

## Alcohol Consumption and the Gastrointestinal Tract

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### Abstract

Alcohol is one of the most commonly abused drugs, with a per capita consumption of approximately 10 L pure ethanol per year in the United States and even higher in Spain and France. In terms of mortality, the effect of alcohol on the liver and the pancreas is probably more significant than on the tubular gastrointestinal tract. However, alcohol is a very important cause of morbidity in the tubular gastrointestinal tract. Alcohol influences the motility in the esophagus, stomach and small bowel and has direct effects on the mucosa of the upper tract. While the stimulation of gastric acid secretion is inversely correlated with the alcohol concentration of the beverage, a direct pathogenetic role in peptic ulcer disease has not been demonstrated. Some alcohols, like red wine, have been shown to possess an anti-*Helicobacter pylori* effect. Alcohol also has a role in the development of tumors of the gastrointestinal tract.

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### Alcohol and the esophagus

The development of esophageal varices secondary to liver disease and portal hypertension is the most serious *indirect* effect of alcohol on the esophagus. Less serious but more frequent are the *direct* effects of alcohol on the esophagus. It is noteworthy that alcohol has a different pathophysiologic action in the lower esophageal sphincter depending on whether the ingestion is acute (as in the occasional drinker) or chronic (as in alcoholics). It was found that small [1,2] and large [3] quantities of alcohol in healthy volunteers had a direct effect on the sphincter by reducing its resting pressure; furthermore, alcohol reduces the motility in the esophagus itself. These facts are of clinical relevance since gastric contents that have regurgitated as a consequence of the reduced pressure in the lower sphincter will remain in the body of the esophagus for longer periods. The result will be an increased sensation of heartburn and damage to the esophageal mucosa with the consequent development of esophagitis. By contrast, in chronic alcoholics the pressure on the lower esophageal sphincter is increased. However, since spontaneous sphincter relaxations are frequent in alcoholics, the result is increased gastroesophageal reflux [4]. Alcohol also damages the esophageal mucosa directly, facilitating hydrogen ion penetration [5]. Interestingly, with the gastroesophageal reflux, esophageal mucosal damage is produced regardless of the type of alcoholic beverage ingested (wine, beer or liquor) [6]. Superficial tears of the mucosa at the lower part of the esophagus (Mallory-Weiss syndrome) due to vomiting after an alcoholic binge are not rare in alcoholic patients [7].

### Alcohol and the stomach

#### Acid secretion

Intravenous, oral, and intragastric alcohol at a concentration of up to 5% increases acid secretion principally by stimulating the secretion of gastrin and to a lesser extent by a direct effect on the parietal cells [8,9]. By contrast, an alcohol concentration of higher than 5% has no effect on gastric acid secretion. An intriguing fact is that the process of distillation of beer, white or red wine, liquors and champagne causes a loss of their stimulating properties [10]. This capacity of producing gastric acid stimulation has been attributed to the presence of maleic and succinic acids, which are found in non-distilled beer and wine [11].

#### Gastritis and peptic disease

The effects of alcohol on the gastric mucosa are dose-dependent, and the damage appears as early as 30 minutes after ingestion and reaches a peak at about 60 minutes [12]. The mucosal changes range from subtle erythema to hemorrhagic gastritis. Gastroscopy performed 24 hours after ingestion revealed that only whiskey or another alcohol solution with a concentration of over 10% caused some degree of mucosal hemorrhagic damage. It has been speculated that alcoholic beverages probably contain some protective substances [12] and that low alcohol concentration of beverages like beer and wine possess more of these substances than whiskey or brandy. In contrast to the gastric mucosa, the duodenum is apparently protected from the damage of alcohol, and no changes are visible macroscopically or by light microscopy. However, under the electron microscope, some degree of fibrosis of the villi can be observed [13]. Although both superficial and chronic gastritis are common in alcohol-abusing individuals, surprisingly, alcohol has been incriminated neither in the etiology of gastroduodenal peptic ulcer nor in impairing the healing of established ulcers [2].

