U.S. Department
of Transportation

**National Highway
Traffic Safety
Administration**

DOT HS 808 065

October 1992

Final Report

The Incidence and Role of Drugs in Fatally Injured Drivers

TECHNICAL REPORT STANDARD TITLE PAGE

1. Report No.	2. Government Accession No.	3. Recipient's Catalog No.
DOT HS 808 065		

4. Title and Subtitle	5. Report Date
The Incidence and Role of Drugs in Fatally Injured Drivers	October 30, 1992
	6. Performing Organization Code

7. Author(s)	8. Performing Organization Report No.
K.W. Terhune, C.A. Ippolito, D.L. Hendricks J.G. Michalovic, S.C. Bogema, P. Santinga, R. Blomberg, D.F. Preusser	8023-1

9. Performing Organization Name and Address	10. Work Unit No.
Accident Research Group Calspan Corporation Advanced Technology Center P.O. Box 400 Buffalo, NY 14225	11. Contract or Grant No. DTNH22-88-C-07069
12. Sponsoring Agency Name and Address	13. Type of Report and Period Covered
U.S. Department of Transportation National Highway Traffic Safety Administration 400 Seventh Street, S.W. Washington, D.C. 20590	Final Report Sept. 1988 - Oct. 1992
	14. Sponsoring Agency Code

15. Supplementary Notes

16. Abstract

Blood specimens were collected from a sample of 1882 drivers from 7 States, during 14 months in the years 1990 and 1991. The sample comprised operators of passenger cars, trucks, and motorcycles who died within 4 hours of their crash. Alcohol and 43 other drugs were studied, to determine their prevalence rates, their causal role in the crashes, and associated driver, vehicle, and crash factors. Coroner or medical examiner reports were obtained on all cases to ascertain cause of death, time of death, and other details. Police accident reports and reports from the Fatal Accident Reporting System (FARS) were also obtained. Alcohol was found in 51.5% of the specimens, and other drugs were found in 17.8%. Other than alcohol, the most prevalent drugs were cannabis (6.7%), cocaine (5.3%), benzodiazepine tranquilizers (2.9%), and amphetamines (1.9%). Responsibility analysis was used to suggest impairment effects, which were found for alcohol alone and for alcohol-drug combinations. Further research was recommended to follow up indications of alcohol-drug additive effects.

17. Key Words	18. Distribution Statement
Drugs; drugs and driving; drug crashes; drinking drivers; highway safety; crash causation	Document is available to the public through the National Technical Information Service, Springfield, VA 22151

19. Security Classif. (of this report)	20. Security Classif. (of this page)	21. No. of Pages	22. Price
Unclassified	Unclassified		

Form DOT F 1700.7 (8-69)

METRIC CONVERSION FACTORS

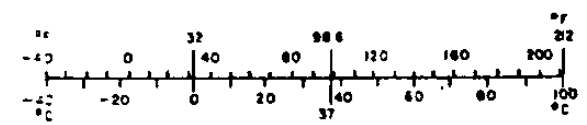
Approximate Conversions to Metric Measures

Symbol	When You Know	Multiply by	To Find	Symbol
	LENGTH			
in	inches	*2.5	centimeters	cm
ft	feet	30	centimeters	cm
yd	yards	0.9	meters	m
mi	miles	1.6	kilometers	km
	AREA			
in^2	square inches	6.5	square centimeters	cm^2
ft^2	square feet	0.09	square meters	m^2
yd^2	square yards	0.8	square meters	m^2
mi^2	square miles	2.6	square kilometers	km^2
	acres	0.4	hectares	ha
	MASS (weight)			
oz	ounces	28	grams	g
lb	pounds	0.45	kilograms	kg
	short tons (2000 lb)	0.9	tonnes	t
	VOLUME			
tsp	teaspoons	5	milliliters	ml
Tbsp	tablespoons	15	milliliters	ml
fl oz	fluid ounces	30	milliliters	ml
c	cups	0.24	liters	l
pt	pints	0.47	liters	l
qt	quarts	0.95	liters	l
gal	gallons	3.8	liters	l
ft^3	cubic feet	0.03	cubic meters	m^3
yd^3	cubic yards	0.76	cubic meters	m^3
	TEMPERATURE (exact)			
°F	Fahrenheit temperature	5/9 (after subtracting 32)	Celsius temperature	°C

*1 in = 2.54 (exactly). For other exact conversions and more detailed tables, see NBS Misc. Publ. 286, Units of Weights and Measures. Price $2.25 SD Catalog No. C13.10:286.

Approximate Conversions from Metric Measures

Symbol	When You Know	Multiply by	To Find	Symbol
	LENGTH			
mm	millimeters	0.04	inches	in
cm	centimeters	0.4	inches	in
m	meters	3.3	feet	ft
m	meters	1.1	yards	yd
km	kilometers	0.6	miles	mi
	AREA			
cm^2	square centimeters	0.16	square inches	in^2
m^2	square meters	1.2	square yards	yd^2
km^2	square kilometers	0.4	square miles	mi^2
ha	hectares (10,000 m^2)	2.5	acres	
	MASS (weight)			
g	grams	0.035	ounces	oz
kg	kilograms	2.2	pounds	lb
t	tonnes (1000 kg)	1.1	short tons	
	VOLUME			
ml	milliliters	0.03	fluid ounces	fl oz
l	liters	2.1	pints	pt
l	liters	1.06	quarts	qt
l	liters	0.26	gallons	gal
m^3	cubic meters	35	cubic feet	ft^3
m^3	cubic meters	1.3	cubic yards	yd^3
	TEMPERATURE (exact)			
°C	Celsius temperature	9/5 (then add 32)	Fahrenheit temperature	°F



ADDENDUM

NHTSA has some concerns regarding Recommendation (3) on page 107 of this report. This recommendation suggests the implementation of a procedure for monitoring crash drug prevalence over time using the FARS system. Since the FARS system is already collecting information on drug presence, when these data are available, this recommendation appears superfluous. The current FARS system relies on the individual states to provide the drug data. NHTSA believes that this is more appropriate than imposing, as the Recommendation implies a federally mandated and funded drug data collection system.

TABLE OF CONTENTS

TABLE OF CONTENTS (Continued)

EXECUTIVE SUMMARY

This study examined drug presence in 1,882 fatally injured drivers from seven States. Alcohol and 43 other drugs in the crashes were studied, to determine (a) their prevalence rates in the drivers, (b) their causal role in the crashes, and (c) their patterns of associated driver characteristics, vehicle types, and crash circumstances.

Background. A 1988 Report to Congress by the National Highway Traffic Safety Administration estimated from previous research that 10 to 15 percent of fatally injured drivers had nonalcoholic drugs in their systems, but the samples were limited and the extent to which the drugs caused the crashes was mainly unknown. It was concluded that a study was needed to more broadly reveal the scope and effects of drug involvement in U.S. driver fatalities.

Drugs studied. Examined were (a) the major drugs of abuse, including cannabis, cocaine, amphetamines, phencylidine (PCP), LSD, and heroin, and (b) common prescription drugs, including benzodiazepine tranquilizers, sedatives, antihistamines, antidepressants, narcotic analgesics, and antipsychotics.

Research approach. There was a total of 13 sampling sites, encompassing three entire States (Massachusetts, North Carolina, and Wisconsin) and selected counties from California, Nevada, Texas, and Virginia. These sites were chosen to achieve broad regional representation and for their previously demonstrated ability to provide blood alcohol data to the Fatal Accident Reporting System (FARS). Over a 14-month period, coroners and medical examiner (ME) staffs at the field sites obtained whole blood specimens from driver fatalities meeting the sampling criteria: survived a maximum of 4 hours; driver of car, motorcycle, or truck; death due to crash; etc. The specimens, with preservatives, were express shipped to American Medical Laboratories (AML) for analysis. To provide information about the crashes and causes of death, the project team collected FARS reports, police accident reports, coroner and ME reports, and special project records. From these data sources, variables were extracted to permit analyses addressing the research objectives.

In addition to AML's internal quality control, quality checks were made by inserting specially prepared test specimens into the site shipments. AML succeeded in detecting all drugs in the test specimens. It was also found that AML's results agreed highly with the substance detections at a field site whose own laboratory performed comprehensive drug testing.

When FARS data became available after the conclusion of data collection, a sampling completeness check was made. Heavy caseloads at one site precluded it from participating effectively

in the project, but the remaining sites succeeded in obtaining specimens from 79 percent of the drivers eligible for the study according to FARS. Our sample was also found to closely resemble a FARS national population on key dimensions of age, gender, vehicle type, time of day, etc., with the exception that our sample slightly oversampled urban crashes.

Responsibility analysis was used to suggest which drugs contributed to the occurrence of the crashes. The method, which was further developed for the study, involves the rating of each driver's crash responsibility, without knowledge of any drug involvement. If proportionately more drug-present drivers are judged responsible than are those free of drugs, this is considered evidence of drug impairment effects.

Main results.

(1) The drivers were found to comprise the following groups:

Drugfree	- 42.1%
Alcohol alone	- 40.1%
Alcohol & drugs	- 11.4%
1 drug, no alcohol	- 4.8%
2+ drugs, no alcohol	- 1.6%
Total driver sample	- 100.0%

(2) The drug prevalence rates found were: alcohol at BAC 0.10% or higher (42.6% of the drivers); alcohol at BAC below 0.10% (9.0%); cannabis (6.7%); cocaine (5.3%); benzodiazepines (2.9%); amphetamines (1.9%); barbiturate sedatives (1.5%); narcotic analgesics (1.2%); antidepressants (0.8%); hallucinogens (0.3%); antiarrhythmics (0.1%); and muscle relaxants (below 0.1%). Neither antipsychotics nor nonbarbiturate sedatives were found.

(3) Regional variations in drug prevalence were found. Amphetamines were nearly exclusively found in California, while alcohol, cannabis, and cocaine were unusually prevalent in the Dallas-Fort Worth area of Texas. The Wisconsin drivers were lowest in abuse-drug detection. Also, 20.9% of urban drivers had drugs other than alcohol in their systems, compared to 15.1% of the rural drivers.

(4) Alcohol was found in 83.3% of the cocaine cases, 68.8% of the THC (cannabis) cases, 66.7% of the antidepressant cases, 61.9 % of the benzodiazepine cases, and in about half the analgesic, amphetamine, antihistamine, and hallucinogen cases.

(5) Driver survival time was generally unrelated to drug prevalence rates except for alcohol, whose rates declined among drivers living longer. The time interval between death and specimen collection was related to alcohol and amphetamine prevalence; delays in obtaining specimens may have elevated those prevalence rates, apparently due to the postmortem redistribution of drugs phenomenon.

(6) The 25-54 age range was overrepresented among drivers involved with abuse drugs, including alcohol. Drivers over 55 tended to be overrepresented among the cases in which prescription drugs were detected.

(7) In comparisons with the drugfree drivers, statistically significant elevations in responsibility rates were found in drivers with alcohol alone and with all high-BAC-drug combinations. The responsibility rates of drivers with THC-only or cocaine-only were not higher than the drugfree rates.

(8) Logistic regression analysis indicated that responsibility rates increased significantly with the number of non-alcohol drugs in a driver; this analysis controlled for potentially confounding variables. The results suggested that alcohol and drug impairment effects were additive.

(9) An analysis controlling for blood alcohol concentration suggested that an alcohol-drug additive effect may be especially important at subintoxication levels of alcohol; however, this needs further investigation.

(10) Among FARS-reported driver factors, speeding was associated primarily with alcohol.

(11) Drivers who had ingested alcohol and/or drugs of abuse presented similar patterns, dominated by the age range of 25-54, male drivers, and drivers with at least one previous speeding or other traffic violation. They also tended to use their restraint systems less than the drugfree drivers.

Conclusions.

(1) Alcohol is still the predominant drug problem; it was by far the most prevalent substance, it was found mainly at intoxication levels, and drivers with alcohol in their systems had the highest crash responsibility rates.

(2) Drugs other than alcohol had relatively low prevalence rates, which limited the capability of the responsibility analyses to find impairment effects. However, there was evidence that multiple drug use impairs drivers, and an alcohol-drug combined impairment effect was suggested by the responsibility data.

(3) Apparent drug impairment effects (as suggested by responsibility analysis) may be due in part to broader behavioral patterns associated with drug use. That is, drug use may be just one manifestation of a broader pattern of high-risk behavior, as suggested by a history of traffic violations.

ACKNOWLEDGEMENTS

The conduct of this project involved a large number of people across the country, and their cooperation is gratefully acknowledged. Space permits us listing only those individuals with primary responsibilities in the project, but we are aware that many more participated, e.g. local coroners who obtained blood specimens for the study. Our appreciation is expressed to all unlisted participants as well as those listed below.

Participants at the Sampling Sites

Alameda County, California:	Smiley Anderson* Gerald Anderson Lt. Duane Gull Lt. Timothy Buckhout
Solano County, California:	James O' Brien Nita Ladd* William Lovelace Jon Garsee Phillip Brown
San Bernadino County, California:	Nelson Alexander Douglas Colwell* Mark Schrader Debra Lichthart
San Diego County, California:	Dr. Brian Blackbourne Richard Shaw*
Los Angeles County, California:	Gary Siglar James Njavro Scott Patterson*
The State of Massachusetts:	Dr. Joann Richmond Sherie Chestis Linda Pothier* Janet Linhares Dr. Stanton Kessler John Laliberte Jerome DiNicola Steve Greziano Peter Burke
The State of North Carolina:	Dr. Andrew Mason John Lacey Paula Hendricks* Carol Popkin

Washoe County, Nevada:	Vernon McCarty*
Fairfax County, Virginia:	Dr. James Beyer Dr. Frances Patricia Field Barbara Gage*
Madison, Wisconsin:	Dr. Patricia Field Thomas Ecker* Richard Schmidt
Milwaukee County, Wisconsin:	Dr. Jeffrey Jentzen Steve Lackey* Warren Hill
Tarrant County, Texas:	Dr. Angela Springfield Thomas Martin*
Dallas County, Texas:	Dr. Charles Petty Dr. Jeffrey Barnard Jody Andreotta* Hamo Meguerditchian

Participants in the FARS Offices

California:	Michael Sowvlen*
Massachusetts:	Anthony Conti*
Nevada:	Mary Lynn Allison Sharon Temple*
North Carolina:	Barbara Mills*
Texas:	James Templeton Susan Vecera*
Virginia:	Sheila Taylor*
Wisconsin:	William Hoebel Joseph Bauknecht*

*These people served as site coordinator.

Others

Dr. Paul J. Kostyniak, Director of the Toxicology Research Center at the State University of New York at Buffalo, served as toxicological consultant to the project during its design phase.

At the American Medical Laboratories, Inc., Anthony Constantino provided valuable information on assay processes and on the postmortem redistribution of drugs phenomenon.

LIST OF TABLES

LIST OF TABLES (Continued)

LIST OF TABLES (Continued)

LIST OF FIGURES

1.0 INTRODUCTION

In a 1988 Report to Congress by the National Highway Traffic Safety Administration, it was concluded that the extent to which drugs other than alcohol were a highway safety problem could not be specified precisely (Compton,1988). Crash studies found that somewhere between 10 and 15 percent of fatally injured drivers had nonalcoholic drugs in their systems, though none of the studies was broadly representative and the extent to which the drugs helped to cause the crashes was mainly unknown. This situation led to the study reported here, which examines drug presence in 1,882 fatally injured drivers from seven States. The study does not use a fully representative national sample, but its inclusion of locations across the country provides an approximate national picture while indicating regional variations. In addition, the study examines the causal role of drugs in the crashes.

Why study fatally injured drivers, since they constitute less than one percent of crash drivers in the United States (National Safety Council, 1991)? One reason is that highway deaths may be one of the more serious consequences of drug use and abuse. A less apparent reason is that a representative sample of the blood specimens needed to study drugs in crashes is more readily obtained from driver fatalities than from crash survivors or from a noncrash driver population.

This study examines 44 different drugs, including cocaine and cannabis as well as prescription drugs such as tranquilizers, antidepressants, and sedatives. All are drugs which scientists and highway safety experts have believed capable of impairing drivers sufficiently to cause crashes.

1.1 Research Objectives

The objectives of this study were as follows:

(1) To determine the extent to which potentially impairing drugs are found in fatally injured drivers. Here we are interested in the question of magnitude; how prevalent are the various drugs found in the drivers? Further, does the extent of prevalence vary regionally across the country? And, are the various drugs found mostly at high blood concentrations, or are they more often at minimal 'trace' levels?

(2) To examine the role drugs play in causing fatal crashes. This addresses the basic question: which drugs, if any, are impairing drivers sufficiently to cause crashes? While all the drugs examined have been judged capable of such behavioral effects, it is only by examining crash data that we can learn whether drugs are contributing significantly to crash causation.

(3) To clarify the circumstances of crash drug presence. This objective addresses a number of questions to help us understand how the drugs are linked to crashes: Are particular drugs found more in some age groups than in others? Do some age groups seem more sensitive than others to the effects of drugs? What kinds of crashes are linked with the drugs? Are any drugs associated with particular vehicle types, crash times, or road conditions?

1.2 Background

We begin this section by considering drug prevalence rates in previous crash studies. Prevalence rates express the percentages of drivers in whom various drugs were found. It is important to understand that a drug's prevalence rate, by itself, implies nothing about its impairment effects (if any) or the crash risks associated with it. These considerations of crash causation are addressed in Section 1.2.3. It is also important to recognize that studies may differ in their drug test sensitivities and in other test factors, which may influence their reported drug prevalence rates.

In Compton's 1988 Report to Congress, he reported four North American studies of drugs in driver fatalities, and one study of injured-but-surviving drivers. From the results, Compton estimated that drugs other than alcohol were present in 10 to 22 percent of the drivers, with the higher figure reported in the single injured-driver study. Cannabis (i.e., marijuana, hashish) was the most commonly found drug, while other prominent drugs were diazepam (Valium (R)), cocaine, amphetamines, and prescription drugs including tranquilizers and sedatives. In the majority of cases where drugs were found, alcohol was also present.

Since Compton's report, researchers continued to study drugs in crash drivers. Some examined other bodily substances in addition to blood, which can result in inflated prevalence rates. For example, Root (1988) reported that drugs of abuse were found in twenty-two percent of car and truck drivers, but this figure may have been inflated by the analysis of urine and other bodily substances as well as blood. In descending order, the most prominent drugs were amphetamines, cocaine, cannabis, phencyclidine, and morphine. Another California study examined driver fatalities in Los Angeles (Budd, Muto, and Wong, 1989). The total drivers with drugs in their systems appeared to be about 28 percent, though it was not clear that detection was always in blood. The most prominently found drugs were cannabis (19%) and cocaine (8%), while barbiturates and phencyclidine were found at low rates (<2%). Finally, a New York City study focused on cocaine, finding it in the blood of 20 percent of the drivers (Marzuk, Tardiff, Leon, Stajic, Morgan, and Mann, 1990).

Drugs in injured drivers taken to a regional trauma center in Toronto, Canada were studied by Stoduto, Vingilis, Kapur, Sheu, McLellan, and Liban (1991). They found nonalcoholic drugs in 41 percent of these drivers, an unusually high rate. That was probably inflated by reporting detection in either blood or urine. In descending order, the drugs found were cannabis, benzodiazepines, cocaine, morphine, barbiturates, codeine, meperidine, diphenhydramine, and pheniramine.

As in the research reviewed by Compton, the subsequent studies usually found that when drugs were in a driver's system, alcohol was present in at least half the cases. In the Budd et al (1989) study, for example, alcohol was present in 67 percent of the cannabis cases and 78 percent of the cocaine cases.

It is apparent from the studies reviewed by Compton and the later studies, that drug prevalence rates in crashes vary over time and among the locations studied. In addition, variations may be caused by the bodily fluids sampled, the drugs tested for, and test sensitivities. These variations reinforced the need for a comprehensive study of drivers in a geographically broad American sample.

1.2.1 Drugs and Driver Populations

Drugs tend not to be uniformly distributed within crash driver populations. Alcohol and other drugs are generally more prevalent among young drivers and male drivers (Terhune, 1982; Donelson, Haas, and Walsh, 1986; Marzuk et al., 1990). Drugs of abuse seem to be particularly prominent among motorcycle drivers (Warren, Simpson, Hilchie, Cimbura, Lucas, and Bennett, 1981; Terhune, 1982; Root, 1988).

1.2.2 Drug Use Trends

Crash studies conducted at various times presumably reflect societal trends in drug use. Consequently, it is useful to briefly examine data on these trends.

A National Household Survey on drug use is conducted annually by the National Institute on Drug Abuse (NIDA). The survey revealed a declining trend in drug use during the 'eighties, with the exception of a small peak in cocaine use in 1985 (Figure 1.1).

To complement self-reports of drug use, trends in hospital emergency room episodes, i.e., overdose emergencies, are relevant. Table 1.1 shows data from the NIDA Drug Abuse Warning Network (DAWN). Although these data are not wholly comparable from year to year because of sampling variations, the data in Table 1.1 exhibit an increase in marijuana reports from 1985 through 1987, and an increase in cocaine reports from 1985 through 1989. In contrast, declines were exhibited in amphetamine, phenobarbital, and diazepam

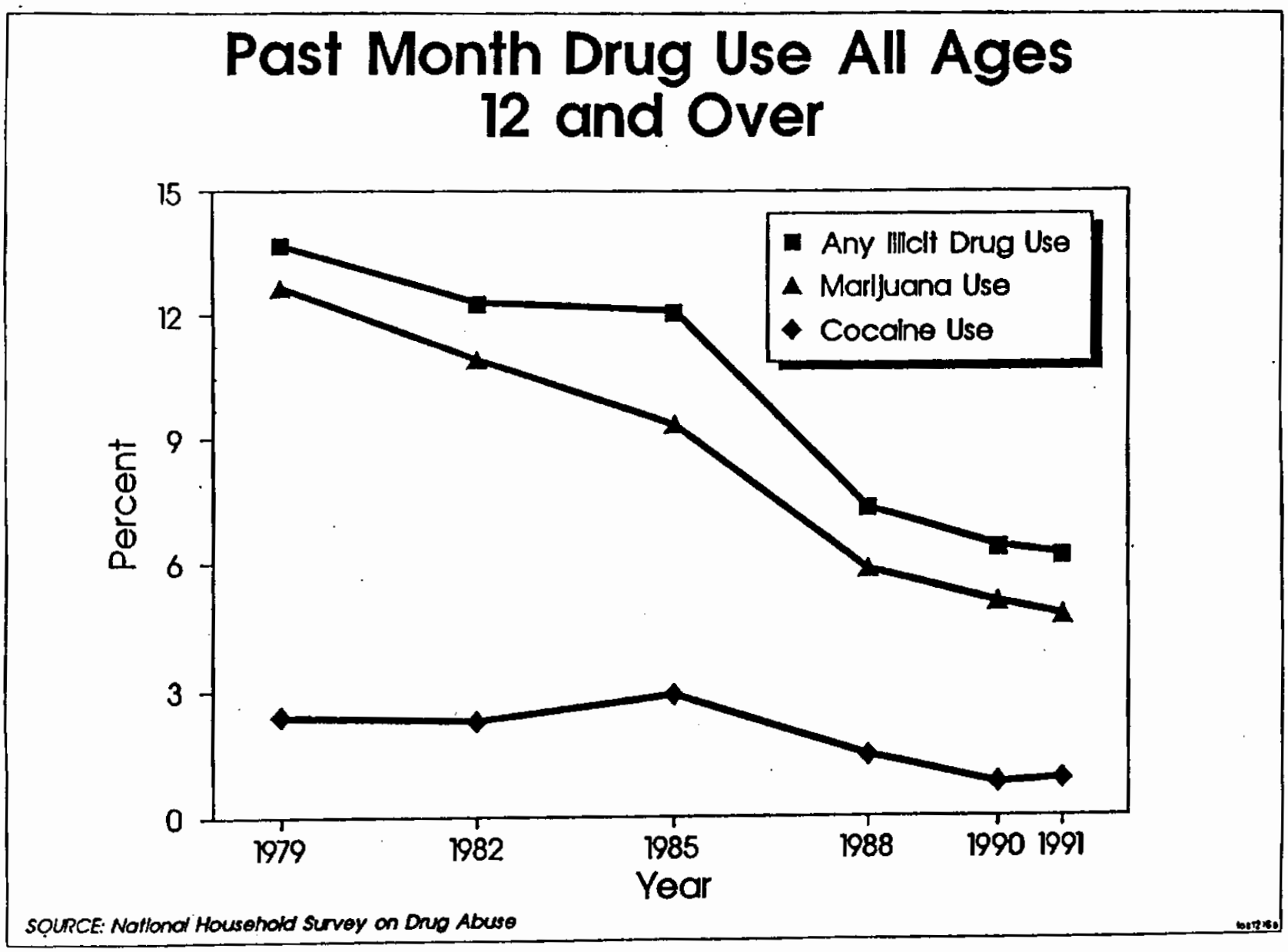


Figure 1.1 DRUG ABUSE TRENDS (NATIONAL INSTITUTE ON DRUG ABUSE, 1992)

Table 1.1

Trends in Emergency Room Episodes Concerning Selected Drugs
Drug Abuse Warning Network (DAWN) - 1985-1989

	Report Series G, No. 21 (1988) (Unweighted Data)			Report Series G, No. 24 (1990) (Unweighted Data)		
	1985	1986	1987	1987	1988	1989
Marijuana	4336	5072	7938	6656	7376	6738
Trend*	<----------(+)---------->			<----------(0)---------->		
Cocaine	12,195	22,586	39,363	33,888	45,697	45,684
Trend	<----------(+)---------->			<----------(+)---------->		
Amphetamine	1035	1029	1089	875	1008	742
Trend	<----------(0)---------->			<----------(-)---------->		
Phenobarbital	1601	1394	1292	954	894	755
Trend	<----------(-)---------->			<----------(-)---------->		
Diazepam	7713	7207	6625	5105	4391	3419
Trend	<----------(-)---------->			<----------(-)---------->		

*Trend test results were given in the DAWN reports. (+) = increasing over time; (-) = decreasing; (0) = no trend.
See the original reports for slope magnitude and other details.

episodes. Thus, the NIDA National Household Survey and the DAWN data convey opposite pictures of trends in the drugs of abuse; cannabis and cocaine use were decreasing according to the household study, but increasing in emergency department episodes.

A possible explanation of the apparently contradictory trends is provided by an observation recently made by Robert Martinez of the administration's Office of National Drug Control Policy. He noted that the number of casual users of abuse drugs has declined "markedly and steadily" in recent years, while "hard-core" users have been more entrenched in their habits. He maintained that the DAWN figures are a measurement of hard-core drug use (Buffalo News, 1992). It may be inferred that NIDA's household survey reflects mainly casual use. Highway accidents are likely to reflect both hard-core and casual use, and it is difficult to predict which trend would dominate.

1.2.3 Drugs and Crash Causation

The causal role of drugs in crashes has been difficult to establish. The main reason for this has been the lack of a control group of on-the-road drivers, as used in the classic Grand Rapids study of alcohol crash effects (Borkenstein, Crowther, Shumate, Zeil, and Zylman, 1974). When a control group is available, greater prevalence of a drug in crash drivers than in the control group indicates that the drug raises crash risks. A major obstacle to such a control group in drug studies is that it requires stopping drivers and obtaining blood specimens from a representative sample. A less powerful but readily used alternative method is responsibility analysis, in which each crash driver is rated for her/his responsibility in causing the crash, preferably without the rater's knowledge of any drug involvement. If proportionately more drug-present drivers are judged responsible than are those free of drugs, this is considered evidence of an impairment effect of the drugs. This method revealed the effects of alcohol in several studies (Terhune, 1983), but it has not consistently indicated impairment by other drugs. Part of the difficulty may lie in the variations of responsibility rating methods used, but small numbers of drug-present cases have also been a problem. Small numbers greatly reduce the capability of responsibility analysis to detect impairment effects.

Of the few studies using responsibility analysis, the study by Warren et al. (1981) found evidence of impairment by cannabis, tranquilizers, and antihistamines, among driver fatalities. No statistical significance was indicated, however. Terhune (1982) found statistically marginal evidence that cannabis was impairing injured drivers. Williams et al. (1985) found no evidence that cannabis contributed to the crashes of fatally injured young male drivers, but they did find that responsibility rates increased with multiple drug use.

In the injured-driver study by Stoduto et al. (1991) cited above, it was found that single-vehicle accidents constituted 32 percent of the crashes of the drug-present drivers, compared with only 20 percent of the drugfree drivers. Researchers generally judge drivers in single-vehicle accidents to be responsible for their crashes, so the results suggest that the drug-present drivers had responsibility rates much higher than the drugfree drivers.

Quite different in approach was a study by Ray, Fought, and Decker (in press). Instead of assaying drugs in the body fluids of crash drivers, they determined risks of an injurious crash for drivers who had received prescriptions for various drugs. They used prescription and crash records of 16,262 elderly (65-84) drivers in Tennessee. Within this group, the relative risks found were: 1.5 for any psychoactive prescription drug; 2.2 for cyclic antidepressants; 1.5 for benzodiazepines; and 1.1 for antihistamines or opiod analgesics. The relative risks were as high as 5.5 for high dosages of antidepressants. To put these figures in perspective, a relative risk of 1.5 to 2.0 is associated with a blood alcohol concentration (BAC) of around .06%, while a 5.5 relative risk resembles alcohol effects at concentrations above .10% (Hurst,1970).

1.3 The Value and Limitations of a Driver Fatality Study

To conclude this Introduction, it is useful to review what a study of drugs in driver fatalities can and cannot accomplish. The first advantage is one that epidemiological (crash) studies generally have over experimental ones. While experimental research (using a driving simulator, closed-course driving, or other controlled situations) is valuable for showing impairment capabilities of a drug, a crash study can reveal (a) the actual extent of drug involvement in accidents, and (b) whether the drug concentrations are at significant levels. Second, a driver fatality study has the previously noted advantage of addressing the most serious outcome i.e., deaths, that drugs may have. Third, a driver fatality study is generally able to include a far higher and more representative proportion of a population of drivers than can an injured-driver or noninjured-driver study. The latter require voluntary cooperation of drivers, which can significantly reduce the completeness and representativeness of a sample.

On the debit side, driver fatality crashes provide a very restricted view of the total crash scene. The conditions and causes of fatal crashes may be very different from those in less severe crashes, and it is possible that drugs play a larger role in the latter. Second, the dominance of alcohol in fatal crashes can make it difficult to discern the particular contribution of drugs; when drugs are found in a driver fatality, alcohol is frequently found, at intoxication levels. Third, driver fatality samples are generally found to have very high responsibility rates, even among drugfree drivers. For drug effects to be statistically significant, the drugs must have extremely high responsibility

rates, substantial prevalence rates, or both.

Finally, it is important for the reader to understand that if responsibility analysis indicates that a specific drug is not contributing to fatal crashes, this does not imply the general conclusion that it is safe for anyone to drive after ingesting the drug. The hazards for an individual will depend on the dosage taken and the individual's response to the drug.

In summary, a driver fatality study has excellent capability to reveal the extent of drug prevalence in the most serious kind of crash, but its capability to reveal drug causal effects is more limited. That was the opportunity and the challenge of this study.

2.0 THE DRUGS OF INTEREST

The objectives of this study required that we examine a highly comprehensive list of the drugs thought capable of causing crashes. Determining drug presence comprehensively (the first objective) required the ability to capture many if not most of the drugs that the drivers had ingested. To study causal contributions of drugs (the second study objective), the responsibility analysis required a control group of drivers who were "drugfree." Clearly, the control group could only be shown "free" of the drugs tested for, which again argued for a comprehensive drug list.

In developing the drug test list, we began with a NHTSA-provided list of 55 different "drugs of interest," including 29 of "most interest." In submitting the list to candidate assay laboratories, we found that the analysis costs would well exceed the funds. Consequently, we sought to eliminate drugs of the least importance, add others if warranted, and establish an order of importance for the drugs on the list. "Importance" was operationally defined as the product of (a) estimated prevalence of a drug and (b) its ability to impair drivers. Estimated prevalence was determined through a literature review of recent studies, from medicinal drug sales data, and reports of NIDA's Drug Abuse Warning Network. Impairment capability was more loosely evaluated through ratings of a panel of experts participating in a NHTSA-sponsored drug workshop (Joscelyn and Donelson, 1980). These data sources were supplemented by contacting thirteen experts on drugs and highway safety across the United States. This group included research scientists, officials in drug abuse centers, law enforcement officials, and toxicologists in the States scheduled to participate in the data collection. They gave their recommendations of the drugs to be included and estimates of drug prevalence rates based on their experience.

In selecting the individual drugs, it was necessary to consider their assay costs. While processes have been developed to efficiently provide screenings for entire classes of drugs, a few drugs of interest were so costly to assay that they were omitted from the test list. (The main example here is triazolam, a tranquilizer.) At the same time, a few drugs of lesser importance were included because they would be captured, at no extra cost, by the screening processes for the more important drugs. (These "add-on" drugs were mainly antidepressants, antipsychotics, and antiarrhythmics.) Interested readers will find in Appendix A a more detailed description of how the drug test list was established.

The drug test list is shown in Table 2.1. It includes the main drugs of abuse found in previous studies: alcohol, cannabis, cocaine, amphetamines, and opiates, as well as the hallucinogens phencyclidine (PCP) and lysergic acid diethyliamide (LSD). Also on the list are important prescription drug classes: benzodiazepines (minor tranquilizers including diazepam), barbiturate sedatives, nonbarbiturate sedatives, and others.

Table 2.1

The Drugs Studied

Substance	Examples of Trade Names or Street Names
Alcohol (ethanol)	booze, juice, sauce
Cannabis	pot, ganja, grass, weed, mary jane
Hallucinogens	
Phencyclidine	PCP, angel dust
Lysergic acid diethylamide	LSD, acid
Benzodiazepine Tranquilizers	
Diazepam	Valium(R)
Lorazepam	Ativan(R)
Flurazepam	Dalmane(R)
Alprazolam	Xanax(R)
Oxazepam	Serax(R)
Chlordiazepoxide	Librium(R)
Barbiturate sedatives	
Phenobarbital	Barbita(R)
Secobarbital	Seconal(R)
Butabarbital	Butisol(R)
Butalbital	Sandoptal(R)
Pentobarbital	Nembutal(R)
Amobarbital	Amytal(R)
CNS Stimulants	
Cocaine	coke, blow, snow, nose-candy
Amphetamine	speed
Methamphetamine	meth, crystal meth
Caffeine	----------
Non-barbiturate sedatives	
Ethchlorvynol	Placidyl(R)
Methaqualone	Quaalude(R)
Meprobamate	Equanil(R), Miltown(R)
Antihistamines	
Diphenhydramine hydrochloride	Ingredient of Benadryl(R), Diahist(R), Nytol(R), Sominex(R)
Chlorpheniramine	Ingredient of Chlortab(R), Chlor-Trimeton(R)
Antidepressants	
Amitriptyline	Elavil(R), Amitril(R)
Imipramine	Trofranil(R)
Doxepin	Adapin(R)
Fluoxetine	Prozac(R)
Narcotic Analgesics	
Meperidine hydrochloride	Demerol(R)
Methadone	Dolophine(R)
Propoxyphene	Darvon(R)
Oxycodone	Ingredient of Percodan(R)
Codeine	----------
Morphine	M, white stuff
Heroin	H, smack, horse, junk
Antipsychotics	
Chlorpromazine	Thorazine(R)
Thioridazine	Mellaril(R)
Mesoridazine	Serentil(R)
Antiarrhythmics	
Quinidine	Cin-Quin(R), Quinora(R)
Procainamide	Promine(R)
Lidocaine	Xylocaine(R)
Flecainide	Tambocor(R)
Muscle relaxant	
Cyclobenzaprine	Flexeril(R), Lisseril(R)

There are various ways by which the drugs may impair driving skills: decreasing alertness, degradation of motor skills, reducing visual acuity, disinhibition with attendant increase in risk-taking, slowing reaction time, degradation of judgment and decision-making, and so on. While experimental research has revealed performance decrements caused by specific drugs, the linkage of these decrements to crash risks in the "real world" has been difficult to establish. For the interested reader who wishes to know more about the effects of drugs in our test list, a brief review is provided in Appendix B.

3.0 RESEARCH DESIGN AND METHODOLOGY

Preliminary Note: This chapter is intended for the readers who want only a summary description of the procedures used. Scientific readers who need more detail will find it in the appendixes. Casual readers may wish to read only the Overview paragraph below, then skip to Chapter 4.

* * *

Overview: The research plan of this study called for the collection of blood specimens from fatally injured drivers meeting our sampling criteria within selected States. The sampling criteria were intended to select crashes in which drugs could have played a causal role, while also enhancing chances of finding any drugs that were affecting the drivers at the moment of their crashes. Sampling sites included three entire States (Massachusetts, North Carolina, and Wisconsin) and selected counties from California, Nevada, Texas, and Virginia. These sites were chosen to achieve broad regional representation and for their previously demonstrated ability to provide blood alcohol data to the Fatal Accident Reporting System (FARS). Data collection was planned for approximately one year, during which blood specimens were to be collected from as complete a driver sample as possible. These specimens were express-shipped to our assay laboratory, where they were analyzed for all drugs on our test list. To provide information about the crashes and cause of death, the project team collected FARS reports, police accident reports (PARs), coroner/medical examiner reports, and certain records created specially for this research. From these data sources, variables were extracted to permit analyses addressing the research objectives. The data were encoded and entered into an automated database, which was used for statistical analyses. The central analyses were (a) the generation of drug prevalence rates for each sampling site and for the entire sample, and (b) responsibility analyses to suggest which drugs contributed to the occurrence of the crashes.

Details of these procedures are provided in the sections that follow. They discuss these topics:

- The driver sampling plan
- The data collection system
- Blood assay procedures
- The responsibility analysis method

3.1 The Driver Sampling Plan

NHTSA initially suggested a sample of 2550 drivers for our consideration. With this figure as a guideline, we performed a statistical power analysis to determine a sample size that would be capable of (a) estimating drug prevalence rates within a few percentage points, and (b) detecting a drug-associated driver responsibility rate that was at least 10 percent higher than the rate for drugfree drivers. A description of the power analysis is provided in Appendix D. Our conclusions from this effort were as follows:

(1) The targeted sample of 2,550 drivers would be able to estimate drug prevalence rates within plus or minus 1 percent, an acceptable level.

(2) Because a driver-fatality sample is expected to have very low prevalence rates of drugs present by themselves, the target of 2,550 drivers has minimal ability to reveal impairment effects of individual drugs other than alcohol, when using responsibility analysis. Even a 3500-driver sample appears capable of detecting drug effects only under best-scenario conditions, e.g. a drug present by itself at a minimum prevalence rate of 5 percent and a minimum responsibility rate 10 percent higher than the drugfree rate.

(3) A 2,550-driver sample has a somewhat greater possibility of detecting impairment effects of alcohol-drug combinations than of drugs present by themselves.

These conclusions were discussed with the NHTSA staff overseeing the project, and it was decided that the original target of 2,550 drivers was acceptable. It would more than adequately meet the objective of estimating drug prevalence rates. While that sample size would only marginally meet the statistical needs for responsibility analysis, a sample significantly improving that capability would have to be at least twice as large. That sample size was considered prohibitive in its acquisition and assay costs, as well as in the size and complexity of the data acquisition system that would be needed. Consequently, plans went forward for obtaining a driver sample of approximately 2,550 drivers.

3.1.1 Driver Eligibility Criteria

The objective of using driver screening criteria was to obtain a sample of fatally injured drivers (a) whose crashes could have been the result of drug influence, (b) from whom useful blood specimens were available, and (c) who represent the predominant driver groups on the road. The criteria are shown in Table 3.1 and are explained below.

Table 3.1

Driver Eligibility Criteria

A driver fatality was included in the study only if it met all the following sampling criteria:

(1) Driver of a car, motorcycle, or truck.

(2) Driver age 15 years or older.

(3) Crash vehicle was in transport.

(4) Driver died within 4 hours of crash.

(5) Blood specimen was taken within 96 hours after death.

(6) Uncontaminated blood specimen available.

(7) Death not due to suicide, homicide, or natural causes.

- Driver of a car, motorcycle, or truck. These are the predominant vehicles on the road, and they include light trucks (pickups, vans, and utility vehicles).

- Driver age 15 years or older. This criterion excludes unusually young drivers, who would likely be driving illegally and in very atypical situations.

- Crash vehicle was in transport. This means that the vehicle was in motion, operated by a driver. Drivers of parked vehicles were excluded because their driving behavior could not be a cause of the crash. Included, however, were vehicles stopped momentarily while in transport.

- Driver died within 4 hours of crash. This criterion is a compromise between competing considerations. On the one hand, it is desirable to include drivers who died in the crash or shortly thereafter, to minimize the opportunity for metabolism and absorption processes to reduce the concentrations of drugs to nondetectable levels. On the other hand, including only drivers who died within an hour of the crash would, according to FARS data, exclude around 40 percent of all driver fatalities. Not only would this make it more difficult to obtain the desired sample size, it could introduce serious sample bias.

  Most previous drug studies sampled only drivers who died within one or two hours of their crashes. The decision to deviate from this common practice was made only after an in-depth review of literature on drug time-dosage-concentration relationships and conferring with pharmacological experts.

- Blood specimen available within 96 hours after death. Like the previous criterion, this one was established only after extensive discussion, including careful consideration of the trade-offs. To minimize evaporation of volatile substances and clotting of blood, a specimen taken within a few hours of death is preferable. However, some of the coroner and medical examiner offices reported that backlogs develop in processing incoming fatalities during peak demand periods, especially on weekends. They expressed the need for a 96-hour time limit, in order not to lose a significant number of otherwise eligible cases. This was allowed after conferring with the toxicologists at American Medical Laboratories. Their opinion was that the

96-hour limit was tolerable, and they increased the sensitivity of their benzoylecgonine test to better the chances of cocaine detection. The field sites agreed to refrigerate the cadavers if the blood specimens couldn't be taken immediately.

- Uncontaminated blood specimen available. Only blood specimens that are not mixed with other bodily fluids or foreign contaminants were desired, for they could produce misleading or incorrect drug assay results. In practice, this criterion necessarily relies on appearances and the judgment of the person taking the specimen. Because crash fatalities may have severe trauma and internal injuries, it may not always be possible to obtain a specimen that assuredly meets this criterion.

- Death not due to suicide, homicide, or natural causes. Given the objective of determining whether drugs cause crashes through impairment, these kinds of fatalities are irrelevant. During the study, all three kinds of disqualifying deaths occurred at our sample sites.

Where possible, screening on these criteria was made at the sample sites, which were provided the explicit criteria in writing. All site staffs were instructed to obtain a blood specimen if there was any doubt about any criterion. This would occur, for example, when there was initial uncertainty concerning a victim's role as a driver or passenger. A similar situation obtained when cause of death was not established until later. In all cases, the final determination of case eligibility was made by the Calspan project staff. This was done by reviewing the police accident reports (to determine, for example, the type of vehicle and whether the victim was a driver), the coroner/medical examiner reports (to determine, for example, the cause of death), and other case documents.

It was our expectation that all eligibility determinations could be completed within a maximum of two months, allowing for delays in arrival of case documents from the field sites. However, coroner or other reports were sometimes unavailable as long as six months after a crash.

3.1.2 Site Selection

Prior to award of this contract, NHTSA had made preliminary contact with several States and had suggested five as candidates for participation in this study. The development of our sampling plan began with this list. It was modified as necessary after initial discussions evaluated the capability of the sites to meet our operational needs. In adding new sites, an effort was made to achieve regional diversity. A strict probability sample of all U.S. driver fatalities was not intended, since feasibility

considerations limited our choice of sampling sites (a) to those having demonstrated the needed capabilities, and (b) to a manageable number for system monitoring.

The site selection process involved a review of the most recent FARS blood alcohol data from each State. It was felt that any State that had demonstrated the capability for obtaining blood alcohol tests on 85 percent or more of fatally injured drivers for the FARS system was a good candidate to participate in this study. The alcohol tests require (a) that blood be properly drawn from fatally injured drivers (b) that the blood be appropriately stored, and (c) that the integrity of the specimen be maintained in transmittal to a test laboratory. The FARS records showed that about half the U.S. States met the 85 percent criterion.

With the NHTSA-provided list and the FARS data, the candidate sampling States were contacted by telephone and in-person visits to evaluate site capabilities in relation to our data acquisition needs. It was found that States vary in the degree of centralization by which they assign responsibility for obtaining and analyzing driver blood specimens: it may be assigned to individual counties (as in California), to districts comprising several counties (as in Virginia and Massachusetts), or to the entire State (as in Wisconsin and North Carolina). These variations mandated that we would work with county personnel, with district personnel, or centralized State personnel in obtaining our blood specimens and coroner/ME reports. Each specific location or agency assuming responsibility for sending specimens and coroner/ME reports became a sampling "site".

The States also varied according to whether (a) the blood specimens were collected by individual coroners and shipped to a central location for analysis, or (b) both the specimen acquisition and analysis were performed at the central location. The latter generally occurred in States which perform autopsies on driver fatalities at one or more autopsy centers within the State. The implication for our project operation was that only at the latter would our project staff routinely interact with the people drawing the blood specimens, allowing a closer degree of monitoring of the specimen acquisition process. For this reason, preference was given to selecting autopsy-center types of sites.

Because of the variations of within-State systems, it was feasible and cost-effective to obtain blood specimens from entire States in some instances, and from parts of States in others. Thus, our final sampling system comprised the complete States of Massachusetts, North Carolina, and Wisconsin, selected counties from California, Nevada, and Texas, and the large Northern District of Virginia. Since there were frequently several sampling sites within a State, there was a total of 13 sites. The map in Figure 3.1 shows their locations.

For each of the sites, the expected number of cases was estimated from their record of providing FARS with blood alcohol

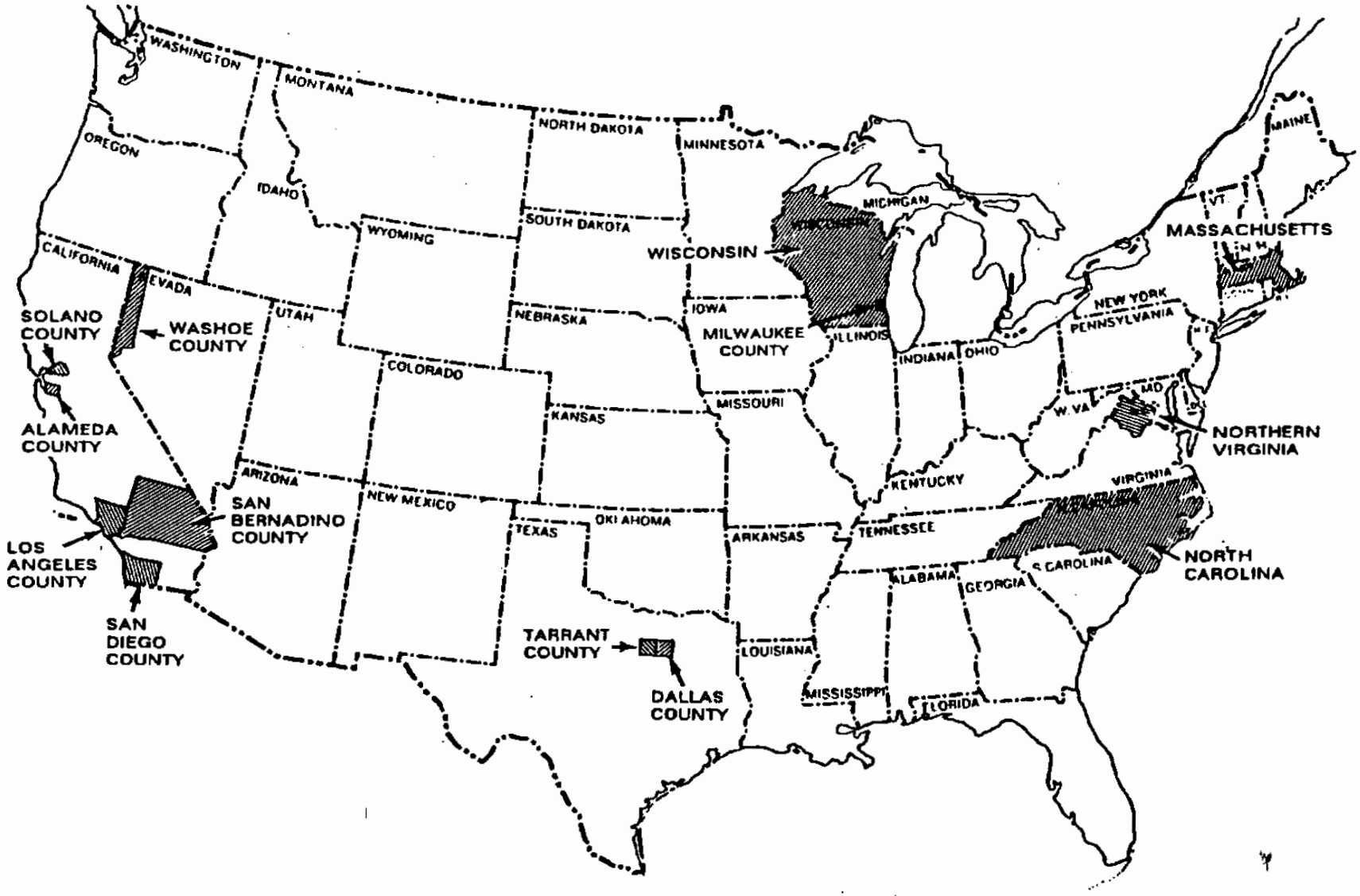


Figure 3.1 SAMPLING SITES FOR THE STUDY

data. It should be noted, however, that the FARS data are based on where the crashes occurred. Because traffic victims may be transported to nearby adjacent counties or States, and subsequently processed by the coroners or medical examiners there, the FARS data do not exactly show the cases handled by the coroners or medical examiners within a county or State. Table 3.2 lists the initially estimated contribution of each site to the sample.

Because our site selection was heavily influenced by considerations of logistic feasibility and effectiveness, the sample should be considered "a sample of convenience." As previously noted, however, effort was made to achieve a sample with sufficient regional diversity to at least approximate a national sample. It included States from the East Coast, the West Coast, the North Central region, and South Central U.S., and it encompassed both urban and rural environments. In Chapter 4, our achieved extent of national representativeness is detailed.

At each of the 13 sampling sites, a person was designated by the responsible chief coroner or ME to be the Site Coordinator for the drug study. These people were our primary site contacts during the study. Usually, however, there were others who also assisted in obtaining blood specimens and providing documents. In Wisconsin and North Carolina, for example, these included the individual coroners distributed across each State.

In addition to the sampling sites, we dealt with the FARS offices in each of the seven participating States. There is one FARS office per State, and each of these had a person designated as our contact for the study. It was through this person that we obtained the needed PARs and FARS reports.

3.2 The Data Collection System

A system was designed to acquire the needed drug and crash data, and to monitor all steps in the procedures. Necessarily it was a complex one, comprising various subsystems. The five major subsystems were:

- Specimen acquisition system. This is the system comprising all the coroners, medical examiners, and their staffs participating in the collection of blood specimens. The specimens were collected in tubes containing sodium fluoride preservative, and express-shipped to the assay laboratory. The coroners/MEs also provided copies of their own reports on each driver fatality (coroner reports, death certificate, etc.) as well as forms completed specially for this project. This system in turn had several subsystems, comprising the components and procedures "tailor-made" to fit the particular requirements of each site.

Table 3.2

Sampling Yields Initially Projected

Site	No. Cases Originally Projected*
California	
Alameda County	53
Los Angeles County	470
San Bernadino County	189
San Diego County	156
Solano County	29
Massachusetts (Entire State)	268
Nevada	
Washoe County	29
North Carolina (Entire State)	646
Virginia	
Northern District	99
Texas	
Dallas County	128
Tarrant County	99
Wisconsin	
Milwaukee County	46
Rest of State	305
Total	2517

*Based on FARS data, which may not reflect the exact numbers of cases processed by coroners or medical examiners within a county or State.

