EPIDEMIOLOGICAL AND LABORATORY STUDIES ON ALCOHOL, DRUGS AND TRAFFIC SAFETY

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EPIDEMIOLOGY

Alcohol

Epidemiological studies indicate that about 50% of all fatal automobile accidents in the United States involve alcohol use.[1] The most common legal criterion for alcohol intoxication in the U.S. is a blood alcohol concentration (BAC) equal to or in excess of 0.10%. Actually a large proportion of the population may be impaired on sensory and motor tasks at lower concentrations, as suggested by most epidemiological studies indicating a significantly increased risk of an accident slightly below or at 0.08%.[2]

Epidemiological studies show that a disproportionate number of fatal auto crashes involve 16-24 year old men. In one study, this group was found to be involved in 42% of alcohol-related fatal crashes but constituted only 20% of licensed drivers.[1] The reasons for this discrepancy are complicated.[3] The relative risk of a fatal automobile accident in the 16-19 year old group is higher than in the older age groups at the same BAC.[2] Roadside surveys show that the lowest frequency of high BAC's (>0.15%) occurs in the 16-17 year olds and that the frequency increases up to the 30-34 year age group.[4,5] The frequency of drinking and driving, according to self-report, is age dependent and increases with age.[6,7] Thus, inexperience with both drinking and driving may account for some of the disproportionate number of fatal auto accidents occurring in the younger group.

The higher prevalence of drinking among older drivers may partially explain why more of them are arrested. The arrest ratio (% of arrested DWI's at a given age ÷ % of fatally injured drinking drivers in the same age group) is lower for the <30 than the >30 age group.[8] A low arrest ratio is especially true for drinking drivers <20 years of age.

Drivers involved in alcohol-related fatal crashes are predominantly men[9] even though women tend to become more intoxicated and have higher BAC's after similar intake.[10] Even with the same BAC, female drivers tend to be more impaired and are more likely to become involved in an auto accident than men.[11] The degree of impairment at a given BAC may vary in women with phase of menstrual cycle or use of oral contraceptives.[12,13] The reason that the proportion of alcohol-involved accidents is smaller for women than men appears to reflect fewer women driving after drinking.[9]

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The majority of alcohol-related auto accidents (>50%) occur at the weekend, especially during the evening hours, and in the summer. In the United States there is also a variation in accident rate depending on locale, with more fatalities occurring in rural areas.[14]

Licit Drugs

In addition to alcohol, other drugs may contribute to an increased accident risk[15,16]. Although the presence of prescription and nonprescription drugs can be documented in drivers involved in accidents it is difficult to ascertain their contribution to the risk of a traffic accident. Drugs with various mechanisms of action, such as sedative/hypnotics, antianxiety medications, antidepressants and antihistamines can cause psychomotor impairment. Many of these drugs have been shown to impair skilled performance in the laboratory. The benzodiazepines cause psychomotor impairment when tested in the laboratory setting and their use appears to be associated with an increased accident risk. Neuteboom et al.[17] found that drugs were present in 9.7% of samples from drivers suspected of drunk driving in the Netherlands. Of drugs which might cause psychomotor impairment, the benzodiazepines were the most common. In a study performed in California on 15-34 year-old victims of fatal car accidents, alcohol was present in 70% and diazepam and phenylpropanolamine were found in 4 and 3% of the samples[18]. Skegg et al.[16] found, when he compared prescriptions for the previous 3 months in fatal auto accident victims to controls, that those involved in the accidents were 4.9 times more likely to have obtained a prescription for a minor tranquilizer such as a benzodiazepine. Honkanen et al.[15] also found that a specific benzodiazepine, diazepam, might be a contributory factor in 1-5% of car accidents in Helsinki. Futhermore, many benzodiazepines appear to produce sufficient hangover when taken as hypnotics to cause some psychomotor impairment in simulated, closed course and real road driving tasks. The benzodiazepines with longer halflives, e.g. flurazepam, cause more hangover than those with shorter halflives, such as triazolam [19,20].

In a recent review of the literature, Tsuang et al. [21] reported on the roles of psychopathology and personality variables in traffic accidents and concluded that it is not always clear which is the greater risk factor for accidents, the prescribed medication or an underlying illness. Although numerous studies have attempted to define personality variables associated with an increased risk for traffic accidents, few have studied this relationship in psychiatric populations. Eelkema et al.[22] performed the only case control study in which the pre and post hospital driving records of 238 discharged psychiatric patients were compared to 238 control subjects matched

for age, sex and locale. They found that psychotic and psychoneurotic men and women had higher prehospital accident rates when compared with controls'. However, their accident rates after discharge were lower than the controls'. Men with personality disorders showed higher accident rates than controls both pre and post discharge, and overall exhibited the least improvement after discharge.

