National Drug Driving Study

Project Update, June 2021

Prepared By: Road Safety & Public Health Research Team at Department of Emergency Medicine, University of British Columbia

CENTRE for Clinical Epidemiology and Evaluation

Vancouver Coastal Health Research Institute
Healthier lives through discovery

UBC

THE UNIVERSITY OF BRITISH COLUMBIA

National Drug Driving Study

Project Update, June 2021

This study has been made possible through funding from Health Canada, the Canadian Institutes of Health Research, the Canadian Centre on Substance Use and Addiction, Ministry of Transportation Ontario, Saskatchewan Government Insurance and Alberta Transportation. The views expressed herein do not necessarily represent the views of the funders.

Suggested Citation: Brubacher JR, Chan H, Masud M, Yuan Y, Erdelyi S, Likhodi S, and the National Drug Driving Research Group. The 2021 National Drug Driving Study. Vancouver, BC. Department of Emergency Medicine, University of British Columbia.

Contributions of the following individuals have made this project possible and successful across Canada. Thank you, for your hard work.

Canadian Drug Driving Study Research Group:

Dr. Paul Atkinson	Dalhousie University (Dalhousie Medicine New Brunswick)
Dr. Jeff Brubacher	University of British Columbia
Dr. Herbert Chan	University of British Columbia
Dr. David Clarke	Dalhousie University
Dr. Raoul Daoust	University of Montreal
Dr. Phil Davis	University of Saskatchewan
Dr. Jeff Eppler	University of British Columbia
Dr. Marcel Emond	Université Laval
Dr. Chrystal Horwood	Memorial University
Dr. Jacques Lee	University of Toronto
Dr. Adam Lund	University of British Columbia
Dr. Andrew MacPherson	University of British Columbia
Ms. Manal Masud	University of British Columbia
Dr. Kirk Magee	Dalhousie University
Dr. Éric Mercier	Université Laval
Dr. Jagadish Rao	University of Saskatchewan
Dr. Brian Rowe	University of Alberta
Dr. John Taylor	University of British Columbia
Dr. Christian Vaillancourt	University of Ottawa
Dr. Ian Wishart	University of Calgary
Ms. Yue Yuan	University of British Columbia

Site Research Coordinators and Assistants:
Faiyzah Ahmed, Gabi Andrus, Lorelei Audas, Alexandra Beaudin, Manya Charette, Stephanie Couperthwaite, Kathryn Cyr, Ashlee de Medeiros, Corinne DeMone, Frédérique Bahary-Dionne, Madison Donoghue, Carly Ethier, Jacqueline Fraser, Fabien Henri, Katrina Koger, Nelofar Kureshi, Chantal Lanthier, Meagan Larson, Kaidi Liu, Megan Martin, Dr. Alexandra Nadeau, Dawn Parker, Amanda Pearce, Tracy Peatt, Lulu Pei, Lydia Redjah, Natalie Runham, Andrea Stringer, Amanda Swirhun, Mariane Martel-Thibault, Lauren Tierney, Jill Vaughan, Hina Walia, Taylor Weir, Queenie Wong, Joanna Yeung, Gabriele Zitikyte

Table of Contents

Terminology and Definitions

Cannabinoids

Marijuana contains over 60 active compounds known as cannabinoids. When absorbed into the blood, cannabinoids exert their effects by binding to receptors in the brain and throughout the body.

THC (delta-9-tetrahydrocannabinol)

THC is the main psycho-active compound found in marijuana and is responsible for most of marijuana's impairing effects.

THC Levels

THC levels are best measured in blood but interpreting THC levels is complicated. Immediately after smoking a "joint", whole blood THC levels typically peak at >100 ng/mL within 15 minutes and then drop rapidly so that, in occasional users, THC is usually <2ng/mL within 4 hours after a single acute exposure.[1] However, in habitual marijuana users, THC accumulates in body fat and is then slowly released back into the blood. As a result, habitual users can have THC levels in the range of 1 -3 ng/mL for days or even weeks after last use.[2] In most cases, however, THC > 5 ng/mL indicates recent use (acute exposure). After smoking a marijuana joint, the psychotropic (impairing) effects typically peak at 20–30 minutes and resolve by 4 hours. Ingesting cannabis delays the onset and extends the duration of effect.

COOH-THC (11-nor-9-carboxy-delta-9-tetrahydrocannabinol)

The main metabolite (breakdown product) of THC. COOH-THC does not cause impairment and persists in blood and urine long after impairment has resolved. Thus COOH-THC provides evidence of previous cannabis exposure but does not necessarily indicate impairment or recent use.

Polysubstance Use

People who use drugs often take more than one substance at the same time. This is referred to as polysubstance use. Taking several drugs in combination can lead to worse impairment than would be seen from either substance taken alone.

