NUTRITIONAL AND HEALTH BENEFITS OF BEER

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Internal Medicine Grand Rounds

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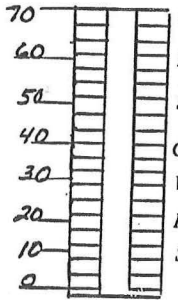
A MORAL AND PHYSICAL THERMOMETER

A scale of the progress of Temperance and intemperance—Liquors with effects in their usual order.

TEMPERANCE
Health and Wealth.



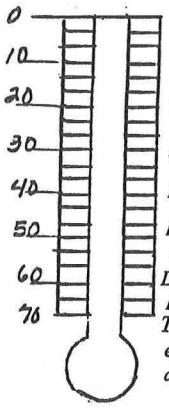
Water,
Milk and Water,
Small Beer,

} Serenity of Mind, Reputation, Long Life and Happiness

Cider and Perry,
Wine,
Porter,
Strong Beer,

} Cheerfulness, Strength, and Nourishment, when taken only in small quantities, and at meals

INTEMPERANCE.



Punch,
Toddy and Egg Rum,
Grog-Brandy and Water,
Flip and Shrub,
Bitters infused in Spirits and Cordials.
Drams of Gin, Brandy, and Rum, in the morning,
The same morning and evening, The same during day & night.

Vices	Diseases	Punishments
Idleness, Gaming, peevishness quarrelling Fighting, Horse-Racing, Lying and Swearing, Stealing & Swindling Perjury, Burglary, Murder,	Sickness, Tremors of the hands in the morning, puking, bloatedness, Inflamed eyes, red nose and face, Sore and swelled legs, jaundice, Pains in the hands, burning in the hands, and feet Dropsy, Epilepsy, Melancholy, palsy, apoplexy, Madness, Despair	Debt. Jail Black eyes, and Rags, Hospital or Poor house. Bridewell. State prison do. for Life Gallows.

Reproduced from pages 2 and 3 of Benjamin Rush's *An Inquiry Into the Effects of Ardent Spirits Upon the Human Body and Mind*, the eight edition.

reproduced from reference 53

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Interests: Nutrition in the pathogenesis and treatment of disease

The benefits of moderate alcohol consumption have not been generally endorsed by physicians[1]. Enthusiasm for promoting moderate alcohol to prevent disease is necessarily tethered by the consequences of excessive ingestion[2]. While acknowledging the double-edged sword of recommending alcohol ingestion for the population at large, this medicine grand rounds will focus on the nutritional and health benefits associated with moderate consumption of alcohol as part of a healthy lifestyle. Physicians and health care professionals seeking continuing medical education regarding the physician's role in diagnosing, treating and preventing alcohol-related disorders can attend the annual ***NOVA Conference,*** *which was cosponsored this year by UT Southwestern and the Texas Association of Addiction Professionals. The* ***Greater Dallas Council on Alcohol and Drug Abuse*** *(214) 522-0300 is an excellent resource for healthcare professionals and the public at large concerning alcohol use and abuse.*

The production of alcohol has a long and rich history that is interwoven with the history of man. This medicine grand rounds will explore the history of alcohol and health, focusing on beer, the oldest alcoholic beverage of record.

Alcoholic Beverages: Where did they come from?

Alcoholic beverages are ancient beverages created accidentally during intentional food storage. Legend suggests yeast from the genus *Saccharomyces* spontaneously colonized a storage jar of food sugars, fermenting them into a sweet, alcoholic beverage.[3]

Grapes are one of the few plants that store carbohydrates as fructose and glucose instead of starch and pectins. The marriage of yeast to grapes was easily arranged[4]. *Saccharomyces* lives off the sap of oak trees and colonizes bark. *Saccharomyces* co-harvested with grapes growing on oak-supported vines could easily ferment mature grapes when they ruptured. The natural acidity of ethanol produced by *Saccharomyces* combined with the tartaric acid from grapes limited the growth of other yeast and bacteria. Wine as a beverage was born. Wine making is depicted in Amanemhat's Tomb, circa 1400 BC. Improvements in the process (e.g., cultivating higher sugar varieties of grapes, fermentation in closed vessels, addition of sulfur dioxide as a bactericidal agent as well as an antioxidant, aging in oak casks etc.) were soon to follow.

Although the prolific use of wine by the Greeks and Romans leads many people to assume that wine is our only ancient alcoholic beverage, beer is an even older beverage[5]. Beer manufacture requires the cultivation of cereal grains, the most common one being barley. Unlike grapes that are suitable to grow in only a narrow

latitude range, barley is a native grass common throughout most of Europe, the Americas, Asia and Africa. Barley has a short growing season (~90 days) and can grow in cool, moist climates as well as dry, hot climates. Barley cultivation began 8,000 years ago in Egypt and 5,000 years ago in northwestern Europe, becoming a staple in the diet. The unleavened bread of the Hebrews, Greeks, and Romans was made from barley flour.

In contrast to grapes, where a glucose substrate is readily available, barley cannot be made into an alcoholic beverage without pretreatment of the grain to release simple sugars from carbohydrates. Release of simple sugars occurs naturally during germination, and it is likely that the discovery of beer occurred when barley seeds in a storage vessel were drowned by a rain and began to germinate. Not wanting to waste the grain or allow it to spoil, the cook dried out the seeds over a fire. These malted barley seeds were ground for flour and baked into bread that had a pleasing, sweet taste.

The discovery of malt led to another accident. Malted bread stored in a vessel also became wet during a rain. *Saccharomyces* brought by the wind colonized the vessel wall, and began fermenting the sugars present in the bread. A frothy liquor, beer, was born. The aroma and taste was pleasing, and the cook worked on reproducing the accident and controlling conditions to improve taste.[6]

Beer as a mass-produced beverage has roots in Mesopotamia where barley was grown in irrigated fields. The Egyptians believed that beer was a gift from the gods. Ibis, the deity of nature, brought the blessing. Hather invented brewing. Menqet was "the goddess who makes beer". Perhaps the ability of the foamy product to "multiply" from the storage vessel during fermentation contributed to beer's mystique. In the temple of Dendra, 2400 BC, multi-step production of beer is documented. An Egyptian brewing recipe was outlined by the chemist Zosimus. Roman historians Pliny and Tacitus reported beer production and consumption among Saxons, Celts, Nordic and Germanic tribes.

The adaptability of beer for travel and trade was unique. Malt cakes could be carried to a distant location and reconstituted. Competition for tasty beer led to nearly infinite variations in production technique. Although beer manufacture was commonplace, the fermentation process was not well understood until the careful, methodical work of Louis Pasteur. In *Études sur la Bière,* Pasteur[7] documented that exposure to air (source of *Saccharomyces*) was necessary for the fermentation process. Germans introduced hops into beer during the first century, but this practice was not broadly adopted until the 16th century. The biochemical steps as we know them today are:

1. **Formation of green malt:** When barley grains are allowed to germinate, the root embryo of the barleycorn secretes gibberellic acid, a plant hormone. Gibberellic acid initiates the synthesis of α-amylase. β-amylase, already present in the barley, works with α-amylase to convert starches in the grain to maltose. Other enzymes including proteases and β-gluconases, convert insoluble proteins and starches into amino acids and glucose.

