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RESEARCH REPORT

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Developing limits for driving under cannabis

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ABSTRACT

Objective Development of a rational and enforceable basis for controlling the impact of cannabis use on traffic safety. **Methods** An international working group of experts on issues related to drug use and traffic safety evaluated evidence from experimental and epidemiological research and discussed potential approaches to developing *per se* limits for cannabis. **Results** In analogy to alcohol, finite (non-zero) *per se* limits for delta-9-tetrahydrocannabinol (THC) in blood appear to be the most effective approach to separating drivers who are impaired by cannabis use from those who are no longer under the influence. Limited epidemiological studies indicate that serum concentrations of THC below 10 ng/ml are not associated with an elevated accident risk. A comparison of meta-analyses of experimental studies on the impairment of driving-relevant skills by alcohol or cannabis suggests that a THC concentration in the serum of 7–10 ng/ml is correlated with an impairment comparable to that caused by a blood alcohol concentration (BAC) of 0.05%. Thus, a suitable numerical limit for THC in serum may fall in that range. **Conclusions** This analysis offers an empirical basis for a per se limit for THC that allows identification of drivers impaired by cannabis. The limited epidemiological data render this limit preliminary.

Keywords Accident risk, adverse effect, cannabis, driving, drug, DUIC, DUID, limit, marijuana, Psychomotor impairment.

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INTRODUCTION

The rising prevalence of driving under the influence of illegal and medicinal drugs (DUID) and its potential impact on traffic safety have raised awareness among media, scientists and policy makers in many countries and prompted calls for more effective control. Driving under the influence of cannabis (DUIC) is of particular concern, because the recreational use of cannabis products, i.e. marijuana and hashish, is often second only to alcohol. This highlights the need for effective legal control of the potential risks posed by DUIC.

Current approaches to assessment and control of DUIC

Current DUID laws use one of three basic approaches to determine whether a driver involved in an accident or stopped at a roadside checkpoint, is impaired or under the influence of a particular drug. One is the traditional impairment or effect-based approach; the others are two versions of the '*per se*' approach. The *per se* approach uses, as in the case of alcohol, a science-based finite limit or employs a zero limit for the tolerable concentration of a drug or its metabolites in a driver's blood or other body fluids. In either case, exceedance of this limit is deemed automatically to prove (legal) impairment.

In theory, the impairment approach best meets the objectives of DUID laws. It observes and assesses the fitness of drivers and potentially penalizes those who are actually impaired. Impairment may arise from several, often-synergistic factors, including fatigue and the consumption of multiple drugs. The main limitation of the impairment approach is the lack of standardized methods for measuring and judging the impairment caused by drug consumption. Standardized sobriety tests are sensitive and reliable when used by trained officers to detect blood alcohol contents of more than 0.1%. These tests

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also detect drug-induced impairment reliably, particularly with drugs that depress the central nervous system. However, sobriety tests for drugs are less sensitive to modest impairment [1,2]. Procedures for handling drivers suspected of drug use are often not standardized. This renders the assessment of their impairment somewhat arbitrary. Legal disputes are thus common with DUID cases and make the enforcement of impairment-based laws costly.

Because of these shortcomings of the impairment-based approach, many jurisdictions have adopted *per se* limits for DUID. Many of them have been set at the limit of detection and are *de facto* zero limits. This avoids the need for a reliable science-based correlation between drug concentration and level of impairment and facilitates enforcement. However, zero limits by design penalize the presence in body fluids of an active drug ingredient or its metabolites, which does not necessarily correspond to actual impairment. This is a particular concern with cannabis. Its main psychoactive constituent, delta-9-tetrahydrocannabinol (THC), is detectable in blood for up to 2 days. Depending on the frequency of use, its metabolites are detectable in blood and urine for days or weeks after cannabis use. In contrast, even a high dose of smoked THC typically causes acute impairment of driving skills for only 3–4 hours. The slow disappearance of THC from serum is particularly pronounced with heavy users, who consume more than one marijuana cigarette (joint) per day, or even with moderate users of cannabis. Their blood may contain THC concentrations of between 1.0 and 6.4 ng/ml serum even 24–48 hours after smoking the last joint [3]. Thus, blood samples taken from moderate users may still test positive for THC even when they observe a sufficiently long waiting period between cannabis use and driving and impairment has dissipated. Heavy passive exposure to cannabis smoke may also result in measurable THC concentrations in blood serum without causing concurrent impairment [4–6].

There are several potential indicators of cannabis use and its potential impact on driving skills. Because of its good correlation with measured impairment during the later phase of a cannabis high, i.e. more than 2 hours after cannabis consumption, the concentration of THC in blood is still the most meaningful indicator of impairment during that period [7]. Note that during the first hour of a cannabis 'high' no unimodal relationship between impairment and THC concentration exists. However, during this phase, THC concentrations in blood clearly exceed the range considered by the authors for a legal limit. Thus, drivers under the acute impact of cannabis and presenting with THC concentrations in the serum of 20 ng/ml or more would invariably be found in violation.

Commercially available less invasive alternatives to measuring THC concentration in blood, such as testing urine for THC metabolites or analysing hair and sweat samples, suffer from long detection windows and/or poor reproducibility and do not qualify as the sole method for determining cannabis-induced impairment. THC concentrations in saliva appear to correlate reasonably well with THC concentrations in blood, and saliva testing may emerge as a non-invasive screening test for the use of cannabis and other drugs in road checks, to be confirmed by blood analysis.

As is commonly conducted with alcohol, *per se* laws for DUIC may adopt a set of two legal limits for the concentration of THC in blood. These limits will reflect varying degrees of impairment and corresponding risk and translate into varying levels of punishment and the intended educational effect. Violation of the lower limit would result in a fine and a temporary suspension of driving privileges, intended to warn the driver to separate drug use and driving. For example, several European countries have set a lower blood alcohol concentration (BAC) limit at 0.05%. Exceeding the higher limit above which most people will be unfit to drive would result in a higher fine, extended revocation of the driver's licence and, depending on the circumstances, criminal prosecution. Several European countries have adopted such a higher BAC of 0.11%.

Approach and objectives of study

This paper summarizes the findings and recommendations by an interdisciplinary working group of international scientists, convened in 2004/05. Its objectives were to conduct a comprehensive review and discussion of scientific evidence on DUIC from experimental and epidemiological studies and to propose a numerical range for a *per se* limit for THC concentrations in blood, which may serve as indicator of cannabis-induced impairment. Selection of the limit was also to consider physiological, toxicological and analytical factors that may modify the correlation between blood THC concentration and the impairment of a driver.

SCIENTIFIC BASIS FOR A LEGAL THC LIMIT

Epidemiological and experimental studies are the two main sources of evidence on the potential impact of cannabis use on driving skills and accident risk.

Epidemiological studies

Findings from epidemiological studies have historically been the basis for *per se* limits for alcohol and driving. These studies examine the statistical association between rare events (traffic crashes, injury or death) and a risk factor, such as the consumption of alcohol or a drug, and the corresponding indicators, such as the BAC. Using a

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case–control or culpability approach, epidemiology assesses the actual risk of a drugged driver causing an accident, relative to that of a sober person driving under similar road conditions. That relative risk is expressed as odds ratio (OR). An OR greater than 1 corresponds to a higher accident risk for the 'case group', i.e. drivers under the influence of a drug, compared to the 'control group'.

Epidemiological studies measure the effect of drug use on driving performance and accident risk under 'real life' conditions and are thus suited to correlate the concentrations of a drug use indicator to an actual risk. For alcohol, scientists have developed, based on the results of numerous epidemiological studies, hazard curves that assign each alcohol concentration to a certain accident risk. As with all epidemiological findings, the validity of each study depends critically on the number of cases included. Driving under the influence of alcohol is a widespread phenomenon and screening of drivers for alcohol using breath analysers is non-invasive. This allowed researchers to collect, for a given time of day, region, road condition and for each BAC class enough cases to yield statistically significant ORs.

Fortunately for traffic safety but unfortunately for epidemiological research DUIC is far less common. Furthermore, meaningful testing for cannabis use requires the collection of blood samples, a procedure that in most countries cannot be used unless a driver is suspected of DUI. Thus, epidemiological studies on DUIC do not usually have sufficient THC positive cases to calculate reliably concentration-dependent ORs.

Detailed overviews of epidemiological studies on DUIC have been provided by Bates & Blakely [8], Chesher & Longo [9], Ogden & Moskowitz [10] and Ramaekers *et al.* [11]. Drummer *et al.* conducted one of only few epidemiological studies that correlated THC concentrations in blood and accident risk and met quality criteria not met by other such studies [12]. The study used accident data from drivers fatally injured in accidents in Australia and found that THC concentrations in whole blood in the range of 0–5 ng/ml were associated with an OR of 0.7 and concentrations between 5 and 100 ng/ml with an OR of 6.6 (95% CI: 1.5–28). Note that both ORs represent an average for the entire respective range of THC concentrations, so the average OR for a driver with a THC concentration in blood of anywhere between 5 and 100 ng/ml is 6.6. Because OR and blood THC concentration are probably correlated by a linear or even exponential function, the point risk at 5 ng/ml THC in whole blood is considerably much lower than 6.6.

To differentiate more clearly the correlation between OR and THC concentration in the 0–20 ng/ml range G. Berghaus and G. Sticht (personal communication) developed the data by Drummer *et al.* into a polynomial function. The results in Fig. 1 show that THC concentrations

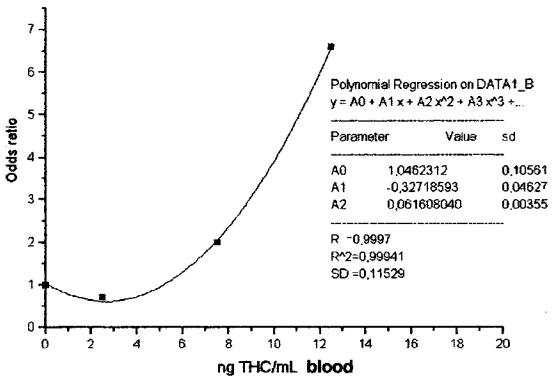

Figure 1 Correlation between delta-9-tetrahydrocannabinol (THC) concentration in whole blood and accident risk (odds ratio) calculated with the data of the study by Drummer *et al.* [12]

in blood are not associated with an elevated risk (OR > 1) until they exceed about 6 ng/ml.

Comparison of these cannabis-induced risks to those associated with driving under the influence of alcohol yields a first approximation to a numerical *per se* limit for DUIC. A BAC of 0.05% alcohol is associated with an OR of about 1.5–2 [13–15]. According to Fig. 1, that range corresponds to a THC concentration in whole blood of about 6–8 ng/ml, equivalent to a THC concentration in serum of about 12–16 ng/ml. The latter assumes a typical conversion factor of 2 between THC concentrations measured in blood versus serum.