- Crash data acquisition system. This system comprised the FARS offices in each participating State. They provided copies of the police accident reports (PARs) and FARS reports on each fatal crash. These two reports contained all the needed information about the crash and crash conditions, including the reporting officer's description. Variations of this system were required to accommodate the particular organization of FARS offices in each State and the way in which they would coordinate with the medical examiner offices to identify project cases.

- Drug assay system. This system comprised three laboratories. The prime one was American Medical Laboratories, Inc., of Fairfax, Virginia. They performed the drug assays on all the specimens and transmitted the reports to Calspan. The other two laboratories were the Quality Assurance Service Corporation of Augusta, Georgia, and the Chemical Toxicological Institute of Foster City, California. They were used in checking accuracy of the assay results, using specially prepared test specimens.

- Monitoring system. To keep track of operations in the above systems, and to coordinate among them, Calspan operated the monitoring system. This involved continuous interaction with coordinators for the coroner/medical examiners, the FARS offices, and the assay laboratory. To monitor the status of all cases and supplies, the staff employed two computerized databases: the Case Monitoring Database and the Site Supply Database.

- Case preparation system. In operating this system, Calspan logged in all field documents and assembled them. The staff determined case eligibility and encoded information from the field documents into the needed study variables; these data were then entered into an automated database where final quality checks were made preparatory to data analysis.

Figure 3.2 illustrates how each case began with a driver fatality and was completed when its data forms were in final storage. Sometimes, a case became a "noncase" when it failed to meet the sampling criteria and was deleted from the system.

Details of the data collection operations are provided in Appendix D.

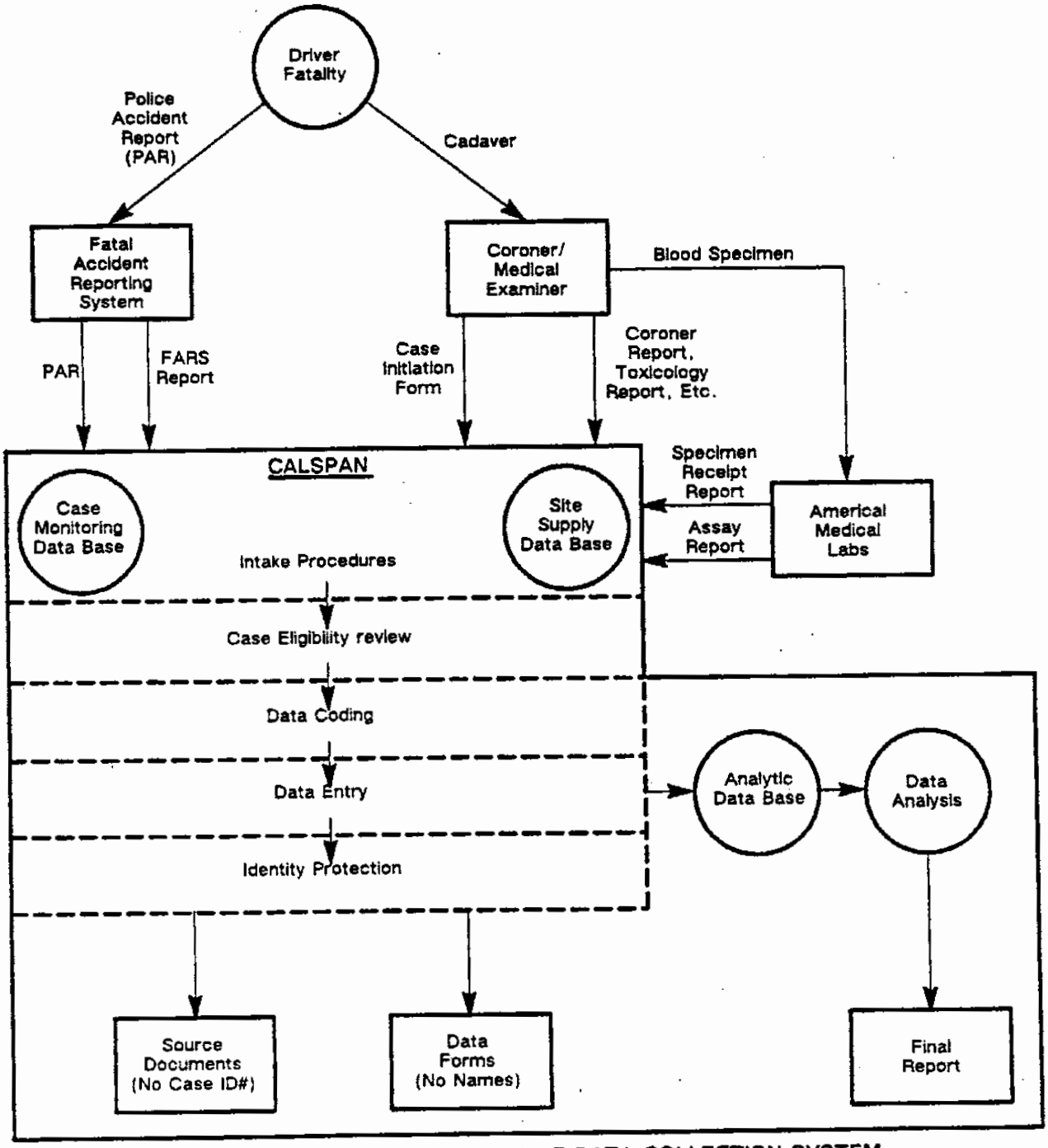


Figure 3.2 CASE FLOW THROUGH THE DATA COLLECTION SYSTEM

3.3 Drug Detection Procedures

Whole blood was selected as the specimen fluid because psychoactive substances found in the blood are more likely to have affected driving performance at the time of the crash than are substances detected in other fluids such as urine. In addition, parent drugs found in blood are more likely to indicate recent ingestion.

While many laboratories are capable of testing for drugs in urine specimens, a much smaller number have the capability of performing blood assays in the volume required for this study. From among the qualified candidates, the American Medical Laboratories (AML) were selected to analyze our specimens. They determined the assay methods to be used for the parent drugs of interest, and they also chose the metabolites to be tested for.

3.3.1 Identifying the Drugs

The general procedure for assaying each of the substances began with highly sensitive screening tests, which signal the presence of particular drugs or drug classes. For example, radioimmunoassay (RIA) was used to identify the presence of cannabinoids, the chemical constituents of Cannabis sativa and their metabolites. The screening tests may have false positives by reacting to irrelevant substances, so they were followed by confirmation tests, which identify particular drug molecules. For example, after a positive cannabinoid screen on a specimen, the presence of tetrahydrocannabinol (THC) or carboxy-THC was determined by gas chromatography and mass spectrometry. At the same time, the concentrations of the substances were measured, in nanograms (billionths of a gram) per milliliter.

Table 3.3 shows the assay methods used for each substance in the test list. Notice that the sensitivity, or detection threshold, is specified for each substance. These thresholds result from the particular methods used and AML's calibrations. The sensitivity limits are critical considerations, because the tests should be able to detect blood concentrations resulting from dosages moderately below those normally taken. Further, the drugs should be detected for a few hours after ingestion, a period during which concentrations usually peak and decline. AML's test methods are described in more detail in Appendix E.

Notice that the list in Table 3.3 includes eleven substances designated as metabolites. These are compounds produced by metabolism of the originally ingested substances, called "parent" drugs. When metabolite tests are included in drug assays, it is usually for one or both of the following reasons:

(a) <u>The metabolite is more likely to be detected than the parent drug</u>. This will be the case when the parent substance is so rapidly metabolized that

Table 3.3
Substances Assayed for, Tests Used,
and their Sensitivities

	Screening Test		Confirmation/Quantitation Test	
Substance	Test Used	Sensitivity	Test Used	Sensitivity
Alcohol (ethanol)	GC/FID	0.005% w/v	GC/FID	0.005% w/v
Cannabinoids				
Delta-9 tetrahydrocannabinol (THC)	RIA	13 ng/ml	GC/MS	1 ng/ml
Carboxy THC (metabolite)	RIA	13 ng/ml	GC/MS	2 ng/ml
Hallucinogens				
Phencyclidine	RIA	12.5 ng/ml	GC/MS	5 ng/ml
LSD	RIA	0.5 ng/ml	HPLC	0.1 ng/ml
Benzodiazepine Tranquilizers				
Diazepam	RIA	100 ng/ml	GC/ECD	100 ng/ml
Nordiazepam (diaz. metab.)	RIA	100 ng/ml	GC/ECD	100 ng/ml
Lorazepam	RIA/GC/MS	50 ng/ml	GC/ECD	5 ng/ml
Flurazepam	RIA/GC/MS	50 ng/ml	GC/ECD	20 ng/ml
Desethylflurazepam (fluraz. metab.)	RIA/GC/MS	50 ng/ml	GC/ECD	20 ng/ml
Alprazolam	RIA/GC/MS	50 ng/ml	GC/ECD	5 ng/ml
Oxazepam	RIA/GC/MS	25 ng/ml	GC/ECD	20 ng/ml
Chlordiazepoxide	RIA/GC/MS	50 ng/ml	HPLC	100 ng/ml
Desmethylchlordiazepoxide (chlordiaz. metab.)	RIA/GC/MS	50 ng/ml	HPLC	100 ng/ml
Barbiturate sedatives				
Phenobarbital	RIA	1000 ng/ml	GC/MS	100 ng/ml
Secobarbital	RIA	1000 ng/ml	GC/MS	100 ng/ml
Butabarbital	RIA	1000 ng/ml	GC/MS	100 ng/ml
Butalbital	RIA	1000 ng/ml	GC/MS	100 ng/ml
Pentobarbital	RIA	1000 ng/ml	GC/MS	100 ng/ml
Amobarbital	RIA	1000 ng/ml	GC/MS	100 ng/ml
Central Nervous System Stimulants				
Cocaine	RIA/GC/MS	50 ng/ml	GC/MS	50 ng/ml
Benzoylecgonine (cocaine metab.)	RIA/GC/MS	50 ng/ml	GC/MS	10 ng/ml
Amphetamine	RIA	50 ng/ml	GC/MS	50 ng/ml
Methamphetamine	RIA	150 ng/ml	GC/MS	50 ng/ml
Caffeine	GC/MS	20,000 ng/ml	HPLC	20,000 ng/ml
Non-barbiturate sedatives				
Ethchlorvynol	Spectrophotometry	50 ng/ml	GC/MS	50 ng/ml
Methaqualone	GC/MS	50 ng/ml	GC/MS	50 ng/ml
Meprobamate	GC/MS	1000 ng/ml	Spectrophotometry	1000 ng/ml
Antihistamines				
Diphenhydramine hydrochloride	GC/MS	20 ng/ml	GC/MS	20 ng/ml
Chlorpheniramine	GC/NPD	50 ng/ml	GC/MS	50 ng/ml
Antidepressants				
Amitriptyline	GC/MS	50 ng/ml	HPLC	5 ng/ml
Nortriptyline (amitrip. metab.)	GC/MS	50 ng/ml	HPLC	5 ng/ml
Imipramine	GC/MS	50 ng/ml	HPLC	5 ng/ml
Desipramine (imipramine metab.)	GC/MS	50 ng/ml	HPLC	5 ng/ml
Doxepin	GC/MS	50 ng/ml	HPLC	5 ng/ml
Desmethyldoxepin (dox. metab.)	GC/MS	50 ng/ml	HPLC	5 ng/ml
Fluoxetine	GC/MS	50 ng/ml	HPLC	5 ng/ml
Norfluoxetine (Fluox. metab.)	GC/MS	50 ng/ml	HPLC	5 ng/ml
Narcotic Analgesics				
Meperidine hydrochloride	GC/MS	50 ng/ml	GC/MS	50 ng/ml
Methadone	GC/MS	50 ng/ml	GC/MS	50 ng/ml
Propoxyphene	GC/MS	100 ng/ml	GC/MS	50 ng/ml
Norpropoxyphene (propox. metab.)	GC/MS	100 ng/ml	GC/MS	50 ng/ml
Oxycodone	GC/MS	50 ng/ml	GC/MS	50 ng/ml
Codeine	RIA/GC/MS	50 ng/ml	GC/MS	10 ng/ml
Morphine	RIA	50 ng/ml	GC/MS	50 ng/ml
Heroin	RIA	50 ng/ml	GC/MS	50 ng/ml

Table 3.3
(Continued)

Substance	Screening Test		Confirmation/Quantitation Test	
	Test Used	Sensitivity	Test Used	Sensitivity
Antipsychotics				
Chlorpromazine	GC/MS	100 ng/ml	HPLC	10 ng/ml
Thioridazine	GC/MS	100 ng/ml	HPLC	10 ng/ml
Mesordiazine	GC/MS	100 ng/ml	HPLC	10 ng/ml
Antiarrhythmics				
Quinidine	GC/MS	500 ng/ml	Immunoassay	100 ng/ml
Procainamide	GC/MS	500 ng/ml	Immunoassay	500 ng/ml
N-Acetylprocainamide (Procainamide metab.)	GC/MS	500 ng/ml	Immunoassay	500 ng/ml
Lidocaine	GC/MS	100 ng/ml	Immunoassay	200 ng/ml
Flecainide	GC/MS	200 ng/ml	HPLC	100 ng/ml
Muscle-relaxant				
Cyclobenzaprine	GC/MS	50 ng/ml	HPLC	10 ng/ml

GC = gas chromatography
RIA = radioimmunoassay
FID = flame ionization detector
MS = mass spectrometry
ECD = electron capture detector
HPLC = high pressure liquid chromatography

chances are much better of finding the metabolite. It may also be the case that available assay methods are more capable of detecting the metabolite than the parent drug. Benzoylecgonine (metabolite of cocaine) and norpropoxyphene (metabolite of propoxyphene) are on our test list because they are more likely to be detected than their parent drugs. The specimens were also analyzed for the presence of the parent drugs.

(b) The metabolite is active. Some metabolites such as benzoylecgonine are inert, while others are important to detect because they can have psychological or physical effects that could impair driving. The effects may be similar or different from those of the parent. Active metabolites on our list are nordiazepam, desethylflurazepam, nortriptyline, nordoxepin, norfluoxetine, desipramine, desmethyldoxepin, and n-acetylprocainamide.

Conversely, if a parent drug is usually detectable, and its metabolites are not known to contribute substantially to psychoactivity, the metabolites are not routinely measured. Also, a psychoactive but difficult-to-detect metabolite may not be tested for if the parent is more readily detected. Reflecting the complexities of biochemical processes, some substances can be either a parent drug or metabolite.

In preparing the drug data for analysis, algorithms were written to identify parent drugs from the specific substances found in the blood specimens. These algorithms were based on the metabolite and parent drugs detected. Because of the complex relationships that may obtain among the substances, it was sometimes necessary to incorporate "best bet" inferences in these algorithms. For example, if codeine and morphine were both present and the codeine/ morphine ratio was less than 0.1, then morphine was identified as the parent drug present. It was sometimes necessary, however, for some ambiguity to remain in the identification of a drug. For example, when amphetamine was found in a specimen but methamphetamine was not, then the drug was simply identified as the amphetamine group. The algorithms are presented along with the programs for other derived variables in Appendix H.

3.3.2 Assay Quality Checks

The basic quality control on the assay procedures was performed by AML, as part of their standard procedures. In addition, independent quality checks were made by Calspan. They are described here.

The main quality check involved the preparation of 52 test specimens spiked with prespecified concentrations of selected drugs

of interest. They were not distinguishable from the other study specimens and were inserted into the shipments from one of the sites. AML's assay reports for these specimens were compared with those of another laboratory, the Chemical Toxicological Institute of California. The results were very satisfactory. AML identified all spiked substances except in one instance when spiked phencyclidine was found by AML at a level below their sensitivity limit, and they properly reported the substance as not detected. The variances in AML's reported concentrations also appeared satisfactory. A detailed report of the quality check is presented in Appendix F.

A secondary check on AML's analyses was made by comparing their results with findings from San Bernadino County's toxicological laboratory. San Bernadino performed the most extensive drug testing of the field sites. As Figure 3.3 shows, there was satisfactory agreement on the prevalence rates.

Another secondary check made was with the BAC results provided by the Madison, Wisconsin laboratory. A Pearson correlation of 0.99 was found between the Wisconsin readings and AML's, an excellent result.

3.4 The Responsibility Analysis Method

One of the contract requirements was to select, develop and apply a responsibility analysis method for detecting drug impairment effects. We had reviewed various methods of responsibility analysis in a previous publication (Terhune, 1983), so that effort was updated for this project. More recent studies found to use responsibility analysis were by Williams et al (1985) and Donelson, Haas, and Walsh (1986). The first of these rated driver responsibility on a two-point scale, while the second used a three-point scale. Both provided some evidence of validity (an increase of responsibility rates with BAC), and the Donelson et al. method evidenced high test-retest reliability on a small sample (39 drivers). Limited information was available on how to use the two methods. Since there was no compelling reason to choose either of these methods over the 5-point scale we employed in a previous study for NHTSA (Terhune, 1982), we decided to build on our previous work. In so doing, we performed the following:

(1) A coding manual was developed. It clarifies the meaning of the responsibility scale, it presents explicit definitions of terms, and it gives coding guidance for particular kinds of crashes. It also provides practice cases and their responsibility codes. The responsibility scale is shown in Table 3.4.

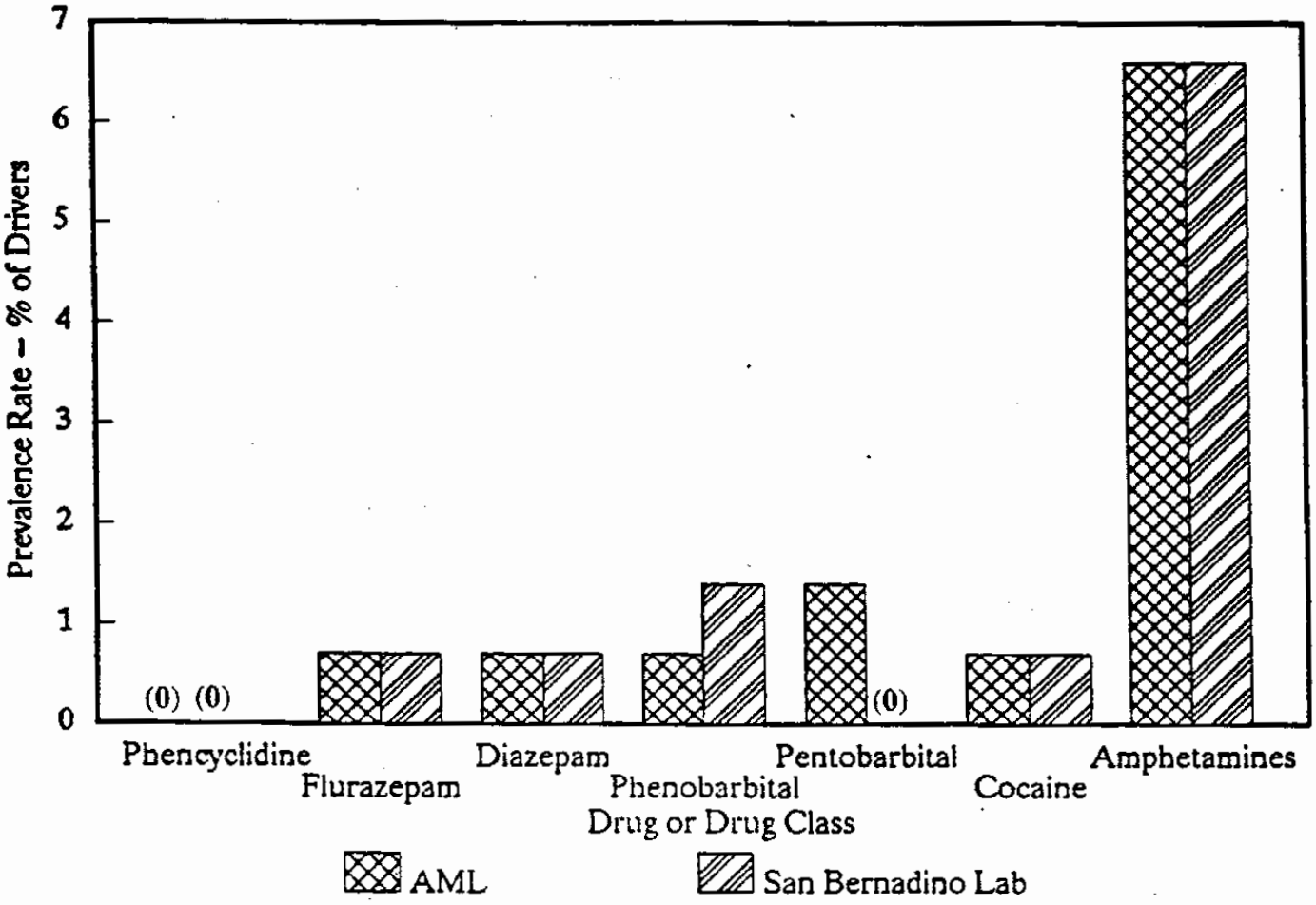


Figure 3.3 COMPARISON OF ASSAY RESULTS: 142 SAN BERNADINO CASES -- AML'S ASSAYS VS. SAN BERNADINO'S LAB ASSAYS

Table 3.4

Explication of the Crash Responsibility Scale

(4) Responsible -- Actions of the subject driver-vehicle created the critical situation.

(3) Responsible/contributory -- Driver had some responsibility, but it is not clear whether he was responsible or contributory.

(2) Contributory -- Another vehicle or agent created the critical situation, but the subject driver could have avoided the crash by a normal evasive maneuver or by driving defensively or by giving a warning signal (e.g., horn, flashers)

(1) Contributory/neither -- At most, the driver's responsibility was only contributory.

(0) Neither responsible nor contributory -- Driver had no responsibility for the accident.

(8) Unknown -- Information is insufficient for rating responsibility. Score when driver may be fully responsible or not responsible at all. Use rarely.

Definitions

Agent -- The precipitator, animate or inanimate, of an event; may be another vehicle, a person (e.g., pedestrian), an animal, or a natural phenomenon such as a tree falling on the road.

Critical situation -- A condition in which a crash is imminent, though it may still be avoidable. (Note: Lack of defensive driving does not in itself define a critical situation.)

Defensive driving -- Driving so as to minimize chances of a critical situation developing. Consists of maintaining alertness, anticipating possible hazards, taking precautionary actions. Examples are: sounding one's horn when a vehicle encroaches on one's travel lane; slowing and watching for crossing vehicles at a yellow blinker light; slowing when a pedestrian appears about to cross the street.

(2) Coding reliability and validity were evaluated with the refined technique. Reliability was assessed via intercoder agreement when different coders independently coded the same cases. Three different coders were employed in this task. It was found that after an initial practice on 40 cases, intercoder correlations averaged above 0.90. Validity was demonstrated by showing that responsibility rates increased systematically with BAC. (Further evidence on this is provided with the results in Chapter 5.)

(3) Theoretical aspects were developed. The theoretical considerations underlying the notion of crash responsibility were discussed and related to concepts of causation.

These three products were incorporated into "A User's Guide to Rating Crash Responsibility," which is presented as Appendix G of this report.

Two trained coders did the coding of driver responsibility in all the cases of the study. The cases were assigned in approximately random fashion, and each coder rated responsibility in half the cases. To evaluate the consistency of their coding over time, the coders exchanged 50 cases and recoded them at approximately three-month intervals. Their agreement on each set was measured by the Pearson correlation coefficient of their ratings. Table 3.5 shows the very high correlations maintained.

3.5 Database Assembly

Needed information about the cases came to Calspan from the various field documents, and this information was encoded into relevant variables on code sheets. The data sources and the code sheets are summarized in Table 3.6. An automated database was created on a microcomputer, using the SPSS Data Entry program. This involved assigning names, value labels, field widths, etc. to all the variables.

3.5.1 Data Quality Control

The data for all cases were keypunched a second time to check their correctness, using the SPSS verification program. Other checks made on the data were as follows:

- Drug concentration ranges. The SPSS Data Entry program was used to identify all drug concentrations with unusually high values. These were reviewed with our assay laboratory for confirmation or correction.

Table 3.5

Intercoder Reliability Checks on Responsibility Coding

Date of check	08/08/90	11/19/90	01/24/91	06/11/91
Number of single-vehicle crashes	25	27	22	24
Number of multi-vehicle crashes	25	23	28	26
Pearson r, multi-vehicle crashes*	.98	.99	.97	.92
Pearson r, all crashes*	.98	.99	.98	.93

*Between-coder agreement level is shown by Pearson correlation coefficient of two coders ratings on the same cases.

Table 3.6

Field Documents and Code Sheets Used in Database Preparation

Information Category	Field Document Information Source	Data Entry Code Sheet
Specimen collection time factors	Case Initiation Form Coroner/M.E. Report Specimen Receipt Report	Time Variables Code Sheet
Specimen condition & assay results	Drug Assay Report Therapeutic Drug Report	Drug Assay Report
Driver responsibility, collision type	Police Accident Report	PAR Data Code Sheet
Crash circumstances	Fatal Accident Reporting System Report	FARS Data Code sheet

- Internal consistency checks. Particular values on some variables logically require certain values on other variables. These connections were checked via computer and any inconsistencies were corrected.

- Review of univariate distributions. The frequency distributions or descriptive statistics (means, ranges, etc.) were printed for all variables. These data were inspected for any anomalous values, and questionable cases were identified for confirmation or correction.

3.5.2 Derived Variables

In addition to the basic variables, another 152 variables were derived from the basic variables. (See Appendix H.) Predominantly, these captured central drug dimensions, derived from the basic variables of drug concentrations. The derived drug variables comprised the following:

- Dichotomized variables - These specified the simple presence or absence of each drug.

- Parent drugs ingested - Taking into account the presence of metabolites and whether a drug had been administered in treating the driver, these variables indicate the parent drugs ingested by the drivers.

- Categorized concentration variables - To provide a basis of comparison for the concentrations of the various drugs, these variables expressed the concentrations in categories of "None", "Trace", "Low", "High", and "Toxic". The "trace" categories are concentrations at or slightly above AML's detection thresholds, while the "toxic" levels were provided by the toxicology staff at AML. The range between these extremes was divided into equal-sized "low" and "high" categories. If a toxic level was unknown, then the midpoint of the obtained range of values above trace was made the divider between "low" and "high". Necessarily, other researchers might create different categories. Table 3.7 shows the categories created.

- Drug classes - Each of the drugs was assigned to a class, such as hallucinogen, minor tranquilizer, etc. (See Table 2.1.) Since such classes have not yet been standardized by the pharmacological discipline, they are somewhat arbitrary. Our classes were established in consultation with the AML toxicological staff. Each of the drug class

Table 3.7

Assignment of Drug Concentrations to Categories*

Substance	Concentration Category			
	Trace	Low	High	Toxic
Ethanol	0.01	.02-.05 Low .06-.09 Intermed.	0.10-0.14	≥ 0.15
Delta-9 THC	1-2	3-19	≥ 20	**
Carboxy THC	1-4	5-249	≥ 250	**
Phencyclidine	1-7	8-48	49-89	≥ 90
Diazepam	1-120	121-2499	2500-4999	≥ 5000
Nordiazepam	1-120	121-1099	≥ 1100	**
Chlordiazepoxide	1-120	121-2499	2500-4999	≥ 5000
Desmethylchlordiazepoxide	1-120	121-1999	≥ 2000	**
Phenobarbital	1-120	121-17,499	17,500-34,999	≥ 35,000
Butalbital	1-120	121-4,999	5000-9,999	≥ 10,000
Pentobarbital	1-120	121-4,999	5000-9,999	≥ 10,000
Cocaine	1-60	61-499	500-999	≥ 1000
Benzoylecgonine	1-60	61-44,999	45,000-89,999	≥ 90,000
Amphetamine	1-60	61-99	100-199	≥ 200
Methamphetamine	1-60	61-4,999	≥ 5000	**
Diphenhydramine hydrochloride	1-24	25-4,999	5000-9,999	≥ 10,000
Chlorpheniramine	1-60	61-349	≥ 350	**
Amitryptyline	1-7	8-249	250-499	≥ 500
Nortryptyline	1-7	8-249	250-499	≥ 500
Fluoxetine	1-7	8-699	≥ 700	**
Norfluoxetine	1-7	8-899	≥ 900	**
Meperidine hydrochloride	1-60	61-499	500-999	≥ 1000
Propoxyphene	1-60	61-249	250-499	≥ 500
Norpropoxyphene	1-60	61-999	1000-1999	≥ 2000
Codeine	1-12	13-99	100-199	≥ 200
Morphine	1-60	61-99	100-199	≥ 200
Lidocaine	1-240	241-2,999	3000-5,999	≥ 6000

* Categories were assigned only to substances found in 3 or more drivers. Ethanol concentrations are in % weight/volume; all other substances are in nanograms per milliliter.

** No toxic level was established.

variables indicates the presence/absence of at least one drug in the class.

- Driver classes by drug involvement - Since the units of study are drivers, it is extremely useful to have a variable that places the drivers into mutually exclusive and mutually exhaustive categories according to the array of substances detected or not detected in their blood. The variable created for this purpose is named SUBSAMPL, and its values are shown in Table 3.8.

  In constructing SUBSAMPL, drivers with THC were distinguished from those in which the inert metabolite carboxy-THC was found without THC, so that each could be examined separately. A similar distinction was not made between cocaine and its metabolite benzoylecgonine, because of the low frequency with which cocaine was found alone.

In addition to the derived drug variables, other derived variables were mainly recodes of basic variables into categories useful for data analysis. The algorithms for all the derived variables are presented in Appendix H.

Table 3.8

The Variable SUBSAMPL: The Driver Sample Divided Into Mutually Exclusive and Mutually Exhaustive Categories

Value of SUBSAMPL	Substance Group
0	Drugfree drivers
	Drivers with 1 substance only in system
1	Alcohol, BAC < 0.10%
2	Alcohol, BAC ≥ 0.10%
3	Tetrahydrocannabinol (THC)*
4	Carboxy THC
5	Cocaine/Benzoylecgonine
6	Benzodiazepines
7	Amphetamines
8	Other single substance
	Drivers with alcohol plus another drug
9	Alcohol and THC*
10	Alcohol and carboxy-THC only
11	Alcohol and cocaine/ benzoylecgonine
12	Alcohol and benzodiazepines
13	Alcohol and amphetamines
14	Alcohol and 1 other not above
15	Alcohol and 2 or more other drugs (may include cannabis or stimulant)
	Drivers with non-alcohol combinations
16	2 drugs
17	3 or more drugs

*With or without carboxy THC

4.0 THE OBTAINED SAMPLE

The sites yielded substantially fewer cases than was projected, though this was not apparent until well into the data collection phase. The first problem to be recognized was at the Los Angeles County site. Accumulating case numbers were far below expectations, and investigation revealed that the county medical examiner's staff was overwhelmed by inordinate numbers of fatalities, particularly homicides. Data collection for the study suffered accordingly. Despite efforts to rectify the problem, there was little improvement, and Los Angeles County ultimately yielded only 98 of its projected 470 cases. Clearly, this was a significant loss to the project.

The fact that other sites were not yielding the expected cases became apparent only later, due to the time lags in the arrival of field documents and subsequent identification of eligible cases. To increase the sample size, data collection was extended two months, through May 1991. However, neither Los Angeles County nor North Carolina participated in the additional months. Other sites were finding the continued participation a burden on their staffs, so data collection was not extended further. The final count of eligible cases was 1,882.

After data collection was completed, the FARS automated database for the study period became available for analysis. From this, we made a count of the driver fatalities occurring within the geographical areas and time period sampled by the project. This revealed a total of 2,548 cases meeting those of our sampling criteria that are measured by FARS variables. This suggests that had there been no losses due to other factors, the sites would have provided close to our targeted sample size. However, FARS does not indicate whether sufficient uncontaminated blood was available, whether it was possible to obtain a specimen within 96 hours after death, or whether the fatality was processed at our sites or elsewhere. (Some victims may be transported to adjacent counties or States.) Hence, the FARS data indicate the cases that would have been eligible, had these other factors not intervened.

Table 4.1 compares the FARS counts with the counts provided by each of our sampling sites, including only crashes occurring within each site's geographic boundary (a county or State). The last column lists the "completion rates" for each site, an approximate index of its success in providing specimens for the cases that ideally would have been included in the project. The median site completion rate was 78.9 percent, somewhat below our anticipated completion rate of 85 percent or higher.

(Note that the two smallest sites yielded a few more cases than the FARS counts, resulting in "completion rates" exceeding 100 percent. Since only aggregate statistics were compared, the reasons for the mismatch were not identified.)

Table 4.1

The Eligible Driver Population and the Obtained Sample

Site	(1) Eligible Population, per FARS*	(2) Total Obtained Sample	(3) Within-Boundaries Sample**	(4) "Completion Rate": (3)/(1)
California				
Alameda County	62	40	40	64.5%
Lassen County***	11	12	12	109.1%
Los Angeles County	417	98	98	23.5%
San Bernadino County	174	132	131	75.3%
San Diego County	159	145	145	91.2%
Solano County	26	25	25	96.2%
Massachusetts	234	173	173	73.9%
Nevada-Washoe County	11	42	14	127.3%
North Carolina	668	530	530	79.9%
Virginia-Northern district	109	83	83	76.1%
Texas				
Dallas County	110	115	63	57.3%
Tarrant County	84	106	81	96.4%
Wisconsin	483	381	381	78.9%
Totals	2548	1882	1776	69.7% (Including L.A.)
				78.7% (Omitting L.A.)

*FARS cases designated "eligible" were all victims who met the following criteria: fatally injured driver of car, truck, or motorcycle; died within sampling time frame; died within 4 hours after crash; age 15 or older; crash occurred within geographical boundaries of State/County.

**Includes only victims from crashes within the State or County sampled; cases from adjacent counties were omitted in column (3).

***Lassen County cases were processed at the Washoe County, Nevada site.

4.1 Comparison with a National Population

Although our sample was intended to be only an approximation of a national sample, it is useful to know how similar our sample is to a national population of driver fatalities. The latter was provided by obtaining descriptive data for the entire country from FARS. However, rather than compare our sample with all driver fatalities, it was considered more appropriate to identify that national population of driver fatalities which occurred during our data collection period and which met our sampling criteria of survival time, driver age, and vehicle type. Thus, any differences in the characteristics of our sample and that population could be attributable only to our geographical sampling areas and to any biases introduced by incomplete sampling.

Table 4.2 compares our sample with the national population on variables likely to be related to alcohol and drug use. The sample and population are highly similar in all respects except that our sample is slightly more urban than the national population. The latter aspect likely results from our site selection procedure.

4.2 Adjusting for Sampling Bias

As noted previously, most sites evidenced omission of some eligible drivers from the samples provided. This raised the possibility of sample bias and distortion of our findings regarding drug prevalence rates. To determine the presence of any biases, the sample provided by each site was compared with the eligible drivers at that location identified through FARS. In other words, we compared the drivers listed in the first and third columns of Table 4.1. Comparisons were made on the variables shown in Table 4.2. Large differences were not found, but some sites evidenced bias on driver age, weekends vs. weekdays, crash time of day, and single-vehicle vs. multivehicle crash. For example, the largest bias was at the Dallas site, where weekend fatalities were underrepresented in the sample: 60 percent of the eligible FARS fatalities occurred on weekends, while only 41 percent of the sample fatalities did. Such a bias could lead to an undercount of alcohol and other drugs in the sample.

To provide an accurate picture of drug prevalence rates at the sampling sites, a bias adjustment was effected by adding a weighting variable to our automated database. This weighting variable was created by the following procedure:

(1) For each site, a count was made of the sample drivers in each combination of driver age categories (15-24, 25-54, 55 and older), weekend vs. weekday, daytime (6AM-6PM) vs. nighttime (6PM-6AM), and single-vehicle vs. multivehicle.

Table 4.2

Comparison of the Drug Study Sample With Drivers in the FARS National Population Who Met the Study Sampling Criteria*

Variable of Comparison	FARS Drivers	Drug Study
Driver Age		
15-17	4.6%	4.8%
18-20	11.0	11.6
21-24	13.1	14.0
25-34	26.8	27.8
35-44	16.3	15.8
45-54	9.6	9.8
55-64	7.4	5.8
65 & older	11.3	10.3
Total	100.0%	100.0%
Driver Gender		
Male	76.4	76.3
Female	23.6	23.7
	100.0%	100.0%
Crash Time of Day		
12:01 AM-6:00 AM	26.0	27.1
6:01 AM-12:00 Noon	18.2	18.2
12:01 PM-6:00 PM	26.5	25.4
6:01 PM-12:00 Midnight	29.3	29.3
	100.0%	100.0%
Land Use		
Rural	65.3	58.6
Urban	34.7	41.4
	100.0%	100.0%

*Both the drug study sample and the selected FARS drivers met the following criteria: fatally injured drivers of car, truck, or motorcycle; died within 4 hours after crash; age 15 or older. The time period covered is April 1990-May 1991.

Table 4.2 (Continued)

Variable of Comparison	FARS Drivers	Drug Study
Crash Day of Week		
Sunday	16.2	17.0
Monday	11.3	10.2
Tuesday	11.5	11.7
Wednesday	11.7	12.1
Thursday	13.0	13.0
Friday	16.7	16.7
Saturday	19.7	19.2
	100.0%	100.0%
Manner of Collision		
Noncollision	52.4	51.5
Rear-end	5.0	5.3
Head-on	21.4	23.6
Angle	19.3	16.1
Sideswipe-same dir.	0.9	1.6
Sideswipe-opp. dir.	0.9	2.0
Rear-to-rear	0.1	0
	100.0%	100.0%
Body Type		
Automobile	62.7	65.2
Motorcycle	10.4	10.7
Light truck	24.2	22.2
Medium/heavy truck	2.7	1.9
	100.0%	100.0%
Police Reported Alcohol Involvement		
Alcohol not involved	32.0	30.4
Alcohol involved	29.0	27.3
Not reported	16.4	23.3
Unknown	22.6	18.9
	100.0%	100.0%

(2) For the geographical region of each site, a count was made of the eligible FARS drivers in each of the preceding combinations.

(3) In each of the combinations, every sampled case was assigned the following weight:

$$\frac{\text{No. FARS cases in combination}}{\text{No. sample cases in combination}}$$

Example. At one site, FARS indicated that there were 36 driver fatalities in the combination: weekend crash occurring in the daytime, collision (2 or more vehicles), driver age 25-54. Our sample for that site had only 30 drivers in the combination. Consequently, each of the 30 cases was assigned a weight of 36/30, or 1.20.

By applying the weight variable, the drug prevalence rates estimate what they would have been if the sample had distributions of the adjustment variables (age, etc.) similar to their distributions in the FARS population. These results are presented in the next chapter.

5.0 RESULTS

This chapter presents the results under three main headings. The first is the nature and scope of the drugs found, wherein we present the drug prevalence rates and the patterns of single and multiple drug use. The second is crash circumstances, which are described in terms of driver, vehicle, and environmental characteristics linked to crash drug presence. The third is crash causation, wherein we examine the data for any evidence that drugs helped to cause the crashes. These sections respectively address the first, third, and second objectives of the study.

5.1 The Nature and Scope of Drugs Found

Throughout this section, the prevalence rates have been weighted to compensate for sampling bias at the sites. Interested readers will find unweighted prevalence rates in Appendix I. The weighting had a negligible effect on the prevalence rates for the whole sample, with somewhat greater effect on the rates for the individual sites.

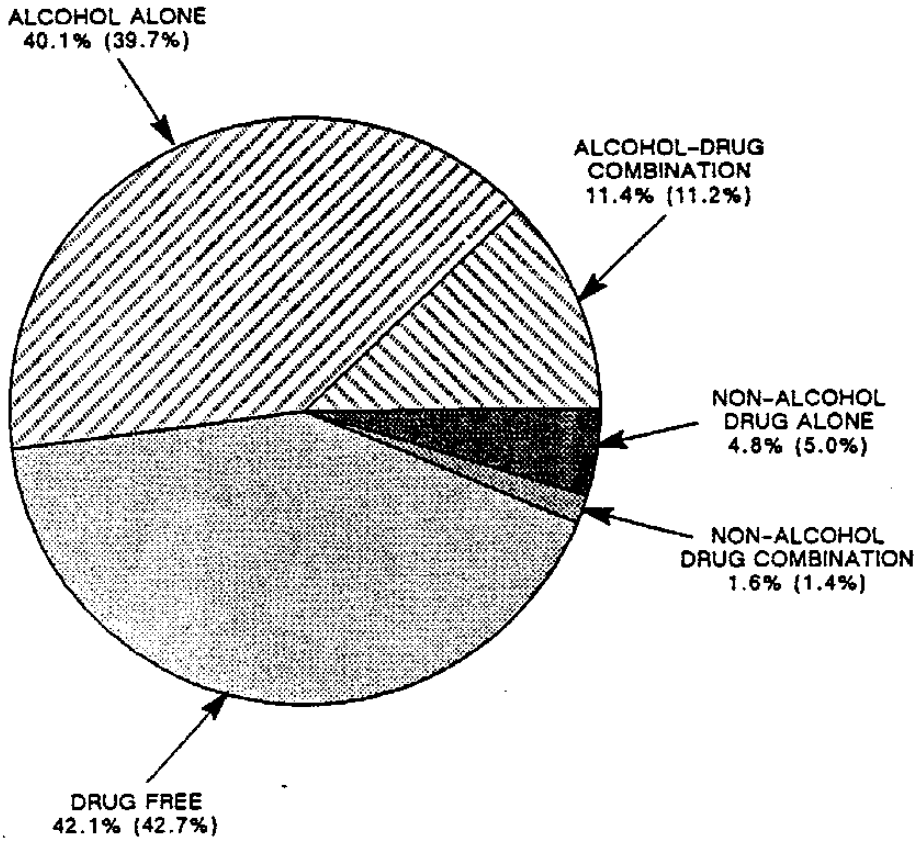
Confidence intervals are provided with the prevalence rates in Appendix I. Although they are not directly applicable to the bias-adjusted rates, they provide a useful reference for the latter.

5.1.1 The Drugs Detected

Figure 5.1 succinctly summarizes the drug involvement of the entire driver sample. Altogether, 57.9 percent of the drivers had at least one substance (alcohol, drug) detected. Alcohol was by far the most prevalent substance, appearing in 51.5 percent of the specimens, mostly in the intoxication range above 0.10% BAC. Nonalcoholic drugs were found in 17.8 percent of the drivers. As the pie chart shows, when drugs were found, alcohol was usually found. In only 6.4 percent of the drivers was a drug detected without alcohol.

Drugs of abuse dominated the substances detected. After alcohol, the most common drugs were cannabis (detected as THC or carboxy-THC in 6.7 percent of the drivers) and cocaine (detected as cocaine or benzoylecgonine in 5.3 percent). Amphetamines appeared at the much lower rate of 1.9 percent, while the hallucinogens phencyclidine (PCP) and LSD were rarely found.

Medicinal drugs were noteworthy for their low frequencies. Even the comprehensive group of benzodiazepine tranquilizers appeared in only 2.9 percent of the specimens, while barbiturates were half as prevalent. Somewhat surprisingly, not a single driver was found to have ingested antipsychotic drugs or nonbarbiturate sedatives.



DRUG or DRUG CLASS	PREVALENCE RATE	
ALCOHOL: BAC $\geq$ .10%	42.6%	(42.6%)
ALCOHOL: BAC < .10%	9.0	(8.3)
CANNABIS	6.7	(6.9)
COCAINE	5.3	(4.5)
BENZODIAZEPINE TRANQUILIZERS	2.9	(3.3)
AMPHETAMINES	1.9	(2.0)
BARBITURATE SEDATIVES	1.5	(1.5)
NARCOTIC ANALGESICS	1.2	(1.0)
ANTIDEPRESSANTS	0.8	(0.8)
ANTIHISTAMINES	0.6	(0.6)
HALLUCINOGENS	0.3	(0.2)
ANTIARRHYTHMICS	0.1	(0.1)
MUSCLE RELAXANTS	< 0.1	(0.1)
ANTIPSYCHOTICS	0.0	(0.0)
NONBARBITURATE SEDATIVES	0.0	(0.0)

Figure 5.1 DRUG PREVALENCE IN THE ENTIRE SAMPLE OF 1882 DRIVERS (DATA WERE WEIGHTED TO COMPENSATE FOR SAMPLE BIAS; RESULTS FOR UNWEIGHTED DATA ARE IN PARENTHESES)

Figure 5.1 provides the prevalence rates of the drugs with and without the adjustments for sampling bias. It can be seen that the effects of the adjustments were slight, and the relative frequencies of the drugs were unaffected.

A breakdown of the individual drugs is given in Table 5.1. Note that the prevalence rates for cannabis and cocaine drop to 4.3 percent and 2.8 percent respectively, when only their parent forms are considered.

Table 5.2 indicates the concentration levels that were found. Very high BACs are reflected in the predominance of toxic concentrations for alcohol. In contrast, the cannabis and cocaine substances were found at trace or low levels. It should be understood that a low concentration could reflect either a low dosage at ingestion or a higher dosage from which the blood concentration had declined substantially by the time of death.

5.1.2 Locational Variations

Particularly interesting are the variations in drug prevalence rates across regions and land use (rural/urban) areas. Regional variations are suggested by Table 5.3. The data are grouped by State, but it should be understood that the statewide representativeness of the California, Virginia, and Texas results is unknown; their sampling sites are mainly urban. The table shows that amphetamines were nearly exclusively found in California, while alcohol, cannabis, and cocaine were unusually prominent in the sampled area of Texas i.e., Dallas-Fort Worth. In contrast, the Wisconsin drivers tended to be lowest in abuse-drug involvement.

Table 5.4 provides detailed results for all the substances -- parent drugs and metabolites -- for each of the specific sampling sites. These data suggest further variations within States. Among the California sites, for example, cannabis was most frequent in San Diego County, while cocaine was most prevalent in the Alameda and Los Angeles samples. In Texas, cocaine was far more common in Dallas County than in Tarrant County (Ft.Worth). Great caution is necessary in viewing the results of individual sites, however, for smaller samples sizes necessarily have lower statistical reliability. At the same time, the results suggest the hazard in making generalizations from single-city or even single-State studies.

A comparison of rural and urban results is provided by Figure 5.2. It shows that drugs were more frequently found in the urban crashes; 20.9 percent of the urban drivers had drugs other than alcohol in their systems, compared to 15.1 percent of the rural drivers. The greatest differences were in the stimulant drugs: cocaine and amphetamines were twice as prevalent in the urban crashes. This is somewhat misleading, however, for the amphetamines were mainly a California phenomenon. A restricted but

Table 5.1

Prevalence Rates of the Drugs Detected*

Data Weighted to Compensate for Sample Bias on Driver Age, Manner of Collision, Time of Day, and Weekend/Weekday

(Note: Percentages are not additive, because more than one drug may be found in a driver's blood.)

Drug	Parent Drug**	Parent or Metabolite**
Alcohol	51.5%	51.5%
Cannabis	4.3	6.7%
Benzodiazepine tranquilizers		
Diazepam	2.2	2.2
Diazepam/chlorazepate/chlordiazepoxide	--	0.3
Flurazepam	<0.1	<0.1
Chlordiazepoxide	0.3	0.3
CNS Stimulants		
Cocaine	2.8	5.3
Amphetamine group	--	0.1
Methamphetamine	1.8	1.8
Caffeine	<0.1	<0.1
Barbiturate Sedatives		
Phenobarbital	0.7	0.7
Butalbital	0.6	0.6
Pentobarbital	0.2	0.2
Narcotic Analgesics		
Meperidine hydrochloride	0.1	0.1
Methadone	0.1	0.1
Propoxyphene	0.6	0.6
Codeine	0.3	0.3
Heroin/codeine/morphine	--	0.1
Antidepressants		
Amitriptyline	0.3	0.3
Imipramine	<0.1	0.2
Doxepin	0.1	0.1
Fluoxetine	0.3	0.3
Antihistamines		
Diphenhydramine hydrochloride	0.4	0.4
Chlorpheniramine	0.2	0.2
Hallucinogens		
Phencyclidine	0.2	0.2
LSD	<0.1	0.1
Antiarrhythmics		
Procainamide	0.1	0.1
Muscle Relaxant		
Cyclobenzaprine	<0.1	<0.1

*Drugs tested for and not found: lorazepam, alprazolam, oxazepam, secobarbital, butabarbital, amobarbital, ethchlorvynol, methaqualone, meprobamate, oxycodone, chlorpromazine, thioridazine, mesordiazine, quinidine, flecainide.

**Data under "Parent Drug" are the percentage of cases in which the specific (parent) drug was detected. Data under "Parent or Metabolite" are the percentage of cases in which either the parent drug of its metabolite was detected.

Table 5.2

Concentration Categories of Substances Found in 3 or More Drivers

Data Weighted to Compensate for Sample Bias on Driver Age, Manner of Collision, Time of Day, and Weekend/Weekday

Substance	# Cases with drug	Concentration Category*				
		Trace	Low	High	Toxic	Total
Alcohol (ethanol)	1314	2.1%	15.3%	19.9%	62.6%	100.0%
Cannabis						
Delta-9 THC	109	22.9	69.7	7.3	**	100.0
Carboxy-THC	172	0.0	99.4	0.6	**	100.0
Hallucinogens						
Phencyclidine	5	0.0	0.0	20.0	80.0	100.0
Benzodiazepines						
Diazepam	56	33.9	66.1	0.0	0.0	100.0
Nordiazepam	53	24.5	73.6	1.9	**	100.0
Chlordiazepoxide	8	0.0	87.5	0.0	12.5	100.0
Desmethylchlordiazepoxide	7	28.6	57.1	14.3	**	100.0
Barbiturate Sedatives						
Phenobarbital	17	0.0	88.2	11.8	0.0	100.0
Butalbital	15	0.0	93.3	0.0	6.7	100.0
Pentobarbital	6	0.0	100.0	0.0	0.0	100.0
CNS Stimulants						
Cocaine	70	30.0	61.4	5.7	2.9	100.0
Benzoylecgonine	134	4.5	95.5	0.0	0.0	100.0
Amphetamine	26	15.4	3.8	46.2	34.6	100.0
Methamphetamine	46	8.7	87.0	4.3	**	100.0
Antihistimines						
Diphenhydramine	9	0.0	88.9	0.0	11.1	100.0
Chlorpheniramine	4	0.0	50.0	50.0	**	100.0
Antidepressants						
Amitriptyline	6	0.0	33.3	33.3	33.3	100.0
Nortriptyline	6	0.0	16.7	66.7	16.7	100.0
Fluoxetine	7	0.0	14.3	85.7	**	100.0
Norfluoxetine	7	0.0	71.4	28.6	**	100.0
Narcotic Analgesics						
Propoxyphene	14	0.0	28.6	0.0	71.4	100.0
Norpropoxyphene	14	0.0	28.6	0.0	71.4	100.0
Codeine	9	0.0	55.6	11.1	33.3	100.0
Morphine	9	22.2	22.2	11.1	44.4	100.0

*See Table 3.8 for concentration ranges of the categories.
**A toxic level was not identified.

