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The Prevalence of Drugs and Alcohol in Fatally Injured Truck Drivers

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ABSTRACT: To assess the impact of alcohol and other drug use in the trucking industry, the National Transportation Safety Board, in collaboration with The National Institute on Drug Abuse investigated fatal-to-the-driver trucking accidents in eight states over a one year period.

Comprehensive drug screens were performed on blood specimens collected from 168 fatally injured drivers. One or more drugs were detected in 67% of the drivers and 33% of the drivers had detectable blood concentrations of psychoactive drugs or alcohol. The most prevalent drugs were cannabinoids and ethanol, each found in 13% of the drivers. Cocaine or benzoylecgonine was found in 8% of the cases. Seven percent of the driver's blood specimens contained amphetamine or methamphetamine and 7% contained phenylpropanolamine, ephedrine, or pseudoephedrine.

A panel of toxicologists reviewed the accident investigation report and the toxicology findings for each case and determined that impairment due to marijuana use was a factor in all cases where the delta-9-tetrahydrocannabinol concentration exceeded 1.0 ng/mL and that alcohol impairment contributed to all accidents where the blood alcohol concentration was 0.04% wt/vol or greater. In 50 of 56 cases where psychoactive drugs or alcohol were found, impairment due to substance use contributed to the fatal accident.

KEYWORDS: toxicology, ethanol, cocaine, THC, THC-COOH, caffeine, impairment, fatal-to-the-driver trucking crashes, accidental death

A number of accidents with serious consequences have sensitized the American public to drug and alcohol abuse by transportation workers. For example, cannabinoid use was

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detected in the 1981 crash on board the aircraft carrier U.S.S. Nimitz, in which 14 died [*1*]. A 1986 railroad accident near Chase, Maryland, resulted in the death of 16 passengers and impairment of a crew member due to drug use was determined to be a cause [*2*]. Impairment from alcohol consumption played a major causative role in the grounding and oil spill of the Exxon Valdez in Alaska in 1989 [*3*]. These incidents accentuated the need to evaluate the effects of alcohol and other drug use on public and environmental safety. Media coverage of these events stimulated public awareness and focused public concern on the impact of drug and alcohol use on transportation safety.

Borkenstein et al. performed one of the first controlled field studies of the effects of ethanol (alcohol) on driving [*4*]. Since that classic study, researchers have continued to investigate the role of alcohol in impaired drivers and have expanded their efforts to include the effects of drugs. Numerous studies reporting the prevalence of drug use by impaired drivers, injured drivers, and fatally injured auto drivers have been published [*5–12*]. Studies of fatally injured driving populations, such as that reported by Garriott et al. in 1977, were undertaken to gain an understanding of the prevalence and forensic implications of drug induced driving errors [*9*]. Williams et al. reported on a "high risk" sample of 440 young male auto drivers killed in California traffic accidents [*10*]. Seventy percent of blood specimens collected from these drivers contained alcohol, and over 40% contained other drugs [*10*]. Substance use and transportation safety is not a problem unique to the U.S., as demonstrated by Robinson in Ireland in 1979, and Cimbura et al. in Canada in 1980 [*7,11*].

Early studies of the prevalence of drug use by drivers were limited because the analytical methods used lacked the sensitivity to screen and reliably quantitate many drugs including cannabinoids in biological fluids. The 1979 study by Reeve et al. provided some of the first insight into the extent of marijuana use by motor vehicle operators [*6*]. Reeve et al. showed that 16% of a selected sample of arrested drivers had cannabinoids in their urine [*6*]. The 1984 report of Mason and McBay also addressed the question of cannabinoid use by drivers. This study of 600 driver fatalities in North Carolina demonstrated that over 79% had detectable blood alcohol concentrations and 7.8% contained cannabinoids [*12*]. Results from comprehensive drug screens on specimens collected from fatally injured auto drivers in Canada showed that cannabinoids were found in the urine of 9% and in the blood of 3% of the 484 drivers [*11*]. This study is significant because presumptive positive marijuana finds were confirmed by Gas Chromatography/Mass Spectrometry (GC/MS). GC/MS confirmations were not routinely performed in earlier studies.

