ABC of alcohol
Alcohol in the body

Alex Paton

Alcohol (ethanol) is a drug, and health professionals should know something of its physiological and pathological effects and its handling by the body. It is a small, water soluble molecule that is relatively slowly absorbed from the stomach, more rapidly absorbed from the small intestine, and freely distributed throughout the body. Alcoholic drinks are a major source of energy—for example, six pints of beer contain about 500 kcal and half a litre of whisky contains 1650 kcal. The daily energy requirement for a moderately active man is 3000 kcal and for a woman 2200 kcal.

This article is adapted from the 4th edition of the *ABC of Alcohol*, which will be available in February

Absorption

Rate of absorption of alcohol depends on several factors. It is quickest, for example, when alcohol is drunk on an empty stomach and the concentration of alcohol is 20-30%. Thus, sherry, with an alcohol concentration of about 20% increases the levels of alcohol in blood more rapidly than beer (3-8%), while spirits (40%) delay gastric emptying and inhibit absorption. Drinks aerated with carbon dioxide—for example, whisky and soda, and champagne—get into the system quicker. Food, and particularly carbohydrate, retards absorption: blood concentrations may not reach a quarter of those achieved on an empty stomach. The pleasurable effects of alcohol are best achieved with a meal or when alcohol is drunk diluted, in the case of spirits.

Alcohol is distributed throughout the water in the body, so that most tissues—such as the heart, brain, and muscles—are exposed to the same concentration of alcohol as the blood. The exception is the liver, where exposure is greater because blood is received direct from the stomach and small bowel via the portal vein. Alcohol diffuses rather slowly, except into organs with a rich blood supply such as the brain and lungs.

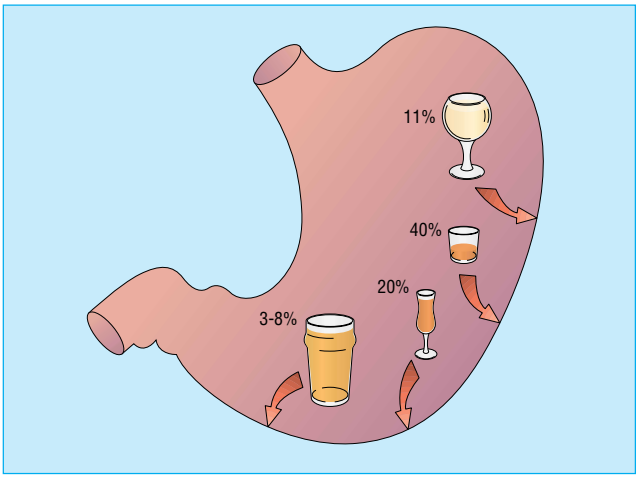
Other factors

Very little alcohol enters fat because of fat's poor solubility. Blood and tissue concentrations are therefore higher in women, who have more subcutaneous fat and a smaller blood volume, than in men, even when the amount of alcohol consumed is adjusted for body weight. Women also may have lower levels of alcohol dehydrogenases in the stomach than men, so that less alcohol is metabolised before absorption. Alcohol enters the fetus readily through the placenta and is eliminated by maternal metabolism.

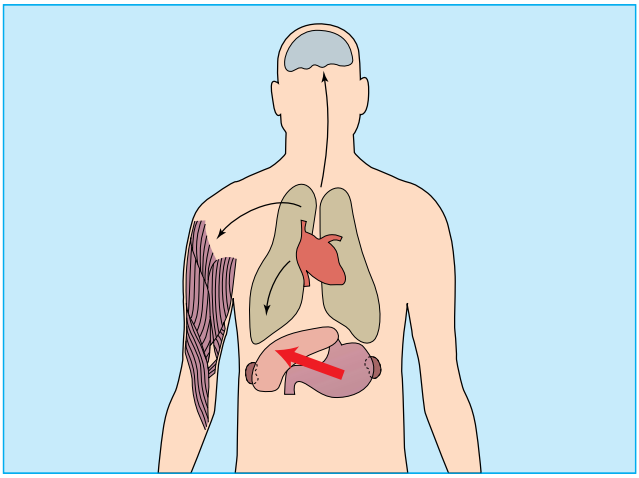
Blood alcohol concentration varies according to sex, size and body build, phase of the menstrual cycle (it is highest premenstrually and at ovulation), previous exposure to alcohol, type of drink, whether alcohol is taken with food or drugs, such as cimetidine (which inhibits gastric alcohol dehydrogenase) and antihistamines, phenothiazines, and metoclopramide (which enhance gastric emptying, thus increasing absorption).