Liquid chromatography/tandem mass spectrometry (LC-MS/MS)

An advanced laboratory technology that is used to detect and/or quantify a wide range of drugs using standards of known substances and concentrations.

Gas Chromatography-Flame Ionization Detection

A standard laboratory technique used for measuring blood alcohol levels.

Phlebotomists

Specially trained technicians who obtain blood samples from patients

Drug-driving vs drug-impaired driving

"Drug-impaired driving" means that the driver is impaired by drugs. Presence of drugs in body fluids indicates drug use but not necessarily impairment. We use the term "drug driving" to indicate driving after using drugs.

Sedating Medications

Many prescription medications cause sedation either as a desired therapeutic effect or as an unwanted side effect. In the Drug Evaluation and Classification System, these drugs would be classified as CNS (central nervous system) depressants. Common sedating medications include:

Benzodiazepines

These are mild sedatives most commonly prescribed as “sleeping pills” or to treat anxiety

Antihistamines

Antihistamines cause sedation as an unwanted side effect. Over the counter antihistamines are used to treat allergies (e.g. diphenhydramine - “Benadryl”), or motion sickness (e.g. dimenhydrinate - “Gravol”).

Antidepressants

Antidepressants, especially the older antidepressants, have sedation as a side effect.

Z-drugs

These drugs are sedatives that act like benzodiazepines and are prescribed mostly as sleep aids. In Canada the most common Z-drug is zopiclone.

Opioids

Opioids are narcotic analgesics (pain killers) that can cause marked sedation or even coma along with respiratory depression. Opioids include prescription medications such as codeine, hydromorphone, oxycodone, and morphine. The street drug heroin is also an opioid.

Psychomotor Skills

This refers to ability to perform tasks involving perception, interpretation and action. Examples include reaction time, tracking ability (e.g. ability to drive a car in a straight line without weaving), coordination, and tasks that require attention.

Background

Drug driving is a threat to road safety. The epidemiology and risk of crashing in drinking drivers is well understood as a result of intense research conducted over the past 50 years.[3-6] This knowledge has facilitated the development of effective measures targeting drunk driving. Alcohol-impaired driving and related fatalities are declining as a result of visibly enforced laws, administrative licensing sanctions, and social marketing campaigns.[7-11] Drug driving is also viewed as a major threat to road safety,[12] and the prevalence of drug driving may be increasing.[13] In fact, there is evidence that *drug driving has become as common as driving after drinking alcohol in Canada*.[14-18] With cannabis legalization, there is concern that the prevalence of drug driving, especially driving after using cannabis, will increase. Cannabis legalization could also result in more drivers combining cannabis with alcohol or other drugs, resulting in additive impairment.[19-21]

Unlike driving after drinking alcohol, which involves a single easily measured substance, drug driving is difficult to study. We know that *many drugs impair the psychomotor skills and/or judgment required for safe driving*. Cannabis intoxication causes attention deficits, slows reaction time and impairs automated tasks such as tracking ability (*e.g.*, staying within a lane) or monitoring the speedometer.[19, 22-26] Several expert panels compared experimental studies of impairment from THC with that from alcohol, in both males and females, and concluded that a blood alcohol concentration (BAC) of 0.05% causes a similar degree of impairment as THC levels in whole blood of 2-5ng/mL.[27-29] However, it is important to note that habitual cannabis users may develop tolerance to some of the impairing effects of cannabis. [30-32] This calls into question the ability to predict impairment based on THC level, especially in habitual users. Although cannabis-impaired driving is very topical, it is important to realize that many other drugs also cause impairment. Stimulants, such as cocaine and amphetamines, impair judgment, impair inhibitory control (ability to "tune out" and not react to irrelevant stimuli), and alter mood.[33-40] Sedating medications, such as benzodiazepines, antihistamines, antidepressants, Z-drugs (non-benzodiazepine sedatives such as zopiclone), and opioids, cause drowsiness, slow reaction time, impair cognitive function and impair tracking ability.[41-51] For these reasons, *many drugs are suspected to increase the risk of crashing*. Several recent meta-analyses all concluded that cannabis increases the risk of crashing, albeit to a lesser extent than alcohol.[52-55] There is epidemiological evidence that other drugs (amphetamines, cocaine, benzodiazepines, antihistamines, antidepressants and opioids) also increase crash risk. In fact, the crash risk with many of these drugs, although lower than that with alcohol, appears to be as high as or even higher than the risk associated with cannabis.[55-58]

