# Alcohol-related road traffic accidents: promoting a lower alcohol strategy

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*Clin Med* 2007;7:348–50

**ABSTRACT – Impairment is the result of unpredictable genetic variation, coexisting disease, or drug interaction, and is modified by hepatic metabolism. The introduction of alcohol tsars and alcohol health workers in trauma centres would capitalise on the 'teachable moment' (when the conceptual link between drinking and its consequences can be demonstrated at a time when the consequences are obvious) to prevent recurring injuries and to lower alcohol intake. Accident fatalities have reached a plateau and the only way of reducing them further is to lower the European legal limit of blood alcohol concentration to a harmonised 50 mg/100 ml.**

**KEY WORDS: accidents, alcohol, driving, fatalities, impairment, intervention, legal limit, metabolism, teachable moment**

The reduction of alcohol-related road traffic casualties will be no accident. Drink-drive accident fatalities have reached a plateau.[1] Legislation regarding upper permissible levels of blood alcohol therefore requires reappraisal. Impairment due to alcohol is the result of genetic variation, coexisting disease, or drug interaction, and is modified by hepatic metabolism. These variables are unpredictable and idiosyncratic. The level of impairment due to an amount of alcohol which would theoretically, in an ideal subject, give a threshold level of 80 mg/100 ml might in fact cause a significantly higher impairment of cerebral function and muscular incoordination. This would have serious consequences for road traffic safety. Many of the aspects which would be required to positively influence the accident statistics are poorly recognised or understood and more research on them is needed if alcohol-related road traffic accidents are to be reduced further.

## Alcohol metabolism

Alcohol and drug interactions are frequently unrecognised but may be important. In elderly subjects there are higher peak ethanol concentrations with alcohol.[2] Drugs that suppress central nervous system activity, such as benzodiazepines, narcotics, and hypnotics, often show increased sedation in elderly people. It is well known that the combination of these drugs and alcohol causes serious impairment of the motor system and judgement needed for driving. Serum phenytoin levels may be reduced significantly in patients who drink alcohol regularly and can therefore be the cause of epileptiform seizures in previously well-controlled patients. Hepatic metabolism is the main factor controlling the impairment caused by alcohol. There are three main pathways for ethanol metabolism: the alcohol dehydrogenase (ADH) pathway; the microsomal ethanol oxidation system (MEOS); and catalase which under physiological conditions plays no role. Alcohol dehydrogenase and catalase activities were significantly lower in patients with liver disease. The activity of the nicotinamide adenine dinucleotide phosphate (NADPH)-dependant MEOS was significantly greater in patients with liver disease and it is suggested that the greater activity of the NADPH system may compensate for the low ADH activity found in patients with liver disease and maintain normal rates of ethanol metabolism. This is a particularly difficult area and alcohol metabolism may be relatively normal in patients even with liver disease and quite severe derangement of liver enzymes. Alcohol metabolic rate, however, can be lower in patients who have cirrhosis, particularly with jaundice. The rate of absorption of alcohol is highly variable depending on food intake, gender,[3] and first-pass metabolism.[4] When the metabolic rate for alcohol is lower it results in raised blood alcohol levels and increased cerebral impairment.

## Genetics

A polymorphism in the gene[5] responsible for ADH type 3 (ADH3) alters the rate of alcohol metabolism and pharmacokinetic studies show a 2.5-fold difference in the maximal velocity of ethanol oxidation between the homodimeric y1 isoenzyme (associated with a fast rate) and the homodimeric y2 isoenzymes (associated with a slow rate). In addition a variant allele of the aldehyde dehydrogenase gene 2 (ALDH2), gene ALDH2*2,[6] has been shown to result in reduced ALDH activity and increased levels of acetataldehyde after the administration of alcohol.

## Alcohol-related harm

There is a well-marked association between alcohol consumption and all dimensions of alcohol-related harm. In the UK, Ireland, Luxembourg and Denmark, the increase in alcohol consumption per capita between 1970 and 1979 was accompanied by increased hospital admissions for alcoholism and drink driving, and an increase in the proportion of road users killed with excessive blood alcohol concentrations (BACs). Conversely, in France between 1970 and 1979 when per capita alcohol consumption fell so did mortality caused by liver cirrhosis, alcoholism and the rate of alcohol-related road deaths.[7] Alcohol intoxication is the leading risk factor for injury. Brief alcohol interventions in trauma patients have been shown to reduce subsequent alcohol intake and injury recidivism by capitalising on a 'teachable moment' wherein the healthcare worker can moderate the conceptual link between drinking and its consequences at a time when the consequences are obvious.[8] Injury is the most common presentation of alcohol problems and perceptive recognition can promote timely intervention. This work from the United States relating to injury trauma has been instrumental in placing alcohol health workers into trauma centres and has initiated screening and counselling. Advanced trauma and life support training and certifying trauma centres were found to significantly benefit outcome and reduce alcohol intake.[9] A Royal College of Physicians working party highlighted the significant drain on hospital resources caused by alcohol and recommended that every hospital should have an alcohol tsar and an alcohol health worker.[10]

## Legal levels of alcohol in the blood

Binge drinking causes severe impairment of judgement and can lead to road accidents. There is also a well-marked association between binge drinking and head injury particularly in more severe accidents. Although of major importance for the understanding of the immediate causes of the crash, a post mortem alcohol test detects only recent use of alcohol whereas the question of chronic use is more difficult to elucidate by alcohol tests only. Karhunenen and Pentula[11] found that one third of heavy consumers of alcohol died without alcohol detectable in their blood. Ostrom and Eriksson[12] reinforce the view that significant impairment is possible even when the BAC is below the current legal limit. The European Commission has made alcohol one of the key health determinants in the European Union. The seminal report by Anderson and Baumberg[13] strongly recommends a maximum BAC limit of 50 mg/100 ml throughout Europe with a lower limit of 20 mg/100 ml for young drivers and drivers of public service and heavy goods vehicles. Some countries such as Slovakia, the Czech Republic and Sweden already have levels below 50 mg/100 ml and the Anderson and Baumberg report recommends that these countries retain these lower levels. Moskowitz and Fiorentino[14] in their analysis of driver impairment found that 27% of the studies reported impairment by 39 mg/100 ml, 47% of studies reported impairment by 49 mg/100 ml, and 92% of studies reported impairment by 79 mg/100 ml. Moskowitz[15] has demonstrated that major driving-related skills were impaired by BACs as low as 20 mg/100 ml. Moskowitz and Robinson[16] concluded that scientific evidence supported a reduction of the BAC driving limit to 50 mg/100 ml. The need therefore is for a European harmonised level of 50 mg/100 ml. At the interface between the teachable moment of public health and the opposing interest of those who resist change lies the challenge of accident reduction. Seasonal publicity campaigns, random breath testing, and the potential use of lockmeters on cars address part of the problem but successful accident reduction depends upon the excellence of medical advocacy.

The correct interpretation of blood alcohol levels and clinical effects requires considerable knowledge of the many factors which affect absorption and metabolism in the body. The correct strategy for reduction of accident fatalities must be to reduce the level of impairment, and in order to do this one must lower the legal blood alcohol limit from 80 mg/100 ml to 50 mg/100 ml.

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