Table 5.3

Drug Class Prevalence Rates Within State Groups*

Data Weighted to Compensate for Sample Bias on Driver Age, Manner of Collision, Time of Day, and Weekend/Weekday

Drug Class	California (5 counties)	Mass.	N.C.	Virginia (15 counties)	Wisconsin	Texas (2 counties)
Alcohol (ethanol)	51.8%	54.8%	46.1%	53.3%	48.9%	67.7%
Cannabis	7.7	9.1	4.3	5.9	4.6	13.3
Cocaine	5.3	5.4	5.1	4.8	2.3	12.9
Benzodiazepines	1.5	5.5	4.9	1.2	1.5	4.0
Amphetamines	9.4	0.0	0.0	0.0	0.3	2.1
Barbiturates	1.3	1.1	1.4	0.0	1.4	3.6
Narcotic Analgesics	1.6	0.5	1.3	0.0	0.9	1.5
Antidepressants	1.0	1.6	0.8	0.0	0.5	0.5
Antihistimines	0.4	0.7	0.5	0.0	0.8	1.0
Hallucinogens	0.6	0.0	0.0	0.0	0.3	0.0
Antiarrhythmics	0.0	0.0	0.2	0.0	0.0	0.0
Muscle Relaxants	0.0	0.0	0.0	0.0	0.2	0.0
Nonbarbiturate Sedatives	0.0	0.0	0.0	0.0	0.0	0.0
Antipsychotics	0.0	0.0	0.0	0.0	0.0	0.0
Misc. Other Drugs	1.4	1.8	1.1	1.4	1.8	0.5
Total Cases (Weighted)	830	234	669	108	459	197

Note: Columns do not add up to 100% because more then one drug class can be found for any one driver.

* Washoe, Nevada site was excluded in this analysis due to small sample size.

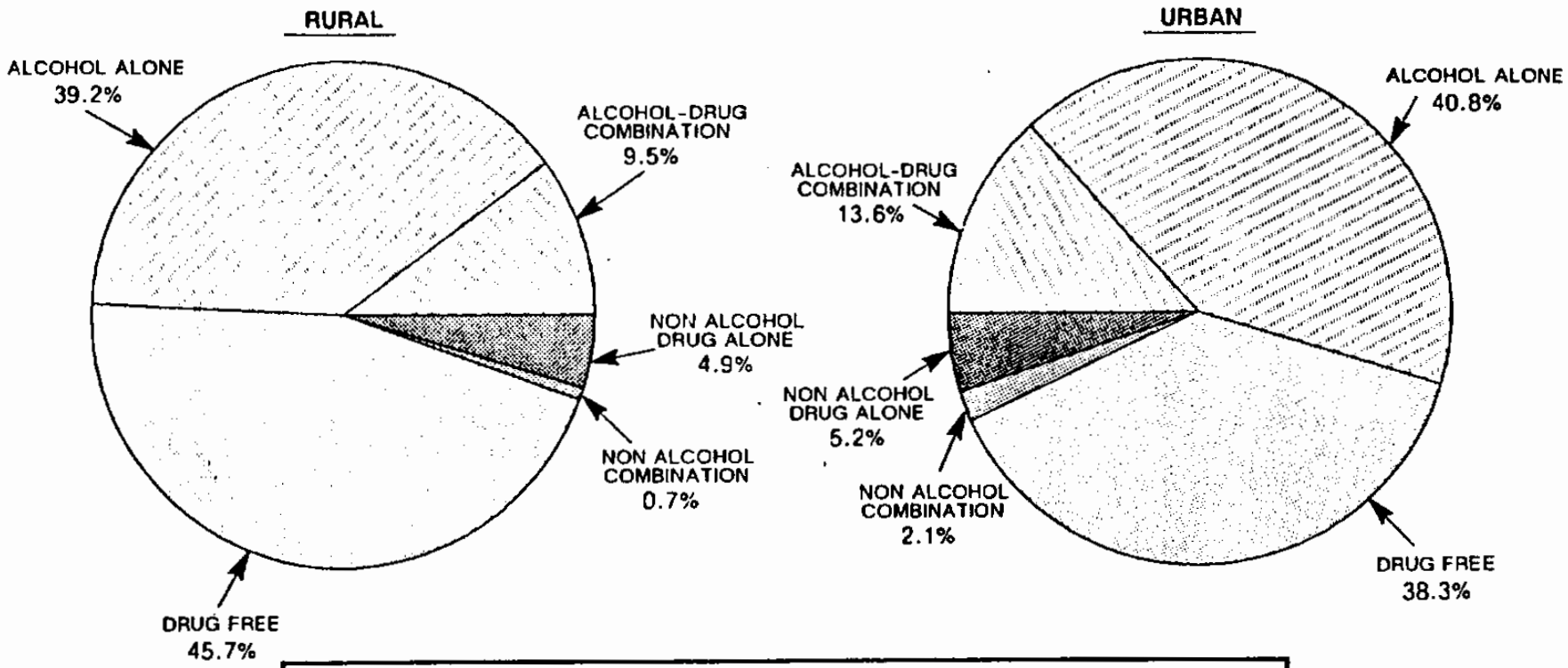
Table 5.4

Individual Substance Prevalence Rates by Sampling Site
Data Weighted to Compensate for Sample Bias on Driver Age, Manner of Collision, Time of Day, and Weekend/Weekday

Substance	Alameda CA	Solano CA	San Bernadino CA	San Diego CA	Los Angeles CA	Massachusetts	North Carolina	Washoe NV	Northern Virginia	Wisconsin Exc. Milw.	Milwaukee WI	Tarrant TX	Dallas TX	All Sites
Alcohol (ethanol): BAC <.10	12.0%	7.7%	9.4%	11.6%	16.8%	4.6%	5.8%	9.3%	11.0%	7.4%	8.0%	12.3%	3.5%	9.0%
Alcohol (ethanol): BAC ≥.10	38.5%	43.6%	30.8%	45.3%	38.1%	50.2%	40.3%	50.9%	42.3%	41.0%	45.1%	56.9%	62.9%	42.6%
Cannabis														
Delta-9 THC	5.6	3.8	3.2	9.1	5.0	5.2	1.8	5.6	1.9	3.3	0.0	9.4	11.4	4.3%
Carboxy THC (metabolite)	5.6	7.7	6.0	13.7	6.4	9.1	4.3	7.4	5.9	5.0	0.0	12.9	13.6	6.7%
Hallucinogens														
Phencyclidine	2.1	0.0	0.0	0.0	0.8	0.0	0.0	0.0	0.0	0.0	2.8	0.0	0.0	0.2%
LSD	0.0	0.0	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	<0.1%
Benzodiazepine tranquilizers														
Diazepam	4.3	9.0	0.6	1.3	0.2	3.7	3.8	0.0	1.2	1.2	0.0	6.1	1.1	2.2%
Nordiazepam (diaz. metab.)	4.3	7.7	0.0	1.3	0.2	2.8	3.9	0.0	1.2	1.2	0.0	6.1	1.0	2.1%
Lorazepam	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Flurazepam	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	<0.1%
Desethylflurazepam (fluraz. metab.)	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	<0.1%
Alprazolam	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Oxazepam	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Chlordiazepoxide	0.0	0.0	0.0	0.7	0.0	1.0	0.5	0.0	0.0	0.2	0.0	0.0	0.0	0.3%
Desmethylchlordiazepoxide (chlordiaz. metab.)	0.0	0.0	0.0	0.7	0.0	0.6	0.5	0.0	0.0	0.2	0.0	0.0	0.0	0.3%
Barbiturate sedatives														
Phenobarbital	0.0	3.8	1.1	1.2	0.0	0.4	0.4	0.0	0.0	0.5	0.0	1.1	4.9	0.7%
Secobarbital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Butabarbital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Butalbital	0.0	0.0	0.0	2.0	0.0	0.7	0.9	1.9	0.0	0.7	0.0	1.1	0.0	0.6%
Pentobarbital	0.0	0.0	1.7	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.2%
Amobarbital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
CNS Stimulants														
Cocaine	3.4	0.0	0.0	1.3	5.9	1.4	2.2	1.9	4.8	0.2	3.3	1.1	13.9	2.6%
Benzoylecgonine (cocaine metab.)	7.8	9.0	0.6	2.2	8.0	5.4	5.1	3.7	4.8	1.9	5.9	5.4	19.4	5.3%
Amphetamine	1.7	9.0	3.7	4.3	1.5	0.0	0.0	0.0	0.0	0.0	0.0	3.3	0.0	1.0%
Methamphetamine	7.8	9.0	7.1	8.5	1.8	0.0	0.0	3.7	0.0	0.3	0.0	0.0	1.1	1.8%
Caffeine	0.0	0.0	0.0	0.6	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Non-barbiturate sedatives														
Ethchlorvynol	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Methaqualone	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Meprobamate	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antihistamines														
Diphenhydramine hydrochloride	0.0	0.0	0.0	0.0	0.0	0.7	0.4	0.0	0.0	0.5	2.8	2.2	0.0	0.4%
Chlorpheniramine	0.0	0.0	1.3	0.7	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.2%

Table 5.4
(continued)

Substance	Alameda	Solano	San Bernadino	San-Diego	Los-Angeles	Mass-achusetts	North Carolina	Washoe	Fairfax	Madison	Milwaukee	Tarrant	Dallas	All Sites
Antidepressants														
Amitriptyline	0.0	0.0	0.0	1.3	0.0	0.5	0.4	0.0	0.0	0.2	0.0	0.0	0.0	0.3%
Nortriptyline (amitrip. metab.)	0.0	0.0	0.0	1.3	0.0	0.5	0.4	0.0	0.0	0.2	0.0	0.0	0.0	0.3%
Imipramine	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Desipramine (imipramine metab.)	0.0	0.0	0.0	0.0	0.8	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.2%
Doxepin	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Desmethyldoxepin (dox. metab.)	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Fluoxetine	0.0	0.0	0.0	0.0	0.7	1.1	0.0	0.0	0.0	0.0	0.0	1.1	0.0	0.3%
Norfluoxetine (fluoxemetab.)	0.0	0.0	0.0	0.0	0.7	1.1	0.0	0.0	0.0	0.0	0.0	1.1	0.0	0.3%
Narcotic Analgesics														
Meperidine hydrochloride	0.0	0.0	0.0	0.0	0.6	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Methadone	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Propoxyphene	0.0	0.0	0.9	0.0	2.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.6%
Norpropoxyphene (propox. metab.)	0.0	0.0	0.9	0.0	2.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.6%
Oxycodone	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Codeine	0.0	0.0	0.0	0.7	0.0	0.5	0.6	0.0	0.0	0.6	0.0	0.0	0.0	0.3%
Morphine	0.0	0.0	0.0	2.0	0.0	1.0	0.0	0.0	0.0	0.3	0.0	1.1	1.9	0.4%
Heroin	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antipsychotics														
Chlorpromazine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Thioridazine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Mesoridazine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antiarrhythmics														
Quinidine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Procainamide	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
N-Acetylprocainamide (Procainamide metab.)	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Lidocaine	7.3	16.7	7.3	13.5	6.6	6.5	4.9	6.5	7.7	3.7	3.1	6.3	9.1	6.4%
Flecainide	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Muscle relaxant														
Cyclobenzaprine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0%
Other drugs	0.0	9.0	4.2	0.6	0.2	1.8	1.1	0.0	1.4	2.0	0.0	1.1	0.0	1.3%
TOTAL CASES (unweighted)	40	25	134	145	96	173	530	54	83	345	36	106	115	1882
TOTAL CASES (weighted)	58	26	174	163	409	234	669	54	108	416	43	92	105	2551



RURAL	DRUG OR DRUG CLASS	URBAN
32.9%	ALCOHOL: BAC ≥ .10%	33.2%
6.3	ALCOHOL: BAC < .10%	7.6
5.3	CANNABIS	8.3
3.5	COCAINE	7.2
3.2	BENZODIAZEPINES	2.6
1.3	AMPHETAMINES	2.5
1.2	BARBITURATES	1.8
1.1	NARCOTIC ANALGESICS	1.3
0.6	ANTIDEPRESSANTS	1.0
0.6	ANTHISTAMINES	0.5
0.1	HALLUCINOGENS	0.4
0.1	ANTIARRHYTHMICS	0.0
0.1	MUSCLE RELAXANTS	0.0

Figure 5.2 DRUG PREVALENCE IN RURAL AND URBAN DRIVER FATALITIES (WEIGHTED DATA)

perhaps more accurate rural-urban comparison is in Table 5.5, which is limited to the States providing statewide samples. Amphetamines hardly enter the picture there, but otherwise the preponderance of drugs in urban crashes is upheld.

5.2 Patterns of Single and Multiple Drug Use

In Figure 5.1, the pie chart gave a succinct overview of the proportions of drivers who were drugfree, alcohol-involved, and so on. Now we shall examine the driver groups in greater detail, using the categories defined by the variable SUBSAMPL (Table 3.8). Table 5.6 shows the driver breakdown. The following points are noteworthy:

(1) Among drivers with one substance only in their blood, alcohol dominated, at intoxication levels. No other single-substance group comprises more than 1 percent of the drivers. Even if we combine THC and its inactive metabolite, only 1.1 percent of the drivers were in the cannabis-only group. Combining cocaine and amphetamines, 1.2 percent of the drivers had only stimulants in their systems. The remaining single-substance cases (the 34 "other" drivers) involved mainly medicinal drugs.

(2) Of the alcohol-drug combinations, alcohol was combined mainly with drugs of abuse. When alcohol was combined with one other drug, the latter was cannabis, cocaine, or amphetamines in nearly 2/3 of the cases. Benzodiazepines were also prominent in the alcohol-drug combinations. In combinations involving alcohol and two other drugs, cannabis, cocaine, and benzodiazepines were predominant. In all alcohol-drug combinations, the alcohol was mainly at intoxication levels.

(3) Multiple drug use not involving alcohol was rare. Table 5.6 shows that a drug combination not involving alcohol was found in only 1.3 percent of the drivers. In these few cases, abuse drugs and benzodiazepines were again prominent.

* * *

[At this point, the analyses move away from drug prevalence rates. The remainder of this chapter examines relationships among variables, particularly the relation of drugs to other variables. Unweighted data were used for these analyses in order to permit statistical tests of significance. Thus, all relationships that follow pertain to the original unweighted data.]

Table 5.5

Drug Prevalence Rates in Rural and Urban Fatal Crashes: Combined Rates of Massachusetts, North Carolina, and Wisconsin

Data Weighted to Compensate for Sample Bias on Driver Age, Manner of Collision, Time of Day, and Weekend/Weekday

	Rural	Urban
Alcohol: BAC $\geq$.10%	42.3%	42.5%
Alcohol: BAC <.10%	6.3	6.2
Cannabis	4.7	6.6
Cocaine	3.8	5.2
Benzodiazepines	3.5	5.0
Amphetamines	0.1	0.0
Barbiturates	1.1	2.0
Narcotic analgesics	1.1	0.6
Antidepressants	0.6	1.3
Antihistamines	0.6	0.8
Hallucinogens	0.0	0.3
Antiarrhythmics	0.0	0.0
Muscle relaxants	0.0	0.0
Total cases	983	376

Table 5.6

The Driver Sample Divided Into Mutually Exclusive and Mutually Exhaustive Groups by Drugs Present

Data were weighted to compensate for sample bias on Driver Age, Manner of Collision, Time of Day, and Weekend/Weekday

Substance Group	Actual Number[8]	Weighted Prevalence
Drugfree drivers	803	42.1%
Drivers with 1 substance only in system		
Alcohol: BAC <0.10%	120	6.9
Alcohol: BAC ≥0.10%	627	33.2
Delta-9 THC[1]	19	0.9
Carboxy THC[2]	6	0.2
Cocaine/Benzoylecgonine	7	0.5
Benzodiazepines	18	0.8
Amphetamines	12	0.7
Other[3]	34	1.9
Drivers with alcohol-drug combination[9]		
Alcohol & delta-9 THC[1]	37 (32)	2.0 (1.7)
Alcohol & carboxy-THC[2]	29 (26)	1.5 (1.3)
Alcohol & cocaine/benzoylecgonine	49 (41)	3.1 (2.7)
Alcohol & benzodiazepines	18 (13)	0.8 (0.7)
Alcohol & amphetamines	13 (9)	0.6 (0.4)
Alcohol & 1 other not above[4]	19 (13)	1.1 (0.7)
Alcohol & 2 or more other[5]	46 (38)	2.3 (1.9)
Drivers with non-alcohol combinations		
2 drugs[6]	20	1.1
3 or more drugs[7]	5	0.2
Total	1882	100.0%

Notes

[1] With or without carboxy THC

[2] Without THC

[3] Includes barbiturates (6), antihistamines (6), narcotic analgesics (5), and miscellaneous others

[4] Includes barbiturates (8), antihistamines (3), and miscellaneous others

[5] Includes cannabis (31 drivers), cocaine (21), benzodiazepines (21), barbiturates (7), and miscellaneous others

[6] Includes barbiturates (7 drivers), benzodiazepines (5), cannabis (5), cocaine (4), amphetamines (4), and miscellaneous others

[7] Includes amphetamines (4), cocaine (3), cannabis (2) and miscellaneous others

[8] The actual numbers cannot be used to calculate the weighted prevalence rates, though they will approximate them

[9] Figures in parentheses are for BAC ≥ 0.10%

* * *

Alcohol-drug patterns. Since alcohol dominated the substance combinations, the specific drug-alcohol combinations are further described in Table 5.7. An interesting implication of the data is that alcohol use and cocaine or cannabis use are not just co-occurrences; they are correlated to a modest degree. This is especially true of cocaine use, which involved alcohol in 83 percent of the cases. The odds ratios in Table 5.7 suggest that not only is cocaine use likely to involve alcohol, but a converse relationship is also indicated: the chances of finding cocaine were 4.9 times higher when alcohol was present than when it was not. Similar but weaker patterns were found with cannabis (THC) present.

The amphetamines did not exhibit the same pattern as cocaine, although both are central nervous system stimulants. In fact, alcohol was less likely to be found when amphetamines were present than when they were not. In inquiring to the National Institute of Drug Abuse as to why the cocaine and amphetamine patterns differ, we learned that users report preferring cocaine with alcohol because it takes the "edge" off the cocaine effect. They apparently do not have the same experience with amphetamines. It is also possible that amphetamines are less frequently combined with alcohol because they are used to combat fatigue.

Table 5.7 shows that the correlations of alcohol presence with the presence of the medicinal drugs were nil. It appears, then, that abusing alcohol implies a tendency to use other drugs of abuse. This suggests a behavioral pattern of users, which we take up in the next section.

5.3 Circumstances of Drugs in Crashes

Here we address the third objective of the study: to describe the crash circumstances in which drugs are found. These were examined in terms of driver variables, vehicle variables, and ambient conditions of the accidents. To explore the network of relationships involving these factors, the variable SUBSAMPL was used.

5.3.1 Driver Patterns

Table 5.8 shows characteristics of the drivers in each of the SUBSAMPL groups. Those groups differed significantly on every one of the variables. The comparisons of most interest are between the alcohol/drug groups and the drugfree drivers.

Age differences. The age differences of the substance groups were pronounced. The 25-54 age range was overrepresented in drivers who had ingested alcohol and/or abuse drugs. The youngest drivers were pronounced in the cannabis and cocaine groups, with or without alcohol also present. The oldest drivers were

Table 5.7

Associations Between Drug Use and Alcohol Use

	Cocaine	THC	Antide-pressant	Benzodi-azepines	Narcotic Analgesic	Amphet-amines	Barbit-urates	Antihist-amines	Halluc-inogen
Correlation of Drug Presence With Alcohol Presence: Phi Coefficient	0.14*	0.08*	0.03	0.04	0.01	0.01	0	0.01	0
Odds ratio: chances of finding alcohol when drug present vs. when not present	1.7	1.4	1.3	1.2	1.3	0.9	1.0	0.8	1.0
Odds ratio: chances of finding drug present when alcohol present vs. when not present	4.9	2.1	1.9	1.6	1.2	0.9	1.1	0.6	1.0
% of cases where alcohol is present when drug is present	83.3%	68.8%	66.7%	61.9%	55.6%	47.4%	51.7%	41.7%	50.0%

*Statistically significant at P< 0.01

Table 5.8

Comparison of the SUBSAMPL Groups on Driver Characteristics

		Single-Substance Groups							Alcohol-Drug Combinations						
	Drugfree	Alc BAC < .10	Alc BAC > .10	Cannabis Only	Cocaine Only	Benzo-diazep. Only	Amphet-amines Only	Any Other Drug	Alc + Cannab.	Alc + Cocaine	Alc + Benzod.	Alc + Amphet	Alc + 1 Other	Alc + 2 Others	Non-Alc Comb.
Age of Driver****															
15-24 yrs.	30.2%	37.5%	33.4%	37.5%	42.9%	5.6%	25.0%	9.1%	37.9%	48.2%	11.1%	7.7%	15.8%	23.9%	16.0%
25-54 yrs.	43.4	51.7	60.1	54.2	57.1	22.2	75.0	51.5	62.1	51.7	83.3	92.3	63.2	76.1	72.0
55-95 yrs.	26.2	10.2	6.5	8.3	0.0	72.2	0.0	39.4	0.0	0.0	5.6	0.0	21.1	0.0	12.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Gender of Driver****															
Male	64.3	82.5	88.2	92.0	71.4	44.4	91.7	78.8	90.9	81.6	66.7	92.3	63.2	87.0	72.0
Female	35.7	17.5	11.8	8.0	28.6	55.6	8.3	21.2	9.1	18.4	33.3	7.7	36.8	13.0	28.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. Speeding Violations***															
None	77.2	67.0	67.9	43.4	71.4	94.4	50.0	77.4	69.2	65.3	94.4	61.5	73.7	61.4	73.9
1 or more	22.8	33.0	32.1	56.6	28.6	5.6	50.0	22.6	30.8	34.7	5.6	38.5	26.3	38.6	26.1
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. Other Violations****															
None	83.3	80.9	72.7	56.5	71.4	100.0	41.7	77.4	63.1	65.3	83.3	76.9	84.2	68.2	82.6
1 or more	16.7	19.1	27.3	43.5	28.6	0.0	58.3	22.6	36.9	34.7	16.7	23.1	15.8	31.8	17.4
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Restraint System Use****															
Not used	57.3	69.6	81.3	56.5	50.0	46.7	72.7	67.9	79.7	79.5	76.9	84.6	87.5	77.5	65.2
Lap/shoulder belt (any)	34.2	25.5	12.3	8.7	33.3	46.7	18.2	28.6	11.9	9.1	15.4	7.7	12.5	17.5	26.0
Helmet	5.5	4.9	6.2	30.4	16.7	6.7	9.1	3.6	6.8	9.1	7.7	7.7	0.0	2.5	8.7
Used; unknown type	3.0	0.0	0.2	4.3	0.0	0.0	0.0	0.0	1.7	2.3	0.0	0.0	0.0	2.5	0.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. drivers (denominator for all %'s except when missing data)	803	120	627	25	7	18	12	33	66	49	18	18	19	46	25

*Chi square significant at .10 level
**Chi square significant at .05 level
***Chi square significant at .01 level
****Chi square significant at .0001 level

overrepresented in the benzodiazepine-only group and in the group that had ingested one "other" drug, which included mainly medicinal drugs.

Gender differences. Males were overrepresented in all the alcohol and drug groups except the benzodiazepine-only group, where females predominated.

Previous traffic violations. Drivers with previous speeding violations were overrepresented in most of the alcohol/drug groups. (Exceptions were drivers with benzodiazepines, with one "other" drug, and perhaps alcohol + 1 other drug.) Drivers with a record of other traffic violations had a similar pattern.

Restraint system use. Nearly every alcohol and/or drug-involved group had used their restraint systems less than had the drugfree drivers. An exception was the benzodiazepines-only group. Another was the cannabis-only group: several drivers in this group were motorcyclists wearing helmets.

Driver patterns summary. Drivers who had ingested alcohol and/or drugs of abuse presented similar patterns, dominated by the age range of 25-54, male drivers, and drivers with at least one prior traffic violation. They also tended to use their restraint systems less than the drugfree drivers. The pattern did not apply to those who had ingested only benzodiazepines or "other" drugs, which were mainly medicinal. Those drivers were likely to be older than 54 and have fewer past traffic violations. The benzodiazepine-only drivers were more frequently female, and used their restraint systems more, than the drug-free drivers.

5.3.2 Vehicle and Collision Circumstances

Table 5.9 shows the composition of the alcohol-drug groups in terms of vehicle and collision variables.

Vehicle types. Vehicle types differed significantly among the driver groups. Passenger cars predominated in the sample, but motorcycles were overrepresented among most of the alcohol and drug groups. Light trucks were also overrepresented in several of those groups. Heavy truck drivers constituted only a small part of the total sample, and there were none in any of the alcohol-drug combinations. No stimulants were found in the heavy-truck drivers.

Number of occupants. The number of vehicle occupants did not differ significantly among the substance groups. The majority of all crashes involved a single occupant.

Manner of collision. The manner of collision data in Table 5.9 were obtained from the FARS reports. Differences among the substance groups were highly significant. "Noncollision" events, i.e., single vehicle crashes, were substantially overrepresented in virtually every substance-detected group, the only exceptions being

Table 5.9

Comparison of the SUBSAMPL Groups on the Vehicle and Its Role

		Single-Substance Groups							Alcohol-Drug Combinations						
	Drugfree	Alc BAC ≤ .10	Alc BAC > .10	Cannabis Only	Cocaine Only	Benzo-diazep. Only	Amphet-amine Only	Any Other Drug	Alc + Cannabis	Alc + Cocaine	Alc + Benzod.	Alc + Amphet.	Alc + 1 Other	Alc + 2 Others	Non-Alc Comb.
Vehicle Type****															
Car	69.7%	65.8%	60.8%	40.0%	42.9%	77.8%	58.3%	57.6%	62.1%	75.5%	72.2%	46.2%	73.7%	69.6%	44.0%
Motorcycle	8.3	9.2	11.0	32.0	28.6	5.6	16.7	9.1	13.6	12.2	22.2	38.5	10.5	15.2	20.0
Light Truck	18.4	20.9	28.1	24.0	28.6	16.7	25.0	33.3	24.3	12.2	5.6	15.4	15.8	15.2	32.0
Heavy Truck	3.5	4.2	0.2	4.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. of Occupants															
1	69.4	66.7	71.8	72.0	71.4	72.2	75.0	84.8	68.2	55.1	77.8	84.6	73.7	69.6	72.0
2	18.4	23.3	19.9	8.0	28.6	27.8	25.0	12.1	25.8	32.7	16.7	7.7	26.3	21.7	20.0
3 or more	12.2	10.0	8.3	20.0	0.0	0.0	0.0	3.0	6.0	12.2	5.6	7.7	0.0	8.7	8.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Manner of Collision****															
Noncollision	32.2	48.7	72.0	36.0	42.9	50.0	50.0	51.5	71.2	63.3	66.7	69.2	63.2	69.6	52.0
Rear end	6.4	6.7	3.4	8.0	14.3	5.6	0.0	15.2	6.1	0.0	5.6	7.7	10.5	4.3	0.0
Head-on	30.6	24.4	16.5	24.0	28.6	33.3	16.7	18.2	13.6	24.5	16.7	15.4	15.8	21.7	16.0
Angle	25.7	15.1	6.4	24.0	0.0	11.1	25.0	15.2	9.1	10.2	5.6	7.7	10.5	4.3	20.0
Sidesup, same dir.	2.1	2.5	0.6	8.0	0.0	0.0	0.0	0.0	0.0	2.0	5.6	0.0	0.0	0.0	8.0
Sidesup, opp. dir.	3.0	2.5	1.1	0.0	14.3	0.0	8.3	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. drivers (denominator for all %'s except when missing data)	803	120	627	25	7	18	12	33	66	49	18	13	19	46	25

* Chi square significant at .10 level
** Chi square significant at .05 level
*** Chi square significant at .01 level
**** Chi square significant at .0001 level

the cannabis-only group. Note that the noncollision percentages were highest in the alcohol-intoxication and alcohol-plus-drug groups. These important differences bear on the subject of crash causation, a topic addressed in section 5.4.

5.3.3 Patterns of Crash Ambient Conditions

Table 5.10 shows that the substance groups differed significantly on several ambient condition variables.

Day of week. Weekends were predominant in the crashes of most of the alcohol-involved groups, the cocaine-only group, and the benzodiazepine only group. Weekdays were somewhat over-represented among the cannabis-only drivers.

Time of day. Group differences in crash time of day were highly significant. The early morning midnight-to-6 AM hours were overrepresented in all the alcohol-involved groups, the amphetamine-only group, and the non-alcohol-combination group. Drivers in the mainly medicinal "other drugs" group tended to be involved more in daytime crashes, similar to the drugfree drivers.

Season. There were no significant differences among the substance groups in the season of crash occurrence.

Land use. As noted previously in the report, drugs tended to be found more frequently in the urban crashes of our mainly rural sample.

Number of road lanes. A large majority of the crashes occurred on two-lane roads. Deviations from this tendency were generally not dramatic, and broad patterns were difficult to discern. The data suggest a tendency for the stimulant-detected crashes to occur more frequently on 4-lane highways than the crashes of other driver groups.

Horizontal alignment. Most of the crashes happened on straight (tangent) sections of highway, but all the driver groups involving alcohol had an overrepresentation of curve crashes. With the possible exception of the "any other drug" drivers, the groups with drugs but not alcohol resembled the drugfree drivers in their predominance of straight-road crashes.

Surface condition. A large majority of the crashes occurred on dry pavement. This tendency was more pronounced among nearly all the alcohol and drug-present groups. The one exception was the benzodiazepine-only group, which had proportionately more wet-surface crashes than the drugfree.

Atmospheric condition. The results for atmospheric conditions paralleled those for surface conditions; dry conditions prevailed, and they were more frequent for the alcohol and drug groups.

Table 5.10

Comparison of the SUBSAMPL Groups on the Ambient Conditions of the Accidents

		Single-Substance Groups							Alcohol-Drug Combinations						
	Drugfree	Alc BAC <.10	Alc BAC >.10	Cannabis Only	Cocaine Only	Benzo-diazep. Only	Amphet-amine Only	Any Other Drug	Alc + Cannabis	Alc + Cocaine	Alc + Benzod.	Alc + Amphet.	Alc + 1 other	Alc + 2 others	Non-Alc Comb.
Day of Week****															
Fri, Sat, Sun.	43.5%	60.0%	64.1%	36.0%	71.4%	66.7%	33.3%	45.5%	48.5%	71.4%	50.0%	46.2%	63.2%	52.2%	44.0%
Weekdays	56.5	40.0	35.9	64.0	28.6	33.3	66.7	54.5	51.5	28.6	50.0	53.8	36.8	47.8	56.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Time of Day****															
Midnite - 6AM	10.3	32.5	45.0	4.0	14.3	0.0	41.7	9.1	40.9	53.1	22.2	30.8	36.8	52.2	16.0
6AM - Noon	30.9	15.0	4.6	24.0	42.9	33.3	16.7	33.3	1.5	10.2	5.6	15.4	15.8	2.2	20.0
Noon - 6PM	38.5	15.0	12.6	40.0	28.6	38.9	16.7	27.3	12.1	16.3	22.2	7.7	15.8	15.2	44.0
6PM - Midnite	20.3	37.5	37.8	32.0	14.3	27.8	25.0	30.3	45.5	20.4	50.0	46.2	31.6	30.4	20.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Season															
Spring	34.1	39.2	33.0	24.0	14.3	50.0	41.7	27.3	24.2	38.8	22.2	69.2	47.4	34.8	24.0
Summer	25.0	21.7	27.8	36.0	14.3	16.7	16.7	36.4	51.5	24.5	38.9	7.7	15.8	26.1	40.0
Autumn	20.8	20.0	22.6	16.0	28.6	27.8	25.0	18.2	16.7	20.4	22.2	15.4	15.8	21.7	16.0
Winter	20.0	19.2	16.6	24.0	42.9	5.6	16.7	18.2	7.6	16.3	16.7	7.7	21.1	17.4	20.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Land Use**															
Urban	38.0	39.5	41.9	56.0	28.6	50.0	50.0	30.3	48.5	57.1	38.9	61.5	47.4	47.8	64.0
Rural	62.0	60.5	58.1	44.0	71.4	50.0	50.0	69.7	51.5	42.9	61.1	38.5	52.6	52.2	36.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. of Travel Lanes**															
1	0.9	0.0	0.8	4.3	0.0	0.0	0.0	0.0	1.5	0.0	5.6	7.7	0.0	0.0	4.3
2	80.7	78.4	82.2	87.0	71.4	88.9	66.7	84.8	84.6	67.3	88.9	61.5	94.4	71.1	69.6
3	6.3	6.0	6.9	4.3	14.3	5.6	0.0	3.0	4.6	14.3	0.0	0.0	0.0	11.1	8.7
4 or more	12.1	15.5	10.1	4.3	14.3	5.6	33.3	12.1	9.2	18.4	5.6	30.8	5.6	17.8	17.4
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Horizontal Alignment****															
Straight	75.0	63.6	59.0	75.0	71.4	77.8	75.0	63.6	45.5	53.1	61.1	61.5	63.2	64.4	80.0
Curved	25.0	36.4	41.0	25.0	28.6	22.2	25.0	36.4	54.5	46.9	38.9	38.5	36.8	35.6	20.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Surface Condition*															
Dry	80.8	82.5	86.3	88.0	71.4	66.7	91.7	93.9	90.9	93.9	83.3	92.3	94.7	87.0	96.0
Wet, snow, ice, etc.	19.2	17.5	13.7	12.0	28.6	33.3	8.3	6.1	9.1	6.1	16.7	7.7	5.3	13.0	4.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Atmospheric Conditions*															
No adverse cond.	84.7	85.8	88.4	92.0	71.4	72.2	91.7	93.9	93.9	95.9	83.3	100.0	100.0	89.1	96.0
Rain, sleet, etc.	15.3	14.2	11.6	8.0	28.6	27.8	8.3	6.1	6.1	4.1	16.7	0.0	0.0	10.9	4.0
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
No. drivers (denominator for all %'s except when missing data)	803	120	627	25	7	18	12	33	66	49	18	13	19	46	25

* Chi square significant at .10 level
** Chi square significant at .05 level
*** Chi square significant at .01 level

Ambient conditions summary. There were marked differences among the driver groups in relation to time, location, and road conditions. Early-morning and weekend crashes were characteristic of several of the substance-present groups, especially those drivers who had ingested alcohol. The latter also were found in curve crashes more than the other groups. Wet conditions were more likely to be a factor in crashes of the drugfree than of the drug-present drivers.

5.3.4 Crash Circumstances, THC, and Carboxy-THC

Since our SUBSAMPL variable distinguished cannabis-present drivers according to whether THC was found, the crash circumstances of the various cannabis groups were examined. In Table 5.11, the carboxy-THC-only and alcohol + carboxy-THC groups differed from their THC-present counterparts in various ways. Consistently, those with carboxy but not THC had higher proportions of (a) the youngest (15-24) drivers, (b) motorcyclists, and (c) summer crashes. Other differences were less consistent, but overall the data indicate that the various groups of cannabis users were not entirely equivalent.

5.4 Indications of Drug Causal Effects

In this section we turn to the question of whether drugs helped to cause the fatal crashes. Our main method was responsibility analysis, in which statistical connections between drug detection and driver crash responsibility were used to identify causal effects. This was supplemented by a brief examination of collision types associated with drug use.

5.4.1 Results of the Responsibility Analysis

It may be recalled from Chapter 3 that trained coders rated each driver's responsibility for the crash on a scale from 0 (not responsible) to 4 (responsible). The ratings were made without the coders knowing (a) whether alcohol or other drugs were present in the crash, (b) the driver's gender, or (c) the driver's age. Table 5.12 shows the distribution of the responsibility ratings. Note that 79.9 percent of the drivers were judged at least contributory to their crashes, and only 15.0 percent were found not responsible.

The responsibility analyses used responsibility rates, the percentage of drivers in a group who were responsible for their crash. To generate these rates, all drivers who were rated 3 or 4 on the responsibility scale were designated "responsible".

Validity. Chapter 3 presented data showing the high intercoder reliability of the responsibility method. It is also important to demonstrate the validity of the method. This was achieved by showing that the driver responsibility rates increased

Table 5.11

Crash Circumstances, THC, and Carboxy-THC

Variable	Drugfree	Cannabis Only: THC Present	Cannabis Only: Carboxy THC Only	Cannabis + Alcohol: THC Present	Cannabis + Alcohol: Carboxy Present, No THC
Age of Driver					
15 - 24 yrs.	30.2%	26.3%	80.0%	29.7%	48.2%
25 - 54 yrs.	43.4	63.2	20.0	70.3	51.7
55 - 95 yrs.	26.2	10.5	0.0	0.0	0.0
Gender of Driver					
Male	64.3	89.5	100.0	91.9	89.7
Female	35.7	10.5	0.0	8.1	10.3
No. Speeding Violations					
None	77.2	50.0	16.7	62.2	78.6
1 or more	22.8	50.0	83.3	37.8	21.4
No. Other Violations					
None	83.3	66.7	33.3	56.8	71.4
1 or more	16.7	33.3	66.7	43.2	28.6
Restraint System Use					
Not used	57.3	70.6	16.7	84.8	73.1
Lap/shoulder belt(any)	34.2	11.8	0.0	9.1	15.4
Helmet	5.5	17.6	66.7	6.1	7.7
Used; unknown type	3.0	0.0	16.7	0.0	3.8
Vehicle Type					
Car	69.7	42.1	33.3	62.2	62.1
Motorcycle	8.3	21.1	66.7	5.4	24.1
Light truck	18.4	31.6	0.0	32.4	13.7
Heavy truck	3.5	5.3	0.0	0.0	0.0
No. of Occupants					
1	69.4	63.2	100.0	67.6	69.0
2	18.4	15.8	0.0	29.7	20.7
3 or more	12.2	21.0	0.0	2.7	10.3
Manner of Collision					
Noncollision	32.2	36.8	33.3	64.9	79.3
Rear end	6.4	10.5	0.0	8.1	3.4
Head-on	30.6	26.3	16.7	16.2	10.3
Angle	25.7	21.1	33.3	10.8	6.9
Sideswp, same dir.	2.1	5.3	16.7	0.0	0.0
Sideswp, opp. dir.	3.0	0.0	0.0	0.0	0.0
Day of Week					
Fri, Sat, Sun	43.5	36.8	33.3	43.2	55.2
Weekdays	56.5	63.2	66.7	56.8	44.8
Time of Day					
Midnite - 6 AM	10.3	5.3	0.0	40.5	41.4
6 AM - Noon	30.9	21.1	33.3	2.7	0.0
Noon - 6 PM	38.5	47.4	16.7	10.8	13.8
6 PM - Midnite	20.3	26.3	50.0	45.9	44.8
Season					
Spring	34.1	42.1	16.7	59.5	41.4
Summer	25.0	5.3	50.0	8.1	27.6
Autumn	20.8	26.3	16.7	2.7	13.8
Winter	20.0	26.3	16.7	29.7	17.2
Land Use					
Urban	38.0	47.4	83.3	48.6	48.3
Rural	62.0	52.6	16.7	51.4	51.7
No. of Travel lanes					
1	0.9	5.6	0.0	2.8	0.0
2	80.7	88.9	66.7	77.8	93.1
3	6.3	5.6	0.0	8.3	0.0
4 or more	12.1	0.0	33.3	11.1	6.9
Horizontal Alignment					
Straight	75.0	72.2	83.3	54.1	34.5
Curved	25.0	27.8	16.7	45.9	65.5
Surface Condition					
Dry	80.8	94.7	66.7	89.2	93.1
Wet, snow, ice, etc.	19.2	5.3	33.3	10.8	6.9
Atmospheric Conditions					
No adverse cond.	84.7	94.7	83.3	91.9	96.6
Rain, sleet, etc.	15.3	5.3	16.7	8.1	3.4
No. Drivers	803	19	6	37	29

Table 5.12

Distribution of Ratings on Driver Responsibility

Value	Label	Frequency	Percent
4	Responsible	1436	76.3
3	Responsible/contributory	67	3.6
2	Contributory	55	2.9
1	Contributory/none	33	1.8
0	Not responsible	283	15.0
8	Unknown resp.	8	0.4
	Total	1882	100.0%

with BAC, reflecting the well-established relationship of BAC to driver impairment and relative crash risk. Figure 5.3 shows the BAC-responsibility relationship for those drivers in our study who had no drugs other than alcohol in their blood. In addition to the results for all crash types, the figure provides data for just the multivehicle crashes. The latter are necessary to demonstrate that responsibility rates do not merely reflect an increase of single-vehicle crashes with BAC. Figure 5.3 shows that the responsibility rates generally increased with BAC, with the sharpest gain as BAC moved into the intoxication range beyond 0.10%. Ideally, responsibility rates should monotonically increase with BAC. While our data did not quite meet this ideal, they did support the general validity of the responsibility ratings.

Table 5.13 gives a more detailed breakdown of the responsibility rates at lower BAC levels. The data exhibit a systematic increase in responsibility rates as BAC moves from the .01-.04% range to the .08-.10% range.

Responsibility, drugs, and alcohol. Responsibility analysis requires division of drivers into groups, and the SUBSAMPL variable provided relevant categories. Table 5.14 shows the responsibility rates of all the SUBSAMPL drivers. Note that the drugfree drivers had a responsibility rate of 67.7 percent, the baseline against which all the other groups are compared.

The first important set of comparisons is with the drug groups listed under "Drivers With 1 Substance Only" in Table 5.14. These indicate the effects of a drug or drug group when it and only it was found in the driver's blood. A major handicap to the responsibility analysis is the small number of cases in these groups, except for alcohol. Only the drivers with alcohol in their blood had a responsibility rate significantly higher than that of the drugfree drivers. The 83.3 percent responsibility rates of the carboxy-only and the amphetamines-only drivers deserve comment. The carboxy-THC result is suspect because of the small sample (n=6), and the previously noted characteristics of this group, i.e. young motorcyclists. The amphetamines result suggests that this drug deserves further study.

Note that the responsibility rates of the THC-only and cocaine-only groups are actually lower than that of the drugfree drivers. Although these results too are inconclusive, they give no suggestion of impairment in the two groups. The low responsibility rate for THC was reminiscent of that found in young males by Williams and colleagues (1986).

The responsibility rate of the benzodiazepine-only drivers was nearly identical to that of the drugfree group. Here also, there is no suggestion of impairment.

The second important comparison set is under "Drivers With Alcohol-Drug Combinations" in Table 5.14. Every one of the listed groups had a responsibility rate significantly higher than that of

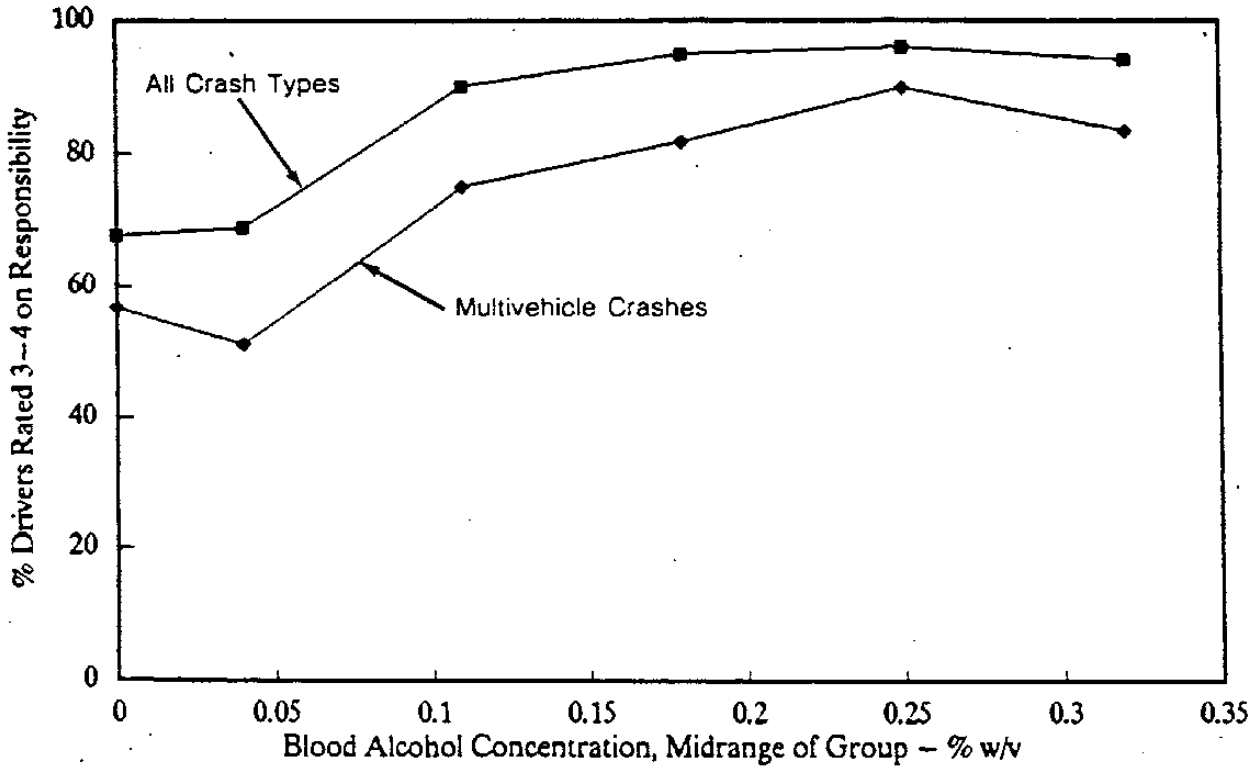


Figure 5.3 RESPONSIBILITY RATES AND BAC: Drivers With No Drugs Other Than Alcohol

Table 5.13

Driver Responsibility Rates at Low BAC Levels
(For all drivers with no drugs other than alcohol; n=1544)

BAC Level	No. Drivers	% Responsible (Rated 3-4)	Chi-Square*	Statistical Significance
0.00% (drugfree)	799	67.7%	--	--
.01-.04%	53	62.3%	0.5	N.S.
.05-.07%	31	80.6%	1.7	N.S.
.08-.10%	54	94.4%	15.8	P<.001
≥ 0.11%	607	93.7%	139.1	P<.001

* Chi-square tests compared BAC groups with the drugfree group.

Table 5.14

Driver Responsibility Rates in Major Substance Groups

Note: The groups below are mutually exclusive and mutually exhaustive.

Substances Present	N (Drivers)	% Responsible[1]	Chi-Square[2]	Statistical Significance
Drugfree	799	67.7%	--	--
Drivers With 1 Substance Only				
Alcohol: BAC <.10%	120	75.8	2.9	P<.1
Alcohol: BAC ≥.10%	625	93.9	144.7	P<.001
THC (with or without carboxy)[3]	19	57.9	0.4	N.S.
Carboxy-THC only	6	83.3	0.1	N.S.
Cocaine	7	57.1	0.6	N.S.
Benzodiazepines	18	66.7	0.0	N.S.
Amphetamines	12	83.3	1.3	N.S.
Any other single drug[4]	34	73.5	0.3	N.S.
Drivers With Alcohol-Drug Combination				
Alcohol + THC (with or without carboxy)[5]	37	94.6	10.7	P<.01
Alcohol + Carboxy THC only	29	93.1	7.2	P<.01
Alcohol + Cocaine	49	87.8	8.7	P<.01
Alcohol + benzodiazepines	17	100.0	6.6	P<.02
Alcohol + Amphetamines	12	91.7	3.1	P<.1
Alcohol + 1 other drug not above	19	100.0	7.5	P<.01
Alcohol + 2 or more other drugs	46	95.7	14.7	P<.001
Drivers With Non-Alcohol Combinations[6]	25	84.0	2.3	N.S.
Missing	8			
Total drivers	1882			

[1]Drivers who were rated as "responsible" or "responsible/contributory" were both considered "responsible" for this analysis.

[2]Chi-square tests compared substance group with the drugfree group.

[3]For the 25 drivers with THC and/or carboxy THC, the responsibility rate is 64.0%, which does not differ significantly from the drugfree rate.

[4]"Any other drug": These included barbiturates (6), antihistamines (6), antidepressants (3), narcotic analgesics (5), antidepressants (3), and miscellaneous others.

[5]For the 66 drivers with alcohol plus (THC and/or carboxy THC), the responsibility rate is 93.9%, which differs significantly from the drugfree rate.

[6]Non-alcoholic drug combinations included stimulants plus another (11 drivers), barbiturates plus another (8), and miscellaneous others.

the drugfree drivers. However, since alcohol alone exhibited a responsibility rate of 93.9 percent for BACs above .09%, the high responsibility rates of the drug-alcohol combinations do not by themselves support an inference of drug contributions to driver impairment.

The last drug group analyzed comprised the 25 drivers who had two or more nonalcoholic drugs in their systems. Their 84.0 percent responsibility rate was substantially higher than the 67.7 percent drugfree rate, but statistical significance was not reached. Here again, the results were suggestive but inconclusive.

A major limitation of the results in Table 5.14 is that there are no controls for potentially confounding variables. Attempts at control are made in the following sections.

Further examination of alcohol-drug combinations. Because the question of alcohol-drug additive or interactive (synergistic) effects is an important one, the analyses of Table 5.14 were extended by controlling for BAC. This was done by subdividing the alcohol-drug combinations into those with BACs below 0.10% and those at or above that level. In comparing each drug-alcohol group with its alcohol-only counterpart, no results were statistically significant (Table 5.15), hence an inference of additive or interactive effects was not supported. Among the low-BAC drivers, however, the elevated responsibility rates for those other than the cocaine-alcohol and amphetamine-alcohol drivers at least suggest the possibility of drug contributions. Among the high-BAC drivers, any drug contribution is harder to see because of the small difference among the responsibility rates.

Table 5.15 also compares the responsibility rates of the alcohol-drug groups with the drugfree drivers. Because of the low numbers in the low-BAC combinations, none of their responsibility rates differed significantly from the drugfree rates. When the low-BAC combinations were aggregated, however, their 83.8 percent responsibility rate reached marginal statistical significance. Among the high-BAC combinations, all the responsibility rates were significantly different from the drugfree rate, except for the small high-BAC amphetamine group.

Since the results for alcohol-drug combinations at least suggested the possibility of synergism, the analysis was carried a step further by controlling more precisely for BAC. To permit the generation of responsibility rates for several different BAC levels, the analysis combined the alcohol-drug groups which previously suggested synergism. They involved THC, amphetamines, and the "other" drugs. Alcohol-cocaine drivers were omitted because they had no indication of the drug adding to the alcohol effect. Figure 5.4 plots the responsibility rates of this group in relation to BAC. For comparison, the alcohol-only graph is also shown. The alcohol-drug responsibility rates are clearly higher than the alcohol-only rates throughout the BAC range. The greatest difference appears for BACs below 0.10%, where an otherwise slight

Table 5.15

Responsibility Rates of Alcohol-Drug Groups
Compared With Alcohol-Only and Drugfree Drivers

				Comparisons with Alcohol-Only Drivers		Comparisons with Drugfree Drivers	
		N (Drivers)	% Resp[1]	Chi-Square[2]	Statistical Significance	Chi-Square	Statistical Significance
A.	Low-BAC Groups: BAC < 0.10%						
(1)	Low-BAC only	120	75.8	--	--	2.9	P<.1
(2)	Low-BAC + cannabis[3]	8	87.5	0.1	N.S.	0.7	N.S.
(3)	Low-BAC + cocaine	8	50.0	1.4	N.S.	0.5	N.S.
(4)	Low-BAC + benzodiazepine	3	100.00	0.1	N.S.	<0.1	N.S.
(5)	Low-BAC + amphetamines	4	75.0	0.3	N.S.	<0.1	N.S.
(6)	Low-BAC + 1 other not above[4]	6	100.0	0.8	N.S.	1.6	N.S.
(7)	Low-BAC + any 2 or more drugs	8	100.0	1.3	N.S.	2.5	N.S.
(8)	Groups (2)-(7) combined	37	83.8	0.6	N.S.	3.5	P<.1
B.	High-BAC Groups: BAC ≥ 0.10%						
(1)	High-BAC only	625	93.9	--	--	144.7	P<.001
(2)	High-BAC + cannabis[3]	58	94.9	0.0	N.S.	16.6	P<.001
(3)	High-BAC + cocaine	41	95.1	0.0	N.S.	12.5	P<.001
(4)	High-BAC + benzodiazepines	14	100.0	0.1	N.S.	5.2	P<.02
(5)	High-BAC + amphetamines	8	100.0	0.0	N.S.	2.5	N.S.
(6)	High-BAC + 1 other not above[4]	13	100.0	0.1	N.S.	4.8	P<.05
(7)	High-BAC + any 2 or more drugs	38	94.7	0.0	N.S.	11.1	P<.001
(8)	Groups (2)-(7) combined	172	95.9	0.7	N.S.	55.4	P<.0001

[1]Drivers who were rated as "responsible/contributing" were both considered "responsible" for this analysis.