Although these studies supported the concern about drug use (particularly marijuana) by auto drivers, they did not address the question of use by truck drivers. A systematic study to evaluate drug use by truck drivers was performed by Lund et al. in 1987 [*13*]. Their findings revealed that less than 1% of 300 paid volunteer drivers randomly selected at an interstate weigh station were positive for ethanol and that 29% were positive for other drugs. In a British Columbia project where truck drivers were also randomly selected at weigh stations, 2% of the drivers admitted to using alcohol on the day of the interview and 9.6% tested positive for drugs [*14*]. Although data from both of these studies demonstrate the extent of drug use by truck drivers, neither assessed driver impairment due to drug use.

The study reported here represents perhaps the most thorough study of drug and alcohol use by drivers involved in fatal-to-the-driver truck crashes conducted to date. It was designed to determine the prevalence of drug and alcohol use by drivers of heavy trucks who were fatally injured in a highway accident, to assess driver impairment, and to identify the cause of the accidents. The design contained a number of critical and unique features: (1) a detailed on-site accident investigation; (2) specimen collection and toxicology testing; (3) development of a detailed factual report on the accident; (4) an

assessment of the contribution of drug or alcohol impairment to each accident; and (5) a determination of probable cause by the National Transportation Safety Board (NTSB).

Methods

Sample Selection

Specimens were collected from drivers killed in highway accidents while operating trucks of greater than 10 000 pounds gross vehicle weight from October 1, 1987, through September 30, 1988.

California, Colorado, Georgia, Maryland, New Jersey, North Carolina, Tennessee, and Wisconsin were selected as target collection states because of their geographic dispersion; their representation of rural, suburban, and metropolitan areas; the nature of their interstate highway systems; and each state's willingness to participate in accident reporting and specimen collection. According to Fatal Accident Reporting System (FARS) data, approximately 800 drivers of large trucks are killed each year on American highways [*15*]. It was estimated from the FARS data that conscientious monitoring of accidents in these eight states would produce the desired sample of approximately 25% to 30% of nationwide fatal-to-the-driver truck accidents in a 1 year time frame.

Each fatal accident that occurred in a selected state resulted in an investigation by the nearest NTSB regional office. NTSB regional offices were located in Los Angeles, Denver, Kansas City, Atlanta, Washington, D.C., New York, Seattle, and Chicago. The investigators evaluated accident parameters including the mechanical condition of the truck, road and weather conditions, and activities of the driver prior to the accident. To determine the drivers' activities, an hours-of-duty audit was conducted. This audit included an examination of receipts for gasoline purchases and minor service repairs, a review of the driver's log book to determine compliance with break and sleep regulations, and interviews. Insurance coverage, maintenance, and load information were obtained on the vehicle. In conjunction with the vehicle assessment, the driver's medical records were examined for evidence of clinical conditions that could have been a factor in the accident. Background data were also obtained on the education, training, and experience of the driver.

Methods

Specimen Collection

Blood was selected as the specimen of choice for analysis because it allows a reasonable probability of interpreting the relationship between drug use, drug concentration, and driver impairment. However, when blood could not be obtained, vitreous humor and urine specimens were collected for testing. Specimens were obtained only from those drivers who died within 4 hours of the crash and who received limited medical attention prior to death. All specimens were collected in shatter-proof silanized glass containers containing sodium fluoride and potassium oxalate and fitted with teflon lined screw caps. Specimens were shipped on ice by overnight courier service by the NTSB investigator or local medical examiner to the Center for Human Toxicology, University of Utah, for toxicology testing.