The prevalence of drug driving in Canada is poorly studied. Previous research on the prevalence of drug use in Canadian drivers is based on roadside surveys, coroner's reports, police crash reports, or self-reported surveys.[14, 16, 59-61] These methods, although useful, have significant limitations. In <u>*roadside surveys*</u>, police pull over drivers and direct them to a safe parking spot. Researchers then ask the drivers about drug and alcohol use and obtain samples for drug testing. Roadside surveys are limited by high refusal rates which could result in selection bias if drivers who used drugs are more likely to refuse than other drivers. For practical reasons, roadside surveys use saliva rather than blood and, as a result, they provide less insight into actual driver impairment. For logistic reasons, roadside surveys typically sample a large number of drivers over a few days during the summer (when weather is good) making these surveys poorly suited for long term monitoring of drug driving. Another limitation is that, because of high cost and logistic challenges, roadside surveys are seldom performed. <u>*Coroner's data*</u> provide another estimate of the

prevalence of drug use in drivers. In 2008, 61.0% of fatally injured Canadian drivers were tested for drugs and 36.7% were positive for an impairing drug other than alcohol, including 16.1% who tested positive for cannabis. Females were less likely than males to be positive for alcohol but equally likely to test positive for drugs.[14] Coroner's data are useful but can be susceptible to selection bias if drug testing is based on suspicion of drug use and not performed routinely on all drivers. In Canada, the percentage of fatally injured drivers tested for drugs (2008) varies by province, ranging from 10% to 100%. The toxicology testing protocols used by coroners differ from province to province - with different protocols detecting different drugs. Coroner's data often fail to distinguish recent *vs* chronic drug use because some coroners measure drug metabolites rather than active drug. If fatally injured drivers survive the crash for a period of time, drug levels will decline with metabolism, making toxicology testing unreliable. Interpreting drug levels from coroner's data is further complicated by postmortem redistribution which for some drugs (such as cannabis) can result in significantly different results for postmortem *vs* pre-mortem drug levels.[62-66] As driving while impaired by drugs is illegal, *police crash reports* allow police to record their suspicion that a driver is impaired by drugs. However, these reports provide unreliable estimates of cannabis/other drug use as police only identify a small fraction of drivers who use cannabis or other drugs.[67] *Self-report surveys* ask questions about driving after using cannabis or other drugs.[68] Surveys are subject to selection, recall and reporting biases. In addition, self-report surveys typically lack precision because they ask about drug use before driving in a given time period (*e.g.*, previous month) instead of before a specific driving episode.

This is a national drug driving research project that aims to study drug use in injured drivers who present to hospital and have bloodwork obtained within six hours of a motor vehicle collision. We study a relevant population (injured drivers) and measure actual concentration of a wide range of impairing drugs in blood. Hence, this research has several advantages over other methods of studying drug driving. We aim to provide relevant data that policy makers and injury prevention groups can use to inform policy and programs designed to prevent people from driving after using drugs.

Methods

Inclusion and Exclusion Criteria

We include all moderately or severely injured drivers of motorized vehicles (e.g. cars, motorcycles, trucks) who visit the emergency department (ED) of a participating hospital and have blood samples obtained within 6 hours of the crash. Injury severity is defined pragmatically as the need to obtain blood for clinical purposes (moderate injury), or need for overnight hospital admission (severe injury). Potentially eligible drivers are identified by daily review of ED visit logs and eligibility is confirmed through chart review. We exclude drivers with minor injuries who do not require blood testing for clinical purposes, cases in which blood was first obtained more than 6 hours after the crash, and cases in which no excess blood remains after clinical use.

Chart Review

ED records of eligible drivers are reviewed, and relevant data is abstracted and entered in REDCap, a secure web application for building and managing online surveys and databases. ED records include ambulance records (filled by paramedics), emergency physician notes, nursing notes, laboratory results including blood alcohol concentration (BAC), and consultant notes (if applicable). The abstracted data includes age, sex, first three digits of postal code, crash time and date, crash type (single vs multiple), vehicle type, blood draw time, prescription medications used in last 30 days, medical history, documentation of alcohol or drug use, disposition and medications given as part of clinical care prior to blood draw (we exclude “post-crash” medications when reporting toxicology results).

Blood Handling

Blood availability is determined by research assistants through review of medical records (to identify drivers who had blood samples drawn) followed by a visit to the hospital laboratory to see if excess blood remains. Excess blood is relabeled with study ID number replacing the clinical label and frozen at -80º C for future analysis. Freezing is important as significant losses of THC/other drugs will occur by two months if blood is stored at room temperature. As blood concentrations of certain drugs, such as cocaine and THC, drop rapidly after use, it is important that time from crash till blood draw is carefully recorded. The time of crash is determined through chart reviews (usually recorded on the ambulance record), and phlebotomists record the time of blood draw. Blood samples are stored in a specimen freezer at each site before shipment on dry ice by overnight courier to the central laboratory in Vancouver where samples are stored at -80º C until ready for analysis.

