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DRUG INVOLVEMENT IN FATAL CRASHES IN MELBOURNE

Author

HENDTLASS, JANE.

Performing Organisation

VICTORIA POLICE,
380 WILLIAM STREET,
MELBOURNE, VICTORIA, 3000,
AUSTRALIA.

Sponsor

FEDERAL OFFICE OF ROAD SAFETY,
P.O. BOX 594.
CIVIC SQUARE, A.C.T., 2608,
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Abstract

Information about drug use was collected from drivers and pedestrians involved in road crashes in Melbourne in which a driver or pedestrian was killed. This involved analysis of body fluid from 191 fatalities combined with other information about drug use from police files. 44% had used drugs *other* than alcohol, caffeine *and* nicotine. Among fatalities, 55% of drivers or riders in single vehicle crashes, 77% of drivers or riders in multivehicle *crashes* and 61% of pedestrians had used drugs. cannabis was the most frequently detected drug other than alcohol, caffeine and nicotine, followed by aspirin.

KEYWORDS Fatal Crashes, Drugs, Alcohol, Drivers, Motorcycle Riders, Pedestrians, Cannabis

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DRUG INVOLVEMENT IN FATAL CRASHES IN MELBOURNE.

JANE HENDTLASS

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Jane Hendtlass,
8 Tyne Street,
Burwood,
Australia 3125.

Definitions

Definitions of terms used in this report:-

Operator	Any driver, motor cycle rider or pedestrian involved in a fatal crash.
Smoker	Any driver, motor cycle rider or pedestrian known to smoke cigarettes, cigars or a pipe or in whom nicotine was detected.
Drinker	Any driver, motor cycle rider or pedestrian known to have been drinking or having a blood alcohol concentration over 0.010g/100 mls.

Abbreviations

Abbreviations used in this report:-

ABS	Australian Bureau of Statistics.
CNS	Central Nervous System.
GC	Gas Chromatography.
LSD	Lysergic acid diethylamide.
MS	Mass Spectrometry.
NH_3	Ammonia.
NHTSA	National Highway & Transport Safety Administration.
No.	Number.
RUM	Road User Movement.
THC	Tetrahydrocannabinol.
U.S.,U.S.A.	United States of America.

Executive Summary.

Many drugs have been shown to impair performance of driving-related skills but the way in which they contribute to road crashes has yet to be established.

The Aim of this study was to determine the incidence of drug use among drivers, motor cycle riders and pedestrians involved in fatal crashes.

The study population comprised 1115 drivers, motor cycle riders and pedestrians involved in a crash in metropolitan Melbourne between April 1980 and April 1982 in which a driver, motor cycle rider or pedestrian died.

The Study collected information about drug use from 225 driver and pedestrian fatalities and 177 survivors of fatal crashes. This involved analysis of body fluid from 191 fatalities combined with other information about drug use from police accident records.

Data about the characteristics of the crash and the operator were also collected.

Results of this survey showed that:

- 44% of the study population had used drugs other than alcohol, caffeine and nicotine. This figure included 55% of drivers or motor cycle riders in single vehicle crashes who died at the scene; 77% of drivers or motor cycle riders in multivehicle crashes who died at the scene; and 61% of pedestrians who died at the scene.

- 11% of the study population had used alcohol in combination with other drugs. A further 21% had used alcohol but no over-the-counter, prescription or illicit drugs were detected.

- 36% of drug users identified in the study population had used more than one substance, excluding alcohol, caffeine and nicotine.

- 32% of the substances detected in driver, motor cycle and pedestrian fatalities were over-the-counter drugs and 46% were drugs which are normally only available under medical supervision.

Cannabinoids had been used by 20% of driver, motor cycle rider and pedestrian fatalities.

Among drug positive fatalities, cannabinoids were detected in 34% of those aged under 25 years and 43% of those aged 25 to 44 years.

37% of driver, motor cycle rider and pedestrian fatalities had used drugs which are known to impair driving-related skills in healthy individuals.

Recommendation 1.

The Federal Office of Road Safety should associate itself with existing organisations involved in designing drug control measures, such as the Federal Department of Health, the various State Police Forces and Education Departments and the Australian Institute of Criminology, in an effort to co-ordinate measures to reduce drug consumption in the community.

Recommendation 2.

An education and awareness programme for medical officers and pharmacists should be established to inform them of the effects which some medicines may have on driving-related skills and the alternative products which are available.

This programme could be administered through the Australian Medical Society on Alcohol and Drug Related Problems and the Pharmaceutical Society of Australia.

Recommendation 3.

Further research should be undertaken into the crash risk of epileptic and diabetic drivers, the effect of available measures to control these conditions on their risk and on driving-related skills, and the most constructive ways of ensuring that these patients do not put other road users unnecessarily at risk.

Recommendation 4.

The role of cannabis in road crashes should continue to be monitored and any attempts to decriminalise the drug should be drafted to include provisions which

maintain and reinforce the social irresponsibility of driving after its use.

One practical way of implementing this recommendation could include proscription of carriage of cannabis in the passenger compartment of motor veh'icles.

Chapter One.

Introduction

Injury sustained in road crashes is now the most frequent cause of death among Australian males aged 1 to 44 years (Australian Bureau of Statistics, 1985). This is due in part to higher exposure resulting from the increased mobility afforded by motor vehicle transport and the consequential change in our social behaviour patterns compared with even fifty years ago.

The relationship between alcohol consumption and road crash potential has been recognised in legislation which has been passed in most western countries from as early as the middle of the last century (Brown, 1983) and road safety efforts frequently emphasise the need to change drink driving behaviour (e.g. Borkenstein et al, 1964; Ross, 1982).

However, the ways in which use of drugs other than alcohol may influence road safety are not well understood and even the incidence of drug use among road users is still open to conjecture because the figures which are cited

depend so much on the way in which they were collected (U.S. Department of Transportation, 1979; Donelson, 1981).

Interpretation of the information which is available about particular drugs in terms of their effect on road crashes is further complicated by the frequent multiple drug use by individuals in the community (e.g. Whitehead & Ferrence, 1976). This means that it is difficult to categorise the influence which individual substances may have on the crash risk of those to whom they are administered.

The Ministry *of* Police & Emergency Services in Victoria was commissioned by the Federal Office of Road Safety, Department of Transport, Canberra, to undertake the survey of the incidence of drug use among drivers and pedestrians involved in fatal crashes which is described in this report. The work was undertaken with the co-operation *of* Victoria Police, the State Laboratory of Victoria and the Melbourne City Coroner (see Appendix G for detail of the financial arrangements).

Information which is available about the ways in which drugs other than alcohol may influence road crashes has been reviewed in Chapters 2 and 3.

Drugs are defined as ANY SUBSTANCE WHICH IS CONSUMED BY THE USER IN AN ATTEMPT TO CHANGE HIS EXISTING PHYSICAL, MENTAL OR EMOTIONAL STATE. This includes:

social drugs such as alcohol, tobacco and caffeine,

over-the-counter drugs which are sold in food outlets and pharmacies and are taken for medicinal purposes such as aspirin, paracetamol and cough medicines;

prescription drugs which are normally available in Australia only under express direction of a medical officer; and

illicit drugs which are either completely banned such as heroin, marijuana and cocaine, or only legally available with a special Health Department permit such as amphetamines.

Also reported are the results of a study of drug use among active participants in road crashes in the metropolitan Melbourne coronial district in which a driver or pedestrian died between April 1980 and April 1982.

Chapter 4 describes the procedures used in the survey and Chapter 5 describes the study population.

Information about use of over-the-counter, prescription and illicit drugs by the study population has been reported in Chapter 6, separately from that describing use of the social drugs alcohol, nicotine and caffeine in Chapter 7.

All these data have been interpreted in the context of drug use in the Australian community in Chapter 8 and recommendations for relevant road safety initiatives have been developed in the light of existing drug control procedures.

Chapter Two.

The Incidence of Drug Use among Road User Casualties - A Literature Review.

Estimates of the incidence of drug use among crash victims are usually obtained by analysis of body samples, from reports in police files or from self-reports by survivors.

Analyses generally understate the number of people who have used drugs because the pharmacologically active levels in blood, urine or breath can be below the sensitivity of the analytical procedure used (U.S. Department of Transportation, 1979).

From the other point of view, metabolites of some drugs, for example methadone and cannabis, can accumulate in the tissue and can be still detected three weeks after last use and after the effects of the parent drug have dissipated (Chesher, 1984).

Surveys which rely on self-reported data yield figures for the frequency of drug use which differ from those based on analytical procedures (Haffner & Lunde, 1974; Ryan, 1978; Macpherson et al, 1983;

Hendtlass, 1983; Bailey, 1984).

Those based on prescription records are not able to take the incidence of multiple drug use or non compliance with medical directions into account (e.g. Maki & Linnoila, 1976; Maddux et al, 1977; Jick et al, 1981).

2.1. Analytical Methods For Determining Presence of Drugs.

Chemical analysis of drugs in biological fluid generally involves extraction of the drugs into solvents which can be introduced into the instruments which separate the components from each other. These drugs are then detected and identified by procedures specific for the substances under consideration.

Although procedures for determining the concentration of particular drugs in blood, urine or saliva are well established and validated, few laboratories in Australia administer routine multidrug screening procedures adequate for law enforcement requirements. Racing laboratories are secretive about their methodology and most hospital and university laboratories cater for high concentrations encountered in overdose patients or for monitoring of particular therapeutic drugs administered

on a continuing basis. Screening of drug dependents undergoing treatment is usually restricted to drugs of abuse.

The procedures used by those attempting to implement routine drug screening procedures for road users must assume that false negatives are as important as false positives because they distort the statistics documenting justification for the development of countermeasures. It has also been assumed that the methods to be used will be open for public scrutiny in the courts; they must be validated and able to be interpreted by analytical chemists as well as other practitioners.

These factors make it relevant to point out several differences between alcohol analysis and multidrug screening:

1. Most drugs are not highly volatile, are active at a low blood concentration and do not lend themselves to analysis directly from biological fluid. Alcohol is volatile and can be analysed directly from blood or urine.

2. Most drugs have several, often many, breakdown products and some of these may be more concentrated an/or more psychotropically active than their parent. Alcohol has only one identifiable breakdown product and this can be expected to have little effect on psychomotor skills because it occurs in low concentrations except when some other medication influences its metabolism.

3. At therapeutic doses the molar concentration of most drugs in the blood is in the same range as that of other normal body compounds. Alcohol requires relatively high levels for it to effect measurable changes in performance of driving related skills.

4. Many drugs have chemical structures which are very similar to normal body substances and to each other. It is often difficult to be sure that the compound is a drug and if it is, that it is identified correctly. Intimate knowledge of each drug's metabolic pathway is essential in its identification.

5. Many drugs are bound to salts, sugars or proteins and are not readily extractable from body fluid without hydrolysis. Further, it is difficult to be sure whether the blood level measured after a therapeutic dose which is reported in the literature is based on hydrolysed or unhydrolysed samples.

6. Different drugs are not always analytically similar to each other, even if they have the same type of pharmacological effect or come from the same chemical base. This means that they will be different in the way *in* which they respond to extraction and detection procedures. These procedures need to be validated for each particular substance.

There are therefore two main ways of approaching the establishment of drug screening procedures:-

<u>Specific</u> screening procedures for particular compounds or groups of compounds generally use particular extraction and detection methods for each drug or chemically similar group.

Most of these procedures use gas chromatography or high pressure liquid chromatography to separate out the sample components (e.g. Garriott & Latman, 1976; Stevens 1984). Many now rely on mass spectrometry for confirmation of the substanne identity. This is generally considered essential for samples required as evidence in a court of law (e.g. Folz et al, 1980; Vine & Watson, 1982; Crouch et al, 1983; Zimmerman et al, 1984).

This type of procedure will therefore detect drugs at a relatively high minimum level but will identify accurately those which it does detect.

General screening procedures cater for a wide range of drugs at lower concentrations than those detectable by so-called specific screening procedures and in a small volume of body fluid. They use immuno-assay, chemical ionisation, thin layer chromatography or other less specific techniques to detect drug components. This means

that identification of the detected compounds is less likely to be accurate than in the specific procedures unless they revert to mass spectrometry for confirmation (e.g. Adams, 1972; Teale et al, 1978; Smith, 1980; Jeffcoat, 1981; Hawks, 1982; Owens et al, 1983; Peel et al, 1984).

2.2. General Drug Use in Crash Victims.

Several studies have estimated general drug use among driver and pedestrian casualties, Results of this work are summarised in the accompanying Table.

The most extensive of these was performed by Cimbura and his co-workers (Cimbura et al, 1980; Warren et al, 1980) in Ontario, Canada. They analysed blood samples taken from 484 driver and pedestrian fatalities and found that 26% had used drugs other than alcohol prior to the fatal crash. This figure included 26% of drivers and 29% of pedestrians. The most frequently reported drugs were cannabinoids, salicylates and diazepam.

The Centre for Human Toxicology in Salt Lake City has analpsed blood from 440 young male driver fatalities from California (Williams et al, 1985). They found that 70% of these drivers had used alcohol and 41% had used drugs other than alcohol. In 43% two or more drugs were detected. The most frequently reported drugs were cannabinoids, cocaine and salicylates.

Earlier American studies (Davis, 1974; Turk et al, 1974; Blackburn & Woodhouse, 1977) reported drugs in 5% to 10% of driver fatalities with phenylpropanolamine and chlorpheniramine most frequent. Before that time, others had found barbiturates in around 9% of driver fatalities in the United States (Briglia, 1966; Californian Highway Patrol, 1967).

Vine and Watson (1982) have published data from analysis of the blood of 425 driver and pedestrian fatalities in Sydney. They found that 10% had used drugs other than marijuana, aspirin and paracetamol and a further 6% had used aspirin. The most frequently detected drug was diazepam.

A New Zealand survey (Bailey, 1984) found that, by analysis, 2% of injured drivers and 5% of injured pedestrians *had* used drugs other than morphine, lysergic acid diethylamide, amphetamines, paracetamol, salicylates, low dose phenothiazines and antihistamines and drugs not containing nitrogen, phosphorus or halogens. Self reports gave much higher figures *for* drug use.

Other surveys of general drug use in driver casualties have been undertaken in the United States of America and New Zealand.

Studies of the involvement of cannabis have suggested that over 22% of driver casualties in Sydney have used this drug in the last two days (Chesher &

Use of Drugs Other than Alcohol by Driver & Pedestrian Casualties.

Place	Year	Drug Type	Drivers	Pedestrians	Inj
Australia.					
Melbourne (Ryan,1978)		Prescribed	17%	-	Fatali
Sydney (Vine & Watson, 1982)	1980-82	Comprehensive	10%		Fatal
		Aspirin	6%		
Sydney (Chesher & Starmer,1983)	1983	Cannabinoids	22%	-	Hospit ised.
Tasmania (McLean et al,1985)	1983-4	General	36%	-	Fatals
New Zealand.					
New Zealand (Missen et al, 1978a)	1971-74	Diazepam	2%	-	Hospit
New Zealand (Missen et al, 1978b)	1977-78	Prescription	15%	30%	Fatali
New Zealand (Missen & Wardill, 1981)	1979-80	Prescription	2.2%	-	Hospit ised R Users
New Zealand (Cairns et al, 1984).	1979-80	Prescription	1.8%	4.5%	Fatals
New Zealand (Bailey,1984)		Prescription	24%	33%	Injure
United States & Canada.					
Sacramento, U.S.A. (Briglia, 1966)		Barbiturates	9.3%	-	Multip Vehicl Fatals
California, U.S.A. (California Highway Patrol, 1967)		Barbiturates & Tranquilisers	9%	-	Single Vehicl Fatali
Kansas, U.S.A. (Blackburn & Woodhouse 1977).		General	6%	-	Driver Fatali

Place	Year	Drug Type	Drivers	Pedestrians	Injury
Florida U.S.A (Davis, 1974)	1956-68	Several	5.6%	-	Fatals.
North Carolina U.S.A., (Turk et al, 1974)	1972	Comprehensive	5.1%	21.3%	Fatals.
Ontario (Cimbura et al, 1981)	1978-79	Comprehensive	26%	29%	Fatal
U.S.A., (N.H.T.S.A.,	1982)	Marijuana	18% 10%	-	Hospital
North Carolina (Owens et al, 1983).	1978-79	Cannabinoids Amphetamine Phenylpropanolamine	5.9% - -	-	Single Vehicle Fatals
North Carolina (Mason & McBay, 1984)		Cannabinoids Methaqualone Barbiturates	8% 6% 3%	- - -	Single Vehicle Fatals
California (Williams et al, 1985)		Cannabinoids Cocaine Other	37% 11% 35%	- - -	Fatals Age 15-34 Years
Europe.					
Finland (Honkanen et al, 1980).		Psychotropic	5.0%	-	Emergency Departmen
Norway (Bø et al, 1974)		Diazepam	18%	-	Hospitalised Road Users
England & Wales (Teale et al, 1978)		Cannabinoids	9%	-	Fatal

Starmer,1983). This is much higher than casualty figures from the United States and Great Britain (Teale et al, 1978; Owens et al, 1983). However, it is lower than the 37% incidence of cannabinoids found in 15 to 34 year old driver fatalities in California (Williams et al, 1985).

2.3 Estimates of Crash Risk.

No information has come to notice which adequately allows comparison of drug use by crash victims with a control group of uninjured road users. The direct relationship between drug use and crash risk is therefore unknown.

Estimates based on surrogate measures of drug use give conflicting results. For example in America and Scandinavia the frequency of use of central nervous system depressant drugs among road accident casualties was similar for drivers presumed at fault and Sor passengers and other drivers (Honkanen et al, 1980; Jick et al, 1981; Solarz ,1983).

In contrast, Skegg et al, (1979) found increased risk of accidents when tranquiliser prescribees were compared with the general population and amphetamine users have four times the crash rate of non-users (Smart et al, 1969).