2. **Roasting malt:** The green malt is dried to reduce moisture content and arrest enzyme activity. Approximately 40-60% of the enzyme potential is preserved. Heating or roasting the malt introduces color and flavor. The kilned malt is pressed into cake. At this point, the cake can be stored or carried to another location.

3. **Preparation of wort:** The malt cake is mixed with water and milled to mechanically break down additional plant material. Mashing the malt allows for the starches released by milling to undergo enzymatic cleavage, forming additional maltose as well as smaller starches and proteins. The milling process does not breakdown the husks, which serve as a natural filter to separate solids from the mashed malt or wort. Wort typically has a sugar concentration of 10%.

4. **Fermentation:** The wort is boiled to destroy remaining enzyme activity. Hops are added, the mixture is heated and cooled so that remaining solids can be removed. *Saccharomyces* is added and fermentation begins. *Saccharomyces* can stabilize the flavor of the brew as well as reduce oxidation potential by converting inorganic sulfate in the barley into sulfite. Elimination or attenuation of the MET10 gene can produce a higher sulfite containing beer.[8] Advances in bioscience have improved brewers' ability to manipulate production.[9]

5. **Consumption/Storage:** Beer is now ready to drink or to be stored in airtight containers. In the US, most beer is pasteurized to reduce chances of bacterial contamination and to stop fermentation. A brief description of the differences in the major varieties of beer is outlined in Table 1.

Table 1 Nomenclature for Types of Beer

Type	Distinguishing features
Ale	Top fermented beer using *Saccharomyces cerevisiae*
Pale Ale	Hard water + pale malt (also called Best Bitter)
Mild Ale	Roasted barley or caramel added + less hops + added cane sugar
Stout	Stronger flavored version of mild ale
Strong Beer	First extraction from single batch of kilned malt
Small beer	Third extraction from single batch of kilned malt
Porter	Mixture of malt extracts + more hops
Pilsner	Soft water + more hops
Bock	Highly roasted malt
Lager	Bottom-fermenting yeast using *S. carlsbergensis.* Lager was produced in the winter and stored ("lagern" = to store)
Weissbier	Wheat, not barley, as sugar source (German)
Burukutu and Kaffir	Sorghum, not barley, as sugar source (Nigeria). Some varieties may also contain maize.
Pulque	Cactus, not barley, as sugar source (Mexico)
Sake	Rice, not barley, as sugar source (Japan)

Taste of Beer and Beer Preference: What are the unique substances responsible?

Many factors contribute to the taste attraction of beer.[10] Since different people have different taste preferences, a consensus on the relative importance of flavor factors in beer cannot always be reached. In an attempt to objectify taste, some chemists have evaluated the aroma of beer and linked the presence of volatile components to strains of yeast and conditions of fermentation.[11,12] A comprehensive flavor wheel (Figure 1) designed by the brewing expert, MC Meilgaard, links the sectors of odor and taste to common flavor descriptors.[13] Each descriptor is assigned a number according to the flavor sector, and the relative potential of a factor to influence taste is rated by a defined unit of flavor. Defining a unit of flavor (Table 2), the flavor in beers can be determined as primary, secondary, and tertiary (Table 3). Ultimately, the flavors of different beers can be compared in a quantitative fashion.

Flavor Wheel

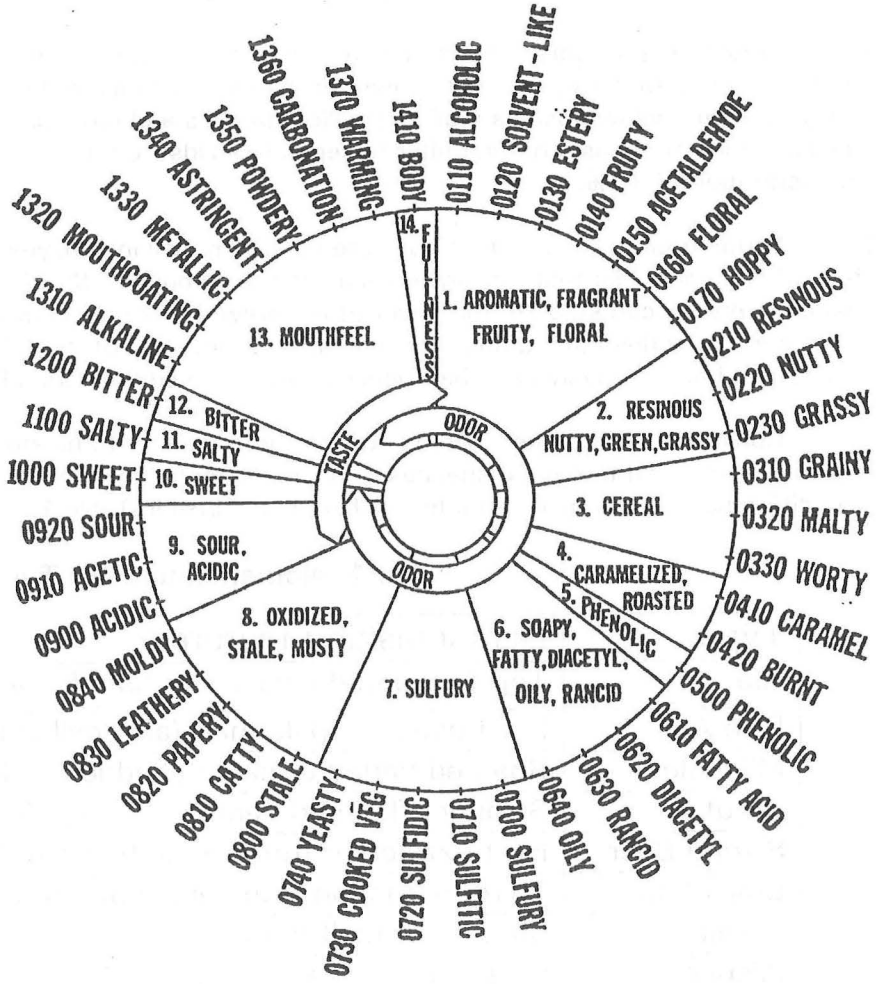

Table 2 PERCEPTION OF DIFFERENCES IN FLAVOR UNIT* (*from reference 13*)

A difference of:	Is Perceived as:
0 - 0.5 FU	Not perceptible
0.5 - 1.0 FU	Perceptible but not identifiable by average taster
1.0 - 2.0 FU	Detectable and identifiable
Over 2 FU	A complete change of flavor

* Flavor Unit = concentration/threshold can be used as a rough measure (±20%) for flavor intensity provided (1) the threshold was determined in a type of beer which varies <50% in composition from that studied; (2) the use is limited to the range from 0.2 FU to 2 FU; and (3) the result is taken as a measure of the difference between a test beer and a control beer.

