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NDLERF

The impact of drugs on road crashes,
assaults and other trauma –
a prospective trauma toxicology study

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The impact of drugs on road crashes, assaults and other trauma – a prospective trauma toxicology study

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Abbreviations

AIS	Abbreviated Injury Scale
AODs	alcohol and other drugs
Amphet	amphetamine
ATSI	Aboriginal and Torres Strait Islander
BAL	blood alcohol level
Benzo	benzodiazepines
CI	confidence interval
Delta-9-THC	Delta-9-Tetrahydrocannabinol – an active ingredient of cannabis (aka THC)
DMA	dimethylamphetamine
DOM	2,5-dimethoxy-4-methylamphetamine
DOTA	drugs other than alcohol
EAST	Eastern Association for the Surgery of Trauma
ED	Emergency Department
ELISA	enzyme-linked immunosorbent assay
ETOH	Ethanol = Alcohol
HDU	High Dependency Unit
ICD-10	International Classification of Diseases Revision 10
ICU	Intensive Care Unit
IMVS	Institute of Medical and Veterinary Science
ISS	Injury Severity Score
MDA	3,4-methylenedioxyamphetamine
MDMA	3,4-methylenedioxymethylamphetamine
MVCs	motor vehicle crashes
NDSHS	National Drug Strategy Household Survey
NISS	New Injury Severity Score
PMA	paramethoxyamphetamine
RAH	Royal Adelaide Hospital
RBT	random breath testing
RTS	Revised Trauma Score
SAPOL	South Australia Police
SDU/HDU	Step Down Unit (aka High Dependency Unit)
THC	Tetrahydrocannabinol – an active ingredient of cannabis (aka Delta-9-THC)
THC acid	11-nor-delta-tetrahydrocannabinol-9-carboxylic acid – an inactive metabolite of THC

TS	Trauma Service
Group 1	Patients with severe injuries seen by the RAH Trauma Team
Group 2	Patients with less severe injuries seen by the RAH Emergency Department
Group 3	Control group comprising non-trauma blood samples from the Institute of Medical and Veterinary Science

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Executive Summary

Overview/Study design

This prospective study is one of the largest of its type ever completed, recruiting 2,127 patients from two trauma groups and one control group.

Its findings provide compelling evidence of the incidence and severity of drug- and alcohol-related trauma in South Australia.

They also have significant implications for health, law enforcement, policy-making and research in relation to the recognition of the impact of drugs on a range of trauma. Patients who are positive for alcohol and other drugs (AODs) create an additional, and presumably otherwise avoidable, financial burden on the health system. Significantly, the findings also add to the growing evidence base for trauma related to drug driving.

The analysis of drug levels in blood rather than urine (as used in most previous related studies) allows for better correlation of results with recent drug usage.

The first arm of the study involved 1,515 patients (Group 1) presenting with injuries severe enough to be seen by the Royal Adelaide Hospital's (RAH) Trauma Team.

The second arm involved 202 patients (Group 2) from the RAH Emergency Department with less severe injuries.

Group 3 comprised 410 patients acquired for comparison from the Institute of Medical and Veterinary Science. This arm looked at random, non-trauma blood samples matched for age, gender and postcode.

SA Police random breath testing data for the period of the study is also included to help identify background alcohol use by South Australian drivers.

Key findings

Alcohol remains the most common recreational drug found in trauma patients. Other recreational drugs, especially cannabis, are also associated with trauma. There is evidence to suggest that use of recreational drugs before/while driving is associated with increased risk of injury occurrence and severity.

With respect to driving and alcohol and other drugs:

- Injury from road trauma appears related to a number of drugs including alcohol, cannabis, stimulants, benzodiazepines, and opiates.
- Alcohol was found in 22.6 per cent of injured car drivers (122 of 539).
- Tetrahydrocannabinol (THC)/THC acid was found in 17.4 per cent of injured car drivers (94 of 539).
- Benzodiazepines were found in 14.7 per cent of injured car drivers (79 of 539).
- Amphetamines were found in 6.9 per cent of injured car drivers (37 of 539).
- Opiates were found in 3.3 per cent of injured car drivers (18 of 539).

Large numbers of patients from other trauma causes were also positive to alcohol and other drugs:

- 72.2 per cent of patients affected by interpersonal violence were positive to alcohol and other drugs (83 of 115).
- 53 per cent of patients affected by interpersonal violence were positive to drugs other than alcohol (61 of 115).
- 34 per cent of patients injured in falls were positive to alcohol and other drugs, predominantly in an older population (67 of 197).
- 24.2 per cent of patients from 'industrial/construction sites' were positive for alcohol and other drugs (15 of 62). 55.7 per cent of patients from a 'trade or service area' were positive for alcohol and other drugs (34 of 61). From an occupational health and safety viewpoint these are concerning figures.

It appears clear from the data gathered in this study that, in addition to alcohol, other drugs are associated with injury from road trauma and other causes.

Despite the limitations of this study, it is clear that the very large numbers, the multiple comparison groups and the consistently very strong statistical findings from the data make these results and statistics perhaps the most comprehensive currently available.

As a ready reference guide, the key findings are summarised in sub-categories as follows.

The use of alcohol and other drugs is associated with an increased incidence of trauma, more severe trauma, longer hospital stays, higher hospital admission rates and a worse clinical condition on arrival at hospital.

- Patients with severe injuries were more likely to test positive for alcohol and other drugs. Of the patients from Group 1, 42 per cent tested positive, compared with 24.3 per cent from Group 2, and 21.7 per cent from Group 3. This was considered highly (statistically) significant.
- There was a positive correlation between increasing injury severity and testing positive to alcohol and other drugs. This was also highly statistically significant.
- Patients who tested positive were more likely to have longer hospital stays, also resulting in increased health costs.
- Patients who were admitted to hospital, or who died before admission, had higher rates of positivity to alcohol and other drugs (521 of 1075 or 48.5 per cent) than those who were not injured severely enough to require admission (206 of 628, or 32.8 per cent).

The use of drugs other than alcohol is associated with an increased incidence of trauma, a greater number of injuries, more severe injuries and longer hospital stays.

- In Group 1, 28.4 per cent of patients tested positive for drugs other than alcohol, compared with 19.3 per cent in Group 2, and 20.2 per cent in Group 3.
- There was a positive correlation between increasing injury severity, increased hospital stays and testing positive to drugs.

The use of alcohol is associated with an increased incidence of trauma, increased number of injuries, more severe injuries and a worse clinical condition on arrival at hospital.

- More patients in Group 1 tested positive for alcohol (23.4 per cent) than in Group 2 (7.9 per cent) and Group 3 (1.5 per cent). Although the patients with less severe injuries were also less likely to have consumed alcohol compared with the more severely injured, the number who tested positive was still considered high.

- The study also found that trauma patients were more likely to test positive for alcohol than the general driving community: 22.9 per cent of patients in Group 1, and 7.4 per cent in Group 2, tested positive for alcohol. This compared with less than 1 per cent of drivers reported for drink-driving (fixed RBT) over the same period.
- There was also a correlation between the number of injuries recorded and the severity of these injuries and testing positive for alcohol.

The use of cannabis is associated with an increased incidence of trauma, increased number of injuries, increased trauma severity and longer hospital stays.

- A greater number of patients tested positive for THC and/or THC acid in Group 1 (19.8 per cent) compared with Group 2 (8.9 per cent) and Group 3 (9 per cent).
- There was also a positive correlation between the number of injuries recorded and a positive reading to cannabis.
- There was also a positive correlation between increasing injury severity and a positive reading to cannabis.
- There was also a positive correlation between increased length of hospital stay (therefore increased health costs) and those testing positive to cannabis who suffered severe injuries.

Amphetamines are associated with an increased incidence of severe trauma occurrence, but are found less commonly than other drugs.

- The greatest number of patients who tested positive for stimulants was in Group 1 (4.4 per cent).
- Fewer patients tested positive for stimulants in all three groups compared with the results for alcohol, cannabis and benzodiazepines.

Benzodiazepines, antidepressants and opiates were not found in significantly different frequencies in the three groups of patients.

All three groups recorded similar frequencies for these three drug groups, although there were significant differences noted in some subsets – one of these subsets being drivers in motor vehicle crashes and benzodiazepines.

The use of alcohol and other drugs and the incidence of trauma appear to be related to a person's age.

- Almost half of Group 1 (48.3 per cent) was aged 18 to 35 years. More than half of this group (50.2 per cent) tested positive – the highest for any group.
- Only 38.6 per cent of those aged less than 18 years recorded a positive reading in Group 1.
- In the 36 to 50 years age group 42.9 per cent recorded a positive reading in Group 1.
- In the 51 to 74 age group 25.4 per cent recorded a positive reading in Group 1.
- In the over 75 age group 20.2 per cent recorded a positive reading in Group 1.

Trauma patients presenting overnight are more likely to record a positive reading for alcohol and other drugs than those presenting during the day.

- The figures show 63.9 per cent of Group 1 and Group 2 patients presenting between 10pm and 6am recorded positive readings for alcohol and other drugs.
- This compares with only 33.1 per cent of patients in both groups presenting between 6am and 10pm.

Patients of Aboriginal or Torres Strait Islander ethnicity, although only comprising a small proportion of injured patients, are more likely to test positive for alcohol and other drugs.

- More than 90 per cent of all Group 1 patients were Caucasian, with 41.6 per cent testing positive for alcohol and other drugs.
- Around 2 per cent of Group 1 patients claimed Aboriginal or Torres Strait Islander background. Ninety per cent of these tested positive for alcohol and other drugs.
- Higher rates of positive recordings for THC and/or THC acid (54.8 per cent), alcohol (48.4 per cent) and benzodiazepines (35.5 per cent) were found among Aboriginal or Torres Strait Islander patients compared with all other patients.

Motor vehicle crashes are the most common cause of injury, with injured drivers more likely to be positive for alcohol, cannabis, benzodiazepines, amphetamines and/or opiates compared with other injured people.

- Motor vehicle crashes were the most common cause of injury in Group 1 and Group 2 patients (70.2 per cent).
- Of those, 38.5 per cent were positive for alcohol and other drugs, while 27.4 per cent were positive for drugs other than alcohol.
- Alcohol was the most common drug found in car drivers, with 21.6 per cent testing positive. This compared with 17 per cent testing positive for cannabis, 14 per cent for benzodiazepines, 6.5 per cent for amphetamines and 3 per cent for opiates.
- While benzodiazepines were found in 7.9 per cent of Group 1 and Group 2 patients, they were found in 14 per cent of injured drivers.
- While benzodiazepines were found in 14 per cent of injured drivers, they were only found in 3 per cent of injured passengers.

Motor vehicle crash victims have a high incidence of blood alcohol levels above 0.05mg%.

- In the cases of trauma patients from motor vehicle crashes, the majority of car occupants who had any alcohol detected in their system (65.4 per cent) had a blood alcohol level above 0.05mg%.
- More than half (50.4 per cent) had a blood alcohol level of 0.11mg% or greater, 30 per cent had a blood alcohol level of 0.16mg% or higher, and 15.4 per cent had a blood alcohol level of greater than 0.2mg%.
- These figures are all statistically significantly higher than police RBT data, both mobile and fixed.

Drivers with a positive blood alcohol level have a 35 per cent incidence of testing positive for another drug.

- Of all drivers with measurable alcohol in their blood, but who had less than the legal limit for committing a driving offence in South Australia (<0.05mg%), 35 per cent were also positive for another drug.

Motor vehicle crash victims who are positive for alcohol and other drugs are less likely to wear safety belts.

- The results show patients not wearing their safety belts were more likely to return a positive blood alcohol level reading (59 per cent) than those wearing seat belts (44.1 per cent).

Falls and positive tests for alcohol and other drugs.

- The second most common cause of injury in Group 1 and Group 2 was falls (11.5 per cent) and was predominantly in an older population, with 34 per cent positive for alcohol and other drugs.

Victims of violence and positive tests for alcohol and other drugs.

- The third most common cause of injury in Group 1 and Group 2 was assault and interpersonal violence (6.7 per cent).
- Of those, 72.2 per cent were positive for alcohol and other drugs – the highest incidence for a single mechanism of injury group in this study.

Industrial accidents and positive tests for alcohol and other drugs.

- Despite a requirement that workplaces be drug-free, the incidence of positive tests for alcohol and other drugs in patients injured at industrial/construction sites was 24 per cent and for those injured in trade/service areas the incidence of positive tests was 55.7 per cent.

Recommendations

It is recommended that:

1. The extent of trauma which appears related to AODs is such that important savings in financial terms, and in terms of injury, death and suffering, should be achievable if the community were to target this issue effectively. New initiatives should be developed which focus not just on alcohol, but also on other drugs, in order to address this problem effectively.
2. Given the apparent strong relationship between use of AODs and increased injury risk from motor vehicle crashes (MVCs), it would seem reasonable to consider the need for additional legislation to improve road safety. This study has provided an indication of the magnitude of the problem for each drug in an Australian context, and this information may be helpful in any such consideration.
3. While it appears clear that there is an association between MVCs and AODs, this study did not specifically address which initiatives might be effective in reducing injury related to AODs. Roadside drug testing is topical and thus worthy of mention. From other people's data, roadside testing for alcohol appears to have been effective. As roadside drug testing was not in place in South Australia during its course, this study provides no 'direct' data as to whether roadside testing for other drugs would have an impact. Despite this caveat, the very significant results from this study add to the debate on the use of roadside drug testing. They also add to the debate on the development of other strategies. From past experience it seems certain that a multi-faceted approach will be needed to address the issue of drugs and trauma.

Chapter one: Introduction

The primary objective of this study was to determine the prevalence and patterns of use of specific recreational drugs in all patients with injuries requiring assessment by a Trauma Team at the Royal Adelaide Hospital over a one-year period.

The other specific objectives included determining an estimate of the prevalence and patterns of drug use, and severity of injury, in patients presenting to the Emergency Department of the Royal Adelaide Hospital (patients of lesser severity of injury not requiring Trauma Service management) following a motor vehicle accident and other trauma.

It also aimed to examine the correlation between drug use and mechanism, pattern, and severity of injury in patients presenting to the Royal Adelaide Hospital following a motor vehicle accident, and the epidemiological and demographic patterns associated with drug use and trauma.

Determining the prevalence of recreational drug use in patients referred by primary care providers (not hospital inpatients) for unrelated blood tests, as an estimate of the prevalence in the general community, was also an objective.