Further, among psychiatric out-patients, those taking medication have a higher crash rate than non-medicated patients (Maki & Linnoila, 1976).

An Australian Study (Macpherson et al, 1982) has implicated analgesics, central nervous system depressants, diabetic agents and anti-arthritic agents in predisposition to crash incidence among drinking drivers.

2.4. Summary.

Analysis *of* alcohol in body fluids is easier and more reliable than measurement of other drugs in driver and pedestrian casualty populations.

Reported use of drugs other than alcohol among driver and pedestrian casualties

ranges from 15% to 40% for those studies based on comprehensive screening procedures.

Cannabis is consistently the most frequently reported drug followed by aspirin.

Chapter Three.

The Effects of Drugs on Driving-Related Skills.

Most of the work which describes the effects of drugs on driving-related skills has involved controlled environment studies such as those performed in laboratories, using driving simulators or on closed driving circuits (e.g. Ashworth, 1975). Different drugs have been found to have different effects on driving-related skills such as reaction time, peripheral vision and divided attention tasks.

The relevance of these results to real life driving experience Is difficult to interpret. For example, experimental requirements which are commonly imposed in drug taking studies may give different results from those obtained if the subjects use the substances according to their own patterns (Morgan & Pickens, 1982; Healey & Pickens, 1983). Further, the relevance of performance of laboratory 'tests or driving simulator tasks to actual driving behaviour has yet to be determined (Naatanen & Summala, 1976; de Gier, 1984; Smiley, 1984).

Studies of the effects of particular substances usually describe healthy young volunteers whereas use of most medicinal preparations is more frequent among the elderly than in the young population (Australian Bureau of Statistics, 1979). The reactions of elderly or ill subjects to particular preparations are often different from those in the healthy or young.

Men and women are also likely to respond differently to different drugs (Clayton et al, 1974; Clayton, 1976; Legg et al, 1973; Peeke et al, 1979; Seixas, 1979; Wetherell, 1979; Spiegel, 1982; Thompson & Triable, 1982; Ellinwood & Heatherley, 1985; Roth & Roehrs, 1985).

In part these differences may be explained by the effects of age, exposure or metabolism of drugs. Distribution patterns for drugs throughout the body are also influential (Greenblatt, 1980). Fatigue, personality, emotional state and mental illness can also affect responses (Linnoila, 1974; Maki & Linnoila, 1976; Peeke et al, 1980; Hobi et al, 1982).

As well, metabolites of many drugs are active and can influence interpretation of results (e.g. Palva & Linnoila, 1978; Bird et al, 1980; Crevoisier, et al, 1983).

The effect of interaction with other *drugs,* especially alcohol, on rate and pattern of absorption and metabolism is also complicated (e.g. Morselli et al, 1971; Mould et al, 1972; Coleman & Evans, 1975; Hemming et al, 1981).

The relationship between accident risk and use of drugs other than alcohol therefore is largely conjectural and will remain so until controlled studies can be undertaken involving road user populations similarly at risk of but not involved in crashes (Waller, 1971; Clayton, 1976; Joscelyn & Donelson, 1981; Friedel, 1979; *U.S.* Department of Transportation, 1979; N.H.T.S.A., 1980).

Information about the effects of particular substances on driving-related skills is summarised in this chapter

according to the pharmacological action of the drug which is most frequently reported. Detailed tables of this information are contained in Appendix A. Some of these data have already been recently reviewed (Ashton, 1983).

Alcohol and other drug combinations can interact through either:-

a) having similar effects on driving-related skills and in this case the effects are <u>additive</u> (e.g. Linnoila, 1973b); or

b) having the opposite effect on driving-related skills and in this case the effects are <u>antagonistic</u> (e.g. Bird <u>et al,</u> 1980);

c) decreasing the metabolism of the other drug so that their combined effects on driving-related skills will be <u>synergistic</u> i.e. greater than additive (e.g. Hart <u>et al,</u> 1976);

d) increasing the metabolism of the other drug so that their combined effects on driving-related skills will be <u>less than additive</u> (e.g. Kato & Vassanelli, 1962).

Effects of interactions of particular substances with alcohol are described in the text appropriate to the relevant drug and are summarised in Appendix B.

3.1. Effects of Stimulants on Driving-Related Skills. (Table A1).

Stimulant drugs range from illicit and permit compounds such as amphetamine and cocaine to the social beverage, caffeine.

Most research on the effects of stimulants focuses on amphetamines or caffeine and this section addresses the former. Information about caffeine is included with that describing alcohol and nicotine in Chapter 7.

Amphetamines appear to-cause greater misjudgement of speed than alcohol (Ashworth, 1975) but otherwise they may even enhance driving-related skills, particularly in those deprived of sleep. However, amphetamine abusers appear heavily overrepresented among accident involved drivers (Smart et al, 1969).

Data regarding the effect of amphetamines on alcohol induced impairment are inconsistent but generally impairment seems less than that observed with alcohol alone. This depends

on the measures used and the degree of tiredness of the subjects (Burst, 1976).

3.2. Effects of Minor Analgesics on Driving-Related Skills (Table A2).

Common over-the-counter analgesics differ in their effects on driving-related skills. Acute administration of aspirin equivalent to 3 tablets has no demonstrable effect whereas indomethacin and phenylbutazone impair performance of co-ordination and divided attention tasks (Linnoila et al, 1974b).

Aspirin has no significant effect on alcohol induced impairment, indomethacin antagonises its effects while the effects of phenylbutazone and alcohol are additive (Linnoila et al, 1974b).

3.3. Effects of Hypnotics, Tranquilisers, Sedatives & Antipsychotics on Driving-Related Skills.

There is a wide range of hypnotics, tranquilisers and sedatives available with diverse effects on driving-related skills.

Anxious and schizophrenic patients under appropriate long term medication have low performance of driving-related skills compared with healthy non-medicated volunteers but this deficit may be caused by either the medication or the disease or both (Hobi et al, 1982).

3.3.1. Benzodiazepines (Table A3):-

Benzodiazepines are known to generally but not always reduce psychomotor performance and to interact with alcohol (e.g. Neuteboom 6 Zweipfennig, 1984; Ellinwood 6 Heatherley, 1985).

Inconsistencies between reports of the effects of these drugs may result from differences in performance measures, protocols for drug administration and characteristics of subjects used in various studies. They may also reflect real variation in the effects of drugs on different people.

Chlordiazepoxide (e.g. Librium) does not seem to affect psychomotor performance at low doses (40mg), but at higher doses the drug does reduce reaction time and other psychomotor skills (Idestrom & Cadenius, 1963).

Chlordiazepoxide does not generally reverse the effect of alcohol but in women it may be antagonistic (Dundee et al, 1971; Linnoila, 1973b; Ashworth, 1975).

Acute doses of diazepam (e.g. Valium) decrease performance of vigilance, choice, divided attention and co-ordination tasks (Linnoila, 1973b; Ashworth, 1975). Peak effects occur about twenty minutes after ingestion (Ellinwood et al, 1981) and some of these effects are still apparent the following morning (Willumeit et al, 1984).

Although performance continues to be reduced during administration of the drug for at least nine days (Moskowitz & Smiley, 1982) some tolerance has been shown to develop after two weeks and the drug may even improve co-ordination and choice tests measures by that time (Linnoila et al, 1974a).

However, de Gier and his coworkers found that patients routinely administered diazepam at therapeutic levels continued to show reduced psychomotor skills and real driving performance when compared to a

control group (de Gier et al, 1981; de Gier, 1984). These effects are greater in men than they are in women taking oral contraceptives and, *among* women, they are less on day 10 than day 28 of the menstrual cycle. The differences seem to be due to differing absorption rates.

Further, acute and chronic use of diazepam increases the effects of alcohol on psychomotor performance and potentiates its sleep inducing properties in women (Dundee et al, 1971; Linnoila, 1973b; Linnoila et al, 1974a; Mørland et al, 1974; Moskowitz & Smileg, 1982).

Nitrazepam (e.g. Mogadon) is a commonly used hypnotic which affects the psychomotor performance of young, healthy men for over 13 hours after a single dose (Idestrom & Cadenius, 1963; Linnoila & Mattila, 1973; Hossman et al, 1980). However, there is little change in the motor skills of anxious patients (Legg et al, 1973).

Temazepam is often used as a sedative or hypnotic. Temazepam affects reaction time and other tasks among elderly patients and healthy volunteers. Both nitrazepam and temazepam tend to accumulate in elderly patients because they are eliminated *or* metabolised more slowly than in healthy, young male or female volunteers taking the same dose. This means that their effects can be greater the longer the dose is maintained. This effect is related to diurnal rhythm (Cook et al, 1983; Subhan.. & Hindmarch, 1983; Wesnes & Warburton, 1984).

Lorazepam and midazolam are short acting benzodiazepines which are used as sedatives and appear to have little residual affect on psychomotor function the following morning (Subhan & Hindmarch, 1983; Hindmarch & Subhan, 1983; Willumeit et al, 1984). However, lorazepam impairs ability to perform driving related tasks when administered acutely (Mattila et al, 1982). Its effects interact with alcohol involvement.

Medazepam affects some skills in anxious patients but not others (Moore, 1977).

Flurazepam is also used as an antianxiety drug and a sedative. It can affect motor task performance and cognitive function and these effects are demonstrable the following morning in healthy volunteers (Seppala et al, 1983; Willumeit et al, 1984; Wesnes & Warburton, 1984). However these affects are not always evident in chronic insomniacs (Hindmarch & Gudgeon 1982; Mattila et al, 1982; Mendelson et al, 1982; Moskowitz & Smiley, 1982; O'Hanlon et al, 1983).

The active metabolites of benzodiazepines, oxazepam (also marketed as Serepax), methyloxazepam and chlordiazepoxide lacten, all contribute to the effects of the parent drugs, alone and in combination with alcohol, on psychomotor skills. N - desmethyl diazepam and another metabolite, benzodiazepin, are not similarly effective (Palva et al, 1976; Palva & Linnoila, 1978; O'Hanlon et al, 1983).

These effects seem to be related to the blood alcohol concentration rather than to any metabolic consequences of the drug combination (Laisi et al, 1979).

3.3.2. Barbiturates (Table A4):-

Barbiturate abusers have lower accident rates than expected for their age and sex groups (Smart et al, 1969) even though all barbiturates reduce performance of driving-related skills in healthy volunteers. However, amylobarbitone had no effect on tests performed by anxious patients (Legg et al, 1973).

The effects of barbiturates on reaction time appear to last for some time after a single dose and interaction with alcohol is usually potentiating (Joyce et al, 1959; Sharma, 1976).

Chronic alcohol use decreases the rate of metabolism of phenobarbital (Kalant et al, 1970). However, among alcoholics receiving phenobarbital which is withdrawn, there is a marked decrease in psychomotor performance.

3.3.3. Other Hypnotics, Tranquilisers, Sedatives & Antipsychotics (Table A5):-

Haloperidol (Serenace) does not seriously affect frequency of flicker fusion, divided attention or co-ordination tasks and does not interact with alcohol though the effects at high doses can be additive (Linnoila, 1973b; Ashworth, 1975).

Thioridazine (Melleril) affects divided attention tasks but not co-ordination or choice reaction tests. Its effects are additive with those of alcohol (Linnoila, 1973b).

Glutethimide (Doriden) reduces the performance of several psychomotor tasks and the degree of this impairment is additive in combination with alcohol (Mould et al, 1972).

Buspirone and loprazolam are used as anti-anxiety agents. Neither display significant effect on psychomotor skills when administered to healthy subjects with or without alcohol; alcohol can even improve the performance of chronic buspirone users (Mattila et al, 1982; Moskowitz & Smiley, 1982).

Chlorpromazine (Largactil) reduces performance of driving-related skills in healthy volunteers (Zirkle et al, 1959; Manton, 1977; Hartley et al, 1978; Liljeuquist et al, 1978; Smart, 1978). The newer drugs, midazolam and zopiclone, have similar effects (Hossman et al, 1980; Crevoisier et al, 1983; Hindmarch & Subhan, 1983; Seppala et al, 1983). However, nomifensine and HOE 8476 have little effect on performance (Hindmarch, 1977a; Parrott et al, 1982).

3.4. Effects of Anticonvulsants on Driving-Related Skills. (Table A6)

Anticonvulsant drugs are routinely prescribed to control seizures among epileptics and those who have undergone head injury or intercranial surgery. Some epileptics have a greater risk of accidents and traffic violation but this seems limited to those who take insufficient medication, have additional alcoholism or suffer epileptic personality change (Ritter, 1976; cited in Hobi, 1982).

The role of anticonvulsants in road accidents must be seen from two viewpoints. On one hand, several anti-epileptic drugs such as phenytoin, clobazam, sodium valproate and

carbamazepine are known to impair performance of psychomotor tasks among epileptics and healthy individuals (Trimble k Thompson, 1981). Similarly, phenobarbital is sometimes used as an anticonvulsant and it also impairs driving-related skills in healthy volunteers. Carbamazepine has a lesser effect than the other drugs.

On the other hand, blood concentrations below the therapeutic range may induce seizures which, of themselves may lead to road accidents (Missen et al, 1978b). A survey of epileptic patients transported by ambulance has shown that 20% were driving a motor vehicle when they had their seizure (Stanaway et al, 1983). Protocols have been drawn up to guide prescribers in their assessment of medication requirements in terms of seizure control vis à vis psychomotor impairment. The fact that this sort of procedure is needed leads to severe doubts about the wisdom of allowing medicated epileptics to drive at all.

3.5. Effects of Antidepressants on Driving-Related Skills. (Table A7).

Clinically depressed patients are routinely treated with chronic anti-depressant medication which can be grouped into two categories, monoamine oxidase inhibitors and tricyclic antidepressants.

Clinically depressed patients under appropriate long term medication with anti-depressants appear to have similar psychomotor performance to medicated healthy controls. Some deviation is demonstrated by patients prescribed mixed or varying psychopharmacotherapy but this has been attributed to physicians' judging therapeutic success less favourably (Hobi et al, 1982).

There seems to be a generally positive relationship between improvement in this psychomotor performance and improvement in clinical symptoms among depressed patients (Hobi et al, 1982; Thompson & Trimble, 1982; Seppala & Linnoila, 1983; Linnoila & Seppala, 1985).

The effects of antidepressants on cognitive function have been reviewed by Thompson & Triable (1982) and Seppala & Linnoila (1983; Linnoila & Seppala, 1985).

Acute doses of tricyclic antidepressants other than amoxapine reduce performance of psychomotor skills and increase the effects of alcohol (Landauer et al, 1969; Ban et al, 1982; Linnoila et al, 1983).

Mianserin also interacts with alcohol (Seppala, 1977) but amoxapine and zimeldine do not have these effects (Ban et al, 1982; Seppala & Linnoila, 1983; Linnoila et al, 1983).

Tolerance to amitriptyline does not reduce this impairment of performance whereas the effects of mianserin, doxepin, imipramine and viloxazine decrease with use (Seppala et al, 1978; Seppala & Linnoila, 1983; Seppala et al, 1984). In contrast, administration of amitriptyline for five days did not impair performance or potentiate the effects of alcohol *in* some studies (Patman et al, 1969). In others it continued to impair critical

flicker fusion and co-ordination for 8 days (Seppala et al, 1984).

Doxepin and chlorimipramine both seem to improve performance of psychomotor tasks by psychiatrically depressed patients (Seppala et al, 1978).

Lithium administered for two weeks impairs choice reaction performance but tends to antagonise the effects of alcohol on attention and choice reaction tests (Linnoila et al, 1974a). It has no significant effects on memory or reaction times in manic depressive patients (Dasso et al, 1982).

3.6. Effects of Cardiovascular Drugs on Driving-Related Skills (Table A8).

Cardiovascular drugs are prescribed for treatment of hypertension, angina and cardiac arrhythmias. They fall into two groups, cardiac depressants which reduce the heart-beat rate by acting directly on the myocardium and/-adrenergic antagonists which reduce stimulation of the heart by the sympathetic nervous system.

Methyldopa and reserpine are cardiac depressants. Both reduce performance of driving-related skills (Clayton, Harvey & Betts, 1977).

Atenolol reduces performance of simple psychomotor tests such as reaction time, flicker fusion and digital copying (Salem & McDevitt, 1983). On the other hand propanolol interferes with visual function but not flicker fusion or actual driving performance (Bryan et al, 1974; Ogle b Turner, 1974; Clayton, Harvey & Betts, 1976).

Clonidine is an antihypertensive drug which is also used as a sedative during opiate withdrawal (Morse & Hughes, 1985). This substance reduces sensory function in healthy volunteers (Hossman et al, 1980).

3.7. Effects of Antihistamines in Driving-Related Skills (Table A9).

Antihistamines are constituents of most hayfever, cold and influenza preparations and some prescription drugs. About half of these antihistamine substances impair driving-related skills and, in general, these same preparations increase the effects of alcohol.

Triprolidine impairs dynamic visual acuity and reduces the threshold for flicker fusion while astemizole, terfenadine and mebhydrolin have little or no effect depending on the time when testing occurs (Franks et al, 1981; Nicholson et al, 1982; Betts et al, 1984).

Dexchlorpheniramine potentiates the effects of alcohol and increases recovery time (Franks et al, 1978).

3.8. Effects of Cannabis and Other Illicit or Permit Requiring Drugs on Driving-Related Skills (Table A10)

Research into the effects of cannabis on driving-related skills is complicated by the varying levels of the active cannabinoid components in the preparation of the drug which is experimentally administered to the test subjects (Bird et al, 1980).

Marijuana grown in different places and from different species of cannabis sativa can have different concentrations of Δ9-tetra-hydrocannabinol ranging from 0.2 to 6.8 per cent by weight. Users do not adjust their

rate of consumption according to the amount of drug present (Chesher, 1984).