Illicit Drugs

When illicit drugs are found they are often in combination with alcohol. In one study, young drivers from fatal crashes in California were tested for drugs and alcohol. It was found that 70% had alcohol and 37% marijuana in their blood[18]. The percentage of samples containing marijuana was higher than the 12% found in a Canadian study[23], although the percentages of drivers with alcohol were similar. In the California study, those with marijuana alone comprised 12% of the sample. Other illicit drugs found in 3-11% of samples included cocaine, phencyclidine and methamphetamine. More than 90% of the alcohol positive drivers were judged responsible for the accident. In the 19 drivers positive for marijuana alone, responsibility for the accident was assigned to 53%.

LABORATORY

Most studies measuring psychomotor effects of drugs and alcohol have been performed in the laboratory. This makes it difficult to extrapolate their results to actual driving, although well designed laboratory studies can provide insight into mechanisms of impairment.

Driving is a complex activity and even relatively simple maneuvering of a car requires perception, information processing and motor coordination. Important for safe driving is the ability of the driver to process information quickly and accurately. A number of tests have been designed to investigate information processing. Tests involving divided attention show impairment at BAC's < 0.05%[24,25,26]. Moskowitz et al.[24] have also reported that marijuana causes a dose-related impairment in the detection of stimuli located in the periphery of the visual field, probably reflecting impairment in information processing.

Vigilance is a measure of the ability of an observer to perform a monotonous task for a prolonged period of time. Laboratory measures of vigilance may provide information relevant to long distance driving on relatively empty highways. Moskowitz and DePry[27] found no impairment in auditory vigilance following a dose of alcohol calculated to produce a BAC between 0.07 and 0.08%. However, Erwin et al.[28] found an increased number of errors of omis-

sion in subjects with BAC's $\geq$ 0.08% in a visual vigilance task. These findings were largely related to drowsiness produced by alcohol[28,29].

TABLE 1
APPROXIMATE SENSITIVITY LIMITS OF PSYCHOMOTOR TASKS TO BLOOD ALCOHOL CONCENTRATIONS (BAC)

Task	BAC
Divided Attention	$\geq$0.02%[26*]
Pursuit Tracking	$\geq$0.04%[29]
Visual Vigilance	$\geq$0.08%[28]
Closed Course Driving - Avoidance Maneuver	$\geq$0.04%[30]

*Superscripts indicate the number of reference

In addition to staying alert and accurately organizing sensorimotor information and utilizing sound judgment, it is necessary for drivers to continously monitor the environment. Many laboratory studies have investigated the effects of alcohol and other drugs on eye movements. Although the results of these studies may not be easily extrapolated to real driving situations they do provide evidence of relevant pharmacological effects that are often difficult to quantify in real driving situations.

Alcohol and barbiturates exert similar effects on eye movements. They both impair smooth pursuit movements, where the eyes involuntarily track an object moving across the visual field. Both the speed and accuracy of these movements are impaired[31-35]. In an effort to correct for this impairment, the eyes initiate saccadic movements which are rapid, conjugate movements of the eyes from one fixation point to another. The threshold speed at which saccades are employed instead of smooth pursuit is decreased by both moderate blood concentrations of alcohol ($\geq$0.05%) and relatively low doses of barbiturates[32,33,35-38]. Saccadic eye movements are also impaired at slightly higher concentrations (>0.10%) of alcohol. Additionally, the length of time from the stimulus presentation to the initiation of an eye movement increases[39-42].

Another type of eye movement affected by both drugs is nystagmus. Nystagmus consists of compensatory jerking eye movements initiated in response to stimulation of the vestibular system. Alcohol causes two types of nystagmus; lateral gaze and positional. Lateral gaze nystagmus occurs at a relatively low BAC of about 0.06% when the eyes are deviated laterally at a

30-40° angle[43]. It may impair the drivers' ability to accurately perceive targets in the periphery of the field of vision.