Toxicology Analysis

Toxicology analyses are performed at BC’s Provincial Toxicology Centre (PTC). In participating hospitals, blood from injured drivers is usually tested for alcohol as part of routine trauma care. If clinical alcohol levels are not available, alcohol will be measured at the PTC using Gas Chromatography-Flame Ionization Detection with a detection limit of 0.01%. In addition, broad spectrum drug screens are performed on each patient’s blood using high-throughput liquid chromatography/tandem mass spectrometry (LC-MS/MS). The extraction process recovers both acidic and basic drugs and is able to detect illicit drugs and their metabolites (cannabinoids, cocaine, amphetamines including their major analogues, and opioids) as well as psychotropic pharmaceuticals (including antihistamines, benzodiazepines, other hypnotics, and sedating antidepressants). The method has detection limits of 0.2 ng/mL for THC and 1 ng/mL for most other substances. When samples are positive for cannabinoids, we quantify both the active ingredient (THC) and the metabolite (COOH-THC). For other drugs, the LC-MS/MS screen will provide a quantitative measure of

drug concentration using ISO-certified reference calibrators. Over 95% of excess blood samples in this study consisted of whole blood. When plasma is available but whole blood was not, we adjust plasma toxicology results to equivalent whole blood results according to previously published studies.

Results

The study received research ethics and operational approval from each of the 15 hospital sites across Canada. Data collection began in British Columbia in 2018 and has since expanded across the country with the latest addition of a site in Edmonton to a total of 16 trauma centres. As of May 2021, approximately 6200 injured drivers met the inclusion criteria. This number represents approximately 49.2% of drivers and motorcyclists who were identified and screened in 15 trauma centres (Royal Alexandra Hospital Edmonton has not started recruitment at the time of this report preparation). The most common reasons for exclusion were either no blood work required (76.7% of excluded cases) or exceeding the 6-hour time frame between crash and blood draw (13.3%). Chart review data from 4976 eligible cases with ED admission date up to January 2021 were completed and their blood samples were analyzed.

Overall, 18.8% of drivers in this sample tested positive for THC (including 7.9% with THC ≥ 2 ng/mL and 3.5% with THC ≥ 5 ng/mL). We also found that 15.5% of drivers tested positive for alcohol including 11.9% with BAC ≥ 0.08%. Opiates were detected in 11% of drivers, recreational drugs (cocaine, amphetamines) in 10.9%, and sedating medications (including the common over-the-counter antihistamine) in 20.7% of injured drivers.

These results, broken down by age, sex and by crash characteristics are shown in Tables 1 and 2 in Appendix A and in Figures 1 to 15 in Appendix B. For comparison purposes, Table 3 in Appendix A summarizes results from all participating hospitals in British Columbia, Alberta, Saskatchewan, Ontario, Quebec, and the Atlantic provinces (Nova Scotia, New Brunswick and Newfoundland). Sites in Atlantic provinces started recruitment last year, therefore their results are aggregated together until a bigger sample size is reached in those provinces. Results between provinces cannot be directly compared without adjusting for age, sex, injury severity and type of crash (singe versus multi-vehicle). Within these limitations, it appears that injured drivers from the Atlantic provinces and Saskatchewan are more likely to have consumed cannabis and are more likely to have been drinking.

Table 4 in Appendix A and Figures 16 and 17 in Appendix B show polysubstance use, the percentage of drivers who used various combinations of alcohol and cannabis (Figure 16) or other drug combinations (Figure 17).

Discussion

In this sample of 4976 injured drivers treated in 15 trauma centres across Canada, half of the drivers (50.8%) tested positive for at least one impairing substance. The most common single substance detected was cannabis with about one in five drivers (18.8%) testing positive for THC, the active ingredient in cannabis. Most of the THC positive drivers had low levels (< 2 ng/mL) which do not necessarily reflect recent use of cannabis, and do not appear to be associated with increased risk of crashing.[69] However, one in 13 drivers (7.6%) had THC ≥ 2 ng/mL which usually indicates recent use of cannabis, and one in 28 drivers (3.5%) had THC ≥ 5 ng/mL which indicates recent use and is often associated with impairment. Cannabis use was more common in drivers under the age of 35 and more common in males than females (22.3% vs 11.4%). It should be noted that these estimates may change as more cases are collected and analyzed. The current state of knowledge indicates that the risk of crashing after using cannabis remains poorly defined but is lower than that for alcohol.[24, 69] Several recent meta-analyses concluded that cannabis increases crash risk, with estimated Odds Ratios (ORs) ranging from 1.36 to 2.66.[52, 54] A recent Canadian study suggests that drivers with THC levels < 5 ng/mL do not have an increased risk of crashing.[69] However, it is worth monitoring the prevalence of drivers with THC ≥ 5 ng/mL over time to analyze whether cannabis impaired driving may be an emerging problem in Canada, especially in younger drivers.