The Committee of the Institute of Medicine (1982) of the American National Academy of Science stated:-
"We can say with confidence that marijuana produces acute effects on mental functions and behaviour. With a severity related to dose, marijuana impairs motor co-ordination and affects tracking ability and sensory and perceptual faculties important for safe driving.'"

The known effects of cannabis on crash risk and driving-related skills have been reviewed by Moskowitz (1985).

Cannabis impairs performance of driving-related tasks which measure or depend on motor and mental performance (Manno et al, 1971; Rafaelsen et al, 1973; Macavoy & Marks, 1975; Chesher et al, 1976; 1977; Belgrave, 1979; Bird et al, 1980; Chesher & Starmer, 1983; Chesher, 1985). No effect has been measured in driving performance tests (Sutton, 1983).

Performance decrements due to cannabis use appear independent of the attention span of the task. That is, divided attention and concentrated attention tasks were similarly impaired. However, sensory processes were not significantly affected (Moskowitz, 1973; Chesher, 1985). There is no significant relationship between the plasma concentration of the drug and the response it elicits in the user but the effects can be expected to extend beyond 3 hours after use (Chesher & Starmer, 1983).

The World Health Organisation (1981) says that cannabis alters the effects of other drugs such as alcohol, barbiturates, nicotine, amphetamines, cocaine, phenycyclidine and opiates. Similarly, other drugs can alter the effects of cannabis. However,literature to support all these statements is difficult to find.

In general, the effects of cannabis and alcohol are additive or synergistic but these can only be measured

at doses above 2.5mg tetrahydrocannabinol (Bird et al, 1980; Chesher & Starmer, 1983; Sutton, 1983).

At lower cannabinoid doses, there is now evidence to suggest that alcohol and cannabis are effectively antagonistic for their effects on driving-related skills and on measures of anxiety and alertness (Chesher, 1985).

Methadone appears to have little effect on driving-related skills in tolerant individuals although it has marked effects on those who do not use the drug routinely (Moskowitz & Sharma, 1969; Gordon, 1976) but phencyclidine reduces performance of both cognitive and sensory function (Catlin et al, 1979).

3.9. summary.

Information about the effects of particular substances on driving-related skills seems limited except in those gsoups of drugs normally used to affect the central nervous system. There is some information available about cardiovascular drugs.

In most cases, substances which can be *shown* to reduce performance of healthy volunteers have less effect on the population for whom they are normally prescribed. Substances such as amitriptyline and some antihistamines which also reduce performance of patients can often be replaced by alternative medication which has little or no demonstrable effect on driving-related skills.

Alcohol interacts with many drugs which impair driving related skills. This effect is generally additive or synergistic except in the case of amphetamines, zimeldine, indomethacin and low doses of cannabinoids.

Chapter Four.

Study Method.

This project was designed to measure the frequency of drug use by drivers, motor cycle riders and pedestrians (operators) involved in fatal road crashes in metropolitan Melbourne Coronial District (Fig. 1).

4.1. Study Population.

The Study Population includes:

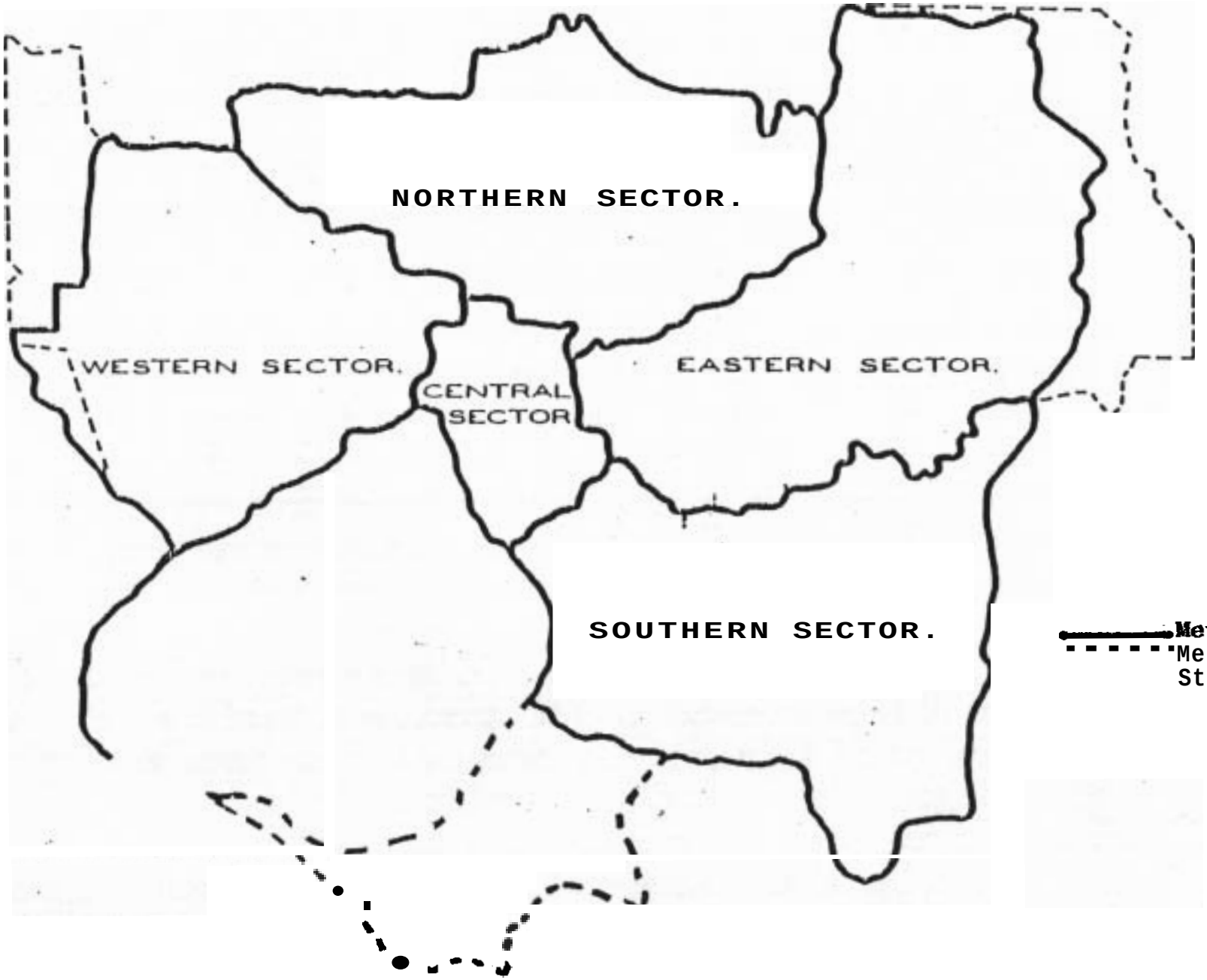
1. All drivers, motorcycle riders and pedestrians killed in the metropolitan Melbourne Coronial district between April 10th, 1980, and April 30th, 1982 (N=630) and

2. All uninjured or hospitalised drivers involved in accidents in the metropolitan Melbourne Coronial district between April 10th, 1980, and April 30th, 1982 in which a driver or pedestrian was killed (N=485).

The crashes were identified from the Fatals Diary maintained by the Statistics Section.; Accident Records, Police Traffic Centre, Melbourne.

OPERATIONAL BOUNDARIES METROPOLITAN MELBOURNE

Fig. 1.



The number of operators in each injury and crash category is shown in Fig. 2.

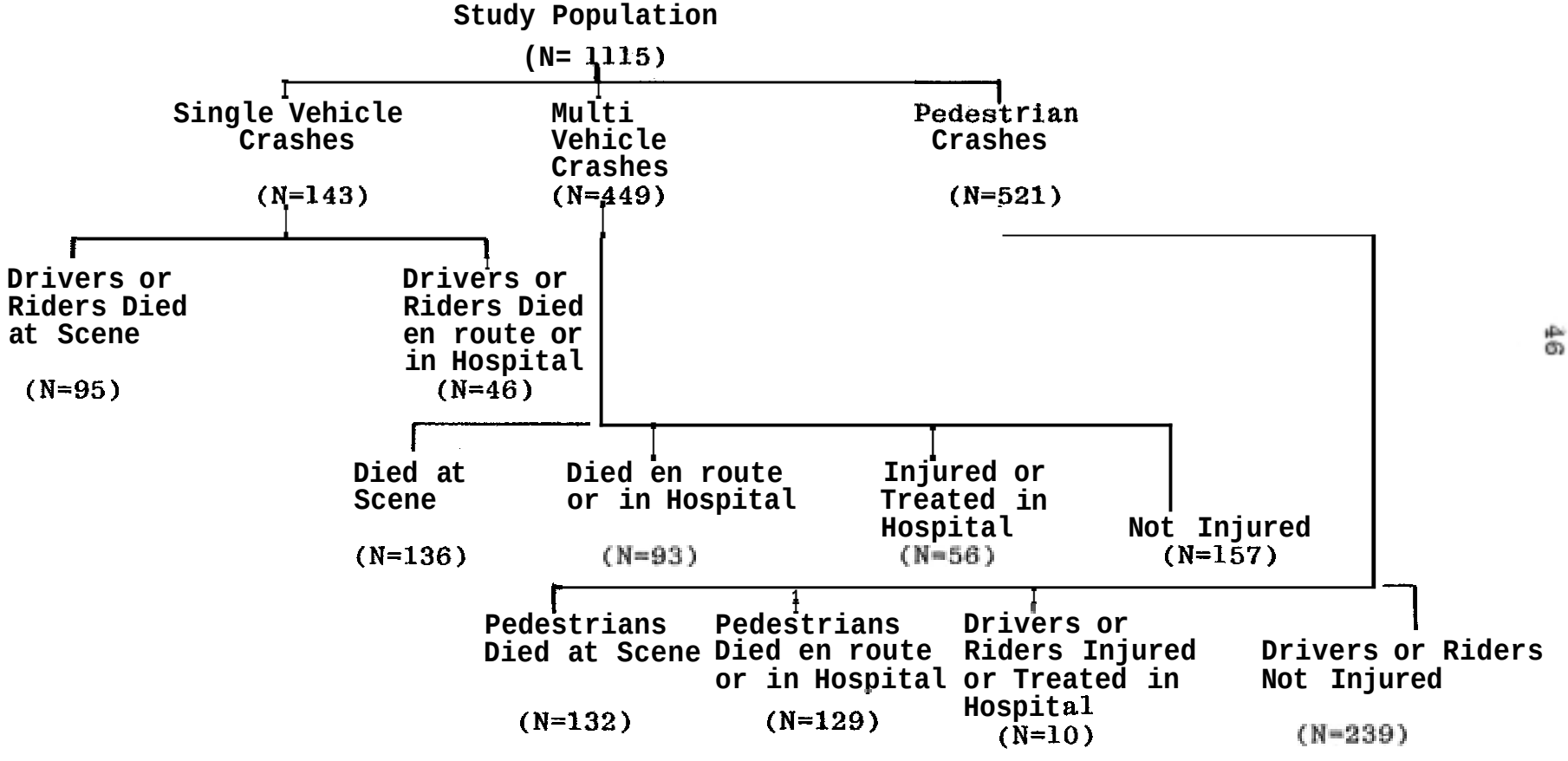
Individuals involved in those crashes for whom Accident Reports had not been returned to the Accident Records Section by 30th August, 1983 (N=17) were not included in the Study Population. All these excluded collisions occurred in 1981 and 1982.

4.2. Data Collection.

Information about the Study Population was gathered from two sources.

(a) The Melbourne Coroner arranged blood and/or urine samples to be taken from drivers and pedestrians by the pathologists during post-mortem examination. These samples of body fluid were analysed for drugs using Chemical Ionisation/Mass Spectrometry, Thin Layer Chromatography and other procedures described in Appendices C & D.
These procedures were capable of detecting each drug at the concentration listed in Table C2.

Crash and Injury Categories of Operators in Study Population. Fig. 2.



Any other drugs present, or drugs present at levels below those indicated will not have been detected.

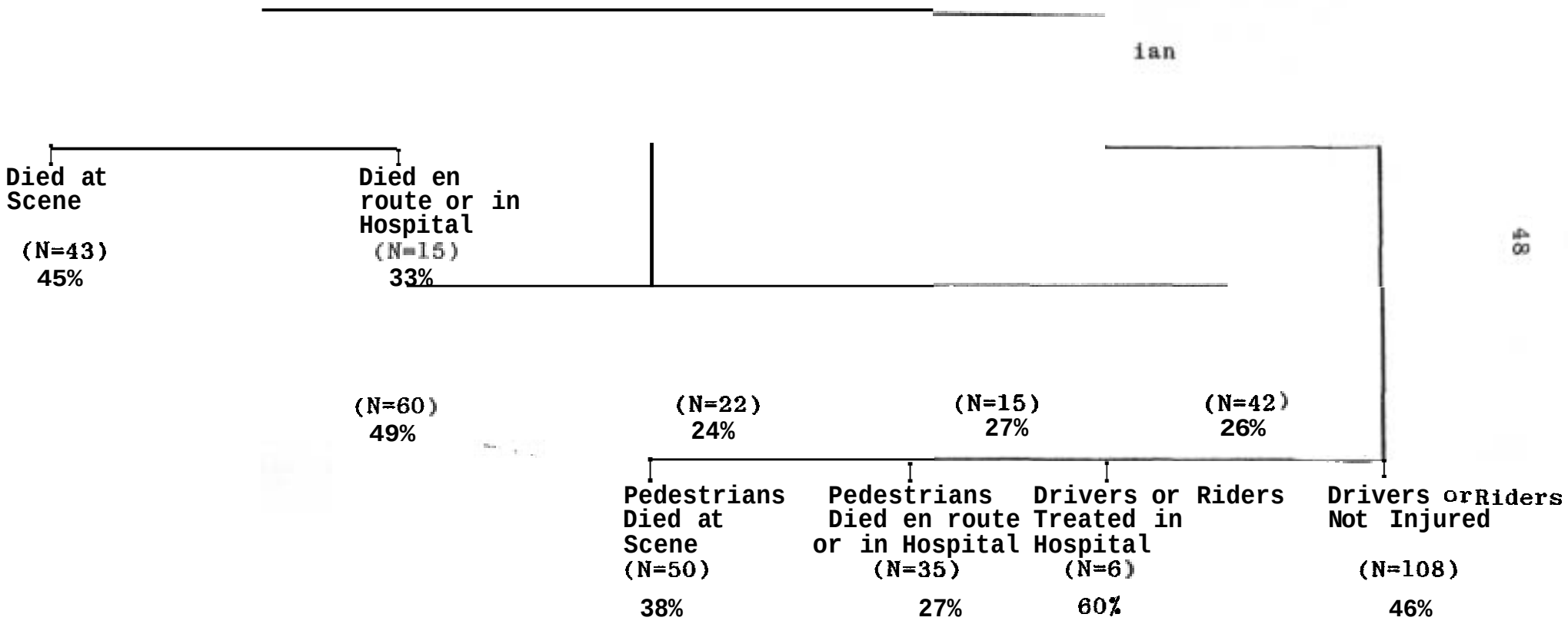
(b) Data about the individual, his alcohol and drug use, and details of the crash which led to his inclusion in the Study Population were extracted from the Fatal Accident Report compiled by the Police. A list of data items collected is contained in Appendix E.

Occupation was categorised according to the Australian Bureau of Statistics (1976) classification.

The results of body fluid analysis were included in this data set.

All data was coded in a format suitable for analysis using the Statistical Package for Social Sciences (Nie et al, 1978). No identifying information was included.

Selection of Operators for Whom Other Drug Information was Available.* Fig. 3.



* percentages indicate proportion of each subgroup *for* whom information was available.

4.3. Data Analysis.

The Fatal Accident Data File was analysed using the Statistical Package for Social Sciences (Nie et al, 1978).

Missing data has been excluded from all statistical analyses.

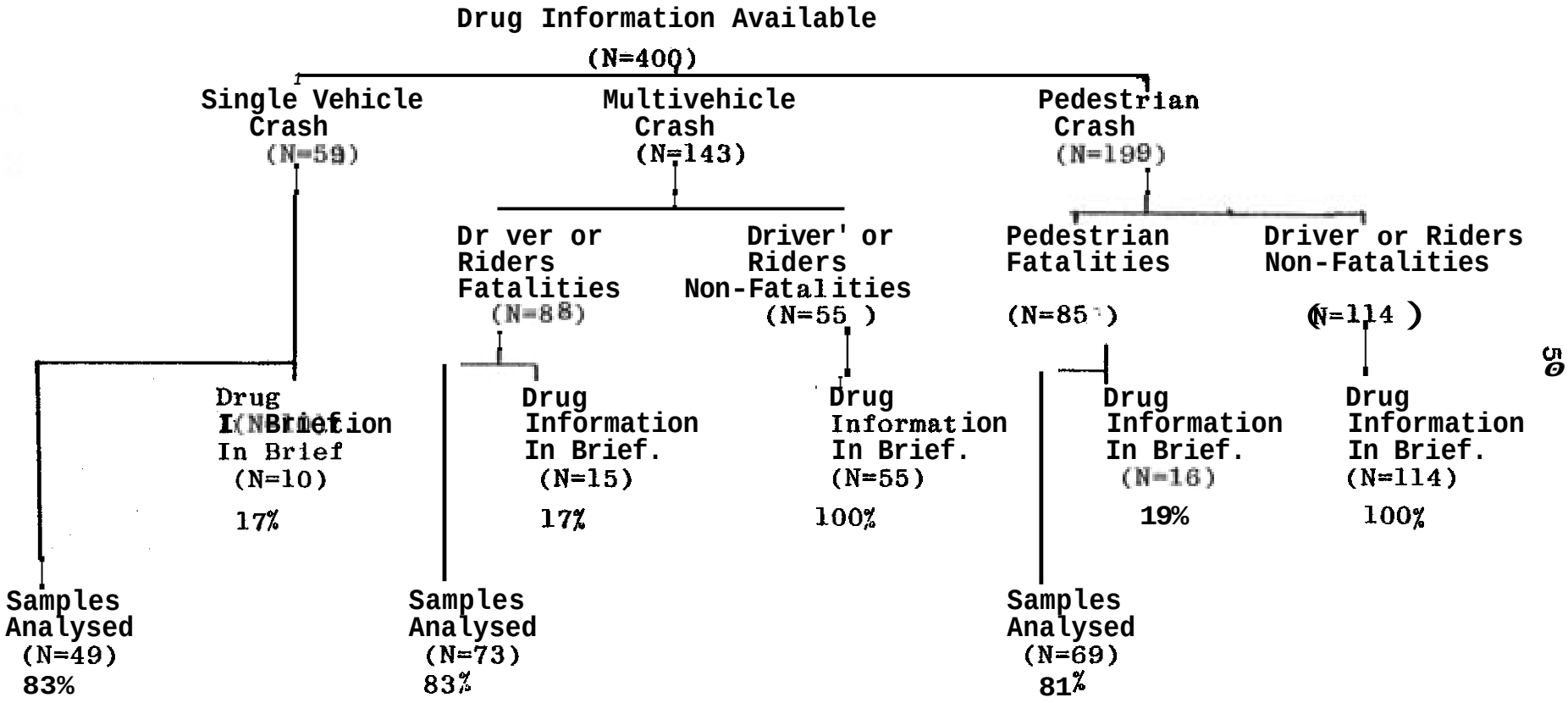
Differences between categorical variables were tested using the Chi Square Test. Differences have been categorised as:

significant at the 0.5% level,
very significant at the 0.1% level,
highly significant at the 0.01% level.