TABLE II
SUMMARY OF EFFECTS OF DRUGS ON EYE MOVEMENTS

	Ethanol	Barbiturates	Benzodiazepines	Marijuana
Saccadic				
Maximum Velocity	slowed	slowed	slowed	unaltered
Latency	increased	increased	unaltered	unaltered
Accuracy	unaltered	unaltered	undershoot?	unaltered
Smooth Pursuit				
Maximum Velocity	slowed	slowed	slowed	unaltered
Nystagmus	produces & modifies	produces & modifies	modifies	unaltered
Vergence	impaired	impaired	impaired	impaired

Vergence eye movements also show alcohol-induced impairment. These movements allow the eye to focus at various distances by utilizing disconjugate movements. These movements work in concert with accomodation of the lens and pupillary constriction to increase the clarity of vision. Alcohol causes outward deviation of the eyes (exophoria) at near, and inward deviation of the eyes (esophoria) at far distances[44-46]. The point of visual convergence moves closer at a BAC of approximately 0.05%[44]. This causes impairment of depth and distance perception. These findings suggest that the alcohol-impaired driver tends to be less able to visually detect potentially dangerous traffic conditions due to a severe impairment of a multitude of oculomotor functions.

The effects of drugs other than barbiturates and alcohol upon eye movements have not been extensively investigated. Moskowitz et al.[24] have reported a dose-related decrease in detection of light stimuli in the periphery of the field of vision after marijuana. This finding may actually reflect a cognitive rather than an eye movement effect. Diazepam 5 mg orally reduces the velocity of saccadic movements[47-49]. Futhermore, Rothenberg and Selkoe[50] reported a tendency for smooth pursuit to be replaced by saccadic movements at a lower velocity following diazepam. Chlordiazepoxide produces effects similar to diazepam[48], and amphetamine[51] causes a slight near esophoria and distance exophoria, in contrast to alcohol. None of these effects are as dramatic as those following relatively low doses of alcohol or barbiturates.

In summary, epidemiological studies suggest that the presence of BAC's $\geq$ 0.08%, young age, and male sex may be the three most significant risk factors associated with a fatal automobile crash. Both licit and illicit drugs may also be contributing risk factors, especially when used in combination with alcohol. Laboratory studies show significant psychomotor and eye movement impairment with BAC's as low as <0.05%. Other drugs may also cause similar impairment but, with the exception of barbiturate-induced eye movement changes, these changes during regular use are usually milder than those caused by relatively low doses of alcohol. An important point is that most of these drugs have additive effects when combined with alcohol, and the segment of population which most often combines recreational drugs with alcohol is young men who form the high accident risk group.

REFERENCES

1. Fell J C (1985). Alcohol involvement in United States traffic accidents: where it is changing. Proceedings of the Ninth international Conference on Alcohol, Drugs and Traffic Safety, San Juan, Puerto Rico, 1983, U.S. DOT, National Highway Traffic Safety Administration pp 439-467
2. Simpson H (1985) Alcohol, Drugs and Driving 1:17-44
3. Jones RK, Joscelyn KB (1978) Alcohol and Traffic Safety in 1978: A review of the State of Knowledge - DOT HS-803-714 Springfield, VA National Technical Information Service
4. Smith G, Wolynetz M, Davidson M, Poulton H (1975) Estimated blood alcohol concentrations of nighttime Canadian drivers, Proceedings of the Annual Conference of the Traffic Injury Research Foundation of Canada, Ottawa: Traffic Injury Research Foundation pp 24-29
5. Wolfe AC (1974) In: Israelstam S, Lambert S (eds), Alcohol, Drugs and Traffic Safety Toronto: Addiction Research Foundation Books pp 41-49
6. Pelz DC, McDole TL,Schuman SH (1975) J Stud Alcohol 36:956-971
7. Wallack L, Barrows D (1981) Preventing alcohol problems in California: Evaluation of the three year "winners" program. Report A-0345-8. Sacramento, CA: California Department of Alcohol and Drug Programs
8. Voas RB, Williams AF (1986) J Stud Alcohol 47:244-248
9. Soderstrom CA, Arias JD, Carson SL, Cowley RA (1984) Alcoholism Clin exp Res 269-271
10. Dubowki KM (1985) J Stud Alcohol Suppl 10:98-108
11. Harrington DM (1972) Accident Analysis Prev 4:191-240
12. Jones BM, Jones MK (1976) Ann NY Acad Sci 273:576-587
13. Jones MK, Jones BM (1984) Alcoholism Clin Exp Res 8:24-28
14. Richman A (1985) J Stud Alcohol Suppl 10:21-31
15. Honkanen R, Ertama L, Linnoila M, Alha A, Lukkari I, Karlsson M, Kiviluoto O, Puro M (1980) Br Med J 281:1309-1312
16. Skegg DCG, Richards SM, Doll R (1979) Br Med J 1:917-919