Table 3. Summary of the Chemical Composition of Beer Flavor (*from Reference 13*)

Flavor Term	Flavor-Active Compound Or Group of Compounds	Typical or Distinctive Member of Group
PRIMARY FLAVOR CONSTITUENTS, PRESENT ABOVE 2 FU		
In typical, Pale Lager Beers:		
1200 Bitter	Hop bitter substances	*trans*-isohumulone
0110 Alcoholic	Ethanol	Ethanol
1360 Carbonation	Carbon dioxide	Carbon disoxide
In Specialty Beers:		
0171 Kettle Hop	Hop oil transformation products	Oxygenated humulenes
0172 Dry Hop	Hop oil constituents	Myrcene, humulene
0410 Caramel	O-heterocylic ketones	Maltol and isomers
0130 Ester	Esters	3-methylbutyl acetate
1000 Sweet	Sugars	Sucrose
SECONDARY FLAVOR CONSTITUENTS, PRESENT BETWEEN 0.5 AND 2 FU		
Compounds Listed Under Specialty Beers above, plus the following:		
0131 Isoamyl acetate	Banana esters	3-Methylbutyl acetate, 2-methylpropyl acetate
0132 Ethyl hexanoate	Apple esters	Ethyl hexanoate, ethyl octanoate
0110 Alcoholic	Fusel alcohols	3-Methylbutanol
0732 DMS	Dialkyl sulfides	Dimethyl sulfide
0611 Caprylic	C-6 to C-12 fatty acids	Octanoic acid
0910 Acetic	Acetic and propanoic acids	Acetic acid
0133 Ethyl acetate	Ethyl acetate	Ethyl acetate
0620 Diacetyl	Vicinal diketones	Diacetyl
0613 Isovaleric	3-Methylbutanoic and 1-pentanoic acids	3-Methylbutanoic acid
0612 Butyric	Butanoic and 2-methylpropanoic acids	Butanoic acid
0920 Sour	Other organic acids	Citric, malic acids
1340 Astringent	Polyphenols	Leucocyanidin
1000 Sweet	Sugars	Maltotriose
1410 Body and other terms	Amino acids, small peptides nucleic acid derivatives	Proline
TERTIARY FLAVOR CONSTITUENTS, PRESENT BETWEEN 0.1 AND 0.5 FU		
Numerous Compounds, such as the following:		
0140 Fruity	Lactones of hydroxy acids	*gamma*-decalactone
0161 2-Phenylethanol	2-Phenylethanol	2-Phenylethanol
0820 Papery	Long-chain aliphatic aldehydes	*trans*-2-nonenal
0500 Phenolic	Volatile phenols	4-vinalguaiacol
0721 H_2S	Hydrogen sulfide	Hydrogen sulfide
0724 Lightstruck	3-Methyl-2-butene thiol, other thiols	3-Methyl-2-butene thiol
1100 Salty	Inorganic salts	Sodium chloride
1330 Metallic	Metal ions	Ferrous ion
BACKGROUND FLAVOR CONSTITUENTS, PRESENT BELOW 0.1 FU		
All Other Flavor Constituents (Probably Thousands)		

Besides flavor, beer has been described as "thirst quenching" and "refreshing." In a comparison of 18 beers, judges rated carbonation and bubble density rather than flavor characteristics responsible for the thirst quenching characteristics of beer.[14]

Nutritional Value

An important definition: one drink

Before addressing the nutritional and health benefits of beer, the unit of consumption must be defined. The USDA defines one drink of alcohol in the US Dietary Guidelines for Health Americans.

US Dietary Guidelines: One Drink

- 12 ounces of regular or light beer
- 5 ounces of wine
- 1.5 ounces of 80 proof distilled spirits (40%) ethanol)

Alcohol content

Alcoholic beverages differ in their alcohol concentration (Table 4). *Saccharomyces* can produce alcohol concentrations up to 16% by volume; higher concentrations of alcohol inhibit fermentation. Distilled spirits were created by Arab alchemists to increase the concentration of alcohol in a beverage, and became widely available 900 years ago.

Table 4. Percent alcohol by volume for common beverages

Beverage	Percent alcohol by volume
Beer	4-5% (maximum by Texas law 5.12%)
Light Beer	Light beer is lower in calories but has a similar alcohol content as regular beer (e.g., Coors beer = 4.86% compared to Coors Light = 4.3%) Calories are reduced by enzymatic treatment converting unfermentable carbohydrates to fermentable sugars.
Low alcohol beer	0.5 – 2.0%; alcohol is removed after fermentation by evaporation or membrane filtration
Alcohol free beer	Less than 0.1%
Malt liquors, Ale, Strong beer, Arctic Ice	> 5.12%. The Texas Alcoholic Beverage Commission does not permit these products to be called "beer"
Wine	8-14%
Wine Cooler	4.5-5%
Distilled Spirits	20-50%

Macronutrients/micronutrients

Alcoholic beverages often contain other macronutrients besides alcohol (Table 5). Barley is a significant source of an incomplete protein that is further modified during fermentation. Enantiomeric purity of the amino acids can be useful in characterizing specific beers by brewing processes.[15] Glutamic acid and proline comprise 16% of the amino acids in beer.

Wine and beer both contain carbohydrates. Oligosaccharides in beer have varying molecular weight and branching patterns.[16]

Wine and beer, and to a lesser extent distilled beverage, contain B vitamins (Table 6).

Alcoholic beverages contain minerals (Table 7). The mineral content of beer varies depending upon the soil where the barley and hops were grown, the temperature and moisture characteristics of the growing season, and the manufacturing practices that increase or decrease mineral concentrations. In a comparison of commercial beers in Spain, the magnesium content varied ten-fold from 9.0 to 82.3 mcg/drink.[17] Similar, broad variations in the cadmium content of alcoholic beverages has been documented (wine =0.01 to 2.31 mcg/drink, beer = undetectable to 0.29 mcg/drink, and spirits = undetectable to 0.52 mcg/drink).[18]

Sorghum and maize beers contain more bioavailable iron than barley beers.[19] Hemochromatosis has been described among beer drinkers in Africa who 1) ferment their own home brew in ungalvanized steel tanks (typically increases Fe concentrations to 29 mg/drink)[20] and 2) have a genetic mutation increasing risk for iron overload.[21]

Alcoholic beverages can contribute to the nutritional value of the diet. In a survey of 940 Italians over age 60,[22] wine was the major source of iron, contributing 20% to overall dietary intake. Wine was the fourth major source of riboflavin, contributing 4% of total dietary intake.