[2]The Chi-square tests compare Low-BAC+drug group with Low-BAC-only group, or High-BAC+drug group with High-BAC-only group.

[3]Cannabis includes tetrahydrocannabinol (THC) and/or its metabolite carboxy-THC.

[4]Other drugs included barbiturates and miscellaneous others.

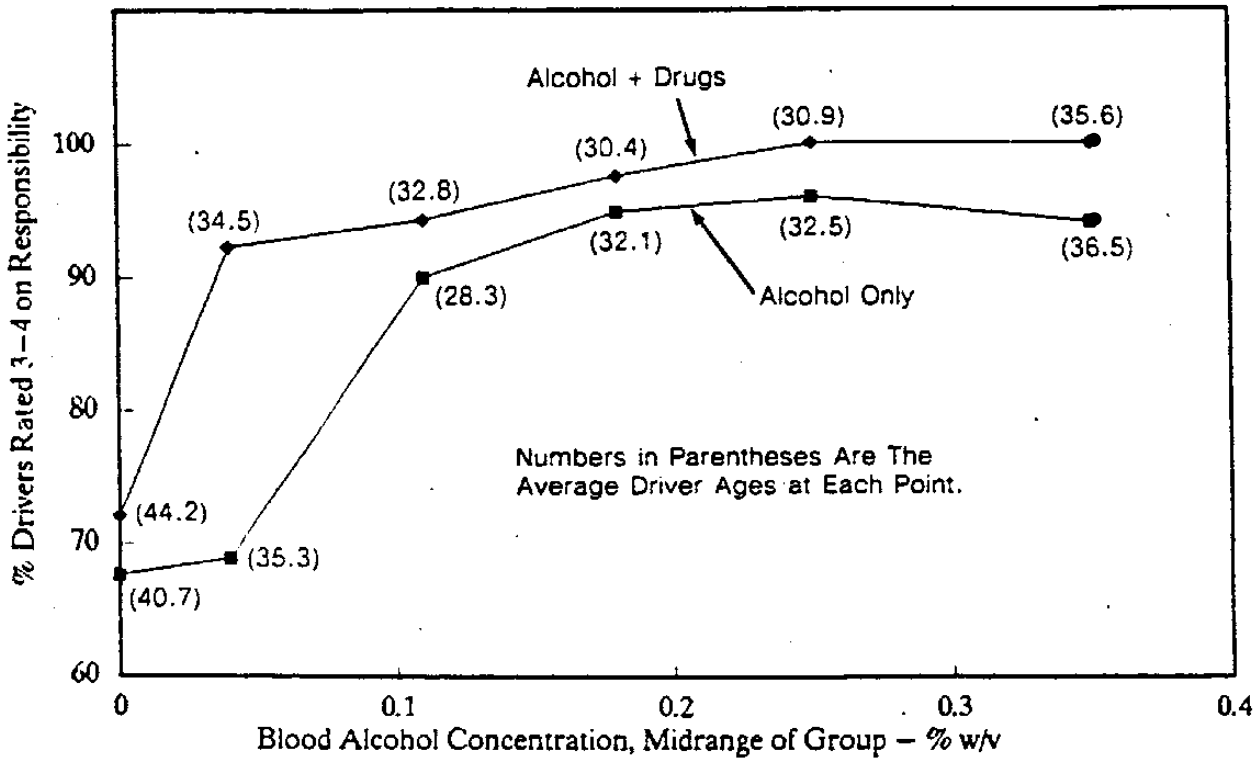


Figure 5.4 RESPONSIBILITY RATES, DRUGS, AND ALCOHOL: Alcohol-Only vs. Alcohol Plus THC, Amphetamines, or Other Noncocaine Drug.

Caution: These Results Are Exploratory and Require Confirmation With Larger Samples And Controls For Possible Confounding Variables Such as Age.

alcohol effect seems to be raised to the equivalent of an alcohol intoxication effect by the drugs. The results in Figure 5.4 must be considered provocative but inconclusive.

A limitation of the analysis in Figure 5.4 is that the necessity of combining data for different alcohol-drug combinations obscures the effects of any one. Another limitation is that it doesn't control for age or other variables. Figure 5.4 shows that the average driver age varied among the points in the graph. In the next section, however, we do impose controls in statistical tests of the alcohol and drug effects.

Controlling for other variables. Section 5.3 showed that the SUBSAMPL groups differed on many variables pertaining to the driver, the vehicle, and the crash environment. Any variable related to drug presence in crashes could be the underlying explanation for apparent drug impairment effects or their absence. This could happen if the variable is independently related to driver responsibility. To explore the possibilities, the responsibility relationships were examined for all variables in Tables 5.8 through 5.10, except for manner of collision. (The latter is an outcome variable, and is examined separately in a later section.) The relationships were examined for the drugfree drivers, in order to avoid the influence of alcohol and drugs. Instead of showing the detailed results of this preliminary examination, the results are summarized in Table 5.16. It shows that only crash time of day and driver age were related to both crash responsibility and to SUBSAMPL. These, then, are variables that need to be controlled in studying drug effects.

Crash time of day is related to driver responsibility as shown in Table 5.17. The data suggest that if a drugfree driver has an accident at night, he/she is less likely to be responsible than if he/she had the accident in daytime. (A possible explanation for this is that the drugfree drivers are more likely to be the victims of alcohol-impaired drivers at night.) The relation of driver age to responsibility is shown in Figure 5.5. It indicates that, without the effects of alcohol and other drugs, the effect of age on responsibility is quite strong. The drivers most likely to be responsible for crashes are the oldest and the youngest.

To examine the influence of alcohol and other drugs on responsibility while controlling for time of day and driver age, logistic regression analysis was used. However, including the individual drugs or classes was precluded because of their low numbers and the complexity it would have added. Consequently, the analysis used NDRUGS, a variable simply counting the number of nonalcoholic drugs in the driver's blood. Age was trichotomized into 15-24, 25-54, and 55-plus groups, while time of day was dichotomized into a simple day-night variable. The results are in Table 5.18. They confirm that crash responsibility is a function of BAC, the number of nonalcoholic drugs in the driver's blood, the driver's age, and the time of day. The probability of a driver being responsible increases with BAC and with the number of nonalcoholic drugs in her/his blood.

Table 5.16

Examination of Crash Circumstance Variables Potentially Confounding of Drug-Responsibility Relationships

Crash Circumstance	Related to Drug Involvement (SUBSAMPL)?	Related to Crash Responsibility?*	Conclusion: Potentially Confounding?
Ambient Conditions			
Day of week	Y(P<.0001)	N	
Time of day	Y(P<.0001)	Y(P<.05)	Yes
Season	N	N	
Land use	Y(P<.05)	N	
No. travel lanes	Y(P<.05)	N	
Horizontal alignment	Y(P<.0001)	N	
Surface condition	Y(P<.05)	N	
Atmosphere cond.	Y(P<.10)	N	
Vehicle Variables			
Vehicle type	Y(P<.0001)	N	
Number of occupants	N	Y(P<.01)	
Driver Variables			
Age	Y(P<.0001)	Y(P<.001)	Yes
Gender	Y(P<.0001)	N	
# Speeding violations	Y(P<.001)	N	
# Other violations	Y(P<.0001)	N	
Restraint system use	Y(P<.0001)	N	

*Relationships to responsibility were determined by examining drugfree drivers.

Table 5.17

Responsibility Rates by Time of Day
(Drugfree Drivers Only)

Time of Day	n	Responsible	Not Responsible	Total
12:01 A.M. - 06:00 A.M.	83	62.7%	37.3%	100.0%
06:01 A.M. - 12:00 Noon	247	71.7	28.3	100.0
12:01 P.M. - 06:00 P.M.	307	70.4	29.6	100.0
06:01 P.M. - 12:00 Midnight	162	59.3	40.7	100.0
Total	799	67.7%	32.3%	100.0%

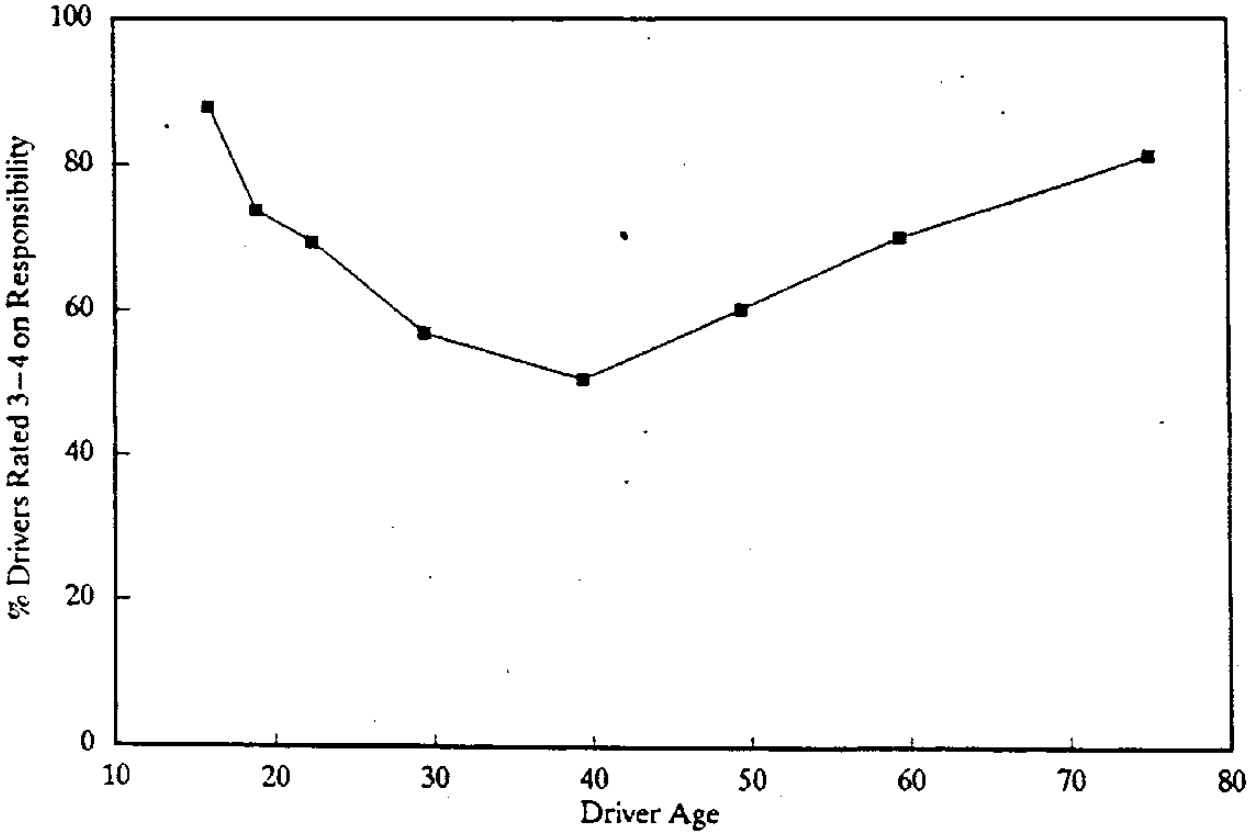


Figure 5.5 DRIVER AGE AND RESPONSIBILITY RATE:
All Drugfree Drivers (n=799)

Table 5.18

Equation for Predicting Crash Responsibility Resulting From Logistic Regression Analysis on Entire Sample (SPSS/PC Advanced Statistics 4.0)

Variables in the Equation

Variable	B	S.E.	Wald	df	Sig	R	Exp (B)
BAC	.1181	.0100	139.7951	1	.0000	.2719	1.1254
NDRUGS	.3684	.1373	7.2022	1	.0073	.0528	1.4454
ACCTIME	-.3224	.1372	5.5235	1	.0188	-.0435	.7244
DRAGEGRP			38.1242	2	.0000	.1353	
YOUNG	.1780	.1944	.8387	1	.3598	.0000	1.1948
MIDLAGE	-.6765	.1707	15.7011	1	.0001	-.0857	.5084
Constant	1.4403	.2190	43.2716	1	.0000		

BAC: = Blood alcohol concentration where 1 unit = .01%.
NDRUGS: = Number of nonalcoholic drugs in driver's blood.
ACCTIME: 1 = 6AM-6PM 2 = 6PM-6AM
YOUNG: 1 = Age 15-24 0 = Other age
MIDLAGE: 1 = Age 25-54 0 = Other age

Probability (Responsible) = 1.44 + 0.118 (BAC) + 0.368 (NDRUGS) - 0.322 (ACCTIME) + 0.178 (YOUNG) - 0.677 (MIDLAGE)

Exp (B) = Effect of a one-unit change in the predictor variable on the odds ratio:

$$\frac{\text{Prob (responsible)}}{\text{Prob (not responsible)}}$$

Classification Table for Responsibility

		Predicted: Resp = 0-2	Predicted: Resp = 3-4
		0	1
Observed			
Resp = 0-2	0	0	371
Resp = 3-4	1	0	1501
Percent Correct		--	80.2%

It should be noted that the logistic regression program was also run with an NDRUGS X BAC interaction term, but it was not found statistically significant. Thus, the results suggested an additive effect of drugs and alcohol, not an interactive or synergistic one. However, representing drugs by such a simple variable as NDRUGS cannot do justice to the effects of individual drugs, and the possibility of alcohol-drug interaction effects cannot be ruled out by the limited analysis.

From the size of their B exponents [Exp(B) in Table 5.18], we can estimate the contribution of the independent variables. Each additional drug in a driver's system seems to have an effect somewhat larger than a .01% increase in BAC. (This is a rough approximation, since the relation of responsibility to BAC was nonlinear; indeed, it was fairly flat above 0.10% BAC) Clearly, the BAC effect is much stronger than the NDRUG effect, but it should be recognized that our analysis necessarily dilutes the effects of impairing drugs by combining them with nonimpairing drugs.

The classification table at the bottom of Table 5.18 compares the observed responsibility ratings with the ratings predicted by the logistic model. It indicates a limitation of a driver fatality sample; since so many were found responsible for their crashes, the "best" prediction was to predict every driver responsible!

Summarizing, the logistic regression analysis provided greater statistical power for testing the role of drugs and controlling for other variables than was possible in the previous analyses focusing on specific drugs and drug combinations. However, the conclusions possible are limited to general inferences about drugs. The results indicate that the chances of a driver being responsible for his/her crash increased with BAC and the number of drugs ingested. Combining drugs with alcohol seems to raise chances of responsible crash involvement above that for alcohol alone.

Relative risk analysis. In a previous article (Terhune, 1983), it was shown that the relative crash risks of alcohol and drugs could be estimated from responsibility data by making the assumption that "nonresponsible" crash drivers comprise a representative sample of drivers on the road. The drug and alcohol proportions in that group, along with the comparable data for the "responsible" drivers, are used to calculate the relative crash risks. Using a sample of injured-but-surviving drivers, the method yielded a relative risk curve for BACs similar to that generated by other studies using the more elegant case-control method. As a suggestive exercise -- though no more than that -- the method was applied to the data of this study. Since this is a driver fatality study, the method estimates the relative crash risks of involvement in a fatal crash.

To calculate the relative crash risks, we advanced the derivation beyond that in the 1983 article. It gives the same result as the previous equation, but it simplifies the calculations. The new equation is:

$$\text{Estimated relative risk} = r_i\,(100-r_o)/[r_o\,(100-r_i)]$$

where r_i = responsibility rate of drug group i
and r_o = responsibility rate of drugfree group

The derivation of the equation is given in Appendix J.

Before considering the results, the reader should understand the following caveats:

(1) Nonresponsible drivers in fatal crashes may not be typical of drivers on the road at the times and places of all driver-fatality crashes.

(2) Small sample sizes for a drug group can produce very misleading results; the relative risk estimates are very sensitive to small changes in high responsibility rates e.g., 85 percent and above. That is, at high responsibility rates, small increases in a responsibility rate translate to a large increase in relative risk. This is effected by the $(100-r_i)$ term in the equation above.

In Table 5.19, we present the relative risk estimates only for the substance groups whose responsibility rates differed significantly from the drugfree rates. Note that for most alcohol-drug combinations, the normalized relative risks exceed that of alcohol-alone for BACs at or above 0.10%.

5.4.2 Collision Type Analysis

Originally planned for this study was an analysis to see if the drugs and drug groups varied in their associated collision types. If so, the collision types could suggest how drugs cause crashes. Unfortunately, the small numbers associated with the individual drugs and drug classes made this approach impractical. Alternatively, it is useful to examine a combination of collision types which may reflect driver impairment. The combination includes single-vehicle-crashes and head-on crashes in which the subject vehicle crossed the road centerline. Both crash types involve the subject vehicle departing its travel lane; indeed, many single-vehicle crashes involving road departure to the left might have been head-on crashes had there been an oncoming vehicle. Consequently, the two crash types were combined in an analysis of "key collision types."

Table 5.20 shows the proportions of the SUBSAMPL drivers who were involved in the key collision types. Note that only 47.9 percent of the drugfree drivers had such crashes, a result actually higher than the 31.6 percent for the THC-present drivers, and the 42.9 percent for the cocaine-present drivers. All the other substance groups were involved in the key collision types at rates

Table 5.19

Estimated Relative Risk of Fatal Crash in Major Substance Groups Whose Responsibility Rate Differed Significantly from the Drugfree Rate

CAUTION: DUE TO LIMITATIONS OF THE METHODS, THESE RESULTS SHOULD BE CONSIDERED SUGGESTIVE ONLY.

Substance Involvement	n	Risk Relative to Drugfree Group[a] Non-Normalized	Normalized[b]	Caveats
Drugfree	799	1.0	1.0	
Drivers With 1 Substance Only				
Alcohol: BAC < .10%	120	1.5	1.2	
Alcohol: BAC ≥ .10%	625	7.3	6.5	
Drivers With Alcohol-Drug Combination				
Alcohol + THC	37	8.4	11.9	
Alcohol + Carboxy THC	29	6.4	8.9	
Alcohol + Cocaine	49	3.4	5.3	
Alcohol + Benzodiazepines	17	Indef. large	Indef. large	Note c
Alcohol + Amphetamines	12	5.3	Indef. large	Note c
Alcohol + 1 other not above	19	Indef. large	Indef. large	Note c
Alcohol + 2 or more other	46	10.6	15.9	

Notes

[a] Estimated relative risk = $r_i (1-r_o)/[r_o (1-r_i)]$
where r_i = responsibility rate of drug group, r_o = resp. rate of drugfree group

[b] Normalization = Responsibility rate of drug group is adjusted by setting proportions on age equal to those for the drugfree group. In some cases normalization was limited only to those age groups where there were sufficient numbers of cases.

[c] Small subsample makes estimates especially tenuous.

Table 5.20

Involvement in Key Collision Configurations by the Sampled Drivers

Note: The groups below are mutually and exclusive and mutually exhaustive.

Substances Present	N (Drivers)	% Key Collisions[1]	Chi-Square[2]	Statistical Significance
Drugfree	803	47.9%	--	--
Drivers With 1 Substance Only				
Alcohol; BAC <.10	120	65.0	11.5	p<.001
Alcohol; BAC ≥.10	627	85.0	208.8	p<.001
THC (with or without carboxy)	19	31.6	1.4	N.S.
Carboxy-THC only	6	50.0	0.1	N.S.
Cocaine	7	42.9	0.01	N.S.
Benzodiazepines	18	66.7	1.8	N.S.
Amphetamines	12	66.7	1.0	N.S.
Any other drug[3]	34	67.6	4.3	p<.05
Drivers With Alcohol-Drug Combinations				
Alcohol + THC	37	78.4	11.9	p<.001
Alcohol + Carboxy THC	29	89.6	17.9	p<.001
Alcohol + Cocaine	49	79.6	17.3	p<.001
Alcohol + Benzodiazepines	18	83.3	7.5	p<.01
Alcohol + Amphetamines	13	69.2	1.6	N.S.
Alcohol + 1 other drug not above	19	78.9	6.0	p<.02
Alcohol + 2 or more other drugs	46	87.0	24.9	p<.001
Drivers With Non-Alcohol Combinations[4]	25	80.0	8.7	p<.01
Total drivers	1882			

[1]The "key collision configurations" are single-vehicle crashes and the vehicle crossing the centerline in head-on crashes. The 2 types are combined in the data above. Example: 47.9% of the drugfree drivers were in a single-vehicle crash or were driving the vehicle that crossed the centerline in a head-on crash.

[2]Chi-square tests compared substance group with the drugfree group.

[3]"Any other drug": These included barbiturates (6), antihistimines (6), narcotic analgesics (5), antidepressants (3), and miscellaneous others.

[4]Non-alcoholic drug combinations included stimulants plus another drug (11 drivers), barbiturates plus another drug (8), and miscellaneous others.

substantially higher than the drugfree rate, although the amphetamine-only and benzodiazepine-only rates did not reach statistical significance. The drivers with non-alcohol drug combinations also had a very high rate, suggesting an effect similar in strength to alcohol intoxication.

In summary, the key collision analysis is a method which may be more sensitive than responsibility analysis, and it bears consideration in future studies. Substantively, it suggests no impairment effects when cannabis or cocaine are present alone, but other drugs and drug combinations may be contributing to fatal crashes. Alcohol again exhibited the predominant impairment effect.

5.4.3 FARS Driver Causal Factors

The FARS database includes a variable that lists any driver factors among the crash causes, as judged by the police and/or the FARS analysts. We have no information on the reliability or validity of those judgments, and it is possible that they contain coding bias due to knowledge of the driver's age, alcohol ingestion, and other factors. As long as these limitations are kept in mind, the FARS factors can suggest how drugs effect crashes.

Driver errors may be coded in FARS as a first, second, or third causal factor. For this analysis, only mentions as a first causal factor were analyzed. Under the heading "Any Error" in Table 5.21, highly significant differences among the SUBSAMPL groups are shown. The substance groups with an error rate much higher than the 69.4 percent rate of the drugfree group were the amphetamine-only group, all the alcohol groups, and the non-alcohol combinations.

Of the specific kinds of error, only speeding and lane-maintenance errors were frequent enough to permit meaningful comparisons among the driver groups. Differences among the groups were significant for both variables. Considering speeding first, note that 14.4 percent of the drugfree drivers had been speeding. Similar speeding rates were found in each of the non-alcohol drug groups, except for the high 42.9 percent rate in the small cocaine-only group. In contrast, every driver group involving alcohol had a speeding rate much higher than the drugfree rate. In the two alcohol-only groups, the speeding rates increased with BAC. These results suggest that speeding is primarily an alcohol effect.

Note that the speeding rate for drivers with alcohol combined with carboxy-THC is much higher than the rate for drivers with an alcohol-THC combination. The high speeding rate of the alcohol-carboxy group may reflect in part a tendency of frequent cannabis users.

Table 5.21

FARS Driver Causal Factors in Relation to the Substance Groups

Substance Involvement	N (Drivers)	None	Any Error	Speeding	Lane Maintenance	Inattentive	Right of Way Error	Other	Total
Drugfree	772	30.6%	69.4%	14.4%	15.5%	5.8%	9.3%	24.4%	100.0%
Drivers With 1 Substance Only									
Alcohol only; BAC <.10	116	18.1	81.9	25.0	25.9	6.0	0.9	24.1	100.0
Alcohol only; BAC ≥.10	614	9.3	90.7	36.6	25.4	5.9	2.1	20.7	100.0
THC (with or without carboxy)	18	44.4	55.6	11.1	11.1	11.1	0.0	22.2	100.0
Carboxy THC only	6	50.0	50.0	16.7	16.7	0.0	0.0	16.7	100.0
Cocaine only	7	42.9	57.1	42.9	0.0	0.0	0.0	14.3	100.0
Benzodiazepines only	18	33.3	66.7	11.1	33.3	0.0	0.0	22.2	100.0
Amphetamines only	12	8.3	91.7	16.7	16.7	16.7	16.7	25.0	100.0
Any other drug	33	24.2	75.8	18.2	21.2	12.1	6.1	18.2	100.0
Drivers With Alcohol-Drug Combination									
Alcohol + THC	37	8.1	91.9	29.7	21.6	8.1	0.0	32.4	100.0
Alcohol + Carboxy-THC	28	10.7	89.3	42.9	21.4	10.7	0.0	14.3	100.0
Alcohol + Cocaine	48	12.5	87.5	41.7	22.9	4.2	0.0	18.8	100.0
Alcohol + Amphetamines	13	0.0	100.0	30.8	30.8	7.7	0.0	30.8	100.0
Alcohol + Benzodiazepines	17	17.6	82.4	35.3	11.8	0.0	5.9	29.4	100.0
Alcohol + 1 other drug	19	10.5	89.5	31.6	21.1	0.0	5.3	31.6	100.0
Alcohol + 2+ other drugs	46	13.0	87.0	32.6	26.1	4.3	2.2	21.7	100.0
Drivers With Non-Alcohol Combinations	24	8.3	91.7	16.7	29.2	8.3	8.3	29.2	100.0
Missing	54								
Chi-square	-	-	127.2	110.4	30.0	-	57.2	-	-
Significance	-	-	P<.0001	P<.0001	P<.01	-	P<.0001	-	-

Lane-maintenance error rates did not differ as dramatically among the substance groups as the speeding rates. The drivers with distinctly the highest of these error rates were those with benzodiazepines only, with alcohol combined with amphetamines, and non-alcohol drug combinations. Whereas speeding seems to have been clearly associated with alcohol, the lane-maintenance results suggest that other drugs may impair a driver's ability to remain in her/his driving lane.

6.0 TIME FACTORS IN DRUG DETECTION

In Chapter 3 we noted that in specifying the sampling criteria of acceptable driver survival time and death-to-specimen time, trade-offs were necessary in balancing methodological ideals against practical considerations at the sampling sites. A useful methodological contribution of this study is provided by taking a retrospective look at these time factors, to see if they had an effect on drug detection.

6.1 Survival Time and Drug Detection

Survival time is operationally defined as the elapsed time between the police-reported time of a crash and the coroner/M.E. reported time of the driver's death. These may be estimates, as when the fatal crash is discovered hours after its occurrence. Studies on drugs in crashes usually are limited to drivers who died within an hour or two after the crash, in order to maximize the chances of finding drugs that were in the driver's blood when the crash occurred. Since this study accepted drivers who survived up to four hours, it is important to see whether the survival time has much effect on drug prevalence rates.

Figure 6.1 shows the overall prevalence of alcohol and other drugs within (a) drivers reported to have died in their crash, (b) drivers who died within one hour, and (c) drivers who died within four hours. (Each of these groups successively includes the previous group.) The groups do not appear to differ much in their results, suggesting that we would have drawn similar inferences of the overall magnitude of drug prevalence regardless of which cutoff between 0 and 4 hours was used in sample selection. Only a slight increase in the "drugfree" drivers may be observed in going from a survival time of zero (died in crash) to survival up to four hours; this suggests that occasionally drugs were lost to detection with the longer survival times. A reason why the survival time would have only a minor effect on the overall prevalence rates is that only 6.3 percent of the sampled drivers survived beyond two hours (Figure 6.2).

A more specific picture of the effect of survival time on drug detection is provided by Table 6.1. It shows that survival time was related mainly to alcohol detection, whereby the detection of intoxication-level BACs was lowest in the fourth hour. Results for the other drugs were encouraging, however, for only the barbiturates exhibited an effect of survival time. Even with those, the deviating value is limited to the third survival hour, and it could have been spurious.

Figure 6.3 provides another view on the alcohol relation to survival time. It may indicate that for drivers surviving beyond two hours, there are substantially increased chances of failure to detect alcohol that was present during the crash. However, the data may also reflect the fact that high-BAC drivers are more

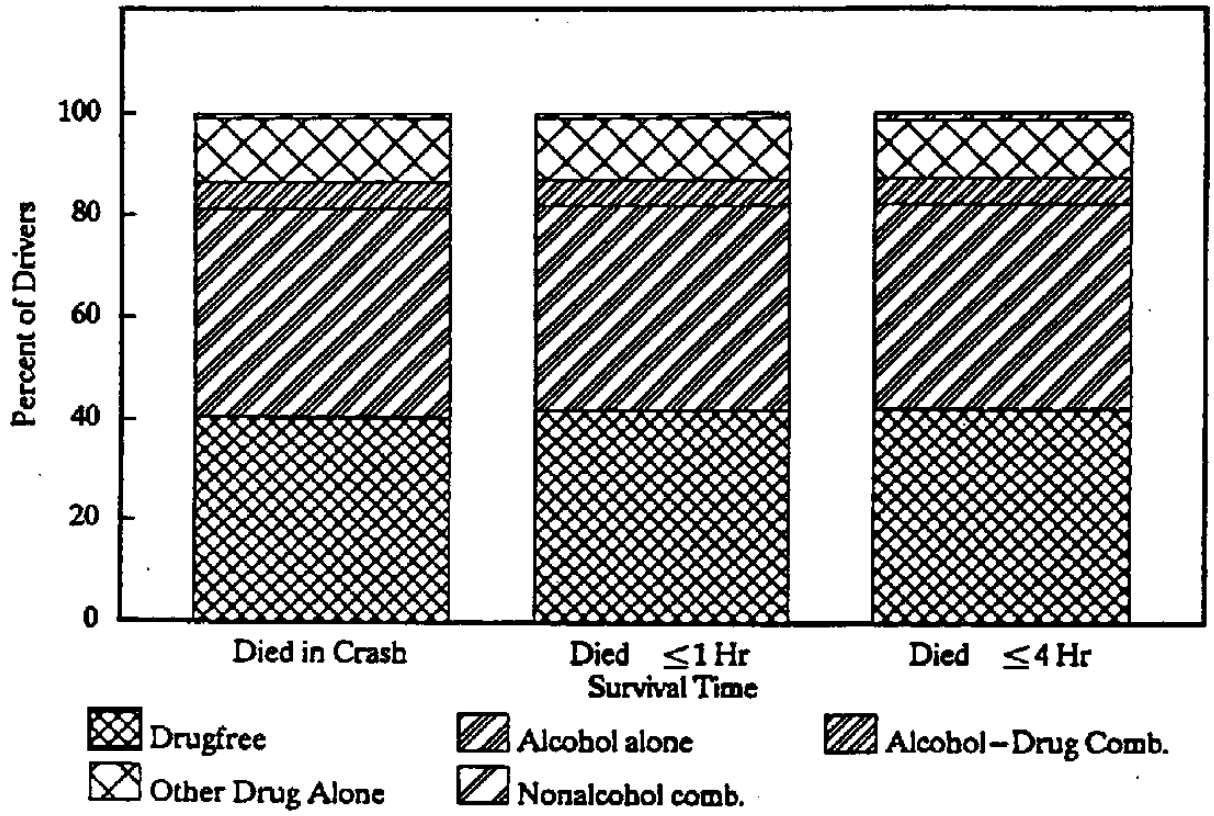


Figure 6.1 BLOOD COMPOSITION IN RELATION TO SURVIVAL TIME
(Weighted Data)

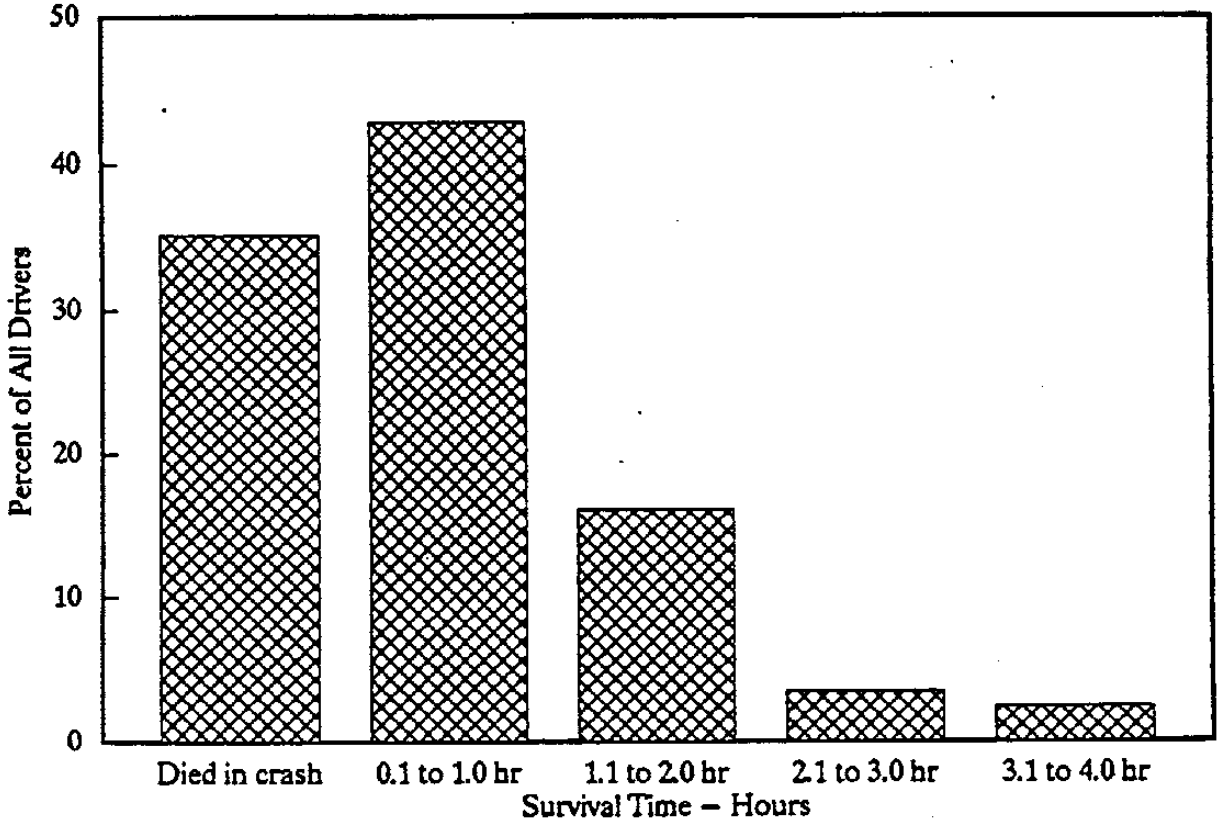


Figure 6.2 DISTRIBUTION OF DRIVER SURVIVAL TIMES

Table 6.1

Drug Class Prevalence Rates in Relation to Time Interval Between Crash and Driver's Death*

Drug Class*	Died in Crash	Time of Death After Crash: 1st Hour	2nd Hour	3rd Hour	4th Hour	Chi-Square	Statistical Significance**
Alcohol: BAC <.10	8.0%	7.7%	9.6%	9.2%	14.9%	3.8	P<.06
Alcohol: BAC >.10	45.1	42.1	44.9	26.2	23.4	16.7	P<.003
Cannabinoids	7.0	8.1	4.3	6.2	2.1	6.7	N.S
Cocaine	4.4	4.7	4.6	0.0	6.4	3.6	N.S.
Amphetamines	2.7	1.5	2.3	1.5	0.0	4.0	N.S.
Benzodiazepines	2.9	3.7	3.3	3.1	4.3	0.9	N.S.
Barbiturates	0.9	1.6	1.7	6.2	2.1	11.0	P<.05
Narcotic Analgesics	0.9	0.9	1.0	1.5	2.1	1.0	N.S.
Antidepressants	1.1	0.9	0.3	0.0	0.0	2.4	N.S.
Antihistamines	0.8	0.5	0.3	1.5	2.1	3.3	N.S.
Hallucinogens	0.2	0.4	0.0	0.0	0.0	2.0	N.S.
Antiarrythmics	0.0	0.0	0.3	0.0	0.0	5.2	N.S.
Muscle Relaxants	0.0	0.1	0.0	0.0	0.0	1.3	N.S.
Nonbarbit. Sedatives	0.0	0.0	0.0	0.0	0.0	--	N.S.
Antipsychotics	0.0	0.0	0.0	0.0	0.0	--	N.S.
No. of Drivers	666	806	303	65	47		

*All percentages in a column are based on the total number of drivers who died within the time interval at the top of the column. For example, of the 666 drivers who died in the crash, 7.0% had cannabinoids in their blood. Note that the percentages are non-additive, for more than one substance may be found in a blood specimen.

**Drug classes include metabolites as well as parent drugs. Specific drugs included in the classes are:
Cannabinoids: Tetrahydrocannabinol, Carboxy THC
Benzodiazepines: Diazepam, Nordiazepam, Lorazepam, Flurazepam, Desethylflurazepam, Alprazolam, Oxazepam, Chlordiazepoxide, Desmethylchlordiazepoxide
Barbiturates: Phenobarbital, Secobarbital, Butabarbital, Butalbital, Pentobarbital, Amobarbital
Narcotic Analgesics: Meperidine, Methadone, Propoxyphene, Norpropoxyphene, Oxycodone, Codeine, Morphine, Heroin
Antidepressants: Amitriptyline, Nortriptyline, Imipramine, Desipramine, Doxepin, Desmethyldoxepin, Fluoxetine, Norfluoxetine
Antihistamines: Diphenhydramine, Chlorpheniramine
Hallucinogens: Phencyclidine, LSD
Antiarrhythmics: Quinidine, Procainamide, N-Acetylprocainamide, Lidocaine, Flecainide
Muscle Relaxants: Cyclobenzaprine
Nonbarbiturate Sedatives: Ethchlorvynol, Methaqualone, Meprobamate
Antipsychotics: Chlorpromazine, Thioridazine, Mesordiazine

***Statistical significance is the result of chi-square analysis comparing incidence rates across the time intervals.

Table 6.3

Drug Class Prevalence Rates in Relation to Time Interval Between Death and Drawing of Blood Specimen*
--For drivers who died within 2 hrs. of crash--

Drug Class**	Time Between Death and Drawing of Blood Specimen					Chi-Square	Statistical Significance***
	LE 8 hr.	8.1-12.0 hr.	12.1-24.0 hr.	24.1-48.0 hr.	48.1-96 hr.		
Alcohol: BAC <.10	7.1%	6.3%	8.2%	8.1%	13.7%	5.7	N.S.
Alcohol: BAC >.10	39.7	71.2	36.2	43.4	40.0	71.8	P<.001
Cannabinoids	5.6	10.5	9.8	6.6	7.4	8.5	P<.08
Cocaine	4.5	3.6	4.0	7.9	2.5	8.2	P<.1
Amphetamines	0.5	1.8	2.2	4.1	7.4	30.3	P<.0001
Benzodiazepines	2.6	4.7	5.3	1.0	2.1	10.2	P<.04
Barbiturates	1.3	1.6	1.9	0.5	0.0	3.2	N.S.
Narcotic Analgesics	0.8	0.5	1.3	0.5	1.1	1.5	N.S.
Antidepressants	0.8	1.0	1.1	1.5	0.0	1.7	N.S.
Antihistamines	0.7	0.0	0.5	0.5	2.1	4.7	N.S.
Hallucinogens	0.3	0.0	0.0	0.5	1.1	4.1	N.S.
Antiarrythmics	0.0	0.0	0.0	0.0	0.0	--	N.S.
Muscle Relaxants	0.2	0.0	0.0	0.0	0.0	1.4	N.S.
Nonbarbit. Sedatives	0.0	0.0	0.0	0.0	0.0	--	N.S.
Antipsychotics	0.0	0.0	0.0	0.0	0.0	--	N.S.
No. of Drivers	605	191	378	198	95		

*All percentages in a column are based on the total number of drivers whose blood specimen was drawn within the time interval at the top of the column. For example, of the 605 drivers whose blood was drawn within 8 hours after death, 5.6% had cannabinoids in their blood.
Note that the percentages are non-additive, for more than one substance may be found in a blood specimen.

**Drug classes include metabolites as well as parent drugs. Specific drugs included in the classes are:
Cannabinoids: Tetrahydrocannabinol, Carboxy THC
Benzodiazepines: Diazepam, Nordiazepam, Lorazepam, Flurazepam, Desethylflurazepam, Alprazolam, Oxazepam, Chlordiazepoxide, Desmethylchlordiazepoxide
Barbiturates: Phenobarbital, Secobarbital, Butabarbital, Butalbital, Pentobarbital, Amobarbital
Narcotic Analgesics: Meperidine, Methadone, Propoxyphene, Norpropoxyphene, Oxycodone, Codeine, Morphine, Heroin
Antidepressants: Amitriptyline, Nortriptyline, Imipramine, Desipramine, Doxepin, Desmethyldoxepin, Fluoxetine, Norfluoxetine
Antihistamines: Diphenhydramine, Chlorpheniramine
Hallucinogens: Phencyclidine, LSD
Antiarrhythmics: Quinidine, Procainamide, N-Acetylprocainamide, Lidocaine, Flecainide
Muscle Relaxants: Cyclobenzaprine
Nonbarbiturate Sedatives: Ethchlorvynol, Methaqualone, Meprobamate
Antipsychotics: Chlorpromazine, Thioridazine, Mesordiazine

***Statistical significance is the result of chi-square analysis comparing incidence rates across the time intervals.

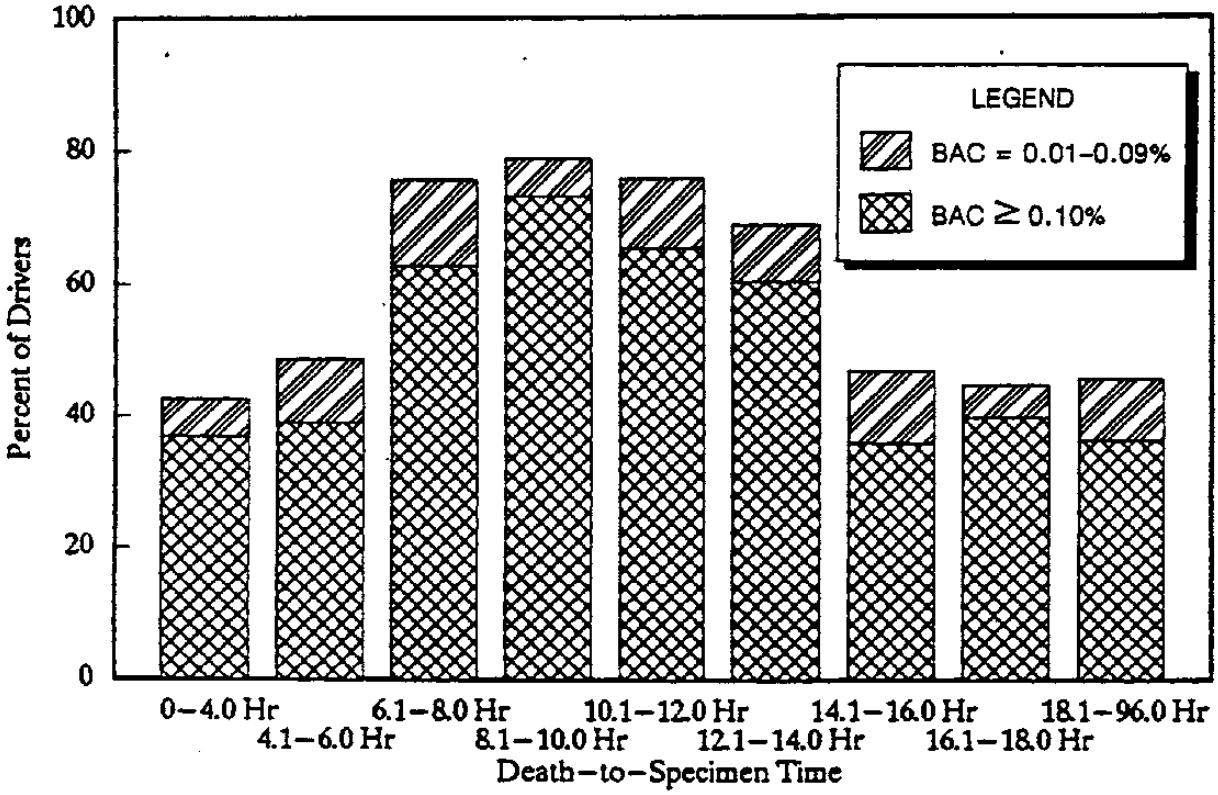


Figure 6.5 ALCOHOL PREVALENCE VS. DEATH-TO-SPECIMEN TIME: Drivers Who Died Within 2 Hr.

in Figure 6.6. Among the four drug groups, the amphetamines most clearly suggest a systematic relation to elapsed time. The amphetamine rates increased with time, which could result from amphetamines stored in tissue migrating into the bloodstream. This may be a characteristic of those who were longtime users of amphetamines.

The relationships in Table 6.3 and Figures 6.4 and 6.5 led us to inquire further for an explanation. We learned that postmortem change of drug concentrations in blood specimens is a phenomenon known as "postmortem redistribution of drugs" (Anderson and Prouty, 1989). While the alcohol and amphetamine relationships to death-specimen time may be examples of this phenomenon, the relationships for the other drugs may not be. All the relationships will be influenced by variations among the sites in the rapidity with which they were able to extract the specimens. Table 6.4 shows that there were highly significant differences among the sites on that dimension. These, combined with site differences in drug prevalence rates, may have effected some of the relationships in Figure 6.6. A check was made to see if those relationships were maintained within individual sites, but the lower numbers and low prevalence rates produced only erratic patterns. Consequently, the reliability of the patterns in Figure 6.6 could not be established.

6.3 Another Look at the Prevalence Rates

The analyses above suggest that the reliability of alcohol and drug prevalence rates in epidemiological studies of alcohol and drugs in driver fatalities will be better assured by limiting the acceptable survival time to two hours for the sample, and by obtaining blood specimens no more than six hours after death. To approximate the effects of such limits, Figure 6.7 shows the alcohol and drug results for the 629 drivers who died within two hours and whose blood was taken within six hours. The effect is to reduce the alcohol and drug prevalence rates somewhat; the drugfree rate increased by 8 percent over its level in the entire sample (Figure 5.1). It must be understood, however, that Figure 6.7 cannot represent the results that would be obtained had all the specimens been obtained within the specified time limits; selecting the data that way biased the sample toward sites like North Carolina and Wisconsin, which collected much of their data shortly after the drivers died.

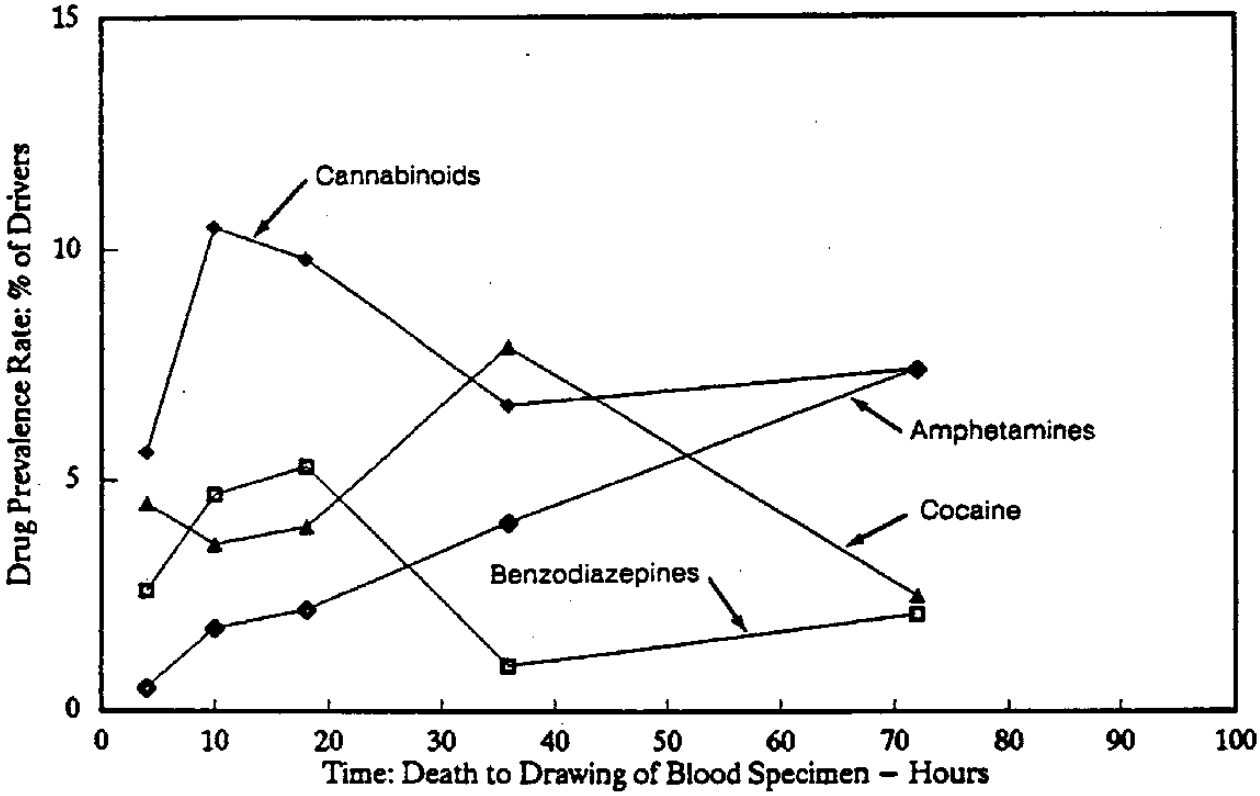


Figure 6.6 DRUG PREVALENCE RATES & DEATH-TO-SPECIMEN TIME INTERVAL:
Drivers Dying Within 2 Hr of Crash

Table 6.4

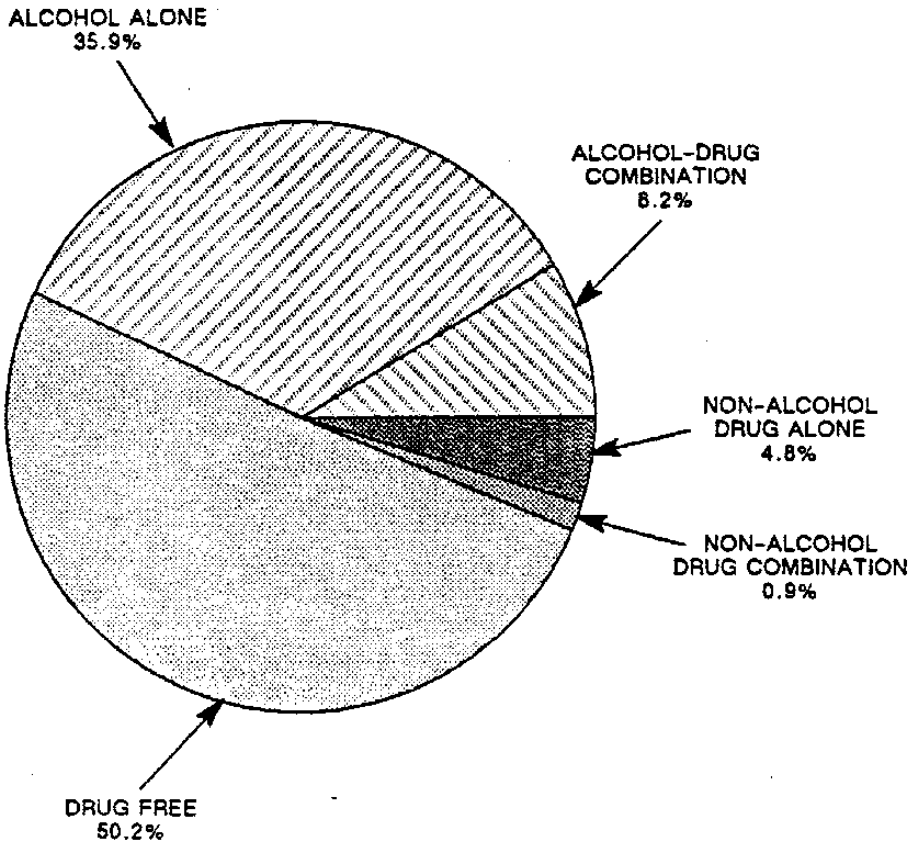
Site Comparison
Time Interval Between Death and Drawing of Blood Specimen

	California					Massachusetts	N. Carolina	Virginia	Wisconsin		Texas	
Time Interval	Alameda	Solano	San-Bernadino	San-Diego	Los Angeles			No. District	Milwaukee	Else	Tarrant	Dallas
LE 1 hr.	2.5%	0.0%	0.0%	0.0%	1.0%	1.7%	26.6%	1.2%	0.0%	17.7%	0.0%	0.9%
1.1-2.0 hr.	0.0	0.0	0.0	0.0	0.0	0.0	15.7	0.0	0.0	31.0	0.9	0.0
2.1-3.0 hr.	0.0	0.0	0.0	0.0	0.0	1.7	8.3	0.0	2.8	17.7	0.9	0.9
3.1-4.0 hr.	0.0	0.0	0.0	0.0	0.0	1.7	5.1	1.2	0.0	7.8	2.8	2.6
4.1-8.0 hr.	17.5	4.0	0.7	4.1	0.0	13.9	11.7	3.6	16.7	9.6	11.3	22.6
8.1-12.0 hr.	20.0	24.0	3.7	20.0	3.1	20.8	10.8	6.0	16.7	5.2	29.2	19.1
12.1-24.0 hr.	52.5	48.0	6.7	53.1	10.4	40.5	18.5	28.9	47.2	9.6	49.1	48.7
24.1-48.0 hr.	5.0	24.0	31.3	22.8	54.2	17.3	2.8	43.4	13.9	1.4	5.7	4.3
48.1-72.0 hr.	2.5	0.0	26.1	0.0	19.8	1.7	0.4	15.7	2.8	0.0	0.0	0.9
72.1-96.0 hr.	0.0	0.0	31.3	0.0	11.5	0.6	0.2	0.0	0.0	0.0	0.0	0.0
Total %	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Total Drivers	40	25	134	145	96	173	530	83	36	345	106	115
Mean Time (Hours)	14.9	18.9	55.2	18.5	42.1	17.3	6:9	30.0	17.6	4.4	13.6	13.1

Chi-Square of site differences*: $X2 = 1916.79$ $p<.0001$

ANOVA test of difference among means: $F12,1869 = 239.01$ $p<.001$

*To meet the expected frequency requirements for chi-square analysis, the time intervals 2.1-3.0 and 3.1-4.0 were combined. Also, the Alameda and Solano sites were combined, as were the Wisconsin sites.