Alcohol was the second most detected impairing substance in this sample. Approximately, one in six drivers (15.5%) had been drinking (BAC > 0), and one in eight (11.9%) had BAC ≥ 0.08%. In this sample, alcohol was more commonly found in drivers between the ages of 19 to 34 and more common in male than female drivers (18% vs 10.3%). It is well known that drivers with BAC > 0.08%, especially younger drivers, have a very high crash risk[69-71]. These data suggest that alcohol impaired driving remains a bigger problem than drug impaired driving in Canada.

Recreational drugs (cocaine, amphetamines) were detected in one in ten injured drivers (10.9%). The highest prevalence of recreational drugs was found in drivers between the ages of 25 to 34 with increased prevalence in males (12.4%) compared to females (7.7%). Sedating medications (including over-the-counter antihistamines were found in approximately one in five drivers (20.7%) with a greater prevalence in females (23.9%) than males (19.1%). The highest prevalence of sedating medications was found in drivers over the age of 55 (24%). These results are not surprising since sedating medication use is typically more common among older population. Opiates were detected in one in nine drivers (11%) and were detected more commonly in males (11.5 %) than females (10%) in this sample. These results are shown in Table 1 and Figures 11 and 12. Cocaine, amphetamines, sedating medications and opiates are known to impair the psychomotor skills required for safe driving.[55, 72] The crash risk associated with these substances is also poorly defined but appears to be less than that associated with alcohol and in the range of that associated with cannabis.[69]

Strengths

Our methods have several advantages. We measured drugs in blood, which, for THC and most other drugs, correlates better with impairment and/or recent use (acute exposure) than drug levels measured in saliva or urine. Our methods quantify alcohol, THC and 85 other impairing drugs and medications. Further, we use blood obtained shortly after the crash, in most cases within 1.5 hours, so our toxicology results closely approximate drug levels at time of crash.[18] This short time interval between crash and blood draw simplifies interpretation of toxicology findings. Third, the decision to obtain blood is *not* based on suspicion of drug use: blood is obtained when clinically indicated for managing the patient's injuries, based on crash mechanism and/or physical examination. This process eliminates the selection bias that would occur if drug testing was based on suspicion of drug use. Also, because this study has ethics approval for waiver of consent due to our innovative methods of anonymizing linked data, we avoid the bias that would arise if drivers who used drugs were less likely to consent for testing, as might be the case in roadside surveys. Most important, we study recent drug use in a relevant population (drivers injured in a crash).

Limitations

There are also several limitations to this study. Because we rely on blood that was obtained for clinical purposes, we do not have control over which drivers are actually tested. As a result, our sample does not include minimally injured drivers even if they caused a crash that seriously injured another road user. It is also possible that the decision to obtain blood tests varies from hospital to hospital which may make results from different hospitals difficult to compare. A final limitation is that we do not examine, or interview injured drivers and are unable to assess whether drivers are actually impaired.

Summary

Driving after cannabis use appears to be an emerging problem in Canada and may now be more common than driving after drinking alcohol. However, given the very high crash risk associated with alcohol, and the fact that most "cannabis positive" drivers had low THC levels, it can be concluded that driving after drinking remains a bigger problem in Canada. Sedating medications, opiates, and other recreational drugs were also commonly detected. Another striking feature of this study was the prevalence of polysubstance use, with approximately one in seven drivers (14.1%) testing positive for more than one impairing substance. Social marketing campaigns or traffic policy designed to prevent impaired driving should continue to target alcohol as well as cannabis and other drugs and should be sensitive to the fact that many drivers use combinations of multiple impairing substances. The high prevalence of sedating medications, in multiple age ranges, suggests the need for better education on prescription practices and on use of sedating medications by drivers (including over the counter antihistamines).

Appendix A: Tables

Table 1. Count (percent) of injured drivers who test positive for impairing substances by age and sex