As the study by Drummer *et al.* was based on only 58 cases whose blood samples contained only THC and no other indicators of drugs, the above considerations do not yield a statistically acceptable basis for an enforceable *per se* limit. The latter would require epidemiological data from a far larger number of cases.

A more recent epidemiological study, conducted in France by Laumon *et al.* [15], evaluated a much larger sample of THC-positive drivers ($n = 681$) who were involved in fatal accidents. Of them, 285 also tested positive for alcohol with a BAC > 0.05%. The adjusted OR (adjustment for alcohol, driver's age, type of vehicle and time of crash) for all THC positive cases was 1.78 (95% CI: 1.40–2.25), with the OR of cases with THC concentrations in blood of less than 1 ng/ml being 1.57 (95% CI: 0.84–2.95) and the OR of the subgroup with the highest THC concentrations (≥ 5 ng/ml whole blood) being only slightly higher (OR = 2.12, 95% CI: 1.32–3.38). The overall OR of 1.78 reported by Laumon *et al.* [15] is similar to that found by Drummer *et al.* [12] (OR = 2.7, 95% CI: 1.02–7.0), and in line with other studies that found only a small overall increase of accident risk in THC positive drivers, e.g. Terhune [16] (OR = 2.1), or even no increase, e.g. Longo *et al.* [17] (OR = 0.9). However, the findings by Laumon *et al.* [15] contradict those from all

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other experimental and epidemiological studies in that they suggest an increased risk for THC blood concentrations below 1 ng/ml and only a slightly higher risk for blood concentrations above 5 ng/ml. A possible explanation for the weak dose–effect relationship is that many of the blood samples were collected 3 or 4 hours after the accident. Delayed sample collection causes a decrease in THC concentration, artificially inflates the calculated accident risk for a given THC concentration and blurs the differences between THC concentration classes. The study also suffered from other flaws, such as the classification of concurrent low concentrations of alcohol as 'null BAC', all of which reduces the value of the obtained data and the study's conclusions.

Overall, current epidemiological evidence on the effects of cannabis on accident risk is much less conclusive than for alcohol and must be considered insufficient for deriving a science-based legal limit for THC in blood. However, it suggests that the presence of THC as the sole drug in whole blood at concentrations above some 5 ng/ml correlates with a gradually increasing accident risk.

Experimental studies on impairment by cannabis

With inadequate epidemiological evidence, the extensive body of experimental research on cannabis use and driving skills may offer a second line of evidence and an alternate approach to deriving *per se* limits for THC. To date, some 150 experimental studies have tested the impact of cannabis use on skills that are essential to driving performance under laboratory conditions, in driving simulators and under road conditions. Most of these studies tested participants who had smoked or ingested a known dose of THC for significant impairment of one or several relevant skills. A typical result of such a test would read: a group of drivers who consumed a specific dose of a drug performed 'significantly worse' on a specific test compared to the performance of a control group who had not taken the drug.

Smiley reviewed driving simulator and on-road studies, which had examined the impact of THC on driving, and compared the latter to the effects by alcohol [18]. In summary, simulator and on-road studies showed that cannabis may impair some driving skills at smoked THC doses of as low as 6.25 mg. However, results varied considerably between the skills tested and among studies, and some of the skills tested were not impaired at doses as high as 18 mg. The impairment caused by cannabis appeared to be partially mitigated because subjects were aware of their impairment and, where possible, tended to compensate by not overtaking, by slowing down and by focusing attention in anticipation of a required response. Such compensation is not always possible in response to an unexpected event. In blind ratings, police officers rated drivers with a BAC of 0.08% as more impaired than those who had taken moderate to high doses of cannabis, and driving instructors rated subjects with a BAC of 0.04% as impaired, while those who had consumed a dose equivalent to 7 mg THC were rated as unimpaired.

Meta-analysis and comparison with alcohol

Findings from experimental studies may vary considerably because the outcome of a particular study is largely a function of study design and the choice of critical parameters, such as drug dose, smoking versus eating, time lapsed between drug use and testing and type and severity of tests during on-road driving. The apparent variability is best addressed through a meta-analysis of experimental studies. Scientists commonly perform meta-analyses of published research on a particular subject to evaluate and compare the results from a multitude of studies that meet a set of minimum quality criteria. Key results from the analysed studies are coded, compiled and analysed statistically. If a sufficiently large number of studies meet these entrance criteria, the meta-analytical approach strengthens the significance of findings from individual experimental studies.

A meta-analysis of a sufficiently large number of compliant experimental studies on cannabis and driving skills balances the variability in key design parameters. It also allows for a comparison with results from a meta-analysis of experimental studies on the impact of alcohol on driving skills, for which risk-based *per se* limits for BAC are well established. Such comparison will suggest a range of THC concentrations in blood from which a *per se* limit for DUIC may be selected.

The following factors support further the rationale for this approach. First, experimental studies on the effects of cannabis and other drugs on driving skills use the same methods, equipment and procedures as those for alcohol, i.e. laboratory tests of isolated skills, driving in simulators and on-road driving. They also use the same statistical methods to process data and report results. Secondly, most studies report information on cannabis dose, mode of application (smoked versus oral) and the time lapsed between consumption and test [19,20]. Using a pharmacokinetic model one can then estimate the THC concentration in blood at the time of testing. THC concentrations in blood show a considerable intra- and interindividual variation after consumption of the same dose [7,21] and the modelling results use mean concentrations. Thirdly, the large number of epidemiological studies on alcohol and driving has produced a strong correlation between BAC and accident risk and jurisdictions world-wide now typically use BAC concentrations of between 0.05 and 0.11% as indicators of various degrees of impairment by

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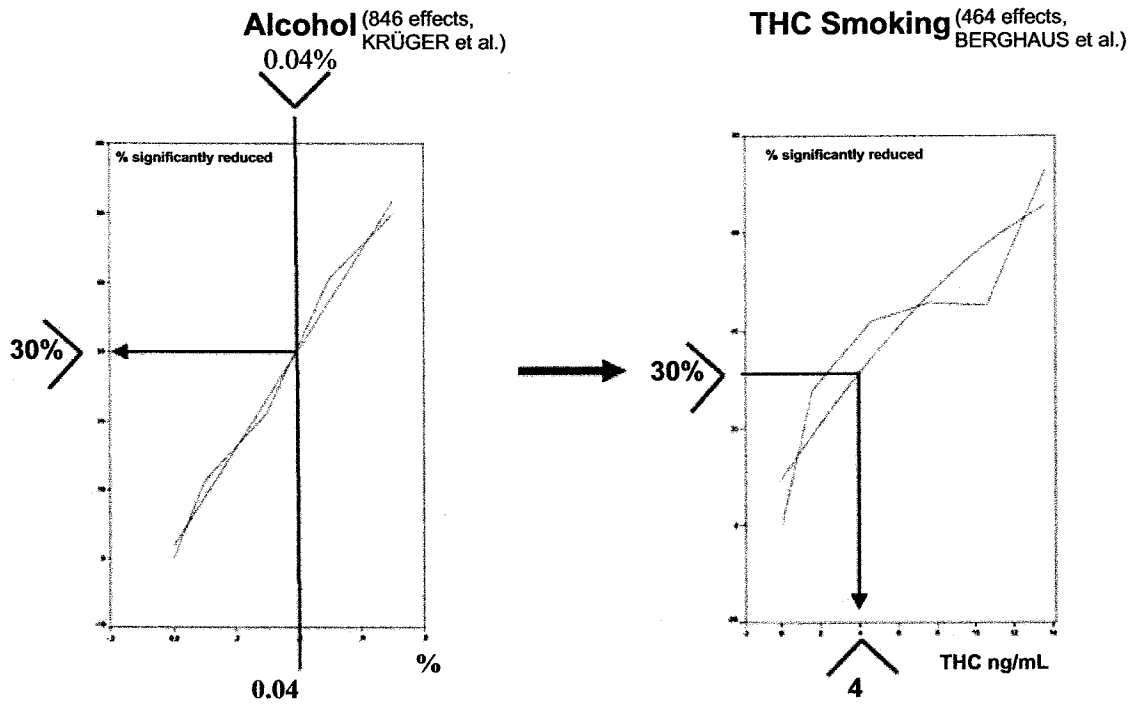

Figure 2 Comparison of survival functions for delta-9-tetrahydrocannabinol (THC) (in serum) and alcohol (in whole blood) and establishment of points of equal impairment. One curve represents the data really measured (unequal curve), the other curve represents the linear (blood alcohol concentration) or exponential (THC) smoothing

alcohol. A major shortcoming of this approach is its failure to consider whether the influence of alcohol and cannabis, respectively, promote different adaptive behaviors that may modify accident risk under actual road conditions [18]. Another limitation of this meta-analysis, as described below, is that it included test results for indicators with no clear link to driving performance, such as flicker fusion.

Within these limitations, a comparison of results from meta-analyses for alcohol and THC, respectively, then generates, for a given THC blood concentration, the corresponding BAC that causes the same level of impairment in test skills and for which accident risk is well established. For example, one may regard the THC concentration in blood at which the same percentage of all test results shows impairment as with a BAC of 0.05% as the THC concentration equivalent to that BAC.

The working groups of Krüger and of Berghaus conducted, in the 1990s, meta-analyses of suitable experimental studies on the effects of low doses of alcohol and cannabis [19,22,23]. Their work allowed a first systematic and quantitative comparison of the results of experimental research on the effects of THC and alcohol, respectively. For their meta-analysis of experimental studies on cannabis, Berghaus *et al.* first selected, out of more than 120 studies, those published in English or German and meeting the following minimum criteria: testing of at least one driving-relevant skill, a minimum of five human participants per study, information given on THC dose and mode of administration; number, age and gender of subjects; time delay between consumption and testing; type of test performed (e.g. tracking, visual function), and the specific tasks (e.g. two-hand-coordination, flicker fusion). Test results had to be coded as 'significant improvement or impairment', at least at the 5% level or as 'no significant change' [19].

Studies in which THC had been taken together with other drugs or alcohol were excluded. Overall, 66 studies in which cannabis had been smoked and 21 with oral intake of cannabis were selected, including laboratory tests, driving simulator and on-road studies. Blood THC concentrations at the time of testing were estimated from the information on THC dose and other factors using the pharmacokinetic model by Sticht & Käferstein [21].

Figure 2 summarizes the key results from the two meta-analyses. For alcohol and smoked cannabis, respectively, each graph shows a set of two 'survival functions'. The respective curves give the percentage of results from all tests that showed significant impairment at a given BAC or THC concentration in serum. One curve represents the original data; the other curve shows the results of linear (BAC) or exponential (THC) smoothing. Comparison of the two graphs thus suggests that a BAC of 0.04% and a serum THC concentration of 4–5 ng/ml

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both impair driving related skills by about 30%. Thus, a lower legal limit for the concentration of THC in serum that produces the same level of impairment and possibly accident risk as a BAC of 0.05% would be somewhere above 4–5 ng/ml of serum. Note that the correlation between THC serum concentrations and impairment did not depend on the route of administration of cannabis (inhalation, oral ingestion).