The final objective was to compare the prevalence of recreational drug use in patients presenting following trauma with the estimate of that in the general community.

This was a prospective observational study of recreational drug use in consecutive patients presenting to the Trauma Service at the Royal Adelaide Hospital. This therefore included all mechanisms of trauma, such as assault – not just motor vehicle crashes. It ran for a period of 12 months, and involved the identification and quantitative analysis of blood samples for the presence of ethanol, opiates, methadone, amphetamines, benzodiazepines, cannabinoids and cocaine.

A medical Project Manager (Caldicott) was employed full-time for six months and half-time for nine months, and a Research Nurse (Pfeiffer) was employed half-time for 15 months to manage the sample and data collection. The results were compared with each patient's prescribed medications, thereby yielding an estimate as to the prevalence of recreational drug use in patients presenting to hospital following a trauma.

The study also examined the demographics and patterns of drug use in this population, as well as the correlation between specific drug use and mechanism, pattern and severity of injury. Quantitative analysis potentially allowed inferences to be drawn on the degree of impairment of the trauma victim.

While the study was planned to begin in March 2003, the final commencement date was 6 August 2003. This was due to a combination of factors, including awaiting legal advice and the relocation of the Royal Adelaide Hospital Emergency Department and Trauma Service.

The only impact of this delay was to also delay the completion of the study. An interim report was produced in March 2004.

A provisional final report was submitted in June 2005. Extensive revisions and rewriting have resulted in this report being completed and submitted in December 2006.

Chapter two: Background

Extensive work has been published on the prevalence and role of alcohol in trauma, particularly motor vehicle-related trauma. These studies have repeatedly identified alcohol as a major factor in trauma-related morbidity and mortality, and have shown the enormous cost that is imposed on health care and the associated loss of productivity.

Although there is a widely-held view that other recreational drug use may have a similar impact on trauma, little prospective data exists on the prevalence and patterns of use in the context of road or other trauma, such as assault, or occupational and sporting injuries. Indeed, data relating to the use of illicit drugs in general is limited and much that is available is based on self-report and personal interview, the limitations of which are clear. To illustrate this point, it is worth noting that the major sources of information on illicit drug use cited by the National Drug and Alcohol Research Centre include the National Drug Strategy Household Survey, and the Illicit Drug Reporting System (which incorporates an Injecting Drug User Survey and a Key Informant Interview process). Other data are derived from police seizures and arrests, and from opioid-related deaths.

The 1998 National Drug Strategy Household Survey (NDSHS) (Fitzsimmons and Cooper-Stanbury 2000) reported that 22.6 per cent of South Australians admitted to using illicit drugs in the previous 12 months. Among South Australian residents older than 13 years of age, 7.3 per cent admitted to having driven a motor vehicle under the influence of illicit drugs in the previous year. This was higher than the reported Australian average of 6.1 per cent. Notably, the sample size for this state was only 861.

The 2001 NDSHS reported that the proportion of South Australians who had admitted to using illicit substances in the past 12 months had fallen to 17.8 per cent. Among Australian residents over 13 years of age, 3.9 per cent admitted to having driven a motor vehicle while under the influence of illicit drugs, while 2.3 per cent had attended work under their influence. The survey also reported that approximately 6 per cent of all Australians suffered an injury (non-self-inflicted) as a result of an alcohol- or other drug-related incident in the 12 months preceding the survey.

The majority of published trials examining recreational drug use and trauma have relied on urine acquisition and drug analysis, with which there are inherent difficulties and limitations. TraumaTox used blood analysis, providing more representative measures of drug levels at the time of the incident. For example, blood quantitative analysis of Delta-9-THC levels provides a more accurate reflection of recent use than urine sampling, which may remain positive for 7 to 10 days following the use of cannabinoids.

In 1995, Sugrue and Seger (1995) conducted a prospective study examining the prevalence and levels of alcohol and other drugs in urine samples of road trauma patients who met the criteria for activation of the Liverpool Hospital's trauma team. Their study examined a total of 164 drivers, 12 pedal cyclists, 55 passengers and 31 pedestrians. Although cannabinoids were detected in 15 per cent of the subjects, cocaine, heroin and amphetamine were found in only one case each. The authors concluded that, based on their study results, 'there is little justification for the routine use of toxicology screens in emergency departments for all trauma patients at the present time'. Limitations of this Australian study included the use of urine sampling and the limited sample size, and we contend that the prevalence and patterns of drug use are likely to have significantly changed since the completion of that study by Sugrue and Seger (1995).

Illustrating the change in patterns of drug use over time, a retrospective study from the University of California (Schermer and Wisner 1999) found a near-doubling of positive methamphetamine rates in trauma patients between 1989 (7.4 per cent) and 1994 (13.4 per cent), compared with a decrease in blood alcohol rates (43 per cent to 35 per cent). There is no published Australian data examining trends over time.

A prospective study of toxicological screening of consecutive road trauma patients presenting to an English university hospital was conducted by Carrigan and Field (2000). Urine analysis was used, and detected the presence of drugs other than alcohol in 51 per cent of patients, with the most frequent being cannabinoids (13 per cent), followed by codeine (11 per cent), morphine (8 per cent) and amphetamine (6 per cent). It is questionable as to whether this data can be directly extrapolated to the Australian situation.

The correlation between specific drug use and the mechanism and pattern of injury was examined in a prospective study from the United States in 1998, in which urine toxicology and blood alcohol screening were performed on 516 patients (Cornwell and Belzberg 1998). The study found that 71 per cent of patients returned positive screens for alcohol and/or drugs. Of this number, 52 per cent tested positively for alcohol and 42 per cent tested positively for drugs, with cocaine and opiates accounting for 91 per cent of the positive drug screens. Correlations between use and pattern and severity of injury, and between use and hospital course and outcome, were also examined, as were patterns of use among specific population demographics. However, the degree of direct relevance of this data to the Australian situation is again unclear.

In one of the few studies assessing occupational fatalities, drug or alcohol use was evident in 19.4 per cent of cases (Fullerton and Olson 1995). There appears to be no other significant Australian data in this area.

To date, most of the work in this area of drugs and traffic accidents in Australia has been undertaken by Drummer (1994, 1995, 1999), who concluded that, while drugs have the potential to adversely affect motor and coordination skills – and are represented in drivers who are assessed by specially trained police officers, or clinical forensic physicians, as being visibly impaired – it has been unclear if this translates to an increased accident risk. Accordingly, he writes that any link between drug use and increased accident risk is equivocal. He has summarised the forensic aspects of drug use in the following way.

‘While a drug may belong to a group that is known to cause impairment, its use by a person or its presence in a bodily fluid does not mean that it caused impairment.’

‘In relation to prescribed medications there are very poor correlations between the dose of most drugs and blood concentrations. Except in extreme cases it is very difficult to predict from blood concentrations a likely dose used and if impairment was likely.’

‘It is inappropriate for blood tests to be performed unless there is a measure of likely impairment at the time that the blood sample is taken.’

This measure of impairment serves two functions:

- It provides the police officer with a reasonable cause for a blood (or urine) sample to be taken.
- It provides a quantitative pharmacological measure of impairment which can be linked to a blood test for an impairing drug.

In 1999, Drummer, Caplehorn and Gerostamoulos conducted a study to determine the presence of drugs in drivers killed on New South Wales roads, and to compare this data to previous years. The purposes of the study were to establish the incidence of the use of alcohol and other drugs by drivers from 1997 to 1998, and to calculate the odds ratio of drug use compared to drug-free drivers. There are clear differences, both in the study population and the outcome measures, between this and the TraumaTox project. The Drummer, Caplehorn and Gerostamoulos (1999) study found that 51.7 per cent of the study population (411 drivers) tested negative to either drugs or alcohol or both. Drugs other than alcohol were detected in 24.1 per cent of all driver fatalities. The most common drug detected was cannabis (12.2 per cent) followed by opioids (7.3 per cent), stimulants (4.9 per cent) benzodiazepines (3.9 per cent) and other psychoactive drugs (2.4 per cent). In addition, the study found that stimulants were detected in 25 per cent of all truck driver fatalities.

Hunter et al. (1998) published an extensive review of the literature on the effects of various drugs on driving performance in 1998. Their study was conducted on behalf of State Forensic Science, South Australian Department of Administrative and Information Services. They reviewed the laboratory studies of the effects of cannabis, stimulants and benzodiazepines on the psychomotor tasks related to driving and driving simulators. As part of their study, they examined the blood samples of people requiring blood alcohol analysis – following non-fatal motor vehicle crashes in 1995/96 – for the presence of alcohol, cannabinoids, benzodiazepines and stimulants. In this group of patients, they found that at least one of these drugs was present in 14.8 per cent of samples – cannabinoids in 10.8 per cent, benzodiazepines in 2.7 per cent, and stimulants in 1.3 per cent. Opioids were not studied. It is also worth noting that blood toxicological analysis was only performed on a specific group of people who had forensic blood alcohol samples taken following vehicle crashes.

In contrast, TraumaTox examined all patients presenting to the Trauma Service, irrespective of the mechanism of injury. It provided observational data on the prevalence and patterns of recreational drug use in this population, as well as determining an estimate of the background prevalence in the general community, against which this may be compared. In addition, it examined the correlation between drug use and the mechanism, pattern and severity of injury, which was not studied in the above report.

Hunter et al. (1998) did not find any research evidence to suggest that, when used on its own, cannabis is associated with increased culpability for crashes. Citing studies conducted by Drummer (1994) and Williams et al. (1985), they noted that there is evidence that cannabis can be associated with lower culpability, although they cautioned that Drummer's (1994) results did not achieve statistical significance. In their own study of 2,500 non-fatally injured drivers in South Australia, Hunter et al. (1998) found no evidence of any increase in the likelihood of crash culpability in those injured drivers in whom cannabis alone was detected.

Swann (1999) argued against these results, on the basis that the methodology used in previous studies to determine culpability associated with cannabis was flawed. In particular, he argued that previous Australian studies had used the presence of 11-nor-delta-9-tetrahydrocannabinol-9-carboxylic acid (THC acid). This is the major metabolite of the impairing constituent in cannabis Delta-9-Tetrahydrocannabinol (Delta-9-THC). THC acid has the ability to remain in the body after the effects of Delta-9-THC have worn off. According to Swann (1999), if THC acid is used to indicate cannabis use and is compared with accident culpability, it is likely to significantly underestimate the actual impact of cannabis consumption on accident culpability.

To overcome this anomaly, Swann (1999) suggested that the presence of Delta-9-THC should be tested for at the time of the crash. He contended that the only way to accurately measure the impact of cannabis was to take samples from the bodies of those killed (where the driver has

died at the time of the crash). To confirm this, he analysed four years of results obtained from studies (involving four years of fatalities in NSW) that identified drivers who tested positive to the impairing part of cannabis (Delta-9-THC). This allowed drivers who were impaired by cannabis to be identified. In essence, the results of Swann's (1999) analysis indicated that there is a high risk of being killed when driving whilst impaired by Delta-9-THC.

He concluded that only fatality studies (where the driver has died at the time of the crash) should be used to estimate the real risk of driving whilst impaired by cannabis. The reasons for this included:

- Upon inhalation, Delta-9-THC is rapidly absorbed from the lungs into the bloodstream – peak blood concentrations are reached within 8 minutes of commencing smoking.
- These blood concentrations decline rapidly, and the time between the crash and when the driver's blood sample is taken in hospital is critical.
- Within approximately 2 hours of the crash, the level of Delta-9-THC has been reduced by approximately 90 per cent.
- Delays of 2 hours between crash and blood being taken in hospital occur regularly.
- Significant losses of Delta-9-THC occur when blood is stored at the normal laboratory storage temperature of -20°C (over 50 per cent at 8 weeks) while only marginal losses are observed when blood is stored at -60°C .

However, it equally could be argued that the correlation between post-mortem blood analyses and blood levels at the time of accident is even less clear. This relationship has not been formally studied, and the pharmacokinetics (including metabolism) of these drugs following death is unclear. Metabolism and redistribution of drugs does not cease as soon as death occurs. In addition, it would be necessary to know how soon after the accident the person died, and there would likely still be a significant delay to blood sampling in this group. In contrast, much more is known about drug pharmacokinetics in the living, potentially allowing more reliable inferences to be made.

The TraumaTox study recorded the times of the incident and of blood sampling. With the acceptable assumption of no further ingestion of drug from incident to sampling, and with some clinical data on the pharmacokinetics of the drugs involved, TraumaTox is in a stronger position than previous studies to comment on drug levels and the potential degree of incapacity at the time of incident. TraumaTox also examined a number of recreational drugs, not just cannabis.

Berghaus, Scheer and Schmidt (1995) reviewed cannabis studies and selected 60 studies with a combined total of 1,344 reported observations to develop a ranking order for THC-related impairment. They reported that 'all performance areas' (e.g. tracking, psychomotor skills, attention, divided attention, visual functions, simulator/driving, reaction time) are affected at 11ng/ml, whereas the driver deaths (reported by Drummer, Caplehorn and Gerostamoulos 1999) occurred at average values of 38ng/ml in 1995/96 and 24ng/ml in 1997/98.

Kruger and Berghaus (1995) note that:

- A plasma concentration of 11ng/ml THC results in an equivalent impairment to that of a blood alcohol level (BAL) of 0.073% – this value of 11ng/ml will be reached approximately 1 hour after smoking a standard cigarette containing 10mg of cannabis.
- It is difficult to decide which substance is more dangerous – cannabis or alcohol – as they cause performance failures in different traffic situations.
- Scientific arguments concerning the real dangers of cannabis will not be easily or quickly resolved.

- The evidence presented on the deaths of drivers who were positive to Delta-9-THC alone, and who were fully responsible for their deaths, indicates that counter-measures for cannabis drivers need to be developed further.
- Relative fatality risks for drivers who have used Delta-9-THC are approximately six times greater than for drug/alcohol-free drivers.
- Although the numbers are small and statistical confidence limits wide, in 24 (4.7 per cent) of the sample of 511 drivers who were killed in road crashes, the only drug these drivers tested positive to was the active constituent of cannabis, Delta-9-THC.

