
Alcohol

- *Alcohol abuse* and *alcoholism* are common, especially in doctors, who have three times the general population rate.
- The effects of alcohol in impairing intellectual and motor function and increasing aggression are due to the drug itself, whereas the effects upon antisocial behaviour, increased consumption and sexual arousal are due to LEARNED EXPECTANCIES.
- Alcoholism results from operant conditioning, classical conditioning and imitation, and in addition there may be genetic predispositions.
- Cross-tolerance between alcohol and opiates suggests that alcohol may have its effects upon central endorphin-mediated mechanisms.
- Total alcohol consumption in society is directly related to the real price of alcohol, taxation and the amount spent on advertising.

Alcohol is perhaps the only drug of addiction which is sometimes used by most of the adult population. That many individuals become physically addicted, or suffer serious acute or chronic side effects is therefore hardly surprising. Table 25.1 shows the risks associated with different levels of alcohol consumption measured in STANDARD UNITS.

Alcohol use must be distinguished from ALCOHOLISM, which is a true addiction. Although most of us use alcohol, and most are not alcoholics, a clear dividing line is not obvious. Certainly the old joke that 'an alcoholic is a person who drinks more than their doctor' is inappropriate, since doctors have three times the general rate of alcoholism and alcohol-related diseases. Probably a suitable definition is that of the World Health Organization: 'Alcoholics are those excessive drinkers whose dependence on alcohol has attained such a degree that it shows a noticeable mental disturbance or interference with their bodily health, their interpersonal relations, and their smooth social and economic functions, or who show the prodromal signs of such development. They therefore require treatment'.

The acute effects of alcohol are well-known: a decrease in sensory and motor abilities, proportional to the dose; increased sociability and decreased social inhibition, the 'ice-melting effect' at parties; and a

Table 25.1

Units of alcohol ('standard drinks') in commonly dispensed measures of different drinks. Men drinking less than 36 units per week (women 24 units) are unlikely to suffer health problems. Men drinking between 36 and 50 units per week (women 24–36) are probably drinking too much, and are likely either to become dependent or to risk their health. Men drinking over 50 units per week (women 36) are probably suffering ill health as a result of their drinking, and men drinking over 96 units per week (women 64) are almost certainly dependent upon alcohol and feeling the consequences. Reproduced with permission from Health Education Council (1989), *That's the limit*, London.

		STANDARD DRINKS
BEERS AND LAGER		
Ordinary strength beer or lager	1/2 pint	1
	1 pint	2
	1 can	1 1/2
Export beer	1 pint	2 1/2
	1 can	2
Strong ale or lager	1/2 pint	2
	1 pint	4
	1 can	3
Extra strength beer or lager	1/2 pint	2 1/2
	1 pint	5
	1 can	4
CIDERS		
Average cider	1/2 pint	1 1/2
	1 pint	3
	quart bottle	6
Strong cider	1/2 pint	2
	1 pint	4
	quart bottle	8
SPIRITS		
	1 standard single measure in most of England and Wales (1/6 gill)	1
	1 standard single measure in Northern Ireland (1/4 gill)	1 1/2
	1/5 gill measure	1 1/4
	1/4 gill measure served in some parts of Scotland	1 1/2
	1 bottle	30
TABLE WINE		
(including cider wine and barley wine)	1 standard glass	1
	1 bottle	7
	1 litre bottle	10
SHERRY AND FORTIFIED WINE		
	1 standard small measure	1
	1 bottle	12
<i>These figures are approximate.</i>		

lowered threshold for antisocial or unsociable acts, so that, as Oliver Zangwill (1914–1987) put it, 'the super-ego is that part of the mind which is soluble in alcohol'. Although most psychomotor tasks deteriorate with alcohol, two are interesting as they are unexpected. Slowly moving objects are tracked visually by SMOOTH TRACKING MOVEMENTS, which as objects speed up eventually break down into SACCADES or jumps; the threshold at which smooth tracking changes to saccades is directly proportional to blood alcohol concentrations, effects being seen even at 20 mg%, well below 80 mg%, the legal maximum for driving. Alcohol also diminishes ability to recover from sudden bright lights ('glare'), the effect being cortical not retinal in origin. The implications for the most serious social consequence of acute alcohol intoxication, road traffic accidents, are obvious, and it is beyond doubt that many road traffic accidents result from alcohol consumption. Less well known is that alcohol levels are raised in pedestrians involved in road accidents, in persons injured in non-traffic accidents, be they industrial or domestic, and in drowning accidents. The final effect of alcohol is that upon sexual function, described well by the porter in *Macbeth*, 'Lechery, sir, it provokes and unprovokes; it provokes the desire, but it takes away the performance'.