The above comparison lumps together the results from tests for a range of driving-related skills, including automatic and controlled functions. A closer analysis of the respective impact of alcohol and THC on these functions suggests that across the BAC range of 0.05–0.11% an increase in BAC further impairs automatic and controlled functions equally (data not shown). In contrast, an increase in serum THC concentrations beyond 5 ng/ml further impairs automatic functions while performance of tasks requiring cognitive control remains stable up to concentrations of 10 ng/ml [20]. This supports the above-mentioned observations from driving studies that drivers under the influence of cannabis may compensate consciously for some of the impairment of their automatic performance, for example by reducing speed or keeping more distance.

These meta-analytical data are in good agreement with the results of a recent experimental study on the relationship between THC concentrations in serum after smoking cannabis and impairment [24]. First signs of impairment were found at THC serum concentrations in the range of between 2 and 5 ng/ml. This degree of impairment may correspond to the impairment at a BAC of 0.03%, where the impairment by alcohol becomes significant. Because the observation pertains to the entire THC concentration range of between 2 and 5 ng/ml, impairment may have started at a THC concentration between 3 and 4 ng/ml serum.

PROPOSAL FOR A *PER SE* LIMIT FOR DUIC

In summary, current evidence from scientific studies offers the following conclusions on the correlation between THC concentrations in blood and cannabis-induced potential impairment of driving performance. Evidence from the few meaningful epidemiological studies on cannabis use and driving is insufficient for deriving a risk-based *per se* limit for DUIC. While based on too few cases of drivers who had used cannabis and not other drugs, the study by Drummer *et al.* suggests that a serum THC concentration of 12–16 ng/ml may correspond to the same accident risk as a BAC of 0.05% [12]. More culpability studies using a larger number of cases, considering non-fatally injured drivers and conducting accurate and timed measurement of blood THC concentrations are needed for a reliable determination of the accident risks associated with different THC blood concentrations.

Alternatively, experimental studies offer a preliminary basis for *per se* limits for DUIC.

Specifically, the results from a comparison of two meta-analyses on alcohol and cannabis, respectively, suggest that a BAC of 0.04% and a serum THC concentration of 4.2 ng/ml cause comparable impairment of driving-related skills.

When using this equivalency as the basis for a *per se* limit, two areas of uncertainty must be considered. First, for a given time-lapse between smoking and blood testing, the correlation between a smoked THC dose of THC and the resulting THC blood concentration shows considerable inter- and intra-individual variability. According to the pharmacokinetic model of Sticht & Käferstein, which was used in the above meta-analysis to estimate THC blood concentrations, a male weighing 70 kg and smoking a THC dose of 19 mg will, after 3 hours, present with a serum concentration of 4.9 ng/ml with a confidence interval of 3.1–7.7 ng/ml [21]. To minimize false positive test results among drivers with THC concentrations at the upper end of this range without being impaired, a risk-based lower serum limit of 7 ng/ml, rather than 4.2 ng/ml, is thus suggested.

Secondly, enforceable legal limits for DUI must consider the effects of analytical errors made during blood analysis. For example, in Germany, the lower legal BAC limit of 0.05% includes the risk-based limit of 0.04% plus a safety margin for analytical errors of 0.01%. That margin is based on interlaboratory proficiency tests and reflects typical variability. Similar proficiency tests conducted for THC have shown a much larger variation. A recent comparison by the German Society of Toxicological and Forensic Chemistry suggested a suitable safety margin for THC of 3.4 ng/ml [25]. Adding such a safety margin yields a lower THC limit of 7–8 ng/ml in serum (4.2 + 3.4), or 3.5–4 ng/ml THC in whole blood, which corresponds to a lower BAC limit of 0.05%. Other countries, including Australia, ask laboratories to assess accuracy of their measurements and to consider it when comparing results to a legal limit. This allows for differences between laboratories with regard to analytical accuracy. For example, a laboratory with a documented internal accuracy of ±2.5 ng/ml for THC in serum at the measured concentration would report samples exceeding 6.7 ng/ml as in violation of a 4.2 ng/ml *per se* limit. Combining these two correction factors would render serum THC concentrations in the range of between 7 and 10 ng/ml (3.5–5 ng/ml in whole blood) equally impairing as a BAC of 0.05% and suggest that range for the selection of a lower legal limit based on the meta-analysis of experimental studies.

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Other modifying factors

Three other potentially modifying factors must be considered when setting legally binding numerical *per se* limits for THC. First, the epidemiological study by Drummer *et al.* suggests that THC concentrations indicate elevated accident risk at levels higher than indicated by experimental studies [12]. This may be due to the more pronounced adaptive behaviours (slowing down, reduced risk-taking) observed with cannabis-affected drivers in driving simulator and on-road studies, both of which represent more closely real-life conductions. In that case, comparison of experimental studies for alcohol and THC, respectively, would result in systematically lower *per se* limits for THC than derived from epidemiological studies.

Secondly, cannabis consumption produces measurable THC residues in blood long after smoking. At 10 hours after smoking residual THC concentrations in the serum of occasional or even frequent users have declined to typically less than 5 ng/ml. The suggested *per se* limit in the range of 7–10 ng/ml safely avoids misclassification of drivers presenting with THC residues from previous cannabis use. It would also spare drivers with low but measurable THC concentrations caused by passive exposure to cannabis smoke or by smoking or oral intake of low THC doses for medicinal purposes [26–31].

Finally, a legal *per se* limit for cannabis must consider that the concurrent use of alcohol and cannabis impairs driving skills more than each drug individually [32]. For drivers presenting with measurable THC concentrations and a BAC exceeding 0.03% or 0.05%, a lower *per se* limit for THC than proposed above may be appropriate.

Using current scientific evidence on cannabis-induced impairment of psychomotor skills and the related accident risk, this paper suggests a range of 7–10 ng/ml THC in the serum for an initial non-zero *per se* limit. It offers reasonably reliable separation of drivers whose driving is in fact impaired by cannabis from those who are not impaired. Inadequate evidence from epidemiological studies renders this limit preliminary and suggests the need for review and possibly revision in the future. Our findings also suggest that using a zero limit for legal determination of impairment by cannabis, which in practice corresponds to the limit of detection for THC in blood, would classify inaccurately many drivers as driving under the influence of, and being impaired by, the use of cannabis.

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SB 212

Patients & Families United
Post Office Box 1471
Helena, MT 59624

EXCERPTS from:

STATE OF THE KNOWLEDGE OF DRUG IMPAIRED DRIVING

U.S. Department of Transportation, HS 809 642

September 2003

(emphases added)

BLOOD TESTING

"Due to the invasiveness of the collection procedure and the cost of laboratory analysis, routine screening of blood for drugs in drivers has generally been viewed as impractical. Augsburger (2002) recommends a three-step laboratory-analysis process for determining the effect of drugs on driving performance. However, in recent years, forensic laboratories have seen an increasing number of specimens for determination of drugs in blood as a result of "zero tolerance" laws and better trained police officers who have been trained to recognized drivers under the influence of drugs (Moeller and Kraemer, 2002). This is especially true in Europe where several European countries (e.g., Sweden, Germany, and Belgium) have enacted per se laws for driving under the influence of drugs. These laws stipulate urinalysis as the preliminary screening test, and require a blood test if the urine is positive for drugs. Under these laws, any level of prohibited drug detected in the blood is considered evidence of driving under the influence.

"In terms of attempting to link drug concentrations to behavioral impairment, blood is probably the specimen of choice. However, forensic toxicologists generally have failed to agree on specific plasma concentrations that could be designated as evidence of impairment (Consensus Development Panel, No Date). The lack of consensus about per se levels of drugs where impairment could be deemed makes it difficult to identify, prosecute or convict drugged drivers in most states."

DRUG-CRASH RISK

"Two U.S. studies were found that conducted a formal assessment of drug-crash risk. The first study (Terhune, Ippolito, Hendricks et al., 1992) used the responsibility-analysis approach (4) and found that **no increased crash risk was associated with marijuana or cocaine alone**, but that multiple drug use may be associated with increased responsibility.

"Drummer (1995) used data from some 1,000 fatal crashes Victoria, New South Wales, and Western Australia to develop fatal-crash risk factors for several drugs. Again, the

responsibility analysis approach was used. Drummer computed odds ratios for drugs / no-drugs for each drug and found that only alcohol gave a statistically significant odds ratio greater than one (odds ratio=7.6, $p < 0.001$). The odds ratio for cannabis approached significance ($p=0.065$) and was actually less than one (0.60), **suggesting a beneficial effect of marijuana use.**

"The study by Longo, Hunter, Lokan et al. (2000) cited earlier in this report analyzed the causal role of alcohol, cannabinoids, benzodiazepines and stimulants in crashes involving 2,500 injured Australian drivers. The responsibility analysis approach also was used in the analysis. Benzodiazepine use was associated with higher culpability when those with very low concentrations were excluded (percentage ratio 3), **but THC was not associated with increased culpability**. Relatively few drivers tested positive for stimulants and there was no clear evidence of greater culpability."

EPIDEMIOLOGICAL RESEARCH

"In sum, recent epidemiologic research indicates that:

* A significant amount of new information has been added to the pool of knowledge about the role of several classes of drugs in traffic crashes since the last state of knowledge update. However, gaps still exist on certain drug classes that are in widespread use, for example, antihistamines and antidepressants.

* Of the drugs appearing in epidemiologic studies of U.S. driver populations, marijuana has been found the most often by a wide margin. This should not be surprising, given the findings of the 2001 national household survey on drug abuse that 76% of current users of illicit drugs were users of marijuana (U.S. Department of Health and Human Services, 2002).

* For drugs that have been studied, the percentage of drug-positive drivers in crashes is much lower than the percentage of alcohol-positive drivers in crashes, but still not negligible.

*** The role of drugs as a causal factor in traffic crashes involving drug-positive drivers is still not understood**. Drug risk factors are still not known with acceptable precision, with some evidence suggesting little or no increase in crash risk at drug levels being detected by current chemical test procedures. **Further, current research does not enable one to predict whether a driver testing positive for a drug, even at some measured level of concentration, was actually impaired by that drug at the time of crash.** This is in sharp contrast to alcohol where BAC measurements can provide a good estimate of impairment."

DETECTION AND MEASUREMENT OF DRUGS IN DRIVERS

"Conclusions

* A variety of specimens can be assayed for drugs, including urine, blood, sweat, saliva, and hair, among others. Each specimen is unique, and each offers different patterns of information about drug use over time (see reference).

* Most laboratories use immunoassay screening technology with gas chromatography-mass spectrometry (GC/MS) confirmation. Over the last 20 years the cost of using these technologies have become affordable, and most laboratories now have the equipment, the assays, and the expertise to identify the most commonly used drugs (see reference).