Any interpretation of the results reported by Berghaus, Scheer and Schmidt (1995) and Kruger and Berghaus (1995) need to be treated with caution due to the relatively small sizes of the samples where drivers had only Delta-9-THC in their bodies at the time of the fatal crash. Nevertheless, if these results are corroborated by further research, it would suggest that drivers intoxicated with Delta-9-THC are six times more likely to be involved in crashes than alcohol-free or other drug-free drivers. This compares with alcohol impaired drivers who are 7.5 times more likely to be involved in crashes than alcohol-free or other drug-free drivers.

In his report, *Drugs and Driving in Australia*, prepared for the Working Group on Drugs and Driving, Potter (2000) perhaps best summarises the current situation in Australia in relation to the lack of research evidence on the issue of drugs and their impact on motor vehicle-related trauma. The Working Group argued that policy on drug use and driver impairment must be soundly based on research.

As a result, more data is required on the contribution of drug use to crash involvement and causation. In order to achieve this, Potter (2000) contended that mandatory blood samples should be taken from the following categories of drivers (listed in order of priority) and tested for the presence of the active components of potentially impairing drugs:

- all driver fatalities;
- all drivers involved in a fatality-causing crash;
- all drivers treated in hospital after an injury-causing accident; and
- all drivers involved in an injury-causing accident.

This was supported by the Injury Control and Violence Prevention Committee of the Eastern Association for the Surgery of Trauma (EAST) (Soderstrom 2001), which published a series of recommendations, including:

- All patients admitted for treatment of injuries should be tested for alcohol and drugs. The intent of such testing is to aid clinical management, to identify patients at risk for withdrawal, for anaesthetic/pain management, and to identify patients at risk of an underlying substance use disorder.
- EAST clinicians should assume leadership roles in the identification and institution of treatment for substance use disorders in their patients by:
 - requiring alcohol and drug testing on admission for all trauma patients;
 - using interview screening tests for alcohol/drug abuse;
 - reporting substance use results to their patients;
 - referring patients for formal evaluation and treatment/intervention; and
 - reinforcing treatment recommendations of substance abuse clinicians.

- EAST should promote research designed to:
 - define the epidemiology of alcohol/drugs and trauma;
 - evaluate the impact of alcohol/drugs on clinical management and outcomes;
 - identify patients at risk of substance use disorders.

Despite significant advances in the development of trauma centres and trauma care systems, and the management of patients with traumatic injuries, trauma clinicians have devoted relatively little effort to disrupting one of the major pathways to traumatic injury (and repeated injury) – the abuse of alcohol and other drugs (Gentilello and Rivara 1999).

In a prospective, randomised, controlled trial in a population of patients treated in a Level 1 trauma centre, Gentilello and Rivara (1999) demonstrated significant decreases in drinking at 12-month follow-up in those patients receiving a single in-hospital motivational intervention, as compared with controls. In addition, they had a 47 per cent reduction in injury episodes requiring medical care, and fewer traffic violations, including impaired-driving violations. In a review of alcohol interventions in trauma centres, Gentilello and Donovan (1995) concluded that these centres should become 'major sites for the incorporation and integration of community agencies available for treating patients with alcohol problems, and screening, intervention and referral should be routine'. It would appear appropriate and desirable that this philosophy be expanded to include patients with drug problems.

Potter (2000) argued that drug driving is a multi-faceted problem. He contended that a coordinated approach combining legislation, enforcement, information and education – that is consistent with aims and methods of the National Drug Strategic Framework – appears to be most likely to meet with success. Blood analysis for recreational drugs in the context of trauma may also permit, in the future, the offering of appropriate counselling and support services prior to the patient's discharge, at a time when behavioural change may be more likely.

Chapter three: Study methods

Subjects

Trauma patients presenting to the Royal Adelaide Hospital are triaged to either the Trauma Service (TS) or the Emergency Department (ED), based on a series of pre-determined historical and clinical criteria. This system has been developed to identify patients most likely to have more serious injuries, and for these patients to be referred to the Trauma Service. Patients not fulfilling these predetermined criteria are assessed and managed in the Emergency Department. This previously well-established practice provided the basis for separating the two groups of patients presenting to hospital.

There was data collected for three groups. These were the Trauma Service Group (Group 1), the Emergency Department Group (Group 2) and the Institute of Medical and Veterinary Science Comparison Group (Group 3).

The arms for the study (including criteria) were:

- Group 1:** In order to determine the prevalence and patterns of drug use in patients presenting to the Trauma Service, Group 1 was to include all patients presenting to the Trauma Service over the study period, other than those who fulfilled any of the exclusion criteria*.
- Group 2:** In order to determine the prevalence and patterns of drug use in patients presenting to the Emergency Department (but not requiring assessment by the Trauma Service) following a motor vehicle crash, excess blood taken from a random selection of ED trauma patients over the course of the 12 months was to be analysed. Inclusion of Group 2, therefore, enabled conclusions to be drawn about all patients presenting to the Royal Adelaide Hospital following motor vehicle accidents, not just those presenting to the Trauma Service, thereby reducing potential selection bias.
- Group 3:** In order to provide an estimate of the prevalence of recreational drug use in the community, and to serve as a comparison group, a group of excess blood samples at the Institute of Medical and Veterinary Science (IMVS) was analysed for the presence and levels of the same recreational drugs as Groups 1 and 2. This blood would otherwise have been discarded. The IMVS analyses samples forwarded from multiple collection points around South Australia. Samples taken from hospital inpatients were excluded, so that the vast majority of patients in this group had been referred by primary health care providers in the community for unrelated blood tests. Although limitations (e.g. potential selection bias) still clearly existed in using this sample group as an estimate of community drug practice, it was considered to be the most feasible and representative option. Sample selection was distributed over the 12 months of the study, with the proportion of samples from metropolitan and rural sites reflecting the pattern of trauma site distribution in patients presenting to the Royal Adelaide Hospital over a 12-month period. In addition, there was cohort matching for age and gender, based on Royal Adelaide Hospital Trauma Registry statistics over the same period. Samples were de-identified prior to the analysis. In addition to the results of the drug screen, the date of birth, gender and postcode were recorded on each IMVS Sample Data Collection Sheet.

*Exclusion criteria for the study were: age under 14 years; and refusal to have any blood sampling.

Sample collection

Each enrolment in Groups 1 and 2 was randomly ascribed a specific study number which related to a corresponding Study Pack. The Study Pack contained a pre-numbered fluoride oxalate bottle (identical to that currently used for forensic blood alcohol analysis) and a corresponding pre-numbered RAH Data Collection Form.

Patients presenting to the Trauma Service were assessed and managed in the usual manner. As part of their routine management, all patients had an intravenous cannula placement and blood drawn from that cannula at the time of its insertion. All patients involved in a motor vehicle accident have part of that sample sent for blood alcohol analysis, unless consent is refused (Section 47(i) of the *Road Traffic Act (1961)* of South Australia). If a patient is incapable of informed consent, under South Australian law a sample is taken and analysed. An additional 5ml of blood was drawn from enrolled patients at the time of their other routine blood tests, and was placed in the designated fluoride oxalate bottle. The bottle was placed in a locked box until cleared by either the Project Manager or the Research Nurse to be forwarded to the forensic laboratory. Access to the box was available only to the Project Manager and the Research Nurse. The sample bottle was labelled only with the date of collection and the study number sticker.

Results of the blood tests were forwarded by mail from the laboratory to one of the RAH Study Investigators other than the Project Manager. The results were stored in a locked cabinet with access restricted to that investigator only. Once all other data had been collected and entered into the database, and the patient identifier removed from the RAH Data Collection Form and destroyed (see 'Data Collection' below), the blood results were entered into the database.

In this way anonymity and confidentiality were maintained.

Analysis was carried out under contract by the Toxicology Group, Forensic Science SA, Department for Administrative and Information Services.

Analytical methods used in the analysis of blood samples

Ethanol was quantified in blood using gas chromatography with flame ionisation detection.

The blood samples were screened by enzyme-linked immunosorbent assay (ELISA) for the following compounds:

- opiates (including morphine, codeine and dihydrocodeine);
- methadone;
- amphetamines (including amphetamine, methylamphetamine and 3,4-methylenedioxymethylamphetamine (MDMA));
- benzodiazepines (including alprazolam, bromazepam, clonazepam, diazepam, flunitrazepam, lorazepam, nitrazepam, nordiazepam, oxazepam, temazepam and triazolam);
- cannabinoids (including tetrahydrocannabinol (THC) and carboxy-tetrahydrocannabinol); and
- cocaine (including cocaine and benzoylecgonine).

Samples with positive ELISA screening results were then confirmed and quantified by the following methods.

Amphetamines/ketamine:

Extracted using liquid/liquid extraction and analysed by gas chromatography with nitrogen phosphorus detection (including amphetamine, chlorphentermine, diethylpropion, dimethylamphetamine (DMA), 2,5-dimethoxy-4-methylamphetamine (DOM), ephedrine, fenfluramine, mephentermine, methylamphetamine, 3,4-methylenedioxyamphetamine (MDA), 3,4-methylenedioxymethylamphetamine (MDMA), methylphenidate, paramethoxyamphetamine (PMA), pseudoephedrine, phentermine).

Limit of detection: amphetamine, methylamphetamine, MDMA (0.1mg/L), ketamine (0.1mg/L).

Benzodiazepines:

Extracted using liquid/liquid extraction and analysed by gas chromatography with electron capture detection and liquid chromatography/mass spectrometry (including alprazolam, bromazepam, clobazam, clonazepam/7-aminoclonazepam, diazepam/nordiazepam, flunitrazepam/7-aminoflunitrazepam, lorazepam, midazolam, nitrazepam, oxazepam, temazepam and triazolam).

Limit of detection: alprazolam (0.01mg/L), clonazepam/7-aminoclonazepam (0.005mg/L), diazepam/nordiazepam (0.02mg/L), flunitrazepam (0.002mg/L), midazolam (0.02mg/L), nitrazepam (0.002mg/L), oxazepam (0.1mg/L), temazepam (0.1mg/L).

Cannabinoids:

Extracted using solid phase extraction and analysed by gas chromatography/mass spectrometry (including THC, 11-nor-9-carboxy-THC).

Limit of detection: THC (1ng/mL), 11-nor-9-carboxy-THC (5ng/mL).

Opiates:

Extracted using solid phase extraction and analysed by liquid chromatography/mass spectrometry (including morphine, codeine and monoacetylmorphine).

Limit of detection: morphine, codeine, monoacetylmorphine (0.01mg/L).

Methadone:

Extracted using liquid/liquid extraction and analysed by gas chromatography with nitrogen phosphorus detection.

Limit of detection: methadone (0.03mg/L).

Cocaine:

Extracted using liquid/liquid extraction and analysed by liquid chromatography/mass spectrometry (including cocaine and benzoylecgonine).

Limit of detection: cocaine and benzoylecgonine (0.01mg/L).

Data collection

A Project Manager and a Research Nurse were employed to collect and manage data using funds from the study grant.

In addition to demographic details, past medical history was recorded as per the International Classification of Diseases Revision 10 (ICD-10) category, as well as nature of trauma and injuries sustained, also as per ICD-10. Within two weeks of discharge from hospital the Research Nurse reviewed the patient's case notes and collected data on:

- New Injury Severity Score (NISS), Injury Severity Score (ISS), Revised Trauma Score (RTS);
- surgery required;
- length of hospital stay; and
- demographic details not already recorded.

Data was entered into an Access Database program by the Research Nurse or Project Manager, excluding the potential patient identifier of the hospital Unit Record Number. The RAH Data Collection Form then had the Unit Record Number removed and destroyed. At this stage, identification was only by the study number. Following this, the blood results were independently added to the database, thus preserving patient de-identification.

The following strategies to maintain accuracy and minimise inconsistencies in the data collection were implemented.

Data collectors received a period of instruction and training in completion of the RAH Data Collection Form and in data abstraction from the Case Records prior to commencement of the study. Regular meetings were held between the Principle Investigators, Research Nurse and the participating Trauma Registrars to review coding rules and interpretations and to monitor the chart/data abstracters.

The chart/data abstracters were blinded to any interim results of the blood tests.

In order to provide an estimate of the background use of recreational drugs in the broader community, approximately 400 excess blood samples at the Institute of Medical and Veterinary Science (IMVS) were analysed for the presence and levels of the same drugs. This blood would otherwise have been discarded. Specimens obtained from hospital inpatients were excluded. Sample selection was distributed over the 12 months of the study, with the proportion of samples from metropolitan and rural sites reflecting the pattern of trauma site distribution in patients presenting to the Royal Adelaide Hospital over a 12-month period. In addition, there was cohort matching for age, gender and postcode based on Royal Adelaide Hospital Trauma Registry statistics over the same period.

The specimens were labelled with an identifying barcode, and were stored at the IMVS until analysis. The same barcode was recorded on the IMVS Sample Data Collection Form, together with the age, gender and postcode of address of the provider, but without any potential patient identifiers (such as the name). The Data Collection Forms were sent to the Research Nurse, who entered the demographic details into a separate database. The selected blood sample results (identified only by the barcode) were forwarded to the Research Nurse for entry into this database.

Statistical methods

Descriptive statistics were produced for all data collected. If a patient admitted using at least one recreational drug they were classified as admitting usage. If there was a positive result for more than one recreational drug from a class (for example benzodiazepines) for a patient, then the maximum value per patient was used in all statistical analyses. Drug positivity prevalences were reported with exact 95 per cent confidence intervals. Comparison of prevalences between Groups 1 and 2 was done using the Pearson chi-square test, and differences in prevalences and their asymptotic 95 per cent confidence intervals were reported. The Pearson chi-square test was also used for comparisons between other groups such as drivers and non-drivers etc. Spearman correlation coefficients were used to examine the relationship between drug level and injury severity, since the data were not normally distributed. The accuracy of the physician suspicion of drug use was assessed using the kappa statistic to measure beyond chance agreement. Pearson chi-square tests were used to examine associations between drug positivity and patient characteristics. A probability value of <0.05 was considered statistically significant.

Statistical analysis was carried out under contract by University of South Australia bio-statisticians, and also in part by the Principal Investigator.

Ethics approval

Ethics approval for this study was received from the Royal Adelaide Hospital Ethics Committee prior to commencement.

Governance

A Steering Committee was established to oversee the study. This committee had expert representation and met regularly to consider the study's progress. Financial statements and other interim reports were produced on a regular basis and reviewed by the Steering Committee. A formal interim report was submitted to NDLERF. This final report was also reviewed by the Steering Committee prior to its submission.