	National	Age group						Sex	
		15-18	19-24	25-34	35-44	45-54	55+	Female	Male
Total injured drivers	4976 (100.0%)	165 (100.0%)	654 (100.0%)	1121 (100.0%)	804 (100.0%)	787 (100.0%)	1445 (100.0%)	1614 (100.0%)	3362 (100.0%)
Alcohol									
BAC > 0	773 (15.5%)	30 (18.2%)	151 (23.1%)	248 (22.1%)	140 (17.4%)	100 (12.7%)	104 (7.2%)	167 (10.3%)	606 (18.0%)
0 < BAC < 0.05%	120 (2.4%)	6 (3.6%)	18 (2.8%)	36 (3.2%)	23 (2.9%)	12 (1.5%)	25 (1.7%)	22 (1.4%)	98 (2.9%)
0.05% ≤ BAC < 0.08%	62 (1.2%)	2 (1.2%)	13 (2.0%)	15 (1.3%)	12 (1.5%)	10 (1.3%)	10 (0.7%)	11 (0.7%)	51 (1.5%)
BAC ≥ 0.08%	591 (11.9%)	22 (13.3%)	120 (18.3%)	197 (17.6%)	105 (13.1%)	78 (9.9%)	69 (4.8%)	134 (8.3%)	457 (13.6%)
Cannabinoids									
COOH-THC > 0	1513 (30.4%)	69 (41.8%)	322 (49.2%)	461 (41.1%)	236 (29.4%)	169 (21.5%)	256 (17.7%)	346 (21.4%)	1167 (34.7%)
THC > 0	933 (18.8%)	40 (24.2%)	233 (35.6%)	284 (25.3%)	143 (17.8%)	105 (13.3%)	128 (8.9%)	184 (11.4%)	749 (22.3%)
THC ≥ 2 ng/mL	392 (7.9%)	18 (10.9%)	105 (16.1%)	120 (10.7%)	55 (6.8%)	34 (4.3%)	60 (4.2%)	86 (5.3%)	306 (9.1%)
THC ≥ 5 ng/mL	174 (3.5%)	7 (4.2%)	39 (6.0%)	64 (5.7%)	25 (3.1%)	11 (1.4%)	28 (1.9%)	38 (2.4%)	136 (4.0%)
Other recreational drugs[1]	540 (10.9%)	15 (9.1%)	79 (12.1%)	169 (15.1%)	121 (15.0%)	88 (11.2%)	68 (4.7%)	124 (7.7%)	416 (12.4%)
Sedating medications[2]	1029 (20.7%)	23 (13.9%)	122 (18.7%)	201 (17.9%)	167 (20.8%)	169 (21.5%)	347 (24.0%)	386 (23.9%)	643 (19.1%)
Opiates	547 (11.0%)	16 (9.7%)	53 (8.1%)	126 (11.2%)	100 (12.4%)	84 (10.7%)	168 (11.6%)	161 (10.0%)	386 (11.5%)
Any substance[3]	2526 (50.8%)	85 (51.5%)	407 (62.2%)	630 (56.2%)	443 (55.1%)	359 (45.6%)	602 (41.7%)	724 (44.9%)	1802 (53.6%)

1. Cocaine, Amphetamines
2. Antihistamines, Benzodiazepines, Z drugs, Antidepressants, Anticonvulsants, Antipsychotics
3. Alcohol, THC (excludes COOH-THC), other recreational drugs, sedating medications, and opiates

Table 2. Count (percent) of injured drivers who test positive for impairing substances by crash characteristics

	National	Treated and released	Admitted	Nighttime	Daytime	Multi-vehicle	Single-vehicle
Total injured drivers	4976 (100.0%)	3068 (100.0%)	1840 (100.0%)	1854 (100.0%)	3043 (100.0%)	2958 (100.0%)	2009 (100.0%)
Alcohol							
BAC > 0	773 (15.5%)	392 (12.8%)	372 (20.2%)	526 (28.4%)	227 (7.5%)	223 (7.5%)	548 (27.3%)
0 < BAC < 0.05%	120 (2.4%)	50 (1.6%)	69 (3.8%)	62 (3.3%)	56 (1.8%)	55 (1.9%)	65 (3.2%)
0.05% ≤ BAC < 0.08%	62 (1.2%)	26 (0.8%)	34 (1.8%)	38 (2.0%)	21 (0.7%)	23 (0.8%)	38 (1.9%)
BAC ≥ 0.08%	591 (11.9%)	316 (10.3%)	269 (14.6%)	426 (23.0%)	150 (4.9%)	145 (4.9%)	445 (22.2%)
Cannabinoids							
COOH-THC > 0	1513 (30.4%)	867 (28.3%)	627 (34.1%)	645 (34.8%)	841 (27.6%)	753 (25.5%)	755 (37.6%)
THC > 0	933 (18.8%)	525 (17.1%)	396 (21.5%)	405 (21.8%)	510 (16.8%)	459 (15.5%)	470 (23.4%)
THC ≥ 2 ng/mL	392 (7.9%)	225 (7.3%)	163 (8.9%)	172 (9.3%)	213 (7.0%)	190 (6.4%)	202 (10.1%)
THC ≥ 5 ng/mL	174 (3.5%)	106 (3.5%)	67 (3.6%)	82 (4.4%)	90 (3.0%)	83 (2.8%)	91 (4.5%)
Other recreational drugs[1]	540 (10.9%)	316 (10.3%)	217 (11.8%)	236 (12.7%)	298 (9.8%)	240 (8.1%)	298 (14.8%)
Sedating medications[2]	1029 (20.7%)	584 (19.0%)	427 (23.2%)	373 (20.1%)	639 (21.0%)	559 (18.9%)	467 (23.2%)
Opiates	547 (11.0%)	264 (8.6%)	273 (14.8%)	219 (11.8%)	317 (10.4%)	275 (9.3%)	270 (13.4%)
Any substance[3]	2526 (50.8%)	1429 (46.6%)	1060 (57.6%)	1079 (58.2%)	1399 (46.0%)	1266 (42.8%)	1253 (62.4%)

1. Cocaine, Amphetamines
2. Antihistamines, Benzodiazepines, Z drugs, Antidepressants, Anticonvulsants, Antipsychotics
3. Alcohol, THC (excludes COOH-THC), other recreational drugs, sedating medications, and opiates

Table 3. Count (percent) of injured drivers who test positive for impairing substances by region.