17. Neuteboom W, Zweipfenning PGM (1985) Driving and the combined use of drugs and alcohol in the Netherlands, Proceedings of the Ninth International Conference on Alcohol, Drugs and Traffic Safety, San Juan, Puerto Rico, 1983, US DOT National Highway Traffic Safety Administration pp 1023-1035

18. Williams AG, Peat MA, Crouch DJ, Walls JK, Finkle BS (1985) Public Health Rep 100:19-25

19. Laurell H, Tornros J (1985) The carry-over effects of triazolam compared with nitrazepam and placebo in acute emergency driving situations and in monotonous driving. Proceedings of the Ninth International Conference on Alcohol, Drugs and Traffic Safety, San Juan, Puerto Rico, 1983, US DOT National Highway Traffic Safety Administration pp 809-830

20. O'Hanlon JF, Volkerts ER, de Vries G, van Arkel A, Meijer T (1985) Effects of flurazepam HCL upon the actual driving performance of occasional hypnotic users: I. Acute experiment. Proceedings of the Ninth International Conference on Alcohol, Drugs and Traffic Safety, San Juan, Puerto Rico, US DOT National Highway Traffic Safety Administration pp 1037-1055

21. Tsuang MT, Boor M, Fleming J (1985) Am J Psychiatry 142:538-546

22. Eelkema RC, Brosseau J, Koshnich R, McGee C (1970) Am J Public Health 60:459-469

23. Cimbura G, Warren RA, Bennett RC, Lucas D, Simpson HM (1980) Drugs detected in fatally injured drivers and pedestrians in the province of Ontario, Ottawa; Traffic Research Foundation of Canada

24. Moskowitz H, Sharma S, McGlothin W (1972) Percept Mot Skills 35:875-882

25. Huntley MS Jr (1973) Q J Stud Alcohol 34:89-103

26. Moskowitz H, Burns MM, Williams AF (1985) J Stud Alcohol 46:482-485

27. Moskowitz H, DePry D (1968) Q J Stud Alcohol 29:54-63

28. Erwin CW, Wiener EL, Linnoila M, Truscott TR (1978) J Stud Alcohol 39: 505-516

29. Linnoila M, Erwin CW, Cleveland WP, Logue PE, Gentry WD (1978) J Stud Alcohol 39:745-758

30. Laurell H (1977) Accident Analysis Prev 9:191-201

31. Drischel H (1968) Prog Brain Res 22:161-174

32. Barnes GR (1984) Acta Otolaryngol Suppl 406:161-166

33. Guedry FE Jr, Gilson RD, Schroeder DJ, Collins WE (1975) Aviation, Space and Environmental Medicine 46:1008-1013

34. Lehtinen I, Nyrke T, Lang AH, Pakkanen A, Keskinen E (1982) Psychopharmacology 77:74-80

35. Wilkinson IMS, Kime R, Purnell M (1974) Brain 97:785-792

36. Norris H (1968) Br J Pharmacol Chemother 33:117-128

37. Rashbass C (1959) Nature 183:897-898

38. Rashbass C, Russell CFM (1961) Brain 84:329-335

39. Baloh RW, Sharma S, Moskowitz H, Griffith R (1979) Aviation, Space and Environmental Medicine 50:18-23

40. Jantti V, Lang AH, Keskinen E, Lehtinen I, Pakkanen A (1983) Psychopharmacology 79:251-255

41. Levett J, Hoeft G (1977) Aviation, Space and Environmental Medicine 48: 612-612

42. Levett J, Jaeger R (1980) Effects of alcohol on retinal potentials, eye movements, accomodation, and the pupillary light reflex. In: Merigan WH, Weiss G (eds) Neurotoxicity of the Visual System, Raven: New York, pp 87-100

43. Aschan G (1958) Acta OtoLaryngol Suppl 140:69-78

44. Wilson G, Mitchell R (1983) Aust J Ophthalmol 11:315-319

45. McNamee J, Piggins D, Tong J (1981) Am J Optom Physiol Opt 58:761-765

46. Hogan RE, Gilmartin B (1985) Ophthalmic Physiol Opt 5:43-51

47. Aschoff JC (1968) Arch Psychiatr Nervenkr 211:325-332

48. Gentles W, Thomas EL (1971) Clin Pharmacol Ther 12:563-574

49. Jurgens R, Becker W, Kornhuber HH (1981) Biol Cybern 39:87-96

50. Rothenberg SJ, Selkoe D (1981) Psychopharmacology 74:232-236

51. Westheimer G (1963) Arch Ophthalmol 70:830-836