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# Effects of Alcohol and Other Drugs on Driver Performance

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# Effects of Alcohol and Other Drugs on Driver Performance

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In the past century we have learned that driving performance is impaired by alcohol even in low dosage, and that many other drugs are also linked to impairment. This article is a summary of some of the more relevant studies in the past fifty years—an overview of our knowledge and unanswered questions.

There is no evidence of a threshold blood alcohol concentration (BAC) below which impairment does not occur, and there is no defined category of drivers who will not be impaired by alcohol. Alcohol increases not only the probability of collision, but also the probability of poor clinical outcome for injuries sustained when impaired by alcohol. This article samples the results of the myriad studies that have been performed during the last half century as experiments have moved from examination of simple sensory, perceptual, and motor behaviors to more complex measures of cognitive functioning, such as divided attention and mental workload. These more sophisticated studies show that significant impairment occurs at very low BACs (<0.02 gm/100 ml).

However, much remains to be determined regarding the more emotional aspects of behavior, such as judgment, aggression, and risk taking. Considering that the majority of alcohol-related accidents occur at night, there is a need for increased examination on the role of fatigue, circadian cycles, and sleep loss.

The study of the effects of drugs other than alcohol is more complex because of the number of substances of potential interest, the difficulties estimating drug levels and the complexity of the drug/subject interactions. The drugs of current concern are marijuana, the benzodiazepines, other psychoactive medications, the stimulants, and the narcotics. No single test or group of tests currently meets the need for detecting and documenting impairment, either in the laboratory or at the roadside.

**Keywords** Alcohol Impairment; Drug Impairment; Alcohol; Drugs; Driver Performance; Traffic Safety; Driving Skills; Impairment Tests

Few people in developed countries can imagine life without their car. While most drivers would know the economic cost of their vehicle, how many of them stop to count the social cost, or to consider the associated morbidity and mortality? Collisions involving vehicles have left countless people crippled, maimed, or bereaved. Road trauma is the most common cause of premature death among young adults in most countries not embroiled in war. Although transport safety continues to be improved by better engineering of roads and vehicles, the most prominent causal factor in collisions remains the human element.

Transport mishaps related to alcohol impairment are not new. It was common knowledge in the pre-industrial age that drunkards were at increased risk when riding horses or driving horsedrawn vehicles, but to a limited extent a good horse could share the responsibility for safe transport with an incompetent driver, compensate for human impairment, and plod homeward in relative safety. The advent of the horseless carriage brought the era of forgiving transportation to an end. The consequences of combining recreational drug usage and transportation became more serious.

One of the most remarkable features about vehicle driving is that a very large fraction of the human race can master the basic concepts and acquire rudimentary skill at the task relatively quickly and without expending large amounts of time or energy (Evans, 1991). Yet the complexity of the man-machine-traffic interaction can make the tasks of driving complex and hazardous.

Although the increased injury toll associated with motor transportation became apparent soon after the beginning of the twentieth century, there was only limited research on driving

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behavior in general, or driving under influence of drugs or alcohol in particular. It was perhaps the massive increase in private motor vehicle ownership after World War II (WWII) that led to the increasing research concern with the human costs of mass private transportation. Prior to the 1950s, for example, although there were frequent prevalence studies on alcohol in driving accidents, there was only one primitive case-controlled study. The 1950s saw the beginning of a major spurt in epidemiological studies of driving accidents and the influence of alcohol.

The growth in motor vehicle usage was matched by growth in research into the behavioral effects of alcohol. A series of large case-controlled epidemiological studies after WWII provided the evidence for the close association between increased BAC and increased accident, injury, and fatality rates. These pioneering studies included Lucas et al. (1955) in Toronto, Canada, Vamosi (1960), in Bratislava, Czechoslovakia, McCarroll and Haddon (1962) in New York City, and Borkenstein et al. (1964) in Grand Rapids, Michigan. These studies were highly influential in making the public aware of the real accident frequency associated with alcohol.

At the same time the nature of the impairment that underlies the increased crash rate was subjected to research at several levels: studies of the individual components of the driving task, studies of simulated driving, and studies of on-road driving. This article has its major focus on alcohol because alcohol is associated with as many fatal collisions as all other drugs combined. For instance, Drummer et al. (2004) describe the toxicological analysis of 3,398 collisions in which the driver died. Of these, 1,704 cases (50.1%) screened negative for alcohol and drugs. Alcohol was present in 29% of cases (n = 990), drugs were present in 27% of cases (n = 907).

Unfortunately, there have been only a few studies that have examined the nature of the driving impairment from an epidemiological perspective. This is partly because of the difficulty in assigning a particular driving deficit to an associated behavioral failure, since the same driving error could be due to many behaviors.

Police descriptions of crashes are typically assigned to the cause of current interest. For example, Moskowitz (2004) examined a series of crashes at T-intersections where drivers drove through into the wooded area beyond the junction where the road no longer existed. From the 1950s through the 1980s the majority of police reports characterized this as "loss of control." Nowadays "inattention" has become a favorite explanation, although there is no evidence that driver behavior has changed. There have only been a few multi-disciplinary accident investigation studies with professional human-factors personnel aware of research in cognitive psychology. More studies of this type would help enlighten the epidemiological record.

Moskowitz and Robinson (1988) and Moskowitz and Fiorentino (2000) have extensively reviewed the experimental literature on alcohol and driving. Jones et al. (2003) have reviewed the literature on drugs. This article does not purport to be a comprehensive literature review, so much as an overview of the field and the directions research has taken.

# ALCOHOL

Alcohol is a drug whose main acute effect is on the central nervous system. Unlike anesthetic agents that depress all brain functions, the effects of alcohol are first manifest in the brain centers involved in highly integrated functions, such as skilled performance. The analysis of sensory information, the control of intricate movement patterns and short-term memory are especially sensitive to alcohol. The effects on human skills and performance commence at the lowest measurable BACs and increase in a roughly dose-related manner (Moskowitz & Robinson, 1988).