Analytical Testing

Screening, confirmation, and quantitative analyses for a variety of illicit, commonly prescribed, and over-the-counter (OTC) drugs (Table 1) were performed using the methods of Crouch et al. 1983 [*16*]. Table 2 summarizes the screen and confirmation testing

TABLE 1—*Drugs tested in the analytical protocol.*

Class/Drugs	Class/Drugs
Sedatives and tranquilizers	Opiates and opoids
(Barbiturates)	Morphine
Butalbital	Codeine
Amobarbital	Meperidine
Secobarbital	Methadone
Pentobarbital	Propoxyphene
Butabarbital	Pentazocine
Phenobarbital	Oxycodone
Methaqulaone	Hallucinogens
	Phencyclidine
(Benzodiazepines)	
Diazepam/Nordiazepam	
Flurazepam/Desalkylflurazepam	Cannabinoids
Chlordiazepoxide/Norchlordiazepoxide/	Delta-9-THC
Demoxepam/Nordiazepam	Delta-9-THC-COOH
Stimulants and sympathomimetic amines	OTC analgesics
Cocaine/Benzoylecgonine	Acetaminophen
Amphetamine	Salicylates
Methamphetamine	Ibuprofen
Phentermine	
	Anticonvulsants
Ephedrine	Phenytoin
Pseudoephedrine	Carbamazepine
Phenylpropanolamine	Phenobarbital
Caffeine	
	Antihistamines
Volatiles	Diphenhydramine
Ethanol	Chlorpheniramine
Methanol	Brompheniramine
Isopropanol	
Acetone	

limits for each drug or drug class tested. All detected drugs and metabolites were quantified. GC/MS was used for confirmation and quantitation of most drugs detected in the screening protocols. The OTC analgesics and the anticonvulsants, carbamazepine and phenytoin, are an exception since they were screened and confirmed by high performance liquid chromatography.

Assessment of Impairment

A panel of pharmacologists and toxicologists, (see Appendix 1), met three times during the course of the study to review the accident investigation reports, to integrate information from the factual report with the toxicology results, and to determine whether impairment due to drug or alcohol use was likely to have contributed to the accident. The role of drug or alcohol impairment in the accident was determined by considering the pharmacology of the drug(s) involved, the blood concentrations of the drug(s) and metabolite(s), the impairment potential of the drug or drugs in question, and the circumstances surrounding each crash. These data were summarized and presented to the NTSB that made the final determination of probable cause for the accident [*17*].

TABLE 2—*Drugs tested, analytical methods and testing limits.*

Drug/class	Screen method	Confirmation method	Testing limits Screen—Confirm (ng/mL)	
Volatiles	GC/FID[a]	CG/FID	0.01% W/V	0.01% W/V
(Ethanol, Methanol, Acetone, & Isopropanol)				
Barbiturates	RIA[b]	GC/MS[c] or HPLC[d]	200	200
Cannabinoids	RIA	GC-CI/MS[e]	25	
THC				1
THC-COOH				2
Cocaine/BE	RIA	GC-CI/MS	25	10
Methaqualone	RIA	GC-CI/MS	1000	500
Opiates	RIA	GC-CI/MS	50	25
PCP	RIA	GC-CI/MS	25	10
OTC Analgesics	HPLC	HPLC	10 000	10 000
Stimulants	GC-NPD[f]	GC-CI/MS	200	100
Antihistamines	GC-NPD	GC-MS	200	100
Opioids	GC-NPD	GC-MS	250	100
Caffeine	GC-NPD	CG-MS	1000	1000
Benzodiazepines	GC-ECD[g]	GC-MS	100	100
Chlordiazepoxide	HPLC	HPLC	3000	300
Anticonvulsants	HPLC	HPLC	5000	5000

[a]Gas Chromatography-Flame Ionization Detection.
[b]Radioimmunoassay.
[c]Gas Chromatography-Mass Spectrometry
[d]High Performance Liquid Chromatography.
[e]Gas Chromatography-Chemical Ionization Mass Spectrometry.
[f]Gas Chromatography-Nitrogen Phosphorus Detection.
[g]Gas Chromatography-Electron Capture Detection.

Statistical Tests

Data for the most prevalent drugs were subjected to computer assisted descriptive statistical analyses to determine the mean, median, standard deviation, range, and distribution of the analytical results. The Kolmogorov-Smirnov test was used to determine the goodness of fit to a theoretical normal distribution. This statistical test is particularly well suited for this project because it is sensitive to dispersion and skewness and is effective with small samples. A priori statistical significance was set at the 0.05 level.

