Cancer Risk Factors in Ontario

Alcoholic Drinks
Cancer Risk Factors in Ontario: Evidence Summary

ALCOHOLIC DRINKS

<table>
<thead>
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<th>Risk factor/exposure</th>
<th>Cancer</th>
<th>Direction of association</th>
<th>Magnitude of risk*</th>
<th>Strength of evidencea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol consumption</td>
<td>Oral cavity and pharynx</td>
<td>↑</td>
<td>1.75–1.86bc</td>
<td>Sufficient</td>
</tr>
<tr>
<td></td>
<td>Esophagus†</td>
<td>↑</td>
<td>1.4–1.5bc</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Colon and rectum</td>
<td>↑</td>
<td>1.18–1.20</td>
<td></td>
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<tr>
<td></td>
<td>Larynx</td>
<td>↑</td>
<td>1.38–1.43bc</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Breast</td>
<td>↑</td>
<td>1.25–1.33bc</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Liver</td>
<td>↑</td>
<td>1.18–1.19bc</td>
<td></td>
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<tr>
<td></td>
<td>Pancreas</td>
<td>↑</td>
<td>...</td>
<td>Limited</td>
</tr>
</tbody>
</table>

Sources: aIARC, 2012; bBagnardi et al., 2001; cCorrao et al., 2004; dFedirko et al., 2011

* Relative risk (RR) estimate comparing 25 g/day (approximately two drinks) of alcohol intake to non-drinking.
. . . Magnitude of risk not shown in table if strength of evidence is “probable” or “limited.”
† Association is primarily restricted to squamous cell carcinoma. The association, if any, with adenocarcinoma is weak.

Background

- Alcoholic beverages contain ethanol (commonly referred to as alcohol) produced through the fermentation of sugars by yeasts.24
- Beers, wines and spirits are the most common alcoholic drinks commercially produced; other alcoholic beverages, such as fermented milks, fermented honey-water (mead) and fermented apples (cider), may be particularly important in some populations or geographic areas.24
- Alcohol content differs among types of alcoholic drinks, usually ranging from 3%–7% in beers, 9%–15% in wines and 35%–50% in spirits or liquors.24
- Standard serving sizes for alcoholic beverages vary among countries but one drink usually contains 12–15 g of alcohol.25 In Canada, one standard drink is usually defined as: a 341 ml or 12 oz bottle of regular strength beer (5%), a 142 ml or 5 oz glass of wine (12%), or a 43 ml or 1.5 oz shot of distilled liquor.26

- Alcoholic beverages have been classified by the International Agency for Research on Cancer (IARC) as carcinogenic to humans (Group 1), causing cancers of the oral cavity, pharynx, larynx, esophagus (primarily squamous cell carcinoma), colon and rectum, female breast, and liver.1,24 There is limited evidence that alcohol consumption may also cause cancer of the pancreas.1
- Meta-analyses have estimated that compared to non-drinkers, people who drink 25 g (~ 2 drinks) of alcohol/day have a 75%–86% increased risk of cancers of the oral cavity and pharynx, a 40%–50% increased risk of esophageal cancer, a roughly 40% increased risk of laryngeal cancer, a 25%–31% increased risk of breast cancer, and a roughly 20% greater risk of colorectal cancer.27,28 The risk of developing these cancers increases substantially with alcohol intake equivalent to 4 or more drinks/day.29–34
The risk of liver cancer is estimated as 18%–19% higher in people who drink 25 g/day of alcohol compared to non-drinkers; however, these estimates should be interpreted cautiously because alcohol-related liver cancers generally follow cirrhosis, which often leads to a reduction of alcohol consumption. For this reason, a large expert panel report by the World Cancer Research Fund (WCRF) and the American Institute for Cancer Research (AICR) acknowledges that alcohol is causally related to cirrhosis of the liver but has classified the evidence for an association between alcohol consumption and liver cancer as “probable.”

Despite a protective effect of light to moderate alcohol consumption for other chronic diseases, such as cardiovascular disease, no clear “safe limit” of alcohol intake to prevent an increased risk of cancer has been determined.

A dose-response relationship exists between alcohol consumption and cancer risk. For most cancers, the increase in risk is continuous and is apparent even at low levels of intake. For example, each 10 g/day (< 1 drink) increase in alcohol consumption is associated with a 21% increase in oral cavity and pharyngeal cancer risk and a 7%–10% increase in female breast cancer risk. For colorectal cancer, a dose-response is evident, but it is unclear whether there is a lower threshold below which no increased risk of colorectal cancer is observed.

Increased cancer risk exists regardless of the type of alcoholic drink consumed, suggesting that the risk is due to ethanol, another IARC Group 1 carcinogen. The effects of duration and cessation of the consumption of alcoholic beverages and the lifetime period of exposure on cancer risk remain uncertain.

Alcohol consumption interacts synergistically with tobacco smoking to influence the risk of some cancers, particularly of the oral cavity, pharynx, larynx and esophagus (see tobacco section on page 7).

Susceptibility to alcohol-related cancers may be higher among individuals with certain functional variants in the genes involved in alcohol metabolism, including those that encode the major alcohol-metabolizing enzymes, alcohol dehydrogenases and aldehyde dehydrogenases (ALDH). The variant allele ALDH*2 is prevalent in Asian populations and has been shown to increase the risk of upper aerodigestive tract cancers in moderate and heavy drinkers.

Evidence suggests several ways that alcohol may increase cancer risk, including:

- Reactive metabolites of alcohol may be carcinogenic. Acetaldehyde has been identified as a carcinogen by IARC and has been shown to form DNA adducts. The resulting genetic damage may lead to increased proliferation of tumour cells.
- Alcohol may act as a solvent, allowing other carcinogens to penetrate cells more easily. This may contribute to the observed synergistic effect between alcohol and tobacco smoking.
- The production of prostaglandins, lipid peroxidation and the generation of free radical oxygen through the metabolism of alcohol may mediate the effects of alcohol.
- In the case of breast cancer, evidence suggests that the carcinogenic effect is due to increased estrogen production in response to alcohol consumption.
- The diets of people who are heavy alcohol consumers may be lacking essential nutrients, which may make body tissues more susceptible to carcinogenesis.