There is no evidence of a threshold effect for alcohol because some impairment of performance occurs at the lowest levels that can be measured; nor is there a level at which a sudden transition from unimpaired to impaired can be expected: whatever the level of BAC examined, at least some skills can be demonstrated to be significantly impaired. The effects of alcohol are dependent both on the quantity consumed and the nature of the performance required. There is no evidence that low BACs improve any human skill (Moskowitz, 1985).

The variation in individual performance at BACs below 0.10% is sufficiently broad that uncertainties must attach to any prediction of the precise effects of a given quantity of alcohol on an individual. All individuals are impaired at any level of alcohol and the impairment increases as the BAC increases. Individual psychomotor abilities vary at the no-alcohol baseline, so that viewing an individual behavior at low BAC can give only limited information about the BAC. Conversely, as impairment can be demonstrated at all levels of BAC there is general agreement that the skills relating to driving can be presumed to be adversely affected below 0.10% (the limit in many jurisdictions) and many of the skills related to driving are significantly impaired below 0.05% (Dunbar et al., 1987; Mitchell, 1985; Moskowitz, 1985; Starmer et al., 1988). Moskowitz and Robinson (1988) concluded: "The legislature is free to prohibit driving at any B.A.C., since such a limit would not contradict the scientific data demonstrating no lower limit to impairment."

#### Subjective Measures of Impairment

For the purpose of this article, impairment has been defined as the occurrence of a change for the worse in the performance required for safe driving. Impairment is not the same as intoxication. Dictionaries give multiple definitions of intoxication using terms like "drunk," "inebriation," and "stupefaction," which are not scientific concepts and have no relevance to the driving task.

When the layman thinks of impairment, he probably envisages the obvious signs of lack of judgment, poor self-control, and loss of gross motor skills. Widmark (1981) reported on the experience in Sweden of testing drivers arrested on suspicion of being under the influence of alcohol. Swedish law required these drivers to be examined by physicians at a police station using a standard seven-item behavioral test battery. At 0.15 g/100 ml physicians assessed only 50% of the arrested drivers as being under the influence and only assessed everyone over 0.26 g/100 ml as intoxicated. Perper (1986) describes a 87 patients entering an alcohol treatment program with positive a BACs. Twenty-four percent of the subjects over 0.20 g/100 ml showed no evidence of intoxication and 26% of those over 0.30 g/ 100 ml passed many behavioral tests. Physicians fared little better when grading patients in an emergency room. Those patients

evaluated as being sober were found to have a mean BAC of 0.272 g/100 ml. One patient declared not to be intoxicated had a BAC of 0.54 g/100 ml (Urso, 1981).

The probability of a police officer detecting drivers on the road with a BAC above the legal limit is estimated to less than 1% (Borkenstein et al., 1963). Wells (1997) examined the ability of police to detect impairment at the roadside. When police cleared drivers from a sobriety checkpoint as "not under the influence," researchers requested breath samples for confidential analysis. Eighty-seven percent of drivers between 0.05 and 0.079 g/100 ml were not arrested, 62% of those between 0.08–0.99 g/100 ml; 64% of those between 0.10 and 0.119 g/100 ml; and, 62% of those at or above 0.12 g/100 ml were not arrested. Whether contemporary education of police has improved this detection rate has not been tested.

The gross intoxication that the layman associates with being "drunk" bears no relationship to the impairment that is significant for road safety.

### PERFORMANCE MEASURES

#### Skills Related to Driving

The 1988 review by Moskowitz and Robinson of the effects of low levels of alcohol on driving-related behavior summarized studies on reaction time, tracking, concentrated attention, divided attention, information processing, visual functions, perception, psychomotor skills, and performance in simulators and on the road.

They obtained more than 500 studies from a sampling of the literature. From these 500 studies, 177 were selected for incorporation in the report because they met their criteria of adequate statistical analysis, sufficient information that the BAC at the time of behavioral testing could be determined, and the presence of placebo treatment. Of the 177 studies 158 reported impairment of one or more behavioral skills at one or more BACs ranging from 0.01g/100 ml to 0.10 g/100 ml and above. Only 19 studies failed to report impairment at the levels examined.

A more recent review of the literature summarized 112 studies published from 1981 to 1997 and screened using similar criteria (Moskowitz & Fiorentino, 2000). In this review 27% of the studies reported finding impairment by .039 g/100 ml, 47% by 0.049 g/100 ml and 92% by .079 g/100 ml. The greater sensitivity of the later studies may be accounted for by improvements in methodology, better instrumentation, and more frequent examination of multiple BACs. Some studies reported impairment at BACs less than 0.01 g/100 ml. The studies showing greatest sensitivity to alcohol impairment were on-the-road and simulator studies of driving, divided attention tasks, and measures of drowsiness. The least sensitive tests were simple reaction time and studies of critical flicker fusion. In between were tasks such as vigilance, tracking, perception, visual functions, and cognitive tasks. There is strong evidence that some driving-related skills were impaired with any departure from zero BAC. By 0.05 g/100 ml the majority of studies reported impairment of some skill by alcohol.

#### **Reaction Time**

Although reaction time is adversely affected by alcohol, the level at which significant effects are noted depends on the complexity of the reaction demanded and the complexity of the stimulus. Some studies have demonstrated deterioration at levels as low as 0.02%, but a level of 0.07% is needed to produce significant deficit with common tasks (Starmer, 1989). The nature of the stimulus and the reaction required are complicating factors in interpreting the reported results. In a complex task such as driving at night deterioration of reaction time is observed at low levels. Tiredness is an important factor in increasing reaction time (Corfitsen, 1982).

Simple reaction time is the only experimental variable that has failed to consistently and overwhelmingly demonstrate impairment by alcohol (Moskowitz et al., 2000). No systematic study of the variation in results from reaction time experiments has been undertaken to explain this variability. However, reaction time experiments involving complex situations tend to show more impairment at lower levels than simpler experiments with fewer demands. Since the driving task is intrinsically a complex task, studies on simple reaction time under the influence of alcohol appear less relevant than studies that are more analogous to the complexity of real driving.

#### Tracking

The ability to follow a complex path under the influence of alcohol was one of the earliest performance measures studied. Tracking is analogous to car control because the subject uses a control device, such as a steering wheel, to follow a target that moves on a screen or (in actual driving) to follow the contours of the road. Tracking is the essence of what most people have in mind when they conceive of car control.