Not all alcohol's effects are pharmacological, some being due to LEARNED EXPECTANCY. Placebo studies using alcohol (usually vodka in dilute orange juice) or placebo (orange juice only, in a glass with vodka smeared around the rim to give the smell of alcohol) find many effects are produced as strongly by the placebo as by alcohol; they are produced in expectation, presumably from previous experience. Deviant antisocial behaviour, including excessive consumption itself, illicit behaviour and sexual arousal are mainly a result of expectancy, whereas impaired intellectual and motor performance, and increased aggression are truly a result of alcohol consumption rather than expectancy.

Alcohol apparently does not affect people equally, although many differences reflect difference in usage of words describing states of intoxication. Whereas some people describe themselves as 'drunk' when feeling slightly impaired, others only 'feel drunk' when losing gross bodily control of walking, or perhaps only on being unable to remember the night before do they conclude retrospectively that they were 'drunk'. Given such perceptions, one can understand how individuals can both agree that 'drunken driving is wrong', and then drive after six whiskies because they do not perceive themselves as being 'drunk'. In taking a drinking history, doctors must also be careful, particularly as many patients will underestimate consumption by as much as 50%; this is mainly a response to implied criticism by the doctor, since more accurate responses are given to an interactive computer program.

Alcoholism transcends normal alcohol use, even with its temporary

excesses, and becomes a state of DEPENDENCE, in which it is 'normal' to have alcohol in the blood stream (or 'blood in the alcohol stream'). Dependent behaviours revolve around alcohol, often rather than alcohol being a small part of daily life; often there is LOSS OF CONTROL, in which a single drink initiates a 'bender', when the patient is unable to stop drinking. Dependent drinkers also consume alcohol at unusual times (before or instead of breakfast, or at work) or in places where alcohol is normally unacceptable (and alcohol on a patient's breath in a GP's surgery is said to be a good indicator of dependent drinking).

Dependent drinkers narrow their drinking repertoire, drinking regularly, day in, day out, rather than in relation to social events. Although CRAVING is conventionally said to be a separate symptom of dependence, both placebo and alcohol in abstinent alcoholics have equivalent effects upon craving suggesting that it is merely a re-description of the alcoholic's other symptoms. Alcoholics also show personality changes, and changed relationships with spouse and friends, becoming argumentative and uncaring, particularly when drinking is frustrated; the drinking itself is blamed upon the uncontrollable actions of inexorable fate, or else, by the defence mechanism of paranoid projection, upon the faults of family, employer, etc.

What makes some people become alcoholic? An immediate explanation is that it is learnt, and in a trivial sense this is true, since alcohol, like cigarettes, is unpleasant at first. Learning theories take three forms. OPERANT CONDITIONING says alcohol consumption is rewarded by reduction in anxiety or stress, or the easing of social relationships, and as each glass of alcohol is rewarding, so the operant response, drinking, will increase in frequency. Certainly in animals alcohol reduces stress reactions, and stressed animals work to drink alcohol rather than water. Alcohol *in vitro* acts like a benzodiazepine and *in vivo* can have an anxiolytic action. The CLASSICAL CONDITIONING theory says that as in operant conditioning, alcohol reduces anxiety, but that eventually the unconditioned response (anxiety reduction) produced by the unconditioned stimulus (alcohol consumption) becomes conditioned to other stimuli occurring concurrently (such as the bar or pub, or the people one drinks with), and those objects themselves become anxiety reducing; however when in a bar the obvious thing to do is to have a drink, and so drinking behaviour increases in frequency. IMITATION is the third form of learning and takes two forms. Parental drinking can provide a model of appropriate behaviour for their children and be imitated; of especial interest is that heavy drinking men often have heavy drinking fathers (but not mothers) and vice-versa in heavy drinking women. Reaction formation can also occur to parental role-models, and TEETOTALLERS (total abstainers from alcohol) also report *higher* incidences of alcoholic parents than do moderate drinkers. Imitation can also involve social modelling. Society regards drinking as normal and allows advertisers to use all