DRUG or DRUG CLASS	PREVALENCE RATE
ALCOHOL: BAC $\geq$ .10%	37.5%
ALCOHOL: BAC < .10%	6.6
CANNABIS	5.3
COCAINE	3.2
BENZODIAZEPINE TRANQUILIZERS	2.6
AMPHETAMINES	0.3
BARBITURATE SEDATIVES	1.3
NARCOTIC ANALGESICS	0.9
ANTIDEPRESSANTS	0.8
ANTIHISTAMINES	0.4
HALLUCINOGENS	0.2
ANTIARRHYTHMICS	0.2
MUSCLE RELAXANTS	< 0.1
ANTIPSYCHOTICS	0.0
NONBARBITURATE SEDATIVES	0.0

Figure 6.7 DRUG PREVALENCE IN 629 DRIVERS WHO DIED WITHIN 2.0 HOURS AND WHOSE BLOOD SPECIMEN WAS TAKEN WITHIN 6.0 HOURS AFTER DEATH. (WEIGHTED FOR SAMPLE BIAS)

7.0 DISCUSSION AND CONCLUSIONS

Most of this chapter is devoted to discussion and conclusions on substantive matters -- the prevalence and role of drugs in driver fatalities. After that, methodological considerations for future research are discussed.

7.1 The Prevalence and Role of Drugs in Driver Fatalities

The main objectives of this study were to learn the extent to which drugs are found in fatal crashes, and to determine the causal role of drugs in those crashes. An additional objective was to learn about the circumstances in which drugs were detected. The results concerning these objectives were presented in Chapter 5, and here we draw our conclusions from those results. In so doing, we take into account the methodological limitations of the study.

7.1.1 Alcohol: Still a Dominant Problem

Although drugs other than alcohol were the basic focus of this study, the dominance of alcohol in the fatal crashes was inescapable. Fully 40 percent of the drivers had only alcohol in their systems, and another 11 percent had alcohol combined with drugs. No single drug, nor all the other drugs combined, approached the prevalence of alcohol. And, of all the drivers with alcohol in their blood, 83 percent had BACs over 0.10%, and 63 percent were at or above .15%, well into the intoxication range.

To be sure, there are reasons to believe these figures are inexact as estimates of the total involvement of alcohol. Our sample included drivers surviving as long as four hours after their crashes, and alcohol prevalence may be underestimated among those surviving the longer periods. On the other hand, the data in Chapter 6 suggested that overestimation of blood alcohol presence could have resulted when the specimens were taken more than a few hours after death. These phenomena offset each other to some extent, and the obtained rate of alcohol prevalence may not be far from the actual one.

Alcohol is also ubiquitous. Unlike some drugs whose popularity seems limited to certain regions, alcohol involvement was high at every sampling site. Its prevalence ranged from 40 percent in the fatalities from California's San Bernadino County to 69 percent in the Fort Worth area of Texas. It was about as prevalent in rural crashes as in urban ones.

Yet it is not its prevalence alone that makes alcohol a great highway safety problem. Judging by its effects on drivers, it is one of the most impairing of drugs. Among the 625 drivers who had BACs at or above 0.10%, the responsibility rate was an extraordinary 94%, well above that found for any other single substance. Alcohol presence was also associated with curve crashes and with speeding.

Finally, the study provided evidence suggesting that even at BACs below 0.10%, alcohol combined with some drugs may have an impairing effect equivalent to alcohol intoxication. This is a finding needing clarification in future research, but it does denote another dimension of the alcohol problem.

Certainly, there is nothing new in acknowledging that alcohol is a most serious highway safety problem, a fact which the public has long recognized. This study adds emphasis, however, by showing alcohol's predominance when compared with other drugs.

7.1.2 Drugs Other Than Alcohol: A Limited Problem

In overview, the following were learned about drugs in fatal crashes at the locations sampled:

(1) Drugs were significantly less prominent than alcohol in the fatal crashes. Altogether, 18 percent of the drivers had one or more of the tested drugs in his or her system, and only 6 percent had drugs without alcohol. We found that the overall prevalence rate for drugs other than alcohol was about 5 percent higher in urban than in rural areas.

(2) The drugs most prominent were cannabis (7 percent of the drivers), cocaine (5 percent), benzodiazepine tranquilizers (3 percent), and amphetamines (2 percent). Regional variations were apparent, with amphetamines being limited mainly to California counties.

(3) Evidence of causal contributions of the drugs to the crashes was very limited. The analyses were handicapped by the small numbers of drivers with specific drugs, which limited our capability to control for key variables such as driver age. In the absence of alcohol, no drug or drug group evidenced a driver responsibility rate significantly different from the drugfree control group. When drugs were combined with alcohol, no drug or drug group exhibited a responsibility rate significantly different from alcohol by itself. However, responsibility rates increased with the simple number of drugs in a driver's system, a statistically significant result similar to that found among young males by Williams and colleagues (1985). We also found that when controlling for specific BAC levels, the aggregated alcohol-drug combinations (excluding cocaine) had consistently higher responsibility rates than the drivers with alcohol alone, an effect most pronounced at BAC levels below 0.10%. This last finding, however,

needs confirmation with samples permitting controls for confounding variables like driver age.

(4) Alcohol tended to be combined with certain drugs. It was present in 4 out of 5 drivers with cocaine in their systems, and in 2 out of 3 drivers with cannabis, antidepressants, or benzodiazepines in their blood.

The most prevalent drugs deserve additional commentary, as follows.

Cannabis. While cannabinoids were detected in 7 percent of the drivers, the psychoactive agent THC was found in only 4 percent. Although cannabinoids were found in few drivers over 55 years old, cannabis was not entirely a drug of youth. A majority of those with THC in their blood (indicating recent ingestion) were in the 25-54 age range. Among those with only carboxy-THC in their blood (indicating less recent ingestion), most of these drivers were in the 15-24 age range, and a majority were motorcyclists. Both cannabinoids involved substantially more male than female drivers.

The THC-only drivers had a responsibility rate below that of the drugfree drivers, as was found previously by Williams and colleagues (1985). While the difference was not statistically significant, there was no indication that cannabis by itself was a cause of fatal crashes. However, the responsibility rate for the alcohol-plus-THC combination was 95%, and the normalized relative risk for the combination was higher than alcohol by itself in the intoxication range. Again, small numbers of cases and lack of statistical significance justify only the conclusion that the possibility of a cannabis-alcohol additive effect is suggested by the data and it merits further research.

For the six drivers with only carboxy-THC in their blood, the responsibility rate was 83%. While higher than the drugfree rate, the difference was not statistically significant. (Further commentary on these drivers is given in Section 7.1.3.)

Cocaine. By itself, cocaine was found in only 7 drivers. Such a small number hardly justifies a responsibility rate, but it may be noted that it was about the same as the THC-only rate and not significantly different from the drugfree rate. Like cannabis, the CNS-stimulant driver groups involved males much more than females.

Cocaine was most frequently combined with alcohol. Like all other drugs, the combination did not yield a responsibility rate significantly different from alcohol alone. Unlike THC, the results did not suggest the possibility that cocaine adds to the impairment of alcohol. Again, however, no firm conclusion is justified because of the statistical limitations.

Amphetamines. Amphetamines alone were present in only 12 drivers, but their 83 percent responsibility rate was well above the 68 percent rate of the drugfree drivers. Again, results were not statistically significant, so the only conclusion justified is that the data suggest the possibility of an impairment effect.

Alcohol was combined with amphetamines less frequently than with cocaine. There were suggestions in the results, e.g. with the normalized relative risk data, that amphetamines may add to the impairment of alcohol. Once again, statistical limitations justify no firm conclusions.

Medicinal (prescription) drugs. These substances were detected infrequently in the sampled drivers. The largest medicinal group was the benzodiazepines, found in only 3 percent of the drivers. Most of the benzodiazepines were diazepam (Valium (R)). The medicinal drugs were generally found in older drivers, about half being 55 and older. Unlike the other groups, the benzodiazepine drivers included mainly women.

The responsibility analysis for these groups gave little indication of impairment effects when these drugs were present alone. The responsibility rate of the benzodiazepine-only group was virtually identical to the drugfree rate. A major caveat is necessary here, however: the small numbers for any particular drug or class prohibited analyzing them separately. Even smaller numbers would be involved by concentrating on the elderly drivers, a group found to experience elevated crash risks from medicinal drugs (Ray et al., 1992). Those relative risks were mostly in the range of 1.1 to 2.2, which correspond to responsibility rates around 69-81 percent, assuming a drugfree rate similar to our sample. For the drug rates to be statistically significant with individual drugs in an elderly subsample, a sample size much larger than in this study would be needed.

7.1.3 Effects of Drugs or Effects of Users?

A nagging question which qualifies conclusions from epidemiological studies of drugs in crashes is: If certain drugs are linked to elevated crash risks, how much of the elevation is due to characteristics of the people who use those drugs? In Section 5.3.1 we reported that the driver substance-group categories were significantly related to every driver variable examined: age, gender, number of speeding violations, number of other traffic violations, and restraint system use. We had no data on personality characteristics, but there may be personal attributes common to some user groups that can increase their crash risks. For example, Terhune's (1986) review of research revealed a striking similarity between the personal correlates of marijuana use and the correlates of crash involvement. Rebellious, deviant, youthful males were prominent among marijuana users and among those in crashes.

Unfortunately, the data of this study include nothing on the personalities or psychosocial history of the drivers. There were, however, clues suggesting that something besides drug impairment contributes to the crashes of drug-present drivers. The clues were:

- Drivers with carboxy-THC or amphetamines in their blood had high responsibility rates, but the active form of the drug may not have been in the driver's blood during the crash.

- Drivers with previous traffic violations, especially speeding, were overrepresented in most of the drug groups. While drug use could have caused that behavior, the drug use may simply have been part of a broader behavior pattern.

Consider the drivers in whom only carboxy-THC was detected. The carboxy showed that they had ingested cannabis at some time previous to their accident, but the psychoactive THC was no longer in their blood. Nevertheless, this group had an 83 percent responsibility rate, well above the drugfree rate. It is possible that this reflects cannabis impairment, but then we would expect the THC-only drivers to have a responsibility rate much higher than the 58 percent that was found. While it is true that impairment has been found up to 24 hours after cannabis ingestion (Leirer, Yesavage, and Morrow, 1991), it is also true that carboxy-THC may be found in plasma several days after marijuana smoking (Barnett and Willette, 1989). It is noteworthy that the carboxy-THC group comprised mainly motorcyclists under age 25 (all males), with the highest record of previous speeding violations of any of the SUBSAMPL driver groups. Again we are hampered by small numbers (only 6 drivers), but we would not infer that cannabis caused the high responsibility rate of these drivers.

Another questionable group comprised the 12 drivers with only amphetamines in their blood. They too had an 83 percent responsibility rate. Unlike the carboxy-THC group, this one was predominantly in the 25-54 age range. This group also had a history of driving violations greater than the drugfree group, although the record was not as pronounced as in the carboxy-THC drivers. Our analysis in Chapter 6 suggested that amphetamines could have been in the blood of some drivers as the result of the specimens being taken several hours after death, during which time the amphetamines could have leached from tissue back into the blood. There may have been a history of amphetamine use, but the amphetamines may not have been in the blood at the time of the crash. If this is true, what would explain the high responsibility rate of this group? It is quite possible that the amphetamines were used by some drivers to compensate for lack of sleep, and that fatigue was the dominant cause of their crashes.

The confounding of drug effects by personality and behavioral patterns is a possibility that must be considered in making inferences from research on drugs in crashes. The problem is as applicable to studies using control groups of on-the-road drivers as to crash studies using responsibility analysis. It is less of a problem regarding alcohol, where the correlation of BAC to crash risk is convincing evidence of the effect of alcohol. With drugs, unfortunately, a parallel relationship of concentration to crash risk has not been demonstrated.

7.2 Methodological Considerations

A drug study as extensive as this is expensive to conduct, particularly because of the costs of operating the data collection system and the costs of comprehensive blood assays. It behooves us, consequently, to identify any methodological improvements that could increase what is learned in future research, and perhaps reduce costs. That we do here.

7.2.1 Driver Fatality Studies

Chapter 1 concluded by reviewing the main benefits and limitations inherent in a driver fatality study. In addition, factors that may be controlled to increase the successfulness of the research are as follows:

(1) Employ an on-site field staff. Without exception, the coroner and medical examiner staffs participating in this study were most cooperative, but necessarily and properly they had to give first priority to the conduct of their official duties. Many were still able to meet the needs of this study very well, but the sample completion rates showed this was not always the case. Having field representatives of the research organization at the sampling sites could facilitate the review of incoming cases to see that all relevant ones are included. Other very significant benefits could be achieved as well. Arrangements might be made with the State and local personnel for the acquisition of the blood specimens shortly after death, which would aid drug detection and enhance comparability of results across sites. Uniformity of specimen acquisition and shipping procedures would also be facilitated. Finally, more relevant details on the crash occurrence might be obtained, as a valuable supplement to the sometimes sparse police accident reports.

(2) Use more efficient sample designs. This study found nonalcoholic drugs in only 18 percent of the drivers, meaning that well over $300,000 in

acquisition, shipping, and assay costs was spent for blood specimens containing none of the drugs. A more cost-beneficial approach might be targeted at samples where drugs are most pronounced, such as younger males. To study medicinal drugs, an older --perhaps elderly-- population is more appropriate.

It should be recognized that sampling from a restricted population of drivers limits generalizations to that population. That may not be a handicap if the population is one of special interest, such as the young or elderly drivers.

If the objective is not to study causation but to monitor national drug prevalence rates, a different sample design is necessary. In that case, random or stratified samples would be cost-effective.

(3) Concentrate on fewer drugs. This and other studies have found that there are relatively few drugs which have prevalences large enough to present a highway safety problem. These were mainly drugs of abuse. Assay costs could be greatly reduced by concentrating on these few prominent drugs.

While improvements in methods may be possible, we may question whether a fatal study is the best way to study the role of drugs in crashes. In addition to the operational and statistical difficulties we encountered, we have also seen problems introduced by postmortem blood specimens. Toxicologists Anderson and Prouty gave the following warning:

> Caution must also be exercised in interpreting analytical results as they relate to the physiological effects of drugs on drivers in motor-vehicle-related deaths. Predicting the effects of drugs on driving skills is a nebulous exercise in the living subject; difficulties are compounded when attempts to make such predictions are based on postmortem measurements (Anderson and Prouty, 1988, p.99).

Some of the problems of postmortem specimens would be avoided by studying injured but surviving drivers. There are advantages to that kind of study, which are discussed next.

7.2.2 Injured Driver Studies

For the objective of determining whether drugs play a causal role in crashes, using hospitals and trauma centers to study injured but surviving drivers offers several advantages. Briefly, these are as follows:

(1) Broader accident sample. Injury accidents cover a broader portion of the accident spectrum than do fatal crashes, thus they may provide more broadly relevant results.

(2) Opportunity to study more subtle effects. By necessity, a fatal sample will be heavily weighted in favor of the extreme behaviors that produce high-severity crashes-- speeding, falling asleep, etc. Single-vehicle crashes are frequently involved. Nonfatal crashes are more likely to involve multi-vehicle collisions and high-demand situations (traffic, intersections, etc.). Even slight driver impairments produced by drugs may be critical in those situations.

(3) Better specimen quality. Severe thorax trauma is common in fatal crashes, which can result in heart blood contamination and cause misleading assay results. Specimens taken from injured drivers are more likely to be of good quality. In addition, they will not have the problems of postmortem drug redistribution.

(4) Lower overall responsibility rate. Fatal crashes, which are dominated by single-vehicle crashes and behavioral extremes, tend to have high responsibility rates, even among drugfree drivers. This limits the degree to which drugs can elevate responsibility rates, especially when comparing drug-alcohol combinations with alcohol alone. Nonfatal injury crashes tend to involve more multi-vehicle crashes and lower overall responsibility rates, providing a greater opportunity for significant impairment effects to be detected.

On the debit side, there are two main limitations to sampling injured drivers. The first is that relatively few hospitals and trauma centers are likely to be used in the study; the unwillingness of some, and the cumbersomeness of logistics are deterrents to using several. Consequently, this lack of broad geographical representation makes this approach less amenable to estimating prevalence rates. That is why we emphasize its value for determining causation rather than prevalence rates. The second limitation is the difficulty of obtaining an unbiased sample of drivers. When the patient's permission is needed to draw a blood specimen, the refusal rate can be substantial (Terhune, 1982). This limitation is best avoided by using hospitals that routinely obtain blood specimens on all injured drivers.

7.2.3 The Vanderbilt Approach

Chapter 1 cited a study of the crash risks associated with prescription drugs in the elderly, by Ray and colleagues at Vanderbilt University (Ray et al., in press). By making use of prescription records and accident records, the researchers were able to achieve a sample size of over 16,000 drivers, including data on drivers not in accidents as well as in accidents. This provided a high statistical sensitivity capable of detecting the relative risks of prescription drugs. It also permitted the study of effects of dosage level. While this approach is generally not applicable to drugs of abuse, for prescription drugs it is a much more effective (and cheaper) method than studies requiring the collecting and assaying of blood specimens. A possible limitation of the method is the required inference that elevated crash risks of drugs were due to the drugs and not the problem that led to their use. However, the researchers' controls for dosage level and likelihood of drug use offset this limitation somewhat.

8.0 RECOMMENDATIONS

It is recommended that the National Highway Traffic Safety Administration consider the following for implementation.

(1) Further research on alcohol-drug effects. Provocative but inconclusive evidence was provided in this study on the effects of drugs combined with alcohol. The data suggested that combining some drugs with subintoxication levels of alcohol could increase relative crash risks to the range of alcohol intoxication. Unfortunately, it was necessary to combine data involving different drugs, and the contribution of any one was not discerned. This lead should be followed up in other studies, which may include crash studies and experimental research. The results may provide an important basis for educational and other preventive programs conducted by NHTSA.

(2) Research isolating drug effects from personality and behavioral patterns. Evidence of this study and previous ones suggests that abuse drugs may be used by people whose life style involves high-risk behaviors. A better understanding of the effects of drugs may be achieved if the contribution of life style can be distinguished from the impairment effects of drugs. This is a difficult challenge for research methodology, but it may be amenable to creative solutions. For example, benefits might be gained by studying drug-impaired drivers identified within NHTSA's police drug detection training program. They could be compared with a control group on relevant dimensions, such as their traffic record, criminal record, social deviance, risk-taking behaviors, and so on.

(3) Monitor selected drugs through FARS. Interesting regional variations in crash drug involvement were indicated by this study, which included approximately 8 percent of drivers killed nationwide and meeting our sampling criteria during the study period. Developing a more complete picture is desirable. Since trends in drug use are clearly evident in surveys of the National Institute on Drug Abuse, monitoring crash drug prevalence over time is suggested. Since the FARS system already does this with alcohol, expanding the data collection to other drugs is appropriate. Since blood assays are so expensive, it is recommended that only a fractional systematic sample be used, concentrating on a few drugs of greater importance. Cannabis, cocaine, amphetamines, benzodiazepines, and barbiturates should be considered. Careful control of specimen collection methods should be achieved, which suggests the use of relatively few test sites.

(4) Automating responsibility analysis. Responsibility analysis was made more objective in this study by specifying coding guidelines for assigning responsibility in specific collision

types. The guidelines are generally based on vehicle actions and collision configurations. There is a good possibility that a computer program could be written to estimate driver responsibility from key indicators that could be readily coded from accident reports. It is recommended that NHTSA consider developing such a method, which could provide a valuable research and monitoring tool.

(5) Injured driver study. For reasons discussed in the previous chapter, it is recommended that NHTSA consider an injured driver study to further our understanding of the causal contribution of drugs to crashes.

9.0 THE RESEARCHERS

Kenneth W. Terhune of Calspan's Accident Research Group was Principal Investigator for the project. Dr. Terhune holds a Ph.D. in Psychology and degrees in automotive and mechanical engineering. He has nearly forty years experience in behavioral science and highway safety research. His research included the topics of crash causation, vehicle rollover, vehicle crashworthiness and aggressivity, accident surrogates, and injury countermeasures. He previously directed a major study of drugs in injured drivers, and he is an authority on responsibility analysis and the epidemiological study of drugs in drivers.

Carol A. Ippolito of Calspan's Accident Research Group was Research Associate for the project. She served as Operations Manager during the data collection phase, when she was the primary contact with all field sites. Ms. Ippolito holds an M.A. degree in psychology and has four publications, including co-authorship of the responsibility analysis manual for this study.

Donald L. Hendricks of Calspan's Accident Research Group was Data Collection Manager for the project. He was the primary coordinator of subcontractors during data collection. Mr. Hendricks holds a B.A. in Business Administration, and he has over thirty years experience in highway safety. He supervises the field activities of all in-depth accident investigations for the Accident Research Group.

John G. Michalovic was director of Calspan's analytical chemistry laboratory at the time of the study. (He presently is Manager, Safety and Environment.) He was instrumental in evaluating candidate assay laboratories for the project, and he designed the quality check procedures. He holds an M.S. in Chemistry and he has over thirty publications.

Stuart C. Bogema, Ph.D. managed project operations for American Medical Laboratories, which performed the drug assays. Dr. Bogema is Vice President, Research and Development, and Director, Toxicology and Therapeutic Drug Monitoring at American Medical Laboratories. Dr. Bogema's Ph.D. is in pathology, and he is a legally qualified expert in forensic and chemical toxicology. He has nearly thirty published articles and abstracts.

Philip Santinga of American Medical Laboratories was analytical director of the specimen assays for the project. He holds a Master's degree in medical technology, a B.A. in chemistry, and has been trained in forensic toxicology. Mr. Santinga has nearly twenty-five years experience in toxicology.

Richard D. Blomberg is President of Dunlap and Associates, Inc. of Norwalk, Connecticut. Through a subcontract, Dunlap was responsible for planning all field arrangements to provide the needed sample and source documents for the study. Mr. Blomberg had managerial responsibility for the Dunlap subcontract. He holds an M.S. degree in industrial and management engineering. He has conducted numerous studies in highway safety including a study of drug abuse and driving performance.

David F. Preusser, Ph.D. was at Dunlap and Associates, Inc. during its participation in the project. (He is now at PRG, Inc.) He was responsible for technical management of the Dunlap subcontract. He has a Ph.D. in psychology and he has over fifty publications, mostly in highway safety. He has analyzed drug abuse in relation to driving performance, and he directed a study on drug involvement of heavy truck drivers.

10.0 REFERENCES

Anderson, W.H. and Prouty, R.W. (1989). Postmortem redistribution of drugs. In R.C. Baselt (Ed.), Advances in Analytical Toxicology. Chicago: Yearbook Medical Publishers.

Barnett, G. and Willette, R.E. (1989). Feasibility of chemical testing for drug-impaired performance. In R.C. Baselt (Ed.), Advances in Analytical Toxicology. Chicago: Yearbook Medical Publishers.

Buffalo News (May, 1992). Hard-core drug use in U.S. getting worse, report says.

Budd, R.D., Muto, J.J., and Wong, J.K. (1989). Drugs of abuse found in fatally injured drivers in Los Angeles County. Drug and Alcohol Dependence, 23, 153-158.

Borkenstein, F.R., Crowther, R.F., Shumate, R.P., Zeil, W.B., and Zylman, R. (1974). The role of the drinking driver in traffic accidents (The Grand Rapids Study). Blutalkohol, 11, Supplement 1.

Constantino, A.G. (1990). Distribution of Amitriptyline and Other Tricylic Antidepressant Drugs in the Postmortem Interval. Unpublished Ph.D. dissertation, University of Maryland, College Park, Maryland.

Donelson, A.C., Haas, G.C., and Walsh, P.J. (1986). The Etiology of Fatal Traffic Accidents Involving Alcohol and Cannabis. Ottawa, Ontario: Traffic Injury Research Foundation of Canada.

Fell, J.C. and Hertz, E.S. (1990). The effects of blood alcohol concentration on time of death for fatal crash victims. Proceedings, 34th Annual Conference of the Association for the Advancement of Automotive Medicine. Des Plaines, IL: AAAM Publishing.

Hurst, P.M. (1970). Estimating the effectiveness of blood alcohol limits. Behavioral Research in Highway Safety, 1, 87-99.

Joscelyn, K.B. and Donelson, A.C. (1980). Drug Research Methodology, Vol. 2. The Identification of Drugs of Interest in Highway Safety. National Highway Traffic Safety Administration, Report DOT-HS-805-299.

Leirer, V.O., Yesavage, J.A., and Morrow, D.G. (1991). Marijuana carry-over effects on pilot performance. Aviation, Space, and Environmental Medicine. March, 221-227.

Marzuk, P.M., Tardiff, K., Leon, A.C., Stajic, M., Morgan, E.B., and Mann, J.J. (1990). Prevalence of recent cocaine use among motor vehicle fatalities in New York City. The Journal of the American Medical Association, 263, 250-256.

National Institute on Drug Abuse (1988). Semiannual Report, Trend Data Through July - December 1987: Data from the Drug Abuse Warning Network (DAWN). Series G, Number 21. Washington, D.C.: U.S. Dept. of Health and Human Services.

National Institute on Drug Abuse (1990). Semiannual Report: Data from the Drug Abuse Warning Network (DAWN). Series G, Number 24. Washington, D.C.: U.S. Dept. of Health and Human Services.

National Institute on Drug Abuse (1992). 1991 National Household Survey on Drug Abuse. DHHS Publication No. ADM921887. Washington, D.C.: U.S. Dept. of Health and Human Services.

National Safety Council (1991). Accident Facts 1991 Edition. Chicago, IL: National Safety Council. Ray, W.A., Fought, R.L., and Decker, M.D. (1992). Psychoactive drugs and the risk of injurious motor vehicle crashes in elderly drivers. American Journal of Epidemiology, (in press).

Root, I. (1988). Drugs of abuse and fatal automobile accidents. Scalpel and Quill: The Bulletin of the Pittsburgh Institute of Legal Medicine, 22(2).

Smiley, A. (1986). Marijuana: On-road and driving simulator studies. In H. Moskowitz (Ed.), Marijuana, Cocaine, and Traffic Safety. Alcohol, Drugs, and Driving: Abstracts and Reviews, 2(3-4).

Stoduto, G., Vingilis, E., Kapur, B.M., Sheu, W., McLellan, B.A., and Liban, C.B. (1991). Alcohol and drugs in motor vehicle collision admissions to a regional trauma unit: Demographics, injury and crash characteristics. Proceedings, 35th Annual Conference of the Association for the Advancement of Automotive Medicine. Des Plaines, IL: AAAM Publishing.

Terhune, K.W. (1982). The Role of Alcohol, Marijuana, and Other Drugs in the Accidents of Injured Drivers. Final report, Contract DOT-HS-5-01179, National Highway Traffic Safety Administration.

Terhune, K.W. (1983). An evaluation of responsibility analysis for assessing alcohol and drug crash effects. Accident Analysis and Prevention, 15, 237-246.

Terhune, K.W. (1986). Problems and methods in studying drug crash effects. Alcohol, Drugs, and Driving: Abstracts and Reviews, 2(3-4), 1-13.

Warren, R., Simpson, H., Hilchie, J., Cimbura, G., Lucas, D., and Bennet, R. (1981). Characteristics of fatally injured drivers testing positive for drugs other than alcohol. In L. Goldberg (Ed.), Alcohol, Drugs, and Traffic Safety, Vol. I. Stockholm: Almquist & Wiksell International.

Williams, A.F., Peat, M.A., Crouch, D.J., Wells, J.K., and Finkle, B.S. (1985). Drugs in fatally injured young male drivers. Public Health Reports, 100(1), 19-25.

Appendix A
The Drug Selection Procedure

We sought to identify the drugs potentially the most important to crash causation in forming the list of drugs for examination in the present study. We began with a list of 55 "Drugs of Interest" provided by the COTR, who designated 29 of them as being of "most interest." Since costs to assay all the drugs on the list would have been prohibitive and well exceed estimates in the initial budget, we sought to eliminate drugs of minimal importance, add others if warranted, and establish an order of importance for the drugs on the list. For the purposes of selection, importance was defined as a function of estimated incidence (frequency of use) and impairment effects. Impairment effects were estimated through ratings of the 1980 NHTSA workshop's panel of experts (see Joscelyn and Donelson, 1980). Estimated incidence was determined through a literature review of recent studies, from drug sales data, and other reports of drug usage. Incidence was emphasized for the following reasons:

(a) Incidence in crash studies will reflect both incidence of use and impairment effects.
(b) Incidence is more objectively determined.
(c) All the drugs on the COTR's list are likely to be impairing under certain dosages.

A.1 The Final Drug List

The final drug list for study (44 drugs and 11 metabolites) in this project is shown in Table A-1. Of the COTR's original list of 55 drugs, our list includes 35 of those, among which are all those designated of greatest interest by the COTR. The table shows the ranges of prevalence rates found in previous studies of drivers and the number of experts mentioning the drug as important to our study.

It is important to point out that our recommended drug list was governed in part by the objective of keeping the assay costs within the level designated in the contract budget. The COTR's initial list of suggested drugs would have far exceeded that level, hence we proceeded to reduce the list by eliminating drugs of less indicated importance. After an initial list was decided upon (our "core" list of drugs we determined to be of most interest and importance), we found that our laboratory of choice, American Medical Laboratories, was able to include some other drugs at no extra cost.

A.2 The Selection Process

The selection of the drugs of interest began with the COTR's proposed list, which was amended after reviewing other sources of information. The first avenue explored was the literature review of

Table A-1

Study Drug List

(Drugs are listed in order of estimated incidence/importance)

Group*	Substance	Incidence in Studies	No. Experts Mentioning
I: May exceed 8%	Alcohol (ethanol)	25-79%	13
	Delta-9 THC	7-45%	12
	Carboxy-THC (THC metabolite)	7-45%	12
II: 4-8% expected	Benzodiazepines	4-8%	8
	Diazepam		
	Nordiazepam (diazepam metabolite)		
	Lorazepam		
	Flurazepam		
	Desethylflurazepam (flurazepam metabolite)		
	Alprazolam		
	Oxazepam		
	Chlordiazepoxide		
III: 2-4% expected	Cocaine	1-12%	13
	Benzoylecgonine (cocaine metabolite)		
	Phencyclidine	1-56%	3
	Barbiturates	0-3%	6
	Phenobarbital		
	Secobarbital		
	Butabarbital		
	Butalbital		
	Pentobarbital		
	Amobarbital		
	Non-heroin opiates	1-20%	6
	Codeine		
	Morphine		
	Amphetamines	0-3%	7
	Amphetamine		
	Methamphetamine		
	Caffeine		
IV: Less than 2% expected	Heroin	Not reported	8
	Non-barbituate sedatives	0-6%	1
	Ethchlorvynol		
	Methaqualone		
	Meprobamate		

Study Drug List (Continued)

Group*	Substance	Incidence in Studies	No. Experts Mentioning
IV: (cont.)	Antihistamines	1%	3
	Diphenhydramine hydrochloride		
	Chlorpheniramine		
	LSD	.5%	4
	Antidepressants	.5%	3
	Amitriptyline		
	Nortriptyline** (amitriptyline metabolite)		
	Imipramine**		
	Desipramine** (imipramine metabolite)		
	Fluoxetine**		
	Norfluoxetine** (fluoxetine metabolite)		
	Doxepin**		
	Desmethyldoxepin (doxepin metabolite)		
	Analgesics	0.5-1%	0
	Meperidine hydrochlorine		
	Methadone		
	Propoxyphene		
	Norpropoxyphene (propoxyphene metabolite)		
	Oxycodone		
	Antipsychotics	0-1%	0
	Chlorpromazine**		
	Thioridazine**		
	Mesoridazine**		
	Antiarrhythmics	0-1%	0
	Quinidine**		
	Procainamide**		
	N-Acetylprocainamide (procainamide metabolite)		
	Lidocaine**		
	Flecainide**		
	Muscle relaxant	0-1%	0
	Cyclobenzaprine**		

*Groups are based on expected incidence rate for the drug or drug-groups; e.g., benzodiazepines as a group are expected to be in the 4-8% range, hence all benzodiazepines are listed in Group II.

**These drugs were included because the recommended assay laboratory included them at no extra cost.

recent (past 5-7 years) studies involving drug/alcohol use and highway accidents. The studies reviewed are listed in Table A-2. This review provided expected incidence rates for most of the drugs on the COTR's list and this information served as a starting point for prioritizing the drug list. Next, experts in the field were contacted by phone to ensure that the drug list proposed was the most "up-to-date." This group of experts consisted of scientists conducting research in the field, directors of drug abuse centers (or their associates), law enforcement officials, and toxicologists from the States targeted for data collection. These experts gave their recommendations for drugs to be included and estimates of their incidence rates based on their own research findings and/or contact with these substances. The experts contacted are listed in Table A-3.

Also examined was the 1987 report from the Drug Abuse Warning Network (DAWN) provided by the National Institute of Drug Abuse. This report consists of statistics on types of drugs found in emergency room episodes and their prevalence rates. The report revealed that 90% of the total emergency room episodes involving drugs listed the following 6 substances: cocaine (32%), alcohol in combination with another drug (28%), heroin/morphine (13%), marijuana/hashish (7%), PCP/PCP combinations (6%), and diazepam (5%). This information was also used in the development of the final list of drugs of interest.

A.3 Rationale for Choosing the Final "Drugs of Interest"

In developing the final list of drugs of interest, most weight was given to the prevalence rates reported in the reviewed studies. Other sources, such as the consensus of experts in the field, were also taken into consideration.

Eight drugs in Table A-1 were frequently mentioned by experts in the field as drugs used most often and potentially dangerous to driving ability. These were alcohol, marijuana, cocaine, diazepam, PCP, heroin, morphine, and barbiturates. All of these substances were on the list suggested by the COTR for inclusion in the present study.

Excluded from Table A-1 are volatile solvents and appetite suppressants. These substances were excluded from the list mainly because of low incidence rates. The incidence rate of volatile solvents is extremely low and their presence in the blood can be detected within only a short time of their ingestion. The effects of caffeine and appetite suppressants were given the lowest possible rating by the 1980 NHTSA workshop's panel of experts (Joscelyn and Donelson, 1980).

Other drugs which were on the COTR's list, such as mescaline, MDA, and some antidepressants were eliminated after assay cost estimates, prevalence rates, and expert opinions were considered.

Table A-2

Studies Used in Literature Review to Obtain Incidence Rates

Cimbura, G., Lucas, D. M., Bennett, R. C., Warren, R. A., & Simpson, H. M. (1982). Incidence and toxicological aspects of drugs detected in 484 fatally injured drivers and pedestrians in Ontario. Journal of Forensic Sciences, 27(4), 855-867.

Compton, R. P. (1986). Field evaluation of the Los Angeles Police Department Drug Detection Program. NHTSA Technical Report No. DOT HS 807 012.

Donelson, A. C., Haas, G. C., & Walsh, P. J. (1986). The etiology of fatal traffic accidents involving alcohol and cannabis. Report from the Traffic Injury Research Foundation of Canada, 171 Nepean Street, Ottawa, Ontario, Canada, K2P 0B4.

Mason, A. P., & McBay, A. J. (1984). Ethanol, marijuana, and other drug use in 600 drivers killed in single-vehicle crashes in North Carolina, 1978-1981. Journal of Forensic Sciences, 29(4), 987-1026.

Nakabayashi, K., Aronson, S. C., Siegal, M., Sturner, W. Q., & Aronson, S. M. (1984). Traffic fatalities in Rhode Island: Part II. The timing of accidents and the role of marital status, alcohol, and psychoactive drugs. Rhode Island Medical Journal, 67, 171-178.

Soderstrom, C. A., Trifillis, A. L., Shankar, B. S., Clark, W. E., & Cowley, R. A. (1988). Marijuana and alcohol use among 1023 trauma patients: A prospective study. Archives of Surgery, 123, 733-737.

Terhune, K. W. (1982). The role of alcohol, marijuana, and other drugs in the accidents of injured drivers. Calspan Field Services, Inc., Buffalo, New York, Perf. for the U.S. Dept. of Transportation, Contract No. DOT-HS-5-01179.

Williams, A. F., Peat, M. A., Crouch, D. J., Wells, J. A., & Finkle, B. S. (1985). Drugs in fatally injured young male drivers. Public Health Reports, 100(1), 19-25.

Table A-3

List of Experts Contacted

Mr. Paul Cascarano
Asst. Director of the National Institute of Justice
Director of the Drug Use Forecasting Project
Washington, D.C.

Dr. Alan Donelson
Traffic Injury Research Foundation
Ottawa, Canada

Mr. Christopher Hanson
Asst. Director of the Bureau of Alcoholism and Substance Abuse
State of Washington

Dr. Arthur McBay
Chief Toxicologist
State of North Carolina

Mr. Dennis McCarty
Head of Statistics
Division of Drug Rehabilitation
State of Massachusetts

Dr. Herbert Moscowitz
Director of Southern California Research Institute

Dr. Alfonse Polklis
Director, Dept. of Pathology
MCV Station
Richmond, Virginia

Mr. David Polley
New York State Regional Coordinator for Drug Abuse Services

Mr. Michael Quirke
Head of Statistics
Office of Alcohol and Other Drug Abuse
State of Wisconsin

Dr. Vidmantas Raisys
Head of Toxicology
State of Washington

Mr. Edward Reese
Coordinator of the New York State Division of
Substance Abuse Services

Mr. Richard Swartz
Asst. Director of the Dept. of Alcohol and Drug Abuse
State of California

Sgt. Richard Ward
DUI Division
California Highway Patrol

A.4 Summary

Our objective in developing a "Drugs of Interest" list was to recommend the most important drugs with regard to highway safety that could be assayed within the contract budget. In so doing, the COTR's suggested list of 55 drugs was reduced by considering assay costs in relation to indicated importance of each drug.[1] Importance was determined through evaluation of several sources of information: a literature review conducted on related studies completed in the last seven years, phone conversations with experts in the field, NHTSA's 1980 workshop report on identification of drugs of interest in highway safety, and the Pharmacy Times list of the top 30 prescription drugs for 1987. The final drug list contained 44 parent drugs and 11 metabolites.

References

Joscelyn, K. B., & Donelson, A. C. (1980). Drug research methodology, Volume II: The identification of drugs of interest in highway safety. NHTSA Technical Report No. DOT HS-7-01530.

Top 30 Generic Drugs - 1987. Pharmacy Times, April, 1988.

[1]It is important to note that the COTR's original recommended list of 55 drugs was not the same as the final list of 55 drugs.

Appendix B
Descriptions of the Drugs Studied

The objective of this appendix is to provide the interested reader with information on the nature of the drugs examined in the study, particularly with regard to the behavioral effects that suggested them as a potential highway safety problem. This is not intended as a technical discussion of psychodynamics or psychokinetics.

The drugs are discussed below within the classifications used in this report. The primary references used in compiling this information were: Disposition of Toxic Drugs and Chemicals in Man, by R.C. Baselt and R.H. Cravey; NHTSA Report No. DOT-HS-805-461 - Drugs and Highway Safety, 1980, by K. B. Joscelyn et al.; and The Pill Book, by G.I. Simon and H.M. Silverman.

B.1 Alcohol

Alcohol (ethanol) is considered primarily a recreational or "social" drug, one rarely used therapeutically. Alcohol is quickly and evenly absorbed throughout the body. It is a central nervous system depressant, i.e. it depresses the activity of all excitable tissues, which results in changes that range from a slight lethargy or sleepiness, to anesthesia, to death from breathing and heart depression (Schuckit, 1989). Alcohol impairs several functions related to driving: motor coordination, attention and alertness, visual acuity, mood (disinhibition), judgment, reaction time, and decision-making (Jones and Joscelyn, 1978). However, the degree of impairment or specific effects on the user are dependent upon the amount of alcohol consumed and on age, sex, weight, and the user's history of alcohol use. Further, when combined with other drugs, a lack of sleep, or a lack of food in the stomach, the impairment effects of alcohol may be enhanced.

Tests to detect alcohol are among the simplest and least expensive and are quite accurate. Blood alcohol concentrations (BAC) can most reliably be obtained through blood or breath (using the "Breathalyzer" test), and can be estimated from urine or vitreous humor specimens.

B.2 Cannabis

Cannabis is derived from the Cannibis sativa plant, and it may be the most widely-used recreational drug after alcohol. It has been used for thousands of years in various countries (and cultures) around the world. Cannabis is generally smoked, although it is sometimes ingested orally. The cannabis user may experience euphoria, increased hunger, paranoia, inability to keep track of time, sleepiness, and short-term memory loss. At high doses, hallucinations, confusion, panic, and disorientation may occur. Experimentally, cannabis has been found to effect performance

decrements on tracking, sensory and perceptual functions, motor coordination, and reaction time (National Academy of Science, 1982). Some common street slang that refers to marijuana includes pot, grass, ganja, weed, and mary jane.

Delta-9 tetrahydrocannabinol. Delta-9 tetrahydrocannabinol (THC) is the most active substance found in the marijuana plant (Cannabis sativa). The presence of this substance in a blood specimen indicates recent ingestion of cannabis. It is believed that delta-9 tetrahydrocannabinol is the ingredient in cannabis that produces all of the effects experienced by the individual and is the psychoactive ingredient that produces the "high".

Carboxy-THC. Carboxy-THC is an inactive metabolite of delta-9 tetra-hydrocannabinol. Found by itself, carboxy-THC does not indicate recent use, or a "high," but it is thought to accumulate in some fatty tissue in regular users (Jaffe, 1985, as cited in Schuckit, 1989).

B.3 Hallucinogens

Hallucinogens are chemical combinations which distort the user's perceptions of reality, causing him/her to visually and/or auditorially experience sensations, sights, or delusions that deviate from "objective" reality. They can be ingested in several different ways, including orally, intranasally, intravenously, or smoking, and they operate by stimulating the central nervous system. Hallucinogens are considered "recreational" drugs and are created from natural and/or synthetic compounds. For the most part, hallucinogens are illegal and are among the drugs of abuse.

Phencyclidine. Phencyclidine (PCP) can be used (in its legitimate form) as an animal tranquilizer; however, it is also used as a street drug, ingested by humans. PCP can cause disorientation and loss of coordination, as well as full-blown hallucinations. It is the distortion of objective reality that makes a driver under the influence of PCP potentially very dangerous. Since the driver may see things that aren't really there, his or her actions may be inappropriate and increase the likelihood of causing an accident. Another name by which PCP is commonly known on the street is "angel dust."

Lysergic acid diethylamide. Lysergic acid diethylamide (LSD) is a synthethic compound manufactured and taken only for its mind-altering properties. As with other types of hallucinogens, the user can experience visual, auditory, and thought distortions, all of which have the potential to cause a person driving a vehicle to take inappropriate or dangerous actions. LSD is also known as "acid."

B.4 Benzodiazepine Tranquilizers

Benzodiazepines are often classified as "minor" tranquilizers prescribed by doctors for the relief of anxiety and tension. Benzodiazepines depress the central nervous system and work by relaxing the large skeletal muscles. Although generally considered safe (without harmful side effects) by the medical community, these drugs have the potential for abuse, and may impair driving performance by decreasing motor coordination, reaction time, alertness, and decision-making ability (Joscelyn et al., 1980). This impairment effect may be enhanced when the drug is combined with alcohol (Scharf, 1988, as cited in Schuckit, 1989). Most of the drugs listed below that belong to this class are chemically similar and exert similar effects. Important differences are noted.

Diazepam. Diazepam is commonly known by its trade name - Valium(R), at one time the most often prescribed drug of the benzodiazepine group. In recent years, the number of prescriptions for Valium(R) have declined, partly due to the allegation that many doctors were overprescribing the drug.

Nordiazepam. Nordiazepam is the metabolite of diazepam, usually found in the blood with diazepam.

Lorazepam. Lorazepam is a minor tranquilizer commomly known by the trade name Ativan(R). It is prescribed for relief of anxiety, tension, or agitation.

Flurazepam. Flurazepam is a minor tranquilizer often prescribed for insomnia or sleeplessness. Its brand name is Dalmane(R).

Desethylflurazepam. Desethylflurazepam is the metabolite of flurazepam.

Alprazolam. In low doses (0.75-4 mg.), alprazolam is effective as an anti-anxiety agent. When prescribed at higher doses (6-9 mg.), the drug is effective in treating phobic disorders and panic attacks (Baselt and Cravey, 1989). The common trade name for alprazolam is Xanax(R).

Oxazepam. Oxazepam is an anti-anxiety agent, somewhat less potent than diazepam. Its common trade name is Serax(R).

Chlordiazepoxide. Chlordiazepoxide is commonly known as Librium(R), prescribed as an antianxiety agent, an anticonvulsant, a muscle relaxant, or a hypnotic. It is considered the "prototype" of the benzodiazepine class, being the earliest to have been approved for human use (Baselt and Cravey, 1989).

Desmethylchlordiazepoxide. Desmethylchlordiazepoxide is the metabolite of chlordiazepoxide.

Appendix C
Statistical Power Analysis

NHTSA's original solicitation for this project specified a tentative sample size of 2,550 drivers. To determine whether this or a different sample size was appropriate for the objectives of the study, we applied statistical power analysis (Cohen, 1969) to the central analysis planned, responsibility analysis. A basic question addressed was: What sample size is needed in order to determine whether the responsibility rates (percentages) of drug-involved drivers are significantly different from the rates of the "drugfree" drivers? To perform the power analysis, it was necessary to know or assume the following:

- The statistical test to be used. In our case, this was the Chi-square test.

- The expected drugfree proportion of drivers. Previous studies of fatally-injured drivers indicated this to be about 35%.

- The expected responsibility rate of the drugfree drivers. The previous studies suggested this would be around 68%.

- How small a drug effect is important to detect? It seemed reasonable to require that the analyses be able to detect a drug-associated responsibility rate at least 10% higher than the drugfree rate. Consequently, we considered an elevation of that magnitude "an important effect."

- How much statistical power is desired? Power is the probability that a test will detect a genuine difference between two groups; the more powerful a test, the more likely one is to find small differences to be statistically significant. While a power level of 0.80 is desirable (Cohen, 1969), we decided that a level of 0.60 was acceptable.

- The expected prevalence rate for the specific drug or drug group of interest, when present by itself. To indicate a drug's contribution to crashes, we have to examine its responsibility rate in drivers who have only that drug in their systems. Evidence from other studies of fatally injured drivers indicated that drugs of abuse are most often found combined with alcohol. For example, Donelson et al. (1986) found THC present in 11% of driver fatalities, but the THC was by itself in less than 1%. Considering the prevalence rates found in previous studies, it was our judgment that the highest (most optimistic) prevalence rates for drugs present alone would be 3%

for cannabis, cocaine, and phencyclidine respectively, and 5% for the benzodiazepine group.

Calculations were extensive; what follows here is a summary of the results from those calculations.

It was found that for a power of 0.80, a sample size of over 3,400 drivers would be needed to detect "an important effect" of a drug having a 5% incidence rate by itself, and over 5,700 drivers would be needed for a rate of 3%. For a power of 0.60, the figures changed to 2,150 and 3,500. It appeared that the sample size of 2550 targeted in NHTSA's solicitation for proposals had minimal statistical power, and could produce inconclusive statistical results. Even a sample size of 3,500 appeared to offer adequate statistical power only for a drug with a 5% prevalence rate by itself.

Since the most prevalent drugs of interest were expected to be found with alcohol much more than they are found alone, we performed power analysis to answer a second basic question: What sample size is needed to determine whether an alcohol-drug combination has a significantly higher responsibility rate than alcohol by itself? On the basis of the research literature, we estimated that about 40% of the drivers will have only alcohol in their systems, while 6-10% would have alcohol combined with cannabis, cocaine, or PCP, and 3-4% would combine benzodiazepines with alcohol. But since alcohol alone was expected to have a responsibility rate around 88%, it seemed unlikely that the alcohol-drug combination would raise responsibility much more than 5%, to 93%. Detecting an effect of this magnitude is possible with a sample size of 2550 and drug-alcohol combinations with 8-10% prevalence rates, according to our calculations. A sample of 3500 drivers appeared necessary for drug-alcohol combinations with a 6% prevalence rate. It seemed, then, that the 2550-driver sample had more power to detect the effects of drug-alcohol combinations than of drugs (or drug groups) present by themselves.

A further important consideration regarding sample size was the size of the confidence intervals in projecting drug prevalence rates in the population of driver fatalities. Table C-1 suggested that we would be able to estimate the population rates to within 1%, based on a 2550-driver sample, or within 1-4%, for driver subgroups as small as 10% of the total sample.[1] (This is relevant, for example, to our provision of prevalence rates for individual States.) While it must be recognized that our sample was not to be a true probability sample, it did appear that the 2550-driver sample size was more than adequate for approximating prevalence rates.

---

[1]The calculations are based on the assumption that the sample of 2550 drivers would be taken from an infinitely large population.