Chapter four: Results

Recruitment

A total of 2,127 patients, samples were analysed in the period from 6th August 2003 to 6th August 2004. Of these, 1,515 were from Group 1, 202 were from Group 2 and 410 were from Group 3. The total Trauma Service attendance for the same period was 1,717 patients, giving a recruitment rate of 80.7 per cent (Table 1a).

Table 1a. Total numbers recruited to TraumaTox study.

	Negative Toxicology	Positive Toxicology (% of total, each arm)	Total
Group1	879	636 (42%)	1,515
Group 2	153	49 (24.3%)	202
Total trauma patients	1,032	685 (39.9%)	1,717
Group 3	321	89 (21.7%)	410
Total	1,353	774 (36.4%)	2,127

These figures make this the largest ever study of this type in Australia. The use of two comparison groups allowed comparisons not possible in previous studies.

Mobile and fixed random breath testing (RBT) data for essentially the same time period were also collected. Mobile RBT data for August 2003 and February 2004 were not available. These data provide yet another source of comparison (Table 1b).

Table 1b. Mobile and fixed RBT data.

Mobile RBT				
Year	Month	Testing	Positives	Detection Rate
2003	August*	-	-	-
	September	2,766	37	1.3%
	October	3,456	153	4.4%
	November	1,849	62	3.4%
	December	7,744	277	3.6%
2004	January	7,377	270	3.7%
	February †	-	-	-
	March	1,040	38	3.7%
	April	8,843	209	2.4%
	May	2,807	74	2.6%
	June	2,463	66	2.7%
	July	7,551	290	3.8%
Total		45,896	1,476	3.2%

Fixed RBT				
Year	Month	Testing	Positives	Detection Rate
2003	August	35,869	144	0.4%
	September	38,444	189	0.5%
	October	67,903	575	0.8%
	November	49,113	642	1.3%
	December	78,297	613	0.8%
2004	January	55,136	457	0.8%
	February	42,175	550	1.3%
	March	51,111	520	1.0%
	April	73,851	400	0.5%
	May	34,216	465	1.4%
	June	29,417	351	1.2%
	July	29,792	410	1.4%
Total		585,324	5,316	0.9%

* Prior to start of mobile RBT

† No mobile RBT periods

Demographics

Prevalence (see Tables 1c–d)

There were greater numbers of patients who tested positive for AODs in Group 1 (42 per cent) compared with Group 2 (24.3 per cent) and Group 3 (21.7 per cent). This was highly statistically significant (P-value <0.001 for Group 1 versus Group 2 and Group 1 versus Group 3).

28.4 per cent of patients in Group 1 tested positive for drugs other than alcohol (DOTA), compared with 19.3 per cent in Group 2 and 20.2 per cent in Group 3. This was highly statistically significant (P-value <0.001).

There were greater numbers of patients who tested positive for alcohol in Group 1 (23.4 per cent) compared with both Group 2 (7.9 per cent) and Group 3 (1.5 per cent). This was highly statistically significant (P-value <0.0001 for Group 1 versus Group 3).

There were greater numbers of patients who tested positive for cannabis and/or THC (in any combination) in Group 1 (19.8 per cent) compared with both Group 2 (8.9 per cent) and Group 3 (9 per cent). All of these were highly statistically significant (P-values <0.0001).

There were greater numbers of patients who tested positive for amphetamines in Group 1 (4.4 per cent) compared with Group 3 (0 per cent). This was highly statistically significant (P-value <0.0001).

There were greater numbers of patients who tested positive for opiates in Group 3 (5.4 per cent) compared with Group 1 (2.7 per cent). This was statistically significant (P-value 0.01). This may be explained in part by the possibility that some of Group 3 were receiving opiates as prescription agents.

Table 1c. Recreational drug prevalence (per cent with 95% confidence intervals (CI)) by study group.

	Group 1	Group 2	Group 3
Alcohol (BAL>0.05)	22.9 (20.8, 25.1) ‡	7.4 (4.2, 12.0) †	1.5 (0.5, 3.2)
Any drug	42.0 (39.5, 44.5) ‡	24.3 (18.5, 30.8)	21.7 (17.8, 26.0)
Alcohol	23.4 (21.3, 25.6) ‡	7.9 (4.6, 12.5) †	1.5 (0.5, 3.2)
DOTA	28.4 (26.2, 30.8) ‡	19.3 (14.1, 25.4)	20.2 (16.5, 24.5)
THC/THC acid	19.8 (17.8, 21.9) ‡	8.9 (5.4, 13.7)	9.5 (6.9, 12.8)
THC acid	19.7 (17.7, 21.8) ‡	8.9 (5.4, 13.7)	9.0 (6.4, 12.2)
THC	16.1 (14.3, 18.1) ‡	6.4 (3.5, 10.8)	6.3 (4.2, 9.2)
Benzodiazepines	7.7 (6.4, 9.2)	9.4 (5.8, 14.3)	7.6 (5.2, 10.6)
Amphetamines	4.4 (3.4, 5.5) †	2.5 (0.8, 5.7)	0.0 (0.0, 0.9)
Opiates	2.7 (1.9, 3.7)	5.9 (3.1, 10.1)	5.4 (3.4, 8.0)*
Antidepressants	0.2 (0.0, 0.6)	0.0 (0.0, 1.8)	0.7 (0.2, 2.1)

† Statistically significant compared with Group 3

‡ Statistically significant compared with both Group 3 and Group 2

* Statistically significant compared with Group 1

Table 1d. Percentage difference between drug prevalence in Group 1 and Group 3 with 95% CI and P-value for statistical significance.

	Difference (Group 1–Group 3)	Lower 95% Limit	Upper 95% Limit	P-value
Any drug*	20.3	15.6	25.0	<0.0001
Alcohol*	22.0	19.5	24.4	<0.0001
Any drug not alcohol*	8.2	3.7	12.7	0.0009
THC/THC acid*	10.3	6.8	13.8	<0.0001
THC acid*	10.6	7.2	14.1	<0.0001
THC*	9.8	6.8	12.8	<0.0001
Benzodiazepines	0.2	-2.7	3.1	0.91
Stimulants*	4.4	3.3	5.4	<0.0001
Opiates**	-2.7	-5.0	-0.3	0.01
Antidepressants	-0.5	-1.4	0.3	0.09

* Items in bold are statistically significant with Group 1 occurrence being greater than Group 3

** Opiates are statistically significant in the reverse direction (i.e. more in Group 3)

Gender (see Tables 2a–e)

Within Group 1, 71.5 per cent (1,083 of 1,515) of patients were male and 28.5 per cent (432 of 1,515) were female. Within this group, 46.1 per cent (499 of 1,083) of males recorded positive results for alcohol or other drugs (AODs), and 31.7 per cent (137 of 432) of females recorded positive results for AODs. Males were more likely to test positive. This was highly statistically significant (P-value <0.0001).

Within Group 2, 56.4 per cent (114 of 202) of patients were male and 43.6 per cent (88 of 202) were female. Only 28.9 per cent (33 of 114) of males recorded positive results for AODs, and 18.2 per cent (16 of 88) of females recorded positive results for AODs. This was not statistically significant (P-value 0.07).

Within Group 3, 73.2 per cent (300 of 410) of patients were male, and 26.8 per cent (110 of 410) were female. 21.3 per cent (64 of 300) of males recorded positive results for AODs, and 24.5 per cent (27 of 110) of females recorded positive results for AODs. This was not statistically significant (P-value 0.44).

From these results it can be seen that in both groups of patients presenting to the hospital there is a male predominance in patients positive for AODs.

Adjusting for numbers of presentations, both alcohol and cannabis have higher incidences in male populations. Benzodiazepine use is more common in the female patients.

Within Group 3, the incidence of use by women was greater than the incidence of use by men in four out of the six classes of drugs detected. Men showed nearly twice the rate of cannabis positivity of women (10.3 per cent versus 5.4 per cent), and all methadone detected was from men. However, in all other categories, including alcohol, women predominated when adjusted for presenting numbers.

Table 2a. Positive toxicology results in Group 1, by gender.

Gender – Group1	Negative Toxicology	Positive Toxicology (% positive)	Total
Male	584	499 (46.1%)	1,083 (71.5%)
Female	295	137 (31.7%)	432 (28.5%)
Total	879	636 (42%)	1,515

Table 2b. Positive toxicology results in Group 2, by gender.

Gender – Group2	Negative Toxicology	Positive Toxicology (% positive)	Total
Male	81	33 (28.9%)	114 (56.4%)
Female	72	16 (18.2%)	88 (43.6%)
Total	153	49 (24.3%)	202

Table 2c. Positive toxicology results in Group 3, by gender.

Gender – Group3	Negative Toxicology	Positive Toxicology (% positive)	Total
Male	236	64 (21.3%)	300 (73.2%)
Female	83	27 (24.5%)	110 (26.8%)
Total	319	91 (22.2 %)	410

Table 2d. Drug positivity by gender (Groups 1 and 2).

	ETOH	THC	Benzo	Amphet	Opioids	Methadone	Cocaine	Heroin	Other
Male	296	267	117	67	32	7	2	2	5
Female	75	49	63	27	16	5	1	0	2

Table 2e. Drug positivity by gender (Group 3).

	ETOH	THC	Benzo	Amphet	Opioids	Methadone	Cocaine	Heroin	Other
Male	3	31	24	0	16	2	0	0	4
Female	5	6	14	0	6	0	0	0	2

Age (see Tables 3a–e)

Nearly half of Group 1 (48.3 per cent, 731 of 1,515) were in the 18–35 year age bracket. More than half of this group (50.2 per cent, 367 of 731) tested positive for AODs – the highest percentage for any age group.

Using arbitrary age ranges (see Tables 3a–e), in this group there was a difference in positivity to AODs between the different age ranges.

The less than 18 year-old age group has a lower rate (38.6 per cent, 39 of 101) of positivity to AODs than the 18–35 age group (50.2 per cent, 367 of 731). This is statistically significant (P-value <0.05).

The 18–35 age group has a higher rate (50.2 per cent, 367 of 731) than the 36–50 age group (42.8 per cent, 148 of 345). This is statistically significant (P-value <0.05).

The 36–50 age group has a much higher rate than the 50–74 age group (25.4 per cent, 63 of 248). This is highly statistically significant (P-value <0.0001).

The 51–74 age group rate (25.7 per cent, 64 of 249) is not statistically different to the over 75 years group (20.2 per cent, 18 of 89).

Cannabis was found more frequently than alcohol in the 18 years or under age group. Positivity to alcohol remained fairly consistent across all age ranges.

Approximately identical rates of cannabis and alcohol presence were found in patients in the 18–35 age group.

Benzodiazepines were the most evenly spread drugs across the age groups.

The age distribution in the lower acuity Group 2 was similar with 50.5 per cent (102 of 202) within the 18–35 year old age bracket. The Group 1 pattern of AOD positivity was not reflected in Group 2 where AOD positivity marginally climbed with age.

The background prevalence of AOD positivity in Group 3 demonstrated a similar distribution to Group 2 with the exception of alcohol, which was significantly higher in Group 2 compared with Group 3.

Alcohol positivity remained fairly consistent across the age brackets.

The 18–35 year age group accounted for more than 80 per cent of all amphetamine positive results.

Within Group 3 such sharp variations with respect to age were not as prevalent. For cannabis, positivity for cannabis was similar in the 36–50 age bracket (12.8 per cent, 12 of 94) compared with the 18–35 age bracket (10.2 per cent, 19 of 186). Benzodiazepines were found in 16 per cent (15 of 94) of the 51–74 age bracket and in 24 per cent (6 of 25) of the 75+ age bracket.

Table 3a. Positive toxicology results in Group 1, by age.

Age – Group1	Negative Toxicology	Positive Toxicology	Total
Less than 18	62	39 (38.6%)	101
18–35	364	367 (50.2%)	731
36–50	197	148 (42.9%)	345
51–74	185	64 (25.7%)	249
75+	71	18 (20.2%)	89
Total	879	636 (41.9%)	1,515

Table 3b. Positive toxicology results in Group 2, by age.

Age – Group2	Negative Toxicology	Positive Toxicology	Total
Less than 18	9	2 (18.2%)	11
18–35	82	22 (21.2%)	104
36–50	33	13 (28.3%)	46
51–74	22	9 (29%)	31
75+	7	3 (30%)	10
Total	153	49 (24.3%)	202

Table 3c. Positive toxicology results in Group 3, by age.

Age – Group3	Negative Toxicology	Positive Toxicology	Total
Less than 18	9	2 (18.2%)	11
18–35	145	41 (22%)	186
36–50	76	18 (19.1%)	94
51–74	73	21 (22.3%)	94
75+	18	7 (28%)	25
Total	321	89 (21.7%)	410

Table 3d. Drug use by age (Groups 1 and 2).

Age	ETOH	THC	Benzo	Amphet	Opioids	Methadone	Cocaine	Heroin	Other
<18	17	28	1	1	0	0	0	0	0
18–35	212	213	78	79	23	7	2	2	1
36–50	91	69	57	14	14	5	1	0	3
51–74	46	6	31	0	8	0	0	0	1
75+	5	0	13	0	3	0	0	0	2
Total	371	316	180	94	48	12	3	2	7

Table 3e. Drug use by age (Group 3).

Age	ETOH	THC	Benzo	Amphet	Opioids	Methadone	Cocaine	Heroin	Other
<18	0	1	0	0	1	0	0	0	0
18–35	2	19	10	0	12	2	0	0	3
36–50	1	12	7	0	5	0	0	0	1
51–74	2	5	15	0	3	0	0	0	2
75+	1	0	6	0	1	0	0	0	0
Total	6	37	38	0	22	2	0	0	6

Ethnicity (see Tables 4a–c, 5)

It is acknowledged that reported ethnicity may not be accurate and that many people are of mixed ethnicity and could reasonably claim that they fit into more than one group. However, for this study we accepted the claims of the participants at face value, recognising these limitations.

The overwhelming majority of attendances to Group 1 or Group 2 were by persons classifying themselves as Caucasian (93.2%, 1,412 of 1,515, and 94.6 per cent, 191 of 202, respectively). The rate of positivity to AODs for Caucasians in Group 1 was 41.6 per cent (587 of 1,412), with Caucasians responsible for 93.4 per cent of all positive results.