Thirty years ago, Moskowitz (1973) reviewed tracking under the influence of alcohol and concluded that there was variability in alcohol effects depending on whether the tracking task was compensatory or pursuit. A compensatory tracking task has the operator observing only the difference between the desired position and the actual position of a controlled element and acting to reduce the error. A pursuit-tracking task has the observer attempting to follow a moving target on a course. Compensatory tracking tasks performed alone have generally failed to find alcohol impairment, except at very high BACs.

There is marked impairment of tracking at quite low alcohol levels if tracking is not the only task. The Moskowitz and Robinson report examined 28 tracking studies, where the tracking was accompanied by some additional task, and found that the BAC at which impairment occurs is as low as 0.02 g/100 ml (Moskowitz & Robinson, 1988).

# Vigilance

Concentration is not particularly sensitive to alcohol and no effects are demonstrated below 0.05%. However, when the task also involves speed and accuracy, such as clerical tasks, impairment can be demonstrated in the range of BAC from 0.005% to 0.009% (Nash, 1962). The most consistent finding is an increase in error rates (Starmer, 1989). Low doses of alcohol interfere with learning and adaptation to unfamiliar tasks (Ogden et al., 1995).

#### **Divided Attention Tasks**

Any experiment that requires subjects to do more than one thing at a time is highly sensitive to drug effects (Moskowitz, 1984). Deterioration is detected on some tests at levels below 0.02% and many studies show deterioration below 0.05%. Small quantities of alcohol impair the ability to perform a secondary task while driving, long before the effect on the mechanics of driving are demonstrable (Brown, 1970).

It has been suggested that one of the reasons for this deterioration in performance is that the alcohol-affected brain processes information more slowly (Moskowitz & Austin, 1983). Since the mental workload required to divide attention is a component of nearly all studies, the challenge is to isolate the effects of alcohol on divided attention from the effects on the constituent components of the task. A study by Moskowitz, Burns, and Williams (1985) demonstrated that divided attention performance was impaired for all subjects at a BAC of 0.015 g/100 ml.

#### Visual Functions

Vision is peculiarly sensitive to sedatives including alcohol causing abnormal eye movements, difficulty in accurate eye tracking of moving objects, impaired color discrimination, tunnel vision, and even temporary blindness (Colson, 1940; Grant, 1974; Thompson-Crawford & Slater, 1971; Wallgren & Barry, 1970; Wilkinson et al., 1974).

Alcohol may impede recovery from glare and impair visual acuity, although the studies have produced conflicting results. Moskowitz, Wilkinson, and Burg (1993) reviewed 112 studies of visual performance under alcohol, and suggested that the discrepancy in findings resulted from experimental techniques that confounded alcohol effects on glare and acuity with concomitant presence of other visual functions, such as search behavior, that have also been demonstrated to be impaired by alcohol. Alcohol effects on visual performance are most marked for moving objects or when there is a simultaneous demand to process other information (Adams et al., 1978; Zeidman et al., 1980).

Eye movement control is the most sensitive of the various components of eye function and is affected at very low BACs. A BAC of 0.04% is enough to induce nystagmus (Aschan et al., 1956; Grant, 1974; Katoh, 1988; Stapleton et al., 1986).

Moskowitz and Robinson (1988) reviewed 28 studies of optometric vision tasks such as saccade velocity, nystagmus, tracking, and acuity, and reported impairment at low BACs but estimated that the magnitude of the impairments were unlikely to be important for the driving tasks. More cognitive visual functions exhibit impairment at even lower BACs and are more likely to influence driving.

Alcohol changes the way that the subject uses vision. Belt (1969) used eye movement recordings in drivers on the road to demonstrate that BACs as low as 0.04 g/100 ml produced changes in the distribution of eye fixation. Similar results have been found by other investigators including a form of tunnel vision with fewer visual excursions to the periphery and a shift in the distribution and duration of eye fixation (Buikhuisen & Jongman, 1972; Moskowitz et al., 1976).

It seems that alcohol slows processing of visual information requiring longer time spent fixed on an object in order to perceive its nature. One consequence of this slower visual processing is that fewer fixations are possible in any given time, which in turn means that fewer things can be seen. Drivers are literally looking less, because each look takes longer under the influence of alcohol (Moskowitz et al., 1976). Alcohol-affected drivers are unable to discern the meaning of road signs until they are closer to the sign compared to driving when unimpaired (Davis W., 1998). This difficulty is even more evident with poor lighting (Hicks, 1976).

# Driving Skills

The actual skills required for driving have been studied in simulators, on the road in instrumented cars and as a cause of epidemic disease. The complexity of the task and the number of variables to be considered in "safe driving" make simple models impossible.

Driving simulators have been used because of the inherent safety advantage of simulated driving. The more complex the driving challenge, the lower the BAC at which errors occur. Steering errors are noticed at an alcohol concentration of 0.03% and collision frequencies rise. Subjects tend to ignore rules and instructions before reaching 0.05%. Subjects are more sluggish to correct positional errors and steering control responsiveness deteriorates after low to moderate doses of alcohol (Starmer, 1989). Drinking experience does not make any difference to driving ability (Laurell et al., 1990). Prior driving skill does not reduce impairment (Beirness & Vogel-Sprott, 1982).

Closed driving course assessments are also used as a model of on-the-road driving tasks. In common with the results of simulator testing, the more complex the task required when actually driving, the greater the deficit produced by alcohol. Increasing blood alcohol levels result in progressive impairment of driving performance. This impairment is clearly demonstrated in non-competitive drivers at 0.05% and in competition drivers at 0.08% (Starmer, 1989). The alcohol-impaired driver may use past experience and learning to cope with normal routine driving demands, but cannot do so in an emergency situation (Lovibond & Bird, 1971).

# EPIDEMIOLOGY OF VEHICULAR ACCIDENTS

#### Driving Survey Data

One way of studying the drinking driver is to study those people apprehended for driving offenses. Such surveys cannot provide information about the general population because they are, by definition, studies of a selected subgroup. Excluding "random breath-testing," police officers do not apprehend drivers randomly and police are not randomly distributed in time or place. Police patrol known "trouble spots" and select individuals because of their driving style, vehicle, or other attributes. The results of such studies are as much a measure of policing biases as of driver characteristics.