Results

Prevalence

There were 761 fatal-to-the-driver trucking deaths in the United States between October 1, 1987 and September 30, 1988. Data in this study were obtained from 182 qualifying accidents, that included 186 trucks and drivers and represented 24% of the nationwide fatalities [*17*]. One accident was disqualified because the investigators were unable to determine whether the driver or the co-driver was operating the truck at the time of the crash. Seventeen other fatalities did not qualify because an inadequate specimens precluded either screening or confirmation analysis. The actual number of cases tested for each different drug or drug class varied somewhat because of specimen volume constraints. To avoid confusion, all percentages are based on a common denominator of 168 drivers.

Fifteen different drugs or drug metabolites were detected in the drivers. The frequency of detection of each substance is presented in Table 3. OTC analgesics such as acetaminophen, salicylate, and ibuprofen were rarely detected. Caffeine, which is also readily available, was frequently identified (35% of the drivers). Ethanol and cannabinoids were the most commonly detected drugs of abuse, occurring in 13% of the cases. Cocaine was found in blood specimens from 8% of the drivers. The prescription stimulants, amphetamine or methamphetamine, were found in 8% of the drivers. Phenylpropanolamine, pseudoephedrine, and ephedrine, which may be found in OTC cold medications and in clandestinely prepared amphetamine "look-alike" preparations, were detected in 7% of the drivers.

Results

Descriptive Statistics

Of the 168 qualifying accidents, 67% or 112 of the drivers' blood specimens contained one or more of the compounds listed in the testing panel shown in Table 1. Psychoactive drugs or alcohol were found in blood specimens from 56 (33%) of the drivers.

Ethanol, the only volatile alcohol encountered, was found in 23 drivers. Although collection protocols were designed to limit the possibility of ethanol being generated from postmortem production, in two cases, in vitro production was a possible source of the ethanol. The blood alcohol concentrations (BACs) in the remaining 21 cases were normally distributed over the concentration range from 0.017 to 0.31% wt/vol (Table 4).

Parent delta-9-tetrahydrocannabinol (THC) or 11-nor-9-carboxy-tetrahydrocannabinol (THC-COOH) was detected in 21 (13%) of the 168 drivers. However, in one case urine was the only specimen available, therefore, 20 cases were used for the descriptive statistics shown in Table 4. Of the 20 drivers with cannabinoids in their blood, 14 contained parent THC. The parent THC concentrations were normally distributed, ranged from 1 to 12 ng/mL, had a mean concentration of 41 ng/mL, and a standard deviation of 3.6 ng/mL (Table 4). The blood THC-COOH concentrations were also normally distributed, ranging from 5 to 174 ng/mL.

Cocaine or its primary metabolite benzoylecgonine (BE), was detected in 15 drivers; however, one accident was excluded from Table 4 because the investigators were unable

TABLE 3—*Substances detected and number of occurrences.*

Substance	Number of occurrences
Acetaminophen	2
Amphetamine	7
Carbon monoxide	11
Caffeine	55
Chlorpheniramine	1
Cocaine/benzoylecgonine	14
Codeine	1
Delta-9-Tetrahydrocannabinol/COOH metabolite	21 (20 blood and 1 urine)
Ethanol	21
Ephedrine	7
Ibuprofen	2
Methamphetamine	12
Phenylpropanolamine	1
Pseudophedrine	5
Phencyclidine	1
Salicylates	11

TABLE 4—*Descriptive statistics for selected drugs.*

Statistic/drug	Ethanol	THC	THC-COOH	BE
Concentration	Mg%	Ng/mL	Ng/mL	Ng/mL
n	21	14	20	14
Mean	149.8	4.08	31.04	404.9
Median	170	3	22	197
Standard deviation	89.4	3.6	36.6	587
Maximum	310	12	174	2300
Minimum	17	1	5	57
Range	293	11	169	2243
Distribution stat	0.16	0.29	0.30	0.31
Significance[a]	0.23	0.13	0.09	0.13
Distribution	Normal	Normal	Normal	Normal

[a]Calculated statistics of >0.05 indicate normal distribution.

to determine which of the two occupants of the tractor was operating the vehicle. Therefore, 14 drivers (8% of the sample population) were considered in these data. Parent cocaine, at concentrations of 80, 200, and 500 ng/mL, was found in only 3 of the 14 cocaine-positive drivers.