the subtle forms of persuasion in their armamentarium to imply that alcohol is 'good', makes one appear 'masculine' or 'feminine', produces success with the opposite sex, etc., in the same way that cigarette advertising promotes similar ends (and heavy drinkers tend to be heavy smokers). Alcohol advertising undoubtedly works; a 10% reduction in advertising results in a 1.3% reduction in alcohol consumption, so that a total ban on advertising could decrease consumption by 13%.

Alcoholism differs in frequency between societies and between groups within a society; for instance Jewish, Chinese and Italians in America have low rates, whereas Irish Americans have high rates of alcoholism. Cross-cultural studies by anthropologists suggest that alcoholism relates to lax social organization, tightly organized groups with strong social hierarchies having low rates. Similar factors are probably important within social groups, and perhaps even within families in our society.

Another explanation of alcohol use and abuse is genetic or constitutional. Alcohol use and alcoholism are more similar in monozygotic than dizygotic twins, and although this might reflect more similar environments, studies also show that drinking habits and alcoholism are more similar to those of biological than adoptive parents in adopted children. Studies in experimental animals show that alcohol preference and rate of alcohol metabolism are highly heritable, being readily selected for. Some individuals may therefore have a genetic predisposition to consume alcohol heavily or to become alcoholic; nevertheless such individuals must first be exposed to readily available alcohol before the gene could manifest.

A neurobiological theory of alcohol action says its effects are due to stimulation of normally inhibitory GABA receptors, which are also stimulated by barbiturates and benzodiazepines, resulting in cross-tolerance between these drugs and alcohol. Recent work shows that Ro15-4513, a specific competitive inhibitor of GABA, can block alcohol's behavioural effects in rats (general stupor, staggering gait and impaired righting reflex), supporting the idea that alcohol acts on these receptors. Ro15-4513 cannot treat the life-threatening complications of alcohol, such as respiratory depression and coma, which are mediated by direct effects upon cell membranes; it is also unlikely to prevent the long term metabolic effects of abuse.

Learning theories of alcoholism are only partly able to account for the final stage of DEPENDENCE when the consequences of drinking become more and more unpleasant. Although the alcoholic may still be drinking for learned reasons, physical dependence means that drinking is also the operant behaviour which avoids the punishing effects of the unpleasant consequences of withdrawal. Acute alcohol withdrawal produces DELIRIUM TREMENS with its tremor, hallucinations, fits and delirium, starting eight hours or so after the last drink, lasting

several days and producing reduced serum magnesium (causing the fits), hyperventilation, respiratory alkalosis and hypoglycaemia. Opiate addicts have long known that opiates reduce the symptoms of alcohol withdrawal, and alcohol reduces the symptoms of opiate withdrawal, resulting in a theory that physical dependence upon alcohol is endorphin mediated. Injection of naloxone into mice during acquisition of alcohol dependence prevents subsequent alcohol withdrawal symptoms. In man alcohol is metabolized to acetaldehydes, which are precursors of the tetrahydroisoquinolines, addictive opiate-like substances. Both alcoholics and their relatives have more efficient systems for converting alcohol to acetaldehyde, suggesting a possible genetic mechanism.