# Post Accident Surveys

Post-accident studies have similar limitations except that the driver is selected by involvement in a collision. Several controlled epidemiological studies have been performed. Borkenstein et al. (1964) performed an influential study in Grand Rapids, Michigan. They compared breath alcohol levels in roughly 6,000 crash-involved drivers with 7,600 control drivers who had not crashed. The probability of involvement in a collision was determined for each BAC by comparing the relative number of collision-involved drivers at each BAC in the crash group with the relative number of non-collision-involved drivers at the same BAC in the control group.

The Grand Rapids study indicated that the probability of causing an accident was a sharply rising exponential function of the driver's blood alcohol concentration. At 0.10 g/100 ml there was a roughly six-fold increase in crashes compared with the crash rate for drivers with no alcohol. At 0.15 g/100 ml the odds ratio was 25 to 1. Young drivers (16 and 17 years) had a five-fold increase in crashes with BACs below 0.04 g/100 ml. At every blood alcohol concentration, drivers under 21 years and over 70 years of age had greater crash rate than drivers age 25 to 45 years.

The original Grand Rapids report created some confusion with its J-shaped curve. It not only showed no increase in overall crashes for alcohol levels below 0.04 g/100 ml, it even suggested that drivers might do better with low levels of alcohol rather than none!

Many researchers have since argued that the Grand Rapids study failed to compensate for factors other than alcohol that influence crash rate. For valid comparison, the control group should have shared the characteristics that influenced outcome. The Grand Rapids study was biased by a zero BAC group with a greater proportion of both younger and older drivers than the "crash" group. Both younger and older drivers have higher crash rates with no alcohol present than drivers aged 25 to 55 years. Other variables that affect crash rates that were not equally distributed among the various groups in the study include educational level, number of miles driven, occupation, and frequency of drinking. Determining the relationship between BAC and crash probability requires controlling for these other variables. The Grand Rapids data has subsequently been analysed by several groups using more sophisticated statistical methods revealing some apparent paradoxes (Allsop, 1966; Hurst, 1973). Daily drinkers had the lowest accident rate compared to weekly, monthly, or yearly drinkers. The youngest and oldest drivers, who tend not to drink daily, have higher crash rates than 25–55-year-olds who might be drinking daily. Once the variable "drinking frequency" is controlled, the probability of involvement in collision increases with any departure from zero BAC and the rate of increase is greatest for the least frequent drinkers. The curve loses its J shape.

The US Department of Transportation sponsored another epidemiological study of alcohol and crash probability, collecting data from drivers involved in crashes in Long Beach, California, and Fort Lauderdale Florida for more than a 12-month period (Moskowitz et al., 2000, 2002). This study represented an improvement over prior study designs by sampling control drivers at the same site, time, and direction of travel as the original crash drivers. Two control drivers were obtained for each crash driver one week after the collision. Extensive efforts were made by the police to capture as many hit-run drivers as possible. BAC estimates were obtained from drivers who refused to participate by passive breath sampling techniques. These factors proved important since more than 69% of the apprehended hit-run-drivers had positive BACs, typically in the higher ranges. Almost 50% of the crash drivers who refused to participate had positive BACs in contrast to fewer than 16% of the control drivers who refused to participate.

Had it not been for the apprehension of some 20% of the hit-run drivers and the use of the passive alcohol sensors to determine alcohol presence in drivers who refused to participate, it was estimated that more than 46% of all drivers involved in crashes who had positive blood alcohol would not have been detected. The study used logistic regression analysis to adjust the control and crash samples for variation in age, sex, drinking practice, and other variables. This improved analysis of the probability of crash involvement as a function of BAC indicated that crash probability increased for alcohol involved drivers at all levels from 0.01 g/100 ml, and the probabilities for crash involvement were considerably greater than in any other prior study.

# Single-Vehicle Collisions

The Grand Rapids study reported that the probability, of a single vehicle collision at various BACs was greater than that of a multiple-vehicle collision. That finding was supported by reports in the 1950s and 1960s that roughly 70% of fatally injured drivers in single-vehicle collisions had alcohol present (NHTSA, 1997).

Zador (1991) argues that single-vehicle crashes provide the only true measure of the contribution of alcohol to increasing the rate of crash involvement. Non-impaired drivers may be able to compensate for the impairment of other drivers and so avoid becoming involved in collisions. When Zador examined the probability of fatal single-vehicle crashes involving alcohol as a function of driver age and sex using data from the National Highway Traffic Safety Administration, he determined that 0.02–0.04 g/100 ml BAC increased fatal crash involvement by 40%; BACs between 0.05 and 0.09 g/100 ml increased fatal crash involvement by 1,100%; BACs between 0.10 and 0.14 g/100 ml increased fatal collision probability by 4,800%; and at levels of 0.15 g/100 ml or higher, the fatal collision rates increased by 38,000%. Thus the role of alcohol in fatal crashes is even more significant than the Grand Rapids report suggested for all collisions.

Stein (1990) looked at drivers at 0.10% or above and found their chances of being involved in a fatal collision were 100 times greater than the sober driver regardless of time of day. However the sober driver's chances of being in a fatal collision with such a driver rose dramatically between 1 A.M. and 3 A.M. because of the increased concentration of drunk drivers in the early hours of the morning.

# **RATE OF ALCOHOL CONSUMPTION**

The rate at which consumption of alcohol has been undertaken influences the outcome. Moskowitz and Burns (1976) studied four groups of subjects who drank alcohol at different rates to achieve a peak BAC of 0.10 g/100 ml, and a control group with a placebo beverage. The 40 subjects were tested on a performance battery that included measures of information processing, motor control, hand steadiness, and body sway. The duration over which they drank ranged from 15 minutes to four hours. The group that consumed the greatest amount of alcohol was the group that took the longest period of time to achieve 0.10 g/100 ml, since they were eliminating alcohol as they were consuming it. Nevertheless, the most impaired individuals were in the group that drank the fastest, even though they drank the least total amount, and the least impaired were in the group that drank the greatest amount at the slowest rate.