Sympathomimetic amines (see Tables 1 and 3) were found in 19 cases (11.3% of the drivers). Amphetamine and/or methamphetamine was found in specimens from 12 drivers. The OTC sympathomimetic amines phenylpropanolamine, ephedrine and pseudoephedrine, were also detected in 12 drivers. Blood from drivers in 5 of the 12 OTC sympathomimetic amine-positive cases contained methamphetamine and/or amphetamine. Phenylpropanolamine was detected in the blood from only one driver and this driver had also taken pseudoephedrine.

Table 5 presents cases in which combinations of more than one abused substance were found. Fourteen percent of the drivers' blood contained more than one drug of abuse. Blood from one driver contained cocaine and prescription stimulants. Five drivers had taken OTC sympathomimetic amines and prescription anorectics. Compounds from three or more classes of abusable substances were detected in only three drivers: two cases contained cannabinoids, cocaine/BE and ethanol; and one driver had cannabinoids, cocaine/BE and controlled stimulants. Although not shown in the table, methamphetamine and/or amphetamine, was found in combination with cannabinoids in three drivers.

Impairment

An objective of the study was to assess the contribution of drug and alcohol impairment to accident causation. This was achieved by the panel case-review procedures and by the assignment of probable cause for each accident by the NTSB. All of the factors surrounding the accident were integrated into the decision. Multiple factors, in addition

TABLE 5—*Combination of drugs of abuse.*

Drug combination	Number of occurrences
1. Marijuana and cocaine/BE	8
2. Ethanol and marijuana	4
3. More than 2 DOA[a] classes	3
4. Amphetamine or methamphetamine with OTCs	5

[a]Drugs of abuse.

to drug or alcohol induced impairment, were causal in most accidents. For example, drug impairment contributed to the accident in all of the cases where parent THC was detected; however, it was seldom the sole cause of the accident. The consumption of other drugs or alcohol, as well as fatigue and mechanical problems, were involved in accident causation in a majority of the crashes.

Discussion

Alarmingly, in 50 of the 56 cases where psychoactive drugs or alcohol were found, impairment due to substance abuse was indicated as the cause, or contributed to the cause, of the fatal accident. A more in-depth breakdown of these 50 accidents revealed that in 10 accidents, driver fatigue in combination with driver impairment from substance use caused the accident. In 3 of the 50 accidents, the mechanical condition of the truck in combination with driver impairment from substance use caused the fatal crash. A variety of factors including medical problems, driver inexperience, driver fatigue, failure to heed warning signs or to yield, load shifts and mechanical failures contributed to those accidents where probable cause was not substance use by the driver. A complete list of the probable cause determinations is provided by the NTSB [*17*].

Carbon monoxide was detected in the blood of 11 drivers. The mean percent carboxyhemaglobin saturation in crashes with fires was 31 and in crashes without fires 12.5. It is difficult to determine whether any of the carbon monoxide in the fire related cases was present prior to the crash and, therefore, may have contributed to the crash. In the drivers with carboxyhemaglobin detected, but the crash did not involve a fire, the mean percent saturation was very close to the analytical testing limit of 10% saturation. Given the closeness to the testing limit, and the analytical precision of + or − 10%, establishing a causative role for carbon monoxide in these crashes was problematic.

Establishing a threshold concentration at which impairment occurs even for a drug as extensively studied as ethanol remains controversial. In flight stimulator studies, performance has been significantly impaired at BACs of 0.025% wt/vol [*18*]. Department of Transportation regulations establish 0.04% wt/vol as the legal BAC for truck drivers [*19*]. Historically, 0.05% wt/vol ethanol has been accepted as a minimum blood concentration capable of producing impairment to operate a motor vehicle [*20*]. Most states have enacted per se legislation of 0.08 or 0.10% wt/vol BAC as prima facie evidence of impairment to operate a motor vehicle [*20,21*]. Impairment from ethanol intoxication was assigned as probable cause by the NTSB in only one (BAC 0.04% wt/vol) of four accidents where the BAC was less than or equal to 0.05% wt/vol. The remaining three cases had BACs of 0.02% wt/vol or less. Alcohol impairment contributed to each of the four cases accidents in which the drivers had BACs exceeding 0.05% wt/vol, but less than or equal to 0.10% wt/vol. Alcohol impairment was a causative factor in all drivers (8) who had BACs between 0.10% and 0.20% wt/vol, and in all drivers (5) whose blood ethanol concentration exceeded 0.20% wt/vol.