For Americans, the contribution of alcohol to the nutritional content of the diet was evaluated by 3 day food records collected monthly for 6-12 months.[23] Except for calories, alcoholic beverages provided little to diets for patients who consumed < 5% of calories from alcohol. Not surprisingly, in subjects who consumed > 5% of calories from alcoholic beverages, the source of alcohol determined the micronutrient contribution to the diet. For subjects consuming 1-2 beers per day, alcoholic beverages provided 14% of calories, 11% of dietary protein, 12% of dietary carbohydrates, 9% dietary phosphorus, 7% dietary riboflavin, and 5% of dietary niacin. The overall quality of dietary intake among drinkers was no different than non-drinkers. In those subjects who reported drinking and non-drinking days, the calories from alcoholic beverages were "added on" to the overall calories.

The USDA Handbook 8 reports the following values for one drink:

Table 5. Calorie Sources for One Drink of An Alcoholic Beverage

Beverage	Protein	Carbohydrate	Ethanol
Beer	1.00g	13.0g	12.8g
Light Beer	0.71	4.6	11.3
White Wine	0.15	1.2	13.5
Red Wine	0.30	2.5	13.5
Gin	--	--	15.9
Rum	--	--	14.0
Vodka	--	--	14.0
Whiskey	--	0.49	15.1

Table 6. B Vitamin Content for One Drink of An Alcoholic Beverage

Beverage	Thiamin B_1 mg	Riboflavin B_2 mg	Niacin B_3 mg	Panto thenic acid mg	Pyridoxine B_6 mg	Folate mcg	Cobalamin B_{12} mcg
Beer	.021	.093	1.613	.206	.178	21	.07
Light Beer	.032	.106	1.388	.127	.120	15	.04
White Wine	.005	.005	0.100	.030	.020	--	--
Red Wine	.005	.040	0.120	.050	.050	5	--
Gin	--	--	--	--	--	--	--
Rum	--	--	--	--	--	--	--
Vodka	.002	.003	--	--	--	--	--
Whiskey	.003	--	0.021	--	--	--	--

Table 7. Mineral Content for One Drink of An Alcoholic Beverage

Beverage	Calcium mg	Iron mg	Magnesium mg	Phosphorus mg	Potassium mg	Sodium mg	Zinc mg	Copper mg	Manganese mg	Selenium mcg
Beer	18	0.10	21	43	89	18	0.07	.032	.043	4.3
Light Beer	18	0.14	18	42	64	11	0.11	.085	.057	4.2
White Wine	15	0.45	15	20	120	5	0.10	.030	.675	0.5
Red Wine	10	0.65	20	20	165	5	0.15	.030	.880	0.5
Gin	--	--	--	-	--	1	--	.002	--	--
Rum	--	0.05	--	2	1	--	0.03	.021	.008	--
Vodka	--	--	--	2	--	--	--	.004	--	--
Whiskey	--	0.01	--	2	1	--	0.02	.009	.006	--

Phytochemicals

Alcoholic beverages contain a variety of biologically active compounds.[24] Polyphenols (formerly referred to tannins[25]) are present in beer and wine.[26] All polyphenols prevent LDL oxidation in vitro when added to a tissue culture system; the magnitude of protection depends upon the phenolic concentration of the beverage and the oxidation system used.[27]

Table 8 Total polyphenols of grape juice, wine or beer, mg/drink

beverage source	juice	wine
Shiraz grape	41.7	114.7
Cabernet Sauvignon grape	24.8	124.9
Grenache grape	146.4	
Sauvignon Blanc		24.6
Chardonnay		35.3
lager beer	112.3	
low alcohol beer	119.9	

An exhaustive list of all phytochemicals in alcoholic beverages cannot be made. Not all biologically active compounds have been identified. For example, an unidentified, non-alcoholic ingredient of beer and fermented glucose stimulates trypsin, amylase, gastrin and cholecystokinin secretion.[28] Not all compounds identified may have biologic activity in the dose ingested. For example, the phytoestrogens content of beer ranges from 0.45 – 10.47 nmol/drink;[29] whether or not this concentration is sufficient to cause the estrogenic effect described for the micromolar concentrations in soy is unclear. A phytoestrogen dose equivalent to 2.6 drinks/d of de-ethanolized bourbon produced corresponding changes in LH, FSH, SHBG, and HDL cholesterol levels consistent with an estrogen effect.[30] Some speculate that the phytoestrogens in certain alcoholic beverages contribute to the development of gynecomastia in chronic drinkers.

The phytochemicals in alcoholic beverages may be modified during the fermentation process. Polyphenols in wine may undergo structural changes during aging of wine, so that wine is more potent than grapes at producing physiologic benefits.[31] Distilled spirits may contain unique phytochemicals that elute with alcohol and are thus concentrated in one drink of spirits, but their presence has not been systematically studied.

An interesting biologic compound present in beer and wine and produced following alcohol ingestion may explain some of the physiologic effects of alcohol ingestion. Beta-carbolines found in a Brazilian Amazon plant are thought to be hallucinogens.[32] The beta-carboline, harman, is present in beer and wine (Table 10).[33] Another beta carboline, norharman, is present in tobacco smoke and produces increases in norharman levels during smoking.[34] Norharman and harman are detectable in human plasma of alcoholics, and are thought to derive either directly from the diet or by condensation of dietary aldehydes from alcohol with neurotransmitters

dopamine and indoleamines.[35] Norharman and harman bind with variable affinity to 5HT receptors,[36] and may explain the ability of alcohol to produce sedation, tremor and hallucinations. The alkaloid hypothesis of alcoholism suggests that beta-carbolines may be involved in addiction.

Table 9 Summary of identified phytochemicals in alcoholic beverages

alcohol source	primary material	additives	Phytochemicals
beer	barley other cereal	hops	Phenolic acids (ferulic acid) Flavonoids (formononetin, genistein[37], biochanin À daidzein, prodelphinidin[38] B3, procyanidin B3, catechin, epicatechin) Harman
wine	grapes rice or fruit		Phenolic acids: p-coumaric, cinnamic, caffeir, gentisic, ferulic, vanillic) trihydroxy stilbenes (resveratrol, polydatin) Flavonoids (catechin, epicatechin, quercetin) Harman
distilled spirits gin	unmalted grain or molasses	herbs e.g. juniper	?
rum	sugar cane byproduct		?
vodka	rye malt potato starch apples, grapes, berries, plums		?
whisky	cereal grain		?
bourbon	maize		beta-sitosterol[39] biochanin A[40]
scotch	malt & grain		?
tequila	guava		?