Although patients reporting Aboriginal and Torres Strait Islander (ATSI) origin represented a small number of the patients in Group 1, (2 per cent, 31 of 1,515), a large percentage (90.3 per cent, 28 of 31) of these were positive for AODs (see Table 5).

For alcohol, cannabinoids and benzodiazepines, ATSI patients had at least twice the incidence of AODs positive samples as other ethnic groups; in the case of benzodiazepines and cannabinoids, more than three times the incidence.

Table 4a. Positive toxicology results by reported ethnicity.

	Negative Toxicology	Positive Toxicology (% positive in group)	Total (% population)
Caucasian	971	632 (39.4%)	1,603 (93.4%)
ATSI	3	28 (90.3%)	31 (1.8%)
Asian	27	14 (34.1%)	41 (2.4%)
African	13	1 (7.1%)	14 (0.8%)
Other	18	9 (33.3%)	27 (1.6%)
Unknown	0	1 (100%)	1 (0.06%)
Total	1,032	685 (39.9%)	1,717 (100%)

Table 4b. Positive toxicology results in Group 1, by reported ethnicity.

Ethnicity–Group1	Negative Toxicology	Positive Toxicology (% positive in group)	Total (% population)
Caucasian	825	587 (41.6%)	1,412 (93.2%)
ATSI	3	28 (90.3%)	31 (2%)
Asian	21	14 (40%)	35 (2.3%)
African	13	1 (7.1%)	14 (0.9%)
Other/ unknown	17	6 (26.1%)	23 (1.5%)
Total	879	636 (42%)	1,515 (100%)

Table 4c. Positive toxicology results in Group 2, by reported ethnicity.

Ethnicity–Group2	Negative Toxicology	Positive Toxicology (% positive in group)	Total (% population)
Caucasian	146	45 (23.6%)	191 (94.6%)
ATSI	0	0	0
Asian	6	0	6 (3%)
African	0	0	0
Other/ unknown	1	4 (80%)	5 (2.5%)
Total	153	49 (24.3%)	202

Table 5. Subdivision of types of drugs used by reported ethnicity for Groups 1 and 2.

	ETOH (% of people in this ethnic group)	THC (% of people in this ethnic group)	Benzo (% of people in this ethnic group)	Amphet (% of people in this ethnic group)	Opioid (% of people in this ethnic group)	Number of people positive* (% of people in this ethnic group)	Total of people in each ethnic group
Caucasian	341 (21.3%)	291 (18.2%)	174 (10.9%)	88 (5.5%)	47 (2.9%)	632 (39.4%)	1,603
ATSI	15 (48.4%)	17 (54.8%)	11 (35.5%)	1 (3.2%)	0	28 (90.3%)	31
Asian	8 (19.5%)	6 (14.6%)	3 (7.3%)	2 (4.9%)	0	14 (34.1%)	41
African	0	1 (7.1%)	0	0	0	1 (7.1%)	14
Other/ unknown	7 (25%)	1 (3.6%)	3 (10.7%)	3 (10.7%)	1 (3.6%)	10 (35.7%)	28
Total	371 (21.6%)	316 (18.4%)	191 (11.1%)	94 (5.5%)	48 (2.8%)	685 (40%)	1,717

* may be positive to more than one test

Mechanisms of injury

Nature of incident (see Table 6)

Motor vehicle crashes (MVCs) are clearly the leading cause of presentation to the hospital following trauma. In Group 1, two-thirds of hospital presentations were due to motor vehicle crashes (66.2 per cent, 1,004 of 1,515). Within this group, 41.2 per cent were positive for AODs (414 of 1,004). If Groups 1 and 2 are combined, 70.2 per cent of presentations in this study were due to MVCs (1,206 of 1,717). Of the 1,206 MVC patients, 463 (38.4 per cent) were positive for AODs.

The next most common cause for hospital presentation was falls (11.5 per cent, 197 of 1,717), with 34 per cent (67 of 197) of patients being positive for AODs.

Although assault/interpersonal violence was only the third most common cause for presentation (6.7 per cent, 115 of 1,717), a very high 72.2 per cent (83 of 115) of those presenting following assault were positive for AODs. This was highly statistically significant when compared with MVCs (P-value <0.0001).

Of the 115 assault patients, 61 were positive for DOTA, the highest incidence for a single mechanism group in this study. This was also highly statistically significant when compared with MVCs (P-value <0.0001).

Table 6. Nature of injury-causing incident (Groups 1 and 2).

	Negative Toxicology	Positive Toxicology (% positive)	Total (% of total)
MVC	743	463 (38.4%)	1206 (70.2%)
Falls	130	67 (34%)	197 (11.5%)
Assault	32	83 (72.2%)	115 (6.7%)
Contact with inanimate objects	46	20 (30.3%)	66 (3.8%)
Self harm	20	19 (48.7%)	39 (2.3%)
Exposure to radiation, smoke, fire, flames, heat	13	15 (53.6%)	28 (1.6%)
Animal rider	19	4 (17.4%)	23 (1.3%)
Contact with animate objects	16	4 (20%)	20 (1.2%)
Water accident	3	5 (62.5%)	8 (0.5%)
Other transport	4	3 (42.9%)	7 (0.4%)
Accidental poisoning	4	0	4 (0.23%)
Legal intervention	1	2 (66.7%)	3 (0.17%)
Forces of nature	1	0	1 (0.06%)
Total	1,032	685	1,717

Place of incident (see Table 7)

Consistent with the high incidence of MVCs as a cause for hospital presentation is the finding that many patients arriving in the Trauma Service are from the street or highway. Along with those arriving from home, these groups make up over 80% of patients (81.7 per cent, 1,403 of 1,717).

High levels of positivity for AODs are seen in those patients arriving from trade and service areas (which include bars and nightclubs but also work areas) (55.7 per cent, 34 of 61). Presentations from street/highway, of course, may also be the result of incidents that began indoors.

The lowest levels, similar to those found in Group 3, were found in patients arriving from either industrial/construction sites (24.2 per cent, 15 of 62) or farms (18.9 per cent, 7 of 37).

Despite the relatively lower levels, the incidence of positivity to AODs from industrial sites is still alarmingly high (24.2 per cent, 15 of 62) considering the potentially dangerous nature of these workplaces and the presumed workplace requirement for zero levels of AODs.

Table 7. Place of injury-causing incident (Groups 1 and 2).

Place	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Street/highway	723	480 (39.9%)	1,203 (70%)
Home	116	84 (42%)	200 (11.6%)
Recreational/sports venue	44	24 (35.3%)	68 (3.9%)
Industrial/construction site	47	15 (24.2%)	62 (3.6%)
Trade/service area	27	34 (55.7%)	61 (3.6%)
Other/unspecified	23	29 (55.8%)	52 (3.0%)
Farm	30	7 (18.9%)	37 (2.2%)
Residential institution	11	5 (31.3%)	16 (0.9%)
School/public building	8	6 (42.9%)	14 (0.8%)
Hospital	1	1 (50%)	2 (0.1%)
Mine/quarry	2	0	2 (0.1%)
Total	1,032	685	1,717

Hospital data

Severity of injuries (see Tables 8a–c)

There are a large number of scoring systems used to assess severity of injury. One of the best-recognised and most widely used is the Injury Severity Score (ISS). To create an ISS the following process is applied. Each separate injury is coded and assigned a body region, with scores ranging from 0–6 (6 being non-survivable). These scores are called the Abbreviated Injury Scale (AIS). The worst AIS scores for three different body regions are then squared and added together, giving a range from 0 to 75 for the ISS (note: any one score of 6 immediately equates to an ISS of 75).

Using an arbitrary scale within this 0–75 range we classified injuries as no injury (0), minor (1–8), moderate (9–15), serious (16–24), severe (25–49), critical (50–74) and maximum (75).

In Group 1, using ISS, more than two-thirds of the patients fell into the ‘minor injury’ category. As severity of injury increased up to the level of ‘serious injury’, the rate of positivity for AODs increased to a maximum of 51.9 per cent (54 of 104).

New Injury Severity Score (NISS) is a variant on ISS where the three worst injuries may all be scored from one body region.

The Group 2 data confirms that – at least in the case of motor vehicle crashes – lower acuity patients were seen in this group. Interestingly, the rates of positivity for AODs for those of equivalent injury severity scores were considerably less in Group 2 than in Group 1. A number of factors may be involved here, including skewing of Group 1 by the high levels of positives from assaults. It is also possible that the presence of AODs affected the patients’ clinical condition on presentation to make them appear even more unwell, thus causing them to be more likely to be triaged to Group 1.

Table 8a. Injury Severity Score (Groups 1 and 2).

Injury Severity Score (ISS)	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
0	32	5 (13.5%)	37 (2.2%)
1–8	715	436 (37.9%)	1,151 (67%)
9–15	182	151 (45.3%)	333 (19.4%)
16–24	51	54 (51.4%)	105 (6.1%)
25–49	43	37 (46.3%)	80 (4.7%)
50–74	7	1 (12.5%)	8 (0.5%)
75	2	1 (33.3%)	3 (0.2%)
Total	1,032	685 (39.9%)	1,717 (100%)

Table 8b. Injury Severity Score (Group 1).

Injury Severity Score (ISS)	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
0	14	3 (17.6%)	17 (1.1%)
1–8	583	390 (40.1%)	973 (64.2%)
9–15	180	150 (45.5%)	330 (21.8%)
16–24	50	54 (51.9%)	104 (6.9%)
25–49	43	37 (46.3%)	80 (5.3%)
50–74	7	1 (12.5%)	8 (0.5%)
75	2	1 (33.3%)	3 (0.2%)
Total	879	636 (42%)	1,515

Table 8c. Injury Severity Score (Group 2).

Injury Severity Score (ISS)	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
0	18	2 (10%)	20 (9.9%)
1–8	132	46 (25.8%)	178 (88.1%)
9–15	2	1 (33.3%)	3 (1.5%)
16–24	1	0	1 (0.5%)
25–49	0	0	0
50–74	0	0	0
75	0	0	0
Total	153	49 (24.3%)	202

Drug use according to severity of injury (see Table 9)

There was a positive correlation between increasing injury severity (as measured by ISS and/or NISS) and positivity to AODs. This was highly statistically significant (P-values <0.0001).

There was a positive correlation between the number of injuries recorded and positivity to AODs. This was highly statistically significant (P-value 0.0001).

There was a positive correlation between increasing injury severity (as measured by ISS and/or NISS) and positivity to DOTA. This was highly statistically significant (P-values <0.001).

There was a positive correlation between the number of injuries recorded and positivity to DOTA. This was statistically significant (P-value <0.02).

There was a positive correlation between increasing injury severity (as measured by ISS and/or NISS) and positivity to alcohol. This was statistically significant (P-values <0.05).

There was a positive correlation between the number of injuries recorded and positivity to alcohol. This was statistically significant (P-value <0.05).

There was a positive correlation between increasing injury severity (as measured by ISS and/or NISS) and positivity to cannabis. This was highly statistically significant (P-values <0.0001).

There was a positive correlation between the number of injuries recorded and positivity to cannabis. This was highly statistically significant (P-value <0.0001).

Thus alcohol, cannabis, AODs and DOTA were all found to be independently related to a number of trauma indicators.

Table 9. Correlations between drug levels and injury severity.

	Spearman Correlation Coefficients Prob > r under H0: Rho=0 (P-value) Number of Observations					
	NISS	ISS	RTS	Length of stay	Number of injuries	Number of complications
Alcohol	0.05761	0.06473	-0.10616	0.03319	0.05156	-0.01109
BAL > 0.05%	0.0171	0.0074	<0.0001	0.1699	0.0329	0.6465
	1,717	1,717	1,717	1,717	1,717	1,717
AODs	0.11052	0.11231	-0.09902	0.07653	0.09199	0.03519
	<0.0001	<0.0001	<0.0001	0.0015	0.0001	0.1450
	1,717	1,717	1,717	1,717	1,717	1,717
Alcohol	0.05299	0.05873	-0.09281	0.03048	0.05276	-0.00844
	0.0281	0.0149	0.0001	0.2068	0.0288	0.7268
	1,717	1,717	1,717	1,717	1,717	1,717
DOTA	0.08505	0.08086	-0.03695	0.06419	0.05648	0.05839
	0.0004	0.0008	0.1259	0.0078	0.0193	0.0155
	1,717	1,717	1,717	1,717	1,717	1,717
THC acid	0.11448	0.11070	-0.02858	0.06788	0.11667	0.00806
	<0.0001	<0.0001	0.2365	0.0049	<0.0001	0.7385
	1,717	1,717	1,717	1,717	1,717	1,717
THC	0.10870	0.10927	-0.03304	0.07634	0.10546	0.00425
	<0.0001	<0.0001	0.1711	0.0015	<0.0001	0.8602
	1,717	1,717	1,717	1,717	1,717	1,717
Benzodiazepine	0.01779	0.01455	-0.02912	-0.00639	-0.03779	0.03516
	0.4614	0.5469	0.2277	0.7914	0.1175	0.1453
	1,717	1,717	1,717	1,717	1,717	1,717
Stimulant	0.01291	0.00457	0.03775	0.00879	0.01872	-0.00796
	0.5931	0.8499	0.1179	0.7158	0.4381	0.7416
	1,717	1,717	1,717	1,717	1,717	1,717
Opiate	0.00736	0.00856	-0.00087	-0.01386	-0.01447	0.02258
	0.7607	0.7231	0.9712	0.5661	0.5491	0.3497
	1,717	1,717	1,717	1,717	1,717	1,717
Antidepressant	0.03368	0.04014	-0.03397	-0.01265	-0.00240	-0.01211
	0.1631	0.0963	0.1594	0.6004	0.9210	0.6160
	1,717	1,717	1,717	1,717	1,717	1,717

ISS = Injury Severity Score

NISS = New Injury Severity Score

RTS = Revised Trauma Score (scores for severity of physiological derangement on presentation)

Note: Statistically significant P-values are in bold

Disposal (discharge status) of those attending hospital (see Tables 10a–c)

More patients were admitted to hospital from Group 1 than were discharged to home after treatment (69 per cent, 1,046 of 1,515 versus 29.5 per cent, 447 of 1,515). The incidence of AOD positivity in those admitted (44 per cent, 460 of 1,046) was higher than in those discharged (36.7 per cent, 164 of 447). This was statistically significant (P-value < 0.01).