Maximum blood THC concentrations are reached within minutes after smoking and decrease rapidly to undetectable levels due to redistribution and tissue absorption [*22,23*]. However, the psychological effects from THC last for at least 1 to 3 hours after smoking and carry-over effects have been demonstrated for up to 24 hours after use [*24,25*]. Depending on the dose and frequency of use, it would be expected from the cited research that blood THC concentrations would rapidly fall below the 1 ng/mL analytical sensitivity used in this study. Unlike THC, THC-COOH is not pharmacologically active and may be detected for days after chronic use [*26*]. The pharmacokinetics and analytical sensitivity for THC and THC-COOH were important in interpreting whether cannabinoid use led to driver impairment. In the 14 fatal crashes in which parent THC was found, the panel concluded that the drivers were impaired due to use of the drug. We may have

been conservative in our interpretation since whole blood concentrations of THC are approximately 55% of plasma concentrations [*27*]. This partitioning resulted in lower THC concentrations in this study than those cited in the literature that are primarily based on plasma or serum THC data [*23,28*]. In the six drivers where only THC-COOH and no parent THC was identified (Table 5 contains cases where THC or THC-COOH was found), three contained other psychoactive drugs and drug impairment was assigned as probable cause of the accidents; in a fourth case, THC-COOH and acetaminophen were detected, and probable cause was not assigned to drug impairment; and in the two remaining cases no drugs other than THC-COOH were found, and drugs were not deemed contributory to the accident.

Cocaine and BE were found in the blood of 3 drivers. Blood from eleven drivers contained only BE. Although the blood specimens were preserved with sodium fluoride, the cocaine may have hydrolyzed and/or continued to be hydrolyzed in vitro to BE [*29*]. Cocaine has a half life of approximately 1 hour so specimens must be collected within a few hours of exposure to ensure detection of the parent drug [*30*]. In vitro hydrolysis and short half life may have both contributed to reducing cocaine concentrations below the analytical sensitivity of the GC/MS analysis and provide possible explanations for the lack of detection of the parent drug.

Amphetamine and phenylpropanolamine may be found in biological specimens as metabolites of methamphetamine and ephedrine, respectively, or from ingestion of the parent compounds. Therefore, the presence in a blood specimen of amphetamine may be indicative of consumption of amphetamine or methamphetamine. Similarly, the detection of phenylpropanolamine might indicate use of either phenylpropanolamine or ephedrine. Amphetamine or methamphetamine was found in the blood of 12 drivers. No drivers had amphetamine alone in their blood specimen, however, in four drivers methamphetamine was detected and no amphetamine was found. Differentiation of the d and l sterioisomers of methamphetamine to address the issue of l-methamphetamine from OTC medications was not performed in the study [*31*]. In two of the four drivers, legal use of OTC preparations containing l-methamphetamine provides a plausible explanation for the presence of methamphetamine since the concentrations were low and other OTC drugs were found.

Research correlating blood concentrations with impaired performance following the use of cocaine and other stimulants is limited. Some data indicate that consumption of stimulants during periods of sleep deprivation can reverse performance decrements [*32–34*]. Use has also been associated with tendencies toward greater risk taking and impairment may occur during "hang over" periods or episodes of depression that often follow discontinuation of use [*32,33*]. Ellinwood noted that single, moderate doses of stimulants may improve performance; larger doses, resulting in elevated blood concentrations, lead to degeneration of performance; and withdrawal can adversely effect performance [*35,36*]. Ingestion of OTC sympathomimetic amines may result in clinical manifestations similar to those found following ingestion of prescription sympathomimetic amines.