Table 10. Harman concentrations in commercial beers and wines sold in the US (ref 33)

beer	mcg/serving	wine	mcg/serving
Budweiser	3.74	Chardonnay	1.75
Coors	4.57	Chablis	0.12
Miller	11.02	Chenin Blanc	0.88
Stroh's	7.7	Sauvignon Blanc	1.57
Michelob	6.66	Bordeaux Blanc	0.46
Michelob dark	4.50	White Zinfandel	0.15
Henry Weinhard's	32.76	Rosè	0.60
Beck's	50.40	Zinfandel	0.46
Bud Light	2.27	Cabernet Sauvignon	0.37
Coors Light	5.80	Gamay Beaujolais	0.22
Lite	41.04	Pinot Noir	0.28
Stroh's Light	12.42	Burgundy	0.52
Weinhard's Light	8.46	Bordeaux	0.51

Contaminants

Not every component of alcoholic beverages is beneficial. Brewing and packaging practices can introduce lead into beer.[41] In an experiment to assess the impact of Chernobyl on food safety, 35% of ^{137}Cesium taken up by barley growing in contaminated areas was recovered in beer[42] Mycotoxins aflatoxin B1, ochratoxin A, Zearalenone, deosynivalenol, Fumonisins B1 and B2 found in contaminated grains[43] survive the brewing process and are measurable in beer made from contaminated grains.[44] Volatile N-nitrosamines have also been detected.[45] Polycyclic aromatic hydrocarbons could contribute to the higher incidence of head and neck cancer among drinkers.[46] Beer has been reported to contain silicon[47] asbestos[48] and aluminum.[49]

The presence of contaminants should not dissuade the reader from drinking beer. Contaminants are a problem in all foodstuffs. Canned soft drinks have a higher aluminum content than canned beer.[49] There are many tradeoffs to food processing; the majority provide greater benefits than risks.

Health Benefits of Alcoholic Beverages

The health benefits of alcoholic beverages were readily apparent to early society. As humans moved from a nomadic lifestyle to an agrarian or urban lifestyle, local sources of water became contaminated with human waste. The requirements for safe water supply became an important focus of educated societies as evidenced by the Romans aqueducts. Unfortunately, not all communities had a ready source of clean water. Alcoholic beverages provided a safe, alternative, dietary liquid. The natural acidity of beer and wine reduces growth of bacteria in water used in their manufacture. Based on the observation that wine drinking was associated with lower disease rates, Hippocrates and Galen supported the common practice of diluting water with an alcoholic beverage:[50]

> *" the best quality waters are those which flow from high-lying earthy places and from the quality of the soil as long as it is not stony and contains no minerals. Waters that are mixed and polluted and salty and those in which there is any bad quality will need a great quantity of wine, great enough to overcome this corrupting badness."*

Although Galen and Hippocrates were not correct that dilute alcoholic beverages could counteract the infectious potential of water,[51] the observation that alcoholic consumption was associated with less dysentery than water consumption lead to societal endorsement of alcoholic beverages. Wine and beer ingestion were sanctioned by religions and held prominent roles in many rituals. After the fall of the Roman Empire, the Catholic church and its monasteries took on the management of ancient Roman vineyards. Monks and nuns commonly maintained breweries to support their livelihood. In English societies, beer was the drink of choice for both noblemen and peasants.

Besides avoiding dysentery, alcohol ingestion had other desirable properties. Alcohol provided pleasure and distraction to the common laborer. Alcohol provided analgesia to suffering people. Beer was often part of a physician's prescription for a malady. For example, one of the forerunners of epidemiology, Thomas Sydenham (1624 – 1689), developed a "cooling treatment" for smallpox. The treatment, by patient report, was successful.[52]

> *"I went abroad by his direction, till I was blind, and then took to my bed. I had no fire allowed in my room, my windows were constantly open, my bedclothes were ordered to be laid no higher than my waist. He made me take twelve bottles of small beer, acidulated with Spirit of Vitriol, every twenty-four hours"*

Early problems of alcohol excess was well documented. Depictions of drunkenness are painted in Khety's Tomb, circa 2100 BC. In the late 1800's, the physician Benjamin Rush characterized drunkenness as a progressive disease of society that should be controlled by abstinence.[53] Advances in the field of public health reduced the prevalence of water borne pathogens,[54] significantly diminishing the benefits of alcohol over water. The temperance movement gathered momentum in the United States, leading to the 1919 constitutional amendment on Prohibition. Although Prohibition as an instrument of social control failed, it was highly successful in promoting a moral perspective on drinking.[55] Only recently has alcohol abuse been viewed as a chronic disease rather than a defect in moral temperance.[56]

Revisiting the Health Benefits of Alcohol: Cardiovascular Benefits

In the 1980's data suggesting a cardiovascular health benefit of alcohol began to emerge. Before reviewing this observational research, two nuances in alcohol research must be reviewed: covariates with intake and validity of self reports.

Covariates with alcohol consumption

On a per capita basis, annual consumption of alcohol in the United States is 2.7 gallons of ethanol. According to 1998 statistics collected by The Texas Alcoholic Beverage Commission,[57] per capita consumption is 3.3 drinks from spirits, 1.3 from wine, and 11.1 from beer each month.

Consumption rates do not disclose the distribution of consumption. The distribution of drinkers in the population is not normally distributed. Approximately one third of the adult population in the United States report abstinence from alcohol. In statistics collected by survey of over 8,000 Texans from the Texas Commission on Alcohol and Drug Abuse, 29% of adult males and 41% of adult females do not consume alcohol.[58] Heavy drinking, defined by the Commission as five or more drinks on five or more days in the past month, was reported by 5% of the adults surveyed. It has been estimated that half of the alcohol sold in the US is bought by the 10% of adults with a drinking problem.[59]

Many health behaviors that impact the risk for heart disease are associated with alcohol intake. Klatsky characterized the taste preferences and cardiovascular risk profile in 53,172 members of the Kaiser Permanente Health Maintenance Program.[60] Beverage preference was reported by

half of consumers. Wine drinkers were more likely to be women, non-smokers, and free of symptoms. Spirit drinkers were more likely to be men, heavier drinkers, less educated, and afflicted with disease symptoms or disease risk factors. Beer drinkers were more likely to be young men who are intermediate between wine and liquor preferrers for most traits.

Increased waist-to-hip ratio ("beer belly") may be another covariate. In a study of 15,800 African American men, alcohol intake and waist to hip ratio were measured.[61] A graded increase in waist to hip ratio was seen for beer and spirit intake but not wine intake. Whether or not this association points to a causal factor or to another covariate with alcohol preference is unknown and deserves more study. In this study, fewer participants reported wine intake. Since wine is more likely consumed with a meal, the association may be due to patterns of alcohol intake rather than specific benefits of wine per se.

The large number of covariates with alcohol consumption reduces the impact of a univariate analysis for predicting risk. Risk assessment should be adjusted for all known risk factors to strengthen the assumption that alcohol intake per se and not some unmeasured covariate is associated with benefit.