In the lower acuity Group 2, the rates of admission were reversed, with 89.6 per cent of patients being discharged. Nevertheless, the rates of intoxication in those admitted from Group 2 were once again higher than the rates of intoxication in those discharged (33.3 per cent, 7 of 21 versus 23.2 per cent, 42 of 181). This was not statistically significant.

Higher admission rates in the positive-for-AODs group may be a reflection of altered clinical conditions rendering physicians less sure of their findings, and thus opting for a period of admission and observation prior to discharge.

It would certainly appear, on admission rates alone, that patients who are positive for AODs create an additional and presumably otherwise avoidable financial burden on the health system.

Table 10a. Patient discharge status – total.

	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Admitted to hospital	600	467 (43.8%)	1,067 (62.1%)
Discharged home	422	206 (32.8%)	628 (36.6%)
Transferred from ED	5	2 (28.6%)	7 (0.4%)
Died in ED	4	4 (50%)	8 (0.5%)
Other	1	6 (85.7%)	7 (0.4%)
Total	1,032	685 (39.9%)	1,717
(Died in total)	29	23 (44.2%)	52 (3%)

Table 10b. Patient discharge status (Group 1).

Patient Status: TS	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Discharged home	283	164 (36.7%)	447 (29.5%)
Admitted to hospital	586	460 (44%)	1,046 (69%)
Transferred from ED	5	2 (28.6%)	7 (0.5%)
Died in ED	4	4 (50%)	8 (0.5%)
Other	1	6 (85.7%)	7 (0.5%)
Total	879	636 (42%)	1,515
(Died in total)	29	23 (44.2%)	52 (3.4%)

Table 10c. Patient discharge status (Group 2).

Patient Status: ED	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Discharged from ED	139	42 (23.2%)	181 (89.6%)
Admitted to hospital	14	7 (33.3%)	21 (10.4%)
Transferred from ED	0	0	0
Died in ED	0	0	0
Other	0	0	0
Total	153	49 (24.3%)	202
(Died in total)	0	0	0

Location of disposal (see Table 11)

In total, 1,067 patients were admitted to hospital from Groups 1 and 2.

The majority were admitted to general hospital wards (66.6 per cent, 711 of 1,067), and in that group the positive rate for AODs was 42.9 per cent (305 of 711).

Of those admitted to hospital, just over one-fifth of patients were admitted to High Dependency or Intensive Care Unit environments (229 of 1,067), but in this smaller group of patients 55 per cent (126 of 229) were positive for AODs. This rate of positivity is highly statistically significant (P-value < 0.002) when compared with admissions to the general ward.

It is possible that this is a reflection of more serious injury in those with intoxicants in their system, and possibly also due, in part, to the effects of AODs mimicking traumatic pathological processes. Either way, the requirement to admit AODs-positive patients to high acuity beds greatly increases the cost of their care.

Table 11. Location post-ED.

	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
ICU/HDU	103	126 (55%)	229 (21.5%)
General Ward	406	305 (42.9%)	711 (66.6%)
Spinal Unit	74	27 (26.7%)	101 (9.5%)
Burns Unit	13	9 (40.9%)	22 (2.1%)
Died in operating theatre	3	0	3 (0.3%)
Unknown	1	0	1 (0.1%)
Total	600	467 (43.8%)	1,067*

*1,067 of 1,717 patients admitted to hospital

Length of hospital stay (see Tables 12a–c)

While the majority of trauma patients were admitted to hospital (62.1 per cent, 1,067 of 1,717), a large group was either discharged from the ED or did not stay in hospital longer than 24 hours (48.6 per cent, 835 of 1,717). The percentage of positive results for AODs in admitted patients was remarkably consistent for different admission lengths although slightly higher in those admitted for less than 24 hours (50.3 per cent, 94 of 187). It is possible that a number of these short-stay patients were admitted because of difficulties in deciding whether their clinical condition was due to intoxication or to the trauma itself.

The positive rates for AODs in those patients admitted from Group 2 were all much less than those from Group 1 (Tables 12b, 12c).

Table 12a. Length of stay for admitted patients – total.

No. of days	Negative Toxicology	Positive Toxicology (% of positives)	Total (% number of total)
<24 hours	93	94 (50.3%)	187 (17.5%)
1	64	37 (36.6%)	101 (9.4%)
2–7	250	201 (44.6%)	451 (42.2%)
8–14	79	60 (43.2%)	139 (13%)
15–21	40	24 (37.5%)	64 (6%)
22–28	21	15 (41.7%)	36 (3.4%)
>28	54	37 (40.7%)	91 (8.5%)
Total	601	468 (43.8%)	1,069

Table 12b. Length of stay for admitted patients (Group 1)*.

No. of days	Negative Toxicology	Positive Toxicology (% of positives)	Total (% number of total)
<24 hours	381	265 (41%)	646 (42.6%)
1	60	36 (37.5%)	96 (6.3%)
2–7	244	199 (44.9%)	443 (29.2%)
8–14	79	60 (43.2%)	139 (9.2%)
15–21	40	24 (37.5%)	64 (4.2%)
22–28	21	15 (41.7%)	36 (2.4%)
>28	54	37 (40.7%)	91 (6%)
Total	879	636 (00%)	1,515

* Note that <24 hr group includes those discharged from the ED

Table 12c. Length of stay for admitted patients (Group 2)*.

No. of days	Negative Toxicology	Positive Toxicology (% of positives)	Total (% number of total)
<24 hours	143	46 (24.3%)	189 (93.6%)
1	4	1 (20%)	5 (2.5%)
2–7	6	2 (25%)	8 (4%)
8–14	0	0	0
15–21	0	0	0
22–28	0	0	0
>28	0	0	0
Total	153	49 (24.3%)	202

* Note that <24 hr group includes those discharged from the ED

Motor vehicle crashes

Nature of crash (see Tables 13a–c, 14, 15, 16a–b, 17a–b)

As previously stated, MVCs represent the majority of the workload presenting to Group 1.

The largest subgroup within the Group 1 MVC group was occupants of cars (either drivers or passengers), who made up more than two-thirds of all vehicular trauma-related attendances (66.2 per cent, 798 of 1,206). Motorcycle-related crashes were in second place (16.8 per cent, 203 of 1,206), and in third place, with half as few again, were pedestrians (8.5 per cent, 102 of 1,206). (Table 13a).

These three subgroups of MVCs had very similar rates for positivity to AODs (all around 40 per cent). Lower levels of positive results were found in Group 2 (between 24 per cent and 30 per cent). (Tables 13b, 13c).

Table 13a. Nature of motor vehicle crash (MVC).

Nature of Accident	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Car occupant	487	311 (39%)	798 (66.2%)
Motorcyclist	119	84 (41.4%)	203 (16.8%)
Pedestrian	61	41 (40.2%)	102 (8.5%)
Cyclist	55	24 (30.4%)	79 (6.6%)
Truck/bus occupant	19	3 (13.6%)	22 (1.8%)
3-wheel/off-road	2	0	2 (0.2%)
Total	743	463 (38.4%)	1,206

Table 13b. Nature of MVC (Group 1).

Nature of Accident	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Car occupant	365	273 (42.8%)	638 (63.6%)
Motorcyclist	110	81 (42.4 %)	191 (19%)
Pedestrian	54	38 (41.3%)	92 (9.2%)
Cyclist	42	21 (33.3%)	63 (6.3%)
Truck/bus occupant	15	2 (11.8%)	17 (1.7%)
3-wheel/off-road	2	0	2 (0.2%)
Total	588	415 (41.4%)	1,003

Table 13c. Nature of MVC (Group 2).

Nature of Accident	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Car occupant	122	38 (23.8%)	160 (79.2%)
Motorcyclist	9	3 (25.0%)	12 (5.9%)
Pedestrian	7	3 (30.0%)	10 (5%)
Cyclist	13	3 (18.8%)	16 (7.9%)
Truck/bus occupant	2	1 (33.3 %)	3 (1.5%)
3-wheel/off-road	1	0	1 (0.5%)
Total	154	48 (23.8%)	202

Most car crashes involved another vehicle (58.6 per cent, 468 of 798). In this group just over one-quarter of the occupants were positive for AODs (27.8 per cent, 130 of 468) (Table 14).

In the car crash group not involving another vehicle (collide with stationary object or rollover etc.), the occupants were positive nearly half of the time (45.4 per cent, 149 of 328) (Table 14). This is highly statistically significant compared with 'versus other vehicle' crashes (P-value < 0.0001).

Table 14. Car crashes (involving another vehicle versus solitary vehicle).

Nature of Accident	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Versus other vehicle	338	130 (27.8%)	468 (58.6%)
Versus stationary object/non-collision	179	149 (45.4%)	328 (41.1%)
Unknown	0	2 (100%)	2 (0.3%)
Total	517	281	798

However, when comparing car drivers only for these two subgroups – the single vehicle crash (80 of 238) and multiple vehicle crash (141 of 339) – positivity for AODs is not statistically significantly different. The difference seen above appears to be due entirely to the non-drivers (Tables 16a, 16b).

The more seriously injured patient group that was triaged to the Trauma Service (Group 1) showed consistently higher rates of positivity for AODs than Group 2, except for passengers involved in multiple vehicle crashes (Tables 15, 16a-b, 17a-b).

Table 15. Vehicle crashes involving single or multiple vehicles.

Car	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Single vehicle	248	236 (48.8%)	484 (44%)
Multiple vehicles	433	184 (29.8%)	617 (56%)
Total	681	420 (38.1%)	1,101

Table 16a. Drivers versus non-drivers in single versus multiple vehicle MVCs (Group 1).

Overall MVCs – Group 1	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total
Single – driver	215	121 (36%)	336
Single – non-driver	38	59 (60.8 %)	97
Multiple – driver	164	150 (47.8%)	314
Multiple – non-driver	74	25 (25.3%)	99
Total	491	355 (42%)	846

Table 16b. Drivers versus non-drivers in single versus multiple vehicle MVCs (Group 2).

Overall MVCs – Group 2	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total
Single – driver	20	6 (23.1%)	26
Single – non-driver	3	1 (25%)	4
Multiple – driver	84	23 (21.5%)	107
Multiple – non-driver	25	11 (30.6%)	36
Total	132	41 (23.7%)	173

Table 17a. Outcome for drivers versus non-drivers of cars in single versus multiple vehicle MVCs (Group 1).

Car only	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total
Single – driver	142	76 (34.9%)	218
Single – non-driver	32	56 (63.6%)	88
Multiple – driver	120	119 (49.8%)	239
Multiple – non-driver	71	21 (22.8%)	92
Total	365	272 (42.7%)	637

Table 17b. Outcome for drivers versus non-drivers of cars in single versus multiple vehicle MVCs (Group 2).

Car only	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total
Single – driver	16	4 (20%)	20
Single – non-driver	3	1 (25%)	4
Multiple – driver	78	22 (22%)	100
Multiple – non-driver	25	11 (30.6%)	36
Total	122	38 (23.8%)	160

Car occupants (see Table 18a–b)

The majority of car occupants in Group 1 were themselves drivers (71.7 per cent, 457 of 637) as were those in Group 2 (75 per cent, 120 of 160). Car occupants in Group 1 had a substantially higher chance of being positive for AODs (42.7 per cent, 195 of 457) than those in Group 2 (21.7 per cent, 26 of 120).

In both Groups 1 and 2, the non-drivers and drivers had similar rates of positivity to AODs, with no significant difference (Tables 18a, 18b).

Table 18a. Car occupant – driver versus non-driver (Group 1).

Occupant	Negative	Positive (% positive of subgroup)	Total
Driver	262	195 (42.7%)	457
Non-driver	103	77 (42.8%)	180
Total	365	272 (42.7%)	637

Table 18b. Car occupant – driver versus non-driver (Group 2).

Occupant	Negative	Positive (% positive of subgroup)	Total
Driver	94	26 (21.7%)	120
Non-driver	28	12 (30%)	40
Total	122	38 (23.8%)	160

Safety devices used in MVCs (see Table 19)

The majority (89.8 per cent, 935 of the 1,041 where use or non-use is known) of vehicular users used safety devices. The two largest groups in our series were drivers wearing their safety belts (93.4 per cent, 761 of 815 of those where use or non-use is known), and passengers wearing their safety belts (78.7 per cent, 174 of 221 of those where use or non-use is known). In those two groups, only around one-third (total 34.1 per cent, 319 of 935 – drivers 34.2 per cent, 260 of 761 and passengers 33.9 per cent, 59 of 174) returned positive blood tests for AODs.

Despite lower numbers of drivers not using their safety belts (6.6 per cent, 54 of 815), or where it was uncertain whether they were using their safety belts or not (7.5 per cent, 66 of 881), these two groups showed much higher tendencies to be positive for AODs (61.1 per cent, 33 of 54, and 53 per cent, 35 of 66 respectively). This was highly statistically significant for all comparisons (drivers alone, passengers alone and 'drivers and passengers' – all P-values < 0.0001).

It is possible that drug positivity was associated with impaired judgement, resulting in either forgetting to secure oneself with a safety device or a diminished perception of the importance of the safety device. Almost exactly the same rates of AODs positivity are seen in those patients who claim to be unsure as to whether or not they were wearing a safety device, as in the unbelted group, leading to speculation that they may in fact belong to the latter group.

Passengers were slightly less likely to wear safety belts overall, but those who did had a marginally lower chance of being positive for AODs (33.9 per cent, 59 of 174). Once again, although lower numbers of passengers did not use their safety belts (21.3 per cent, 47 of 221), or it was uncertain if they had or had not (9.4 per cent, 23 of 244), these two groups again showed an increased likelihood to be positive for AODs (55.3 per cent, 26 of 47 versus 56.5 per cent, 13 of 23). This was highly statistically significant (P-values <0.0001).

Table 19. Drivers and safety devices.

	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Driver, safety	501	260 (34.2%)	761 (67.6%)
Driver, no safety	21	33 (61.1%)	54 (4.8%)
Passenger, safety	115	59 (33.9%)	174 (15.5%)
Passenger, no safety	21	26 (55.3%)	47 (4.2%)
Driver, safety unknown	31	35 (53.0%)	66 (5.9%)
Passenger, safety unknown	10	13 (56.5%)	23 (2%)
Total	699	426 (37.9%)	1,125

Drugs involved in car crashes

The most common drug found in all categories of drivers was alcohol, with 122 of 564 (21.6 per cent) drivers positive. This compared with 96 of 564 (17 per cent) positive for cannabinoids, 79 of 564 (14 per cent) for benzodiazepines, 37 of 564 (6.6 per cent) for amphetamines and 18 of 564 (3.2 per cent) for opiates.