The extent illegal use of multiple substances by professional drivers is shown in Table 5. The threat to the driver and to public safety lies in the potential of these drugs to act in combination to impair driver performance [*37*]. Lundberg et al. found that alcohol in combination with barbiturates "sharply" increased the number of driver fatalities when compared to single drug ingestion [*5*]. In driving simulator studies, low doses of ethanol combined with cannabinoids produced greater impairment than that found when either drug is administered individually [*28*]. On road driving studies have also shown increased reaction times, impaired steering control, and greater risk taking in groups following co-administration of alcohol and marijuana compared to the performance of placebo or individual drug dosed groups [*37*]. The cumulative effects on the driver from the drug combinations presented in Table 5 are difficult to predict given the current body of

scientific knowledge on drug actions and driving performance. However, one can predict that the combinations pose a greater risk than no drugs or a single drug ingestion.

The data from the current study were obtained from a targeted sample of fatally injured truck drivers. However, the similarities of the results of this study with other research findings suggest that drug and alcohol use by truck drivers present a significant traffic safety hazard. Comparable percentages of THC or THC-COOH of 13% and 15%, respectively, were found in the fatally injured truck drivers discussed here and in the Lund study [*13*]. In the Lund study, 29% of the truck drivers were positive for ethanol or drugs and in the current study 33% [*13*]. The 29% and 33% figures are consistent with a 1989 motor carrier survey in which drivers believed that 26% of their peers regularly drive under the influence of illegal drugs [*38*]. In 1988, this estimate was 29%, again remarkably close to the percentage findings in the both the Lund findings and the current data [*13,38*].

Conclusions

Evidence indicates that many variables contribute to highway crashes. A single variable was seldom judged to have been the causal factor of the crash in this study. Driver impairment due to drug and alcohol use, driver fatigue, mechanical and environmental factors all contributed in varying degrees to the accident. Therefore, a thorough accident investigation and toxicology testing are recommended before assigning fault for traffic accidents.

The use of drugs or alcohol by one of every three truck drivers in this study is an alarming statistic with equally alarming public safety implications. This prevalence rate is supported by data from truck drivers volunteering for testing and from self-reported use. Future studies to evaluate drug and alcohol use by truck drivers and to determine mechanisms to deter drug use by drivers of heavy trucks are clearly indicated.

APPENDIX 1

The following groups contributed to the study.

1. The National Institute on Drug Abuse, Division of Applied Research, provided financial support.

2. Members of the toxicology panel alphabetically were: Merritt Birky, Ph.D.; Yale Caplan, Ph.D.; Dennis Crouch; Brian Finkle, Ph.D.; Steven Gust, Ph.D.; Herbert Moskowitz, Ph.D.; Kenneth Rogers; and Douglas Rollins, M.D., Ph.D.

3. The following states and representatives cooperated in the project:
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   b. Colorado. Dr. Ron Cada, Robert Zettl, Donald Lamb and Ralph Martine. The Department of Health, Laboratory Division and the State Patrol.
   c. Georgia. C. R. Pinyan, W. D. Fielding, L. B. Howard and Archie Burnham representing the State Patrol, the Bureau of Investigation and the Department of Transportation.
   d. Maryland. Drs. Smialek and Caplan, William Carson, Ronald Lipps, Rodney Martin and Don Boehm. The State Medical Examiners Office, Department of Transportation and the State Police.
   e. New Jersey. Dr. Robert Goode and Dr. Reng-lang Lin of the State Medical

Examiners Office and William Taylor and Vincent Pagano from the Office of Highway Traffic Safety and the State Police.

f. North Carolina. Dr. Arthur McBay, Chief Toxicologist; W. B. Richardson, State Highway Patrol; and Paul Jones, Governor's Highway Safety Program.

g. Tennessee. Dr. Jerry Francisco, Ronald Marshak and Mike Ellis. The State Medical Examiner, Governor's Highway Safety Program, Department of Public Health, Highway Patrol, and Public Service Commission.

h. Wisconsin. Dr. Patricia Field and Maynard G. Stoehr from the Toxicology Section, Laboratory of Hygiene and Office of Highway Safety respectively. Participating agencies included Highway Safety Coordinators Association, Sheriffs & Deputy Sheriffs Association, Coroners Association, County Police Association and local law enforcement agencies.

4. The Field and Regional Office investigators of the NTSB.

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