# ALCOHOL AND FATIGUE

Sleep disorders have become a recognized medical speciality, and the last decade has seen increased interest in the effects of alcohol on sleepiness (Lyznicki, Doege, Davis, & Williams, 1998), particularly the effects after the BAC has dropped to zero.

Roehrs et al. (1994) compared subjects given sufficient alcohol to produce a peak level of 0.06 g/100 ml at 7.30 A.M., alcohol sufficient to reach a peak at 0.04 g/100 ml given at 10.30 A.M., or placebo. By 3:30 P.M., all subjects were at zero BAC. Subjects were tested for sleep latency (time to fall asleep) at two-hour intervals from 9:30 A.M. until 9:30 P.M. Subjects displayed a shortened time to fall asleep throughout the entire period when alcohol was present and even when the BAC had dropped to zero, compared with the placebo treatment day.

Taking a nap normally combats fatigue and increases the time required to fall asleep. A dose of alcohol that produces a peak level of 0.04 g/100 ml counteracts the effect of the nap (Roehrs et al., 1989).

These laboratory studies are relevant to real-life driving situations. In New Mexico there was an increase in the number and proportion of alcohol-related traffic crashes during the seven days following the change to and from daylight-saving, compared with the week before and the second week after the changes (Hicks et al., 1998).

# HANGOVER

The after effects of alcohol intoxication have been shown to persist after the BAC has fallen to zero. Many people with a hangover report feeling unwell and impaired. Measurable effects of hangover include hormonal changes, depression of brain activity, difficulty with judgment of space-time relationships, irritability, and poor concentration.

The degree of "hangover" is not easily measured, but impairment has been demonstrated in driving simulators, flight simulators, skiing, and administrative tasks. The impairment persists for at least three hours after all the alcohol has been metabolized (Collins & Chiles, 1980; Delin & Lee, 1992; Lemon et al., 1993; Yesavage et al., 1986; York & Regan, 1988).

# ALCOHOL AND AGGRESSION

The last decade has seen increasing concern about aggressive driving and the phenomenon the media call "road rage." There is no hard epidemiological evidence linking aggressive driving and alcohol consumption, but there is extensive laboratory evidence showing increased aggressive behavior under alcohol. Bushman and Cooper performed a meta-analysis of thirty experimental studies and concluded that the evidence supported the conclusion that alcohol causes aggression in male "social drinkers" (Bushman & Cooper, 1990).

There is a vast literature in sociology, criminology, and psychology linking alcohol to acts of violence, as well as the individual characteristics that may predispose some individuals to violence and the situations that promote its expression (Brain, 1986). It has been suggested that alcohol reduces inhibition and unmasks underlying aggressive tendencies. The extent to which this is reflected in crash statistics is not yet known.

# ALCOHOL AND DEGREE OF INJURY

Alcohol not only reduces performance and affects behavior, but the "use of alcoholic beverages predisposes to more severe and extensive injury than would be experienced by non-drinkers given impact of the same severity" (Committee on Trauma Research, 1985). There is an extensive trauma literature on this subject, which is beyond the scope of this article.

Animals subjected to a standardized force with and without alcohol demonstrate increased trauma with alcohol (Brodner et al., 1981). Humans experiencing trauma have altered hormonal responses when alcohol is present that may influence outcome (Woolf et al., 1990), and alcohol affected trauma victims are likely to have sustained more injuries (Fabbri et al., 2001). Waller et al. (1986) examined over a million collision reports in North Carolina from 1979 to 1983. When they controlled for a wide variety of factors such as crash severity, type and weight of vehicle, speed, driver age and sex, and seatbelt use, they found that the presence of alcohol increased the probability of being killed in an collision 225% over that of a matched non-alcohol involved driver.

Evans and Frick (1993) used fatal crash data for two-car crashes where at least one driver was killed and controlled for factors such as relative weight and impact areas. They determined that the presence of a BAC of 0.10 g/100 ml roughly doubled the risk of death from a given impact and a BAC of 0.25 g/100 ml tripled the probability of death.

# DRUGS OTHER THAN ALCOHOL

#### Epidemiology of Drugs and Crashes

While alcohol remains the dominant drug causing impairment of driving performance, other drugs, especially in combination with alcohol, increase collision risk. Reviewing the history of 43,000 outpatients, Skegg et al. (1979) found that the 53 crash-involved drivers in that sample were 4.9 times more likely than their matched controls to have been using a tranquillizer. The relative risk of a driver being killed in a traffic crash (assessed by odds ratio analysis) shows a significant increase for drivers consuming alcohol alone, alcohol with other psychoactive drugs, combinations of psychoactive drugs, and cannabis (Alvarez et al., 1992a, 1992b; Alvarez et al., 1997; Drummer & Gerostamoulos, 1998; Drummer et al., 1998).

Impairment can be predicted from known or expected effects of medication on:

- Alertness (e.g., sedation, stimulation)
- Vision (e.g., visual blurring, delayed recovery from glare)
- Function (e.g., impaired coordination or movement)
- Performance (e.g., impaired performance on skills testing)
- Psycho-social (e.g., changes in behaviour, risk taking)
- Cognition (e.g., changes in processing information)

This information is available from the pharmacology of certain substances, reports of adverse drug reactions, epidemiological data, and specific testing (Ogden & Brous, 1999).

# Major Problems in Interpreting Data on Drugs and Driving

There are many major problem areas that need to be considered when attempting to show the correlation between drug consumption and road trauma.

*Proof That the Drug Has Been Consumed.* Proof of drug consumption requires analysis of a body fluid to identify the drug. There is a large number of potential drugs that could be screened, and many of the drugs of interest may only be present in minute quantities while having significant effects.