4.4. Selection of Operators for Drug Analyses.

Information about drugs other than alcohol was available for about 36% of all drivers, motorcycle riders and pedestrians in the Study Population (Fig. 3). This proportion was higher for operators killed in single vehicle and pedestrian crashes than for those involved in multivehicle crashes; it was also higher for those who died at the scene than for other injury categories. In pedestrian crashes there was more information available about the drivers who were treated in hospital or not injured than there was for pedestrian fatalities.

Source of Information About Other Drug Use.* Fig.4.



* percentages indicate proportion of drug information about subgroup derived from each source.

4.4.1. Sources of Drug Information:- Information about other drug use was available through analyses of body fluid samples taken by the pathologists or from information available in the Brief prepared by the police (Fig. 4).

Questions about drug use are administered as part of the breath test procedures and these were usually the source of information in the Brief.

The sources of drug information about drug use for different crash categories are shown in Fig. 4.

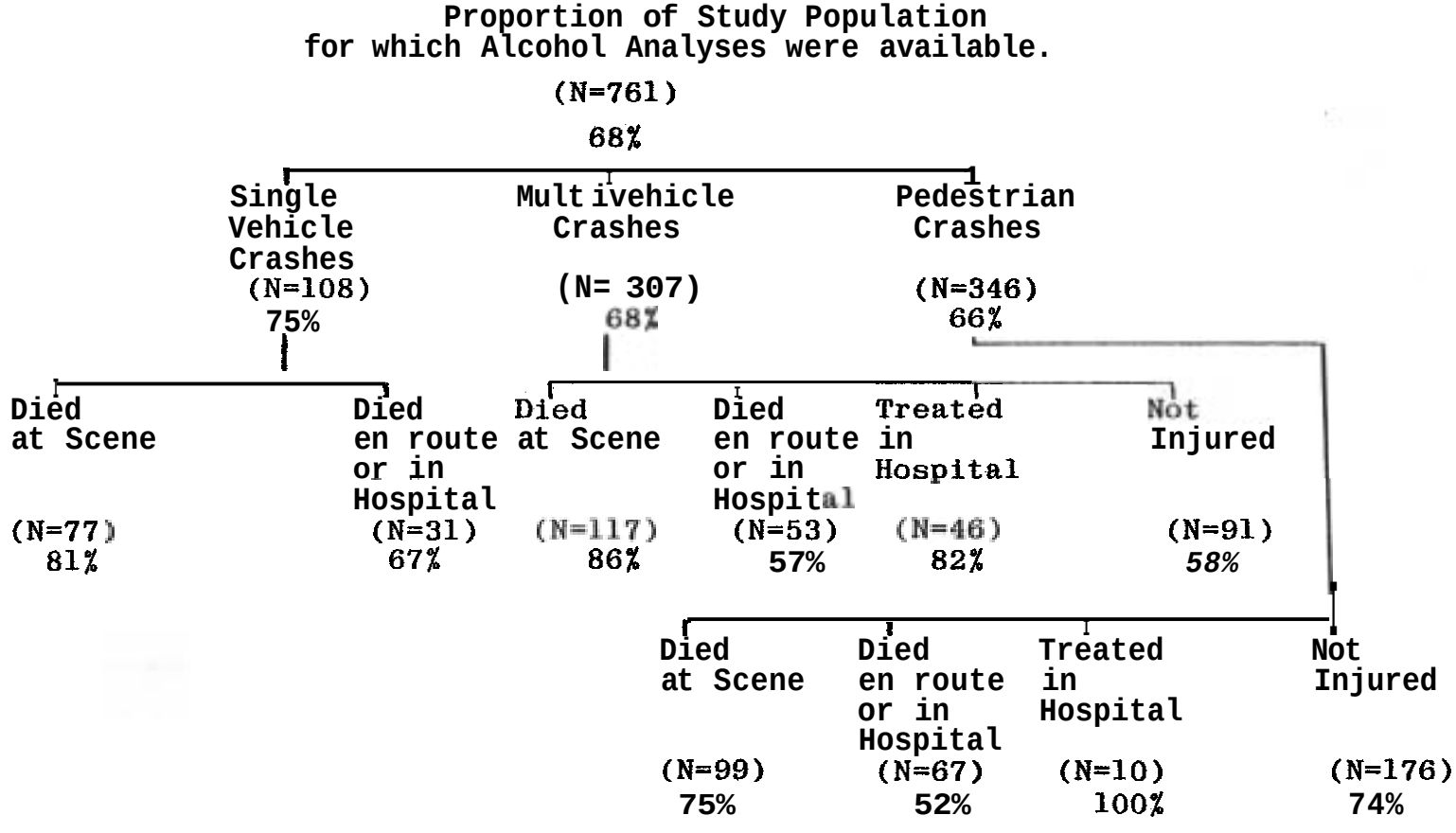
In 10 cases where compatible information about drug use was available from analyses and from the Brief, there was complete agreement in 9 cases and in 1 case the analysis had failed to detect one drug reported in the Brief.

Information about nicotine use in fatalities was collected only for crashes occurring after 20th May 1981.

4.5. Selection of Operators for Alcohol Analyses.

Alcohol information was available for 70% of all drivers and pedestrians in the

Selection *of* Operators *for* whom Alcohol Information was Available.* Fig. 5.



* percentages indicate proportion of each subgroup for whom alcohol information was available.

the Study Population. This proportion was higher for drivers and motorcycle riders killed in single vehicle crashes than it was *for* operators involved in multivehicle or pedestrian crashes (Fig. 5). Further, it was higher for operators killed outright or treated in hospital in the latter two crash categories than *for* those who died en route to or in hospital or were not injured at all.

4.5.1. Sources of Alcohol Information:- All information about alcohol was derived from the Police Accident File. However, it had originally derived from several sources.

All information available about alcohol use by fatalities who died at the scene and 75.3% of those who died en route to or in hospital derived from coronial analysis of post mortem blood. This means that the reported number of drinkers among those who died en route to or in hospital will underestimate the true frequency because of the alcohol metabolism which occurred between the crash and the time of death.

Information about operator casualties and 24.7% of those who died en route to or in hospital derived from Forensic Science Laboratory Analyses of blood samples taken in hospital.

About 96% of information about alcohol use by uninjured drivers derived from breath analysis. The other 4% came from reports made in statements to the police.

Chapter Five.

Characteristics of Crashes Involving Driver and Pedestrian Fatalities.

There were 1115 drivers and pedestrians involved in collisions in metropolitan Melbourne between April 10th 1980 and April 30th 1982 in which at least one driver or pedestrian was killed. In this chapter, information about operators who survive is included with those who have died unless otherwise indicated.

Those operators involved in

- single vehicle fatal crashes,
- multivehicle fatal crashes,
- pedestrian fatal crashes

differed from each other group in several of their personal characteristics, in the degree of injury they sustained, and the characteristics of the crash.

5.1. Personal Characteristics.

5.1.1. Sex:- Operators involved in single vehicle and multivehicle crashes were more likely to be men than those involved in pedestrian crashes (Table 1).

Table 1. Sex of Operators involved in Fatal Crashes.

Sex	Single Vehicle Crash (N= 143) %	Multi Vehicle Crash (N= 444) %	Pedestrian Crash (N= 511) %
Men	86.0	83.8	72.0
Women	14.0	16.2	28.0
	100.0	100.0	100.0

Chi Square Test
Highly Significant.

5.1.2. Age:- Operators involved in single vehicle and multivehicle crashes were more likely to be aged under 30 years than those involved in pedestrian crashes. On the other hand, over 30% of the operators in pedestrian crashes were aged 60 years or over (Table 2).

Table 2. Age of Operators involved in Fatal Crashes.

Age (Years)	Single Vehicle Crash (N= 141) %	Multi Vehicle Crash (N= 441) %	Pedestrian Crash (N= 508) %
Under 20	12.8	12.5	6.9
20 to 29	46.0.	32.4	26.0
30 to 39	14.9	24.0	16.9
40 to 49	8.5	10.2	10.4
50 to 59	5.7	10.7	8.9
60 to 69	5.7	6.6	10.4
70 to 79	6.4	3.6	20.1
Over 79	-	-	0.4
	100.0	100.0	100.0

Chi Square Test
Highly Significant.

There was no significant relationship between age and sex of operators except among pedestrians who died at the scene of the crash. In this group, 60% of women and only 36% of men were aged over 60 years.

5.1.3. Occupation:- Operators involved in pedestrian crashes were more likely to be not working while those involved in single vehicle crashes were more often in the farmers, horticultural workers and miners group. Transport and communication occupations were overrepresented among operators involved in multivehicle crashes (Table 3).

Table 3. Occupations of Operators involved in Fatal Crashes.

	Single Vehicle Crash	Multi Vehicle Crash	Pedestrian Crash
	(N= 102)	(N= 368)	(N= 458)
Occupation	%	%	%
Professional	-	7.1	7.9
Administrative, Managerial	2.0	3.3	2.4
Sales	6.9	7.6	8.2
Farmers, Miners	5.9	1.4	0.7
Transport & Communications	11.8	20.1	8.4
Tradesmen & Labourers	28.4	24.2	17.2
Service	7.8	7.3	4.6
Unemployed	3.9	5.7	4.9
Not working	33.3	23.3	45.7
	100.0	100.0	100.0

Chi Square Test
Highly Significant.

5.1.4. Place of Residence: Eleven percent of all operators involved in the study lived outside the metropolitan area.

Among those who lived in the survey area, place of residence was not significantly different from the general population of metropolitan Melbourne.

Operators involved in single vehicle crashes were most likely to come from the Northern part of the City while those involved in pedestrian crashes were least likely to live outside the metropolitan area (Table 4).

Table 4. Place of Residence of Operators involved in Fatal Crashes.

Place of Residence	Single Vehicle Crash (N=141) %	Multivehicle Crash (N=443) %	Pedestrian Crash (N=511) %
Central City	8.5	8.1	11.4
Western Sector	13.5	11.7	14.1
NorthernSector	12.1	17.6	17.0
Eastern Sector	24.8	21.0	26.5
Southern Sector	27.6	28.2	22.5
Other	13.5	13.4	8.5
	100.0	100.0	100.0
Chi Square Test Significant			

5.1.5. Vehicle:- Two thirds of the vehicles involved in driver and pedestrian fatalities were sedans and another 9% were stationwagons, 8% were vans or utilities, 9% were heavy vehicles and 11% were motor cycles.

There was a highly significant relationship between the sex of the driver and the vehicle driven. Women were more likely to drive sedans while nearly all the heavy truck and motor cycle riders were men.

Table 5. Vehicles Driven by Men & Women in Study Population.

Vehicle Type	Sex	
	Men (N=648)	Women- (N=127)
	%	%
Sedan	58.3	87-3
Station Wagon	9.0.	8.7
Van & Utility	9.1.	2.4
Heavy Truck	11.1	0.8
Motor Cycle	12.5	0.8
	100.0	100.0

Chi Square Test
Highly Significant.

Similarly, 83% of motorcycle riders were aged under 30 years, while sedans were more often driven by drivers aged over 60 pears.

5.1.6. Licence Type:- Nearly 70% of all drivers in the study papulation had full drivers' licences, 3.2% were learners, 11.6% first year probationers, 6.1% probationary drivers with more than one year's experience, 3.6% conditional, 0.8% disqualified and 4.7% unlicenced drivers.

Motor cycle riders were significantly more likely than drivers of other vehicles to be riding without a licence, learners or in their first six months of their probationary licence period (Table 6).

Table 6. Licence Type of Drivers of Different Vehicles.

Licence Type	Vehicle			
	Sedan or Stationwagon (N=537)	van & Utilities (N=58)	Motorcycle (N=77)	Heavy Vehicle (N=73)
	%	%	%	%
Learner	0.7	3.4	23.4	-
Probationary (less than 1 year)	12.1	8.6	18.2	2.8
Probationary (1 year or over)	8.0	6.9	6.5	5.5
Full Licence	70.9	*70.8*	35.0	84.8
Conditional	3.8	5.2	-	5.5
No Licence	3.8	3.4	15.6	1.4
Disqualified	0.7	1.7	1.3	-
	100.0	100.0	100.0	100.0

Chi Square Test
Highly Significant.

5.2. Characteristics of the Crash.

5.2.1. Place : Crashes involving pedestrian fatalities were twice as likely as single vehicle or multivehicle fatal crashes to occur in the Central City indicated in the map on p.44. However single vehicle crashes occurred more frequently *in* those areas designated metropolitan Melbourne which are more distant from the Central Business District (Table 7).

Table 7. Place of Fatal Crashes.

Place	Single Vehicle Crash (N=143) %	Multivehicle Crash (N=449) %	Pedestrian Crash (N=521) %
Central City	7.7	11.1	21.9
Western Sector	14.7	12.5	14.4
Northern Sector	14.7	14.9	15.9
Eastern Sector	29.3	25.0	23.4
Southern Sector	18.9	24.3	17.7
Other	14.7	12.2	6.7
	100.0	100.0	100.0

Chi Square Test
Highly Significant.

5.2.2. Time of the Incident:- Two thirds of the operators were involved in fatal crashes which took place between midday and midnight. The times were related to the type of crash (Table 8) with one third of single vehicle crashes occurring in the early morning, between midnight and 6 am, while a third of multivehicle and pedestrian crashes occurred during the afternoon.

Table 8. Time of Incident of Different Types of Fatal Crashes.

Time (24 hour clock)	Single Vehicle Crash (N=143) %	Multivehicle Crash (N=445) %	Pedestrian Crash (N=520) %
0001 to 0600 hours	32.8	8.5	6.5
0601 to 1200 hours	17.5	26.7	20.6
1201 to 1800 hours	18.2	36.0	34.4
1801 to 2400 hours	31.5	28.8	38.5
	100.0	100.0	100.0

Chi Square Test
Highly Significant.

5.3. Summary.

There are significant differences in characteristics of the operators and the incidents of single vehicle fatal crashes, multivehicle fatal crashes and pedestrian crashes.

Pedestrian crashes are more likely than other crashes to involve women, operators aged over 60 years and individuals not in the work force. They occur more frequently in the Central City.

Single vehicle crashes are more likely than other crashes to involve farmers, miners and horticultural workers and occur in the outer area of metropolitan Melbourne and between midnight and 6 a.m.

Multivehicle crashes are more likely than other crashes to involve people in transport and communications occupations.

Chapter Six.

Use of Drugs Other than Alcohol. Caffeine & Nicotine.

Information about drug use was available for 36% of the study population. Overall, 43.6% of all these operators were positive for drugs other than alcohol, caffeine and nicotine. There were significant differences in the frequency of the drug use by operators in different types of crashes.

Drivers and motor cycle riders killed in single vehicle crashes had a greater frequency of drug use than operators involved in multivehicle crashes. These were both greater than that for operators in pedestrian crashes (Table 9). These figures include:-

52% of drivers and motor cycle riders in single vehicle crashes who died at the scene,

77% of drivers and motor cycle riders in multivehicle crashes who died at the scene, and

61% of pedestrians who died at the scene.

Use of drugs was significantly related to injury category for occupants involved in each of the three crash types. Those who died were more likely to have taken drugs than those who survived in each case. However this could reflect the different data collection procedures used for fatalities and survivors.

Table 9. Drug Use by Operators involved in Fatal Crashes.

Use of Drugs Other than Alcohol, Caffeine & Nicotine.	Single Vehicle Crash (N=59)	Multivehicle Crash (N=146)	Pedestri Crash (N=19
	%	%	%
Yes	59.3	45.9	37.1
NO	40.7	54.1	62.9
	100.0	100.0	100.0

Chi Square Test
Highly Significant.

6.1.1. Sex :- The sex of the operator was significantly related to the likelihood they had used drugs. Women were over one and a half times more likely than men to have used drugs (Table 10).

Table 10. Use of Drugs by Male & Female Operators.

Use of Drugs Other than Alcohol, Caffeine & Nicotine.	Sex: Men (N=319) %	Sex: Women (N=80) %
Yes	38.2	66.2
NO	61.8	33.8
	100.0	100.0

Chi Square Test
Highly Significant.