* While there have been significant improvements in laboratory assays for drugs of abuse, the value of such improvements to highway safety specifically is limited by an insufficient number of laboratories incorporating these improvements.

*** The reliance solely on the forensic laboratory to assay all specimens in all cases limits the number drug-impaired driving cases that can be prosecuted, because there are simply not enough forensic resources currently available.**

* Point-of-contact-testing (POCT) devices offer promise for alleviating this problem. For example, these POCT devices could be used by police officers to routinely screen DUI suspects for illegal drug use and obtain drug test results immediately, as they currently do with alcohol tests (see reference).

*** Until there is adequate capability for rapid, cost-effective drug testing, many drugged drivers will not be identified or prosecuted."**

SB 212

Patients & Families United
Post Office Box 1471
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Types of Common Legal Medications Known to Potentially Impair Driving Even When Used as Directed*

* Anti-anxiety medication
* Amphetamines
* Barbiturates
* Stimulants
* Narcotic pain medications
* Allergy medicines
* Blood sugar medicines
* Antidepressants
* Tranquilizers
* Blood pressure medicines
* Motion sickness medication
* Ulcer medication
* Antibiotics
* Anti-seizure medicines
* Paregoric
* Anti-nausea medicine
* Sedatives
* Cough syrups
* Alcohol-containing medicines
* Caffeine-containing medicines
* Decongestants

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Written by: Larry Axmaker, EdD, PhD
Date Published: November 21,2001 Date Reviewed: November 14,2007

SB212

Edwin L. Stickney M.D.
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TO: Members of the Montana Senate Judiciary Committee

RE: SB 212

As a licensed Montana physician and past president of the Montana Medical Association, I write to explain my strong opposition to SB 212. I urge a "do not pass" recommendation for this proposal.

I wish I could attend your hearing to comment and answer questions in person, but my schedule interferes. However, I want all committee members to know that I would welcome the opportunity to talk with you about this should you wish to before taking action on SB 212. Please feel free to call me at the phone number listed above.

SB 212 seems punitive in nature – severely so – and ultimately makes no scientific sense to me for reasons I'll outline here.

The bill singles out only one legal drug – medical marijuana – that is known to impair driving skills under certain circumstances. But singling out medical marijuana alone makes no sense to me in light of the fact that only about 1,600 Montanans are licensed to use it, in contrast to the many tens of thousands of Montanans known to be using other legal drugs, many of which are far more prone to interfere with driving skills than is medical marijuana.

A long list of prescription drugs, including pain relievers, mood stabilizers and others, can cause severe impairment of driving skills, much more certainly than medical marijuana can – yet SB 212 addresses none of these far more common risks on our roads and highways.

Further, patients who need marijuana as medicine generally suffer severe conditions. In my experience working with such patients, many are unable to drive at any time because of their medical circumstances alone, and not due to their use of marijuana. In addition, it is well understood that patients who use medical marijuana on a regular basis develop physical tolerances to the drug such that its impairment effects on them are much lower than they would be in persons who consume marijuana only occasionally.

One of the most important points related to these themes concerns the longevity of medical marijuana's constituents in the urine and blood. The bill focuses only on THC (tetrahydracannibinol), which is one of more than 60 active cannabinoids known to collectively be responsible for marijuana's benefits as medicine; and, indeed, where medicinal effects are concerned, THC also is believed by scientific researchers to be relatively less significant than other cannabinoids, especially cannabidiol.

406-896-1100 estickney2@bresnan.net

What's important for you to understand when evaluating SB 212 is that the THC blood-limits it specifies are so low that most any medical marijuana patient would fail the blood test – even if those patients were not under the influence of marijuana and hadn't consumed any for several days. A great many patients use marijuana in the evening, before sleeping, to control pain and muscle spasms and to allow a fuller, more beneficial sleep. The following morning or afternoon – long after the effects of marijuana would have any influence whatsoever on their ability to drive – these patients would still fail the blood test required in SB 212.

Perhaps most objectionable, SB 212 proposes an unprecedented penalty that in my judgment is immoral. It would ban a patient from using medical marijuana again – forever – merely for failing a blood test that isn't demonstrative of driving impairment.

This penalty is unacceptable in my judgment.

It already is illegal to drive under the influence of medical marijuana or any other potentially debilitating drug, including alcohol. And current penalties for violating these requirements – loss of driving privileges for a period of time, for example – are entirely appropriate, because they connect directly to the offense.

But banning a sick person from ever again using a medicine that his or her physician recommends, without regard to the person's present medical condition or future prospective condition, strikes me as ridiculous on its face.

I urge you to oppose SB 212 – and to feel free to call me if you have questions or concerns about my comments.

Thank-you for your consideration.

Sincerely,

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SB212

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NORML

Working to Reform Marijuana Laws

Cannabis and Driving:
A Scientific and Rational Review

By Paul Armentano
Deputy Director
NORML | NORML Foundation
E-mail: paul@norml.org
January 10, 2008

NORML

Working to Reform Marijuana Laws

Policy debates regarding marijuana law reform invariably raise the question: "How does society address concerns regarding pot use and driving?" The subject is worthy of serious discussion. NORML's Board of Directors addressed this issue by ratifying a "no driving" clause to the organization's "Principles of Responsible Cannabis Use"[1] stating, "Although cannabis is said by most experts to be safer with motorists than alcohol and many prescription drugs, responsible cannabis consumers never operate motor vehicles in an impaired condition."

Nevertheless, questions remain regarding the degree to which smoking cannabis impairs actual driving performance. Unlike alcohol, which is known to increase drivers' risk-taking behavior and is a primary contributor in on-road accidents, marijuana's impact on psychomotor skills is subtle and its real-world impact in automobile crashes is conflicting.

Drugged Driving: True Threat Or False Panic?

Survey data indicates that approximately 112 million Americans (46 percent of the US population) have experimented with the use of illicit substances.[2] Of these, more than 20 million (8.3 percent of the population) self-identify as "current" or "monthly" users of illicit drugs,[3] and more than 10 million Americans say that they've operated a motor vehicle while under the influence of an illicit substance in the past year.[4] These totals, while far from negligible, suggest that the prevalence of illicit drug use among US drivers is far less than the prevalence of alcohol among this same population.[5]

To date, "[The] role of drugs as a causal factor in traffic crashes involving drug-positive drivers is still not well understood."[6] While some studies have indicated that illicit drug use is associated with an increased risk of accident, a relationship has not been established regarding the use of psychoactive substances and crash severity.[7] Drivers under the influence of illicit drugs do experience an enhanced fatality risk compared to sober drivers. However, this risk is approximately

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[1] Adopted by NORML's Board of Directors, February 3, 1996. Read all of NORML's "Principles of Responsible Use" online at <http://www.norml.org/index.cfm?Group_ID=3417>

[2] US Department of Justice, Bureau of Justice Statistics. *Drug and Crime Facts: Drug Use Among the General Population*. Online document accessed November 24, 2007. <http://www.ojp.usdoj.gov/bjs/dcf/du.htm>

[3] US Department of Health and Human Services, Substance and Mental Health Services Association, Office of Applied Studies. *2006 National Survey on Drug Use and Health: National Results*. Online document accessed November 24, 2007. <http://www.oas.samhsa.gov/nsduh/2k6nsduh/2k6Results.cfm#Fig2-1>

[4] Ibid.

[5] US Department of Transportation, National Highway Traffic Safety Administration. *State of Knowledge of Drugged Driving: FINAL REPORT*. September 2003. <http://www.nhtsa.dot.gov/people/injury/research/StateofKnwlegeDrugs/StateofKnwlegeDrugs/>

[6] Ibid.

[7] Smink et al. 2005. Drug use and the severity of traffic accident. *Accident, Analysis and Prevention* 37: 427-433.

three times lower than the fatality risk associated with drivers who operate a vehicle above or near the legal limit for alcohol intoxication.[8] According to one recent review: "The risk of all drug-positive drivers compared to drug-free drivers is similar to drivers with a blood alcohol concentration of 0.05%. The risk is also similar to drivers above age 60 compared to younger drivers [around age 35]." [9]

Marijuana is the most common illicit substance consumed by motorists who report driving after drug use.[10] Epidemiological research also indicates that cannabis is the most prevalent illicit drug detected in fatally injured drivers and motor vehicle crash victims.[11] Reasons for this are twofold. One, pot is by far the most widely used illicit drug among the US population, with nearly one out of two Americans admitting having tried it.[12] Two, marijuana is the most readily detectable illicit drug in toxicological tests. Marijuana's primary psychoactive compound, THC, may be detected in blood for several hours, and in some extreme cases days after past use,[13] long after any impairing effects have worn off. In addition, non-psychoactive byproducts of cannabis, known as metabolites, may be detected in the urine of regular users for days or weeks after past use.[14] (Other common drugs of abuse, such as cocaine or methamphetamine, do not possess such long half-lives.) Therefore, pot's prevalence in toxicological evaluations of US drivers does not necessarily indicate that it is a frequent or significant causal factor in auto accidents. Rather, its prevalence affirms that cannabis remains far more popular and is far more easily detectable on drug screening tests than other controlled substances.

Cruising On Cannabis: Clarifying The Debate

While it is well established that alcohol consumption increases accident risk, evidence of marijuana's culpability in on-road driving accidents and injury is far less clear. Although acute cannabis intoxication following smoking has been shown to mildly impair psychomotor skills, this impairment is seldom severe or long lasting.[15] In closed course and driving simulator studies, marijuana's acute effects on psychomotor performance include minor impairments in tracking (eye

[8] Franjo Grotenhermen. *Drugs and Driving: Review for the National Treatment Agency, UK.* Nova-Institut (Germany). November 2007.
[9] Ibid.
[10] US Department of Health and Human Services, Substance and Mental Health Services Association, Office of Applied Studies. *Driving After Drug or Alcohol Use, 1998.* Online document accessed November 24, 2007. <http://www.oas.samhsa.gov/driverrprt/toc.htm>
[11] US Department of Transportation. 2003. op. cit.
[12] October 23-24, 2002 CNN/Time poll conducted by Harris Interactive.
[13] Skopp et al. 2003. Serum cannabinoid levels 24 to 48 hours after cannabis smoking. *Archives of Criminology* (Germany) 212: 83-95.
[14] Paul Cary. 2005. The marijuana detection window: Determining the length of time cannabinoids will remain detectable in urine following smoking. *Drug Court Review* 5: 23-58.
[15] According to the US Department of Transportation, 2003. op. cit., "Experimental research on the effects of cannabis ... indicat[e] that any effects ... dissipate quickly after one hour."

movement control) and reaction time, as well as variation in lateral positioning, headway (drivers under the influence of cannabis tend to follow less closely to the vehicle in front of them), and speed (drivers tend to decrease speed following cannabis inhalation).[16] In general, these variations in driving behavior are noticeably less consistent or pronounced than the impairments exhibited by subjects under the influence of alcohol.[17] Also, unlike subjects impaired by alcohol, individuals under the influence of cannabis tend to be aware of their impairment and try to compensate for it accordingly, either by driving more cautiously[18] or by expressing an unwillingness to drive altogether.[19]