This account has mostly asked why individuals become alcoholic. Treatment and prevention may be considered more quickly, since their state is far less satisfactory. Many treatments are entirely empirical, without theoretical rationale. An exception is disulfiram ('Antabuse'), which produces a toxic reaction with alcohol, producing discomfort, due to nausea, vomiting and tachycardia. The drug therefore acts by producing a LEARNED AVERSION, the response (alcohol) producing punishment (nausea), and thus decreasing the rate of responding, and inducing abstinence. Although not as effective as might be hoped, this is partly due to a lack of compliance in taking the drug. With little doubt the most effective treatment for alcoholism is provided by *Alcoholics Anonymous* (AA), and emphasizes the need for individuals to be responsible for their own drinking, and assists this with substantial social support, particularly from ex-alcoholics who have themselves been through the process.

The most important medical role in treating alcoholism is in *recognizing* alcoholics. Each GP is estimated to know only 1 or 2 of the 15 alcoholics in their practice, most of whom are not 'skid row' down-and-outs, but ordinary, respectable individuals with a problem. Effective screening methods include questionnaires (which work well if the patient is co-operative), detection of alcohol on the breath, measurement in serum of alcohol or of raised levels of the liver enzymes, gamma-glutamyl transpeptidase (GGTP) and aspartate transaminase (AspT), and the finding of macrocytosis without anaemia. Alcoholism has been called the great imitator (displacing syphilis and tuberculosis from that role), and undoubtedly in accident and emergency departments, psychiatric units, and general medical and surgical units many cases are misdiagnosed.

Prevention of alcoholism and alcohol abuse is not principally a medical or psychological problem, but instead a case for political action. Alcoholism needs alcohol and ready availability increases alcohol consumption (as for instance in Finland where allowing general stores to sell alcohol produced a sudden and sustained increase in consumption). Alcohol consumption closely follows the price in

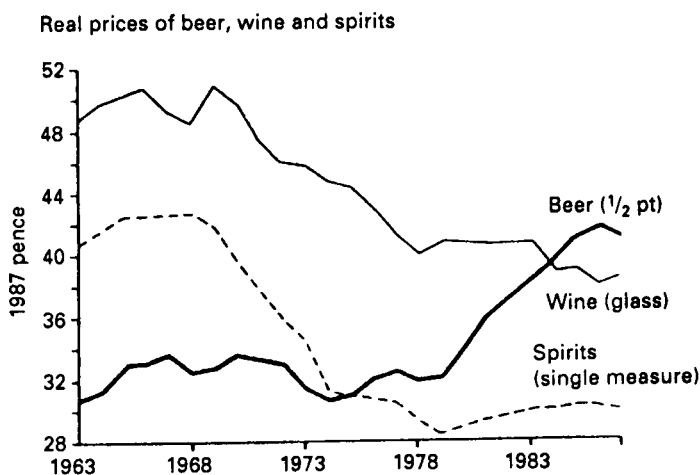
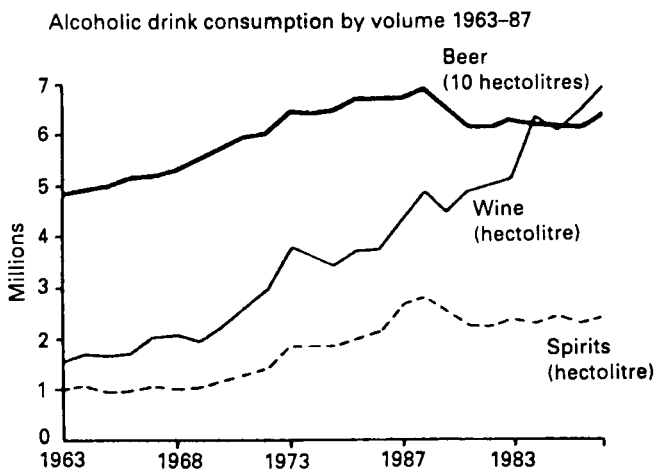


Fig. 25.1 Relationship between alcohol consumption in Britain, 1963-1987, and the real price of beer, wine and spirits. Reproduced with permission from Smith R (1989), *Br Med J*, **298**, 701.

real terms (Fig. 25.1). Advertising undoubtedly increases alcohol intake; and alcohol consumption is directly related to alcoholism rates. Alcohol consumption is reduced by increased taxation, a 10% price increase reducing consumption by 2.5%. The prevention of alcoholism and alcohol abuse is thus primarily in the hands of government.