	National	British Columbia	Alberta	Saskatchewan	Ontario	Quebec	Atlantic Provinces*
Total injured drivers	4976 (100.0%)	1823 (100.0%)	1361 (100.0%)	368 (100.0%)	1005 (100.0%)	241 (100.0%)	178 (100.0%)
Alcohol							
BAC > 0	773 (15.5%)	212 (11.6%)	237 (17.4%)	68 (18.5%)	177 (17.6%)	40 (16.6%)	39 (21.9%)
0 < BAC < 0.05%	120 (2.4%)	42 (2.3%)	27 (2.0%)	10 (2.7%)	26 (2.6%)	12 (5.0%)	3 (1.7%)
0.05% ≤ BAC < 0.08%	62 (1.2%)	18 (1.0%)	14 (1.0%)	7 (1.9%)	17 (1.7%)	3 (1.2%)	3 (1.7%)
BAC ≥ 0.08%	591 (11.9%)	152 (8.3%)	196 (14.4%)	51 (13.9%)	134 (13.3%)	25 (10.4%)	33 (18.5%)
Cannabinoids							
COOH-THC > 0	1513 (30.4%)	480 (26.3%)	399 (29.3%)	140 (38.0%)	340 (33.8%)	58 (24.1%)	96 (53.9%)
THC > 0	933 (18.8%)	304 (16.7%)	233 (17.1%)	93 (25.3%)	200 (19.9%)	39 (16.2%)	64 (36.0%)
THC ≥ 2 ng/mL	392 (7.9%)	114 (6.3%)	99 (7.3%)	45 (12.2%)	77 (7.7%)	22 (9.1%)	35 (19.7%)
THC ≥ 5 ng/mL	174 (3.5%)	42 (2.3%)	49 (3.6%)	24 (6.5%)	30 (3.0%)	12 (5.0%)	17 (9.6%)
Other recreational drugs[1]	540 (10.9%)	176 (9.7%)	159 (11.7%)	44 (12.0%)	94 (9.4%)	30 (12.4%)	37 (20.8%)
Sedating medications[2]	1029 (20.7%)	313 (17.2%)	311 (22.9%)	85 (23.1%)	208 (20.7%)	60 (24.9%)	52 (29.2%)
Opiates	547 (11.0%)	160 (8.8%)	168 (12.3%)	52 (14.1%)	116 (11.5%)	30 (12.4%)	21 (11.8%)
Any substance[3]	2526 (50.8%)	786 (43.1%)	758 (55.7%)	216 (58.7%)	529 (52.6%)	127 (52.7%)	110 (61.8%)

1. Cocaine,
Amphetamines
2. Antihistamines, Benzodiazepines, Z drugs, Antidepressants, Anticonvulsants, Antipsychotics
3. Alcohol, THC (excludes COOH-THC), other recreational drugs, sedating medications, and opiates
*Atlantic Provinces: New Brunswick, Nova Scotia and Newfoundland and Labrador

Table 4. Polysubstance Use: Count (percent) of injured drivers who test positive for one or more classes of impairing substance in Canada.

	National	British Columbia	Alberta	Saskatchewan	Ontario	Quebec	Atlantic Provinces
Total injured drivers	4976 (100.0%)	1823 (100.0%)	1361 (100.0%)	368 (100.0%)	1005 (100.0%)	241 (100.0%)	178 (100.0%)
Number of substances[1]							
1	1555 (31.2%)	497 (27.3%)	490 (36.0%)	118 (32.1%)	328 (32.6%)	77 (32.0%)	45 (25.3%)
2	704 (14.1%)	213 (11.7%)	199 (14.6%)	75 (20.4%)	146 (14.5%)	36 (14.9%)	35 (19.7%)
3 or more	267 (5.4%)	76 (4.2%)	69 (5.1%)	23 (6.2%)	55 (5.5%)	14 (5.8%)	30 (16.9%)
Alcohol and THC							
BAC > 0 & THC > 0	249 (5.0%)	63 (3.5%)	78 (5.7%)	30 (8.2%)	48 (4.8%)	10 (4.1%)	20 (11.2%)
BAC ≥ 0.05% & THC ≥ 2 ng/mL	72 (1.4%)	16 (0.9%)	20 (1.5%)	10 (2.7%)	14 (1.4%)	4 (1.7%)	8 (4.5%)
Alcohol and other substances							
BAC > 0 & Other recreational drugs	165 (3.3%)	41 (2.2%)	50 (3.7%)	13 (3.5%)	34 (3.4%)	11 (4.6%)	16 (9.0%)
BAC > 0 & Sedating medications	194 (3.9%)	52 (2.9%)	60 (4.4%)	12 (3.3%)	42 (4.2%)	13 (5.4%)	15 (8.4%)
BAC > 0 & Opiates	93 (1.9%)	23 (1.3%)	26 (1.9%)	8 (2.2%)	23 (2.3%)	8 (3.3%)	5 (2.8%)
THC and other substances							
THC > 0 & Other recreational drugs	178 (3.6%)	48 (2.6%)	40 (2.9%)	26 (7.1%)	35 (3.5%)	9 (3.7%)	20 (11.2%)
THC > 0 & Sedating medications	217 (4.4%)	66 (3.6%)	49 (3.6%)	21 (5.7%)	46 (4.6%)	10 (4.1%)	25 (14.0%)
THC > 0 & Opiates	123 (2.5%)	34 (1.9%)	29 (2.1%)	13 (3.5%)	29 (2.9%)	8 (3.3%)	10 (5.6%)