*Could the Amount of Drug Detected Produce Impairment?* The fact that a substance is found does not mean that it caused impairment. It is necessary to ask a series of questions: Does this substance cause impairment of human skills? If so, is such impairment universal or idiosyncratic? Does the impairment occur in normal dosages or only when the drugs is used in excess? The presence of a drug may not necessarily mean the driver is impaired (Maki & Linnöila, 1976). There is considerable information on the clinical use of some drugs and on the normal levels expected and on what constitutes a "toxic" concentration (Baselt et al., 1975; Uges, 2004). The clinical concepts of "therapeutic" and "toxic" do not necessarily correlate with impairment. Some individuals will be impaired with levels of a drug normally considered therapeutic (e.g., sedatives), while dangerously toxic levels of other drugs may have no effect on driving skills (e.g., paracetamol) (Pearl et al., 1989). There is no critical level of most drugs above which impairment is present or below which no impairment can be demonstrated (Starmer et al., 1988).

While we are interested in the behavioral effects of drugs, presumably due to activity at some site in the brain, we are limited to taking samples from peripheral sites in the body. The drug levels in blood, urine, saliva, hair, etc., may be

- 1. quite different from that in the CNS and
- 2. not well correlated over time as levels change at the central and peripheral sites.

*Could This Amount of Drug Have Contributed to the Crash?* There are a number of individuals whose behavior and functioning is considerably improved by prescription medications, and without which they would not be fit to hold a drivers licence, such as anti-convulsants for epilepsy. Withdrawal of such drugs may produce a considerable deterioration in driving performance.

The alcohol literature has relied heavily on the utility of measuring the blood alcohol concentration. Alcohol is a relatively easy drug to study: it is taken in large quantities; it is watersoluble; the concentration is easy to measure; and impairment is effectively dose-related. This paradigm does not translate to other drugs that may be impairing in miniscule doses; be protein bound or sequestered into fat; be hard to quantify; and the blood levels may have no correlation with impairment.

THC is a good example of the problems understanding the relationship between drug usage and impairment. When marijuana is smoked, THC in the inhaled smoke is absorbed within seconds. Peak blood levels appear about the time smoking is finished. The cannabinoids are rapidly distributed into fat and blood levels fall within minutes. Maximum impairment is observed an hour after smoking, when THC levels are about 5 to 10% of the peak (Tzambasis, 2001). The half-life of THC is estimated to be as long as 10 days and metabolic products can be found for several weeks after exposure. The presence of THC metabolites is evidence of drug exposure and not of impairment.

The work of Terhune in the United States (Terhune et al., 1992) and Drummer's group in Australia (Drummer & Gerostamoulos, 1998; Drummer et al., 1998; Robertson & Drummer, 1994) has examined the culpability of fatally injured

Table I Risk of culpability for fatal collision

Drug(s)	Percentage of cases	Culpability odds ratio
Alcohol alone	24	9.1
Alcohol plus drugs	9	11
Psychoactive drugs	2	3.4
Drug combinations	3	4.6
THC > 5 ng/ml	1	3.0
Benzodiazapines	4	2.4
All psychotropics	13	1.5
Stimulants	3	1.4

drivers. They have each used the odds ratio to indicate the relative importance of various drugs in fatal collision causation. Drummer summarized this work in Table I (Drummer, 2002).

These results must be taken cautiously, because epidemiological studies to evaluate the role of drugs such as cannabis in live drivers are fraught with difficulties. First, the rate at which subjects agree to participate in providing body fluid samples for drug testing is far below that found in alcohol studies. The results of studies where voluntary participation rates are only in the 80% range may suffer considerable bias.

Second, relative risks for death may be very different from the risk of injury or non-injury collision. Studies of fatal collision may not be comparable with studies of injured or non-injured drivers.

Third, the probability of crash involvement is also a function of non-drug factors including geographic area, traffic conditions, vehicle characteristics, and the individual characteristics of the driver. Few studies have obtained data that would permit the separation of the possible effects of a drug in collision causation from all the other factors that determined the event.

For example, in the United States the National Highway Traffic Safety Administration estimates that alcohol is present in 8% of all motor collisions. Even if one assumes that alcohol was the sole cause of all of those crashes, it leaves 92% of the crashes as due to other causes. How do we determine the degree to which those other causes are also present in alcohol-related crashes? How are the contributions of the various factors to be separated or proportioned? The difficulty of performing epidemiological studies even with respect to cannabis, the most frequently studied drug other than alcohol, can be seen in two recent reviews of the literature that reached differing conclusions. Bates and Blakely, (1999) concluded that "... there is no evidence that consumption of cannabis alone increases the risk of culpability for traffic crash fatalities or injuries for which hospitalization occurs, and may reduce those risks." Ramaekers et al. (2004) concluded that drivers who had recently used cannabis were "about three to seven times more likely to be responsible for their crash as compared to drivers that had not used drugs or alcohol."

#### Impairment Tests

Given the difficulty of obtaining appropriate biological samples, getting timely analysis, and interpreting the result, law enforce-

ment measures aimed at drug-impaired driving have been dependent on behavioral tests to demonstrate impairment (Ogden, 1995). There is worldwide interest in roadside testing for drugs and the establishment of per se definitions of impairment.

The probability of being arrested for driving while impaired by alcohol was estimated to be extremely low with a risk estimated at 0.001 per trip made while intoxicated. This led the US Department of Transportation to commission work during the late 1970s to develop a standardised field sobriety test battery that would facilitate the accurate recognition of intoxicated drivers in the field.

Burns and Moskowitz carried out two large research projects involving over 500 subjects (Burns & Moskowitz, 1977; Tharp et al., 1981). Police officers tested laboratory subjects who had consumed alcohol to simulate intoxicated drivers. They identified three tests (Horizontal Gaze Nystagmus, Walk and Turn, One-Leg Stand) that reliably identified alcohol intoxication (defined BAC > 0.10%). This battery of three relatively straightforward tests was recommended for adoption by police as a roadside screen for sobriety, The three tests became collectively known as the Standardised Field Sobriety Test (SFST).

In the laboratory studies, police officers' estimates of BAC differed from the measured concentration by an average 0.03%. They were able to correctly classify subjects above or below 0.10 g/100 ml, 81% of the time.

Anderson et al. (1983) found that different police departments had predictive accuracy between 76% and 96%, suggesting that reliability may be related to training and careful adherence to protocol rather than inherent test validity. Drugs other than alcohol may have contributed to the apparent over-classification.