Metabolism of alcohol

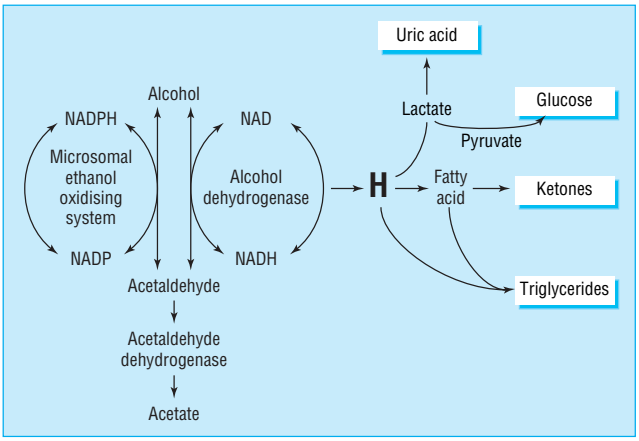
More than 90% of alcohol is eliminated by the liver; 2-5% is excreted unchanged in urine, sweat, or breath. The first step in metabolism is oxidation by alcohol dehydrogenases, of which at least four isoenzymes exist, to acetaldehyde in the presence of



Rate of absorption of alcohol (arrows) is affected by concentration of alcohol



Most tissues are exposed to the same alcohol concentration as in the blood



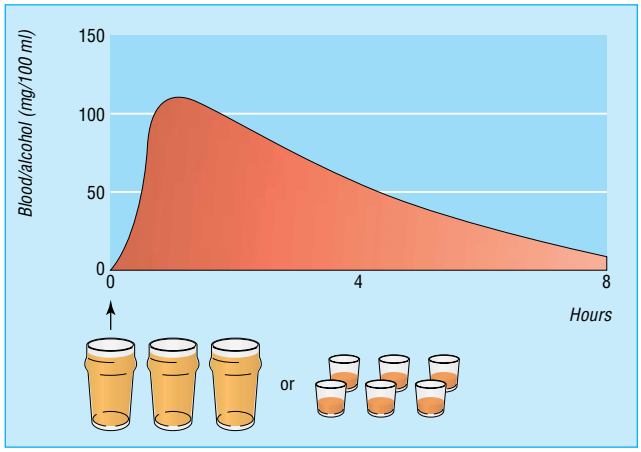
Metabolism of ethanol

cofactors. Acetaldehyde is a highly reactive and toxic substance, and in healthy people it is oxidised rapidly by aldehyde dehydrogenases to harmless acetate.

Several isoenzymes of aldehyde dehyrdrogenase exist, one of which is missing in about 50% of Japanese people and possibly other south Asian people (but rarely in white people). Unpleasant symptoms of headache, nausea, flushing, and tachycardia are experienced by people who lack aldehyde dehydrogenases and who drink; this is believed to be because of accumulation of acetaldehyde. Under normal circumstances, acetate is oxidised in the liver and peripheral tissues to carbon dioxide and water.

On an empty stomach, blood alcohol concentration peaks about one hour after consumption, depending on the amount drunk; it then declines in a more or less linear manner for the next four hours. Alcohol is removed from the blood at a rate of about 3.3 mmol/hour (15 mg/100 ml/hour), but this varies in different people, on different drinking occasions, and with the amount of alcohol drunk.

At a blood alcohol concentration of 4.4 mmol (20 mg/100 ml), the curve flattens out, but detectable concentrations are present for several hours after three pints of beer or three double whiskies in healthy people; enough alcohol to impair normal functioning could be present the morning after an evening session of drinking. Alcohol consumption by heavy drinkers represents a considerable metabolic load—for example, half a bottle of whisky is equivalent in molar terms to 500 g aspirin or 1.2 kg tetracycline.

