcohol and prostate cancer is mixed. Moreover, there is a lack of research investigating early-life alcohol intake as a risk factor for either overall or high-grade prostate cancer. We examined lifetime alcohol intake in association with prostate cancer diagnosis in an equal-access, racially diverse prostate biopsy cohort. Men undergoing prostate biopsy at the Durham Veterans Affairs Medical Center from 2007 to 2018 completed a survey indicating average number of alcoholic beverages consumed per week [categorized as none (ref), 1–6, ≥7] during each decade of life. Multivariable logistic regression was used to test the association between alcohol intake across decades and diagnosis of overall, low-grade [grade group (GG) 1–2] and high-grade prostate cancer (GG 3–5). Of 650 men ages 49–89 who underwent biopsy, 325 were diagnosed with prostate cancer, 238 with low-grade and 88 with high-grade disease. Relative to nondrinkers, men who consumed ≥7 drinks/week at ages 15 to 19 had increased odds of high-grade prostate cancer diagnosis (OR = 3.21, P trend = 0.020), with similar findings for ages 20 to 29, 30 to 39, and 40 to 49. Consistent with these results, men in the upper tertile of cumulative lifetime intake had increased odds of high-grade prostate cancer diagnosis (OR = 3.20, P trend = 0.003). In contrast, current alcohol intake was not associated with prostate cancer. In

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conclusion, among men undergoing prostate biopsy, heavier alcohol intake earlier in life and higher cumulative lifetime intake were positively associated with high-grade prostate cancer diagnosis, while current intake was unrelated to prostate cancer. Our findings suggest that earlier-life alcohol intake should be explored as a potential risk factor for high-grade prostate cancer."

Dermoury, C., Karakiewicz, P. & Parent, M-E. 2016. **Background:** Alcohol intake may increase the risk of prostate cancer (PCa). Many previous studies harbored important methodological limitations.
Methods: We conducted a population-based case-control study of PCa comprising 1933 cases and 1994 controls in Montreal, Canada. Lifetime alcohol consumption was elicited, by type of beverage, during in-person interviews. Odds ratios (OR) and 95% confidence intervals (CI) assessed the association between alcohol intake and PCa risk, adjusting for potential confounders and considering the subjects' PCa screening history.
Results: We observed a weak, non-significant positive association between high consumption of total alcohol over the lifetime and risk of high-grade PCa (OR = 1.18, 95% CI 0.81–1.73). Risk estimates were more pronounced among current drinkers (OR = 1.40, 95%CI 1.00–1.97), particularly after adjusting for the timing of last PCa screening (OR = 1.52, 95%CI 1.07–2.16). These associations were largely driven by beer consumption. The OR for high-grade PCa associated with high beer intake was 1.37 (95%CI 1.00–1.89); it was 1.49 (95%CI 0.99–2.23) among current drinkers and 1.68 (95% CI 1.10–2.57) after adjusting for screening recency. High cumulative consumption of spirits was associated with a lower risk of low-grade PCa (OR = 0.75, 95%CI 0.60–0.94) but the risk estimate no longer achieved statistical significance when restricting to current users. No association was found for wine consumption.
Conclusion: Findings add to the accumulating evidence that high alcohol consumption increases the risk of high-grade PCa. This association largely reflected beer intake in our population, and was strengthened when taking into account PCa screening history.

CANSA's Position on Alcohol:

According to the World Health Organization's International Agency for Research on Cancer (IARC) alcoholic beverages are carcinogenic to humans (Group 1). Acetaldehyde, the breakdown product of alcohol, is also carcinogenic to humans (Group 1).

CANSA supports the evidence that alcohol use is a proven carcinogen (cause of many cancers).

CANSA believes that any level of alcohol consumption increases the risk of developing an alcohol-related cancer.

CANSA further believes that when it comes to cancer, no amount of alcohol consumption is safe. Furthermore, there is sufficient scientific evidence that the level of cancer risk increases in line with the level of alcohol consumption.

CANSA, therefore, advocates against the consumption of alcohol in any form, whether it be beer, wine, or distilled spirits.

Medical Disclaimer

This Fact Sheet is intended to provide general information only and, as such, should not be considered as a substitute for advice, medically or otherwise, covering any specific situation. Users should seek appropriate advice before taking or refraining from taking any action in reliance on any information contained in this Fact Sheet. So far as permissible by law, the Cancer Association of South Africa (CANSA) does not accept any liability to any person (or his/her dependants/ estate/heirs) relating to the use of any information contained in this Fact Sheet.

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Sources and References Consulted or Utilised

Alcohol
http://trendsupdates.com/tobacco-alcohol-cigarette-brands-allowed-to-advertise-their-brand-extensions/
American Cancer Society
http://www.cancer.org/cancer/cancercauses/othercarcinogens/generalinformationaboutcarcinogens/known-and-probable-human-carcinogens

Andersson, T.M., Engholm, G., Pukkala, E., Stenbeck, M., Tryggvadottir, L., Storm, H. & Weiderpass, E. 2018. Avoidable cancers in the Nordic countries – the impact of alcohol consumption. *Eur J Cancer*. 2018 Nov;103:299-307. doi: 10.1016/j.ejca.2018.03.027. Epub 2018 May 5.