Alcohol was also the drug most commonly found in AODs-positive patients who were non-drivers (24.1 per cent, 53 of 220), and in this group there was also a large number of persons positive for cannabinoids (20 per cent, 44 of 220).

In more than two-thirds of all cases involving a car crash, where an occupant returned a positive result, it was the driver who was positive. This was regardless of drug type involved. Benzodiazepines also featured significantly in drivers, as did opioids, with 91 per cent and 85 per cent respectively of affected occupants being drivers.

Table 20. Car crashes by drug type.

	ETOH	THC	Amphet	Benzo	Opioid	Number positive individuals	Number negative individuals
Driver, safety	85	63	26	66	18	160	321
Driver, no safety	19	13	1	6	0	28	12
Driver, safety unknown	18	18	10	7	0	33	10
Passenger, safety	27	26	14	6	3	52	107
Passenger, no safety	17	13	3	0	0	24	14
Passenger, safety unknown	9	5	1	1	0	13	10
Total	175	138	55	86	21	310*	474

* Note: each individual may be positive for more than one agent

Drugs involved in motorcycle crashes (see Table 21)

Of 203 motor cycle crash victims, 88 returned positive AOD tests. Unlike car crashes, the most common drugs detected were cannabinoids, followed by alcohol. Riders were positive for AODs 41.5 per cent of the time (78 of 188) with 28.2 per cent (53 of 188) positive for cannabinoids, 11.7 per cent (22 of 188) positive for alcohol, 7.4 per cent (14 of 188) positive for amphetamines, 6.9 per cent (13 of 188) positive for benzodiazepines, and 5.3 per cent (10 of 188) positive for opioids. This appears to be quite a different pattern to that seen in car drivers.

Table 21. Motorcycle crashes by drug type.

	ETOH	THC	Amphet	Benzo	Opioid	Number positive individuals	Number negative individuals
Rider, safety*	20	51	14	13	10	74	109
Rider, no safety	2	2	0	0	0	4	1
Passenger, safety	2	4	5	0	1	6	5
Passenger, no safety**	0	0	0	0	0	0	0
Unknown safety	1	1	0	0	0	4	0
Total	25	58	19	13	11	88***	115

* Safety – wearing a helmet

** No safety – not wearing a helmet

*** Note: each individual may be positive for more than one agent

Drugs involved in pedal cycle MVCs (see Table 22)

An overwhelming number of cyclists involved in MVCs wore their helmets regardless of their positivity. Roughly one-quarter were positive for AODs. More than half of all positive results were for alcohol or cannabinoids. Riders were positive for AODs 31.1 per cent of the time (23 of 74) with 20.2 per cent (15 of 74) positive for alcohol and also 20.2 per cent (15 of 74) positive for cannabinoids. Other drugs were all positive in less than 5 per cent of cases.

Table 22. Pedal cycle MVCs by drug type.

	ETOH	THC	Amphet	Benzo	Opioid	Number positive individuals	Number negative individuals
Rider, helmet	14	14	1	3	2	23	48
Rider, no helmet	1	1	0	0	0	1	2
Total	15	15	1	3	2	24*	50

* Note: each individual may be positive for more than one agent

Drugs involved in pedestrian MVCs

A total of 102 pedestrians presented to the study, 92 to Group 1 and 10 to Group 2. 41.3 per cent were positive for AODs in Group 1 and 30 per cent in Group 2. The most commonly occurring positive result was alcohol with 26.5 per cent (27 of 102), followed by cannabis with 16.7 per cent (18 of 108) and amphetamines with 11.8 per cent (12 of 102). The most common time for presentation was between 14:00 and 18:00, and the most common time for presenting with positive AODs was between 22:00 and 02:00.

Table 23. Pedestrian MVCs by drug type.

	ETOH	THC	Amphet	Benzo	Opioid	Number positive individuals	Number negative individuals
Pedestrian	27	18	12	4	2	41*	61

* Note: each individual may be positive for more than one agent

Self-harm (see Table 24)

A total of 39 persons presented as a consequence of self-harm. The most common modality of self-harm was through the use of a sharp object (38.5 per cent, 15 of 39), closely followed by attempted hanging. High rates of AODs use (>40 per cent) were found in nearly all traumatic causes of self-harm, although the total numbers were small.

Table 24. Nature of self-harm.

Nature of incident	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Hanging	7	5 (41.7%)	12 (30.8%)
Sharp object	8	7 (46.7%)	15 (38.5%)
Smoke/fire	2	2 (50%)	4 (10.3%)
Firearm	3	1 (25%)	4 (10.3%)
Jump high place	1	2 (66.7%)	3 (7.7%)
Other	1	0	1 (2.6%)
Total	22	17 (43.6%)	39 (100%)

Assault (see Table 25a–25d)

A total of 115 patients presented to the trauma unit as a consequence of assault. This represented a unique group in the study, in that for each sub-category the lowest rate of AOD positivity was more than 60 per cent. Nearly three-quarters (72.2 per cent, 83 of 115) of all assaulted patients presenting to the Trauma Unit were positive for AODs. In no other group studied was the incidence of positivity greater than 50 per cent. Highest rates of AOD positivity were found in victims assaulted with sharp weapons. The most common modality of injury was blunt force trauma (38.3 per cent, 44 of 115), closely followed by sharp or penetrating injury (33 per cent, 38 of 115). The highest rate of AOD positivity was in this last group with 78.9 per cent (30 of 38). In contrast to the rates of firearm injuries elsewhere in the world, only 7 cases presented as a result of assault in a year. The rate of AOD positivity in those assaulted with firearms was 71.4 per cent (5 of 7).

The most commonly occurring positive result was for alcohol with 47.8 per cent positive (55 of 115), then cannabinoids with 35.7 per cent (41 of 115), benzodiazepines with 24.3 per cent (28 of 115), amphetamines with 7.8 per cent (9 of 115) and finally opioids with 5.2 per cent (6 of 115).

The most common place of assault was at home, followed by on the street, then within a trade or services area, generally a pub. The street location, of course, may reflect someone having just left a trade or service area.

Age and gender distribution is shown below.

Table 25a. Nature of assault.

Nature of assault	Negative Toxicology	Positive Toxicology (% positive of subgroup)	Total (% of total)
Sharp	8	30 (78.9%)	38 (33%)
Blunt	12	32 (72.7%)	44 (38.3%)
Bodily force	10	16 (61.5%)	26 (22.6%)
Firearm	2	5 (71.4%)	7 (6.1%)
Total	32	83 (72.2%)	115 (100%)

Table 25b. Place of assault for positive toxicology.

Nature of assault	Home	Street	Trade/service	Other
Firearm	1	2	2	0
Sharp	12	8	8	0
Blunt	10	7	9	6
Bodily force	6	6	2	2

Table 25c. Assault, drugs and gender.

Gender	ETOH	THC	Amphetamines	Benzodiazepines	Opioids
All	55	41	9	28	6
Male	45	33	9	22	4
Female	10	9	0	6	4

Table 25d. Assault, drugs and age.

Age	ETOH	THC	Amphetamines	Benzodiazepines	Opioid
All	55	41	9	28	6
<18	3	3	0	0	0
18–35	36	28	8	17	2
36–50	12	9	1	8	4
51–74	4	1	0	3	0
>74	0	0	0	0	0

Blood alcohol over 0.05mg% (see Table 26a, b)

In the cases of MVCs, assaults and self-harm, the majority of patients who had any alcohol in their system had more than 0.05mg%. In the case of MVCs this was 65.4% (157 of 240) of patients (Table 26a).

Other findings were that:

- More than half had a BAL of 0.11mg% or greater (50.4%, 121 of 240).
- 30% had a BAL of 0.16mg% or higher (72 of 240).
- 15.4% had a BAL of greater than 0.2 mg% (37 of 240).

These are all statistically significantly higher than RBT data (P-value <0.0001).

Considering all mechanisms of injury, even in patients who had BAL < 0.05mg% (113), 36.3% (41 of 113) were positive for DOTA (Table 26b).

In the cases of assaults and self-harm, the majority of patients who had any alcohol in their system had more than 0.05mg%. This was 80 per cent of cases for both assaults (44 of 55) and self-harm (8 of 10).

Table 26a. Blood alcohol levels versus MVCs, assaults and self-harm in Groups 1 and 2 combined, using Group 3 as comparison.

Blood Alcohol mg%	MVC	Assault	Self-harm	Group 3*
< or 0.05	83	11	2	0
0.051–0.10	36	5	2	3
0.101–0.15	49	15	2	0
0.151–0.2	35	14	1	3
>0.2	37	10	3	0
Total	240	55	10	6

* IMVS comparison group (Group 3)

Table 26b. BAL < 0.05 but other drugs present.**

THC	23
Benzos	12
Amphetamines	4
Cocaine	1
Opioids	1
Total	41

** BAL < 0.05 – n=113

Toxicology results

Overall results (see Table 27)

A total of 774 patients were found to have AODs in their blood, with 1,088 positive results (Table 27). In 528 cases, patients were only positive to one drug while 246 patients were positive to two, three or four drugs.

Table 27. Total positive toxicology results*.

Drug	Number	Total	% single drug positives (n=528)	% of all tests for that drug	% of total tests
Alcohol ONLY	225		42.6	60.6	
Alcohol plus other(s)	146	371	-	39.4	35.9
THC ONLY	158		29.9	50.0	
THC plus other(s)	158	316	-	50.0	30.6
Benzos ONLY	87		16.5	48.3	
Benzos plus other(s)	93	180	-	51.7	17.4
Amphetamines ONLY	35		6.6	27.1	
Amphetamines plus other(s)					
plus unspecified	17		-	13.2	
plus MDMA	14		-	10.9	
plus methyl-amphet	63	129	-	48.8	9.1
Opioids ONLY	20		3.8	29.4	
Opioids plus other(s)					
plus codeine	33		-	48.5	
plus morphine	15	68	-	22.1	4.6
Methadone** ONLY	2		0.4	16.7	
Methadone** plus other(s)	10	12	-	83.3	1.2
Drugs not elsewhere listed	1		0.2	14.3	
Drugs not elsewhere listed plus other(s)	6	7	-	85.7	0.7
Cocaine ONLY	0		0	0	
Cocaine plus other(s)	3	3	-	100	0.3
Heroin** ONLY	0		0	0	
Heroin** plus other(s)	2	2	-	100	0.2

* Total positive toxicology results – n=1088

**All Methadone/ heroin positives were known users

Mono- versus poly-intoxications (see Table 28)

Frequently, drugs were found together (poly-intoxications) (i.e. patients who had one drug found in their system often had another). Alcohol was the drug most commonly found in mono-intoxications, in 42.6 per cent (225 of 528) of all mono-intoxications. This was followed by cannabis (29.9 per cent, 158 of 528), benzodiazepines (16.5 per cent, 87 of 528), amphetamines (6.6 per cent, 35 of 528), and opioids (3.8 per cent, 20 of 528). Cocaine and heroin were never found alone.

Table 28. Ranking of various drug combinations.

Number of drugs	Drug combinations	Number	Subtotal
1	ETOH	225	
1	THC	158	
1	Benzos	87	
1	Amphetamines	35	
1	Opioids	20	
1	Methadone	2	
1	Other *	1	
1	Cocaine	0	
1	Heroin	0	528
2	ETOH/THC	92	
2	ETOH/Benzos	26	
2	THC/Amphetamine	20	
2	THC/Benzos	17	
2	ETOH/Amphetamine	11	
2	Benzos/Opioids	10	
2	Combination of amphetamines	8	
2	Amphet/Benzos	7	
2	ETOH/Opioids	6	
2	THC/Opioids	5	
2	THC/Other*	2	
2	Amphet/Opioids	2	
2	Benzos/Other*	2	
2	Benzos/Methadone	2	
2	THC/Methadone	1	
2	Amphet/Methadone	1	212

Table 28 continued.

Number of drugs	Drug combinations	Number	Subtotal
3	ETOH/ HC/Benzos	12	
3	THC/Amphet/Benzos	4	
3	ETOH/Amphet/THC	3	
3	ETOH / THC/Other*	1	
3	ETOH/Amphet/Cocaine	1	
3	THC/Benzo/Opioid	1	
3	ETOH/Opioids/Benzo	1	
3	Benzos/Opioids/Methadone	1	
3	THC/Benzo/Cocaine	1	
3	Heroin/Benzo/Opioid	1	
3	THC/Benzo/Other*	1	
3	THC/Benzo/Methadone	1	28
4	THC/Amphet/Benzo/Methadone	2	
4	ETOH/THC/Methadone/Benzos	1	
4	Methadone/Heroin/Benzo/Opioid	1	
4	ETOH/Cocaine/Benzo/Amphet	1	
4	THC/Benzo/Amphet/Opioid	1	6

* Other = drugs not otherwise listed

A perhaps surprising number of positive results were found in Group 3. Some of these might be explained as being positive from normal medication, but more than 9% (37 of 410) were positive for cannabinoids, across all age groups (see Chapter four: Results Tables 2c, 3e).

In Group 3 it could be argued that, because the blood samples analysed were taken from an outpatient laboratory service, the incidence of medical drugs (such as benzodiazepines and opioids) might be higher than a random sample of the general population at large. The incidence of samples positive for benzodiazepines was 9.3 per cent (38 of 410) and 5.4 per cent (22 of 410) for opioids. Strictly non-prescription drugs are more difficult to explain this way. No amphetamines were detected in the comparison group, and alcohol was only found in 1.5 per cent of samples (6 of 410). The results for alcohol fall between the values of detection rates of mobile (3.2 per cent) and fixed (0.9 per cent) random breath testing used by SAPOL over the same time period. Of the comparison group, 9 per cent (37 of 410) were positive for THC and/or THC acid.

Drug combinations

The prevalence of different drug combinations is detailed in Tables 28 and 29.

In poly-intoxications with two drugs, the most common mixture was alcohol and cannabis (92 of 212), with less than one-third of that number represented in the next most common combination of alcohol and benzodiazepines (26 of 212). There were 14 further, different combinations of two-drug poly-intoxications.

This pattern is reflected in poly-intoxications involving three drugs, with alcohol, cannabis and benzodiazepines being the most common (12 of 28). There were 12 different three-drug poly-intoxications. There were six occurrences of positivity to four drugs.