6.1.2. Age :- The age of the operator was significantly related to the likelihood that drugs were involved. However, this relationship only held for operators involved in multivehicle crashes when each category was analysed separately. There was no relationship between age and drug use for drivers killed in single vehicle crashes (Table 11).

Table 11. Use of Drugs by Different Age Groups of Ouerators.

Use of Drugs other than Alcohol, Caffeine & Nicotine	Age: Under 25 Years (N=117) %	25 to 44 Years (N=156) %	45 to 64 Years (N=70) %	Over 64 Years (N=58) %
Pes	43.6	32.1	45.7	72.4
No	56.4	67.9	54.3	27.6
	100.0	100.0	100.0	100.0

Chi Square Test
Highly Significant.

6.1.3. Vehicle Type:- 60% of motor cycle riders in the study population had been using drugs compared with 38% of sedan and stationwagon drivers, 19% of van and utility drivers and 12% of drivers of heavy vehicles (Table 12).

Table 12. Drug Use by Drivers of Different Vehicles involved in Fatal Crashes.

Use of Other Drugs other than Alcohol, Caffeine & Nicotine .	Vehicle Type: Sedan or Stationwagon (N=218) %	Motor Cycle (N=45) %	Van or Utility (N=26) %	Heavy Truck (N=24) %
Yes	38.1	60.0	19.2	12.5
No	61.9	40.0	80.8	87.5
	100.0	100.0	100.0	100.0

Chi Square Test

6.1.4. Licence Type :- There was no significant relationship between use of drugs and licence type.

6.1.5. Alcohol:- Overall, 11.3% of the study population had combined alcohol with the use of other drugs. Use of drugs other than alcohol was significantly related to blood alcohol level. Those operators known to have a blood alcohol level over 0.05g/100mls were less than half as likely to have used other drugs than those with zero blood alcohol level (Table 13).

However, when only those operators killed at the scene of the crash are considered, no statistically significant relationship between blood alcohol reading and other drug use-could be demonstrated for drivers or motor cyclists involved in single vehicle or multivehicle crashes or pedestrian fatalities.

In all these groups, there remained a tendency for non drinkers to have a higher frequency of drug use than drinkers.

Table 13. Use of Drugs by Drinking and Non-Drinking Operators.

Use of Drugs Other than Alcohol, Caffeine & Nicotine	Blood Alcohol Level (g/100 mls)		
	Over 0.05 (N=95)	0.01 to 0.05 (N=18)	Zero (N=134)
	%	%	%
Yes	30.5	55.5	78.3
No	69.5	44.5	21.7
	100.0	100.0	100.0

Chi Square Test
Highly Significant

6.1.6. Time of Incident:- The time of the incident was significantly related to the incidence of drug use in single vehicle crashes only. In these crashes, drugs were found in 87% of those involved in daylight crashes (0601 to 1800 hours) but only 40% of those which occurred in the early morning and 56% of those in the evening.

6.2. Categories of Drugs Used.

Drugs can be classified into categories according to their usual pharmacologically active sites in the body. The categories of drugs used by operators in the survey are listed in Table 14.

Table 14. Types of Drugs Used by Operators involved in Fatal Crashes.

Drug Type	Operators using Drugs other than Alcohol, Caffeine & Nicotine. (N=178) %
Amphetamines	2.2
Cannabinoids ***	20.5
Alimentary Tract & Metabolism	5.8
Blood and Blood Forming Organs ****	0.6
Cardiovascular System	11.0
Systemic Hormonal Preparations ****	0.6
General Antiinfectives ****	1.7
Musculo Skeletal System ****	1.7
* Central Nervous System **	97.2
Respiratory System	12.4
Unspecified	7.5

* Includes local anaesthetics, analgesics, antiepileptics, antiparkinsonism, psycholeptics and psychoanaleptics.

** 10, or 13.7% were drugs usually given in hospital i.e. pethidine or morphine.

*** Not assayed in all individuals. This figure is proportion of drug positive fatalities tested for cannabinoids.

**** None of these drugs were identifiable in body fluid i.e. all rely on police reports.

This list excludes alcohol, caffeine and nicotine. Different types of drugs were used by operators with different characteristics.

6.2.1. Sex :- There was no significant difference in the type of substance used by men and women who had taken drugs. There was also no significant relationship between sex and the types of drugs used in any of the three categories analysed separately.

6.2.2. Age:- There was a highly significant difference in the types of drugs used by different age groups (Table 15). This relationship held for drug positive operators involved in multivehicle and pedestrian crashes but not for single vehicle crashes. It also held for male but not for female drug users when these groups were analysed separately.

Drug users aged under 45 years were more likely to have used cannabis or amphetamine than older users. In contrast, drugs for treatment of the cardiovascular system and sedatives, hypnotics and tranquilisers were more prevalent among those aged 44 years to 64 years (Table 15).

Table 15. Proportion of Drug Positive Operators in Different Age Groups Who Use Particular Types of Drugs.

Drug Type	Age (Years) Under 25 (N= 50) %	25 to 44 (N= 50) %	45 to 64 (N= 32) %	Over 64 (N= 42) %
Amphetamines	6.0	1.9	-	-
Cannabinoids***	34.4	42.8	8.1	-
Alimentary Tract & Metabolism	4.0	8.0	9.4	4.8
Blood & Blood Forming Organs ****	-	-	-	2.4
Cardiovascular System	-	1.9	21.9	23.8
Systemic Hormonal Preparations ****	-	-	3.1	-
General Anti-Infectives ****	-	6.0	-	-
Musculo Skeletal****	-	-	6.3	2.4
Anaesthetics* (Local)	-	-	3.1	2.4
Analgesics**	35.9	50.0	31.1	47.6
Anti-epileptics	4.0	8.1	6.3	2.4
Anti Parkinsonism	-	-	-	2.4
Psycholeptics: Sedatives,Hypnotics & Tranqurlisers	20.0	20.0	40.7	28.5
Psychoanaleptics : Antidepressants	6.0	14.0	3.1	4.8
Respiratory	15.9	9.9	15.6	7.2

Table 15 (Continued).

Chi Square Test
Highly Significant.

* Reported to be part of dental treatment.

** 10, or 13.7% of these analgesics were drugs usually given in hospital i.e. pethidine or morphine.

*** Not assayed in all individuals. This figure is proportion of drug positive fatalities tested for cannabinoids.

**** None of these drugs were identifiable in body fluid i.e. all rely on police reports.

6.2.3. Crash Type:- There was a significant difference between the types of drugs used by operators involved in different types of crashes. Drug using drivers and motor cyclists involved in multivehicle fatal collisions were more than twice as likely to have used cannabis than drug users in other crashes. On the other hand, operators involved in pedestrian crashes were more likely to have used cardiovascular drugs, and anti-epileptic drugs were overinvolved in those killed in single vehicle crashes (Table 16).

6.2.4. Alcohol:- There was no significant relationship between alcohol use and the categories of drugs used by drug users.

6.2.5. Polydrug Use:- Over one third of drug users are known to have used more than one substance other than alcohol, nicotine or caffeine. This figure (35.6%) holds for fatalities when they are analysed alone.

There was a significant difference in the number of substances used by different age groups of drug positive operators. Excluding alcohol, operators aged under 25 had taken on

average, 1.12 preparations, 25 to 44 year olds had taken 1.44 preparations each, 45 to 64 year olds had taken 1.47 preparations each, those over 64 years old had used 1.29 preparations each.

6.3 Particular Drugs Identified in Driver and Pedestrian Fatalities.

The particular drugs identified in blood and/or urine from driver and pedestrian fatalities are listed in Table 17.

The most frequently detected substances were, in order of frequency, cannabinoids, aspirin, paracetamol, ephedrine, benzodiazepines, (i.e. diazepam, oxazepam and desmethyldiazepam), seco-barbital, amitriptyline and nortriptyline. Many of these drugs were used in combination with each other though only 11.3% of fatalities combined alcohol use with use of other drugs.

Table 16. Proportion of Drug Positive Operators in Different Crash Types Who Use Particular Types of Drugs.

Drug Type	Single Vehicle Crash (N=35) %	Multivehicle Crash (N=67) %	Pedestrian Crash (N=73) %
Amphetamines	5.7	6.0	1.4
Cannabinoids***	24.1	48.5	19.2
Alimentary Tract & Metabolism	-	6.0	3.5
Blood & Blood Forming Organs****	-	-	1.4
Cardiovascular System	8.6	3.0	19.2
Systemic Hormonal Preparations ****	-	1.5	-
General Anti Infectives ****	-	3.0	1.4
Musculo Skeletal****	-	-	4.1
Anaesthetics* (Local)	-	-	2.7
Analgesics **	34.3	56.7	31.5
Anti-epileptics	8.6	4.5	4.1
Anti Parkinsonism	-	-	1.4
Psycholeptics	40.0	44.8	43.8
Psychoanaleptics	8.5	4.5	8.2
Stimulants	2.8	-	1.4
Respiratory	14.2	11.9	11.0
Unspecified	2.8	10.4	6.8

Table 16 (Continued).

* Reported to be part of dental treatment.

** 10, or 13.7% of these analgesics were drugs usually given in hospital i.e. pethidine or morphine.

*** Not assayed in all individuals. This figure is the proportion of drug positive fatalities tested for cannabinoids.

**** None of these drugs were identifiable in body fluid i.e. all rely on police reports.

Table 17. Particular Drugs Identified in Driver & Pedestrian Fatalities.

	Drug	Number of individuals identified with the drug
	AMPHETAMINE/METHAMPHETAMINE	5
	NOVACAINE	1
	THIOPENTONE ***	1
	PETHIDINE ***	7
	MORPHINE ***	2
	CODEINE	3
	METHADONE	1
	ASPIRIN **	33
	PARACETAMOL	19
*	PHENYTOIN	5
*	CARBAMAZAPINE	1
*	OXAZEPAM **	9
*	DIAZEPAM/DESMETHYLDIAZEPAM **	10
	METHAQUALONE	1
*	CHLORDIAZEPOXIDE	5
	CHLORPROMAZINE	4
*	NITRAZEPAM	4
*	GLUTETHIMIDE	2
*	BUTABARBITONE	4
*	SECOBARBITAL	8
*	AMYLOBARBITAL	3

Table 17 (Continued).

	Drug	Number of individuals identified with the drug
*	QUINALBARBITAL	3
*	PHENOBARBITAL	3
*	AMITRIPTYLINE/NORTRIPTYLINE	6
*	WXEPIN	3
*	IMIPRAMINE	3
*	THIORIDAZINE	1
	EPHEDRINE	15
	DIPHENHYDRAMINE	1
*	CANNABINOIDS **	41
	PHENCYCLIDINE	1
	PHENYLPROPANOLAMINE	1
	DEXTROMORAMIDE	1
	PROPOXYPHENE	1

* drugs known to impair driving-related skills in healthy individuals.

** drugs which were subject to separate analytical procedures and all individuals were not necessarily included. See Appendix D.

*** used in medical treatment in all but one morphine case.

6.3.1. Cannabinoids

Cannabinoids were detected in 20.5% of driver and pedestrian fatalities for whom blood andlor urine was assayed. Alcohol was also present in 28.6% of these operators. Further, although 59.4% of cannabis users and 44.8% of non cannabis users were also nicotine smokers, this difference was not statistically significant. Similarly, 59.5% of cannabis users and 54.1% of non users had used drugs other than alcohol, caffeine or nicotine. This difference was not statistically significant.

Drugs used in combination with cannabis included diazepam/oxazepam or their metabolite desmethyldiazepam (N=6), barbiturates (N=5, including the secobarbital1 amylobarbital combination in 2 cases) and the antidepressants amitriptyline, nortriptyline or doxepin (N=3).

6.3.2. Aspirin

Aspirin was used by 10.3% of fatalities in whom it was assayed. This figure included 8.8% of male and 16.2% of female fatalities.

In 5 cases (13.9% of total) the aspirin users had also been drinking. This

was significantly less than alcohol use among non aspirin users. All of the drinkers in this group were men and 4 were aged under 25 years.

Two thirds (65.9%) of aspirin users had combined use of this drug with others, including cannabis (N=7), diazepam, oxazepam or desmethyldiazepam (N=6), paracetamol (N=8) and ephedrine (N=5).

6.4. Summary.

Drugs other than alcohol, caffeine and nicotine had been used by 43.6% of operators in the study population. This use was related to crash type, sex, age, vehicle type, alcohol use and time of incident.

Among fatalities, cannabinoids and aspirin were the most frequently detected substances. About one third of operators involved in the survey who had used drugs had used more than one substance. The types of drugs used differ for different categories of crash and different age groups but were not

significantly related to their sex or alcohol consumption.

Aspirin users were less likely to have been drinking than non aspirin users, and two thirds had used other drugs, particularly paracetamol and cannabis.

Chapter Seven.

Use of Alcohol. Caffeine & Nicotine.

Alcohol, nicotine and caffeine have been defined as social drugs for the purposes of this report.

All of these substances are known to affect driving-related skills. Details of research in this axea axe contained in Appendix F.

7.1. Alcohol.

Alcohol is known to reduce reaction time, performance of divided attention tasks, choice and reaction tests. At low blood alcohol levels, from 0.015g/100 mls, divided attention tasks appear more sensitive than other measures of psychomotor skill (Moskowitz et al, 1984; Chesher,1985) (Table F1).

The drug is unlikely to impair decision making at blood alcohol levels below 0.075g/100 mls, but if output is maintained accuracy deteriorates (Evans et al, 1974).

Alcohol consumption also increases anxiety levels in test subjects (Linnoila et al, 1980) and, in women, the degree of anxiety induced is higher in the luteal phase than the follicular phase of the menstrual cycle (Logue et al, 1981).

This anxiety is thought to contribute to inconsistencies in reaction to combined alcohol and cannabis observed at low cannabis levels (Chesher, 1985).

Further, alcohol is known to significantly increase drivers' likelihood of crash involvement at blood levels over 0.05g/100mls (Borkenstein et al, 1964; McLean & Holubowycz, 1984). This degree of increased risk is related to the frequency with which drivers drink (Allsop, 1966).

7.1.1. Use of Alcohol in the Study Population:-

Overall, 32.4% of operators involved in fatal crashes had been drinking and operators involved in single vehicle crashes were more than twice as likely as those involved in multivehicle or pedestrian crashes to have a blood alcohol level over 0.05g/100 mls.

These figures for alcohol consumption include:-

- 62% of drivers in single vehicle crashes who died at the scene;
- 38% of drivers in multivehicle crashes who died at the scene; and
- 46% *of* pedestrians who died at the scene.

Alcohol was not significantly related to the operators' injury categories except in the pedestrian crashes. Pedestrians who died at the scene were nearly twice as likely as pedestrians who died in hospital and drivers involved in the same crashes to have been drinking (Table 18).

Table 18. Alcohol Use by Operators involved in Fatal Crashes.

Blood Alcohol Level	Single Vehicle Crash (N=111) %	Multivehicle Crash (N=313) %	Pedestrian Crash (N=354) %
(g/100 mls)			
Over 0.05	53.2	23.0	22.9
0.01 to 0.05	5.4	4.8	5.4
Nil	41.4	72.2	71.7
	100.0	100.0	100.0

Chi Square Test
Highly Significant.

Sex:- Men in the study population were more than one and a half times more likely than women to have been drinking (Table 19). However, when each operator category was analysed separately this relationship did not hold for:

- Drivers *in* single vehicle or multivehicle crashes who died at the scene,
- Drivers in multivehicle crashes who survived,
- Pedestrians who died en route to or in hospital, or
- Drivers involved in pedestrian crashes.

Table 19. Use of Alcohol by Male and Female Operators.

Blood Alcohol Concentration (g/100 mls)	Sex: Men (N=620) %	Sex: Women (N=155) %
Over 0.05	30.0	16.8
0.01 to 0.05	5.3	4.5
Zero	64.7	78.7
	100.0	100.0

Chi Square Test
Highly Significant.

Age: The age of the operator in the study population was significantly related to the likelihood he had been drinking. However, this relationship only held for pedestrians who died at the scene and the drivers who were uninjured in pedestrian crashes when operators from different categories of crash with different injury severity were analysed separately.

Table 20. Use of Alcohol by Different Age Groupsof Operators.

Blood Alcohol Level (g/100 mls)	Age (Years) Under 25 (N=239)	25 to 44 (N=307)	45 to 64 (N=133)	Over 64 (N= 96
Over 0.05	28.0	30.9	27.1	14.6
0.01 to 0.05	7.1	3.9	3.0	6.3
Zero	64.9	65.2	69.9	79.1.
	100.0	100.0	100.0	100.0

Chi Square Test
Significant .

Vehicle Type :- Drivers in the study population who drove heavy trucks were much less likely to have been drinking than those who drove other vehicles (Table 21). This could reflect the police reported nature of this data as few heavy vehicle operators were killed or injured.

Table 21. Alcohol Use by Drivers of Different Vehicles involved in Fatal Crashes.

Blood Alcohol Level	Vehicle Type: Sedan or Station Wagon (N= 424)	Motor Cycle (N=71)	Van or Utility (N= 50)	Heavy Truck (N= 44)
(g/100 mls)	%	%	%	%
Over 0.05	26. 7	32.4	30.0	2.3
0.01 to 0.05	5.9	7.0	2.0	6.8
Zero	67.4	60.6	68.0	90.9
	100.0	100.0	100.0	100.0

Chi Square Test
Significant

Licence Type:- Drivers' licence type was significantly related to the likelihood he had been drinking. About three times more of those drivers with blood alcohol levels over 0.05g/100 mls than those who had not been drinking were unlicensed or disqualified from driving (Table 22).

This relationship was also significant for drivers involved in multivehicle or pedestrian crashes but did not hold for single vehicle operators.

Table 22. Use of Alcohol by Drivers with Different Types of Licence.