Asombang, A.W., Chishinga, N., Nkhoma, A., Chipaila, J., Nsokolo, B., Manda-Mapalo, M., Montiero, J.F.G., Banda, L., Dua, K.S. 2019. Systematic review and meta-analysis of esophageal cancer in Africa: epidemiology, risk factors, management and outcomes. *World J Gastroenterol*. 2019 Aug 21;25(31):4512-4533. doi: 10.3748/wjg.v25.i31.4512.

Australian Institute of Health and Welfare. Australasian Association of Cancer Registries. Cancer in Australia: an overview, 2012. Canberra: AIHW; 2012. Report No.: Cancer series no. 74. Cat. no. CAN 70.

Bagnardi, V., Rota, M., Bottyeri, E., et al. 2013. Light alcohol drinking and cancer: a meta-analysis. Ann Oncol. 2013; 24:301-308.

BreastCancer.Org
http://www.breastcancer.org/risk/factors/alcohol

Burton, R. & Sheron, N. 2018. No level of alcohol consumption improves health. *The Lancet*, Vol 392, Issue 10153:987-988. http://www.thelancet.com/article/50140-6736(18)31571-X/fulltext

Cancer Council Australia
http://wiki.cancer.org.au/policy/Position_statement_-_Alcohol_and_cancer

Cancer Council Victoria
http://www.cancervic.org.au/preventing-cancer/avoid-alcohol

Cancer Research UK
http://www.cancerresearchuk.org/health-professional/cancer-statistics/statistics-by-cancer-type/oesophageal-cancer/risk-factors#heading-Zero

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http://www.cancerresearchuk.org/about-cancer/causes-of-cancer/alcohol-and-cancer/how-alcohol-causes-cancer
http://www.cancerresearchuk.org/about-us/cancer-news/news-report/2009-11-04-iarc-finds-more-cancers-linked-to-tobacco-and-alcohol?_ga=1.146191101.897212213.1483511483
http://scienceblog.cancerresearchuk.org/2010/01/28/alcohol-and-cancer-the-evidence/

Choi, Y.J., Myung, S.K. & Lee, J.H. 2018. Light alcohol drinking and risk of cancer: a meta-analysis of cohort studies. Cancer Res Treat. 2018 Apr;50(2):474-487. doi: 10.4143/crt.2017.094. Epub 2017 May 22.

Dermoury, C., Karakiewicz, P. & Parent, M-E. 2016. Association between lifetime alcohol consumption and prostate cancer risk: a case-control study in Montreal, Canada. *Cancer Epidemiology*. Volume 45, December 2016. 11-17.

Fonseca, A.L.A., Prosdocimi, F., Bianco, B., Perez, M.M., Fonseca, F.L.A., & da Costa Agular Alves, B. 2019. Involvement of repair genes in oral cancer: a systematic review. *Cell Biochem Funct*. 2019 Sep 9. doi: 10.1002/cbf.3428. [Epub ahead of print]

Guénel, P., Cyr, D., Sabroe, S., Lynge, E., Merletti, F., Ahrens, W., Baumgardt-Elms, C., Ménégoz, F., Olsson, H., Paulsen, S., Simonato, L. & Wingren, G. 2004. Alcohol drinking may increase risk of breast cancer in men: a European population-based case-control study. *Cancer Causes Control*, 2004. August, 15(6):571-80.

IARC
http://monographs.iarc.fr/ENG/Monographs/vol96/mono96-6E.pdf

IARC Monograph 71, 1999
http://monographs.iarc.fr/ENG/Publications/techrep42/TR42-12.pdf

IARC Working Group
IARC Monogr Eval Carcinog Risks Hum 1988:44:1-378.

IOGT International
http://iogt.org/news/2015/09/15/study-pinkwashed-alcohol-products-promotions-exposed/

Kawakita, D. & Matsuo, K. 2017. Alcohol and head and neck cancer. Cancer Metastasis Rev. 2017 Sep;36(3):425-434. doi: 10.1007/s10555-017-9690-0.

Lin, H.Y., Fisher, P., Harris, D. & Tseng, T.S. 2019. Alcohol intake patterns for cancer and non-cancer individuals: a population study. *Transl Cancer Res*. 2019 Jan;8(Suppl 4):S334-S345. doi: 10.21037/tcr.2019.06.31.

Liu, Y., Nguyen, N. & Colditz, G.A. 2015. Links between alcohol consumption and breast cancer: a look at the evidence. *Womens Health (Lond).* 2015 Jan;11(1):65-77. doi: 10.2217/whe.14.62.