Table C-1

Drug Prevalence Rate Confidence Intervals*
Using a 2550-Driver Sample Size

A. Estimating Population Incidence from Complete Sample

Incidence Rate	95% Confidence Interval
15%	+ or - 1%
10%	+ or - 1%
5%	+ or - 1%
2%	+ or - 0.5%
1%	+ or - 0.2%

B. Estimating Population Subgroup Incidence Rates from a Subsample of 255 Drivers (10% of Sample)

Incidence Rate	95% Confidence Interval
15%	+ or - 4%
10%	+ or - 4%
5%	+ or - 3%
2%	+ or - 2%
1%	+ or - 1%

*These confidence intervals assumed an infinitely large population.

A final consideration regarding sample size was that our power considerations did not assume subdivision of the sample in any way, as appropriate in attempts to control for driver age and gender, urban vs. rural crashes, time interval between crash and driver's death, etc. Since each subdivision would further reduce statistical power, subdividing did not appear feasible.

In summary, our conclusions on sample size were as follows:

(1) Because a driver-fatality sample was expected to have very low prevalence rates of drugs present by themselves, the RFP target of 2550 drivers had minimal ability to reveal impairment effects of individual drugs or drug groups, when using responsibility analysis. Even a 3500-driver sample appeared capable of detecting drug effects only under best-scenario conditions.

(2) If our assumptions were valid, a 2550-driver sample would have a somewhat greater possibility of detecting impairment effects of alcohol-drug combinations than of drugs present by themselves.

(3) A sample of 2550 drivers appeared to have more than ample capability for estimating incidence rates with small confidence intervals, i.e., + or - 1%.

Thus, the 2550-driver sample would have served well the objective of estimating drug incidence rates, but many results still would have been inconclusive regarding drug impairment effects. Certainly a larger sample would have had greater statistical power, but even raising the sample to 3500 drivers could easily have shown inconclusive results with the responsibility analyses. Considering the greatly added expense and logistic difficulties of a substantially larger sample size, we recommended staying with the 2550-driver sample target.

References

Cohen, J. (1969). Statistical Power Analysis for the Behavioral Sciences. New York: Academic Press.

Donelson, A. C., Haas, G. C., and Walsh, P. J. (1986). The Etiology of Fatal Traffic Accidents Involving Alcohol and Cannabis. Ottawa, Ontario: Traffic Injury Research Foundation of Canada.

Appendix D

Data Collection Procedures

Figure D.1 illustrates how each case began with a driver fatality and was completed when its data forms were in final storage. The steps in case creation were generally as follows.

(1) Driver dies. A fatal crash results in the initiation of a police accident report (PAR) and, upon the driver's death, the cadaver is examined by a coroner or a medical examiner. The body may be transported to an autopsy center.

(2) Site initiates case. The fatality enters our study when the site determines that, as far as is known, the driver is eligible for our study. With that tentative determination, a blood specimen is drawn in accordance with the written project protocol. Heart blood is taken, unless due to trauma none is available, or it appears to be contaminated, e.g., by stomach contents. In these case, the specimen is taken from the best available source. The blood is placed a into project-supplied tube containing sodium fluoride as a preservative. The site promptly completes a Case Initiation Form (a project document), and faxes or mails it to Calspan, where the case is logged into the Case Monitoring Database (CMDB).

(3) Site sends specimen to assay lab. The tube is immediately shipped, or if a large-volume site, tubes are stored up to 6 days at 4° C, then shipped in batches. (Depending on the site organization, shipment is either directly to American Medical Laboratories or through a central coordinating office for the site.) Shipments are sent by air express, for delivery within 24 hours. Site faxes or mails airbill to Calspan.

Note: The specimens were shipped in special protective boxes supplied by the project. No cooling packs were included, when discussion with our toxicological consultants determined that they were not essential.

(4) Lab assays blood. Daily, the American Medical Laboratories teletype to Calspan a report of all specimens received. The lab stores each specimen at 4° C, and within 24 hours of receipt, screening for drugs begins. Positive results are followed by confirmation tests and quantitation assays. (Most are completed within two to four weeks of specimen receipt.) The assay results are teletyped to Calspan.

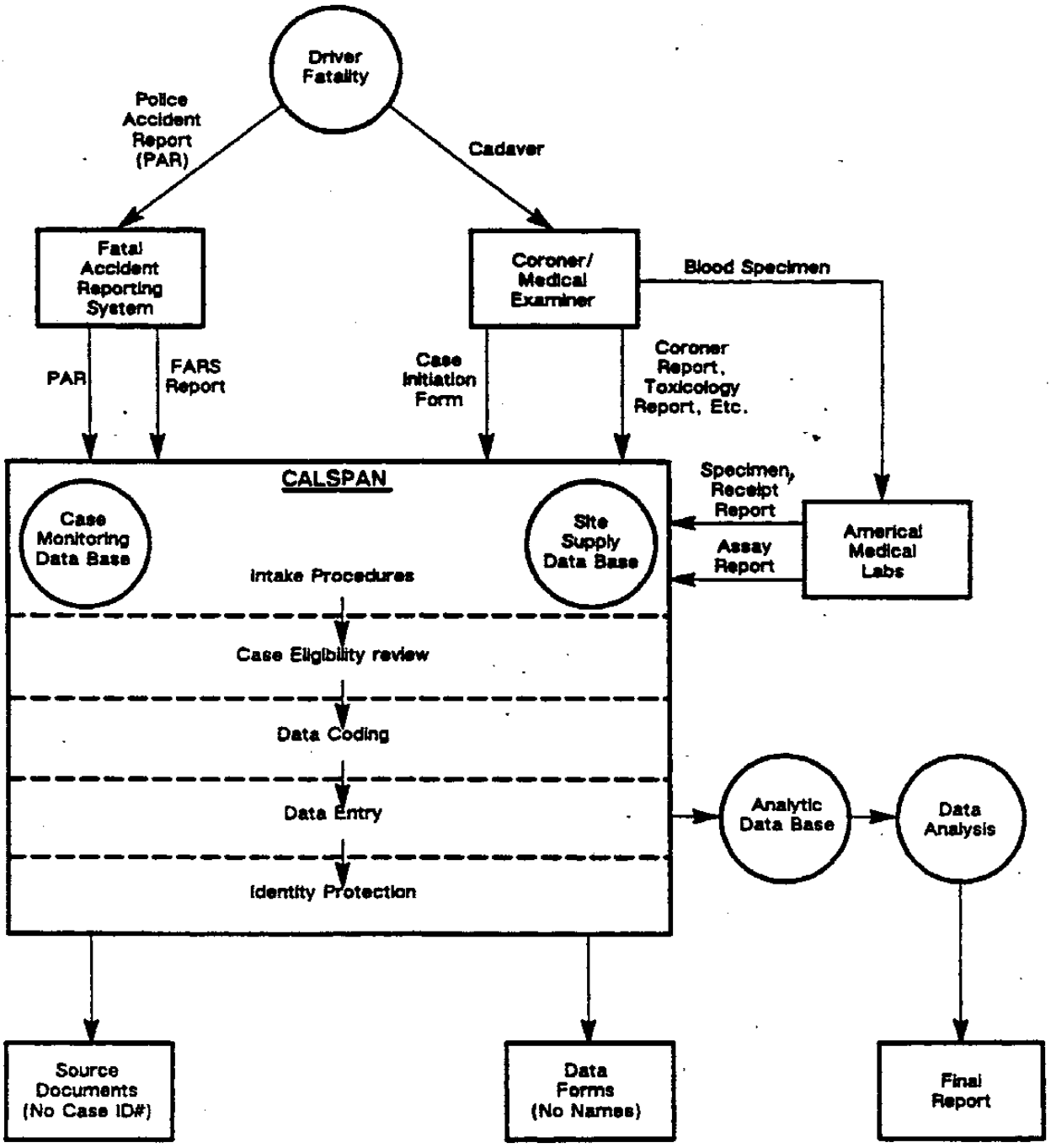


Figure D1 CASE FLOW THROUGH THE DATA COLLECTION SYSTEM

(5) Calspan checks assay report. In addition to checking the assay report for possible aberrations rare), Calspan checks for the detection of these possibly therapeutically administered drugs:

diazepam, cocaine, meperidine hydrochloride, codeine, morphine, and lidocaine. If found, Calspan contacts the site to determine whether the drug was administered to the driver prior to death. If so, this is encoded on the assay report.

(6) Site sends coroner/ME report to Calspan. When the coroner's or medical examiner's report is completed, the site faxes or mails it to Calspan. This is typically about two months after the crash, but it may take up to six months.

(7) State FARS office sends PAR and FARS report to Calspan. From identifying information provided by Calspan, the State FARS office locates the PAR and the FARS report on the case. Copies of both reports are sent to Calspan. Depending on the State, these reports arrive an average of 3 to 4 1/2 months after the crash, and some cases take up to 6 months.

At this point, Calspan has all the field documents for the case. In all subsequent steps, the case is processed at Calspan.

(8) Final case eligibility check is made. Referring to the field documents, the case's eligibility is checked on each of the sampling criteria. The cause of death and time of death is obtained from the coroner/ME report. Confirmation that the victim was a driver and that the vehicle type was eligible comes primarily from the FARS report. The time of the crash, of the victim's death, and the drawing of the blood specimen are used to compute the crash-death and death-specimen lag times, which pertain to two other criteria. Cases found ineligible at this point are removed from the system and filed separately.

As noted in steps 5 and 6 above, coroner/ME reports, police reports, and FARS reports typically arrived at Calspan months after the crashes to which they pertained. Necessarily, determination of case eligibility was delayed accordingly.

(9) The data are encoded. Information on the field documents is encoded into the study variables and recorded on code sheets. While much of this is straightforward, coding of driver responsibility and collision type require trained coders.

(10) The data are entered into an automated database. The data are keypunched and verified. Internal consistency checks are made, and any errors are corrected. The cases is identified by case number, with no identifying information entered into the database.

(11) Calspan implements identity protection. In this process, the hardcopy documents on the case are divided into two groups. In the first are all source documents (coroner/ME reports, PAR, etc.), from which the case identification numbers are obliterated. These are to be destroyed. In the second are all code sheets, which contain no names or other identifying information. Identity protection is now complete.

Upon the completion of these eleven steps for all cases, the data collection task ended.

Appendix E
Drug Assay Methodology

by P. Santinga and A. Constantino
American Medical Laboratories, Inc.

E.1 Specimens

Approximately 20 mL of whole blood preserved with 0.1% sodium fluoride was submitted in glass screw top tubes from the Medical Examiner's Offices participating in this study. A minimum of 6 mL was required for complete identification of the drugs present with additional volumes necessary for quantitation.

E.2 Testing Protocol

Specimens submitted were screened for alkaline, acid, neutral drugs and volatile substances. RIA was the screening procedure used for marijuana, PCP, LSD, cocaine metabolite, opiates, benzodiazepine, and amphetamines. This procedure complemented the combined alkaline/acid/neutral drug screen performed by gas chromatography/mass spectrometry. All specimens were also analyzed for the presence of ethyl alcohol by headspace analysis on a gas chromatograph with a FID detector as well as TDx ethanol assay (Abbott Laboratories, Illinois).

Specimens submitted with a volume of 5 ml or less were tested only by the RIA and alcohol procedures. A summary of the substances tested for, the analytical methods used and their sensitivity limits are listed in Table E-1.

E.3 Sample Preparation

10 mL of the whole blood was transferred to a screw cap polypropylene tube and frozen, then thawed at room temperature to lyse the red blood cells in order to provide a uniform matrix. The thawed specimen was centrifuged at 10,000/g for 10 minutes and 3 aliquots were prepared. One mL was required for the RIA screen and alcohol analysis. Four mL was then aliquoted for the alkaline/acid/neutral drug screen by GC-MS. Sample preparation, extraction methods and instrument conditions are detailed in Tables E-2 through E-7.

E.4 RIA Screening

The Roche Abuscreen Radioimmunoassay was employed for the initial screening of opiates, cocaine metabolite (benzoylecgonine) marijuana metabolite (11-nor-9-carboxy tetrahydrocannabinol), amphetamine, methamphetamine, phencyclidine and the benzodiazepines.

Diagnostic Products' Coat-A-Count® assay for LSD was also used. Manufacturers recommended procedures were followed for each. Modifications were made to the quality control material such that the reference standard was at the sensitivity limit described in Table E-1 and the positive control was concentrated at twice the sensitivity. A drug free control was also analyzed.

The THC procedure required a preliminary treatment of the blood to optimize recovery.

In this treatment, to 100 mcL of sample was added 100 mcL of cold (-10°C) acetonitrile in a microcentrifuge tube and vortex mixed. Optimal recovery was achieved when the acetonitrile was added slowly to the already vortexing specimen. The mixture was centrifuged at high speed for 5 minutes and 100 mcL of the supernatant was removed and placed in a polystyrene tube.

This tube was placed in a water bath at 40°C and evaporated under a stream of nitrogen (approximately 5 minutes). The kits' reagents were added directly to the residue. The manufacturer's protocol was followed from this point in the assay.

E.5 GC/MS Screen

A solid phase extraction using Worldwide Monitoring Corporation Clean Screen® extraction columns (ZCDAU020) was used to extract the acid, basic and neutral drugs which were included in this study.

To prepare the sample 6 ml of 100 mM sodium monobasic phosphate buffer adjusted to pH 5.5 with concentrated sodium hydroxide was added to 4 ml of the whole blood sample. 100 mcl of internal standard (Proadifen SKF 525A, 10 mcg/ml) was also added. The sample was centrifuged before applying to the column.

The column was prepared by aspirating 3 ml methanol, followed by 3 ml of deionized water using a vacuum-extraction apparatus.

The sample was applied to the column and aspirated at a rate of 1 to 2 ml per minute. The vacuum did not exceed 5-7 inches Hg. The column was washed by adding 2.0 ml of 0.1 M phosphate buffer followed by 0.5 ml of 1.0 N Acetic Acid. The column was then dried for 5 minutes under full vacuum of 15-20 " Hg. 1.0 ml hexane was added to the column, which was then aspirated.

The vacuum apparatus was prepared to collect the acid and neutral drug fraction. The acidic and neutral drugs were eluted by the addition of 4 ml of Methylene Chloride. The eluant was set aside to be added to the basic drug fraction later. The column was washed with 3 ml methanol before collecting the basic drug fraction. The basic drug eluant was prepared fresh by mixing 80 parts Methylene Chloride, 20 parts Isopropanol to 2 parts concentrated Ammonium Hydroxide. A 6 ml portion was added to the column and collected. The two eluants were combined in a conical centrifuge tube and evaporated to dryness under a stream of nitrogen in a 40°C water bath.

The extract was prepared for the GC/MS by adding 50 mcl 90% Ethanol and 500 mcl of Hexane. After vortexing and centrifuging, 2 mcl of the lower ethanol layer were injected into the GC/MS in the scan mode. Ion masses in the range of 40-450 amu were scanned. A drug and metabolite library search of all peaks was made along with a search of all base peaks of the drugs listed in Table E-1 at appropriate retention times. A summary of the GC/MS parameters can be found in Table E-2 and E-3.

E.6 Quality Control

The GC/MS screen used selective drugs at the sensitivity levels for the drug classes as the positive control; for example, amitriptyline representing the antidepressants, butalbital representing the barbiturates, etc. (See Table E-8). An internal standard was used in all extractions to insure individual extraction recoveries. Also a GC/MS verified negative blood control was used.

The Confirmation/Quantitation procedures used a high, low and negative control in most procedures. MultiShewhart Rules were used as acceptance criteria for the quality control. See Table E-8 for a complete list of quality control materials.

Table E1

Substances to be Assayed for, and Tests to be Used and their Sensitivities: for "Complete Assay" Specimens

Substance	Screening Test		Confirmation/Quantitation Test	
	Test Used	Sensitivity	Test Used	Sensitivity
Alcohol (ethanol)	GC/FID	0.005% w/v	GC/FID	0.005% w/v
Hallucinogens				
9 THC	RIA	13 ng/ml	GC/MS	1 ng/ml
Carboxy THC (metabolite)	RIA	13 ng/ml	GC/MS	2 ng/ml
Phencyclidine	RIA	12.5 ng/ml	GC/MS	2.5 ng/ml
LSD	RIA	0.5 ng/ml	HPLC	0.1 ng/ml
Benzodiazepine Tranquilizers				
Diazepam	RIA	100 ng/ml	GC/ECD	100 ng/ml
Nordiazepam (diaz. metab.)	RIA	100 ng/ml	GC/ECD	100 ng/ml
Lorazepam	RIA/GC–MS	50 ng/ml	GC/ECD	5 ng/ml
Flurazepam	RIA/GC–MS	50 ng/ml	GC/ECD	20 ng/ml
Desethylflurazepam (fluraz. metab.)	RIA/GC–MS	50 ng/ml	GC/ECD	20 ng/ml
Alprazolam	RIA/GC–MS	50 ng/ml	GC/ECD	5 ng/ml
Oxazepam	RIA/GC–MS	25 ng/ml	GC/ECD	20 ng/ml
Chlordiazepoxide	RIA/GC–MS	50 ng/ml	HPLC	100 ng/ml
Desmethylchlordiazepoxide (chlordiaz. metab.)	RIA/GC–MS	50 ng/ml	HPLC	100 ng/ml
Barbiturate sedatives				
Phenobarbital	GC–MS	1000 ng/ml	GC–MS	100 ng/ml
Secobarbital	GC–MS	1000 ng/ml	GC–MS	100 ng/ml
Butabarbital	GC–MS	1000 ng/ml	GC–MS	100 ng/ml
Butalbital	GC–MS	1000 ng/ml	GC–MS	100 ng/ml
Pentobarbital	GC–MS	1000 ng/ml	GC–MS	100 ng/ml
Amobarbital	GC–MS	1000 ng/ml	GC–MS	100 ng/ml
CNS Stimulants				
Cocaine	RIA/GC–MS	50 ng/ml	GC–MS	50 ng/ml
Benzoylecgonine (cocaine metab.)	RIA/GC–MS	50 ng/ml	GC–MS	50 ng/ml
Amphetamine	RIA	50 ng/ml	GC–MS	50 ng/ml
Methamphetamine	RIA	150 ng/ml	GC–MS	50 ng/ml
Caffeine	GC–MS	20,000 ng/ml	HPLC	20,000 ng/ml
Non–barbiturate sedatives				
Ethchlorvynol	Spectrophotometry	50 ng/ml	GC–MS	50 ng/ml
Methaqualone	GC–MS	50 ng/ml	GC–MS	50 ng/ml
Meprobamate	GC–MS	1000 ng/ml	Spectrophotometry	1000 ng/ml
Antihistamines				
Diphenhydramine	GC–MS	50 ng/ml	GC–MS	50 ng/ml
Chlorpheniramine	GC–MS	50 ng/ml	GC–MS	50 ng/ml
Antidepressants				
Amitriptyline	GC–MS	50 ng/ml	HPLC	5 ng/ml
Nortriptyline (amitrip. metab.)	GC–MS	50 ng/ml	HPLC	5 ng/ml
Imipramine	GC–MS	50 ng/ml	HPLC	5 ng/ml
Desipramine (imipramine metab.)	GC–MS	50 ng/ml	HPLC	5 ng/ml
Doxepine	GC–MS	50 ng/ml	HPLC	5 ng/ml
Desmethyldoxepine (dox. metab.)	GC–MS	50 ng/ml	HPLC	5 ng/ml
Fluoxetine	GC–MS	50 ng/ml	HPLC	5 ng/ml
Norfluoxetine	GC–MS	50 ng/ml	HPLC	5 ng/ml
Narcotic Analgesics				
Meperidine	GC–MS	50 ng/ml	GC–MS	50 ng/ml
Methadone	GC–MS	100 ng/ml	GC–MS	50 ng/ml
Propoxyphene	GC–MS	50 ng/ml	GC–MS	50 ng/ml
Norpropoxyphene (propox. metab.)	GC–MS	50 ng/ml	GC–MS	50 ng/ml
Oxycodone	GC–MS	50 ng/ml	GC–MS	50 ng/ml
Codeine	RIA/GC–MS	50 ng/ml	GC–MS	50 ng/ml
Morphine	RIA	50 ng/ml	GC–MS	50 ng/ml
Heroin	RIA	50 ng/ml	GC–MS	50 ng/ml

Table E1 Continued

Substance	Screening Test		Confirmation/Quantitation Test	
	Test Used	Sensitivity	Test Used	Sensitivity
Antipsychotics				
Chlorpromazine	GC/MS	100 ng/ml	HPLC	10 ng/ml
Thioridazine	GC/MS	100 ng/ml	HPLC	10 ng/ml
Mesordiazine	GC/MS	100 ng/ml	HPLC	10 ng/ml
Antiarrhytmics				
Quinidine	GC/MS	500 ng/ml	Immunoassay	100 ng/ml
Procainamide	GC/MS	500 ng/ml	Immunoassay	500 ng/ml
N–Acetylprocainamide (procainamide metab.)	GC/MS	500 ng/ml	Immunoassay	500 ng/ml
Lidocaine	GC/MS	100 ng/ml	Immunoassay	200 ng/ml
Flecainide	GC/MS	200 ng/ml	HPLC	100 ng/ml
Muscle relaxant				
Cyclobenzeprine	GC/MS	50 ng/ml	HPLC	10 ng/ml

Table E2
GC/MS Qualitative and Quantitative Procedures

Procedure	Extraction	Internal Standard/Conc	Derivatizing Agent
GC/MS Screen			
Acid, Basic and neutral drugs (qualitative)	Solid Phase$_1$ Column: 200 mg Clean Screen Worldwide Monitoring Corp. Horsham, PA (ZSDAU020)	Proadifen (SKF525A) Philadelphia, PA	No derivatization necessary
Opiates			
1. Morphine 2. Codeine	Solid Phase$_1$ Column: 200 mg Clean Screen Worldwide Monitoring Corp. Horsham, PA (ZSDAU020)	Deuterated (D3) Morphine, codeine (MSD isotopes, Merck & Co., Rahway, NJ., (# MD-3452, MD-3455) 1 mg/ml (500 mcl)	Pentafluoropropionic Anhydride (PFPA) Regis Chemical, Morton Grove, IL, (#640113)
Amphetamines			
1. Amphetamine 2. Methamphetamine	Solid Phase$_2$ Column: Chem Elut Varian (CE1005)	Deuterated (D3) Amphetamine, (D5) Methamphetamine Sigma Chemical Co., St. Louis MO (#A-0922, # M-2271) 1.4 mcg/ml (200 mcl)	4-Carbethoxyhexafluorobutyryl chloride PCR Inc. (#12357)
Cocaine			
1. Cocaine 2. Benzoylecgonine	Solid Phase$_1$ Column: 200 mg Clean Screen Worldwide Monitoring Corp. Horsham, PA (ZSDAU020)	Deuterated (D3) Cocaine, Benzoylecgonine Sigma Chemical Co., St. Louis, MO (#C-8162, #B-8021) 5 mcg/ml (200 mcl)	BSTFA with 1% TMCS Pierce Chemical Company Rockford, IL (#12357)
Marijuana			
1. Delta-9-THC 2. Carboxy D-9-THC	Liquid/Liquid	Deuterated (D3) D-9-THC, Carboxy D-9-THC Research Triangle Institute THC 50 ng/ml (300 mcl) THCA 250 ng/ml (300 mcl)	TMAH/DMSO, Iodomethane Sigma Chemical Company St. Louis, MO
Barbiturates			
1. Amobarbital 2. Butabarbital 3. Butalbital 4. Pentobarbital 5. Phenobarbital 6. Secobarbital	Solid Phase$_1$ Column: 200 mg Clean Screen Worldwide Monitoring Corp. Horsham, PA (ZSDAU020)	Tolybarbital Aldrich Chemical Company (#17,957-4) 25 mcg/ml (200 mcl) Milwaukee, WI	Trimethylanilinium hydroxide (TMAH) Pierce Chemical Company Rockford, IL (#49300)
Propoxyphene			
1. Propoxyphene 2. Norpropoxyphene	Solid Phase$_1$ Column: 200 mg Clean Screen Worldwide Monitoring Corp. Horsham, PA (ZSDAU020)	Proadifen (SKF525A) 10 mcg/ml (100 mcl) Philadelphia, PA	No derivatization necessary
Generic Basic Drugs			
1. Phencyclidine 2. Methadone 3. Meperidine 4. Diphenhydramine 5. Chlorpheniramine 6. Oxycodone	Solid Phase$_1$ Column: 200 mg Clean Screen Worldwide Monitoring Corp. Horsham, PA (ZSDAU020)	Proadifen (SKF525A) 10 mcg/ml (100 mcl) Philadelphia, PA	No derivatization necessary

F-GCMS

Table E3
Ions Monitored GC/MS Procedures

	Quantitation Ion	Ions Monitored		ISTD Ion
GC/MS Screen		40–100		86.1
Morphine:	414.2	577.2	430.2	580.2
Codeine	445.2	282.2	446.2	448.2
Amphetamine:	294.1	248.1	266.1	297.1
Methamphetamine	308.1	280.1	262.1	312.1
Cocaine:	182.1	272.1	303.1	185.1
Benzoylecgonine	240.1	346.1	361.1	243.1
D–9–THC:	313.1	328.1		316.1
Carboxy D–9–THC	313.1	357.1	372.1	331.1
Amobarbital	169.1	184.1		246.1
Butabarbital	169.1	184.1		
Butalbital	181.1	196.1		
Pentobarbital	169.1	184.1		
Phenobarbital	175.1	232.1		
Secobarbital	181.1	196.1		
Propoxyphene	58.1			86.1
Norpropoxyphene	44.1	234.1		
Phencyclidine	200.1	186.1	242.1	86.1
Methadone	72.1	165.1		
Meperidine	71.1	247.1		
Diphenhydramine	58.1	165.1		
Chlorpheniramine	203.1	165.1		
Oxycodone	303.1	196.1		

F-GCMS3

TABLE E4
GC PARAMETERS

ASSAY	INJECTION PORT (°C)	TEMP 1	TIME 1	RATE 1	TEMP 2	TIME 2	DETECTOR TEMP
Opiates[1]	250	170	0.1	15°C/min	280	2	280°C
Amphetamines[1]	170	100	0.1	30°C/min	200	2	280°C
Cocaine[1]	250	190	0.1	18°C/min	280	5	280°C
Marijuana[1]	250	235	2.2	18°C/min	290	6	280°C
Barbiturates[1]	250	100	0.1	15°C/min RATE 2 30°C/min	180 TEMP 3 290	0 TIME 3 5	280°C
Propoxyphene[1]	250	170	0.1	20°C/min	290	5	280°C
Alkaline Drugs[1]	250	150	0.1	20°C/min	280	5	280°C
GC/MS Acid,[1] Base, Neutral Screen (Qual.)	250	80	0.1	20°C/min	290	5	280°C
Volatiles[2]	80	80	---	---	---	---	160°C
Benzodiazepines	250	170	0.1	20°C	290	5	300

[1]Instrument/column: HP 5890GC with 5970B MSD/12.5 meter HP-5

[2]Instrument/column:. HP Headspace autosampler, Shumadzu Mini-2 GC/5% Carbowax 20M on 60/80 Carbopak 6'X 1/4" O.D. X 4 MM I.D.

[3]Instrument/column: HP 5880 GC/ECD/Rt-50 (50% methyl-50% Phenylpolysiloxane) 15 meters x 0.25 mm ID X 0.25 film thickness

TABLE3

HPLC Quantitative Procedures – TABLE E5

Procedure	Extraction	Internal Standard Concentration (amount used)	Mobile Phase	Column	Instrument/Wavelength/Flow
Tricyclics					
1. Amitriptyline 2. Nortriptyline 3. Imipramine 4. Desipramine 5. Doxepin 6. Desmethyl Doxepine (doxepin metabolite) 7. Cyclobenzeprine	Solid Phase, Column: Jet Tubes Harlin Associates (#1805) Gibsonia, PA	Haloperidol McNeil Pharmaceutical Spring House, PA (#17,957–4) 500 ng/ml (200 mcl)	63/27/1 Acetonitrile:Methanol (0.32% Heptane Sulphonic Acid/0.2% Acetic Acid) pH 5.8	Cyanopropyl 5 mcm 25 cm (#9525) Zorbax (#880952–705) Chadds Ford, PA	Waters Instrument Co. U.V. Detector 254 nm Milford, MA 1.5 ml/min
8. Fluoxetine 9. Norfluxetine	Solid Phase, Column: Jet Tubes Harlin Associates (#1805) Gibsonia, PA	Flecainide acetate 3M Pharmaceuticals St. Paul, MN	63/27/1 Acetonitrile:Methanol (0.32% Heptane Sulphonic Acid/0.2% Acetic Acid) pH 5.8	Cyanopropyl 5 mcm 25 cm (#9525) Zorbax (#880952–705) Mac–Mod Analytical Chadds Ford, PA	Waters Instrument Co. Fluorometer Detector Excitation wavelength – 227 nm Emission Filter 280 nm Milford, MA 1.5 ml/min
Antipsychotics					
1. Chlorpromazine 2. Thioridazine 3. Mesoridazine	Solid Phase, Column: Jet Tubes Harlin Associates (#1805) Gibsonia, PA	Trimipramine 500 ng/ml (200 mcl) Wyeth–Ayerst Philadelphia, PA	66.5/28.5//5. Acetonitrile:Methanol: (0.32% Heptane Sulphonic Acid/0.2% Acetic Acid) pH 5.8		Waters Instrument Co. U.V. Detector 254 nm Milford, MA 1.5 ml/min
Benzodiazepines					
1. Alprazolam	Solid Phase, Column:Bond–Elute C–18 Analytichem (#1210–2001) Harbor City, CA	Methylclorazepam Hoffman LaRoche 10 mcg/ml (800 mcl) Nutley, NJ	21/14/65. Acetonitrile:Methanol: (0.06 M Methane Sulphonic Acid 50 mM Triethylamine) pH 2.5 with 10 N	NOVA–PAK C18 Waters (#86344) Milford, MA	Waters Instrument Co. U.V. Detector 254 nm Milford, MA 1.5 ml/min
2. Chlordiazepoxide 3. Desmethylchlordiaxepoxid	400 mcg sample 400 mcg cold acetonitrile	Stock R07–9749 Hoffman LaRoche Nutley, NJ 10 mcg/ml (800 mcl)	KOH 28/66/6 Acetonitrile:0.833% Acetic acid: 0.32% Heptane Sulphonic Acid pH 5.8	N/A	Waters Instrument Co. U.V. Detector 254 nm Milford, MA 1.5 ml/min

HPLC Quantitative Procedures – TABLE E5 Continued

Procedure	Extraction	Internal Standard Concentration (amount used)	Mobile Phase	Column	Instrument/Wavelength/Flow
Hallucinogens					
LSD	Solid Phase, Column: Jet Tubes Harlin Associates (#1805) Gibsonia, PA	Ergotamine Tartrate Alltech–Applied Science State College, PA	35/65/06. Acetonitrile:2% Acetic Acid: Heptane Sulphonic Acid pH 5.6	Cyanopropyl 5 mcm 25 cm (#9525) Zorbax (#880952–705) Mac–Mod Analytical Chadds Ford, PA	Waters Instrument Co. Fluorometer Detector Excitation wavelength – 227 nm Emission Filter 280 nm Milford, MA 1.5 ml/min
Stimulants					
Caffeine	Methanol Precipitation 100 mcl Sample 200 mcl methanol	Beta–hydroxyethyltheophylline 4 mcg (200 mcl) S–15531 3M Pharmaceutical St. Paul, MN	35/65/06. Acetonitrile:2% Acetic Acid: Heptane Sulphonic Acid pH 5.6	Ultramex Cyanopropyl 15 cm (#00F–0050EOS) Phenomenex Co. Palos Verdes, CA	Waters Instrument Co. Fluorometer Detector Excitation wavelength – 227 nm Emission Filter 280 nm Milford, MA 1.5 ml/min
Antiarrhythmic					
Flecainide	Solid phase Jet Tube Harlin Associates (#1805) Gibsonia, PA	(n–(2–piperidylmethyl)–2, 5–bis (2,2,2–trifluoroethyl) benzamide hydrochloride Riker Laboratories 250 ng/ml/200	35/65/06. Acetonitrile:2% Acetic Acid: Heptane Sulphonic Acid pH 5.6	Beckman #244070 Ultrasphere Cyano–propyl stainless steel (15 cm)	Excitation 210 nm Emission filter 280 nm Milford, MA 1.5 ml/min

TABLE E6
Solid Phase Extraction GCMS

Procedure	Sample Size	Sample Preparation	Column Preparation	Column Wash	Sample Elution
GC/MS Screen					
Acid, Basic and neutral drugs (qualitative)	4 ml	6 ml 0.1M phosphate buffer Adjust pH to 6.0 with phosphate buffer	3 ml methanol 3 ml DI water 3 ml 0.1M phosphate buffer	Acidd & Neutral Drugs 3 ml DI water 0.5 ml Hexane Basic Drugs 3 ml Methanol	Acid & Neutral Drugs 4 ml Methylene Chloride Basic Drugs 6 ml Methylene Chloride Isopropanol Ammonium hydroxide (78:20:2)
Opiates					
1. Morphine 2. Codeine	2 ml	4 ml 0.1M phosphate buffer Adjust pH to 6.0 with phosphate buffer	3 ml methanol 3 ml DI water 3 ml 0.1M phosphate buffer	3 ml DI water 3 ml 1.0N acetate buffer pH 3.5 3 ml methanol	6 ml Methylene Chloride Isopropanol Ammonium hydroxide (78:20:2)
Amphetamines					
1. Amphetamine 2. Methamphetamine	2 ml	4 ml 0.1M phosphate buffer Adjust pH to 6.0 with phosphate buffer	3 ml methanol 3 ml DI water 3 ml 0.1M phosphate buffer	3 ml DI water 3 ml 1.0N acetic acid 3 ml methanol	6 ml Methylene Chloride Isopropanol Ammonium hydroxide (78:20:2)
Cocaine					
1. Cocaine 2. Benzoylecgonine	2 ml	4 ml 0.1M phosphate buffer Adjust pH to 6.0 with phosphate buffer	3 ml methanol 3 ml DI water 3 ml 0.1M phosphate buffer	3 ml DI water 3 ml 0.1N HCl 3 ml methanol	6 ml Methylene Chloride Isopropanol Ammonium hydroxide (78:20:2)
Barbiturates					
1. Amobarbital 2. Butabarbital 3. Butalbital 4. Pentobarbital 5. Phenobarbital 6. Secobarbital	1 ml	4 ml 0.1M phosphate buffer Adjust pH to 6.0 with phosphate buffer	3 ml methanol 3 ml DI water 3 ml 0.1M phosphate buffer	3 ml DI water 3 ml 1.0N acetic acid 0.5 ml Hexane	6 ml Hexane/Ethyl acetate (50:50)
Propoxyphene					
1. Propoxyphene 2. Norpropoxyphene	2 ml	4 ml 0.1M phosphate buffer Adjust pH to 6.0 with phosphate buffer	3 ml methanol 3 ml DI water 3 ml 0.1M phosphate buffer	3 ml DI water 3 ml 0.1N HCl 3 ml Methanol	6 ml Methylene Chloride Isopropanol Ammonium hydroxide (78:20:2)
Generic Basic Drugs					
1. Phencyclidine 2. Methadone 3. Meperidine 4. Diphenhydramine 5. Chlorpheniramine 6. Oxycodone	2 ml	4 ml 0.1M phosphate buffer Adjust pH to 6.0 with phosphate buffer	3 ml methanol 3 ml DI water 3 ml 0.1M phosphate buffer	3 ml DI water 3 ml 0.1N HCl 3 ml Methanol	6 ml Methylene Chloride Isopropanol Ammonium hydroxide (78:20:2)

F-GCMS-1

TABLE E7
HPLC Extractions

Procedure	Sample Size	Column Prep	Sample Elution	Back Extraction
Tricyclics				
1. Amitriptyline	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
2. Nortriptyline	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
3. Imipramine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
4. Desipramine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
5. Doxepin	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
6. Desmethyl Doxepine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
(doxepin metabolite)	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
7. Cyclobenzeprine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
8. Fluoxetine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
9. Norfluoxetine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4
Antipsychotics				
1. Chlorpromazine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 ml ethanol/0.01N H_2SO_4
2. Thioridazine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 ml ethanol/0.01N H_2SO_4
3. Mesoridazine	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 ml ethanol/0.01N H_2SO_4
Benzodiazepines				
1. Alprazolam	1 ml	1 ml methanol 1 ml DI water	400 mcl methanol	evaporate: reconstitute with 200 mcl acetonitrile/methanol/ 10 mM phosphate buffer 21:14:65
Hallucinogens				
LSD	3 ml	2 ml bicarbonate buffer pH 10.5	18 ml Hexane/ethyl acetate/isopropandl Isoamy alcohol 65:35:5:1	300 mcl 0.01N H_2SO_4
Antiarrhythmic				
Flecainide	1 ml	2 ml bicarbonate buffer pH 10.5	24 ml Hexane:Isoamyl alcohol (99/1)	400 mcl 0.01N H_2SO_4

F-HPLC-E

TABLE E8
Liquid/Liquid Extractions

Assay	Sample	Buffer	Extraction Solvent
Marijuana			
1. d-9-THC	2 ml	none	6 ml cold acetonitrile
2. d-9-Carboxy THC	2 ml	none	6 ml cold acetonitrile
Caffeine	100 mcl	none	200 mcl cold methanol
Benzodiazepines			
1. Chlordiazepoxide	400 mcl	none	800 mcl cold acetonitrile
2. Desmethylchlordiazepoxide	400 mcl	none	800 mcl cold acetonitrile
3. Diazepam	200 mcl	pH 10.5 bicarbonate buffer	Heptane/ethyl acetate 50/50
4. Nordiazepam	200 mcl	pH 10.5 bicarbonate buffer	Heptane/ethyl acetate 50/50
5. Flurazepam	200 mcl	pH 10.5 bicarbonate buffer	Heptane/ethyl acetate 50/50
7. Desalkylflurazepam	200 mcl	pH 10.5 bicarbonate buffer	Heptane/ethyl acetate 50/50

TABLE E9

QUALITY CONTROL QUANTITATIVE ASSAYS

	Procedure	Low	High
Tricyclics			
1.	Amitriptyline	100 ng/ml	400 ng/ml
2.	Nortriptyline	100 ng/ml	400 ng/ml
3.	Imipramine	100 ng/ml	400 ng/ml
4.	Desipramine	100 ng/ml	400 ng/ml
5.	Doxepin	100 ng/ml	400 ng/ml
6.	Desmethyl Doxepine (doxepin metabolite)	100 ng/ml	400 ng/ml
7.	Cyclobenzeprine	100 ng/ml	400 ng/ml
8.	Fluoxetine	100 ng/ml	400 ng/ml
9.	Norfluxetine	100 ng/ml	400 ng/ml
Antipsychotics			
1.	Chlorpromazine	100 ng/ml	400 ng/ml
2.	Thioridazine	100 ng/ml	400 ng/ml
3.	Mesoridazine	100 ng/ml	400 ng/ml
Benzodiazepines			
1.	Alprazolam	20 ng/ml	N/A
2.	Chlordiazepoxide	200 ng/ml	N/A
3.	Desmethylchlordiaxepoxid	200 ng/ml	N/A
Hallucinogens			
	LSD	0.8 ng/ml	N/A
Stimulants			
	Caffeine	10,000 ng/ml	N/A
Antiarrhythmic			
	Flecainide	200 ng/ml	N/A
Opiates			
1.	Morphine	100 ng/ml	300 ng/ml
2.	Codeine	100 ng/ml	300 ng/ml
Amphetamines			
1.	Amphetamine	100 ng/ml	600 ng/ml
2.	Methamphetamine	100 ng/ml	600 ng/ml
Cocaine			
1.	Cocaine	100 ng/ml	200
2.	Benzoylecgonine	100 ng/ml	200

TABLE E9 Continued

	Procedure	Low	High
Marijuana			
1.	Delta-9-THC	2 ng/ml	5 ng/ml
2.	Carboxy D-9-THC	10 ng/ml	20 ng/ml
Barbiturates			
1.	Amobarbital		
2.	Butabarbital		
3.	Butalbital		
4.	Pentobarbital	500 ng/ml	2,000 ng/ml
5.	Phenobarbital		
6.	Secobarbital		
Propoxyphene			
1.	Propoxyphene	200 ng/ml	500 ng/ml
2.	Norpropoxyphene	200 ng/ml	500 ng/ml
Generic Basic Drugs			
1.	Phencyclidine	25 ng/ml	250 ng/ml
2.	Methadone	275 ng/ml	1,500 ng/ml
3.	Meperidine	100 ng/ml	500 ng/ml
4.	Diphenhydramine	100 ng/ml	500 ng/ml
5.	Chlorpheniramine	100 ng/ml	500 ng/ml
6.	Oxycodone	100 ng/ml	500 ng/ml

GC/MS SCREEN
ACID, BASIC AND NEUTRAL
DRUGS (QUALITATIVE)

	Low	High
Butalbital	1,000 ng/ml	2,000 ng/ml
Nordiazepam	100 ng/ml	200 ng/ml
Cocaine	50 ng/ml	200 ng/ml
Chlorphyeniramine	50 ng/ml	200 ng/ml
Meperidine	50 ng/ml	200 ng/ml
Methamphetamine	100 ng/ml	200 ng/ml
Amitriptyline	50 ng/ml	200 ng/ml
Codeine	100 ng/ml	200 ng/ml

P-0003A

Appendix F
Quality Check Procedure for Blood Assays

Formal quality control of the assay procedures was carried out by American Medical Laboratories' (AML) internal quality control, as described in Appendix E. For this project, quality checks independent of AML were also made. The purposes of these checks were twofold: (1) to evaluate AML's success in detecting drugs of interest to the project; and (2) to assess AML's ability to accurately determine the drug concentrations. To keep the expense of these evaluations to a moderate level, only selected drugs of most interest were included.

The quality checks ultimately were implemented through two stages. Originally, the plan called for shipment to AML of test specimens, disguised as regular study specimens. These test specimens consisted of human blood spiked with prespecified concentrations of the selected drugs. The specimens were prepared by the Quality Service Assurance Corporation (QSAC) of Augusta, Georgia. After introduction of this procedure, however, we learned that spiking could not be done with great precision in the drug concentrations, hence it was not possible to adequately assess AML's reports in that regard. Consequently, a new stage of quality checking was introduced. In this second stage, the spiked test specimens were independently assayed by another independent laboratory, the Chemical Toxicological Institute (CTI) of Foster City, California. The assay results from AML and CTI were then compared.

The sections below describe the outcomes in the two stages.

F.1 Stage 1

It was specified in the project's study design that forty test specimens were to be prepared by an independent laboratory and submitted to AML for analysis. Six drugs (in varying combinations and concentrations) were included in the spiked samples: ethanol, cocaine, carboxy-THC (a cannabis metabolite), diazepam, benzoylecgonine (a cocaine metabolite), and phencyclidine. In the first three months of the project's data collection phase, twenty of the forty test specimens were prepared and then sent to AML via the Wisconsin study site. Once in Wisconsin, the samples were given Wisconsin case numbers and labels, and shipped in the normal shipping boxes to AML.

The samples were prepared in lots of five specimens, in which each of the five samples were to be identical. To check on AML's capabilities to detect weak concentrations, spiking was at trace levels, i.e. just above the detection thresholds, in half of the cases. To test AML's variance in measuring concentrations, spiking was at an intermediate concentration for the other half.

The results of this first stage of the quality control operation are presented in Table F-1. All of the spiked substances

Table F-1

Result of Assays of Test Specimens
by American Medical Laboratories:
Stage 1

No. Specimens	Substances	Intended Concentration	Reported Concentrations Mean	Reported Concentrations Std. Dev.
5	benzoylecognine	75 ng/ml	59.0	1.4
	phencyclidine	20 ng/ml	16.2	1.8
	ethanol (3 samples)	(Not intended)	0.006%	0.005
5	carboxy THC	50 ng/ml	59.2	4
	ethanol	0.01% w/v	0.01%	0
	diazepam	150 ng/ml	314	34
5	benzolecognine	250 ng/ml	195	15
	phencyclidine	100 ng/ml	89.4	2.1
	cocaine (3 samples)	(Not intended)	10.6	9.8
	ethanol (5 samples)	(Not intended)	0.02%	0
5	carboxy THC	250 ng/ml	266	27
	ethanol	0.10% w/v	0.07%	0
	diazepam	1000 ng/ml	878	75

were correctly identified, i.e., there were no false negatives. There were false positives however, in the specimens spiked with benzoylecgonine and phencyclidine. In three of these specimems, AML detected cocaine, a result of the assay process converting benzoylecgonine into cocaine (upon notification of this problem, AML took steps to correct this). Also, ethanol was detected in eight of the specimens spiked with benzoylecgonine and phencyclidine; in these cases however, AML was correct, because ethanol was in the solvent necessary for spiking with benzoylecgonine.

Regarding the issue of quantification, Table F-1 indicates that there were significant deviations of the mean concentrations from the intended concentrations of the test specimens. This problem was investigated, and it was determined that there were two important factors involved. One problem was the lack of a foolproof method of preparing test samples to precise specifications (i.e. it is difficult to spike the samples so that they contain exactly the intended amount of the substances). The second problem is a lack of information on how the substances break down (decay) over time. All of the samples were prepared at the same time and then shipped to the Wisconsin site over a period of eight weeks. It is possible that the concentrations declined over time due to this decay.

F.2 Stage 2

To deal with the problems encountered in the first quality control procedure, we revised our plan to include a third laboratory in the process. The Chemical Toxicological Institute (CTI) was chosen to assist in the quality control operations.

A total of thirty-three more test specimens were prepared by Quality Service Assurance Corporation. To ensure that the blood supply itself was not contaminated, the first specimen was substance-free (no drugs were added to the blood). This sample was sent to CTI for analysis and the results showed the blood supply to be clean. The other thirty-three specimens were prepared in four separate lots (eight in each lot), with five of the samples sent to AML and three sent to CTI. The samples were frozen prior to shipment to CTI and the Wisconsin site, and were sent from Quality Service Assurance Corporation using cold packs. Both of these steps were taken to reduce the possibility of decay.

The drug combinations, intended concentrations, positive detections, and reported concentrations by the laboratories are presented in Table F-2. The results were very satisfactory. AML identified all spiked substances except in one instance when spiked phencyclidine was found by AML at a level below their sensitivity limit, when they properly reported the substance as not detected. Interestingly, AML was always able to detect the carboxy-THC, while the confirmation laboratory was not. The variances in AML's reported concentrations also appeared satisfactory in all cases except with a series of diazepam concentrations, where the

Table F-2
Drug Assay Quality Check Results Summary:
Stage 2

Prepared Test Specimens			Assay Results: Chem. Tox. Inst.*			Assay Results: Amer. Med. Lab.*		
Test Lot #	Spiked Substances	Intended Concentration	# Specimens in which Detected	Reported Concentrations		# Specimens in which Detected	Reported Concentrations	
				Mean	Std. Deviation		Mean	Std. Deviation
1	Benzoylecgonine	75 ng/ml	3 out of 3	61.7	0.47	5 out of 5	81.2	4.49
	PCP	8 ng/ml	3 out of 3	6.9	0.90	4 out of 5	5.6	2.80
2	Carboxy THC	50 ng/ml	3 out of 3	Detected, unable to quantify	-	5 out of 5	51.6	5.5
	Ethanol	0.01 w/v	3 out of 3	0.01	0	5 out of 5	0.01	0
	Diazepam	150 ng/ml	3 out of 3	176.7	12.5	5 out of 5	120.6	23.8
3	Benzoylecgonine	250 ng/ml	3 out of 3	159.3	47.3	5 out of 5	142.4	13.9
	PCP	100 ng/ml	3 out of 3	79.3	0.5	5 out of 5	85.4	6.8
4	Carboxy THC	100 ng/ml	3 out of 3	Detected, Unable to quantify	-	5 out of 5	106	8.0
	Ethanol	0.10 w/v	3 out of 3	.09	0	5 out of 5	.086	.005
	Diazepam	1000 ng/ml	3 out of 3	1323	351.5	5 out of 5	1220	146.0

*From each lot, Chem. Tox. Inst. was provided with 3 test specimens and Amer. Med. Lab. was provided with 5 specimens. For each lab, there was a minimum interval of 1 week between shipment of individual specimens.

variability seemed excessive. The situation was improved when AML switched from gas chromatography to liquid chromatography, but the variances in their readings also seemed due in part to an actual deterioration of the concentrations of diazepam in the series of spiked specimens.

APPENDIX G
Responsibility Analysis Methodology

Table Of Contents

CRASH RESPONSIBILITY RATING

This manual tells you how to rate driver crash responsibility, using accident reports and a rating scale. In rating a driver's responsibility for a crash, you will answer the question: To what extent is this crash due to the driver? A driver is "responsible" for a crash when something she/he did or did not do helped to cause the crash. Examples of crash-causing driver actions or inactions are the following:

- The driver lost control of his/her vehicle.
- The driver failed to perceive something, such as a stop sign.
- The driver misjudged something, such as the speed of an approaching vehicle.
- The driver went through a red traffic signal.

It is very important that you understand that in rating responsibility, you are not judging fault, guilt, blame-worthiness, culpability, or making any other moral or legal attribution of wrongdoing. To clarify the difference between responsibility and the other concepts, consider a crash in which the driver had a heart attack, and his vehicle went off the road into a tree. In this case, the driver was "responsible" for the crash (he lost control of the vehicle), but you probably would not consider the driver to be at fault. Of course, sometimes a driver may be responsible for a crash and legally at fault too, but basically the concepts are different, and you must concentrate on understanding responsibility as it is defined here.

The scale you will use was developed from earlier versions (Perchonok, 1978, Terhune 1983). It is shown in Table G-1. In rating responsibility with this scale, you basically judge whether the driver was fully responsible for the accident, contributory to the accident, or not at all responsible. Because information on a crash may sometimes be inexact or ambiguous, you are also allowed to specify "responsible or contributory" or "contributory or neither". Thus, in rating a driver's responsibility, you have a choice among five responses, which makes the scale a 5-point scale.

Table G-1

The Scale of Crash Responsibility

(4) Responsible -- Actions of the subject driver-vehicle created the critical situation.

(3) Responsible/contributory -- Driver had some responsibility, but it is not clear whether he was responsible or contributory.

(2) Contributory -- Another vehicle or agent created the critical situation, but the subject driver could have avoided the crash by a normal evasive maneuver or by driving defensively or by giving a warning signal (e.g., horn, flashers)

(1) Contributory/neither -- At most, the driver's responsibility was only contributory.

(0) Neither responsible nor contributory -- Driver had no responsibility for the accident.

(8) Unknown -- Information is insufficient for rating responsibility. Score this when choice is between full responsibility and none. Use rarely.

Definitions

Agent -- The precipitator, animate or inanimate, of an event; may be another vehicle, a person (e.g., pedestrian), an animal, or a natural phenomenon such as a tree falling on the road.

Critical situation -- A condition in which a crash is imminent, though it may still be avoidable. (Note: Lack of defensive driving does not in itself define a critical situation.)

Defensive driving -- Driving so as to minimize chances of a critical situation developing. Consists of maintaining alertness, anticipating possible hazards, taking precautionary actions. Examples are: sounding one's horn when a vehicle encroaches on one's travel lane; slowing and watching for crossing vehicles at a yellow blinker light; slowing when a pedestrian appears about to cross the street.

DRIVER RESPONSIBILITY: CLARIFICATION OF ITS MEANING

By "responsibility" we mean that the driver's behavior was a causal factor in the the accident; the behavior includes what the driver did do and did not do, i.e., responsibility can be the result of an action taken or not taken, a perception made or not made. A driver will be responsible as the result of any influence "internal" to the driver, i.e., a physical or mental event or condition. Examples are a perception, a heart attack, fainting, anger, excitement, and a judgment.

Note that our working use of the responsibility concept, by excluding culpability, blame, and similar considerations, also omits considerations of motivation and intent. You are not to judge whether the driver was deliberately risk-taking or showing careless disregard of safety. While such driver attitudes may have influenced the behavior causing the accident, it is the behavior and not the underlying attitude that makes the driver responsible.