As a result, cannabis-induced variations in performance do not appear to play a significant role in on-road traffic accidents when THC levels in a driver's blood are low and/or cannabis is not consumed in combination with alcohol.[20-20] For example, a 1992 National Highway Traffic Safety Administration review of the role of drug use in fatal accidents reported, "There was no indication that cannabis itself was a cause of fatal crashes" among drivers who tested positive for the presence of the drug.[21] A more recent assessment by Blows and colleagues noted that self-reported recent use of cannabis (within three hours of driving) was not significantly associated with car crash injury after investigators controlled for specific cofounders (e.g., seat-belt use, sleepiness, etc.)[22] A 2004 observational case control study published in the journal *Accident, Analysis and Prevention* reported that only drivers under the influence of alcohol or benzodiazepines experience an increased crash

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[16] Grotenhermen. 2007. op. cit. and US Department of Transportation. 2003. op. cit. Other summaries include: Ramaekers et al. 2006. Cognition and motor control as a function of Delta-9-THC concentration in serum and oral fluid: Limits of impairment. *Drug and Alcohol Dependence* 85: 114-122; David Hadorn. "A Review of Cannabis and Driving Skills," In: *The Medicinal Uses of Cannabis and Cannabinoids.* (eds: Guy et al). Pharmaceutical Press, 2004; Canadian Senate Special Committee on Illegal Drugs, *Cannabis: Summary Report: Our Position for a Canadian Public Policy.* 2002. (See specifically: Chapter 8: "Driving Under the Influence of Cannabis"); Alison Smiley. "Marijuana: On-Road and Driving-Simulator Studies," In: *The Health Effects of Cannabis.* (eds. Kalant et al) Canadian Centre for Addiction and Mental Health, 1999.

[17] David Hadorn. 2004. op. cit. and US Department of Transportation. 2003. op. cit.

[18] According to the US Department of Transportation, 2003. op. cit., "The extensive studies by Robbe and O'Hanlon (1993), revealed that under the influence of marijuana, drivers are aware of their impairment, and when the experimental task allows it, they tend to actually decrease speed, avoid passing other cars, and reduce other risk-taking behaviors."

[19] Menetrey et al. 2005. Assessment of driving capability through the use of clinical and psychomotor tests in relation to blood cannabinoid levels following oral administration of 20mg dronabinol or of a cannabis decoction made with 20 and 60mg delta-9-THC. *Journal of Analytical Toxicology* 29: 327-338.

[20] United Kingdom Department of Environment, Transport and the Regions, Road Safety Division *Cannabis and Driving: A Review of the Literature and Commentary.* Online document accessed November 24, 2007. <http://www.dft.gov.uk/pgr/roadsafety/research/rsrr/theme3/cannabisanddrivingareviewoft4764> "Overall, we conclude that the weight of the evidence indicates that ... there is no evidence that consumption of cannabis alone increases the risk of culpability for traffic crash fatalities or injuries for which hospitalization occurs, and may reduce those risks."

[20] Gregory Chesher and Marie Longo. "Cannabis and Alcohol in Motor Vehicle Accidents," In: *Cannabis and Cannabinoids: Pharmacology, Toxicology, and Therapeutic Potential.* (eds. Grotenhermen et al.) Haworth Press, 2002.

[21] US Department of Transportation, National Highway Traffic Safety Administration. *The Incidence and Role of Drugs in Fatally Injured Drivers: Final Report.* October 1992.

[22] Blows et al. 2004. Marijuana use and car crash injury. *Addiction* 100: 605-611.

risk compared to drug-free controls. Investigators did observe increased risks – though they were not statistically significant – among drivers using amphetamines, cocaine and opiates, but found, "No increased risk for road trauma was found for drivers exposed to cannabis."[23]

A handful of more recent studies have noted a positive association between very recent cannabis exposure and a gradually increased risk of vehicle accident. Typically, these studies reveal that drivers who possess THC/blood concentrations above 5ng/ml – implying cannabis inhalation within the past 1-3 hours[24-25] – experience an elevated risk of accident compared to drug-free controls.[26-27] (Motorists who test positive for the presence of THC in the blood at concentrations below this threshold typically do not have an increased risk compared to controls.[28]) However, this elevated risk is below the risk presented by drivers who have consumed even small quantities of alcohol.

Two recent case-controlled studies have assessed this risk in detail. A 2007 case-control study published in the *Canadian Journal of Public Health* reviewed 10-years of US auto-fatality data. Investigators found that US drivers with blood alcohol levels of 0.05% – a level well below the legal limit for intoxication – were three times as likely to have engaged in unsafe driving activities prior to a fatal crash as compared to individuals who tested positive for marijuana.[29] A 2005 review of auto accident fatality data from France showed similar results, finding that drivers who tested positive for any amount of alcohol had a four times greater risk of having a fatal accident than did drivers who tested positive for marijuana in their blood.[30] In the latter study, even drivers with low levels of alcohol present in their blood (below 0.05%) experienced a greater elevated risk as compared to drivers who tested positive for high concentrations of cannabis (above 5ng/ml). Both studies noted that overall few traffic accidents appeared to be attributed to driver's operating a vehicle while impaired by cannabis.

Defining A Rational 'Drugged Driving' Policy

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[23] Movig et al. 2004. Psychoactive substance use and the risk of motor vehicle accidents. *Accident Analysis and Prevention* 36: 631-636.

[24] Huestis et al. 1992. Blood cannabinoids: Absorption of THC and formation of 11-OH-THC and THCCOOH during and after smoking marijuana. *Journal of Analytical Toxicology* 16: 276-282.

[25] Mushoff et al. 2006. Review of biologic matrices (urine, blood, hair) as indicators of recent or ongoing cannabis use. *Therapeutic Drug Monitor* 2: 155-163.

[26] Drummer et al. 2004. The involvement of drugs in drivers killed in Australian road traffic crashes. *Accident, Analysis and Prevention* 36: 239-248.

[27] Grotenhermen et al. 2007. Developing per se limits for driving under cannabis. *Addiction* (E-pub ahead of print).

[28] Grotenhermen. 2007. op. cit.

[29] Bedard et al. 2007. The impact of cannabis on driving. *Canadian Journal of Public Health* 98: 6-11.

[30] Laumon et al. 2005. Cannabis intoxication and fatal road crashes in France: a population base case-control study. *British Medical Journal* 331: 1371-1377.

The above review illustrates the need for further education and understanding regarding the effects of cannabis upon driving behavior. While pot's adverse impact on psychomotor skills is less severe than the effects of alcohol, driving under the acute influence of cannabis still may pose an elevated risk of accident in certain situations. However, because marijuana's psychomotor impairment is subtle and short-lived, consumers can greatly reduce this risk by refraining from driving for a period of several hours following their cannabis use.

By contrast, motorists should never be encouraged to operate a vehicle while smoking cannabis. Drivers should also be advised that engaging in the simultaneous use of both cannabis and alcohol can significantly increase their risk of accident compared to the consumption of either substance alone.[31-32] Past use of cannabis, as defined by the detection of inactive cannabis metabolites in the urine of drivers, is not associated with an increased accident risk.[33]

Educational or public service campaigns targeting drugged driving behavior should particularly be aimed toward the younger driving population age 16 to 25 – as this group is most likely use cannabis[34] and report having operated a motor vehicle shortly after consuming pot.[35] In addition, this population may have less driving experience, may be more prone to engage in risk-taking behavior, and may be more naïve to pot's psychoactive effects than older, more experienced populations. This population also reports a greater likelihood for having driven after using cannabis in combinations with other illicit drugs or alcohol.[36] Such an educational campaign[37] was recently launched nationwide in Canada by the Canadian Public Health Association and could readily be replicated in the United States. Arguably, such a campaign would enjoy enhanced credibility if coordinated by a private public health association or traffic safety organization, such as the American Public Health Association or the AAA Automobile Club, as opposed to the federal Office of National Drug Control Policy – whose previous public service campaigns have demonstrated limited influence among younger audiences.[38]

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[31] Ramaekers et al. 2004. Dose related risk of motor vehicle crashes after cannabis use. *Drug and Alcohol Dependence* 73: 109-119. "Experimental studies have shown alcohol and THC combined can produce severe performance impairment even when given at low doses. The combined effect of alcohol and cannabis on performance and crash risk appeared additive in nature, i.e. the effects of alcohol and cannabis combined were always comparable to the sum of the effects of alcohol and THC when given alone."

[32] Williams et al. 1985. Drugs in fatally injured young male drivers. *Public Health Reports* 1: 19-26.

[33] Ramaekers et al. 2004. op. cit.

[34] US Department of Justice, Bureau of Justice Statistics. op. cit.

[35] US Department of Health and Human Services, Substance and Mental Health Services Association, Office of Applied Studies. 1998. op. cit.

[36] Ibid.

[37] Canadian Public Health Association. "The Pot and Driving Campaign." <http://www.potanddriving.cpha.ca/index.html>

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Finally, increased efforts should be made within the law enforcement community to train officers and DREs (drug recognition experts) to better identify drivers who may be operating a vehicle while impaired by marijuana. In Australia, efforts have been made to adapt elements of the roadside Standardized Field Sobriety Test to make it sensitive to drivers who may be under the influence of cannabis. Scientific evaluations of these tests have shown that subjects' performance on the modified SFSTs may be positively associated with dose-related levels of marijuana impairment.[39] Similarly, clinical testing for cannabis impairment among suspected drugged drivers in Norway has been positively associated with identifying drivers with THC/blood concentrations above 3ng/ml.[40]

Though the development of such cannabis-specific impairment testing is still in its infancy, an argument may be made for the provisional use of such tests by specially trained members of law enforcement. In addition, the development of cannabis-sensitive technology to rapidly identify the presence of THC in drivers, such as a roadside saliva test, would provide utility to law enforcement in their efforts to better identify intoxicated drivers. The development of such technology would also increase public support for the taxation and regulation of cannabis by helping to assuage concerns that liberalizing marijuana policies could potentially lead to an increase in incidences of drugged driving.[41] Such concerns are a significant impediment to the enactment of marijuana law reform, and must be sufficiently addressed before a majority of the public will embrace any public policy that proposes regulating adult cannabis use like alcohol.

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Select Research Quotes on THC Levels & Driving Impairment

From:
"Developing limits for driving under cannabis"
Franjo Grotenhermen, et al.
Journal Compilation, 2007, Society for the Study of Addiction

"The results in Fig. 1 show that THC concentrations in the blood are not associated with an elevated risk (OR>1) until they exceed about 6 ng/ml."

"Using current scientific evidence on cannabis-induced impairment of psychomotor skills and the related accident risk, this paper suggests a range of 7-10 ng/ml THC in the serum for an initial non-zero per se limit. It offers reasonably reliable separation of drivers whose driving is in fact impaired by cannabis from whose who are not impaired."