Living Science
http://www.livescience.com/55497-does-alcohol-cause-cancer.html

MacMillan Cancer Support
http://www.macmillan.org.uk/Cancerinformation/Cancertypes/Larynx/Aboutlaryngealcancer/Causes.aspx

Marczinski, C.A. & Fillmore, M.T. 2014. Energy drinks mixed with alcohol: what are the risks? *Nutr Rev.* 2014 Oct;72 Suppl 1:98-107. doi: 10.1111/nure.12127.

Mart, S. & Giesbrcht, N. 2015. Red flags on pinkwashed drinks: contradictions and dangers in marketing alcohol to prevent cancer. Addiction, Oct 2015: 110(10):1541-8. DOI: 10.1111/add.13035.

Matejcic, M., Gunther, M.J. & Ferrari, P. 2017. Alcohol metabolism and oesophageal cancer: a systematic review of the evidence. *Carcinogenesis*. 2017 Sep 1;38(9):859-872. doi: 10.1093/carcin/bgx067.

Michael, J., Howard, L.E., Markt, S.C., De Hoedt, A., Baily, C. Mucci, L.A., Freedland, S.J. & Allott, E. 2018. Early-life alcohol intake and high-grade prostate cancer: results from an equal-access, racially diverse biopsy cohort. *Cancer Prev Res; 1–8.* ©2018 AACR.

MS Magazine

http://msmagazine.com/blog/2012/10/08/what-the-pink-boozing-for-boobs/

National Institute on Alcohol Abuse and Alcoholism
http://pubs.niaaa.nih.gov/publications/aa21.htm
Ogden, G.G. 2018. Alcohol and mouth cancer. *Br Dent J*. 2018 Nov 9;225(9):880-883. doi: 10.1038/sj.bdj.2018.921.

Oze, I., Charvat, H., Matsuo, K., Ito, H., Tamakoshi, A., Nagata, C., Wada, K., Sugawara, Y., Sawada, N., Yamaji, T., Naito, M., Tanaka, K., Shimazu, T., Mizoue, T., Tsugane, S. & Inoue, M. 2019. Revisit of an unanswered question by pooled analysis of eight cohort studies in Japan: does cigarette smoking and alcohol drinking have interaction for the risk of oesophageal cancer? *Cancer Med*. 2019 Sep 1. doi: 10.1002/cam4.2514. [Epub ahead of print]

Parkin, D.M. 2011. Cancers attributable to consumption of alcohol in the UK in 2010. *Br J Cancer* 2011 Dec 6;105 Suppl 2:S14-8
http://www.ncbi.nlm.nih.gov/pubmed/22158312].

Rogha, M., Berjis, N., Laievardi, S.M., Alamdaran, M. & Hashemi, S.M. 2019. Ident6ification of R249 Mutation in P53 gene in tumoral tissue of tongue cancer. *Int J Prev Med*. 2019 Aug 1;10:129. doi: 10.4103/ijpvm.IJPVM_50_17. eCollection 2019.

Schütze, M., Boeing, H., Pischon, T., Rehm, J., Kehoe, T., Gmel, G., et al. 2011. Alcohol attributable burden of incidence of cancer in eight European countries based on results from prospective cohort study. BMJ 2011 Apr 7;342:d1584
http://www.ncbi.nlm.nih.gov/pubmed/21474525].

Setiawan, V.W., Monroe, K.R., Goodman, M.T., Kolonel, L.N., Pike, M.C. & Henderson, B.E. 2008. Alcohol consumption and endometrial cancer risk: the multiethinic cohort. Int J Cancer.. Author manuscript; available in PMC 2009 Apr 10.

Su, L.J. & Arab, L. 2004. Alcohol consumption and risk of colon cancer: evidence from the national health and nutrition examination survey I epidemiologic follow-up study. *Nutr Cancer*. 50(2):111-9.

The Oral Cancer Foundation
http://www.oralcancerfoundation.org/understanding/alcohol-connection.php

Tsai, H.J. & Chang, J.S. 2019. Environmental risk factors and pancreatic cancer. *J Clin Med*. 2019 Sep 10;8(9). pii: E1427. doi: 10.3390/jcm8091427.

Wang, J., Zhang, X., Beck, A.H., Collins, L.C., Chen, W.Y., Tamimi, R.M., Hazra, A., Brown, M., Rosner, B. & Hankinson, S.E. 2015. Alcohol consumption and risk of breast cancer by tumor receptor expression. Horm Cancer. 2015, Sep 18. (Epub ahead of Print).
http://www.ncbi.nlm.nih.gov/pubmed/26385458

WebMD
http://www.webmd.com/cancer/understanding-liver-cancer-basic-information

World Cancer Research Fund, American Institute for Cancer Research. 1009. Policy and action for cancer prevention. Food, nutrition, and physical activity: a global perspective. Washington DC: AICR; 2009.
http://www.dietandcancerreport.org/cancer_resource_center/downloads/chapters/pr/Introductory%20pages.pdf.

World Cancer Research Fund International
http://www.wcrf.org/int/blog/articles/2016/03/how-does-alcohol-increase-cancer-risk

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