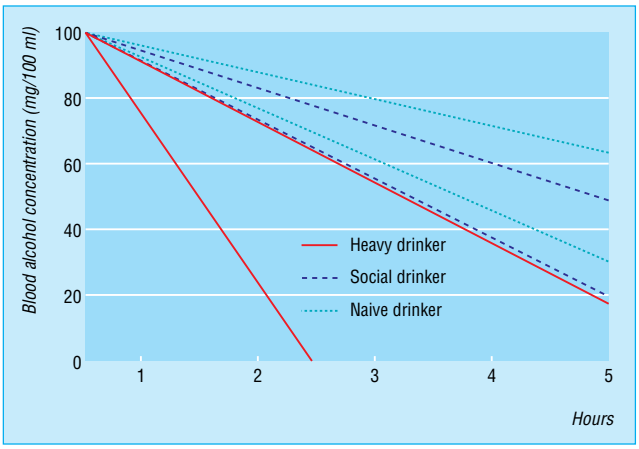


Concentrations of alcohol in the blood after six units of alcohol (equivalent to 48 g alcohol)

Heavy drinkers

Two mechanisms dispose of excess alcohol in heavy drinkers and account for "tolerance" in established drinkers. Firstly, normal metabolism increases, as shown by high blood concentrations of acetate. Secondly, the microsomal ethanol oxidising system is brought into play; this is dependent on cytochrome P450, which is normally responsible for drug metabolism, and other cofactors. This process is called enzyme induction, and the effect is also produced by other drugs that are metabolised by the liver and by smoking.

The two mechanisms lead to a redox state, in which free hydrogen ions build up and have to be disposed of by several different pathways. Some of the resultant metabolic aberrations can have clinical consequences: hepatic gluconeogenesis is inhibited, the citric acid cycle is reduced, and oxidation of fatty acids is impaired. Glucose production is thus reduced, with the risk of hypoglycaemia; overproduction of lactic acid blocks uric acid excretion by the kidneys; and accumulated fatty acids are converted into ketones and lipids.

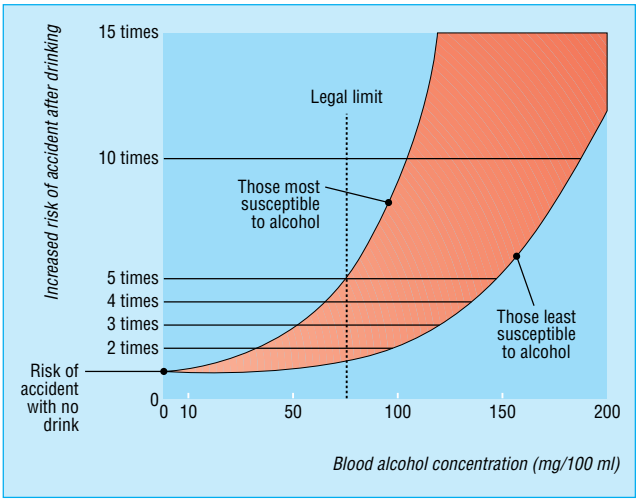


Rate of decrease of concentrations of alcohol in the blood in heavy, social, and naïve drinkers. The two lines represent maximum and minimum rates for each category

Behavioural effects

Alcohol is a sedative and mild anaesthetic. It is believed to activate the pleasure or reward centres in the brain by triggering release of neurotransmitters such as dopamine and serotonin. Alcohol produces a sense of wellbeing, relaxation, disinhibition, and euphoria. These feelings are accompanied by physiological changes such as flushing, sweating, tachycardia, and increases in blood pressure, probably because of stimulation of the hypothalamus and increased release of sympathomimetic amines and pituitary-adrenal hormones. The kidneys secrete more urine, not only because of the fluid drunk but also because of the osmotic effect of alcohol and inhibition of secretion of antidiuretic hormone.

Increasing consumption leads to a state of intoxication, which depends on the amount drunk and previous experience of drinking. Even at a low blood alcohol concentration of