Table 29. Profile of most common drug combinations*.

Event	ETOH/ THC	ETOH/ Benzo	THC/ Amphet	THC/ Benzo	ETOH/ Amphet	Benzo/ Opioids	Total
MVC	78	23	26	23	10	13	173
Assault	21	13	3	10	4	1	52
Falls	5	2	3	2	1	0	13
Self-harm	0	2	0	1	0	0	3
Other	1	1	0	1	1	1	5
Total	105	41	32	37	16	15	246

* may be more than two drugs present for each individual so individuals could be counted more than once (e.g. if someone has ETOH and THC and amphetamines, they would be counted here in three columns)

Diurnal patterns (see Tables 30a-c)

An interesting difference was observed in the distribution of 'time of presentation' between patients positive and negative for AODs. For trauma patients, 63.9 per cent (242 of 379) presenting to hospital between 10pm and 6am were positive for AODs. This compares with a positivity rate of 33.1 per cent (443 of 1,338) for trauma patients presenting between 6am and 10pm. This is highly statistically significant (P-value <0.0001).

Table 30a. Time of incident.

Time	Negative Toxicology	Positive Toxicology (% positive for time period)	Total
Unknown	6	3 (33.3%)	9
0600–0959	167	59 (26.1%)	226
1000–1359	273	103 (27.4%)	376
1400–1759	290	143 (33%)	433
1800–2159	159	135 (45.9%)	294
2200–0159	94	147 (61%)	241
0200–0559	43	95 (68.8%)	138
Total	1,032	685 (39.9%)	1,717

Table 30b. Time of incident (Group 1).

Time	Negative Toxicology	Positive Toxicology (% positive for time period)	Total
Unknown	6	3 (33.3%)	9
0600–0959	129	51 (28.3%)	180
1000–1359	228	93 (29%)	321
1400–1759	261	133 (33.8%)	394
1800–2159	134	125 (48.3%)	259
2200–0159	83	140 (62.8%)	223
0200–0559	38	91 (70.5%)	129

Table 30c. Time of incident (Group 2).

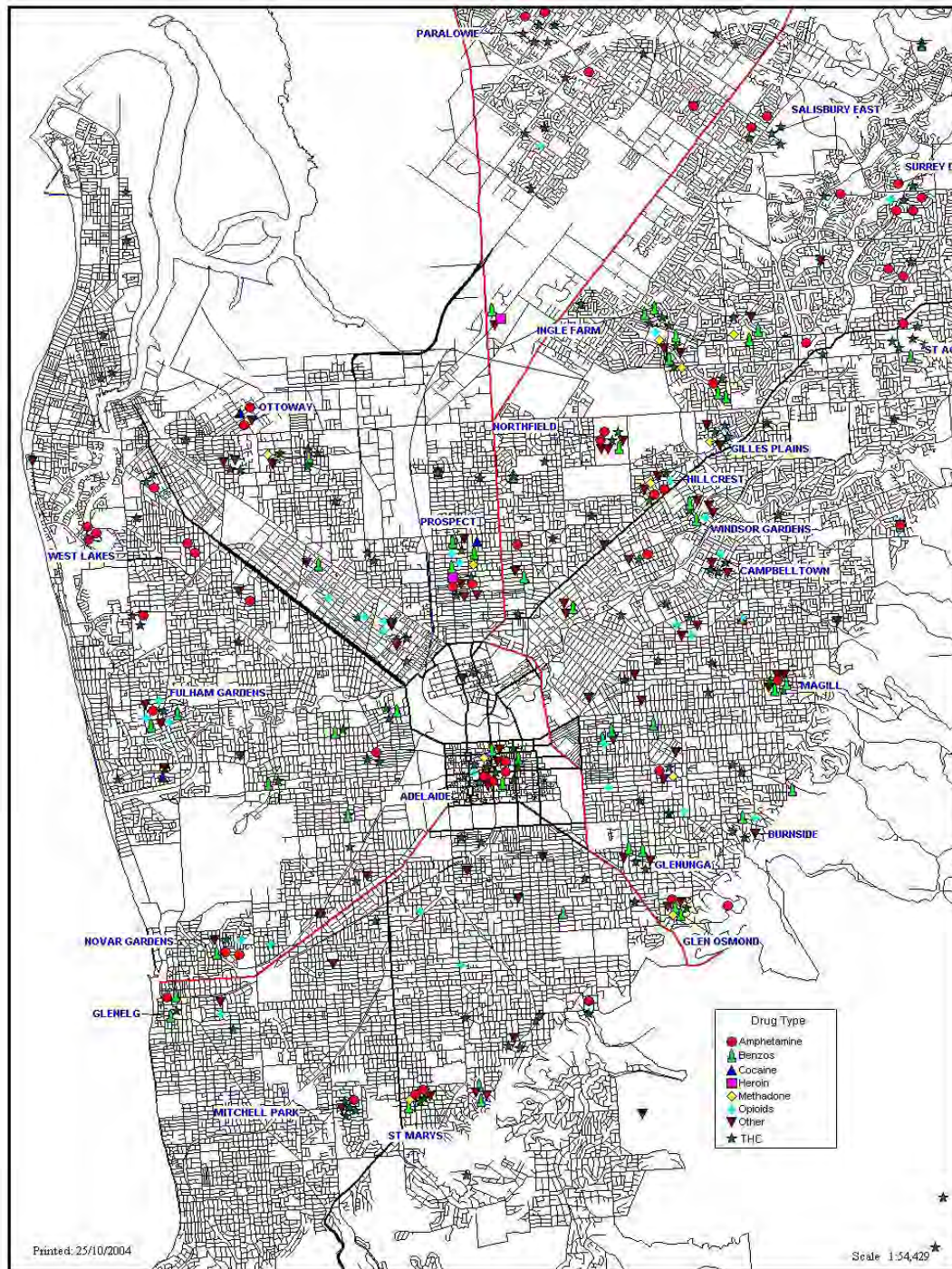
Time	Negative Toxicology	Positive Toxicology (% positive for time period)	Total
Unknown	0	0	0
0600–0959	38	8 (17.4%)	46
1000–1359	45	10 (18.2%)	55
1400–1759	29	10 (25.6%)	39
1800–2159	25	10 (28.6 %)	35
2200–0159	11	7 (38.9%)	18
0200–0559	5	4 (44.4%)	9

GIS data

The residential location of all AOD-positive patients was plotted. This data may become useful in planning and delivering drug-education specific messages.

Figure 1: TraumaTox Oct. 2004 by residence

TraumaTox Oct. 2004 by residence (Example Only)



Chapter five: Conclusion

Potential study limitations

It is acknowledged that this study contained some inherent limitations. The RAH TraumaTox Study Group has been aware of these since the study's inception. Accordingly, it is worth making a brief acknowledgment and comment here on some of those limitations, and the authors' approach to minimising their effects.

Group 3 was used in an effort to provide a comparison with the general community. While it was matched for age, gender and postcode, the selection of people who were having blood tests for some reason may have led to a slightly 'sicker' group than the average community being selected. This may have resulted in this group having a higher incidence of drug usage than the general community. Despite this, there were very large and very significant differences between the findings in this group and those of the trauma victims.

One aim of having Group 2 was to have a comparison group of minor trauma compared with the more major (severe) trauma of Group 1, and the non-trauma of Group 3. However, some lower severity trauma cases were found in Group 1, and some higher ones in Group 2. Despite this, comparisons between these groups confirmed the significantly higher overall (and average) severity of trauma in Group 1. Comparisons regarding severity of trauma were also able to be done across all patients in both groups using internationally recognised severity scoring systems (ISS, NISS). Again, there were very large and very significant differences between the findings in this group and Group 1 and Group 3.

The use of RBT data as a comparison group aimed to provide a comparison to the general community of drivers. It can be argued that RBT does not accurately do this, although the results are remarkably consistent over time. Equally, the fact that mobile RBT data is consistently higher in positive reporting than fixed RBT data is to be expected from the fact that mobile RBT testing is targeted towards road users who may have done something to bring themselves to the attention of a police patrol. However, the extremely large differences (factors of well over 10) in results between TraumaTox blood testing and RBT testing can not be explained away by suggesting RBT is not a completely accurate reflection of driver behaviour. It is worth noting that the positive blood alcohol finding in Group 3 was 2 per cent (8 of 410). This is not dissimilar to the findings in the RBT groups of 0.9 per cent and 3.2 per cent for fixed and mobile RBT respectively, and perhaps adds additional support to RBT being a reasonable indication of baseline alcohol usage in drivers (in those geographical areas where it is used).

While the use of the three comparison groups (Group 1, Group 2 and RBT) allowed many comparisons to be made, as acknowledged above none of these are ideal as true 'control' groups. It is worth noting that many comparisons were made within groups as well. Despite these limitations, the findings within groups and between groups were remarkably consistent. The consistently high level of statistical significance found means that the results should not be dismissed because these groups were not perfect control groups.

Questions may also be raised regarding testing for drugs and the relationship between recent use and drug levels. Cannabis is perhaps the prime example where this has been questioned in the past. The use of blood, rather than urine, to test for drugs makes the results more relevant to time of usage. Regardless of this, it could be argued that our testing could have picked up some

chronic, but non-recent, usage. In reality, this issue in no way invalidates our results as, regardless of whether usage was recent or not, the highly significant findings indicate a relationship between blood levels 'as tested' and trauma in multiple, highly statistically significant ways. The actual contributions of acute versus chronic usage may be slightly unclear; what is clear, however, is that there appears to be a relationship between drugs (including cannabis), as tested in our study, and trauma. The same is true for other drugs.

Final comments

This study was one of the largest of its type ever conducted. The findings have significant implications for health, law enforcement, policy making and research in relation to the recognition of the impact of drugs other than alcohol on a range of trauma. Significantly, the findings also add to the growing evidence base for drug-driving.

The analysis of blood drug levels in this study, rather than analysis of urine drug levels (as used in most previous related studies), allows for better correlation of results with recent drug usage.

Despite the acknowledged limitations of this study, as discussed on the previous page, the very large numbers, the multiple comparison groups and the consistently very strong statistical findings from the data make these results and statistics perhaps the most comprehensive currently available.

References

- Australian Institute of Health and Welfare 2002, *2001 National Drug Strategy Household Survey: First results*. Drug Statistics Series No. 9.
- Berghaus, G., Scheer, N. & Schmidt, P. 1995, Effects of Cannabis on Psychomotor Skills and Driving Performance – A Meta-Analysis of Experimental Studies, Proceedings of the 13th International Conference on Alcohol, Drugs and Traffic Safety, The International Council on Alcohol, Drugs and Traffic Safety, Adelaide.
- Carrigan, T.D. & Field, H. 2000. Toxicological screening in trauma, *Journal of Accident and Emergency Medicine*; vol.17, no. 1, pp. 33–37.
- Cornwell, E.E. 3rd & Belzberg, H. 1998. The prevalence and effect of alcohol and drug abuse on cohort-matched critically injured patients, *American Journal of Surgery*, May vol. 64, no. 5, pp. 461–465.
- Drummer, O. 1994, *Drugs in Drivers Killed in Australian Road Traffic Accidents: The Use of Responsibility Analysis to Investigate the Contribution of Drugs to Fatal Accidents*, Victorian Institute of Forensic Pathology, Melbourne.
- Drummer, O. 1995, *A Review of the Contribution of Drugs in Drivers to Road Accidents*. Victorian Institute of Forensic Pathology. [Online], Available: <http://www.parliament.vic.gov.au/parlpsc/drugdrum.htm>, [Accessed 25 September 2000].
- Drummer, O. 1999, *Involvement of Drugs in Accident Causation*, Report to the AustRoads Drugs and Driving Committee, Victorian Institute of Forensic Medicine, Melbourne.
- Drummer, O., Caplehorn, J. & Gerostamoulos, J. 1999, *Drugs in Drivers killed in New South Wales Road Traffic Crashes 1997 & 1998*, Victorian Institute of Forensic Medicine, Melbourne.
- Fitzsimmons, G. & Cooper-Stanbury, M. 2000, *1998 National Drug Strategy Household Survey: State and Territory results*, Drug Statistics Series No. 5, Australian Institute of Health and Welfare, Canberra.
- Fullerton, L. & Olson, L. 1995, Occupational injury mortality in New Mexico, *Annals of Emergency Medicine*, vol. 26, pp. 447–454.
- Gentilello, L.M. & Donovan, D.M. 1995, Alcohol interventions in trauma centres: current practice and future directions, *Journal of the American Medical Association*, vol. 274, pp. 1043–1048.
- Gentilello, L.M. & Rivara, F.P. 1999, Alcohol interventions in a trauma centre as a means of reducing the risk of injury recurrence, *Annals of Surgery*, vol. 230, pp. 273–283.
- Hunter, C., Lokan, R., Longo, M., White, J. & White, M. 1998, *The prevalence and role of alcohol, cannabinoids, benzodiazepines and stimulants in non-fatal crashes*, Forensic Science, Department for Administrative and Information Services, Adelaide.
- Kruger, H. & Berghaus, G. 1995, Behavioural Effects of Alcohol and Cannabis: Can Equipotencies be Established? Proceedings of the 13th International Conference on Alcohol, Drugs and Traffic Safety, The International Council on Alcohol, Drugs and Traffic Safety, Adelaide.

- Potter, J. 2000, *Drugs and Driving in Australia*, Austroads Incorporated, Sydney.
- Schermer, C.R. & Wisner, D.H. 1999, Methamphetamine use in trauma patients: a population based study, *Journal of American College of Surgeons*, vol. 189, pp. 442–449.
- Soderstrom, C.A., 2001, Injury in America. The role of alcohol and other drugs – an EAST position paper prepared by the Injury Control and Violence Prevention Committee, *Journal of Trauma*, vol. 50, pp. 1–12.
- Sugrue, M. & Seger, M. 1995, Evaluation of the prevalence of drug and alcohol abuse in motor vehicle trauma in South Western Sydney, *Australian and New Zealand Journal of Surgery*, vol. 65, pp. 853–856.
- Swann, P. 1999, *The Real Risk of Being Killed When Driving Whilst Impaired by Cannabis*, VicRoads, Road Safety Department, Melbourne.
- Williams, A., Peat, M., Crouch, D., Wells, J. & Finkle, B. 1985, Drugs in fatally injured young male drivers, *Public Health Reports*, vol. 100, no.1, pp. 19–25.