Reliability of self reports for alcohol intake

All observational studies rely on self reported alcohol intake. Health professionals are taught to be skeptical of self reported alcohol intake. In attempts to improve the accuracy of self report, the "top high" technique has been recommended.[62] In this technique, the patient is asked in a non-judgmental manner whether or not he drinks a larger amount than the physician suspects e.g., "two bottles of whiskey a day". The response of the patient will be denial of this larger amount and perhaps admission to a smaller amount better reflecting true consumption. This teaching arises from the assumption that self reports are affected by a "need for approval"[63] and "denial".[64] Specifically, alcoholics who recognize that alcohol consumption is socially undesirable but who have not confronted their drinking problem will grossly underestimate intake.

Several studies support the assumption that alcoholics do not truthfully report intake. In a long term followup study of 605 men who completed a program on alcohol rehabilitation at a Veteran's hospital, followup reports on drinking from the patient were compared to reports from a cohabiting adult as well as regular blood and urine specimens analyzed for ethanol. Using self report alone, 27% of subjects reported abstinence. When self report was corroborated by the friend/relative's report, 25% were classified abstinent. Excluding those subjects who had one weekly urine specimen or a bimonthly blood specimen test positive for alcohol, the number who were categorized as continuously abstinent fell to 19%.[65] Although the authors report the difference as highly significant, the results are somewhat encouraging -- self report of abstinence in a drinker is accurate 75% of the time. How much these data can be generalized to populations without a drinking problem is unclear.

Critics of self reported intake have been answered by others who recommend rigorous attention to the methodology of data collection. Data collected on alcoholics enrolled in an alcohol treatment program may have undue bias.[66] In the veteran's study, alcoholics may not have disclosed recidivism to their alcohol counselor because of consequences. Similarly, alcoholics enrolled in a treatment program tend to overestimate intake in order to justify their need for treatment.[67] Do problems with data collection diminish the value of self reported intake? Most researchers in the field still feel that the validity of self reported intake can be interpreted by assessing five methodology variables:[68]

1. the sensitivity of the information sought (e.g., individuals filling out questionnaires with requests for other demographic data vs. individuals who are completing information linked to an arrest record or in direct response to a physician's query for an explanation of an alcohol-related disease)
2. The specificity of the validation criteria (e.g., archival records, breath alcohol readings, urine tests, blood tests, collateral reports from other persons)
3. the personal characteristics of the respondents (e.g., sober vs. intoxicated; acute alcohol is well known to alter memory)
4. The time window of the report (e.g., lifetime vs. recent intake; recent intake over the past few months is more reliable)
5. The demand characteristics of the task situation (e.g., clinical interview vs. research evaluation)

Self reports obtained by a trained [66] interviewer correlate well with collateral reports obtained from spouses who were asked to estimate intake,[69] with r values typically > 0.80, even in alcoholics. Although some have argued that comparison to other self-report data is inappropriate, advances in the field are hampered by the lack of specific and sensitive markers of long term alcohol intake. Few methods other than disappearance data are available, and no method other than self report measures individual intake.[70] Although self-reported intake is an imprecise measure, different methods of self report are well correlated to each other (e.g., food frequency questionnaire and four 1-week food records, $r = 0.86$-0.90) and with other factors known to be altered by alcohol ingestion (e.g., HDL cholesterol levels with alcohol intake from food records, $r = 0.33$; HDL cholesterol levels and food frequency questionnaires $r = 0.33$-$.40$).[71]

Several studies have evaluated whether alcohol preference is associated with systematic under or over reporting. In an Italian study comparing intake recorded on a food frequency questionnaire and two seven day diet records, the correlation between methods for wine consumption was $r = 0.70$.[72] Poorer correlations but still $> .50$ for beer and distilled spirits still were seen. The authors suggested that the infrequent intake of beer and spirits in Italy was better reported by food frequency questionnaire than by daily food records. Only 10% of the abstainers on food frequency questionnaire had reported alcohol intake on food records. The amount of alcohol recorded on food records reclassified these individuals as light drinkers. In the MONICA study, a randomly selected subset of 244 Danish women and 249 Danish men completed a food frequency questionnaire in addition to a diet history personal interview.[73]

Intakes of wine, beer and spirits were similar between the methods (r values between 0.60-0.87) and no systematic bias by beverage type could be discerned.

For studies evaluating the health benefits of alcohol consumption in a general population, self reported intakes in the context of a questionnaire collecting a multitude of health behaviors is likely a reasonable estimation of intake.

Moderate Consumption and Total/Cardiovascular Mortality

International data plotting alcohol production versus CHD rates reveals a linear relationship. Countries consuming more alcohol had lower rates of coronary disease than countries consuming less alcohol. Although this type of analysis avoids the problems with self reported alcohol intake, it cannot adjust for other factors known to alter risk.

Several early studies point out the methodological difficulties of analyzing alcohol intake with mortality. In the prospective British Regional Heart Study, [74] 7,735 men age 40-59 were followed for 7½ years. Although a u-shaped curve between alcohol intake and total mortality was observed, the relationship dissipated when adjusted for risk factors smoking, age, and preexisting coronary disease. Other studies, such as the Yugoslavia Cardiovascular Disease study[75] measured frequency of alcohol intake and not quantity, allowing only limited conclusions to be made. In the Framingham study,[76] only a weak negative association between alcohol ingestion and CHD was observed.

The majority of more recent, large, population based studies have observed that moderate drinking in the range of 1-3 drinks daily is associated with a 30-40% lower rate of coronary disease compared to non-drinking. This association is present even after accounting for covariates of cigarette smoking and age. The association between alcohol consumption and cardiovascular disease is not linear but more "u-shaped" with higher death rates found among those who abstain as well as those who drink in excess of 6 drinks a day. The finding has been true for both myocardial infarction[77] as well as CHD death (Table 11). These findings have been replicated in multiple case control studies. As Hein, Suadicani and Gyntelberg wrote in their letter to the editor "The proposal that alcohol has protective effect on the risk of ischaemic heart disease no longer has the charm of novelty."[78]

All forms of alcohol are equally beneficial. In a metaanalysis evaluating benefits of different alcoholic beverages,[79] the findings from 10 studies were compared. The findings of a superior beverage were equally distributed between beer, wine and spirits. This suggests that the majority of the cardioprotective effect is due to the alcohol content of the beverage and not to inherent differences in other components found in these beverages.

The consistency of the data are persuasive. Most studies have observed that moderate alcohol intake afforded a one-third reduction in CHD risk. It should be emphasized that this reduction in risk applies only to the portion of the population who is currently not drinking. Translating this

figure to the population at large will lead to lesser benefits since fewer than 30% of men do not drink.