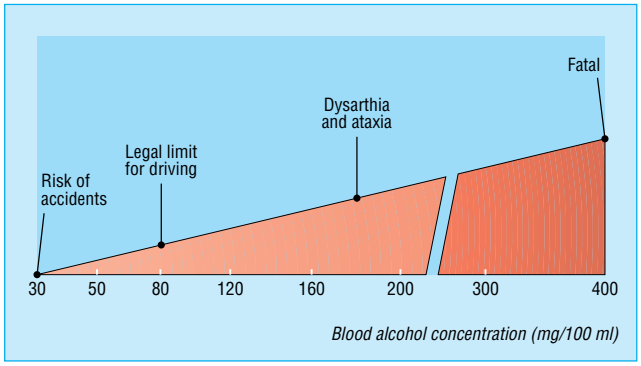


Effect of alcohol on behaviour

around 6.5 mmol/l (30 mg/100 ml), the risk of unintentional injury is higher than in the absence of alcohol, although individual experience and complexity of task have to be taken into account. In a simulated driving test, for example, bus drivers with a blood alcohol concentration of 10.9 mmol/l (50 mg/100 ml) thought they could drive through obstacles that were too narrow for their vehicles. At 17.4 mmol/l (80 mg/100 ml)—the current legal limit for driving in the United Kingdom—the risk of a road traffic incident more than doubles, and at 34.7 mmol/l (160 mg/100 ml), it increases more than 10-fold.

People become garrulous, elated, and aggressive at concentrations above 21.7 mmol/l (100 mg/100 ml) and then may stop drinking as drowsiness supervenes. After effects ("hangover") include insomnia, nocturia, tiredness, nausea, and headache.

If drinking continues, slurred speech and unsteadiness are likely at around 43.4 mmol/l (200 mg/100 ml), and loss of consciousness may result. Concentrations above 86.8 mmol/l (400 mg/100 ml) commonly are fatal as a result of ventricular fibrillation, respiratory failure, or inhalation of vomit (this is particularly likely when drugs have been taken in addition to alcohol).



Risks associated with concentrations of alcohol in the blood

The figure showing the metabolism of ethanol is adapted from C S Lieber et al. *N Engl J Med* 1978;298:356. The figure showing the effect of alcohol on behaviour is adapted from Transport and Road Research Laboratory. *The facts about drinking and driving*. Crowthorne: Berkshire, 1983. The figure showing concentrations of alcohol in the blood in heavy, social, and naïve drinkers is constructed from figures supplied by K Lewis *BMJ* 1987;295:800–1.

Competing interests: None declared.

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Further reading

- Lewis KO. Back calculation of blood alcohol concentration. *BMJ* 1987;295:800-1
- Lieber CS, Salaspuro MP. Alcoholic liver disease. In: Millward-Sadler CHM, Wright R, Arthur MJP, eds. *Wright's liver and biliary disease*, 3rd ed. London: Saunders, 1992:899-964
- Paton A. The body and its health. In: Cooper DB, ed. *Alcohol use*. Oxford: Radcliffe, 2000:25-38.

When I use a word

Medical emoticons

On 19 September 1982 a computer scientist at the Carnegie Mellon University in Pittsburgh, Scott E Fahlman, sent an email message to his colleagues, who had been discussing how to mark jokes in an email text:

"I propose the following character sequence for joke markers: :–) Read it sideways. Actually, it's probably more economical to mark things that are NOT jokes, given current trends. For this, use :–("

This is generally agreed to be the first example of what are now called emoticons, or smileys, the little sets of symbols that are used as markers in email messages. In fact, there is a pre-history of such symbols. According to the *Official Smiley Dictionary* (www.smileydictionary.com), in 1972 a French journalist called Franklin Loufrani invented a picture consisting of a bubble with two dots for eyes and a curve for a smiling mouth, thus ☺; he called it a smiley and used it and derivatives to mark articles in *France soir* and other newspapers.

Whatever their origin, emoticons are popular and have spawned explanatory dictionaries and websites. Here are my suggestions for some medical emoticons. Remember to hold the page sideways.

a€:– |	Male pattern baldness	:–(#)	Tonsillitis
@:–|	Alopecia areata	:–))	Mania
8–)	Myopia	:´–((	Depression
%–)	Strabismus	:–§	Bipolar disorder
!–)	Ptosis	:–(#	Torticollis
::– –))	Diplopia	€:–)X	Harley Street doctor
8–)) 8	Graves' disease	€:–) :–	Man
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:o(	Rhinophyma	€:–(O	Ascites
:¬|	Scleroderma	€:–(O	Hydrocele
:–|= = = =	Marfan's syndrome	€:–(¬	Impotence
:–£	Congenital syphilis	€:–)) \	Viagra
:–/	Bell's palsy	€:–((\	Priapism

There was a time when doctors would write abbreviations and acronyms, such as GOK (God only knows) or TALOIA (there's a lot of it about), in patients' notes. Now instead we can draw emoticons. I expect to see this happen before too long.

Jeff Aronson *clinical pharmacologist, Oxford*