From:
"Marijuana and DUI Laws: How Can We Best Guard Against Impaired Driving?"
Marijuana Policy Project, May 2007

"...the blood serum of moderate to heavy marijuana users may contain more than two ng/mL of THC at 24 or even 48 hours after smoking a single joint, a level that studies have shown does not produce impairment."

"This is a particular concern for medical marijuana patients who are using marijuana in compliance with state laws and their doctors' advice, but who would likely test positive for marijuana while sober."

"Additionally, several studies show that exposure to second-hand marijuana smoke may cause the non-user to show THC concentrations in blood serum of several nanograms per milliliter."

From:
"The involvement of drugs in drivers of motor vehicles killed in Australian road traffic crashes"
Olaf Drummer, et. al.
Accident Analysis and Prevention 36, 2004
[research conducted, November, 2002]

"Many CNS active drugs, particularly cannabis, benzodiazepines, barbiturates and the sedating antihistamines, reduce land control by increasing the standard deviation of lateral position (SDLP)..."

"Logan et. al. (2000) examined the extent of driver impairment of carisoprodol, a skeletal muscle relaxant, and its major matabolite meprobamate, which has sedative properties. The authors found that at therapeutic concentrations impairment was possible with symptoms of intoxication similar to alcohol."

"A study which linked prescription records with hospital admissions from road crashes showed that people who used minor tranquilizers in the past 3 months had a five-fold higher risk of a serious road accident. A similar study showed the odds ratio was elevated for those person taking benzodiazepines, particularly within a few weeks of the first prescription."

From:
"Psychoactive Substance Use and the Risk of Motor Vehicle Accidents"
K.L.L. Movig, et al.
Accident Analysis & Prevention, a peer-reviewed journal, July 2004

"The objective of this study was to estimate the association between psychoactive drug use and motor vehicle accidents requiring hospitalization."

"The risk for road trauma was increased for single use of benzodiazepines and alcohol... High relative risks were estimated for drivers using combinations of drugs and those using a combination of drugs and alcohol. Increased risks, although not statistically significant, were assessed for drivers using amphetamines, cocaine, or opiates."

"No increased risk for road trauma was found for drivers exposed to cannabis."

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http://www.druglibrary.org/schaffer/MISC/driving/ddimp.htm

Drugs and Driving Impairment

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ABSTRACT The objective of this review is to evaluate whether the results of blood drug concentrations could be used by expert witnesses as a basis for scientifically acceptable opinions on driving impairment in adversarial proceedings. Research findings on actual driving performance will be used whenever available.

The adverse effects on driving performance of one drug, alcohol has been well established. Experts can testify to its effects based upon blood and breath alcohol concentrations.

The effects of a few other drugs on actual driving performance have been compared to its likely effects at various blood alcohol concentrations (BACs).

In an actual driving study the impairing effects of the highest smoking dose of marijuana, 3.7% THC, never exceeded those of alcohol's at BAC of 0.8 mg/mL.. Several studies came to the conclusion that it appears to be impossible to conclude anything about a driver's impairment based on THC and THC-COOH blood concentrations. A study of chronic heavy marijuana users which included those who drove trucks, buses and taxis concluded that, no real consequence of prolonged use of the drug was uncovered. Amphetamines and cocaine can improve the performance of fatigued drivers.

The driving impairment BAC equivalencies following the therapeutic doses of drugs used in this review were:

Less than 0.5 mg/mL.. BAC; lorazepam, fluoxetine, flunitrazepam, nitrazepam, paroxetine, loratidine, pseudoephedrine, terefenadine, zoplicone, and doxepin (chronic).

0.5 - 1 mg/mL.. BAC: diphenhydramine, triprolidine and clemastine.

More than 1 mg/mL.. BAC: diazepam, barbiturates, flurazepam, loprazolam, mianserin and doxepin (single dose), all depending on dose and the time between administration and blood sampling.

The ranges of drug concentrations resulting from therapeutic doses, the lack of studies of therapeutic and higher doses for most drugs and combinations, and other factors make expert opinions of drug effects on driving performance questionable.

Keywords: forensic science, forensic toxicology, drug testing, driving performance, expert interpretation, drug concentrations.

The widespread availability of drug testing have led legislators and the public to believe that specimens obtained from drivers can be easily tested and that the results of such tests can be correlated with drug impairment. The objective of this review is to evaluate whether blood drug concentrations could be used by expert witnesses as a basis for scientifically acceptable opinions on driving impairment or improvement in adversarial proceedings.

The adverse effects on driving performance of one drug, alcohol has been well established. Experts can testify to its effects based upon blood and breath alcohol concentrations. Assuming that no alcohol is ingested between the time of an incident and the time the blood is collected, they can calculate within an acceptable range the blood concentration at the time of incident. They can estimate the probable amount of alcoholic beverage which should be ingested to produce certain blood alcohol concentrations. All states have "per se" concentrations. There are many different types of analyses for alcohol which are relatively easy to perform and are inexpensive.

Most of what is known about alcohol and driving performance is not available for most drugs. In 1983 a panel of experts reached a consensus concerning drug concentrations and driving impairment which was reaffirmed in 1989 [1,2]. The panel reported: "In order to establish that use of a drug results in impairment of driving skills and to justify a testing program to respond to this hazard, certain facts must be available. (1) The drug can be demonstrated in laboratory studies to produce a dose related impairment of skills associated either with driving or with related psychomotor functions. (2) Concentrations of the drug and/or its metabolites in body fluids can be accurately and quantitatively measured and related to the degree of impairment produced. (3) Such impairment is confirmed by actual highway experience. (4) Simple behavioral tests such as can be done at the roadside by police officers with modest training, can indicate the presence of such impairment to the satisfaction of courts. (5) A range of concentrations of the drug can be incorporated in laws relating to impaired driving as ipso facto evidence. These criteria have been met for ethanol. It is not certain they can be met for most drugs that are now of concern to highway safety."

The effects of marijuana on actual driving performance have been studied in the Netherlands in a project supported by the U.S. Department of Transportation [3]. Performance of subjects, after smoking standardized marijuana cigarettes, who drove in traffic and on highways for 64 km (40 miles) at speeds up to 100 km/h (62 mph) was evaluated. Plasma specimens were analyzed for tetrahydrocannabinol (THC) and its carboxy metabolite (THC-COOH).

Plasma THC concentrations after smoking 100,200,300 mcg/kg were: 3.3-45.9 ng/mL at 40 min.(minutes), 0.3-15.2 ng/mL at 100 min., 0.5-6.8 ng/mL at 160 min. and 0-5.1 ng/mL at 220 min. The THC-COOH concentrations were from 0-96.4 ng/mL. It was concluded that "THC's effects of SDLP (standard deviation of lateral position) were equivalent to those associated with BACs in the range of 0.3-0.7 mg/mL. Other driving performance measures were not significantly affected by THC." "THC's effects after smoking doses up to 300 mcg/kg never exceed those of alcohol's at BAC's (blood alcohol concentrations) of 0.8 mg/mL.. It appears not possible to conclude anything about a driver's impairment on the basis of his/her plasma concentration of THC and THC-COOH determined in a single sample.." A common standardized test was used that measures driving impairment from vehicular weaving , SDLP (standard deviation of lateral position).

The effects of chronic marijuana use have been reported in a study funded by the National Institute on Drug Abuse [4]. The subjects were 86 chronic marijuana users and 156 non-users. The users smoked an average of 10 (2.5-40) marijuana cigarettes a day for a minimum of 10 years and an average of 17 years. The cigarettes contained 1.3 to 3.7% THC. The report states: "No hard data were obtained regarding the effect of marijuana use on driving ability. However, some of the user subjects did earn their living by driving trucks, buses or taxis, and some preferred to drive while under the influence of the drug." It was concluded that, no real consequences of prolonged use of the drug was uncovered. This was found to be in keeping with the controlled studies carried out in Jamaica and Greece. Until actual driving studies are performed which report blood concentrations in heavy chronic users, one can only speculate as to what its effects might be.

Another NIDA study reported that: "The performance effects of several drug classes were examined using repeated measures design. Eight volunteers were administered two doses of ethanol (0.3and 1.0 g/kg.), marijuana (1.3% and 3.9% THC), amphetamine (10 and 30 mg), hydromorphone (1 and 3 mg), pentobarbital (150 and 450 mg), or placebo on separate days [5]. The larger dose of each increased subjective drug strength; however only, ethanol and pentobarbital impaired performance on circular lights, digit symbol substitution, and serial math tasks. Both ethanol and pentobarbital impaired performance on card-sorting tasks; impairment was evident at lower doses as the cognitive load increased. Results illustrate differences among drugs in producing performance impairment at doses that cause subjective effects. Increasing cognitive requirements uncovered performance impairment at lower doses." "Marijuana had a significant effect on only one of the fourteen performance measures in the present study. On the Serial Addition/Subtraction task, response time was significantly slowed (46%) by the 3.9% THC marijuana cigarette at the 30-minute time point. Our results differ from those of several studies that have shown performance impairment after smoked marijuana. "

Hypnotic drugs were taken by subjects nights before they were to be tested by the aforementioned actual driving method [6]. "All the mean performance changes which occurred after two nights of drug treatment were significant in morning or afternoon tests, or both, except those following nitrazepam 5 mg. and temazepam 20 mg.. The magnitudes of some changes were relatively small for: lorazepam 1 mg., nitrazepam 10 mg., zoplicone 7.5 mg., and flunitrazepam 2 mg. These were equivalent to the amount of impairment caused by BACs (blood alcohol concentrations) in a range from just under 0.5 mg/mL. to about 0.6 mg/mL.. Slightly greater impairment was produced by flurazepam 15 mg. in the morning test. However a very serious degree of impairment, greater than the equivalent of a BAC of 1 mg/mL., was caused by the residual effects of secobarbital 200 mg., flurazepam 30 mg., and loprazolam 2 mg.."

Another study in which temazepam (15 mg.) and temazepam plus ethanol (breath ethanol concentrations, at three testing times were: 30 min. 8 mmol/L, 90 min. 7 mmol/L, 150 min. 4 mmol/L) were administered concluded [7]: "Previous studies have shown that appropriate use (e.g. a therapeutic dose taken before bed with testing the following morning) does not result in residual impairment. This study showed that temazepam, especially coupled with ethanol, does result in impairment by tracking tasks over three hours, the divided attention test providing a more sensitive measure of these effects. The subjects' perception that their performance was unimpaired after taking both drugs in combination is especially important in the light of the

measureable reduction in performance seen with the psychomotor tasks." "The mean plasma temazepam concentrations for the six subjects at 150 minutes was 372 (SD 144) ng/mL."

After a week of taking diazepam 5 mg. 3xd and lorazepam 2 mg. 2xd, driving performance was impaired more than that produced at a BAC of 1 mg/mL..[8].