Licence Type	Blood Alcohol Level (g/100 mls) Over 0.05 (N= 183) %	0.01 to 0.05 (N= 31) %	Zero (N= 395) %
Learner	4.4	3.2	2.5
Probationary (less than 1 year)	10.4	6.5	12.9
Probationary (1 year or over)	8.7	9.7	6.3
Full Licence	64.0	61.2	70.5
Conditional	2.2	6.5	3.8
No Licence	1.6	3.2	0.5
Disqualified	8.7	9.7	3.5
	100.0	100.0	100.0

Chi Square Test.
Significant.

7.2. Caffeine.

The effects of caffeine on visual alertness depend on the subject's tolerance to the drug and the dose administered (Childs, 1978). Heavy users (more than 3 cups of coffee per day) improved performance on administration of 400mg caffeine (equivalent to 3 No Doze tablets) whereas those who rarely drink coffee showed reduced performance at that level (Childs, 1978).

Further, the effect of caffeine is related to the subject's practice in the tasks presented. Perceptual restructuring exercise performance is impaired by caffeine when the task is novel but it is improved when the task has been previously undertaken (Broverman & Casagrande, 1982) (Table F2).

Caffeine has been found to generally reduce the effects of alcohol on some driving-related skills depending on the relative doses of the two drugs (Franks et al, 1975, Moskowitz & Burns, 1981).

7.2.1. Use of Caffeine in Study Population:-

Caffeine was present in 64.4% of fatalities analysed. Drugs other than alcohol

and nicotine were also detected in 65.8% of individuals who had used caffeine and 50.0% of those in whom caffeine was not detected. This difference was statistically very significant.

7.3. Nicotine.

Cigarette smoking appears to improve psychomotor performance including simulated driving tasks (Heimstra et al, 1967; Williams, 1980; Wesnes & Warburton, 1984) (Table F3).

This improvement is related to the nicotine level of the smoke inhaled and seems to be the overt example of the central cholinergic response of the cortex (Wesnes & Warburton, 1983). It can be minimised using nicotine tablets (Wesnes & Revell, 1984). Performance is related to the cigarette smoking procedure, being less effective if subjects smoke according to their normal habits (Morgen & Pickens, 1982).

7.3.1. Use of Nicotine in the Study Population:-

Nicotine was identified in the blood or urine of 44.2% of driver, motor cycle rider and pedestrian fatalities. Although 42.9% of smokers and 32.6% of non-smokers had also used alcohol (Table 23), this difference was not statistically significant.

Table 23. Use of Alcohol by Smokers and Non-Smokers.

Alcohol Use	Smokers (N=70) %	Non-smokers (N=86) %
Yes	42.9	32.6
No	57.1	67.4
	100.0	100.0

Chi Square Test
Not Significant .

With reference to caffeine, 72.4% of smokers and 53.7% of non-smokers had also used caffeine (Table 24). This difference was highly significant.

Use of drugs other than alcohol and caffeine was not significantly related to use of nicotine.

Table 24. Use of Caffeine by Smokers & Non-Smokers.

Caffeine Use	Smokers (N=127) %	Non-smoker (N=108) %
Yes	72.4	53.7
NO	27.6	46.3
	100.0	100.0

Chi Square Test
Highly Significant.

7.4. Summary.

One third of operators involved in fatal crashes had been drinking alcohol, two thirds had used caffeine and 44% had smoked nicotine.

Caffeine use was significantly related to use *of* other drugs including nicotine.

Chapter Eight.

Discussion & Recommendations.

At least 44% of operators involved in fatal crashes have used over-the-counter, prescription or illicit drugs. Included in this figure are:-

- 52% of drivers and motor cycle riders in single vehicle crashes who died at the scene;
- 77% of drivers and motor cycle riders in multivehicle crashes who died at the scene;
- 61% of pedestrians who died at the scene.

These proportions exceed those reported by other studies of drugs in fatal crash populations.

A recent similarly comprehensive Californian survey of young driver fatalities showed 41% had used drugs other than alcohol, caffeine and nicotine (Williams et al, 1985) and a Canadian survey found 26% of their driver fatalities and 29% of pedestrian fatalities had used drugs (Cimbura et al, 1980).

In all cases, that is in the current survey, the Californian survey and the Canadian survey, cannabis was the most frequently reported drug other than alcohol, caffeine and nicotine. In California, cocaine was the next most frequently detected drug, while in Canada, aspirin was second. These substances were excluded from the Vine & Watson (1982) Sydney survey of driver and pedestrian fatalities which found only 10% had used drugs. As well 6% had used aspirin which was analysed and interpreted separately.

Seventeen of the drugs detected in driver and pedestrian fatalities in the current study have been shown to impair driving-related skills in healthy individuals. That is 53% of the drugs detected in the fatality group may have influenced the crash in which they were involved. Use of these substances involved 3% of the fatalities whosa blood or urine was analysed.

Further, 11% of operators involved in fatal crashes combined alcohol consumption with use of over-the-counter, prescription or

illicit drugs.

Figures of 14%, 4% and 40% respectively of fatalities surveyed in Canada, Sydney and California have combined alcohol use with use of drugs other than caffeine or nicotine (Cimbura et al, 1980; Vine & Watson? 1982; Williams et al, 1985).

These high figures mean that any influence which drugs may have on crash risk could be particularly important in designing new road safety initiatives or extending the influence of the drink-driving countermeasures which are already in position. However, in the absence of reliable information about use of drugs other than alcohol by road users not involved in road crashes, it is impassible to determine the real role of drugs in predisposition to crash risk.

8.1. Poly-Drug Use.

Drug users frequently use more than one substance at a time. For example,

nicotine smokers are more likely than non-smokers to use alcohol and cannabis (Australian Bureau of Statistics 1978; Roy Morgan, 1984; Bailey, 1984); cannabis users are more likely than non-cannabis users to use nicotine and, alcohol (Roy Morgan, 1984; Williams et al, 1985).

In the current study drug users had used, on average, 1.4 drugs including alcohol compared with between 1.3 and 1.6 in other similar studies (Cimbura et al, 1980; Williams et al, 1985).

This means that, even with the supporting evidence of controlled studies and pharmacological effects, it will be difficult to determine which particular substances significantly increase crash risk. It is therefore premature to recommend new legislative road safety measures directed specifically at road users who use drugs.

General countermeasures against crashes involving drugs need to be community based and directed through health and education programmes and existing drug law enforcement.

Recomendation 1.

It is recommended that:-
The Federal Office of Road Safety associate itself with existing organisations involved in designing drug control measures, such as the Federal Department of Health, the various State Police Forces and Education Departments, and the Australian Institute of Criminology, in an effort to co-ordinate measures to reduce drug consumption in the community.

8.2. Use of Medication in the Community.

The frequency of drug use in the study population is high, to the extent that fatalities killed in multivehicle and pedestrian crashes had used other drugs more often than they had used alcohol. Most of these substances are legally available.

Over 32% of the substances detected in driver and pedestrian fatalities were over-the-counter medicines sold through pharmacies and food outlets. A further 46% were prescription drugs, which are normally only available with the authority of a medical officer.

Use of medication, i.e.drugs other than illicit drugs, was more frequent among female operators and those aged over 45 years. In particular, prescription drug use was highest among fatalities aged over 64 years. These characteristics concur with those of frequent users of medication in the general population. *For* example, it is also known that community use of medication is high in Australia:

> Non prescribed medication is taken by 23% of men and 29% of women aged over 14 years in any two day period (Australian Bureau of Statistics, 1979). Among both men and women this use is greatest in the 25 to 44 year age bracket. *Common* pain relievers and vitamins or tonics are the most frequently used drug types.

> Prescribed medication is taken by 27% of men and 50% of women in any two day period (Australian Bureau of Statistics, 1979). Over 60% of men and 70% of women aged over 64 years have used these drugs in

> that time period and the most frequently used prescription drugs are contraceptive preparations and medicines for heart conditions, blood pressure or fluid retention.

Further, surveys which are known to underreport the frequency of drug use in the driving population suggest that between 10% and 13% of drivers in Australia and elsewhere have used medication (Bean, 1974; Honkannen et al, 1980; Clayton et al, 1980; Hendtlass,1983).

Among weekend, nighttime drivers in Melbourne, drivers aged over 49 years were more likely to say that they had taken medication although the frequency of drug use was not related to gender. Women aged over 39 years were significantly underrepresented in this study (Hendtlass, 1983).

Drinkers in the community and in the driving population are consistently more likely than non-drinkers to use medication (Carroll et al, 1977; Hendtlass, 1983). This means that the characteristics of road users who are most likely to use medication are similar to those in the .general population who use medicines more frequently.

Further, it is known that substances which impair driving-related skills in the healthy do not always have the same effect on those for whom they are normally prescribed. There are now effective alternatives which usually do not interact with alcohol available for most exceptions to this generalisation.

Therefore, specific counter measures directed against those road crashes in which medication could have played a role should be directed through the suppliers of these drugs.

Recommendation 2.

. It is recommended that:

An education and awareness programme for officers and pharmacists be established to inform them of the effects which some medicines may have on driving-related skills and the alternative products which are available.

Implementation of Recommendation 2.

Drug use, including marijuana and amphetamines, is frequent among young and trainee

physicians (McAuliffe et al, 1984) and historically the medical profession has a high rate of self medication and addiction (Valliant et al, 1970). Further, communication with the medical profession is difficult (Parish,1971). This means that potential prescribers can be expected to be relatively tolerant of drug use and abuse and while prescription drugs are subsidised, they remain an easy, inexpensive form of therapy.

Great care will therefore be needed to design and implement an effective programme which will change this behaviour as well as their knowledge. It is suggested that the programme would best be administered through the Australian Medical Society on Alcohol and Drug Related Problems, and the Pharmaceutical Society of Australia.

8.3. Epilepsy & Diabetes.

Some consideration needs to be given to the special case of drivers known to be epileptics or diabetics. Both these conditions are controlled by medication and its absence may precipitate seizures or lapse of consciousness.

Antiepileptic preparations, particularly phenytoin, were detected in 9 driver and pedestrian fatalities. These drugs can impair driving-related skills even in epileptics, and Seppala and his colleagues (1979) considered epileptics were seldom capable of driving. In New Zealand 1 in 300 driver casualties was reported to have had an epileptic seizure (Bailey, 1984). Of course, some alcoholics are also maintained on anti-epileptic drugs to control their alcoholic seizures.

Further, although our analytical procedures did not detect hormones and drugs such as insulin which are used to control diabetes, these drugs were implicated through statements to police in five cases. They have also been shown to be associated with increased crash risk among drinking drivers (Macpherson et al, 1982).

Recommendation 3.

It is recommended that:

Further research be undertaken into crash risk of epileptic and diabetic drivers, the effect of available measures to control these conditions on their risk and on driving-related skills, and the most constructive ways of ensuring that these patients do not put other road users unnecessarily at risk.

8.4. Cannabis.

Cannabinoids were the most frequently detected *drug* among driver and pedestrian fatalities, excluding alcohol, caffeine and nicotine. They have been detected previously in:-

- 9% in driver fatalities in the United Kingdom (Teale et al, 1977).
- 12% of driver and pedestrian fatalities in Canada (Cimbura et al 1981),
- 6% of drivers killed in single vehicle crashes in North Carolina (Owens et al, 1983),
- 22% of driver casualties in Sydney (Chesher & Starmer, 1983),

- 7% of injured drivers in New Zealand (Bailey, 1984).
- 37% of young driver fatalities in California (Williams et al, 1985).

These figures for cannabis use are very similar to the 20% found among driver and pedestrian fatalities in the current study. All include both active and inactive metabolites of the parent drug and so cannot be interpreted in terms of impairment. However, the apparent frequency of use is three to four times higher than self-report surveys indicate for community (Roy Morgan, 1984).

About 29% of operators who had used cannabis had also been drinking; 69% of operators who had used cannabis had also used medication; 25% had used both alcohol and medication as well as cannabis, Drugs known to impair driving-related skills in healthy volunteers had also been used; for example, oxazepam and/or barbiturates were detected in 45% of cannabis users. Both of these prescription drug groups are known to be frequently diverted to illegal distribution networks and it

therefore seems likely that many of the individuals taking these drugs were not using this medication under medical direction; that is, the drugs will have affected the users in the same way that they affect healthy volunteers.

This suggests that, although cannabis use seems to be overrepresented in the study population, two thirds of the cases involved other drugs including alcohol which *may* have also affected driving-related skills.

Williams and his coworkers (1985) found in their survey of young male driver fatalities in California that no measures of crash responsibility were able to be related to blood levels of active cannabinoids. Their finding is consistent with Chesher & Starmer (1983) who were unable to relate blood levels of Δ9-tetrahydrocannabinol (the active component of cannabis) to impairment of driving-related skills. Interaction between alcohol and cannabis is synergistic among subjects who have not used cannabis previously but antagonistic among regular

cannabis users (Chesher et al, 1979; Chesher 1985).

The frequency with which the young Californian male fatalities combined alcohol with their cannabis use (84%) was much higher than that found in this study (29%). In contrast, combinations of cannabis and other drugs were less frequent in the American survey (33%) than in this Melbourne study (69%). These factors may have been influenced by the gender of the fatalities because men are more likely to have used alcohol, while more women use medication.

All these factors mean that there remains considerable doubt about the role which cannabis may play in causing the crashes in which it is involved.

Recommendation 4.

It is therefore recommended that:-

The role of cannabis in road crashes continue to be monitored and that any attempts to decriminalise the drug be drafted to include provisions which maintain and reinforce the social

irresponsibility of driving after its use.

Implementation of Recommendation 4.

The active components of cannabis are difficult to identify conclusively in blood and their levels cannot be related to effect or to crash risk (Chesher & Starmer 1983; Williams et al, 1985). Further, about one quarter of drug offenders charged with possession or use of cannabis were identified during vehicle checks '(Hendtlass, 1985).

Therefore, one practical way of implementing the recommendation could include proscription of carriage of cannabis in the passenger compartment of a motor vehicle, with penalties similar to those already in place for drink-driving offences. For consistency, open containers of alcohol could also be banned as is already the case in California.

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APPENDIX A. Effects of Drugs on Driving-Related Skills.

Table A.l. Effects of Stimulants on Driving-Related Skills.

Drug (Reference)	Measurement Techniaue	Subject Type	Reduction Performanc
Amphetamine (Ashworth, 1975)	Judgement of Speed	Healthy Volunteers	Improve
Amphetamine (Hurst, 1976)	General Review		-
Amphetamine (Gourevitch & Yanev, 1979)	Reaction Time		+
Amphetamine (Goldstein et al, 1960)	various	Normal Students	-

* + means performance is reduced.

\- means no effect.

Table A.2. Effects of Minor Analgesics on Driving-Related Skills.

Drug (Reference)	Measurement Techniaue	Subject Type	Reduction in Performance
Aspirin (Linnoila et al, 1974b)	Choice Reaction Co-ordination, Divided Attention	Healthy Students	-
Indomethacin (Linnoila et al, 1974b)	Choice Reaction Co-ordination Divided Attention	Healthy Students	+
Phenylbutazone (Linnoila et al, 1974b)	Choice Reaction Co-ordination, Divided Attention	Healthy Students	+

* + means performance is reduced.

\- means no effect.

Table 83. Effects of Benzodiazepines on Driving-Related Skil

Drug (Reference)	Measurement Technique	Subject Type	Reduction Performan
Chlordiazepoxide (Idestrom & Cadenius, 1963)	Driving	Healthy	
Chlordiazepoxide (Betts et al, 1972)	Vehicle Handling	Healthy Volunteers	+
Chlordiazepoxide (Linnoila & Mattila, 1973)	Motor Skills	Male Students	-
Chlordiazepoxide (Austen et al, 1973)	Driving	Healthy	-
Chlordiazepoxide (Linnoila,1973b)	Motor Skills Cognitive Skills	Healthy Volunteers	+ -
Chlordiazepoxide (Teo et al, 1975)	Motor Skills Sensory Function	Healthy Volunteers	+ +
Chlordiazeooxide (Betts & Biake, 1977)	Concentration	Normal Young Women	+
Diazepam (Haffner et al, 1973)	various Laboratory Tests	Healthy Young Males	+
Diazepam (Linnoila,1973b)	Choice Co-ordination Divided Attention Tasks	Healthy	+
Diazepam (Linnoila & Mattila, 1973)	Sensory Function Motor Skills	Male Students	+ -
Diazepam (Linnoila k Hakkinen 1974)	Motor Skills	Male Students	-
Diazepam (Mørland et al, 1974)	Various	Healthy Young Males	+

Table A3 (Continued).

Drug (Reference)	Measurement Technique	Subject Type	Reduction in Performance*
Diazepam (Linnoila et al, 1974a)	Reaction Choice Co-ordination	Healthy Male Volunteers	-
Diazepam (Teo et al, 1975)	Motor & Sensory Function	Healthy Volunteers	+
Diazepam (Liljeuquist et al, 1978)	Motor & Sensory Function	Healthy	+
Diazepam (Wetherell,1979)	Gap Judging	Drivers	Improve.
Diazepam (de Gier et al, 1981)	Driving Performance & Vigilance Tasks	Healthy Male Students	+
Diazepam (Moskowitz & Smiley,1982)	Driving Simulator Divided Attention	Men & Women Volunteers	+ +
Diazepam (O'Hanlon et al, 1982)	Lateral Position Control	Skilled Drivers	+
Diazepam (O'Hanlon et al, 1982)	Lateral Position Control	Healthy Male	+
Diazepam (Griffiths et al, 1984)	Various Psychomotor Tasks	Drug Abusers	+
Diazepam (de Gier, 1984)	Actual Driving	Anxious Patients	-
Flurazepam (Pishkin et al, 1980)	Cognitive Function		+
Flurazepam (Betts & Birtle, 1982)	Weaving Test Gap Test	Women Volunteers	+

Table A3 (Continued).