Table 11: Examples of Reported or Estimated Adjusted Relative Risk for moderate drinkers

study	participants	baseline year	yrs obs	Relative Risk CHD Death RR 1.0= abstainers
British Doctors Study[80]	12,321 physicians age 48-78	1978	6-13	13 drinks/wk RR ~ 0.51
α tocopherol, β carotene study[81]	7,052 male smokers age 50-69	1984-8	4.7	moderate drinkers RR ~0.74
Kaiser Permanente study[82]	128,934 adults	1978-85	3-10	men 1-2/d RR 0.80 women 1-2/d RR 0.80
Puerto Rico[83]	9,150 men age age 35-79	1965-8	12	single 24 hr recall RR .70
Italian Rural Cohort Study[84]	1536 men age 45-64	1965	15	4-5 drinks/d RR 0.69
Copenhagen city heart study[85,86]	6,051 men 3,234 women age 30-70	1976-8	10-12	1-6 drinks/wk RR 0.63
Shanghai Cancer Study[87]	18,244 men age 45-64	1986-9	6	1-28 drinks/wk RR 0.64
Honolulu Heart study[88]	8,006 men born 1900-19	1965-8	6	2 drinks/wk RR 0.74
Nurses Health Study[89]	87,526 women	1980	4	1 drink/d RR 0.6
Health Professionals Study[90]	51,529 men	1986	2	up to 2 drink/d RR 0.74

In a recent paper from Finland, a theoretical projection of benefits on CHD mortality was calculated using characteristics of the Finnish population.[91] For men aged 30-69, an estimated savings of 400 deaths/yr was calculated (12-14% reduction). The benefits for women or older men were less striking. Counting all categories, an annual reduction of 700 CHD deaths, or an 8% reduction in CHD mortality, was projected.

Corroborating evidence: lesion size

In a recent study of coronary atherosclerosis,[92] 484 men undergoing coronary angiography were questioned regarding their alcohol intake. The extent of atherosclerosis by angiography, was inversely associated with alcohol consumption. When HDL cholesterol levels were considered, the trend of protection against heart disease was lessened but still significant. Similar associations have been observed for carotid atherosclerosis[93] determined by ultrasound. In the recently published Bruneck Study,[94] five year progression of carotid lesions by doppler was measured in 826 men and women. The association between progression of disease and alcohol intake was j-shaped. No differences between source of alcohol were seen.

Mechanism of action

Several proposed mechanisms have been forwarded. Alcohol itself is known to raise HDL cholesterol levels, reduce platelet aggregation, reduce fibrinogen levels, and increase fibrinolytic and antithrombin activity. How much each factor contributes to the overall benefit is unclear.

In several studies, 30-50% of the overall reduction in heart disease can be explained by the HDL raising effects of alcohol[95,96]. Alcohol raises HDL cholesterol levels in a dose-dependent fashion. A single, daily alcoholic beverage raised HDL cholesterol levels 4.4% or 2 mg/dL.[97]

Another attractive explanation for the protective effect of alcohol is the effects on thrombosis. It is well known that alcohol intake increases bleeding time. Moderate alcohol consumption impairs platelet aggregation,[98] by affecting production of thromboxane A2.[99] Alcohol may improve the fluidity of red blood cell membranes.[100]

A growing area of investigation is the effects of moderate alcohol intake on insulin resistance. Insulin levels in nondiabetics predict atherosclerosis risk.[101] Moderate drinking is associated with lower insulin levels, even after adjusting for covariates[102,103]

A portion of the beneficial effects of alcohol could be due to the non-alcohol components of alcoholic beverages. Polyphenols can potentially interfere with several steps in the atherosclerosis cascade. Although alcohol itself appears to be a pro-oxidant for LDL, the phenol content in beer (~115 mg/drink) reduces LDL oxidation[104]. Polyphenols can induce endothelium dependent relaxation in rat aorta by enhancing nitrous oxide synthesis[105]. Polyphenols inhibit synthesis of thromboxane in platelets and leukotriene in neutrophils, and modulate the synthesis and secretion of lipoproteins in animals.

Public Policy: Recommendations on Alcohol Intake

Public policy on alcohol intake is a complex subject beyond the scope of this medicine grand rounds format. The book, Alcohol Policy and the Public Good[106] is highly recommended.

Science influenced the repeal of prohibition.[107] Yandell Henderson argued that exposure to alcohol was analogous to carbon monoxide. "Everyone now inhales daily a considerable amount of carbon monoxide...an acute intoxicant in high concentrations is not intoxicating at all in small amounts." He furthered that the toxic effects of alcohol could be assessed using chemistry and physiology, defining a blood alcohol level of 0.1 percent the beginning of intoxication. These data may have defined the intoxication limits commonly used today.

Public policy for alcohol ingestion falls under the purview of the USDA Dietary Guidelines. The current guidelines recommends that if you drink, drink in moderation. Moderation by the USDA

is defined as no more than one drink per day for women and no more than two drinks per day for men. The Dietary Guidelines specify that some people should not drink alcohol, including children and adolescents, individuals of any age who cannot restrict their drinking to moderate levels, women who are trying to conceive or who are pregnant, individuals who plan to drive or take part in activities that require attention or skill, and individuals using prescription and over-the-counter medications.

Last Month, it was announced that the Wine Institute was successful in its lobby to the Bureau of Alcohol, Tobacco and Firearms. Two voluntary statements have been approved for use on either the front or back labels:

> TO LEARN THE HEALTH EFFECTS OF WINE CONSUMPTION, SEND FOR THE FEDERAL GOVERNMENT'S DIETARY GUIDELINES FOR AMERICANS: CENTER FOR NUTRITION POLICY AND PROMOTION, USDA, 1120 20TH STREET, NW, WASHINGTON DC 20036 OR VISIT ITS WEB SITE: HTTP://WWW.USDA.GOV/FCS/CNPP.HTM

and/or

> The proud people who made this wine encourage you to consult your family doctor about the health effects of wine consumption.

These statements do not imply endorsement of wine as a preferred beverage by the USDA. Treasure General Counsel Ed Knight said during the announcement "under existing law, ATF can only deny labeling statements if they are false or misleading." The labeling statements approved met the factual standards as not being false or misleading because the statements do not make any health claim, but simply direct consumers to sources for information about the health effects of alcohol consumption. The above statements were tested for their impact on behavior by the Substance Abuse and Mental Health Service Administrations' Center for Substance Abuse Prevention. For wine drinkers, drinking patterns were not be influenced by the message. The statements to not replace the 1989 mandated government warning label:

> GOVERNMENT WARNING: (1) According to the Surgeon General, women should not drink alcoholic beverages during pregnancy because of the risk of birth defects. (2) Consumption of alcoholic beverages impairs your ability to drive a car or operate machinery, and may cause health problems.