It may help you to think of the driver as an organism or mechanism in the human-machine system comprising the driver and the vehicle. If a crash occurred because the driver-mechanism failed to perform as it's supposed to, then the driver-mechanism is responsible for the crash. To function safely, the driver-mechanism must follow certain rules or programs, represented by traffic laws, "rules of the road" specified in state driver manuals, and safe-driving practices taught in driver education. The responsibility coder should have a sound understanding of these safe-driving rules, for it is a driver's deviation from the rules which make him/her responsible for an accident.

The Driver vs. External Agents

Responsibility for a crash is divided among the driver you are rating (the subject driver) and all other agents "external" to the driver. The external agents may include:

- other driver-vehicles
- other occupants of the subject driver's vehicle
- the subject driver's own vehicle
- features of the roadway environment
- pedestrians
- animals

In judging the responsibility of the subject driver, you consider how much responsibility is shared by external agents. Clearly, if an external agent is primarily responsible for the crash, the subject driver can at most be considered "contributory." Usually, the role of an external agent is easily determined, but occasionally fine distinctions have to be made. For example, if a driver crashed when a painful bee sting caused a sudden disruption of vision, little or no driver responsibility would be assigned,

because the bee was an external agent. If, on the other hand, the driver crashed because a sudden illness caused a loss of vision, the driver would be assigned full responsibility, because the influence was "internal" to the driver and not "external." Fine distinctions will also have to be made when there are distractions to the driver, such as children inside the car. If the driver allows herself/himself to be distracted, then the driver must be assigned responsibility for a resulting crash. If the distraction was so intrusive that the driver could not ignore it, then the driver may bear little or no responsibility for the crash. You will have to make a judgment in the individual case.

Responsibility Through Compensation Failure

As you know, weather conditions, traffic volume, and other environmental circumstances can make driving more hazardous. For a crash in such circumstances, however, you should not automatically reduce the driver's responsibility rating because of these "external agents." The driver's internal "program" should tell the driver to compensate for conditions, such as by slowing down and increasing alertness. Two important considerations here are (a) foreknowledge and (b) normal precautions. For a driver to be judged responsible through compensation failure, we must assume he/she had foreknowledge of the conditions, yet failed to take normal precautions. Such assumptions must have reasonable grounds, e.g., an icy road must be readily visible, or a deer-crossing area must be clearly posted. Sometimes, however, a driver may have no foreknowledge of a road hazard, as in the following examples:

- an isolated icy patch
- a rock that tumbled into the road
- a deer darting into the road where there are no deer-crossing signs
- a sudden snow-squall obscuring vision

In such cases, the driver will have little or no responsibility for a crash that occurs. The coder will have to evaluate the circumstances in the individual case.

HOW TO RATE CRASH RESPONSIBILITY

In order to rate a driver on his/her crash responsibility, you first need to learn what happened in the crash, and then you have to judge why it happened on the basis of the facts in the crash file. In order to do this, you will find it best to examine the following documents, if present in the case file, in the order shown:

- scene diagram - This will provide a bird's eye view of the accident, and it may indicate the sequence of events during the crash.

- police narrative - Since the reporting officer was at the scene, and may have interviewed witnesses of the crash, this may be your best objective description of how the crash happened and why.

- witness reports - If present in the crash file, witness reports can provide useful evidence on what happened and perhaps even why. Witnesses are fallible, of course, but they can provide valuable corroboration (or noncorrobation) of details from other sources. Unfortunately, witnesses are not present in many crashes. Sometimes a police report will merely report the presence of witnesses without actually providing their statements. In these cases, you'll have to assume that the police narrative took into account the witness reports.

- driver interviews - If there are interviews of one or more of the drivers in the crash, these can provide the only source of what the driver(s) was actually doing before and during the crash. You need to be cautious here, for drivers in the crash may provide a biased interpretation of the events.

Rules of Thumb for Responsibility Rating

The next section will provide guidance for rating responsibility in particular crash situations, but there are a few general rules of thumb which you should keep in mind while rating responsibility. These are as follows:

(1) When you have information on what the driver did, take that into account in rating the driver's responsibility. If you do not have information what the driver did, rate the driver's responsibility on the basis of the vehicle's actions leading to the crash. (For example, it is often possible to assign responsibility when knowing a vehicle went through a red light or stop-sign.) This rule will often be useful in rating the responsibility of fatally injured drivers.

(2) Do not assume that a driver must be able to handle every situation; an external agent may assume part or all of the responsbility for a crash.

(3) If a driver attributes crash responsibility to an external agent, do not accept the driver's report unless there is supporting evidence, such as a witness report.

(4) Try to avoid biasing assumptions about crash responsibility on the basis of what you learn about the driver's age, gender, driving experience, vehicle type, or even alcohol use. Code strictly on the basis of the driver's and/or vehicle's actions.

(5) Give special attention to witness reports, including police mention that there was a witness. These add credibility to the police account of the crash, even if the officer does not actually cite the witness's statement.

Guidelines for Specific Situations

Crash patterns which occur commonly are described here. While you may expect to assign responsibility generally as suggested here, there can be exceptions.

Single-driver accidents. In single-vehicle crashes, and crashes of a vehicle with a parked vehicle, you should assume that the driver was fully responsible for the crash, unless an external agent assumed some or all of the responsibility. For example, if the subject driver lost control of his/her vehicle while trying to avoid a crash with another, the other vehicle may bear some of the responsibility for the crash. Other external agents may be pedestrians, animals, the driver's own vehicle (if it failed in some way), or an environmental factor. Caution: when there is no evidence of the presence of an external agent except the driver's own report, do not accept the driver's report as credible unless there is plausible, substantiating detail in the driver's story. Generally, the driver's report should have some corroborating evidence, such as witness reports, a dead animal at the roadside, a vehicle part left at the scene.

Left-turn accidents. A fairly common accident is one where a vehicle making a left turn collides with another vehicle on the same road coming in the opposite direction. Typically, the left-turning vehicle cuts across the path of the oncoming vehicle and is struck by it. The driver of the turning vehicle is usually judged responsible, because a left-turning vehicle is required to yield the right-of-way to oncoming vehicles before completing the turn.

Rear-end crashes. A driver is generally held responsible when his/her vehicle runs into the rear of a vehicle ahead on the same road, for drivers are expected to be vigilant in observing traffic

ahead, and maintain enough distance behind other vehicles to be able to stop safely should the vehicle ahead stop suddenly. An exception to this general rule would the case where the vehicle run into was stopped in the road, without lights, in darkness.

Head-on crashes. Generally, responsibility in head-on crashes is given to the driver of the vehicle which crossed the road centerline. If the collision occurred right on the centerline, or if it is unclear which vehicle crossed the centerline, each driver is considered contributory.

Stop-sign accidents. In intersection collisions where one vehicle had a stop-sign (or yield-sign) and the other did not, the driver-vehicle with the stop-sign is generally assigned responsibility for not yielding the right-of-way to the vehicle on the through-street. In the case of intersections with stop-signs on all approaches, the vehicle reaching the intersection first has the right-of-way. When two vehicles reach an intersection at the same time, the vehicle to the right has the right-of-way. Responsibility is generally assigned to the vehicle which violated the right-of-way of another, but sometimes a driver with the right-of-way may be held partially responsible for failing to show reasonable caution.

Vehicle mechanical failures. A driver usually is not judged responsible if the crash was due to mechanical failure of the vehicle. A driver's claim of vehicle failure is not to be considered valid, however, without some supporting evidence of the failure. This may sometimes be found in the police report of the accident.

Sun glare and other visibility interferences. Drivers in accidents occasionally attribute the crash to their being partially blinded by sun glare. In these cases, you should first look for plausibility of this claim: use the police-reported time of day, weather condition, and direction the driver's vehicle was facing to see if they are consistent with the possibility of sun glare. Second, determine whether the driver made a reasonable choice of action under the circumstances. Similar considerations arise in the case of a vision-obscuring downpour. A driver who cannot see adequately who chooses to make a turn or change lanes takes a high risk, and should be assigned responsibility for any ensuing crash. Generally, the driver with a visibility interference should either proceed very cautiously or move carefully to the side of the road, or not move if already stopped.

REFERENCES

Perchonok, Kenneth (1978). Identification of Specific Problems and Countermeasures Targets for Reducing Alcohol Related Casualties. U.S. Department of Transportation, National Highway Traffic Safety Administration, Report DOT-HS-803716.

Terhune, Kenneth W. (1983). An evaluation of responsibility analysis for assessing alcohol and drug crash effects. Accident Analysis and Prevention, 15, 237-246.

LEARNING TO RATE RESPONSIBILITY

Learning to rate responsibility consistently with experienced coders has been found surprisingly easy. To achieve proficiency, the coder needs to (a) study this manual, and (b) practice rating cases, comparing one's own ratings with those of experienced raters. To facilitate practice, descriptions of 50 varied crashes are provided in Appendix A. Because of space limitations, these cases are not nearly as thorough as the case documents of a professional accident investigation, but rating drivers in these cases will provide a valuable initial lesson.

In our experience, merely practicing on a diverse set of 50 to 100 crash cases helps the first-time rater to learn to distinguish the levels of responsibility that drivers experience in actual crash situations. It is recommended, consequently, that the rater practice on actual police reports or on case records of professional accident investigators. It is essential that those records include at least a narrative of the crash events, and preferably an accident scene diagram showing the paths and positions of the crash vehicles.

APPENDIX G-1: PRACTICE CASES

Note: The following narratives are quoted from actual police accident reports, with identifying information changed. The amount of detail provided is typical of a police accident report. An in-depth investigation (often provided in fatal accidents) will of course provide more detail. Each case is preceded by 4 pieces of information that may be helpful in rating the case: number of vehicles involved, time of day, weather/road conditions, and location description. Record your rating in the space provided. Ratings of original coders for these cases follow the entire set of 50.

Legend: Veh = Vehicle
Oper = Operator

1) 1 veh 3:06 AM clear/dry suburban intersection

1 was southbound on Smith St when (according to skid marks) the driver lost control and swerved from the west lane into the east lane. The veh crossed the Jones Ave intersection, swerving back into the west lane, then off the road into a retaining fence, tearing out approximately 75 feet of the fence before coming to rest across the west lane of Jones Ave south.

Rating Veh 1 _____

2) 2 veh 4:30 PM clear/dry urban intersection

Veh 1 being operated north on Cooper Rd when at Harrison Ave with the front end collided against the rear of Veh 2 being operated north on Smith. Driver 1 states he looked down and when he looked up it was too late.

Rating Veh 1 _____

3) 1 veh 4:43 AM clear/dry suburban 2-lane rd

According to operator of car who related that she was traveling north on Perry Ave, "Suddenly a dog ran out in front of me. I swerved to avoid the dog and swerved to the right and struck a pole."

Rating Veh 1 _____

4) 2 veh 12:10 AM clear/dry urban intersection

Veh 1 being operated in northerly direction on Main St and when at the intersection of Stanford Ave, with the front end of veh collided with the left side of Veh 2 which had pulled into the path of Veh 1 and attempted to negotiate a left turn.

Rating Veh 1 _____

5) 2 veh 9:00 PM snowing/icy urban intersection

Oper of Veh 2 states that while northbound on Ellen St and approaching the intersection at Mary Ave, she did apply her brakes and her veh skidded into Mary Ave. Oper of Veh 2 states she was attempting to stop for the stop sign. Oper of Veh 1 was westbound on Mary Ave at the intersection of Ellen St when Veh 2 skidded into his path. Vehicles collided. Veh 2 also hit a house at 123 Mary Ave.

Rating Veh 2 _____

6) 1 veh 6:00 PM clear/dry urban intersection

Veh 1 being operated west on Culpepper Ave, at the intersection driver did attempt to avoid a defective barricade and struck a light pole. Reflector on barricade defective, nonvisible.

Rating Veh 1 _____

7) 2 veh 12:45 PM clear/dry suburban 4-lane rd.

According to witness who was northbound on Main St ahead of Veh 2, Veh 1 was being operated southbound in the northbound lane and narrowly missed the witness veh prior to striking Veh 2. Witness further stated that the operator of Veh 1 appeared to be asleep as his head was resting against the driver's window and his eyes appeared to be closed. Operator of Veh 1 apparently suffered a stroke as upon arrival of ambulance he stated that his left side was numb.

Rating Veh 1 _____

8) 1 veh 3:23 PM raining/icy urban bridge

Veh 1 eastbound over Johnson Rd on bridge. Driver observed a veh ahead of her slow for an accident. She applied her brakes and went into a skid on the ice-covered bridge. She lost control and struck the guardrail.

Rating Veh 1 _____

9) 2 veh 2:15 AM cloudy/dry urban intersection

Veh 1 being operated in southerly direction when at Jones Ave with right front of veh collided with rear of Veh 2 which was parked at the curb on Smith St. Owner of Veh 2 was under the vehicle attempting repairs. Oper 1 states she did not see Veh 2 (no lights or flashers operating on Veh 2).

Rating Veh 1 _____

10) 1 veh 2:13 AM clear/dry suburban intersection

Driver 1 stated he was north on Johnson Rd when he fell asleep. According to Driver 1, he awoke and found the veh to be at the "Y" intersection of Johnson and Peterson Rds. He attempted to drive off the grass but lost control. The car slid into the light pole which stopped Veh 1.

Rating Veh 1 _____

11) 2 veh 3:47 PM clear/dry urban intersection

Operator of Veh 2 stated that she was southbound on Donovan St and was stopped for the red light on Donovan at the intersection of Patrick Ave. Operator of Veh 2 stated that while she was stopped, her veh was struck in the rear by Veh 1. Operator of Veh 1 stated that she was proceeding southbound on Donovan and when she attempted to stop, the brakes failed. Officer checked Veh 1; no brakes.

Rating Veh 1 _____

12) 2 veh 12:35 PM cloudy/wet urban intersection

According to the operator of Veh 2 who was traveling north on Connecticut St, he was about to turn left onto Maryland Ave and had the green light and it changed to yellow. He said that as he was entering the intersection, he observed another southbound motorist - Veh 1 - sliding toward him, so he stopped his veh and was struck by Veh 1. Oper of Veh 1 related that he had the green light, entered the intersection and Veh 2 made a left turn in front of him and did not stop at all.

Rating Veh 2 _____

13) 1 veh 1:40 PM rain/wet urban intersection

Veh 1 was going west on Greer Ave and when at the intersection of Constitution Ave, the driver in stopping his motorcycle, slipped and lost control of it. Driver fell down with cycle and hit the pavement.

Rating Veh 1 _____

14) 3 veh 3:05 PM cloudy/dry urban 2-lane rd

Veh 3 stopped at a red light, Veh 2 coming to a stop behind Veh 3. Driver of Veh 1 behind Veh 2 was distracted when a cigarette fell inside Veh 1. When driver reached down for cigarette, Veh 1 struck Veh 2 and then Veh 2 struck Veh 3 causing a chain reaction.

Rating Veh 1 _____

15) 1 veh 12:50 AM cloudy/wet urban expressway

Driver of Veh 1 states that while attempting to exit crossway at Peterson Rd, an unknown small vehicle cut in front of her vehicle, causing her veh to swerve from the middle lane past the left lane striking the guardrail. Veh 1 left the roadway, flipped over perpendicular to the roadway and slid eastbound on top of the guardrail. While still flipped over on its top, Veh 1 then struck and knocked over a light pole and came to a stop. Both the driver and the passenger were trapped inside the veh and had to be freed. Both witnesses gave accounts of the accident while driving in front of Veh 1. Statements supported Driver 1's statements and officer's investigation at scene.

Rating Veh 1 _____

16) 2 veh 11:05 PM cloudy/dry urban intersection

Veh 1 going west on Peterson Rd when at the intersection of Johnson Ave and with the front end collided with Veh 2 striking the left front quarter. Veh 1 then left the roadway and struck a tree on the southwest corner. Veh 2 left the roadway and struck a tree on the northwest corner.

Rating Veh 1 _____

17) 2 veh 5:45 PM cloudy/dry urban 2-lane rd

Oper of Veh 2 states as she stopped for traffic ahead of her on Johnson Ave in the area of Flower Park, she was hit in the rear by Veh 1. Oper 1 states she was traveling east in the curb lane in this area and passed into the passing lane and as she did, Veh 2 stopped suddenly. Oper Veh 1 states she attempted to stop but could not and hit Veh 2.

Rating Veh 2 _____

18) 2 veh 4:05 PM cloudy/wet suburban 2-lane rd

Veh 1 operating north on Main St when at about 1234 Main, Veh 1 struck a center median curb causing veh to go out of control and collide with Veh 2 also going north on Main. Oper of Veh 1 states she didn't observe the median curb sticking out as her passengers were distracting her.

Rating Veh 1 _____

19) 2 veh 11:39 AM clear/dry suburban intersection

Oper 1 stated he was driving westbound on Carson Ave when Veh 2 pulled in front of his veh. Oper 2 stated she was traveling eastbound on Carson and began to make a left turn from Carson onto Main St. Oper 2 stated she turned across the eastbound lane of Carson and did not observe Veh 1. Witness stated that Veh 2 pulled

in front of Veh 1, both vehicles struck each other, and Oper 1 was thrown to the pavement.

Rating Veh 1 _____

20) 2 veh 1:10 PM clear/dry urban interchange

Driver 2 stated he was exiting Highway 55 onto Highway 99, stopped at the stop sign, and pulled ahead slowly to see if it was clear to proceed when Veh 1 struck him from behind. Driver 1 stated he was exiting Highway 55 onto Highway 99 and was stopped at the stop sign behind Veh 2, and started up when he thought Veh 2 had started, colliding with Veh 2.

Rating Veh 2 _____

21) 1 veh 3:00 PM clear/dry suburban driveway

Driver 1 stated that he was backing down his driveway and his veh kept stalling. When the veh made it to the roadway, the passenger stated that she would hold the gas pedal down with her foot while the driver braked and shifted. The passenger hit the gas too hard, the driver could not control the veh, and the veh went off the road, striking a mailbox and a tree.

Rating Veh 2 _____

22) 2 veh 6:35 AM clear/dry suburban intersection

Driver 1 said he was southbound when Veh 2 made a left turn in front of him. Veh 1 left 57 feet of skid marks before striking Veh 2. Driver 2 said that Veh 1 was driving without lights at a high rate of speed (accident occurred early morning before sunrise). This was confirmed by both witnesses. It is a 40 MPH zone. Driver 2's view was obstructed by a slight rise in the road also. Veh 2 was struck in the right rear in the middle of southbound lane.

Rating Veh 2 _____

23) 2 veh 8:56 PM cloudy/dry suburban curve

Veh 1 traveling north, failed to negotiate a curve and struck Veh 2 parked on east side of roadway. Veh 2 unattended and parked off highway. Operator of Veh 1 states she did not see curve in roadway.

Rating Veh 1 _____

24) 4 veh 2:00 AM cloudy/dry urban 4-lane hwy

Vehicles 1, 3, and 4 (police vehicles) were westbound chasing Veh 2, also westbound. Driver 2 attempted to make a left turn and Driver 1 hit Veh 2 on the driver's side to stop him. This caused Veh 2 to spin around and strike a utility pole. Veh 3 came up behind Veh 2, and Veh 2 backed up into Veh 3's front end. Driver 2 was trying to get away. Veh 4 came alongside of Veh 2 and Vehicles

1, 3, and 4 pinned Veh 2 at the pole.
Rating Veh 3 _____

25) 2 veh 1:30 AM clear/dry suburban 4-lane hwy

Driver 2 stated that Veh 1 crossed into southbound lane. Driver 2 moved right to avoid collision but Veh 1 kept coming and struck Veh 2. Witness John Feldman stated he observed Driver 1 slumped over the wheel before the collision as if the driver had fallen asleep. Driver 1 could not recall what had happened. She was taken to the hospital by her father. Veh 1 continued on after impact with Veh 2, jumped the curb and took down mail boxes belonging to Jane Shoe.
Rating Veh 1 _____

26) 1 veh 6:36 PM cloudy/wet urban 2-lane rd

Veh 1 was southbound on Faith St when it crossed over the northbound lane and struck a tree. The driver does not remember what happened prior to the collision. The passenger states that the driver tried to avoid a parked vehicle then lost control of the vehicle.
Rating Veh 1 _____

27) 2 veh 2:00 PM clear/dry urban intersection

Veh 1, being operated north on Barbara St, when at Dooley Ave, with front of veh did collide with rear of Veh 2, stopped, preparing to make an illegal left turn. Oper of Veh 1 states he was watching a vehicle to his right and when he looked forward, Veh 2 was there.
Rating Veh 2 _____

28) 2 veh 3:52 PM clear/dry urban intersection

Veh 1 proceeding northbound in the passing lane on Muldoon St. Veh 2 westbound entered the intersection of Muldoon St and O'Leary Ave in the path of Veh 1. Veh 1 struck Veh 2. Driver 2 failed to yield the right-of-way to Driver 1.
Rating Veh 1 _____

29) 2 veh 7:15 PM clear/dry urban/parking lot

Veh 1 southbound in the rear parking lot of the grocery store with its front end struck the front end of Veh 2 which was parked. Owner of Veh 1 is a witness to the accident and states that the driver appeared to have lost control.
Rating Veh 1 _____

30) 2 veh 12:48 AM rain/wet urban intersection

Veh 1 reported to be northbound on Dover St. Veh 2 crossed Dover from Michael Ave. Veh 1 struck Veh 2, Veh 2 was spun around and stopped facing south, Veh 1 was pushed onto the center median and struck a sign. Driver 2 ejected from vehicle.

Rating Veh 1 _____

31) 1 veh 12:14 AM clear/dry suburban 4-lane hwy

Veh 1, driving east on Lincoln Ave, left the roadway and struck a light pole. Driver of veh stated she and driven a long distance and had fallen asleep at the wheel.

Rating Veh 1 _____

32) 2 veh 6:55 PM clear/dry urban intersection

Veh 1 struck Veh 2 when Driver 1 sped up to make the yellow light while Driver 2 was making a left turn during the yellow light.

Rating Veh 2 _____

33) 1 veh 3:15 PM clear/dry urban 2-lane rd

Veh 1 (motorcycle) did strike a pothole with front wheel, causing driver to lose control. The veh then fell on its side.

Rating Veh 1 _____

34) 1 veh 11:54 AM clear/dry suburban 2-lane rd

Driver 1 stated that a red Pinto stopped fast in front of her. She knew she could not stop, drove into a yard, slid sideways about 75 feet, and struck a tree. Other vehicle's actions, veh unknown. Witnesses: Martha Eldridge, Kevin Healy.

Rating Veh 1 _____

35) 2 veh 7:00 AM cloudy/dry urban 6-lane hwy

Veh 1 collided with Veh 2 causing damage. Oper of Veh 1 stated she signalled to change lanes and did not see Veh 2. Oper of Veh 2 states he was going straight and suddenly Veh 1 changed lanes, striking him.

Rating Veh 1 _____

36) 2 veh 9:05 AM rain/wet urban intersection

Driver 1 states he was operating north on Broadway and did not see the stop sign directing his flow of traffic. Veh 2 was operating west on Jennifer Ave and with front, Veh 1 collided with left side of Veh 2 at the intersection.

Rating Veh 2 _____

37) 1 veh 5:15 PM cloudy/dry rural 2-lane hwy

Veh 1 with front end struck a tree off roadway. Oper 1 remembers sneezing before the accident and nothing else.

Rating Veh 1 _____

38) 2 veh 4:59 PM clear/dry suburban plaza

Veh 1 was eastbound through plaza. Driver stated that he went to flick ashes in the ashtray and momentarily took his eyes from the road. Veh 2 was northbound through plaza and was struck by Veh 1.

Rating Veh 2 _____

39) 1 veh 9:55 AM clear/dry urban RR crossing

Veh 1 was northbound on State St on the railroad crossing when the vehicle's front end dropped down and struck the railroad tracks. The veh dropped down due to the crossing grade being uneven as the crossing is in stages of construction. When the veh struck the tracks, Driver 1 struck her face - causing swelling, bleeding, and loss of teeth. Also injured knee. The passenger struck her head on the windshield, cracking it.

Rating Veh 1 _____

40) 2 veh 5:12 PM rain/wet suburban 4-lane hwy

Driver of bus (Veh 2) stated he was stopped at a bus stop letting off passengers. The 4-way flashers were operating and then he was hit from behind. Said bus was facing north in curb lane in front of a city bus stop. Driver of Veh 1 stated he was northbound on Smith St in the curb lane. He observed the stopped bus and tried to go around it. He could not make it because of a car in the passing lane. He made contact with the bus.

Rating Veh 1 _____

41) 2 veh 4:25 PM clear/dry rural intersection

Driver of Veh 1 was northbound on Harrison St approaching Westminster Ave when he observed Veh 2 eastbound on Westminster approaching Harrison St. Driver of Veh 1 stated Veh 2 slowed to about 15 MPH and then continued across Harrison St without stopping

at the stop sign. Driver of Veh 2 stated she did not remember what happened and saw no stop sign. The below named witness was driving directly behind Veh 1 and said he saw Veh 2 slow down but then go through the stop sign.

Rating Veh 2 _____

42) 1 veh 2:20 PM clear/dry urban 4-lane hwy

Veh 1 being operated south in the northbound curb lane did mount curb and continue southbound striking gas pumps located approximately 20 feet from the east curb. As a result of the collision the gas pumps were knocked from the pump island causing fire. Oper of Veh 1 states that she was operating southbound on Main St when the gas pedal became stuck. After attempting to pull the pedal from the floor with her foot and unable to do so, she pulled on the emergency brake in an attempt to stop the bus. She was unable to stop the bus and constantly gaining speed and overtaking vehicles in the southbound lane. Oper did observe a veh operating in the northbound lane and did pull out to avoid this veh. After passing the veh, Oper did return to the curb lane. After passing through the intersection at Main St and Jackson Ave, Oper did mount the curb and strike the gas pumps located approximately 20 feet from the highway.

Rating Veh 1 _____

43) 2 veh 6:55 PM cloudy/dry urban intersection

Veh 1 was northbound on Keifer St approaching the Getzel Ave intersection. Driver 2 was westbound on Getzel Ave and attempted to turn left onto Keifer St. Traffic on Keifer St had the green light. Veh 2 turned in front of Veh 1, Veh 1 struck Veh 2.

Rating Veh 2 _____

44) 2 veh 6:15 PM clear/dry urban intersection

Veh 1 was on Prospect St and when at the River Ave intersection turned left into Veh 2, which was eastbound on Prospect St. Oper 1 stated she could not see Veh 2 due to the glare of the sun, which was bad at time of report.

Rating Veh 2 _____

45) 1 veh 5:30 PM rain/wet urban 4-lane hwy

Veh 1 being operated south on Military Rd when at 1234 Military with the front collided against a telephone pole at same location. Driver 1 states an unidentified veh ran him off the road. Driver apparently didn't have control of veh.

Rating Veh 1 _____

46) 2 veh 3:35 PM cloudy/dry suburban 4-lane hwy

Driver 2 told patrol he observed Veh 1 in his rear view mirror traveling at a high rate of speed before Veh 1 hit Veh 2 in the back end. Veh 1 and Veh 2 were traveling in the middle lane. Driver 1 told patrol he accelerated his veh not realizing how close he was to Veh 2, causing him to run into the back of Veh 2.

Rating Veh 1 _____

47) 2 veh 5:36 PM snow/icy suburban 2-lane rd

Oper of Veh 2 stated that he was proceeding southbound on Dillon St when Veh 1, which was proceeding northbound on Dillon crossed over into the southbound lane and struck Veh 2 head on. Oper of Veh 1 stated that she was proceeding northbound on Dillon and as she was attempting to negotiate a curve in the roadway she lost control of her veh on the slippery pavement and crossed over and struck Veh 2 head on.

Rating Veh 2 _____

48) 2 veh 2:56 PM clear/dry suburban 2-lane rd

Veh 1 southbound on Angelo Rd passed a roadsweeper. When Driver 1 pulled back in line, the front of the veh began to shake, causing the driver to lose control of the veh, causing the driver to fall out onto pavement. Veh 1 then crossed the northbound lane, grass area, and struck a parked car (Veh 2) in the left door. Veh 2 was parked in exit driveway of gas station and unoccupied at time of collision.

Rating Veh 1 _____

49) 2 veh 2:40 PM clear/dry suburban 4-lane hwy

Veh 1 made a right hand turn (northbound) onto Barrister Rd from the grocery store parking lot. Driver 2 made a left hand turn (northbound also) onto Barrister Rd from the department store parking lot. Both drivers stated they did not see the other until impact. Skid marks on the northbound inside lane appear to belong to Veh 1, possibly indicating Veh 1 improperly turned into the inside lane.

Rating Veh 1 _____

50) 1 veh 2:53 AM dry suburban curve

Veh 1 was heading west on Gulver Fork Rd. As Veh 1 was going around a curve just before the accident scene (on wrong side of the road), the driver slammed on the brakes, skidded sideways, and struck (on driver's side) a telephone pole (knocking down pole and wires), a telephone booth and guard rails in front of 9999 Gulver Fork Rd.

Rating Veh 1 _____

Ratings of Original Coders

1) 4
2) 4
3) 3
4) 0
5) 3
6) 1
7) 4
8) 1
9) 2
10) 4
11) 1
12) 4
13) 4
14) 4
15) 2
16) 8
17) 0
18) 4
19) 0
20) 0
21) 4
22) 1
23) 4
24) 1
25) 4
26) 4
27) 2
28) 0
29) 4
30) 8
31) 4
32) 2
33) 3
34) 3
35) 4
36) 0
37) 4
38) 1 (driver may have some responsibility because drivers should be especially cautious in a shopping plaza parking lot)
39) 1 (questionable responsibility; could the driver not see the construction work?)
40) 4
41) 4
42) 0
43) 4
44) 0
45) 4
46) 4
47) 0
48) 1
49) 2
50) 4

APPENDIX G-2

THEORETICAL CONSIDERATIONS OF DRIVER RESPONSIBILITY

In the definition and explanation of the responsibility concept as used in this manual, the driver is treated as a mechanism in the driver-vehicle system. This perspective enables us to evaluate whether a crash can be attributed in part to a malfunction of that mechanism, assuming that the total responsibility for a crash can be divided among the driver, other drivers playing a role in the accident, the vehicles, and the environment. This perspective, we believe, will be consistent with the objectives of most investigations into the role of the driver in crash causation. Researchers generally are more interested in learning about "what went wrong" to cause a crash than they are in divining the moral or legal shortcomings of drivers, for discerning the major "what went wrong" factors can lead to ways to reduce the frequency and severity of highway crashes. Hence, we have endeavored in this manual to distinguish crash responsibility from culpability, malfeasance, blame, fault, and other legal and moral conceptions.

In our initial application of the driver-as-mechanism concept to assessing driver responsibility in actual crashes, a question immediately confronted was: By what standards do we determine whether something was "wrong" in the driver's functioning? We need to know the programs or operating rules of a correctly-functioning driver in order to determine that a particular driver has deviated from those rules. In theory, we would need to know the entire "program" by which the safe driver should operate, and that program should account for virtually all the situations a driver will encounter. To our knowledge, no one has ever written that program, but its basic components have been codified in traffic laws, state driver manuals, and safe-driving courses. While a thoroughly exhaustive responsibility-coding manual would lay out the basic "rules of the road" for the coder to study, no attempt to do that was made for this manual. The guidelines that were provided seem to be sufficient, given the limited information usually available in accident reports. In actuality, we seldom find details on the driver's perceptions, judgments, and actions, especially when the driver was killed. Hence, crash responsibility frequently must be based on observed vehicle actions. For the most part, responsibility is descriptive of the driver-vehicle system and is not a psychological or mentalistic concept. As used here, it certainly is not a moral concept.

Division of responsibility. We recognize that crashes are the result of an interaction between the performance demands on the driver and the driver's response to those demands. The performance demands derive from the driver's own objectives and the demands placed by external agents, including the traffic, traffic controls, the weather, and other agents as listed in the text of this manual. A truly thorough and sophisticated responsibility analysis would

partial responsibility among all the agents, and also show how much the crash is due to a discrepancy between the external demands on the driver and the driver's ability to meet those demands. (A causal analysis system developed by Donelson et al. [1986] attempted to capture such interaction effects.) For example, a driver with limited experience or with imperfect vision may have an accident when he/she misunderstands a confusing traffic signal at a complex intersection. Unfortunately, most of the accident details received in police reports and even in most in-depth reports are insufficient to assess fully the effects of external agents, and this is especially true of passive agents such as traffic signs, road characteristics, and weather conditions. It is also true of the demands of the vehicle, for accident reports almost never address the handling requirements of the vehicle. Consequently, the burden of crash responsiblity is usually assigned to the driver. In effect, the driver is evaluated against a standard in which he/she is supposed to be the perfect mechanism, responding to and compensating for virtually all circumstances. Perhaps this is why most attempts to parcel responsibility among the driver, the vehicle, and the environment report that a large majority of accidents are due to the driver, and few are attributed to the vehicles or environment. Statistical data show that something is wrong with these indications, however. For example, highway crash data show that accidents clearly tend to occur at certain kinds of locations such as intersections and curves on rural roads. Surely this is evidence of a pronounced effect of the environment. Similarly, vehicles have been found to differ in their accident involvements, according to vehicle age, size, and make/model. An ideal responsibility assessment would capture these effects, but it may be impossible to meet that ideal, because of the great difficulty of identifying the environmental and vehicular influences on the individual accident, except in instances of overt failure, such as an inoperative traffic signal or a brake failure. Consequently, a driver typically is assigned the responsibility for most accidents by default.

Unfortunately, the responsibility method described in this manual has similar limitations. While the manual draws attention to the effects of external agents, and it instructs the coder that the driver must not be expected to handle all situations, the coder is restricted nevertheless by the lack of information on the influence of external agents. Hence, much responsibility will be assigned to the driver by default. This is particularly true in the case of single-vehicle crashes, where the coder is instructed to begin with the presumption that the driver was completely responsible for the accident. The limitation of our method should not be exaggerated, however. In many cases, the driver's error is apparent, and it is sometimes admitted by a driver.

Levels of Responsibility. Experts on crash causation have generally agreed that accidents rarely have a single cause. Considering the various external agents that may share crash responsibility with the driver, one might expect to find considerable variation among crash drivers in the degree of

responsibility assigned to them by coders. Yet, the scale we have used basically has only three levels: full responsibility, contributory responsibility, and no responsibility. Theoretically, further delineation of levels of responsibility is possible. We find, however, that the amount of information on crash causation in most accident reports does not support finer distinctions. Consequently, a more fine-grained responsibility scale must rely on the use of in-depth accident investigations using sophisticated methods to identify all crash-contributing factors.

Stages of responsibility. It is important to realize that our coding method assesses the driver's responsibility at the time of the crash. This is the last stage in a sequence, in each of which the driver assumes a different form of responsibility. The sequence begins when the driver enters his vehicle for the particular trip. Depending on his awareness of the condition of himself, his vehicle, and the environment, he assumes some risk in driving the vehicle, and thereby incurs some responsibility for the outcome of the trip. He may be aware that he is tired, or that his vehicle has bald tires, or that weather conditions make driving hazardous. Although he may attempt to compensate for these conditions in his driving, he nevertheless at this stage has assumed what may be called "diffuse responsibility" for a crash that may occur as a partial result of the conditions.

On the road, the driver enters a second stage of responsibility which depends on the way he is driving. He may increase crash risks by excessive speed for the conditions, by following too closely, by distracting his attention through conversation with a passenger. Thus, he has further established the conditions for an accident. This enhancement of crash risk may be called "global responsibility."

The final stage of crash responsibility is entered just before the crash, when the driver's actions at a specific time and location result in a crash. Here, we may say the driver assumes "immediate responsibility" for the crash. In the responsibility method of this manual, only immediate responsibility is rated, although in some cases the coder knows that the driver assumed diffuse or global responsibility. To illustrate, one crash in our files involved a vehicle with a damaged steering mechanism, which repeatedly came loose and required tightening. The driver continued to use the vehicle, aware of the defective steering mechanism. One evening the steering mechanism failed, the driver lost control of his vehicle, and a collision resulted. The driver was not judged immediately responsible for the crash, although he had clearly assumed diffuse responsibility.

Now, it may seem to the reader that a responsibility method should rate any responsibility the driver had for a crash, rather than isolate just immediate responsibility. In that approach, the driver in the example above would have been rated responsible for the crash. The problem with such a general approach is that it can interfere with research objectives, such as learning impairment

effects due to alcohol or other drugs. Suppose, for example, that a driver had smoked marijuana, then drove off and had a crash. The driver could be rated responsible because of the diffuse risk he/she assumed by driving after using marijuana. Were we to similarly rate all crash drivers who had ingested marijuana, it would be impossible for a statistical analysis of the responsibility data to indicate whether marijuana influences crashes by impairing driving performance. Similar losses of analytic capability would occur were we to prejudge any other factors in the first two stages of the responsibility sequence. Hence, our concentration is on the driver's actions in the third and last stage immediately preceding the crash.

Reference

Donelson, A.C., Haas, B.C, and Walsh, P.J. The Etiology of Fatal Traffic Accidents Involving Alcohol and Cannabis. Report, Traffic Injury Foundation of Canada, March 1986.

APPENDIX G-3:

DEVELOPMENT OF CRASH RESPONSIBILITY METHODOLOGY

The final product presented here is based on a method for judging crash responsibility originally developed by Kenneth Perchonok at Calspan Corporation in 1972. The original 5 scale points remain the same, although the definitions and clarifications are recent developments. The original scale demonstrated high inter-coder reliability ($r = .92$) when utilized in Terhune's (1982) study. Further, responsibility rates were found to increase with blood alcohol concentration (BAC), indicating validity of the scale.

The Perchonok scale was evaluated in comparison with another responsibility scale, a method by which coders rated responsibility on a 0 to 100% basis, as used by Smith and Popham (1952). The objective of this comparison was to determine whether one of the scales was clearly superior to the other in terms of reliability or other criteria.

The two scales were first evaluated by having two coders independently rate crash cases on each of the scales using accident reports from Terhune's 1982 study. Intercoder agreement levels were similar for the two scales, with correlations of their scores ranging from .77 to .92. While neither scale was more advantaged in this respect, the Perchonok scale was judged more easily interpretable due to its clearly defined scale points. The points of the "Smith-Popham scale" were undefined, hence it is difficult to interpret a coder's ratings. Rather than undertake a substantial developmental effort to define the points in a meaningful way, the decision was made to concentrate on improving the better-established Perchonok scale. Since no coding guide had ever been written for the Perchonok scale, development of a guide was made the prime objective. Some minor clarifications in the scale points were also made.

In the initial evaluation of the Perchonok scale, comparisons were made of responsibility ratings taken from the 1982 study with those of a new coder unfamiliar with the scale. Percent of exact agreement between the new coder and the original coder was modest (50%) and Pearson correlations fluctuated between .60 and .80. A second coder then became involved and coded 40 cases. Correlations were run between these "new" coders' ratings, with fair results ($r = .65$). Problem cases were singled out and differences discussed. Rules were developed for certain types of cases and incorporated into an early draft of the coding manual. Another 40 cases were independently coded by the two coders, and correlations again computed. Differences were noted and the manual was once again revised so as to encompass more situations.

The second attempt yielded very high correlations; however, it was necessary to determine the consistency of this relationship. Therefore, another set of 40 cases was coded by the same two

coders, and it was found that the strong relationship remained. The progression of intercoder agreement is presented in Tables 1, 2, and 3. Not only were high correlations obtained in the complete data set (Table 1), they were maintained in separate analyses of single-vehicle and multiple-vehicle crashes (Tables 2 and 3). This shows that intercoder agreement is not confined mainly to single-vehicle crashes in which the driver is usually assigned high responsibility for the accident. Comparing the intercoder correlations obtained in the 1982 study with those in this study, the ranges were similar. The correlations overall were slightly higher in the latest effort, but percent exact agreement was lower for single-vehicle accidents than in the previous study. This difference may be due to the new coding manual, in which the coder is encouraged to consider other factors involved that may hold some responsibility for the accident.

The final test for intercoder reliability involved giving the new coding manual to a naive coder who coded the 50 practice cases in Appendix A with no verbal instruction. The correlation was excellent: $r = .91$, and percent of exact agreement was 66%. The coder was next given 50 fatal-accident cases, taken from 7 different States' actual police reports, to code in the same manner. The correlation was again exceptional, $r = .96$, with percent of exact agreement higher than in the first set (80%).

Validity of the revised scale was examined as a final step in the process. If the responsibility ratings should demonstrate a positive relationship to blood alcohol concentration, this would indicate validity, for it would reflect the well-established correlation between BAC and relative crash risk. The BAC-responsibility relationship is shown in Figure 1 which incorporates the 3 sets of 40 cases. The Pearson correlation between BACs amd responsibility ratings was 0.38, which was significant at the .01 probability level.

The relationship presented graphically in Figure 1 was also compared to that found in the 1982 study. While the relationship in the earlier study showed a curious "drop" at the BAC level .15-.19, Figure 1 shows no such anomaly. Responsibility increases monotonically with BAC. It is important to note that the data set used in the latest reliability and validity checks is the same as that used in the 1982 study. In comparing the present reliability scores and the validity checks with those completed earlier, it appears that the revised scale and new manual are an improvement over the original version in terms of reliability and validity.

Table 1:

Intercoder Agreement Using the Perchonok Crash Responsibility Scale

Entire Data Set

First number in each cell is the Pearson correlation between coder ratings.

Second number (in parentheses) is the percent exact agreement between coders.

First Set of 40 cases

	Coder 2	Coder 3
Coder 1	.65 (48%)	.63 (45%)
Coder 2	*	.72 (50%)

Second Set of 40 cases

	Coder 2	Coder 3
Coder 1	.91 (68%)	.94 (83%)
Coder 2	*	.93 (62%)

Third Set of 40 cases

	Coder 2	Coder 3
Coder 1	.88 (54%)	.92 (74%)
Coder 2	*	.93 (68%)

Table 2:

Intercoder Agreement Using the Perchonok Crash Responsibility Scale

Single Vehicle Accidents

First number in each cell is the Pearson correlation between coder ratings.

Second number (in parentheses) is the percent exact agreement between coders.

First Set of 40 Cases N=9

	Coder 2	Coder 3
Coder 1	.79 (67%)	.82 (78%)
Coder 2	*	.97 (89%)

Second Set of 40 Cases N=11

	Coder 2	Coder 3
Coder 1	.69 (73%)	.83 (91%)
Coder 2	*	.93 (73%)

Third Set of 40 Cases N=9

	Coder 2	Coder 3
Coder 1	.95 (67%)	.93 (78%)
Coder 2	*	.96 (78%)

Table 3:

Intercoder Agreement Using the Perchonok Crash Responsibility Scale

Multiple Vehicle Accidents Only

First number in each cell is the Pearson correlation between coder ratings.

Second number (in parentheses) is the percent exact agreement between coders.

First Set of 40 Cases N=31

	Coder 2	Coder 3
Coder 1	.59 (42%)	.57 (35%)
Coder 2	*	.64 (39%)

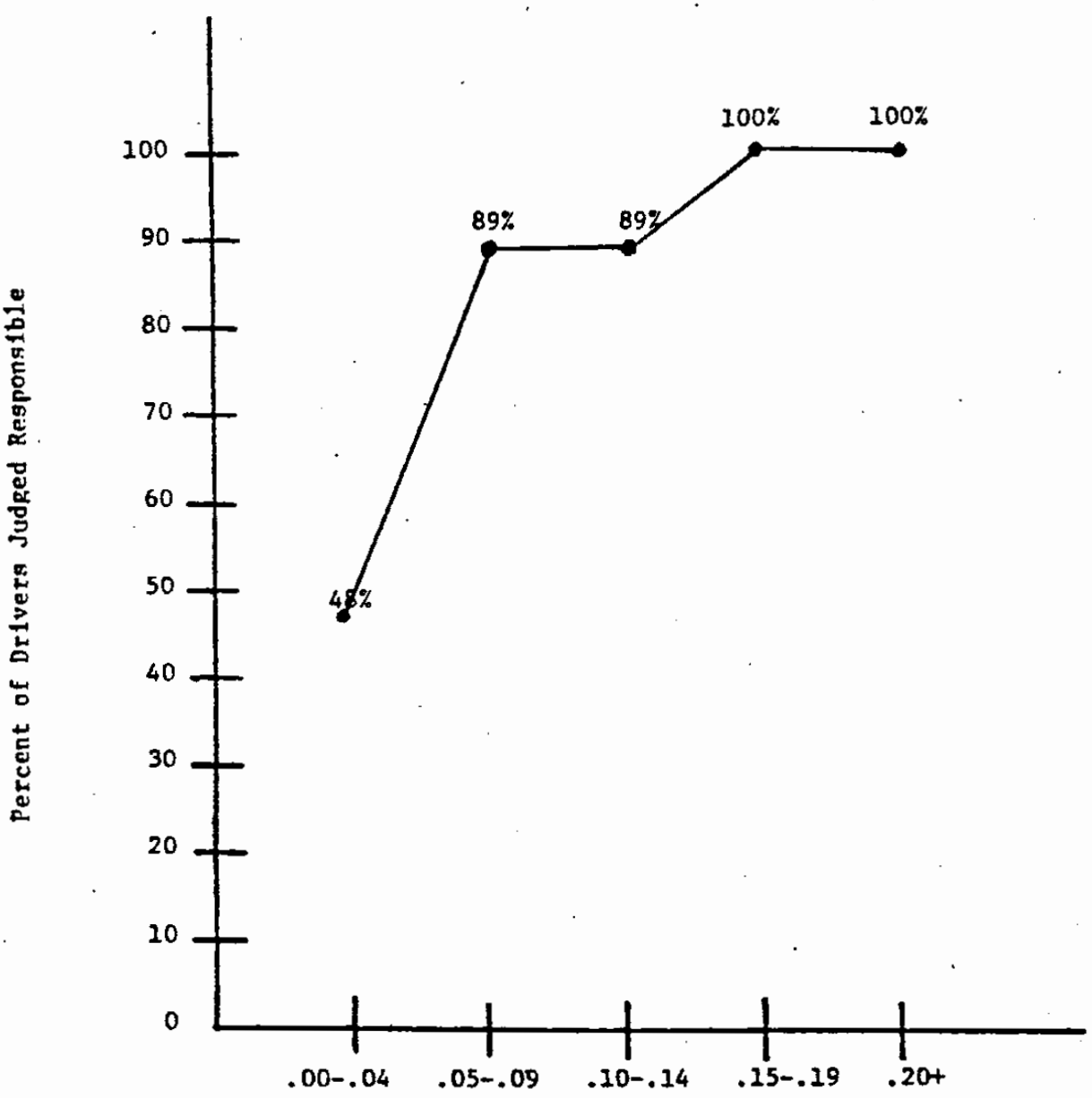
Second Set of 40 Cases N=29

	Coder 2	Coder 3
Coder 1	.93 (66%)	.94 (80%)
Coder 2	*	.93 (58%)

Third Set of 40 Cases N=31

	Coder 2	Coder 3
Coder 1	.85 (50%)	.91 (73%)
Coder 2	*	.92 (65%)

Figure 1: BAC x Driver Responsibility



References

Perchonok, K. Accident cause analysis. Final report, U.S. Department of Transportation, National Highway Traffic Safety Administration, July, 1972.

Terhune, K.W. The role of alcohol, marijuana, and other drugs in the accidents of injured drivers. Final report, U.S. Department of Transportation, National Highway Traffic Safety Administration, January, 1982.