Drug (Reference)	Measurement Technique	Subject Type	Reduction Performa
Flurazepam (Meldelson et al, 1982)	Motor Tasks Reaction Time	Chronic Insomniacs	-
Flurazepam (O'Hanlon et al, 1983)	Actual Driving Performance	Patients	+
Flurazepam (Seppala et al, 1983)	Tracking Reactive Skills Critical Flicker Fusion True Anticipation	Normal Males Subjects	+
Flurazepam (Subhan,1984)	Response Latency	Normal	+
Lorazepam (Hindmarch & Gudgeon,1980)	Actual Driving	Healthy Volunteers	+
Lorazepam (Mattila et al, 1982)	Various	Healthy Male Students	+
Lormetazepam (Subhan,1984)	Response Latency	Normal Volunteers	-
Loprazolam (McManus et al, 1983)	Manual Dexterity Mental Arithmetic Tracking Memory	Healthy Male Students	+ + + +
Medazepam (Moore, 1977)	Cognitive Skills Motor Skills Sensory Function	Anxious Patients	+ - -
Nitrazepam (Idestrom & Cadenius,1963)	Motor Skills		+
Nitrazepam (Malpas et al, 1970)	Card Sorting Digit Symbol Substitution	Healthy Young Volunteers	+ + +

Table A3 (Continued).

Drug (Reference)	Measurement Techniaue	Subject Type	Reduction in Performance
Nitrazepam (Legg et al, 1973)	Various	Anxious Patients	-
Nitrazepam (Linnoila & Mattila, 1973)	Motor Skills	Male Students	+
Nitrazepam (Hossman et al, 1980)	Sensory Function	Healthy Males	+
Oxazepam (Palva & Linnoila, 1978)	Various Psychomotor	Healthy Young	+
Oxazepam (Griffiths et al, 1984)	Various Psychomotor	Drug Abusers	+
Temazepam (Pishkin et al, 1980)	Cognitive Function		+
Temazepam (Betts & Birtle, 1982)	Weaving Test Gap Test	Women Volunteers	+
Triazolam (Subhan, 1984)	Response Latency	Normal Volunteers	+

* + means performance is reduced.

\- means no effect.

Table A4. Effects of Barbiturates on Driving-Related Skills.

Drug (Reference)	Measurement Technique	Subject Type	Reductio Performa
Amylobarbitone (Calloway, 1959)	Attention		+
Amylobarbitone (Idelstrom & Cadenius, 1963)	Motor Skills		+
Amylobarbitone (Turner,1965)	Critical Flicker Fusion		+
Amylobarbitone (Betts et al, 1972)	Driving Gap Estimation Driving Gap Estimation	Normal Male Normal Female	+ + .Improve *Improve*
Amylobarbitone (Legg et al, 1973)	Various	Anxious Patients	-
Amylobarbitone (Ashworth,1975)	Vigilance Critical Flicker Frequency	Drivers	+
Amylobarbitone (Linnoila,1976)	Vehicle Handling	Healthy Volunteers	+
Amylobarbitone (Manton,1977)	Tracking Detection	Male Normal	+ +
Pentobarbitone (Quarton k Tallend 1962)	Attention		+
Phenobarbital (Kielholz,1969)	Driving	Normal Subjects	+
Secobarbital (Goldstein et al, 1960)	Various	Normal Students	+

* + means performance is reduced.

\- means no effect.

Table A5 Effects of Hypnotics, Sedatives, Tranquilisers & Antipsychotics (Other than Benzodiazepines & Barbiturates.

Drug (Reference)	Measurement Techniaue	Subject Type	Reduction in Performance
Buspirone (Moskowitz & Smiley,1982)	Driving Simulator Divided Attention	Male Female Volunteers	Improve Improve
Buspirone (Mattila et al, 1982a)	Various	Healthy Male Students	-
Chlorpromazine (Zirkle et al, 1959)	Various	Volunteers & 'Patients	+
Chlorpromazine (Manton, 1977)	Tracking Task Detection Task	Male Normal	+ +
Chlorpromazine (Hartley et al, 1978)	Serial Reaction Performance	Healthy Male 15-26 years	+
Chlorpromazine (Hartley & Couper Smart, 1978)	Actual Driving Motor Skills	Healthy Males	+ +
Chlorpromazine (Liljeuquist et al, 1978)	Actual Driving Motor Skills Sensory Function		- - -
Glutethimide (Mould et al, 1972)	Reaction Time Tapping Speed	Healthy	+ +
Haloperido1 (Linnoila,1973b)	Choice,Co-ordination & Attention tasks	Healthy Males	+
Haloperidol (Ashworth,1975)	Critical Flicker Fusion		-
Haloperido1 (Girotti et al, 1984)	Motor Performance	Huntingdon's Disease	-
HOE 8476 (Parrott et al, 1982)	Various	Normal	-

Table A5 contd.

Drug (Reference)	Measurement Techniaue	Subject Type	Reductio Performa
Midazolam (Crevoisier, et al, 1983)	Reaction Time Tracking Test	Healthy Volunteers	+
Midazolam (Hindmarch & Subhan,1983)	Car Driving Critical Flicker Fusion	Healthy Female Volunteers	+ +
Nomifensine (Hindmarch,1977)	Night-time Driving	Healthy	Sligh
Nomifensine (Parrott et al, 1982)	Various	Normal	-
Thioridazine (Linnoila, 1973b)	Choice,Co-ordination Attention tasks	Healthy Males	+
Zopiclone (Seppala et al, 1983)	Tracking Reactive Skills Critical Flicker Fusion True Anticipation	Normal Males	-

* + means performance is reduced.

\- means no effect.

Table A6 . Effects of Anticonvulsants on Driving Related Skills.

Reference	Measurement Technique	Subject Type	Reduction in Performance *
Phenytoin (Trimble & Thompson,1981)	Various Cognitive Function tests	Healthy Volunteers	+
Phenytoin (Trimble & Thompson,1981)	Various Cognitive	Epileptics	+
Carbamazepine (Trimble & Thompson, 1981)	Various Cognitive Function tests	Healthy Volunteers	+
Carbamazepine (Trimble & Thompson,1981)	Various Cognitive Function tests	Epileptics	+
Sodium Valproate (Trimble & Thompson, 1981)	Various Cognitive Function tests	Healthy Volunteers	+
Sodium Valprcate (Trimble & Thompson,1981)	Various Cognitive Function tests	Epileptics	+
Clobazam (Trimble & Thompson,1981)	Various Cognitive Function tests	Healthy Volunteers	+
Clobazam (Trimble & Thompson,1981)	Various Cognitive Function tests	Epileptics	+
Clobazepam (Hindmarch & Gudgeon,1980)	Actual Driving	Healthy Volunteers	-
Clobazam (Parrott & Hindmarch,1978)	Choice Reaction Time	Normal	+

* + means performance , is reduced.

\- means no effect .

Table A7 . Effects of Antidepressants on Driving-Related Skills.

Drug (Reference)	Measurement Technique	Subject Type	Reduction i Performance
Tricyclic Antidepressants.			
Amitriptyline (Patman et al, 1969	Several	Normal (Chronic) Dose	+
Amitriptyline (Ban et al. 1982)	Several	Normal (Chronic) Dose	+
Amitriptyline (Crome & Newman, 1978)	Reaction Times Critical Flicker	Healthy Volunteers	+ +
Amitriptyline (Hindmarch et al, 1983)	Brake Reaction Time Critical Flicker Fusion Choice Reaction Time Tracking	Healthy Female Volunteers	+
Amitriptyline (Linnoila et al, 1983)	Continuous Performance & Cognitive Memory Tasks	Healthy Males	+
Amitriptyline (Seppala et al, 1984)	Critical Flicker Fusion Co-ordination	Healthy Volunteers	+ +
Chlorimipramine (Seppala et al, 1978)	Motor Skills Sensory Function	Depressed Patients	+ -
Desipramine (Linnoila et al, 1983)	Continuous Performance k Cognitive Memory Tasks	Healthy Males	+
Doxenin (Seppala et al, 1978)	Motor Skills Sensory Function	Depressed Patients	+ -
Doxepin (Seppala et al, 1978)	Motor Skills Sensory Function	Healthy Volunteers	+ -

Table A7. (Continued).

Drug (Reference)	Measurement Technique	Subject Type	Reduction in Performance *
Imipramine (Clayton et al, 1977)	Closed Course	Male Students	+
Imipramine (Bayliss & Duncan., 1974)	Sensory Function	Male	+
Nortriptyline (Bye et al, 1978)	Reaction Times Audible Vigilance Digit Symbol Substitution	Healthy Volunteers	+ at 25 mg - 12.5 mg
Monoamine Oxidase Inhibitors.			
Amoxine (Ban et al, 1982)	Motor Reflex Motor Co-ordination Depth Perception	Normal Male	-
Hydroxyzine (Pishkin & Shirley, 1983)	Various	Healthy Volunteers	+ at 25 mg - 12.5 mg
Lithium (Linnoila et al, 1974 a)	Choice Reaction	Healthy Male Volunteers	+
Lithium (Dasso et al, 1982)	Memory Reaction Times	Manic Depressives	-
Mianserin (Crome & Neman, 1978)	Reaction Times Critical Flicker	Healthy Volunteers	+ +
Mianserin (Seppala et al, 1984)	Critical Flicker Fusion Nystagnus Reaction Times	Healthy Volunteers	+ + +
Nomifensine (Hindmarch, 1977)	Night-time Driving Performance	Volunteers	-
Viloxazine (Clayton et al, 1981)	Closed Course	Male Students.	-

Table A7 (continued)

Drug (Reference)	Measurement Technique	Subject Type	Reduction Performance
Viloxazine (Bayliss & Duncan, 1974)	Cognitive Skills Motor Skills	Males	- -
Zimeldine (Hindmarch et al, 1983)	Brake Reaction Time Critical Flicker Fusion Choice Reaction Time Tracking	Healthy Female Volunteers	-
Zimeldine (Linnoila et al, 1983)	Continuous Performance & Cognitive Memory Tasks	Healthy Males	-
Zimeldine (Seppala et al, 1984)	Critical Flicker Fusion Reaction Times Co-ordination	Healthy Volunteers	- - -

* + means performance is reduced.

\- means no effect.

Table A8. Effects *of* Cardiovascular Drugs on Driving-Related Skills.

Drug (Reference)	Measurement Techniaue	Subject Type	Reduction i Performance
Atenolol (Salem & McDevitt, 1983)	Various Psychomotor	Healthy Males	+
Atenolol (Betts & Blake, 1977)	Concentration	Healthy Young Women	-
Atenolol (Clayton, Harvey & Betts, 1977)	Visual Acuity	Healthy Males	-
Clonidine (Hossman et al, 1980)	Sensory Function	Healthy Males	+
Methyldopa (Clayton, Harvey & Betts, 1977)	Visual Acuity	Healthy Males	+
Propanolol (Bryan et al, 1974)	Visual Function		+
Propanolol (Ogle & Turner, 1974)	Flicker Fusion		-
Propanolol (Clayton, Harvey & Betts, 1976)	Actual Driving	Healthy Males	-
Propanolol (Clayton, Harvey & Betts, 1977)	Visual Acuity	Healthy Males	-
Reserpine (Clayton, Harvey & Betts, 1977)	Visual Acuity	Healthy Males	+

* + means performance is reduced.

\- means no effect.

Table A9 . Effects of Antihistamine Preparations on Driving-Belated Skills.

Drug (Reference)	Measurement Technique	Subject Type	Reduction i Performance
Astemizole (Nicholson et al, 1982)	Dynamic Acuity Flicker Fusion		-
Chlorpheniramine (Clarke & Nicholson 1978)	Motor Skills	Healthy Females	+
Chlorpheniramine (Hindmarch & Parrott, 1978a)	Complex Reaction Time Flicker Fusion	Healthy Volunteers	- +
Chlorpheniramine (Kulshrestha et al, 1978)	Cognitive Skills	Healthy Females	+
Clemastine (Hindmarch & Parrott, 1978a)	Complex Reaction Time Flicker Fusion	Healthy Volunteers	- -
Demastine (Clarke & Nicholson, 1978)	Motor Skills	Healthy Females	+
Dexchlorpheniramine (Franks et al, 1978)	Various	Healthy Volunteers	+
Dexchlorpheniramine (Mattila et al, 1978)	Driving Cognitive Skills	Male Students	- -
Diphenylpryaline (Manton, 1977)	Tracking Test Detection Task	Male Normal	- +
Ketotifen (Hindmarch & Parrott, 1978a)	Complex Reaction Time Flicker Fusion	Healthy Volunteers	- -
Mebhydrolin (Hindmarch & Parrott, 1978a)	Complex Reaction Time Flicker Fusion	Healthy Volunteers	- -
Promethazine (Clarke & Nicholson, 1978)	Motor Skills Sensory Function	Healthy Adults	- -

Table A9 . (continued).

Drug (Reference)	Measurement Technique	Subject Type	Reduction in Performance *
Promethazine (Hindmarch & Parrott, 1978a)	Complex Reaction Time Flicker Fusion	Healthy Volunteers	- -
Tavegil (Hindmarch & Parrott, 1978a)	Complex Reaction Time Flicker Fusion	Healthy Volunteers	- -
Terfenadine (Mould et al, 1972)	Motor Skills Sensory Function	Normal Males	- -
Terfenadine (Kulshrestha et al, 1978)	Motor Skills Sensory Function	Healthy Females	- -
Terfenadine (Nicholson et al, 1982)	Dynamic Acuity Flicker Fusion		- -
Terfenadine (Betts et al, 1984)	Actual Driving	Experienced Women Drivers	-
Triarolidine (Nicholson et al, 1982)	Dynamic Acuity Flicker Fusion		+ +
Tripolidine (Betts et al, 1984)	Actual Driving	Experienced Women Drivers	+

* + means performance is reduced.

\- means no effect.

Table A10 Effects of Cannabis on Driving-Related Skills.

Reference	Measurement Technique	Subject Type	Reduction Performance*
Manno et al, 1971	Various	Healthy Male Volunteers	+
Moskowitz, 1973	various	Healthy Male Volunteers	+
Rafaelsea et al, 1973	Driving Simulator	Servicemen	+
Chesher & Starmer, 1983	Various	Male & Female Students	+
Sutton, 1983	Driving Performance	Male Volunteers	-
Attwood, 1980	Closed Course Driving	Male Volunteers	+
Chesher, 1985	Various	Cannabis Users	+

* + means performance is reduced.

\- means no effect.

APPENDIX B. Effects *of* Alcohol in Combination with Other Drugs on Driving-Related Skills.

Stimulants	Test	Effect of
Amphetamines (Hurst,1976)	General	Antagonistic
Minor Analgesics.		
Aspirin (Linnoila et al, 1974b)	Choice, Reaction Co-ordination Divided Attention	No effect
Indomethacin (Linnoila et al, 1974b)	Choice, Reaction Co-ordination Divided Attention	Antagonistic
Phenylbutazone (Linnoila et al, 1974b)	Choice, Reaction Co-ordination Divided Attention	Additive
Hypnotics, Tranquilisers, Sedatives & Antipsychotics.		
Benzodiazepines		
Chlordiazepoxide (Linnoila,1973b)	Choice, Co-ordination Attention Tasks Tracking	Additive
Diazepam (Moskowitz & Smiley, 1982)	Critical Flicker Fusion Reactive Tests Time Anticipation	Additive
Diazepam (Dundee et al, 1971)		Additive
Diazepam (Linnoila,1973b)	Choice Co-ordination Reaction task	Additive
Diazepam (Linnoila et al, 1974)	Reaction Time Choice Co-ordination	Additive
Diazepam (Mórland et al, 1974)	Various	Additive
Diazepam (Mattila et al, 1978)	Various	Additive

Antidepressants.

Amitryptyline (Ban et al, 1982)	Response Time	Synergistic
Amitryptyline (Linnoila et al, 1983)	Continunus Performance & Cognitive Learning	Additive
Amitryptyline (Seppala et al, 1984)	Critical Flicker Fusion Co-ordination	Additive Additive
Amoxazine (Ban et al, 1982)	Response Time	Additive
Desipramine (Linnoila et al, 1983)	Continuous Performance & Cognitive Learning Tasks	Additive
Mianserin (Seppala et al, 1984)	Critical Flicker Fusion Reaction Times	Additive Additive

Antihistamines.

Antihistamines (Coleman & Evans, 1975)	Sedation	Additive
Dexchlorpheniramine (Franks et al, 1978)	Various	Synergistic
Diphenhydramine (Burns & Moskowitz, 1980)	various	Additive
Terfenadine (Moser et al, 1978)	Various	No effect
Zopiclone (Seppala et al, 1983)	Tracking Critical Flicker Fusion Reactive Tests Time Anticipation	Additive

Flunitrazepam (Seppala et al, 1983)	Tracking Critical Flicker Fusion Reactive Tests Time Anticipation	Synergistic
Lorazepam (Mattila et al, 1982)	Peripheral Vision Tracking	Synergistic
Loprazolam (McManus et al, 1983)	Manual Dexterity Tracking Memory	Antagonistic Antagonistic Additive
Midazolam (Hindmarch & Subhan, 1983)	Car Driving Critical Flicker Fusion	Additive Additive
Oxazepam (Palva & Linnoila, 1978)	Various Psychomotor	Additive
Oxazepam (Laisi et al, 1979)	Various	Additive

Barbiturates.

Phenobarbitone (Joyce et al,1959)	Various	Synergistic
Phenobarbital (Kalant et al, 1970)	Various	Synergistic

Cannabis.

Cannabis (Rafaelsen et al, 1973)	Driving Simulator	Additive
Cannabis (Chesher et al, 1977)	Various Psychomotor Tests	Additive
Cannabis (Moskowitz et al, 1978)	Driving Simulator	Additive

Cannabis (Sutton, 1983)	Driving Performance	Synergistic
Cannabis (Stein & Allen,1984)	Driving Simulator	Synergistic
Cannabis (Chesher, 1985)	Reaction Time Choice Reaction Time "Little Men"	Slight Additive Slight Additive Antagonism

Other Hypnotics. Tranquilisers, Sedatives & Antipsychotics.