### Alcohol and motility

#### Gastric motility

The effects of ethanol on gastric emptying are probably dependent on the beverage: a low alcohol dose (wine and beer) seems to induce gastric motility whereas higher alcohol concentrations delay gastric emptying [14]. The latter may produce a sensation of epigastric fullness or even nausea.

#### Bowel motility

Small bowel motility is reduced in alcoholic patients probably due to toxic effects on the vagus and by a direct effect upon the muscular layer of the intestine [15].

## Alcohol and gastrointestinal infections

Wine has long been known for its disinfecting and cleansing properties. According to historical sources, wine was used during the Prussian War to prevent dysentery. White and red wine have been shown to have bactericidal effects against *Salmonella enteritidis* and *Shigella sonnei* [16]. A large proportion of this activity was found to be due to the acid pH and the alcohol content in wine [17].

Red wine has been found to exert a marked bactericidal effect on *Helicobacter pylori* that is stronger than of an HCl solution at pH 3.5. Nevertheless, the acid pH of wine and its alcohol concentration were not enough to explain its bactericidal effect [18]. Substances other than alcohol itself have not been identified, and factors such as the increase in acid secretion and motility probably play a role in the bactericidal effect [19]. Clinical studies [20] have shown that a high consumption of alcohol is inversely related to the presence of *H. pylori* and is seen more often in association with wine than with beer.

## Alcohol and food absorption

A decrease in the activity of small intestinal disaccharidase and an increase in the permeability of the mucosa [21] may explain the increased frequency of diarrhea in alcoholic patients. Steatorrhea is less frequently observed and may be attributed to some degree of pancreatic insufficiency [22]. The absorption of several nutrients may be altered in patients with chronic alcohol abuse, such as vitamin B12, folic acid, thiamin, amino acids (leucine, phenylalanine, glycine, methionine, etc), calcium, and magnesium.

## Alcohol and gastrointestinal tumors

Alcohol consumption has been linked to increased risk of tumors in the pharynx, esophagus, stomach and colon. Some of the factors associated with alcohol consumption that may favor the development of tumors include: generation of free radicals (peroxide, superoxide) and other oxidizing factors [23], inorganic arsenic, preservatives, additives, alterations in hormonal balance, and depletion of vitamin deposits [24]. This risk is increased by tobacco smoking [25] in direct relation to the number and type of alcoholic beverages consumed, with wine increasing this risk less than other types of alcohol [26]. Drinkers of more than 21 beers or liquors a week had a relative risk of 5.2 (the relative risk of abstemious being 1), whereas subjects who drunk wine had a relative risk of 1.7. It seems that moderate wine consumption probably does not increase the risk of developing esophageal and oropharyngeal cancer. Conversely, moderate liquor or beer consumption does increase the frequency of such tumors [26]. Many studies have established that antioxidants in fruits and vegetables reduce the risk of tumor development [27]. Alcoholic beverages contain polyphenols (like rutin and resveratrol), which exert antioxidant activity [28]. Indeed, some studies have found that not only does moderate wine consumption, as compared to beer and/or liquor, not increase the risk of esophageal and oropharyngeal cancer, but it even protects against the development of esophageal and gastric tumors [29,30].

Alcohol consumption in a moderate quantity (up to 15 g/alcohol/day) has been related to an increased risk of carcinoma and adenoma of the colon and rectum [31,32], but these results have

not been unanimously confirmed. A lack of increased risk has also been described [33].

## Conclusions

Alcohol is a frequently abused drug and an important cause of morbidity and mortality. In addition to its effects on the tubular gastrointestinal tract, alcohol affects the physiologic motor action of the esophagus, stomach and intestine. In low concentrations it has a direct stimulating effect on gastric acid secretion, although contrary to conventional wisdom, it is not considered to have a pathogenetic influence on the development of peptic disease. Alcohol has a bactericidal effect on *Salmonella*, *shigella*, and *H. pylori*. It also has a tumorigenic role.

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