In Britain, the Royal colleges of Physicians, Psychiatrists, and General Practitioners have defined "sensible limits" of alcohol intake up to 21 units per week for men and 14 units for women, 1 unit being defined as 8 g of ethanol.[108] These guidelines are very similar to the USDA definition of moderation: 168g/wk for men (13 drinks/wk or slightly under 2/d) or 112 g/wk for women (9 drinks/wk or slightly more than 1/d). Although the current guidelines are in general agreement,

some epidemiologists have argued that the protective benefits of alcohol are seen at lower intakes and the guidelines are too liberal for the public good.[109]

An Approach to Alcohol Recommendations from Physician to Patient

Although evidence has been mounting that moderate consumption of alcohol is beneficial, recommending that patients consume 1-2 drinks per day has not been an easy recommendation to make. In a 1979 editorial, William Castelli wrote[110]

> *"With 17 million alcoholics in this country we perhaps have a message for which this country is not yet ready."*

In a 1997 editorial, Criqui reiterated this concern, writing[111]

> *"Alcohol is too dangerous to be employed as a pharmacological agent except in highly selected situations."*

Physicians wishing to recommend moderate alcohol consumption to patients should consider the recommendation only after completing an appropriate history and physical examination. Salient points to include

History

- current drinking habits and family/personal history of alcohol use/abuse.

  Permissive advise for drinking should not be given to patients with a personal or family history of alcoholism because these patients are at a four-fold increased risk for developing alcoholism themselves.

The **CAGE** questions, derived from a longer questionnaire, the Michigan Alcoholism Screening Test (MAST)[112] can be used for a rapid interview for alcohol abuse[113]. Two or more positive answers suggests an alcohol problem.

CAGE
Have you felt the need to **C**ut down?
Have others **A**nnoyed you by criticizing your drinking?
Have you felt **G**uilty about your drinking?
Have you ever had a morning drink (**E**ye-opener) to steady your nerves or get rid of a hangover?

Although the CAGE questions can be helpful, only half of alcoholics answer positively to these questions and the questions themselves may appear confrontational.
Alternative approaches to screening to alcohol use have been proposed[114]:

1. How often do you use alcohol?

The question focuses on frequency and is asked in a non judgmental fashion. Frequency may be more reliable than quantity.

2. How much to you usually use?

Query into drinking pattern. Six or more bottles of beer drank during a single occasion was associated with a three fold increase in mortality compared to less than 3 bottles.[115] *Size of drinks needs to be queried. Spirit drinkers often make their drinks to "taste" rather than ounces.*

3. Have you ever used alcohol more heavily?
4. Does anyone close to you drink heavily?

If concerned, the physician can extend the interview with additional questions:

5. What are the circumstances in which you drink? (social interactions, etc)
6. Do you like yourself better when you drink?
&. Do you use alcohol as medication to relieve pain, anxiety, or trouble sleeping?
8. Have you had "blackouts" – loss of memory for events while intoxicated?
9. Is your drinking different now than it was 5 years ago?

- Diseases that may worsen with alcohol consumption should be tabulated.

1. Patients taking MAO Inhibitors. Ten biogenic amines have been identified in beer, the most common being tyramine.[116] Bottled beers have low tyramine concentrations (0-1.14 mg/drink) and patients taking monoamine oxidase inhibitors can probably consume 1-3 cans of beer in a four hour period without having a reaction. Tap beer, particularly bottom fermentation derived lagers with *S. carlsbergensis*, can have tyramine levels up to 40.8 mg/drink and should not be consumed by patients taking MAO inhibitors.[117,118]
2. Patients with gout or elevated uric acid levels. Alcohol reduces purine excretion, and beer contains guanine and adenine, two purines.[119] Patients with gout should not drink alcohol and should particularly avoid beer.
3. Patients with sprue. The barley prolamin, hordein, is present in beer at a concentration of 1.12 g/drink[120]. Hordein is to barley as gliadin is to wheat – both are prolamins that cross reacts with anti-gliadin antibodies. Beer consumption in patients with coeliac disease may exacerbate their symptoms.
4. Patients who will be undergoing elective surgery. Alcohol increases the likelihood of intraoperative and postoperative bleeding, so patients should be counseled to abstain from alcohol 1-2 weeks prior to major surgery[121]
5. Patients with existing heart disease. Excessive single dose ingestion of >7 drinks of alcohol in a single setting, reducing heart rate variability for 12 hours after the dose.[122] Patients with existing disease should be explicitly instructed to avoid binge drinking.
6. Elderly patients. Geriatric patients in a nursing home become more social when given a beer or a glass of wine during the evening meal.[123] Alcohol dose should be adjusted downward because of reduced tolerance with age.
7. Patients stopping by for a drink before going home. Alcohol is more rapidly absorbed on an empty stomach, resulting in a higher peak blood alcohol concentration.[124]

Table 12. Peak Blood Alcohol Concentration in Men following 1½ drinks

Condition	Whiskey	Beer
preprandial	.048 ± .04	.040 ± .03
prandial	.022 ± .02	.028 ± .03
postprandial	.023 ± .03	.031 ± .04

Physical examination

- signs of trauma
- signs of liver disease: tender hepatomegaly, spider nevi, secondary lunulae, palmar erythema, bruising, parotid enlargement, ascites
- conjunctival injection, facial telengectasia, tongue and hand tremors
- hypertension, obesity
- withdrawl features, commonly anxiety, sweating, and tachycardia
- evidence of intoxication – alcohol on the breath, ataxia, disinhibition

Laboratory evaluation

- half of the cases show some abnormality in liver function tests; γ-glutamyltransferase measurement is the most sensitive
- mean corpuscular volume is elevated

Specific Patient Recommendations

- Patients who report more than moderate alcohol intake or who have signs of alcoholic liver disease should be counseled to reduce their current intake.
- Patients who are light drinkers can be informed of the health benefits of moderate consumption.
- Patients who abstain from alcohol and have no risk factors for alcoholism can be informed of the benefits, realizing that personal choice (e.g., dislike of side effects of alcohol consumption) may continue to influence behavior.
- There is no evidence that cardiovascular benefits are limited to a specific type of alcohol, and people who prefer beer or spirits over wine should be counseled to continue the beverage of their choice in moderation.

Summary and Conclusions: The Nutritional and Health Effects of Beer

1. **Beer is an ancient beverage with rich ties to our culture.**

2. **The flavor characteristics of beer are unique and varied.**

3. **Beer is rich in protein, B vitamins, and certain minerals.**

4. **Beer has equivalent polyphenol content to red wine and contains other phytochemicals that may have biologic activity.**

5. **Similar to other alcoholic beverages, people who drink a moderate intake of beer have a lower rate of cardiovascular disease compared to nondrinkers.**

6. The mechanism for the protective effect of beer has not be determined, but speculations include the effect of beer on raising HDL, increasing bleeding time, improving insulin resistance, and providing phytochemicals to the diet.

7. Recommendations to increase the intake of beer or any other alcoholic beverage should be made cautiously, and only after an appropriate history and physical examination is performed.

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