The driving of subjects using first and second generation antihistamines was evaluated [9]. Single doses of diphenhydramine 50 mg., clemastine 2 mg. and multiple doses of triprolidine 5 and 10 mg. produced changes equivalent to those produced by BAC's of 0.5 - 1 mg/mL..

Terfenadine [9] a second generation "non-sedating" antihistamine, was taken in single doses up to 180 mg. and multiple doses over 4 days up to 120 mg. 2xd. Single doses and multiple doses of 60 mg. 2xd and 120 mg. 4xd never produced a significant rise in SDLP. On the contrary, there was a tendency for 60 and 120 mg. to produce a slight fall in SDLP, suggesting a mild stimulating activity of the drug. When subjects took doses of 120 mg. 2xd for 4 days, impairment was equivalent of up to that of 0.05% BAC.

Loratidine [9] in single doses of 10 and 20 mg. produced no significant rise in SDLP. When given in 20 mg. doses 4xd for 4 days, the impairment was similar to that of terfenadine.

"Cetirizine's [9] effects on SDLP is a matter of contention between different groups of investigators. One showed a significant impairing single-dose effect of cetirizine 10 mg., while the other found no effect of that dose on either the first or fourth day of that dose.

"In Europe acrivistine is available alone in 8-mg. doses and combined with pseudoephedrine in two formulations: acrivistine 8 mg., pseudoephedrine 60 mg. (instant release) and acrivistine 12 mg. (slow release)[9]. Only the combination, Semprex D™, acrivistine 8 mg. and pseudoephedrine 60 mg. (instant release) is available in the U.S.A." "Acrivistine 8 mg. had no effect on mean SDLP but the 8 as well as the 16 and 24 mg. combination preparations had a salutary effect on driving performance. The women who were impaired after 8 mg. of acrivistine alone were not affected by the same dose in combination with pseudoephedrine 60 mg.. Moreover, the men who were treated with that combination and drove, after 4 days of treatment, had a significantly lower SDLP than after placebo. It would appear that the pseudoephedrine's mild stimulating activity physiologically antagonizes acrivistine's correspondingly mild sedating activity, when present; and the former predominates when the latter is low or absent."

The study included two other drugs, mizolastine and ebastine [9]. Mizolastine taken in single doses of 5, 10, and 20 mg. produced effects less than those of 0.5 mg/mL. BAC and at 40 mg. less than those of 0.8 mg/mL. BAC. Ebastine was taken 4xd. "The effects of the 10 mg. doses were stimulating both days. The 20 mg. doses lowered SDLP on day 1, though not significantly." By day 5, the 30 mg. doses produced impairment less than that of 0.5 mg/mL. BAC.

Fluoxetine 20 mg. was administered to 18 healthy volunteers for 22 evenings [10]. "Mean plasma concentrations and (s.d.) for fluoxetine and norfluoxetine were respectively 34.47 (14.41) and 42.47 (17.47) ng/mL on day 8 and 57.83 (24.88) and 75.78 (28.29) ng/mL on day 22 of treatment." "No significant effects were found on any parameters in either the highway driving or the car-following test."

"Amitriptyline 75 mg./day produced severe drowsiness and strikingly impaired performance on nearly every test on the first day but its effects were practically gone after 1 week of treatment [11]. Paroxetine 20 mg., the usual anti-depressant dose, had no effect on performance. Paroxetine 40 mg. did not affect road tracking but slightly impaired performance in some psychomotor tests in a persistent manner."

"Depression itself and the chronic use of one antidepressant, amitriptyline, are associated with greater than normal risk of traffic accidents [12]. Otherwise, impairments associated with depression generally resolve in those patients showing a favorable response to antidepressant therapy, regardless of the drug."

Mianserin 10 mg. 3xd and doxepin 25 mg. 3xd were administered for 8 days. "On day 1, mianserin and doxepin impaired driving [13]. Impairment dissipated after 8 days of treatment with doxepin but not during treatment with mianserin."

"Cocaine effects on driving performance have been examined in a series of studies performed at SCRI (Southern California Research Institute). Twenty-four healthy male subjects, ages 21-40 years, who were self-reported moderate users of cocaine were used. An initial experiment with cocaine (96 mg., intranasally) and alcohol 0.58 g/Kg b.w., found no impairment of driving-related laboratory tasks by cocaine [14]." "In a second experiment with 96 mg. cocaine, subjects performed better with cocaine than with placebo with greatest difference observed during a test battery beginning three hours after dosing. Since that second test time coincided with the afternoon slump, the findings raised questions about the drugs effects with circadian rhythm [15]. Time-of-day differences associated with cocaine's effects were further studied in a nighttime experiment."

In the nighttime experiment [16], "Subjects participated in three two-day treatment sessions. Day 1 began between 18.00 h and 19.30 h. Subjects slept overnight and were awakened at 08.00 h to begin day 2." Each treatment of 96 or 126 mg. of cocaine was divided into three equal amounts given intranasally at half hour intervals. Blood specimens obtained 10 min. after each dose had the following concentrations of cocaine/benzoylecgonine; 3/57, 64/214, and 189/363 ng/mL. "D-A (divided-attention) and VIG (vigilance) data agree with previously-reported data [15] in demonstrating that the effects of cocaine on performance persist past the period of acute stimulation. When subjects were tested near midnight, scores were better with cocaine than with placebo. It was only in the placebo condition that overall D-A performance was significantly worse at the late night hour. D-A RTs were faster with 96 mg. cocaine whereas 126 mg. cocaine prevented slowing of VIG RTs (response times). These data suggest that cocaine effects may be task dependent as well as dose dependent."

A study of methamphetamine and driving impairment concluded: "The net conclusion of the material reviewed in this study was that the circumstances under which any methamphetamine induced performance increment is possible are extremely narrow, and is not guaranteed because of typical side effects associated even with low dose use. Furthermore, there is ample evidence from the epidemiological, clinical, case report and toxicological data to conclude that the behavior displayed in the (28) cases we reviewed is consistent with impairment as a result of methamphetamine use, withdrawal, or combined use of methamphetamine and some other drugs [17]. The author's hysteresis plot showed improved reaction time, relief from fatigue, and

euphoria with blood methamphetamine concentrations of 0.0l to 0.09 mg/L becoming fatigue and exhaustion on withdrawal with the same blood methamphetamine concentrations.

A study of the effects of methadone, as used in methadone maintenance programs, on performance related to driving has been reported [18]. The mean dose of methadone was 70 mg. (range 15 to 150 mg.) . The test battery was sensitive to the effects of alcohol (mean BAC 0.64 mg/mL.) and diazepam 15 mg. orally. "Both alcohol and diazepam produced a significant decrement in performance on the test battery by the control groups and the stabilized methadone clients. However, there was no evidence for an interaction between methadone and either alcohol or diazepam in the group of methadone clients stabilized on the program.." "The insensitivity of these tests of skill performance to the acute effect of methadone on the clients within the methadone maintenance program indicate that these clients should not be considered as impaired in their ability to perform complex tasks such as driving a motor vehicle."

Patients whose pain was controlled with slow-release morphine sulfate tablets in a daily dose range of 60 to 1100 mg. had the following plasma concentrations: morphine 4.5-337ng/mL., morphine-6-glucuronide 20-1014 ng/mL. and morphine-3-glucuronide 139-4857 ng/mL. [19]. The authors stated that, "In conclusion, long-term analgesic medication with stable doses of morphine does not have psychomotor effects of a kind that would be clearly hazardous in traffic."

In another study, subjects who took oral single 10 and 15 mg. doses of morphine sulfate, "had minimal impairment of cognitive and psychomotor function with possible improvement in one test [20]..

Diabetics and epileptics require such drugs as insulin, diphenylhydantoin and phenobarbital in order to live and to control physical and mental conditions that would make driving hazardous if drugs were not used to normalize their driving performance. "Diabetic hypoglycemia produces cognitive motor slowing [21]. Driving performance was not disrupted at mild hypoglycemia nor after recovery from moderate hypoglycemia. Moderate hypoglycemia disrupted steering, causing more swerving, spinning, time over midline and time off road. It also resulted in an apparent compensatory slowing, with more very slow driving." Mean blood glucose levels: Control, euglycemia 6.3 nM/L., mild hypoglycemia 3.6 nM/L., moderate hypoglycemia 2.6 nM/L.

Epileptics should not drive unless they are seizure-free or their seizures are controlled by anticonvulsants and sedatives. "EEG and driving behaviour were monitored in six patients with subclinical focal and generalized eleptiform EEG discharges during 420 km (260 miles) of actual motorway driving in a suitable instrumented vehicle [22]." "Evidence of impaired driving performance during subclinical discharges was significant in three subjects and was suggestive in one. The two patients with greatest impairment had active epilepsy, whereas the others had been seizure-free for upwards of 4 years. No other features appeared to be predictive of altered driving behaviour during discharges."

Discussion:

Adequate methods are available for the identification and determining the amount of drugs in blood, urine, hair, sweat, saliva and other specimens. The major problem is in relating the drug concentrations in the specimens to driving impairment. Studies have reported the numbers of

drivers, injured or dead in crashes, who had drugs in their bodies. It is not known whether the drugs were factors in causing the crashes.

The U.S. Department of Transportation issued regulations that require testing of safety-sensitive employees in transportation industries "for use, in violation of law or Federal Regulation, of alcohol and drugs listed in the Controlled Substances Act." [23] Drivers may use controlled substances, "when the use is pursuant to the instructions of a physician who has advised the driver that the substance does not adversely affect a driver's ability to safely operate a commercial motor vehicle." Could such a statement be supported scientifically? The stated intent of the Federal workplace drug testing program is to identify individuals who use illegal substances [24]. Urine specimens are tested for amphetamines, phencyclidine and the metabolites of marijuana, opiates, and cocaine.

Some "legal drugs" which are controlled substances have adverse effects on actual or simulated driving and must be obtained by prescription. Some of these are: diazepam, flurazepam, loprazolam, barbiturates, mianserin, and clemastine. Diphenhydramine and triprolidine are available without prescriptions. Tests for the above drugs and many others are rarely performed on impaired drivers. If two or more drugs are found, it is essential that the combined effect be evaluated. Combining an antihistamine with pseudoephedrine can overcome the impairing effect of the antihistamine.

Problems such as fatigue, lack of attention, vigilance deficits, suicidal and aggressive tendencies can cause crashes. Many drugs can create such problems. They can influence vision, vigilance and impulsiveness. Concentrations of drugs and metabolites in body fluids can be determined but the concentrations of most drugs cannot be correlated with impairment or improvement of driving.

Specimens other than blood are useful in determining drug use but none is helpful in determining whether there is an active drug in the body which is affecting driving performance. Interpretation of the effects produced at various concentrations of drugs in blood specimens depends on many factors not generally available to an expert witness for use as a basis for formulating acceptable scientific opinions. Some of the factors are: the impossibility of reliably back calculating concentration to a prior time, individual differences in metabolism, single or chronic dosing, tolerance, withdrawal, inter and intralaboratory methods and variances, multiple drug use, method of use, and the ranges of drug concentrations produced in different individuals ingesting the same size dose.