At the roadside, "No decision" is not an option for operational police (Burns, 1991): a decision must always be made. It seems natural that police will err on the side of caution when making roadside assessments: better to make an incorrect release than make an incorrect arrest. "It is apparent that the 'arrest' criterion is lower in the laboratory. The penalties for mistakes in a laboratory setting are, of course, fairly trivial when compared to a real world setting" (Burns & Anderson, 1995). A Finnish study with more than 5,000 subjects found that observations of nystagmus combined with tests of balance and walking was the best screening tool for alcohol (Pentilla et al., 1974).

Evidence of drug impairment traditionally relied on physicians with the appropriate experience and interest. The Los Angeles Police Department pioneered the training of police officers to perform these field evaluations and give expert evidence on drug effects. The program allowed prosecution for drug impaired driving in three discrete steps:

- 1. The arresting officer establishes impairment and calls for a Drug Recognition Expert.
- 2. The drug recognition officer establishes that the impairment is likely to be due to a drug in a particular class.
- 3. The laboratory confirms that a drug in that class is present in blood or urine.

Enthusiasm for the abilities of the police trained Drug Recognition Expert (DRE) performance should be restrained. There are no scientific studies utilizing adequate blind controls to examine the ability of DRE trained officers to correctly identify an individual as drug impaired, and/or identify the drug classification. The studies performed have been primarily field observational studies. It is interesting to note that when one examines the ability of police officers to detect and identify the presence of alcohol beverage on an individual's breath (with adequate controls) how much the results vary from commonly held police opinions as to their ability to detect whether a person has been drinking (Moskowitz et al., 1999).

The DRE program has been refined and renamed Drug Evaluation and Classification (DEC). SFST procedures remain at the core of this process with the addition of some physiological data (pupil reaction, pulse, blood pressure, finger-nose test) to aid drug classification.

No battery has been standardized against the broad range of drugs that are implicated in driving impairment. Tzambasis has shown that observation of head movements and jerks improves the discrimination of SFST for impairment due to marijuana (Tzambasis, 2001; Tzambazis et al., 2000, 2001), but similar work has not yet been done with other substances.

# SPECIFIC DRUGS

There are many ways to look at the literature on drugs and driving. The alcohol review was arranged according to experimental methodology and performance variables. A similar approach to other substances would give a coherent overview of the research methodologies, but make it difficult for the reader to appreciate the specific effects of individual substances. We have chosen to present the data by drug because of the practical implications, acknowledging that this is done at the expense of presenting a somewhat disjointed overview.

#### Marijuana

Marijuana is the common term given to the leaves of the plant *cannabis sativa*. It has been used for centuries in various parts of the world, and has become a popular recreational drug throughout the western world.

Of the many chemical compounds in its leaves, delta-9 tetrahydrocannabinol ( $\Delta$ 9 THC or THC) has been identified as the major psychoactive component. THC has significant effects on the human brain in tiny concentrations both at the time of consumption and long term.

The most frequently detected metabolite of THC is 11-norcarboxy-delta-9-THC (often called THC-carboxylic acid), which is inactive. THC-carboxylic acid can be detected in blood, urine, and other tissues for an extended period. It has a metabolic half-life of 33–40 hours, but its half-life in fatty tissues is significantly longer because of its affinity for fat. It can take up to a month to be eliminated beyond the detection limits of modern toxicological techniques. The detection of the metabolite in a biological sample is therefore evidence of the consumption of cannabis within the last month, but does not enable a more accurate estimate of when it was consumed nor quantification of the dose consumed.

There is no biological measurement of cannabinoid concentration that allows direct estimate of cannabis-induced impairment of driving skills as exists for alcohol (Chesher et al., 1986). Tzambasis's (2001) observation that impairment was greatest 70 minutes after smoking when THC levels had fallen to 5 to 10% of peak level demonstrates this point.

Drivers under the combined influence of marijuana and alcohol have an increased likelihood of initiating a crash and the combination produces an additional decrement in performance of driving related tasks (Burns & Moskowitz, 1980; Chesher et al., 1986; Drummer, 2002; Drummer & Gerostamoulos, 1998; Drummer et al., 1998; Klonoff, 1983; Perez-Reyes et al., 1988; Ramaekers et al., 2004).

Early studies on the effects of marijuana on simulated driving performance established that some driving variables are impaired by the consumption of marijuana. In particular subjects appeared to have delayed or inappropriate reactions, attention deficits, poor speed and distance judgment, and poor hazard perception. People affected by cannabis tend to travel slowly and avoid risk. It is unclear to what extent this is due to conscious recognition of impairment rather than distortion of judgement of time and distance.

More recent studies in more realistic driving simulators show that marijuana increases the variability of speed control and road position. Marijuana-affected participants tend to hit obstacles, miss signs, have delayed responses to the need to change speed (both braking and accelerating are inappropriate), and drive more slowly than when unaffected (Drummer, 2002; Drummer & Gerostamoulos, 1998; Drummer et al., 1998; Tzambasis, 2001; Tzambazis et al., 2000, 2001).

There have been several on-road driving studies examining the effects of cannabis on driving performance. Results show that marijuana results in poor car handling, with drivers exposed to high doses of marijuana five times more likely to strike cones on a driving task than when not affected by the drug (Klonoff, 1974, 1983; McBay & Owens, 1981; Ramaekers et al., 2000).

There does not appear to be a "hangover effect" of the sort seen with alcohol and long-acting sedatives (Chait, 1990; Chait et al., 1985), however there is some prolonged impairment of skilled performance. Leirer et al. (1991) studied nine experienced licensed pilots performing a simulator flight with numerous response variables before and after smoking a cigarette containing 20 mg of delta 9-tetrahydrocannabinol (THC). Marijuana impaired performance at 0.25, 4, 8, and 24 hours after smoking. At 24 hours, only one pilot reported awareness of drug effects.

THC levels greater than 5 ng/ml are associated with a threefold increase in the risk of being responsible for a fatal collision (Drummer, 2002). Yet several other epidemiological studies have failed to find above baseline fatality rates for use of cannabis alone (Bates & Blakely, 1999). The combination of marijuana and alcohol severely impairs performance (NHTSA, 2000).