Appendix H
Algorithms for the Derived Variables

Dichotomous Drug Variables

```
compute dbac = 0.
if (bac gt 0) dbac = 1.
compute dthc = 0.
if (thc gt 0) dthc = 1.
compute dcarbthc = 0.
if (carbthc gt 0) dcarbthc = 1.
compute dpcp = 0.
if (pcp gt 0) dpcp = 1.
compute dlsd = 0.
if (lsd gt 0) dlsd = 1.
compute dalpraz = 0.
if (alpraz gt 0) dalpraz = 1.
compute doxazpam = 0.
if (oxazpam gt 0) doxazpam = 1.
compute dclordyz = 0.
if (clordyz gt 0) dclordyz = 1.
compute ddesclor = 0.
if (desclor gt 0) ddesclor = 1.
compute dfenobrb = 0.
if (fenobrb gt 0) dfenobrb = 1.
compute dsecobrb = 0.
if (secobrb gt 0) dsecobrb = 1.
compute dbutabrb = 0.
if (butabrb gt 0) dbutabrb = 1.
compute dbutalbt = 0.
if (butalbt gt 0) dbutalbt = 1.
compute dpntobrb = 0.
if (pntobrb gt 0) dpntobrb = 1.
compute damobrb = 0.
if (amobrb gt 0) damobrb = 1.
compute dcoke = 0.
if (coke gt 0) dcoke = 1.
compute dbenzlec = 0.
if (benzlec gt 0) dbenzlec = 1.
compute damfet = 0.
if (amfet gt 0) damfet = 1.
compute dmethfet = 0.
if (methfet gt 0) dmethfet = 1.
compute dcafeen = 0.
if (cafeen gt 0) dcafeen = 1.
compute dethclor = 0.
if (ethclor gt 0) dethclor = 1.
compute dmthqual = 0.
if (mthqual gt 0) dmthqual = 1.
compute dmeprob = 0.
if (meprob gt 0) dmeprob = 1.
compute ddiphen = 0.
if (dipben gt 0) ddiphen = 1.
compute dclorfen = 0.
if (clorfen gt 0) dclorfen = 1.
```

Dichotomous Drug Variables (cont.)

```
compute damtryp = 0.
if (amtryp gt 0) damtryp = 1.
compute dnortryp = 0.
if (nortryp gt 0) dnortryp = 1.
compute dimpram = 0.
if (impram gt 0) dimpram = 1.
compute ddespram = 0.
if (despram gt 0) ddespram = 1.
compute ddoxepin = 0.
if (doxepin gt 0) ddoxepin = 1.
compute ddesdox = 0.
if (desdox gt 0) ddesdox = 1.
compute dmeperdn = 0.
if (meperdn gt 0) dmeperdn = 1.
compute dmethdon = 0.
if (methdon gt 0) dmethdon = 1.
compute dpropoxy = 0.
if (propoxy gt 0) dpropoxy = 1.
compute dnorprop = 0.
if (norprop gt 0) dnorprop = 1.
compute doxydone = 0.
if (oxydone gt 0) doxydone = 1.
compute dcodeen = 0.
if (codeen gt 0) dcodeen = 1.
compute dmorfeen = 0.
if (morfeen gt 0) dmorfeen = 1.
compute dheroin = 0.
if (heroin gt 0) dheroin = 1.
compute dclorpro = 0.
if (clorpro gt 0) dclorpro = 1.
compute dthordaz = 0.
if (thordaz gt 0) dthordaz = 1.
compute dmesdaz = 0.
if (mesordaz gt 0) dmesdaz = 1.
compute dquindyn = 0.
if (quindyn gt 0) dquindyn = 1.
compute dprocain = 0.
if (procain gt 0) dprocain = 1.
compute dacetpro = 0.
if (acetpro gt 0) dacetpro = 1.
compute dlydcain = 0.
if (lydcain gt 0) dlydcain = 1.
compute dflecain = 0.
if (flecain gt 0) dflecain = 1.
compute dcycbenz = 0.
if (cycbenz gt 0) dcycbenz = 1.
```

Parent Drug Derivations

```
COMPUTE MYCANNAB=0.
IF (DTHC EQ 1 OR DCARBTHC EQ 1) MYCANNAB=1.
COMPUTE MYDIAZ=0.
COMPUTE MYCLRDYZ=0.
COMPUTE DCCGROUP=0.
IF (DDIAZ EQ 1 AND EDIAZ NE 1) MYDIAZ=1.
IF (DCLORDYZ EQ 1 OR DDESCLOR EQ 1) MYCLRDYZ=1.
IF (DNORDIAZ EQ 1 AND DDIAZ EQ 0 AND DCLORDYZ EQ 0 AND DDESCLOR EQ 0)
DCCGROUP=1.
COMPUTE MYFLURAZ=0.
IF (DFLURAZ EQ 1 OR DDESFLUR EQ 1) MYFLURAZ=1.
VARIABLE LABELS MYCANNAB 'THC/metab'/ MYDIAZ 'Diazepam definite'/
MYCLRDYZ 'Chlordiazepoxide/metab'/DCCGROUP 'Diazepam-Chlorazepate-Chlordiaz'/
MYFLURAZ 'Flurazepam/metab'.
VALUE LABELS MYCANNAB MYDIAZ MYCLRDYZ DCCGROUP MYFLURAZ 0 'Not found'
1 'Found'.
COMPUTE MYCOKE=0.
IF (DCOKE EQ 1 OR DBENZLEC EQ 1 AND ECOKE NE 1) MYCOKE=1.
COMPUTE AMFETGRP=0.
COMPUTE MYMTHFET=0.
IF (DAMFET EQ 1 AND DMETHFET EQ 0) AMFETGRP=1.
IF (DMETHFET EQ 1) MYMTHFET=1.
COMPUTE MYAMTRYP=0.
IF (DAMTRYP EQ 1 OR DNORTRYP EQ 1) MYAMTRYP=1.
COMPUTE MYDOXPIN=0.
IF (DDOXEPIN EQ 1 OR DDESDOX EQ 1) MYDOXPIN=1.
VARIABLE LABELS MYCOKE 'Cocaine/metab'/ AMFETGRP 'Amphetamine Group'/
MYMTHFET 'Methamphetamine Definite'/MYAMTRYP 'Amitryptyline/metab'/
MYDOXPIN 'Doxepin/metab'.
VALUE LABELS MYCOKE AMFETGRP MYMTHFET MYAMTRYP MYDOXPIN 0 'Not found'
1 'Found'.
```

Parent Drug Derivations (cont.)

```
COMPUTE MYPROPOX=0.
IF (DPROPOXY EQ 1 OR DNORPROP EQ 1) MYPROPOX=1.
COMPUTE MYCODEEN=0.
COMPUTE MORFGRP=0.
IF ((DCODEEN EQ 1) AND (CODEEN GT 0.1*MORFEEN)) MYCODEEN=1.
IF ((DMORFEEN EQ 1) AND (CODEEN LE 0.1*MORFEEN)) MORFGRP=1.
COMPUTE MYPROCAN=0.
IF (DPROCAIN EQ 1 OR DACETPRO EQ 1) MYPROCAN=1.
COMPUTE MYLYDCAN=0.
IF (DLYDCAIN EQ 1 AND ELYDCAIN EQ 0) MYLYDCAN=1.
VARIABLE LABELS MYPROPOX 'Propoxyphene/metab'/ MYCODEEN 'Codeine definite'/
MORFGRP 'Heroin/codeine/morphine'/ MYPROCAN 'Procainamide/metab'/
MYLYDCAN 'Lidocaine definite'.
VALUE LABELS MYPROPOX MYCODEEN MORFGRP MYPROCAN MYLYDCAN 0 'Not found'
1 'Found'.
if (emorfeen eq 1) mycodeen=0.
if (emorfeen eq 1) morfgrp=0.
compute mymeprdn=0.
if (dmeperdn eq 1 and emeperdn ne 1) mymeprdn=1.
variable labels mymeprdn 'Meperidine definite'.
value labels mymeprdn 0 'Not found' 1 'Found'.
compute naranalg=0.
if (mymeprdn eq 1 or dmethdon eq 1 or mypropox eq 1 or doxydone eq 1
or mycodeen eq 1 or morfgrp eq 1) naranalg=1.
compute antaryth=0.
if (dquindyn eq 1 or myprocan eq 1 or mylydcan eq 1 or
dflecain eq 1) antaryth=1.
compute impramgp=0.
if (dimpram eq 1 or ddespram eq 1) impramgp=1.
variable labels impramgp 'Imipramine/desipramine'.
value labels impramgp 0 'Not found' 1 'Found'.
```

Drug Class Derivations

```
COMPUTE HALUCGEN=0.
IF (DPCP EQ 1 OR DLSD EQ 1) HALUCGEN=1.
COMPUTE BENDIAZ=0.
IF (DDIAZ EQ 1 OR DNORDIAZ EQ 1 OR DLORAZ EQ 1 OR DFLURAZ EQ 1
OR DDESFLUR EQ 1 OR DALPRAZ EQ 1 OR DOXAZPAM EQ 1 OR DCLORDYZ EQ 1
OR DDESCLOR EQ 1) BENDIAZ=1.
COMPUTE BARBIT=0.
IF (DFENOBRB EQ 1 OR DSECOBRB EQ 1 OR DBUTABRB EQ 1 OR DBUTALBT EQ 1
OR DPNTOBRB EQ 1 OR DAMOBRB EQ 1) BARBIT=1.
COMPUTE CNSSTIM=0.
IF (DCOKE EQ 1 OR DBENZLEC EQ 1 OR DAMFET EQ 1 OR DMETHFET EQ 1
OR DCAFEEN EQ 1) CNSSTIM=1.
COMPUTE OTHSEDTV=0.
IF (DETHCLOR EQ 1 OR DMTHQUAL EQ 1 OR DMEPROB EQ 1) OTHSEDTV=1.
VARIABLE LABELS HALUCGEN 'Hallucinogens' BENDIAZ 'Benzodiazepines'
BARBIT 'Barbiturates' CNSSTIM 'CNS Stimulants' OTHSEDTV 'Other Sedatives'.
VALUE LABELS HALUCGEN BENDIAZ BARBIT CNSSTIM OTHSEDTV 0 'Not found'
1 'Found'.
COMPUTE ANTIHIST=0.
IF (DDIPHEN EQ 1 OR DCLORFEN EQ 1) ANTIHIST=1.
COMPUTE ANTIDEPR=0.
IF (DAMTRYP EQ 1 OR DNORTRYP EQ 1 OR DIMPRAM EQ 1 OR DDESPRAM EQ 1
OR DDOXEPIN EQ 1 OR DDESDOX EQ 1 OR DFLUOX EQ 1 OR DNORFLUX EQ 1)
ANTIDEPR=1.
COMPUTE NARANALG=0.
IF (DMEPERDN EQ 1 OR DMETHDON EQ 1 OR DPROPOXY EQ 1 OR DNORPROP EQ 1
OR DOXYDONE EQ 1 OR DCODEEN EQ 1 OR DMORFEEN EQ 1 OR DHEROIN EQ 1)
NARANALG=1.
COMPUTE ANTPSYCH=0.
IF (DCLORPRO EQ 1 OR DTHORDAZ EQ 1 OR DMESDAZ EQ 1) ANTPSYCH=1.
COMPUTE ANTARYTH=0.
IF (DQUINDYN EQ 1 OR DPROCAIN EQ 1 OR DACETPRO EQ 1 OR DLYDCAIN EQ 1
OR DFLECAIN EQ 1) ANTARYTH=1.
VARIABLE LABELS ANTIHIST 'Antihistamines' ANTIDEPR 'Antidepressants'
NARANALG 'Narcotic analgesics' ANTPSYCH 'Antipsychotics'
ANTARYTH 'Antiarrythmics'.
VALUE LABELS ANTIHIST ANTIDEPR NARANALG ANTPSYCH ANTARYTH 0 'Not found'
1 'Found'.
```

Drug Concentration Variables

```
compute gbac=bac.
recode gbac (0.01=1)(0.02 thru 0.05=2)(0.06 thru 0.09=3)
(0.10 thru 0.14=4)(0.15 thru 1.5=5).
var labels gbac 'BAC Group'.
value labels GBAC 0 'None' 1 'Trace' 2 'Low' 3 'Intermed' 4 'High'
5 'Toxic'.
compute gthc=thc.
recode gthc (1,2=1)(3 thru 19=2)(20 thru 900=3).
var labels gthc 'THC Conc Group'.
compute gcarbthc=carbthc.
recode gcarbthc (1 thru 4=1)(5 thru 249=2)(250 thru 9000=3).
var labels gcarbthc 'COOH-THC Conc Group'.
compute gpcp=pcp.
recode gpcp (1 thru 7=1)(8 thru 48=2)(49 thru 89=3)(90 thru 9000=4).
var labels gpcp 'PCP Conc Group'.
compute gdiaz=diaz.
recode gdiaz (1 thru 120=1)(121 thru 2499=2)(2500 thru 4999=3)
(5000 thru 9000=4).
variable labels gdiaz 'Diazepam Conc Group'.
compute gnordiaz=nordiaz.
recode gnordiaz (1 thru 120=1)(121 thru 1099=2)(1100 thru 9000=3).
compute gclordyz=clordyz.
recode gclordyz (1 thru 120=1)(121 thru 2499=2)(2500 thru 4999=3)
(5000 thru 9000=4).
compute gdesclor=desclor.
recode gdesclor (1 thru 120=1)(121 thru 1999=2)(2000 thru 9000=3).
compute gfenobrb=fenobrb.
recode gfenobrb (1 thru 120=1)(121 thru 17499=2)(17500 thru 34999=3)
(35000 thru 90000=4).
compute gbutalbt=butalbt.
recode gbutalbt (1 thru 120=1)(121 thru 4999=2)(5000 thru 9999=3)
(10000 thru 90000=4).
compute gpntobrb=pntobrb.
recode gpntobrb (1 thru 120=1)(121 thru 4999=2)(5000 thru 9999=3)
(10000 thru 90000=4).
compute gcoke=coke.
recode gcoke (1 thru 60=1)(61 thru 499=2)(500 thru 999=3)(1000 thru 9000=4).
compute gbenzlec=benzlec.
recode gbenzlec (1 thru 60=1)(61 thru 44999=2)(45000 thru 89999=3)
(90000 thru 98000=4).
compute gamfet=amfet.
recode gamfet (1 thru 60=1)(61 thru 99=2)(100 thru 199=3)(200 thru 90000=4).
compute gmethfet=methfet.
recode gmethfet (1 thru 60=1)(61 thru 4999=2)(5000 thru 90000=3).
compute gdiphen=diphen.
recode gdiphen (1 thru 24=1)(25 thru 4999=2)(5000 thru 9999=3)
(10000 thru 90000=4).
```

Drug Concentration Variables (cont.)

```
compute gclorfen=clorfen.
recode gclorfen (1 thru 60=1)(61 thru 349=2)(350 thru 90000=3).
compute gamtryp=amtryp.
compute gnortryp=nortryp.
recode gamtryp (1 thru 7=1)(8 thru 249=2)(250 thru 499=3)(500 thru 9000=4).
recode gnortryp (1 thru 7=1)(8 thru 249=2)(250 thru 499=3)(500 thru 9000=4).
compute gfluox=fluox.
recode gfluox (1 thru 7=1)(8 thru 699=2)(700 thru 9000=3).
compute gnorflux=norfluox.
recode gnorflux (1 thru 7=1)(8 thru 899=2)(900 thru 9000=3).
compute gmeperdn=meperdn.
recode gmeperdn (1 thru 60=1)(61 thru 499=2)(500 thru 999=3)
(1000 thru 99000=4).
compute gpropoxy=propoxy.
recode gpropoxy (1 thru 60=1)(61 thru 249=2)(250 thru 499=3)(500 thru 9900=4).
compute gcodeen=codeen.
recode gcodeen (1 thru 12=1)(13 thru 99=2)(100 thru 199=3)(200 thru 9900=4).
compute gmorfeen=morfeen.
recode gmorfeen (1 thru 60=1)(61 thru 99=2)(100 thru 199=3)(200 thru 9900=4).
compute glydcain=lydcain.
recode glydcain (1 thru 240=1)(241 thru 2999=2)(3000 thru 5999=3)
(6000 thru 99000=4).
variable labels GNORDIAZ 'Nordiazepam Conc Group' GCLORDYZ
'Chlordiazepoxide Conc Group' GDESCLOR
 'Desmethylchlordiazepoxide Conc Group'
GFENOBRB 'Phenobarbital Conc Group' GBUTALBI 'Butalbital Conc Group'
GPNTOBRB 'Pentobarbital Conc Group' GCOKE 'Cocaine Conc Group'
GBENZLEC 'Benzoylecgonine Conc Group' GAMFET 'Amphetamine Conc Group'
GMETHFET 'Methamphetamine Conc Group' GDIPHEN 'Diphenhydramine Conc Group'
GCLORFEN 'Chlorpheniramine Conc Group' GAMTRYP 'Amitryptyline Conc Group'
GNORTRYP 'Nortryptyline Conc Group' GFLUOX 'Fluoxetine Conc Group'
GNORFLUX 'Norfluoxetine Conc Group' GMEPERDN 'Meperidine Conc Group'
GPROPOXY 'Propoxyphene Conc Group' GCODEEN 'Codeine Conc Group'
GMORFEEN 'Morphine Conc Group' GLYDCAIN 'Lidocaine Conc Group'.
Value labels GTHC GCARBTHC GPCP GDIAZ GNORDIAZ GCLORDYZ GDESCLOR GFENOBRB
GBUTALBI GPNTOBRB GCOKE GBENZLEC GAMFET GMETHFET GDIPHEN GCLORFEN GAMTRYP
GNORTRYP GFLUOX GNORFLUX GMEPERDN GPROPOXY GCODEEN GMORFEEN GLYDCAIN
0 'None' 1 'Trace' 2 'Low' 3 'High' 4 'Toxic'.
variable labels GNORDIAZ 'Nordiazepam Conc Group' GCLORDYZ
'Chlordiazepoxide Conc Group' GDESCLOR
 'Desmethylchlordiazepoxide Conc Group'
GFENOBRB 'Phenobarbital Conc Group' GBUTALBI 'Butalbital Conc Group'
GPNTOBRB 'Pentobarbital Conc Group' GCOKE 'Cocaine Conc Group'.
```

Other Drug Data Derived Variables

```
count ndrugs=MYCANNAB DPCP DLSD MYDIAZ DCCGROUP MYFLURAZ MYCLRDYZ
DFENOBRB DBUTALBT DPNTOBRB MYCOKE DMETHFET AMFETGRP DCAFEEN DDIPHEN
DCLORFEN MYAMTRYP DDESPRAM MYDOXPIN DMEPERDN DMETHDON MYPROPOX MYCODEEN,
MORFGRP MYLYDCAN DCYCBENZ OTHRDRUG (1).
variable labels NDRUGS 'Total nonalcoholic drugs ingested'.
compute ndrugs@a=ndrugs+dbac.
variable labels NDRUGS@A 'Total drugs ingested, incl. alcohol'.
compute NOALCDRG=0.
if (NDRUGS GE 1) NOALCDRG=1.
variable label NOALCDRG 'Any Nonalc Drug'.
value labels 0 'Not detected' 1 'Detected'.
compute ANYSUBST=0.
if (NDRUGS@A GE 1) ANYSUBST=1.
variable labels ANYSUBST 'Any Alcohol or Drug'.
value labels 0 'Not detected' 1 'Detected'.
```

Derivation of SUBSAMPL Variable

```
COMPUTE SUBSAMPL=97.
IF (NDRUGS@A EQ 0) SUBSAMPL=0.
IF (GBAC EQ 1 OR GBAC EQ 2 OR GBAC EQ 3 AND NDRUGS EQ 0) SUBSAMPL=1.
IF (GBAC GE 4 AND NDRUGS EQ 0) SUBSAMPL=2.
IF (DTHC EQ 1 AND NDRUGS@A EQ 1) SUBSAMPL=3.
IF (DTHC EQ 0 AND DCARBTHC EQ 1 AND NDRUGS@A EQ 1) SUBSAMPL=4.
IF (MYCOKE EQ 1 AND NDRUGS@A EQ 1) SUBSAMPL=5.
IF ((AMFETGRP EQ 1 OR MYMTHFET EQ 1) AND NDRUGS@A EQ 1) SUBSAMPL=6.
IF (DBAC EQ 0 AND NDRUGS EQ 1 AND MYCANNAB EQ 0 AND
CNSSTIM EQ 0) SUBSAMPL=7.
IF (DTHC EQ 1 AND DBAC EQ 1 AND NDRUGS@A LT 3) SUBSAMPL=8.
IF (DTHC EQ 0 AND DCARBTHC EQ 1 AND DBAC EQ 1 AND NDRUGS@A LT 3)
SUBSAMPL=9.
IF (MYCOKE EQ 1 AND DBAC EQ 1 AND NDRUGS@A LT 3) SUBSAMPL=10.
IF ((AMFETGRP EQ 1 OR MYMTHFET EQ 1) AND DBAC EQ 1
AND NDRUGS@A EQ 2) SUBSAMPL=11.
IF (DBAC EQ 1 AND NDRUGS@A EQ 2 AND MYCANNAB EQ 0 AND CNSSTIM EQ 0)
SUBSAMPL=12.
IF (DBAC EQ 1 AND NDRUGS@A GE 3) SUBSAMPL=13.
IF (DBAC EQ 0 AND NDRUGS EQ 2) SUBSAMPL=14.
IF (DBAC EQ 0 AND NDRUGS GE 3) SUBSAMPL=15.
variable label SUBSAMPL "Driver Drug Involvement Categories".
value labels SUBSAMPL 97 'Other' 0 "'Drugfree'" 1 "AlcOnly-LT.10"
2 "AlcOnly-GE.10" 3 "THC-Only" 4 "Carboxy-Only" 5 "Cocaine-Only"
6 "Amfets-Only" 7 'Othr Singl' 8 'Alc+THC' 9 "Alc+Carbx"
10 'Alc+Coke' 11 "Alc+Amfets" 12 "Alc+Othr" 13 "Alc+2+"
14 "2 Nonalc" 15 "3+ Nonalc" 97 'Other'.
```

Non-drug Crash Descriptor Variables

```
compute landuser=roadtype.
recode landuser (1 thru 9=2)(11 thru 19=1).
variable labels landuser 'Land Use from ROADTYPE'.
value labels landuser 1 'Urban' 2 'Rural'.
compute season=axmonth.
recode season (1,2,3=1)(4,5,6=2)(7,8,9=3)(10,11,12=4).
value labels season 1 'Winter' 2 'Spring' 3 'Summer' 4 'Fall'.
compute surfconr=surfcon.
recode surfconr (2 thru 5,8=2).
variable labels surfconr 'Surface Condition'.
value labels surfconr 1 'Dry' 2 'Other'.
compute vehtype=bodytype.
recode vehtype (1 thru 9,13,67=1) (10,50,51,52=2) (11,40,41,42,48,49,54=3)
(12,55,56,68=4) (53,58,59,69=5) (20,21,27,28,29=6) (70 thru 79=7).
variable labels vehtype 'Vehicle Type'.
value labels vehtype 1 'Car' 2 'Pickup' 3 'Van' 4 'U-Veh' 5 'Oth Lt Truk'
6 'M-cyle' 7 'Hvy Truk'.
compute calaxr=calax.
recode calaxr (111,112,113,118,121 thru 124=1) (211,213,215,217,221=2)
(311,321=3) (511,513=4) (212,214,216,218,222=5) (312,322=6) (512,514=7)
(411,413,415,421,423,425,427=8) (412,414,416,422,424,426,428=9) (else=97).
variable labels calaxr '10-Value CALAX'.
value labels calaxr 1 'Sngl Drvr' 2 'Rear Strkng' 3 'Opp Strkng'
4 'Int Strkng' 5 'Rear Struk' 6 'Opp Struk' 7 'Int Struk' 8 'Turn On'
9 'Turnd Upon' 97 'Other'.
missing value surfconr (9) calaxr (998).
```

```
compute crdthtmg=crdthtym.
recode crdthtmg (0.1 thru 0.5=1) (0.6 thru 1.0=2) (1.1 thru 1.5=3)
(1.6 thru 2.0=4) (2.1 thru 2.5=5) (2.6 thru 3.0=6) (3.1 thru 3.5=7)
(3.6 thru 4.0=8) (4.1 thru 9.8=9).
variable labels crdthtmg 'Crash-Death-Time Group'.
value labels crdthtmg 0.0 '0 hr' 1 '0.1-0.5 hr' 2 '0.6-1.0 hr'
3 '1.1-1.5 hr' 4 '1.6-2.0 hr' 5 '2.1-2.5 hr' 6 '2.6-3.0 hr' 7 '3.1-3.5 hr'
8 '3.6-4.0 hr' 9 '4.0-9.8 hr'.
compute dthbltmg=dthbltym.
recode dthbltmg (0.0 thru 1.0=1) (1.1 thru 2.0=2) (2.1 thru 3.0=3)
(3.1 thru 4.0=4) (4.1 thru 8.0=5) (8.1 thru 12.0=6) (12.1 thru 24.0=7)
(24.1 thru 48.0=8) (48.1 thru 72.0=9) (72.1 thru 96.0=10).
variable label dthbltmg 'Death-Spcmn-Time Group'.
value labels dthbltmg 1 'LE 1 hr' 2 '1.1-2.0 hr' 3 '2.1-3/0 hr'
4 '3.1-4.0 hr' 5 '4.1-8.0 hr' 6 '8.1-12.0 hr' 7 '12.1-24.0 hr'
8 '24.1-48.0 hr' 9 '48.1-72.0 hr' 10 '72.1-96 hr'.
compute dragegrp=agedrvr.
recode dragegrp (15 thru 17=1) (18 thru 20=2) (21 thru 24=3) (25 thru 34=4)
(35 thru 44=5) (45 thru 54=6) (55 thru 64=7) (65 thru 95=8).
variable label dragegrp 'Driver Age Group'.
value labels dragegrp 1 '15-17 yr' 2 '18-20 yr' 3 '21-24 yr' 4 '25-34 yr'
5 '35-44 yr' 6 '45-54 yr' 7 '55-64 yr' 8 '75-95 yr'.
compute drfctr1=trunc(drvrfctr/10000).
compute rem1=drvrfctr-(10000*drfctr1).
compute drfctr2=trunc(rem1/100).
compute drfctr3=rem1-(100*drfctr2).
compute drfctr1r=drfctr1.
compute drfctr2r=drfctr2.
compute drfctr3r=drfctr3.
recode drfctr1r drfctr2r drfctr3r (3, 7 thru 12, 19 thru 25, 29,31,32,37,
40 thru 43,48,49,52 thru 57,59 thru 87,90,91,92=97)(33,34,35=3)(4,5=4).
variable label drfctr1r 'First Driver Factor'.
variable label drfctr2r 'Second Driver Factor'.
variable label drfctr3r 'Third Driver Factor'.
value labels drfctr1r drfctr2r drfctr3r 1 'Sleep/y' 2 'Ill' 3 'Passng Err'
26 'Follwng Err' 27 'Ln Chg Err' 28 'Ln Mnt Err' 30 'Ntry/xt Err' 36 'Rckless'
38 'Rtofway Err' 39 'Trafcntrl Err' 44 'Toofast' 45 'Tooslow'
46 'Eratspeed' 47 'Turn Err' 50 'Wrongway' 51 'Wrong Syd'
58 'Ovrcorct' 97 'Other' 0 'None' 4 'Drugs' 6 'Inattntv'.
missing value drfctr1r drfctr2r drfctr3r (99).
```

Appendix I

Unweighted Drug Prevalence Rates

This Appendix presents the prevalence rates for the drugs and drug classes, using the original data without corrections for sample bias. For some sampling sites, the data include cases that originated from adjacent counties or States.

At the end of this Appendix, Table I-7 provides confidence intervals, based on the sample sizes obtained for each county or State, and the FARS counts of the eligible driver population from each county or State.

Table I-1

Drug Prevalence Rates in Final 1982 Cases
Drugs in Driver Fatalities Study

Substance	Alameda CA	Solano CA	San Bernadino CA	San Diego CA	Los Angeles CA	Massachusetts	North Carolina	Washoe NV	Northern Virginia	Wisconsin Exc. Milw.	Milwaukee WI	Tarrant TX	Dallas TX	All Sites
Alcohol (ethanol): BAC <.10	12.5%	8.0%	9.7%	11.0%	14.6%	5.2%	6.0%	11.1%	12.0%	7.5%	8.3%	11.3%	7.8%	8.3%
Alcohol (ethanol): BAC ≥.10	37.5%	44.0%	31.3%	44.8%%	44.8%	47.4%	40.6%	50.0%	39.8%	41.2%	44.4%	53.8%	46.1%	42.6%
Cannabis														
Delta-9 THC	7.5	4.0	3.7	9.7	5.2	4.6	1.9	5.6	2.4	3.2	0.0	10.4	6.1	4.3%
Carbony THC (metabolite)	7.5	8.0	6.7	13.8	7.3	8.1	4.3	7.4	6.0	4.9	0.0	14.2	8.7	6.9%
Hallucinogens														
Phencyclidine	2.5	0.0	0.0	0.0	1.8	0.0	0.0	0.0	0.0	0.0	2.8	0.0	0.0	0.2%
LSD	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Benzodiazepine tranquilizers														
Diazepam	5.0	8.0	0.7	1.4	1.8	3.5	4.0	0.0	1.2	1.2	0.0	4.7	1.7	2.5%
Nordiazepam (diaz. metab.)	5.0	8.0	0.0	1.4	1.8	2.3	4.0	0.0	1.2	1.2	0.0	4.7	0.9	2.3%
Lorazepam	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Flurazepam	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Desethylflurazepam (flura. metab.)	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Alprazolam	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Oxazepam	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Chlordiazepoxide	0.0	0.0	0.0	0.7	0.0	1.2	0.4	0.0	0.0	0.3	0.0	0.0	0.9	0.4%
Desmethylchlordiazepoxide (chlordiaz. metab.)	0.0	0.0	0.0	0.7	0.0	0.4	0.4	0.0	0.0	0.3	0.0	0.0	0.9	0.4%
Barbiturate sedatives														
Phenobarbital	0.0	4.0	0.7	1.4	0.0	0.4	0.4	0.0	0.0	0.4	0.0	0.9	1.7	0.6%
Secobarbital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Butabarbital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Butalbital	0.0	0.0	0.0	2.0	0.0	0.4	0.9	1.9	0.0	0.4	0.0	0.9	0.0	0.7%
Pentobarbital	0.0	0.0	1.5	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.2%
Amobarbital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
CNS Stimulants														
Cocaine	5.0	0.0	0.0	1.4	7.3	1.2	1.9	1.9	3.6	0.3	2.8	0.9	4.0	2.8%
Benzoylecgonine (cocaine metab.)	10.0	8.0	0.7	0.2	10.4	4.6	4.9	3.7	3.6	2.8	5.6	4.7	9.6	4.5%
Amphetamine	2.5	8.0	3.7	4.1	1.8	0.0	0.0	0.0	0.0	0.0	0.0	2.8	1.7	1.1%
Methamphetamine	7.5	8.0	7.5	8.3	2.1	0.8	0.0	3.7	0.0	0.3	0.0	0.0	1.7	1.8%
Caffeine	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Non-barbiturate sedatives														
Ethchlorvynol	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Methaqualone	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Meprobamate	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antihistamines														
Diphenhydramine hydrochloride	0.0	0.0	0.0	0.0	0.0	0.8	0.4	0.0	0.0	0.6	2.8	0.9	0.0	0.4%
Chlorpheniramine	0.0	0.0	1.5	0.7	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.9	0.3%

Table I-1 (Continued)

Substance	Alameda CA	Solano CA	San Bernadino CA	San-Diego CA	Los-Angeles CA	Mass-achusetts	North Carolina	Washoe NV	Northern Virginia	Wisconsin Exc. Milw.	Milwaukee WI	Tarrant TX	Dallas TX	All Sites
Antidepressants														
Amitriptyline	0.0	0.0	0.0	1.4	0.0	0.6	0.4	0.0	0.0	0.3	0.0	0.0	0.9	0.4%
Nortriptyline (amitrip. metab.)	0.0	0.0	0.0	1.4	0.0	0.6	0.4	0.0	0.0	0.3	0.0	0.0	0.9	0.4%
Imipramine	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Desipramine (imipramine metab.)	0.0	0.0	0.0	0.0	1.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Doxepin	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Desmethyldoxepin (dox. metab.)	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Fluoxetine	0.0	0.0	0.0	0.0	1.0	1.2	0.0	0.0	0.0	0.0	0.0	0.9	0.0	0.2%
Norfluoxetine (fluoxemetab.)	0.0	0.0	0.0	0.0	1.0	1.2	0.0	0.0	0.0	0.0	0.0	0.9	0.0	0.2%
Narcotic Analgesics														
Meperidine hydrochloride	0.0	0.0	0.0	0.0	1.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Methadone	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Propoxyphene	0.0	0.0	0.7	0.0	1.0	0.0	0.6	0.0	0.0	0.0	0.0	0.0	0.9	0.3%
Norpropoxyphene (propox. metab.)	0.0	0.0	0.7	0.0	1.0	0.0	0.6	0.0	0.0	0.0	0.0	0.0	0.9	0.3%
Oxycodone	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Codeine	0.0	0.0	0.0	0.7	0.0	0.6	0.6	0.0	0.0	0.6	0.0	0.0	0.0	0.4%
Morphine	0.0	0.0	0.0	2.8	0.0	1.2	0.0	0.0	0.0	0.3	0.0	0.9	1.7	0.5%
Heroin	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antipsychotics														
Chlorpromazine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Thioridazine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Mesoridazine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antiarrhythmics														
Quinidine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Procainamide	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
N-Acetylprocainamide (Procainamide metab.)	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Lidocaine	7.5	16.0	7.5	13.8	7.3	6.4	4.7	5.6	7.2	3.8	2.8	7.5	8.7	6.4%
Flecainide	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Muscle relaxant														
Cyclobenzaprine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Other drugs	0.0	8.0	3.0	0.7	1.0	1.7	1.1	0.0	1.2	2.0	0.0	0.9	1.7	1.5%
TOTAL CASES	40	25	134	145	96	173	530	54	83	345	36	106	115	1882

Table I-2

Concentration Categories of Substances Found in 3 or More Drivers

Substance	# Cases with drug	Concentration Category: Trace	Low - Intermed.	High	Toxic	Total
Alcohol (ethanol)	958	2.2%	14.1%	18.1%	65.6%	100.0%
Cannabis						
Delta-9 THC	80	20.0	68.8	11.2	*	100.0
Carboxy-THC	129	0.0	99.2	0.8	*	100.0
Hallucinogens						
Phencyclidine	3	0.0	0.0	33.3	66.7	100.0
Benzodiazepines						
Diazepam	47	36.2	63.8	0.0	0.0	100.0
Nordiazepam	43	23.3	74.4	2.3	*	100.0
Chlordiazepoxide	8	12.5	75.0	0.0	12.5	100.0
Desmethylchlordiazepoxide	7	42.9	42.9	14.3	*	100.0
Barbiturate Sedatives						
Phenobarbital	12	0.0	83.3	16.7	0.0	100.0
Butalbital	13	0.0	92.3	7.7	0.0	100.0
Pentobarbital	4	0.0	100.0	0.0	0.0	100.0
CNS Stimulants						
Cocaine	37	24.3	64.9	5.4	5.4	100.0
Benzoylecgonine	84	6.0	94.0	0.0	0.0	100.0
Amphetamine	20	20.0	10.0	30.0	40.0	100.0
Methamphetamine	34	8.8	88.2	2.9	*	100.0
Antihistimines						
Diphenhydramine	7	0.0	85.7	0.0	14.3	100.0
Chlorpheniramine	5	0.0	60.0	40.0	*	100.0
Antidepressants						
Amitriptyline	7	0.0	28.6	28.6	42.8	100.0
Nortriptyline	7	0.0	28.6	57.1	14.3	100.0
Fluoxetine	4	0.0	25.0	75.0	*	100.0
Norfluoxetine	4	0.0	75.0	25.0	*	100.0
Narcotic Analgesics						
Propoxyphene	6	0.0	66.7	0.0	33.3	100.0
Norpropoxyphene	6	0.0	66.7	0.0	33.3	100.0
Codeine	7	0.0	57.1	14.3	28.6	100.0
Morphine	9	22.2	33.3	11.1	33.3	100.0

*A toxic level was not identified.

Table I-3

Site Distribution of Parent Drugs Found in 1982 Fatally Injured Drivers
Drugs in Driver Fatalities Study

Substance (Parent Drugs only)	Alameda	Solano	San Bernadino	San-Diego	Los-Angeles	Mass-achusetts	North Carolina	Washoe	Fairfax	Madison	Milwaukee	Tarrant	Dallas	All Sites
Alcohol (ethanol)	50.0%	52.0%	41.0%	55.9%	59.3%	52.6%	46.6%	61.1%	51.8%	48.7%	52.8%	65.1%	53.9%	50.9%
Cannabis	7.5	8.0	6.7	13.8	7.3	8.1	4.3	7.4	6.0	4.9	0.0	14.2	8.7	6.9%
Hallucinogens														
Phencyclidine	2.5	0.0	0.0	0.0	1.0	0.0	0.0	0.0	0.0	0.0	2.8	0.0	0.0	0.2%
LSD	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Benzodiazepine tranquilizers														
Diazepam	5.0	8.0	8.7	1.4	1.0	3.5	4.0	0.0	1.2	1.2	0.0	4.7	1.7	2.5%
Diazepam/Chlorazepate/ Chlordiazepoxide	0.0	4.0	0.0	0.0	0.0	0.6	0.6	0.0	0.0	0.3	0.0	0.0	0.9	0.4%
Flurazepam	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Chlordiazepoxide	0.0	0.0	0.0	0.7	0.0	1.2	0.6	0.0	0.0	0.3	0.0	0.0	0.9	0.4%
Barbiturate sedatives														
Phenobarbital	0.0	4.0	0.7	1.4	0.0	0.6	0.4	0.0	0.0	0.6	0.0	0.9	1.7	0.6%
Butalbital	0.0	0.0	0.0	2.0	0.0	0.6	0.9	1.9	0.0	0.6	0.0	0.9	0.0	0.7%
Pentobarbital	0.0	0.0	1.5	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.2%
CNS Stimulants														
Cocaine	10.0	8.0	0.7	2.0	10.4	4.6	4.9	3.7	3.6	2.0	5.6	4.7	9.6	4.5%
Amphetamine Group	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.8	0.9	0.2%
Methamphetamine	7.5	0.0	7.5	0.3	2.1	0.0	0.0	3.7	0.0	0.3	0.0	0.0	1.7	1.0%
Caffeine	0.0	0.0	0.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Antihistamines														
Diphenhydramine hydrochloride	0.0	0.0	0.0	0.0	0.0	0.6	0.4	0.0	0.0	0.6	2.8	0.9	0.0	0.4%
Chlorpheniramine	0.0	0.0	1.5	0.7	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.9	0.3%
Antidepressants														
Amitriptyline	0.0	0.0	0.0	1.4	0.0	0.6	0.4	0.0	0.0	0.3	0.0	0.0	0.9	0.4%
Imipramine	0.0	0.0	0.0	0.0	1.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Doxepin	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.3	0.0	0.0	0.0	0.1%

Table I-3 (Continued)

Substance	Alameda	Solano	San Bernadino	San-Diego	Los-Angeles	Mass-achusetts	North Carolina	Washoe	Fairfax	Madison	Milwaukee	Tarrant	Dallas	All Sites
Narcotic Analgesics														
Meperidine hydrochloride	0.0	0.0	0.0	0.0	1.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Methadone	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Propoxyphene	0.0	0.0	0.7	0.0	1.0	0.0	0.6	0.0	0.0	0.0	0.0	0.0	0.9	0.3%
Codeine	0.0	0.0	0.0	0.7	0.0	0.6	0.6	0.0	0.0	0.6	0.0	0.0	0.0	0.4%
Heroin/Codeine/Morphine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.9	1.7	0.2%
Antiarrythmics														
Procainamide	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Muscle relaxant														
Cyclobenzaprine	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Other drugs	0.0	8.0	3.0	0.7	1.0	1.7	1.1	0.0	1.2	2.0	0.0	0.9	1.7	1.5%
TOTAL CASES	40	25	134	145	96	173	530	54	83	345	36	106	115	1882

Table I-4

Drug Class Prevalence Rates in Final 1882 Cases
Drugs in Driver Fatalities Study

Drug Class	Alameda	Solano	San Bernadino	San-Diego	Los-Angeles	Mass-achusetts	North Carolina	Washoe	Fairfax	Madison	Milwaukee	Tarrant	Dallas	All Sites
Alcohol (ethanol)	50.0%	52.0%	41.0%	55.9%	59.3%	52.6%	46.6%	61.1%	51.8%	48.7%	52.8%	65.1%	53.9%	50.9%
Cannabis	7.5	8.0	6.7	13.8	7.3	8.1	4.3	7.4	6.0	4.9	0.0	14.2	8.7	6.9%
Hallucinogens	2.5	0.0	0.7	0.0	1.0	0.0	0.0	0.0	0.0	0.0	2.8	0.0	0.0	0.2%
Benzodiazepine tranquilizers	5.0	12.0	1.5	2.8	1.0	5.2	5.1	0.0	1.2	1.7	0.0	4.7	3.5	3.3%
Barbiturate sedatives	0.0	4.0	2.2	3.4	0.0	1.2	1.5	1.9	0.0	1.4	0.0	1.9	1.7	1.5%
CNS stimulants	15.0	12.0	8.2	11.0	11.5	4.6	4.9	5.6	3.6	2.3	5.6	6.6	12.2	6.3%
Non-barbiturate sedatives	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antihistimines	0.0	0.0	1.5	0.7	0.0	0.6	0.6	0.0	0.0	0.6	2.8	0.9	0.9	0.6%
Antidepressants	0.0	0.0	0.0	1.4	2.1	1.7	0.8	0.0	0.0	0.6	0.0	0.9	0.9	0.8%
Narcotic Analgesics	0.0	0.0	0.7	0.7	2.1	0.6	1.1	0.0	0.0	0.9	0.0	0.9	2.6	1.0%
Antipsychotics	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antiarrhythmics	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0	0.0	0.1%
Muscle Relaxants	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.1%
Other Drugs	0.0	0.0	3.0	0.7	1.0	1.7	1.1	0.0	1.2	2.0	0.0	0.9	1.7	1.5%
Total Cases	40	25	134	145	96	173	330	54	83	345	36	106	115	1882

Note: Columns do not add up to 100% because more than one drug class can be found for any one driver.

Table I-5

Drug Class Prevalence Rates Within State Groups*
Drugs in Driver Fatalities Study
n = 1828 Cases

Drug Class	California (5 counties)	Mass.	N.C.	Virginia (15 counties)	Wisconsin	Texas (2 counties)	All Sites
Alcohol (ethanol)	51.4%	52.6%	46.6%	51.8%	49.1%	59.3%	50.9%
Cannabis	9.3	8.1	4.3	6.0	4.5	11.3	6.9%
CNS Stimulants	10.7	4.6	4.9	3.6	2.6	9.5	6.3%
Benzodiazepines	2.5	5.2	5.1	1.2	1.6	4.1	3.3%
Barbiturates	2.0	1.2	1.5	0.0	1.3	1.8	1.5%
Narcotic Analgesics	0.9	0.6	1.1	0.0	0.8	1.8	1.0%
Antidepressants	0.9	1.7	0.8	0.0	0.5	0.9	0.8%
Antihistimines	0.7	0.6	0.6	0.0	0.8	0.9	0.6%
Hallucinogens	0.7	0.0	0.0	0.0	0.3	0.0	0.2%
Antiarrhythmics	0.0	0.0	0.2	0.0	0.0	0.0	0.1%
Muscle Relaxants	0.0	0.0	0.0	0.0	0.3	0.0	0.1%
Nonbarbiturate Sedatives	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Antipsychotics	0.0	0.0	0.0	0.0	0.0	0.0	0.0%
Misc. Other Drugs	1.8	1.7	1.1	1.2	1.8	1.4	1.5%
Total Cases	440	173	530	83	381	221	1828

Note: Columns do not add up to 100% because more than one drug class can be found for any one driver.

*Washoe, Nevada site was excluded from this analysis due to small sample size.

Table I-6

Prevalence Rates Within Mutually Exclusive Substance Categories
Drugs in Driver Fatalities Study

	Alameda	Solano	San Bernadino	San-Diego	Los-Angeles	Massa-achusetts	North Carolina	Washoe	Fairfax	Madison	Milwaukee	Tarrant	Dallas	All Sites
Substance Group														
Drugfree drivers	37.5%	32.0%	50.7%	34.5%	32.3%	39.9%	47.9%	37.0%	43.4%	46.1%	47.2%	30.2%	38.3%	42.7%
Drivers with 1 substance only in system														
Alcohol; BAC = .10	7.5	4.0	4.5	6.9	11.5	4.6	4.7	3.9	12.0	6.7	8.3	9.4	4.3	6.6%
Alcohol; BAC > .10	30.0	32.0	24.6	33.1	36.5	37.0	31.7	40.7	32.5	35.4	36.1	37.7	30.6	33.3%
Delta-9 THC*	0.0	4.0	0.0	3.4	0.0	1.7	0.6	0.0	0.0	1.2	0.0	0.9	1.7	1.0%
Carbony THC**	0.0	0.0	0.0	0.7	0.0	1.2	0.2	0.0	1.2	0.0	0.0	0.9	0.0	0.5%
Stimulant	5.0	0.0	3.0	2.8	2.1	0.0	0.4	0.0	1.2	0.3	0.0	0.9	2.6	1.1%
Other	2.5	4.0	3.0	0.7	3.1	4.6	3.4	1.9	2.4	2.6	0.0	0.9	1.7	2.7%
Drivers with alcohol-drug combination														
Alcohol & delta-9 THC*	2.5	0.0	2.2	2.8	3.1	1.2	0.6	5.6	2.4	1.7	0.0	4.7	4.3	2.0%
Alcohol & carbony-THC**	0.0	4.0	2.2	2.0	1.0	1.7	1.3	0.0	2.4	1.4	0.0	2.8	0.9	1.5%
Alcohol & stimulant only	7.5	4.0	3.7	5.5	4.2	1.7	3.4	1.9	2.4	1.2	5.6	1.9	7.8	3.3%
Alcohol & 1 other not above	2.5	4.0	2.2	0.7	1.0	3.5	2.6	0.0	0.0	1.4	0.8	1.9	2.6	2.0%
Alcohol & 2+ drugs	0.0	4.0	1.5	4.8	2.1	2.9	2.3	3.7	0.8	0.9	2.8	6.6	3.5	2.4%
Drivers with non-alcohol combination														
2 drugs	2.5	4.0	1.5	2.0	3.1	0.0	0.9	0.0	0.0	0.9	0.0	0.0	1.7	1.1%
3+ drugs	2.5	4.0	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.3	0.0	0.9	0.0	0.3%
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0%

*With or without carbony-THC
**Without delta-9 THC

Table I-7

Confidence Intervals for Prevalence Rates*

Instructions: For a given drug prevalence rate at a sample site, find the value in the column labeled "Drug Prev. Rate", and read the 95% or 99% confidence interval from the corresponding columns. Interpolate as necessary. Example: For a 10% prevalence rate in Solano County, the 95% confidence interval is + or - 2.4%.

	Drug Prev. Rate	95% Confidence Interval + or -	99% Confidence Interval + or -
Total Sample (Within site cases only)			
	50.0%	1.3%	1.6%
Sample Size = 1790	40.0%	1.2%	1.6%
Population Size = 2539	30.0%	1.2%	1.5%
	20.0%	1.0%	1.3%
	10.0%	0.8%	1.0%
	5.0%	0.5%	0.7%
	2.0%	0.4%	0.5%
	1.0%	0.3%	0.3%
Alameda County, CA			
	50.0%	9.3%	12.2%
Sample Size = 40	40.0%	9.1%	12.0%
Population Size = 62	30.0%	8.5%	11.2%
	20.0%	7.4%	9.8%
	10.0%	5.6%	7.3%
	5.0%	4.1%	5.3%
	2.0%	2.6%	3.4%
	1.0%	1.9%	2.4%
Solano County, CA			
	50.0%	3.9%	5.1%
Sample Size = 25	40.0%	3.8%	5.0%
Population Size = 26	30.0%	3.6%	4.7%
	20.0%	3.1%	4.1%
	10.0%	2.4%	3.1%
	5.0%	1.7%	2.2%
	2.0%	1.1%	1.4%
	1.0%	0.8%	1.0%
San Bernadino County, CA			
	50.0%	4.3%	5.6%
Sample Size = 131	40.0%	4.2%	5.5%
Population Size = 174	30.0%	3.9%	5.1%
	20.0%	3.4%	4.5%
	10.0%	2.6%	3.4%
	5.0%	1.9%	2.4%
	2.0%	1.2%	1.6%
	1.0%	0.8%	1.1%
San Diego County, CA			
	50.0%	2.4%	3.2%
Sample Size = 145	40.0%	2.4%	3.1%
Population Size = 159	30.0%	2.2%	2.9%
	20.0%	1.9%	2.5%
	10.0%	1.5%	1.9%
	5.0%	1.1%	1.4%
	2.0%	0.7%	0.9%
	1.0%	0.5%	0.6%
Los Angeles County, CA			
	50.0%	8.7%	11.4%
Sample Size = 97	40.0%	8.6%	11.2%
Population Size = 417	30.0%	8.0%	10.5%
	20.0%	7.0%	9.2%
	10.0%	5.2%	6.9%
	5.0%	3.8%	5.0%
	2.0%	2.4%	3.2%
	1.0%	1.7%	2.3%

Table I-7
(continued)

	Drug Prev. Rate	95% Confidence Interval + or -	99% Confidence Interval + or -
Massachusetts (State)			
	50.0%	3.8%	5.0%
Sample Size = 173	40.0%	3.7%	4.9%
Population Size = 234	30.0%	3.5%	4.6%
	20.0%	3.0%	4.0%
	10.0%	2.3%	3.0%
	5.0%	1.7%	2.2%
	2.0%	1.1%	1.4%
	1.0%	0.8%	1.0%
North Caroline (State)			
	50.0%	1.9%	2.5%
Sample Size = 530	40.0%	1.9%	2.5%
Population Size = 668	30.0%	1.8%	2.3%
	20.0%	1.5%	2.0%
	10.0%	1.2%	1.5%
	5.0%	0.8%	1.1%
	2.0%	0.5%	0.7%
	1.0%	0.4%	0.5%
Washoe County, NV			
	50.0%	0.0%	0.0%
Sample Size = 41	40.0%	0.0%	0.0%
Population Size = 37	30.0%	0.0%	0.0%
	20.0%	0.0%	0.0%
	10.0%	0.0%	0.0%
	5.0%	0.0%	0.0%
	2.0%	0.0%	0.0%
	1.0%	0.0%	0.0%
Northern Virginia			
	50.0%	5.3%	6.9%
Sample Size = 83	40.0%	5.2%	6.8%
Population Size = 109	30.0%	4.8%	6.3%
	20.0%	4.2%	5.5%
	10.0%	3.2%	4.2%
	5.0%	2.3%	3.0%
	2.0%	1.5%	1.9%
	1.0%	1.1%	1.4%
Wisconsin (State)			
	50.0%	2.1%	2.7%
Sample Size = 381	40.0%	2.0%	2.7%
Population Size = 459	30.0%	1.9%	2.5%
	20.0%	1.7%	2.2%
	10.0%	1.2%	1.6%
	5.0%	0.9%	1.2%
	2.0%	0.6%	0.8%
	1.0%	0.4%	0.5%
Tarrant County, TX			
	50.0%	2.1%	2.7%
Sample Size = 81	40.0%	2.0%	2.7%
Population Size = 84	30.0%	1.9%	2.5%
	20.0%	1.7%	2.2%
	10.0%	1.2%	1.6%
	5.0%	0.9%	1.2%
	2.0%	0.6%	0.8%
	1.0%	0.4%	0.5%

Table I-7
(continued)

	Drug Prev. Rate	95% Confidence Interval + or -	99% Confidence Interval + or -
Dallas County, TX			
	50.0%	8.1%	10.6%
Sample Size = 63	40.0%	7.9%	10.4%
Population Size = 110	30.0%	7.4%	9.7%
	20.0%	6.5%	8.5%
	10.0%	4.9%	6.4%
	5.0%	3.5%	4.6%
	2.0%	2.3%	3.0%
	1.0%	1.6%	2.1%

*Note: The confidence intervals shown are based on the obtained sample sizes and the size of the FARS driver population eligible for the study. Both are counted within the geographical boundaries of each site. The confidence intervals pertain only to prevalence rates based on the samples from within those boundaries, i.e., the weighted prevalence rates. Confidence intervals for the samples including cases from outside the site boundaries are not provided, for the populations they represent are ambiguous.

Appendix J

Derivation of Relative Risk from Responsibility Rates

From Terhune (1983), we get the following:

Estimated relative risk of causing a crash with $drug_i$=

$$\frac{\text{No. responsible drivers with } drug_i \times \text{No. nonresponsible drugfree drivers}}{\text{No. nonresponsible drivers with } drug_i \times \text{No. responsible drugfree drivers}} \quad \text{(J1)}$$

Let A= No. responsible drivers with $drug_i$
B= No. nonresponsible drivers with $drug_i$
C= No. responsible drugfree drivers
D= No. nonresponsible drugfree drivers

Equation (J1) becomes $(A/B)*(D/C)$ (J2)

Now, the responsibility rate of the drugfree group=

$$r_o = C/(C + D)$$

and $$1/r_o = 1 + D/C \quad \text{(J3)}$$

Similarly, the responsibilty rate of the $drug_i$ group=

$$r_i = A/(A + B)$$

and $$1/r_i = 1 + B/A \quad \text{(J4)}$$

From (J3) we get, $D/C = (1 - r_o)/r_o$ (J5)

From (J4) we get, $A/B = r_i/(1 - r_i)$ (J6)

Entering equations (J5) and (J6) into (J2),

Estimated relative risk of causing a crash with $drug_i$=

$$\frac{r_i\,(1 - r_o)}{r_o\,(1 - r_i)}$$

Appendix K

Distribution of SUBSAMPL Drivers in Relation to Crash Responsibility

Substance Group	Responsibility Group: Responsibility = 0-2	Responsibility = 3-4
Drugfree drivers	70.3%	35.1%
Drivers with 1 substance only in system		
Alcohol: BAC <0.10%	8.7	6.5
Alcohol: BAC ≥0.10%	10.8	38.8
Delta-9 THC[1]	1.6	0.7
Carboxy THC[2]	0.2	0.2
Cocaine/Benzoylecgonine	0.7	0.4
Amphetamines	0.2	0.8
Other[3]	3.5	2.6
Drivers with alcohol-drug combination		
Alcohol & delta-9 THC[1]	0.2	2.5
Alcohol & carboxy-THC[2]	0.4	1.8
Alcohol & cocaine/benzoylecgonine	1.6	3.4
Alcohol & amphetamines	0.2	0.7
Alcohol & 1 other not aboye[4]	0.0	2.3
Alcohol & 2 or more other[5]	0.4	2.7
Drivers with non-alcohol combinations		
2 drugs[6]	0.5	1.3
3 or more drugs[7]	0.5	0.2
Total	100.0%	100.0%

Notes

[1]With or without carboxy THC

[2]Without THC

[3]Includes benzodiazepines (18 drivers), barbiturates (6), antihistamines (6), narcotic analgesics (5), and miscellaneous others

[4]Includes benzodiazepines (18 drivers), barbiturates (8), antihistamines (3), and miscellaneous others

[5]Includes cannabis (31 drivers), cocaine (21), benzodiazepines (21), barbiturates (7), and miscellaneous others

[6]Includes barbiturates (7 drivers), benzodiazepines (5), cannabis (4), amphetamines (4), and miscellaneous others

[7]Includes amphetamines (4), cocaine (3), cannabis (2) and miscellaneous others.

*U.S. G.P.O.:1994-301-717:80492