About twenty-five drugs are reviewed in this paper. There are thousands of drugs available and millions of combinations of these drugs. It is improbable that by any of the methods now available, that the problems of relating drug concentrations to impairment or improvement of driving performance will be solved.

In criminal court, it must be proven beyond a reasonable doubt that a person drove while his or her physical or mental faculties, or both, were appreciably impaired by an impairing substance. In civil courts the standard of proof is by the preponderance of the evidence or that impairment is more likely than not.

Based on the reports in this review, forensic scientists appearing as experts in adversarial proceedings should be able to offer some opinions. Based on the blood concentrations of THC and/or THC-COOH in this review, an expert could not say with scientific certainty that a driver's impairment would be greater than that of a driver with a 0.5 mg/mL. BAC.

Based on the blood drug concentrations in this paper, knowledgeable experts should be able to rebut opinions of significant impairment by marijuana, cocaine, pseudoephedrine, amphetamines, lorazepam, fluoxetine, terfenadine, paroxetine, loratidine, nitrazepam, zoplicone, flunitrazepam and chronic doxepin. They appear to have little or no impairing effect on driving performance in the concentrations cited in this review. Opinions that higher concentrations results in impairment must be backed by scientifically acceptable evidence.

Expert opinions might be offered that driving impairment is probably greater than that of a driver with a 1 mg/mL. BAC when blood specimens have therapeutic blood concentrations of the following: barbiturates, diazepam, flurazepam, loprazolam, mianserin and doxepin (single dose). Impairment equal to that of a driver with a BAC of 0.5 - 1 mg/mL. was found in drivers with therapeutic blood concentrations of the following: clemastine, diphenhydramine, and triprolidine.

In many countries: only a BAC of < 0.2 or of < 0.5 mg/mL. is acceptable and 1 mg/mL. is considered as an unacceptable impairment level.

Law enforcement officers, including drug recognition evaluators, DRE's, who try to evaluate the performance of drivers should be aware of the reports in this review and elsewhere, before they offer opinions of the source of various signs and symptoms which might be produced by drugs and other factors, and affect the physical and mental conditions of drivers.

The establishment of "per se" concentrations of drugs is not scientifically sound. A discussion of the problems of such an approach is presented in the Consensus Report [1].

Making it a crime to drive while possessing drugs or finding drugs in specimens obtained from drivers cannot be related scientifically to driving impairment for most drugs.

Whatever expert opinions are offered, they must be supported by scientific documentation, experience and by other evidence. Much more scientific research is needed on the effects of drugs and drug combinations on actual driving performance. Experts must be able to show that an impairing substance appreciably adversely affected the driver's physical and/or mental faculties. In adversarial proceedings where performance is a factor, the mention of the possible use of drugs is prejudicial and should be excluded as irrelevant, unless it can proven that performance was adversely affected by the use of drugs.

The ranges of drug concentrations resulting from therapeutic doses and the lack of studies of therapeutic and higher doses for most drugs and combinations, make expert opinions of drug effects on driving performance questionable.

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Therapeutic doses and concentrations:

Baselt [25]

Amitriptyline (Elavil) 50mg(S) 16-35*(2-4 hr), 150mg(P) 38-162.

Diazepam (Valium) 10mg(B) 148 (1hr), 37 (24hrs), 30mg/d(P) 700-1500.

Diphenhydramine (Benadryl) 50mg(P) 83(3hr), 49(6hr), 9(24hr)

.

Doxepin (Sinequan) 75mg(P) 24(2hr), 113mg/d(P) 5-115.

Flurazepam (Dalmane) 90mg(B) 13, none after 30mg/d.

Lorazepam (Ativan) 2mg(P) 18(2hr), 9(P)(24hr), 10mg/d(P) 140-240.

Methadone 15mg. 75(4hr) 30(24hr), 100-200mg/d 570-1060(4hr),

280-790(24hr)

Mianserin (Norval) 20mg(P) 26-30(1.7hr), 60mg/d(P) 12-81.

Morphine 20-30mg above 20(4-6hrs).

Nitrazepam (Mogadon) 5mg(S) 35(2hr), 5mg/d(P) 39.

Secobarbital 200mg(B) 2000(3hr), 1300(20hr)

. Temazepam (Restoril) 10mg(P) 205-430(15-90m), 20mg(P) 363-856(15-75m)

.

PDR. 1997 [26]

Acrivistine (Simplex D) 8mg(P)max.393 + pseudoephedrine 60mg(P) max.1308

Cetrizine (Zyrtec) 10mg/d(P) 311±40(1.0±0.5hr)

Paroxetine 30mg/d 61.7

Terfenedine (Seldane) 60mg(P) 263-423(2.5hr)

*ng/mL, Blood(B), Serum(S), Plasma(P), mg/d=mg/day, m=minutes.

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http://www.mpp.org/library/marijuana-and-dui-laws-how.html

Marijuana and DUI Laws: How Can We Best Guard Against Impaired Driving?

How do laws against driving under the influence of marijuana work?

Blood testing seems to be the only reliable method to determine the actual level of THC in the body, since urine tests cannot show that a person has recently used marijuana. Depending on quantity and strength, a single dose of THC produces metabolites in urine that last for at least 12 days — long after the psychoactive effects of the substance have worn off.[1] However, the key is not necessarily to know the exact level of THC in a driver?s bloodstream, but whether or not the person is impaired and thus incapable of safely operating a motor vehicle.

What is the threshold for considering a driver to be impaired by marijuana?

It is unclear what blood level of THC (the main psychoactive ingredient in marijuana) constitutes actual impairment. Most credible scientists working on the issue acknowledge the difficulty of pegging THC impairment to a number (in a way similar to drunk driving laws), and epidemiological evidence on the risk of accidents associated with marijuana is much less conclusive than data regarding alcohol.

The most meaningful recent study measuring driver "culpability" (i.e., who is at fault) in 3,400 crashes over a 10–year period indicated that drivers with THC concentrations of less than five ng/mL in their blood have a crash risk no higher than that of drug–free users.[2] The crash risk begins to rise above the risk for sober drivers when a marijuana user's THC concentrations in whole blood3 reach five to 10 ng/mL.

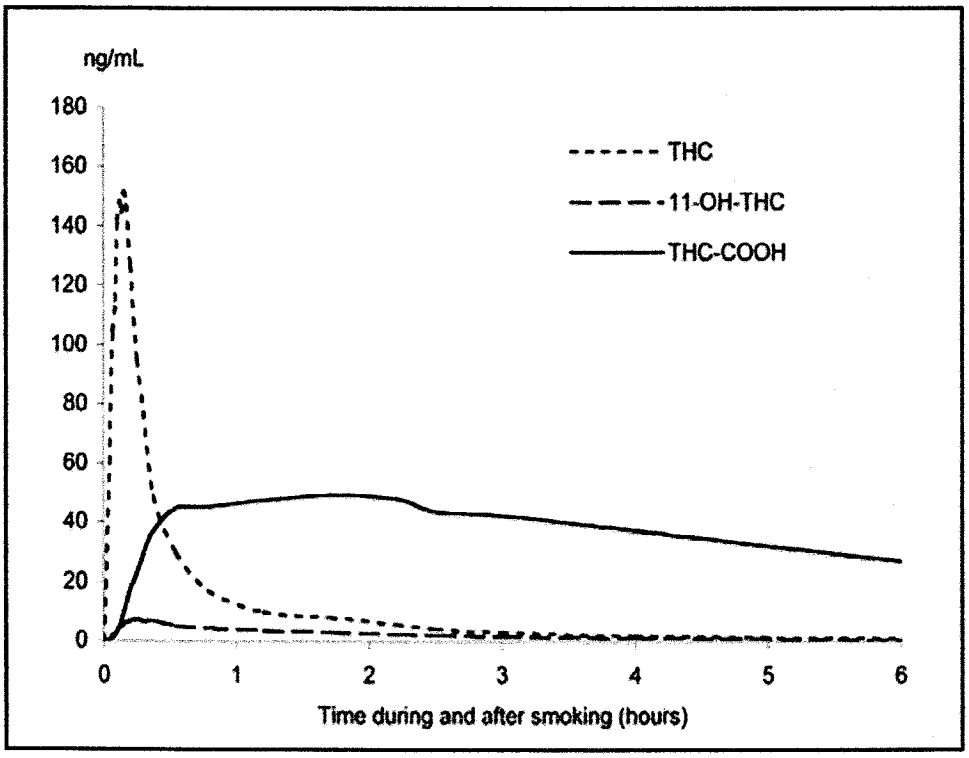
How long does it take for the psychoactive effects of marijuana to wear off?

Because smoked THC is rapidly transferred into the blood stream, THC levels in the blood rise quickly immediately after inhalation. Depending on the dose, THC typically reaches peak concentrations of more than 100 ng/mL five to 10 minutes after inhalation and then rapidly decreases to between one and four ng/mL within three to four hours.

However, the blood serum of moderate to heavy marijuana users may contain more than two ng/mL of THC at 24 or even 48 hours after smoking a single joint, a level that studies have shown does not produce impairment.[4]

This is a particular concern for medical marijuana patients who are using marijuana in compliance with state laws and their doctors' advice, but who would likely test positive for marijuana while sober.

The graphic below shows the mean plasma levels of THC and its metabolites (11-OH-THC) and THCCOOH) for six subjects smoking a marijuana cigarette containing 34mg of THC, following several days of abstinence (which would reflect an occasional user's pattern of usage).[5]



Additionally, several studies show that exposure to secondhand marijuana smoke (which could result from being in the same room with a person who is using marijuana) may cause the non–user to show THC concentrations in blood serum of several nanograms per milliliter.[6]

Does this mean that some DUID laws may actually criminalize sober drivers?

Yes, depending on the threshold of THC that the law sets. Furthermore, arresting and convicting motorists who only have traces of marijuana metabolites in their systems (from having used marijuana days before) will certainly cause people who are completely sober to be arrested and wrongly convicted of driving under the influence of drugs.

The standard for scientists is to test blood and urine, but what about other bodily fluids, like saliva, or performance–based tests?

Because of the invasiveness of blood tests and the inadequacy of urine tests in determining impairment on the roadside (i.e., actual THC levels), police officials hope to institute roadside saliva testing in the near future. However, the technology for reliably testing saliva is still unavailable, and there are no national standards for testing saliva, as there are with blood and urine.

Significant work is being done to develop and implement modified field sobriety tests, which measure the behavior of drivers (reaction time, for example) rather than their bodily fluids.

MPP recommends a policy similar to most state laws on driving under the influence of alcohol: A driver who fails a roadside sobriety test should be required to submit to a blood test by a trained medical professional — or risk criminal and administrative sanctions.

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