# Anti-Anxiety Drugs

The benzodiazepine group of drugs includes minor tranquillizers, sedatives, anticonvulsants, and hypnotics. Representative members of the group are diazepam, oxazepam, nitrazepam, and flunitrazepam. These drugs have largely taken the place of the barbiturates in the treatment of anxiety and insomnia because of their efficacy and safety even with overdose.

Different members of the class depress the central nervous system to varying degrees and in qualitatively different ways. Some are better at relieving pathological anxiety and agitation and are classified as tranquillizers. Others are more sedating and hence are used to treat insomnia. Some members of the group are primarily used in epilepsy as anticonvulsants. There is no sharp distinction between any of these effects and higher doses of any of the benzodiazepines may induce sedation and coma.

Berghaus and Grass (1997) reviewed over 500 experimental studies of driving related tasks. They showed that the serum level of each of the benzodiazepines studied was related to the degree of impairment in the laboratory.

The data suggest that there is an increased risk of personal injury crashes among drivers using anti-anxiety drugs compared with the rest of the population (Skegg et al., 1979) and this is exacerbated by alcohol (Seppälä et al., 1976a). There is a hangover effect and a small dose of alcohol the following day can potentiate the effect. There is a decrement in tasks requiring vigilance at low doses and tolerance is only occasionally noted. The opposite effect, exaggerated impairment, has also been documented (Kolega, 1989).

The benzodiazepine group has been shown to impair driving skills to a similar degree and in similar ways to alcohol. Thomas (1998) concluded that the risk of collision was doubled for patients taking benzodiazepines. The impairment and collision risk are greatest in the first two weeks of treatment (de Gier et al., 1981). The ICADTS working group concluded that patients should be warned not to drive in the first two weeks of treatment (Alvarez & de Gier, 2002). However, not all research has found an association between sedative use and collision risk (Jick et al., 1981). de Gier (1993) reported that clinically anxious patients are also poor drivers. Although treatment with benzodiazepine tranquillizers will improve clinical anxiety, there is no improvement in their driving ability.

# **Psychoactive Medication**

The untreated psychiatric patient is a potentially hazardous driver either because of the underlying illness and the consequent disorder of the mind, or of the associated psychomotor impairment. Once stabilized on medication, patients are better on their medication and a greater risk without it (Seppälä et al., 1976b; Smiley et al., 1981). There is some question about the generality of this conclusion (de Gier et al., 1981) as it has been based primarily on subjective clinical judgment without adequate research backing. There is some interaction with alcohol generally related to the sedative effects of some psychoactive drugs (Bauer, 1984; Hindmarch, 1984). Different members of each class of psychoactive medication have quite different effects on driving. For instance, the tricyclic antidepressants are quite impairing of driving skill, while the selective serotonin reuptake inhibitor (SSRI) antidepressants have lesser effect on driving and little or no interaction with alcohol (Pullen, 1999). Starmer and Mascord (1994) have stated that tricyclic anti-depressants, which are intrinsically sedative in nature and cause driving impairment in normal individuals, will improve the driving ability of depressed patients.

# Stimulants—Amphetamine/Cocaine

There are laboratory studies showing that small doses of stimulants can improve cognitive performance (De Wit et al., 2002; Wachtel & de Wit, 1999) and improve reaction time (Fleming et al., 1995; Halliday et al., 1994). On the other hand, amphetamines cause deficits in divided attention tasks and perception in the peripheral visual fields (Easterbrook, 1955; Mills et al., 2001).

Amphetamine variants (dexamphetamine, methamphetamine, and methylenedioxymethamphetamine (MDMA, or Ecstasy)) have been implicated in traffic fatalities (Drummer, 1994, 2002; Drummer & Gerostamoulos, 1998, 1999; Drummer et al., 1998). A review of the epidemiological evidence in 1987 by Hurst found little to support such a relationship.

There's been little laboratory work with driving simulators or on-the-road performance under amphetamines. However, using a driving simulator, Silber et al. (2004) found that 0.42 mg/kg dexamphetamine significantly impaired overall performance for daytime but not night-time driving, possibly because the visual field is restricted in the night-time simulation and peripheral cues are less important. During the daytime simulation, drivers signalled incorrectly and failed to stop at red traffic lights more frequently.

Laboratory studies are required that replicate the conditions under which the amphetamines are frequently used. Thus, for example, long-distance drivers often take methamphetamine repetitively and so the drug should be examined experimentally under similar conditions. Finally, it is known that methamphetamine depresses neurotransmitters in the brain for extended periods over a week or more, even following single-dose treatments. During that period subjects exhibit depressed behavior that should be examined for impairment.

#### **Opioid Analgesics**

The opiate drugs—heroin, methadone, codeine, and related compounds—are used for pain relief and the suppression of cough. They have a high addiction potential. Acute sedation and impairment is observed in a dose-related manner and there is a deleterious interaction with alcohol, although the effects are slight compared with alcohol (Chesher, 1989). Methadone is used for the long-term maintenance therapy of narcotic addicts. Long-term methadone maintenance is not associated with an increase in collision risk after the initial stabilization period (Chesher et al., 1995; Friedel & Berghaus, 1995; Friedrich et al., 1991; Moskowitz & Robinson, 1985).

#### Minor Analgesics and Anti-Arthritics

There are few central side effects of the common minor painkillers (aspirin, paracetamol) or of the non-steroidal antiinflammatory agents that are used for the treatment of arthritis.

# SUMMARY

Alcohol, the substance most frequently found in crashinvolved drivers, has been extensively examined in experimental and epidemiological studies. While behavioral areas such as judgment, emotion, cognition, and sleep requires further work, the existing large literature describes numerous behaviors impaired by alcohol and their role in traffic safety.

Other drugs have been shown to impair human performance, or have been implicated in epidemiological studies as increasing the risk of crashes. None have been examined in the same detail as alcohol over the wide range of possible behaviors. Such examination would enhance the ability to evaluate the role of these drugs in traffic. The increasing sophistication of behavioral measures examining cognitive processes and judgment will assist researchers in estimating drug safety.

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