Buspirone (Moskowitz & Smiley, 1982)	Tracking Critical Flicker Fusion Reactive Tests Time Anticipation.	Additive
Buspirone (Mattila et al, 1982a)	Peripheral Vision Tracking	Additive Additive
Glutethimide (Mould et al, 1972)	Reaction Time Tapping Speed	Additive
Haloperidol (Linnoila, 1973b)	Choice,Co-ordination & Attention tasks	Additive at high doses.
Haloperido1 (Ashworth, 1975)	Critical Flicker Fusion	
Thioridazine (Linnoila, 1973b)	Choice,Co-ordination & Attention tasks.	Additive

APPENDIX C. General Procedures Used for Analysis of Blood and Urine.

The general analytical procedures used in this project involved:

enzyme hydrolysis

extraction

chemical ionisation screening using the direct insertion probe on the mass spectrometer

thin layer chromatography confirmation.

All analyses were performed in duplicate. Drugs were not recorded as positive unless either

a) The primary molecular ion was present in the direct insertion probe screening and

b) It had the same R_f as the appropriate standard in three thin layer chromatography solvents and

c) It responded appropriately to five thin layer chromatography detection procedures;

or EMIT procedures were used to screen urine for cannabinoids, amphetamines and benzodiazepines. These needed confirmation using the general screening procedure before they were reported (Appendix D).

or Urines were screened for salicylate using the Trinders Reagent (Appendix D).

or Urine or blood extracts containing the primary ion for caffeine or nicotine at a level equivalent to greater than 0.2µg/ml were deemed positive for the purposes of the study.

General Screening Procedure.

1. Enzyme Hydrolysis:-

All samples of blood and urine were hydrolysed for 24 hours at pH 5.2 at room temperature using a mixed glucuronidase-sulphatase enzyme solution. N. Nitrosodiphenylamine was used as an internal standard. This was introduced following hydrolysis *and* before extraction.

2. Extraction:-

Samples were extracted sequentially at pH 7.4, pH 10.8 and pH 2.2, using 1:9 solution of isopropanol in chloroform.

All three solvent phases were pooled. The solution was centrifuged (20 mins x 1500 rpm) and the solvent dried. The percentage extraction for 49 drugs included in the screening procedure is show in Table C1 at the end of this Appendix..

3. Chemical Ionisation/Mass Spectrometry:-

The residue was taken up in 100 µl methanol and 4 X 10 µl aliquots were used to make up two duplicate capillary probes. These were analysed using the temperature programmed direct insertion probe option of the mass spectrometer (initial temperature 200°C, rate 30°C per minute). Ammonia reagent gas was used for best sensitivity for basic drugs and isobutane was used for neutral and acidic drugs.

The abundance of the primary molecular ion for each of the target drugs was integrated over the entire programme. Each ion current relative to that of the internal standard was then calculated off-line. Base line values were calculated from blank blood and urine samples. The least detectable concentration of each drug is listed in Table C2 at the end of this Appendix.

4. Thin Layer Chromatography.

The remaining residue was dried down and taken up in 25 µl methanol. 5 µl aliquots were applied to 3 thin layer silica gel chromatography plates with appropriate standards (Gelman Instrument Company, 1975).

One plate was run in an Acidic Solvent (32.0% v/v ethyl acetate in hexane). This plate was viewed under ultra violet light. It was

then sprayed sequentially with silver acetate, diphenylcarbazone and potassium permanganate.

One plate was run in Basic Solvent (5.3% ethanol & 21.1% chloroform v/v in ethyl acetate. 1.1% ammonia was added immediately before use). This plate was viewed under ultra violet light. It was then sprayed sequentially with ninhydrin and iodoplatinate.

One plate was run in Confirming Basic Solvent (6.7% methanol v/v in ethyl acetate. 5.4% ammonia added immediately before use). This plate was viewed under ultra violet light. It was then sprayed sequentially with ninhydrin and iodoplatinate.

The lowest quantity of each drug which was detectable by thin layer chromatograp methods is shown in Table C2.

Detectable Levels.

The blood concentrations of drugs following therapeutic doses have been reported in the literature (Baselt et al, 1975; Winek, 1976; Stead & Moffat, 1983).

All of these reports appear to be based on free drug levels and do not take account of that which is normally bound to sugars, salts or proteins. In those cases, the reported therapeutic level is therefore lower than that expected in hydrolysed blood.

The concentrations at which drugs were able to be detected and confirmed in blood were generally in the reported therapeutic range *for:-*

Caffeine
Chlorpromazine
Glutethimide
Methadone
Methaqualone
Methprylon
Oxazepam
Paracetamol
Phenytoin
Quinine
Trifluorperazine (see Table C2)

Other substances were detectable in blood if they were present at therapeutic levels but were normally bound to sulphates or glucouronides.

These drugs include:-

Amitriptyline
Codeine
Methadone
Morphine
Nortriptyline
Pethidine.

.Further, many drugs accumulate in the urine and *can* therefore be expected to be normally present at higher levels in urine than they are in the blood. They would therefore be detected in urine even if the level was too low to be detectable in blood.

These drugs include:-

Amphetamine
Chlordiazepoxide
Diazepam
Ephedrine
Imipramine
Methamphetamine
Nitrazepam
Phenylpropanolamine
Pseudoephedrine
Tetrahydrocannabinol
Thioridazine

Table C.l.

Extraction* of Drugs from Spiked Blood & Urine.

Parent Drug.	Percent Extraction	Primary Molecular Ion	Reagent Gas
Acetyl Salicylate	100.0	181.2	Isobutane
Amitriptyline	102.5	278.3	NH_3
Amphetamine	14.7	136.2	NH_3
Amylobarbitone	116.9	227.2	Isobutane
Atropine	100.0	290.3	NH_3
Barbitone	100.0	185.3	Isobutane
Butabarbitone	36.3	213.3	Isobutane
Caffeine	100.0	195.1	NH_3
Chloral Hydrate	68.3	165.2	Isobutane
Chlorpromazine	100.0	319.3	NH_3
Chlordiazepoxide	100.0	300.3	NH_3
Cocaine	26.7	304.4	NH_3
Codeine	100.0	300.3	NH_3
Desmethyldiazepam	61.6	271.2	NH_3
Dextromoramide	25.2	393.2	NH_3
Diazepam	<10	285.2	NH_3
Diphenhydramine	<10	255.2	-
Disulphiram	100.0	297.3	NH_3
Doxepin	39.4	280.4	NH_3
Ephedrine	71.2	166.2	NH_3
Glutethimide	55.1	218.2	NH_3
Imipramine	90.4	281.3	NH_3
Indomethacin	7.9	358.3	Isobutane

Table C1 (Continued).

Methampetamine	25.3	150.2	NH_3
Methadone	100.0	310.3	NH_3
Methaqualone	21.4	251.3	NH_3
Methprylon	109.0	184.3	NH_3
Morphine	37.8	286.2	NH_3
Nicotine	65.2	163.2	NH_3
Nitrazepam	66.2	282.3	NH_3
Nortriptyline	100.0	264.2	NH_3
Oxazepam	61.6	287.2	NH_3
Paracetamol	33.0	152.2	Isobutane
Pethidine	62.1	248.4	NH_3
Pentobarbital	116.9	227.2	Isobutane
Phenobarbital	48.1	233.2	Isobutane
Phenylpropanolamine	103.4	152.2	NH_3
Phencyclidine	100.0	244.4	NH_3
Phenytoin	57.5	253.2	NH_3
Phenacetin	76.7	180.3	NH_3
Promethazine	51.2	285.2	NH_3
Propoxyphene	100.0	340.4	NH_3
Pseudoephedrine	71.2	166.2	NH_3
Quinalbarbitone	63.9	227.2	Isobutane
Quinine	21.2	325.3	NH_3
Tetrahydrocannabinol	84.6	315.4	NH_3
Thioridazine	29.4	371.4	NH_3
Thiopentone	44.5	243.3	Isobutane
Trifluorperazine	106.0	408.3	NH_3

* Calculated from mean size of primarymolecular ion peak in extract minus mean size of that peak in blank over six extractions each analysed in duplicate relative to size of internal standard (Primary Molecular Ion, 170.1).

Table C.2.

Lower Limit of Detection for Each Drug Using General Procedures.

Parent Drug.	Lower Limit of Detection Chemical Ionisation (µg/sample)*	Lower Limit of Detection Thin Layer Chromatography (µg/sample)*	Blood or Plasma Le Reported Therapeut Dose ** (µg/ml).
Atropine	1.4	ND	
Acetyl Salicylate	0.1	77.0	2.0 - 100
Amitriptyline	1.64	1.20	0.035 - 0.
Amphetamine	0.5	11.6	0.02 - 2.0
Amylobarbitone	0.1	71.2	2 - 13
Barbitone	0.1	83.3	5 - 30
Butabarbitone	0.1	229.5	2 - 16
Caffeine	0.1	0.8	2 - 10
Chloralhydrate	0.2	ND	15 - 15
Chlordiazepoxide	0.7	16.7	1 - 8
Chlorpromazine	0.8	0.8	0.1 - 2
Cocaine	2.7	4.7	0.01 - 0.3
Codeine	0.7	0.4	0.01 - 0.1
Desmethyldiazepam	0.3	1.7	0.01 - 0.0
Dextromoramide	10.8	<0.1	
Diazepam	0.3	1.7	0.05 - 2
Diphenhydramine	0.6	20.0	0.05 - 0.(
Disulphiram	16.7	3.8	0 - 6
Doxepin	14.1	20.1	0.03 - 3
Ephedrine	117	120	0.02 - 0.1

Table C.2. continued.

Glutethimide	0.3	>5	0.2 - 45
Imipramine	0.1	0.9	0.01 - 0.25
Indomethacin	8.4	ND	0.7 - 4
Methadone	0.1	0.8	0.3 - 1.1
Methamphetamine	0.3	4.2	0.02 - 0.06
Methaqualone	0.1	3.9	0.4 - 7.0
Methprylon	0.1	0.8	0.5 - 20
Morphine	0.1	1.1	0.04 - 0.5
Nicotine	0.2	31.2	0.01 - 0.3
Nitrazepam	0.3	18.9	0.03 - 0.07
Nortriptyline	2.5	0.8	0.05 - 02
Oxazepam	0.3	0.3	0.5 - 2.0
Paracetamol	1.6	1.0	2.3 - 25
Phenacetin	1.91	45.4	0.01 - 1.0
Pentobarbital	0.1	71.2	1 - 10
Bethidine	2.0	48.2	0.2 - 0.8
Phenobarbital	0.1	173.2	4 - 26
Phenylpropanolamine	4.8	5.6	0.05 - 0.1
Phenytoin	0.9	0.3	3 - 20
Phencyclidine	0.2	6.0	0.- 5
Promethazine	0.8	47.0	1 - 2
Propoxyphene	0.1	<5	0.05 - 0.2
Pseudoephedrine	117	5.8	0.3 - 0.7
Quinalbarbitone	0.1	130.3	2 - 10
Quinine	0.4	1.9	2 - 8
Thioridazine	>500	283	0.05 - 5.0
Thiopentone	0.4	28.1	3 - 7
Tetrahydrocannabinol	6.1	6.5	0.079 - 0.26
Trifluorperazine	0.8	0.5	0.8

** Range reported by Baselt et al, 1975; Winek, 1976; Stead & Moffat, 1983.

* Normal sample size was 2 mls urine or 1ml blood.

These levels do not include drugs bound to sugars, salts or proteins.

APPENDIX D. Special Screening Procedures.

Special screening procedures were used for some substances whose ions had high background level in the routine chemical ionisation screen i.e. acetyl salicylate; or were known to be difficult to interpret i.e. cannabinoids; or showed *low* sensitivity to either the chemical ionisation or thin layer chromatography parts of the routine screen i.e. benzodiazepines and amphetamines.

Cannabinoids.

200 urine and/or blood samples from driver or pedestrian fatalities were analysed for cannabinoids. In 139 cases, urine was screened using EMIT procedures and the results were confirmed using the routine procedures; in 32 cases, only blood was analysed using routine procedures; in 29 cases, blood and urine was available so both types of analysis.was performed.

Cannabinoids were detected in 41 of these samples, that is 20.5% of driver, motor cycle and pedestrian fatalities; this includes 12 or 19.7% of cases in which blood was analysed.

Salicylates.

153 urine samples from driver and pedestrian fatalities were analysed for salicylates using the analytical test described by Clarke (1976). 32% were positive to Trinders reagent. Of these assays 26, or 17.0% of the total were confirmed to contain acetyl salicylate using routine procedures describedin Appendix C.

A further 7 blood samples, or 4.2% of the total analysed were also found to contain acetyl salicylate.

This means that acetyl salicylate was present in 10.3% of those individuals for whom analyses were available.

Benzodiazepines.

150 urine samples from driver or pedestrian fatalities were screened for benzodiazepines using EMIT procedures. Of these assays, diazepam, desmethyloxazepam and/or oxazepam was confirmed in 15 or 10% of the total using routine screening procedures.

In a further 19 blood analyses, oxazepam was detected once, and/or desmethyl twice and/or diazepam once.

This means that diazepam, oxazepam or

their metabolites were confirmed in 11.2% of cases.

APPENDIX E. Information Collected in Survey.

1. Study Number.
2. Date of Event.
3. Day of Week.
4. Time of Incident.
5. Place.
6. Age.
7. Sex.
8. Licence.
9. Occupation ..
10. Place of Residence.
11. Injury.
12. State of Patient on arrival.
13. Hospital.
14. Type of crash - including train, tram etc.
15. RUM Number.
16. Vehicle Type.
17. Vehicle - Year of Manufacture.
18. Cause of Death - Coronial Report.
19. Coroners Verdict.
20. Liability - Report of police at scene.
21. Drugs Reported.
22. Prescribed Medication Reported.
23. Drugs - Source of Information.

APPENDIX E (Continued).

24. Number of Preparations Reported.
25. Total Number of Separate Compounds Xeported.
26. First Drug.
27. Second Drug.
28. Third Drug.
29. Fourth Drug.
30. Benzodiazepine Screen Result.
31. Caffeine.
32. Nicotine .
33. Time of Breath Test. -
34. Other Evidence of Alcohol.
35. Blood Alcohol Reading.

APPENDIX F. Effects of Alcohol, Caffeine & Nicotine on Driving-Related Skills.

Table El. Effects of Alcohol on Driving-Related Skills.

Reference	Measurement Techniaue	Subject Type	Reduction Performar
Haffner et al, 1973	Various Laboratory Tests	Healthy Young Males	+
Moskowitz, 1973	various		+
Evans et al, 1974	Various	Males	+
Haffner et al,	Several	Healthy Young Males	+
Linnoila et al, 1974a,b.	Choice Reaction Co-ordination Attention	Healthy Male Volunteers	+
Franks et al, 1976	various	Healthy Young Students	+
Attwood et al, 1980	Closed-Course	Social Drinkers	+
Antebi, 1982	Four Choice Serial Reaction Time	Male Healthy	+
Linnoila et al, 1983	Continuous Performance & Cognitive Learning, Tasks	Healthy Male Volunteers	+
Glencross et al, 1984	Primary & Secondary Tasks	Healthy Volunteers	+
Moskowitz et al, 1984	Divided Attention	Healthy Volunteers	+
Chesher, 1985	Various	Healthy Marijuana Users	+

* + means performance is reduced.

\- means no effect.

Table F2 Effects of Caffeine on Driving-Related Skills.

Drug (Reference)	Measurement Technique	Subject Type	Reduction in Performance
Caffeine (Franks et al, 1975)	Reaction Time	Volunteers	Improve
Caffeine (Childs,1978)	Target Scanning Performance	Low Caffeine Using Male Volunteers	Improve
Caffeine (Clubley et al, 1979)	Vigilance Reaction Time	Healthy	+
Caffeine (Moskowitz & Burns, 1981)	Tracking Divided Attention Task Information Processing Critical Tracking	Male Moderate Caffeine Users	Improve - - Improve
Caffeine (Broverman & Casagrande 1982)	Perceptual Bestructuring Task		+
Caffeine (Spiegel,1982)	Daytime Vigilance	Elderley	-

* + means performance is reduced.

\- means no effect .

Table F 3. Effects of Nicotine on Driving-Related Skills.

Drug (Reference)	Measurement Technique	Subject Type	Reduction Performance
Nicotine (Heimstra et al, 1967)	Driving Simulator	Smokers	Improve
Nicotine (Williams, 1980)	Letter Cancellation	Smokers	Improve
Nicotine (Wesnes & Warburton. 1983)	Rapid Information Processing	Male	Improve
Nicotine (Wesnes & Warburton, 1983)	Rapid Information Processing	Male & Female Smokers	Improve

* + means performance is reduced.

\- means no effect.

APPENDIX G. Costing of Project.

This project was funded by direct and indirect contributions from the Federal Office of Road Safety, Victoria Police, State Laboratories and the Coroner. The following conservative estimates are based on 1982-83 costs, rounded to nearest $1000.

Federal Office of Road Safety: $74,000
including salaries of research staff* and expenses incurred by them in performing their tasks e.g. analytical requirements, computing, travel.

Victoria Police: - estimate $33,000
at 60% of cost of salaries including office space and costs associated with employ'ing consultants e.g. Workers Compensation, telephone, stationary and some typing.

State Laboratories : - estimate $72,000
including laboratory space, development of analytical procedures (about $20,000), analyses of standards ($6,000) and analyses of body fluid from fatalities (356 samples in duplicate

at $65 each, not including repeats, 846,000).

Coroner: - estimate $5,000 including taking, labeling and storing samples taken during post-mortem at $15 each.

TOTAL $184,000

* ($55,000) - time for extraction of information from police files, analysis *of* data and writing of the report not covered in this amount.