1. Alcohol, THC, other recreational drugs, sedating medications, and opiates are considered distinct substances
*Atlantic Provinces: New Brunswick, Nova Scotia and Newfoundland and Labrador

Appendix B: Figures

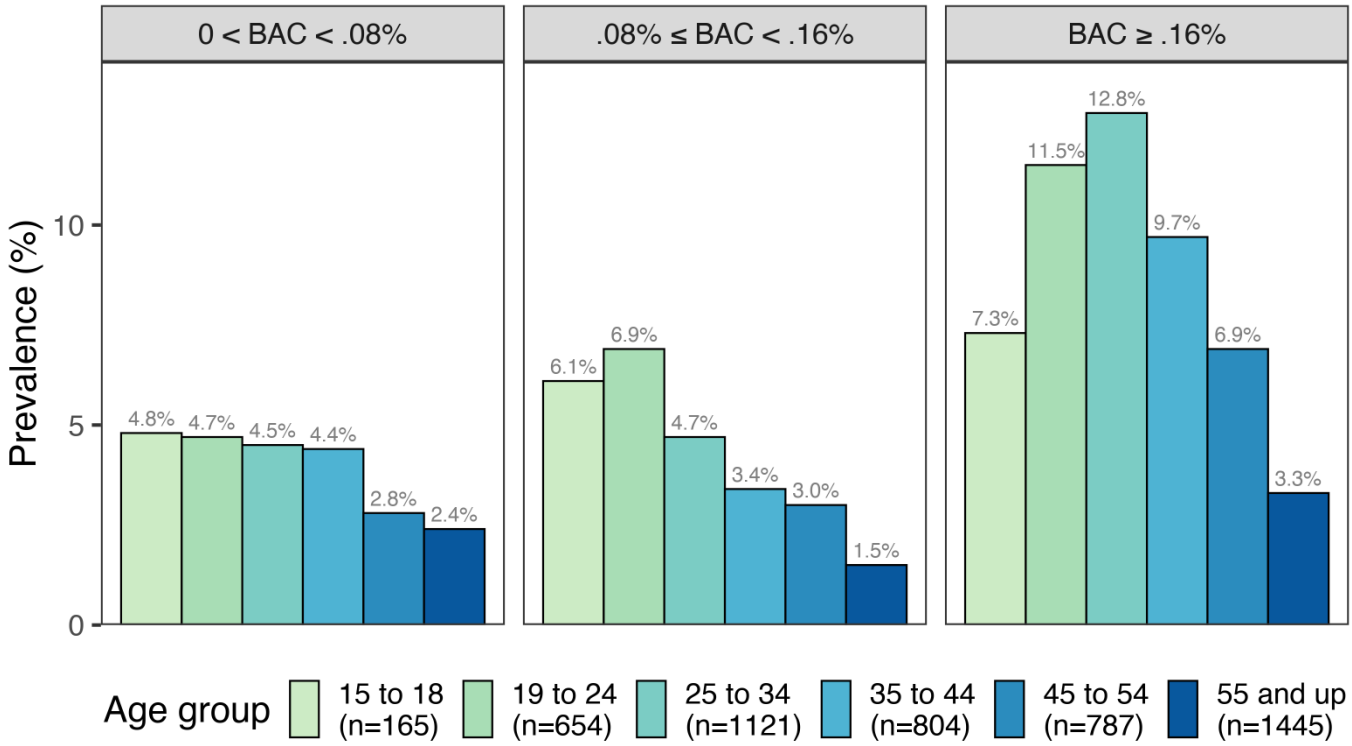

Figure 1. Prevalence of alcohol use among injured drivers in Canada, by age group

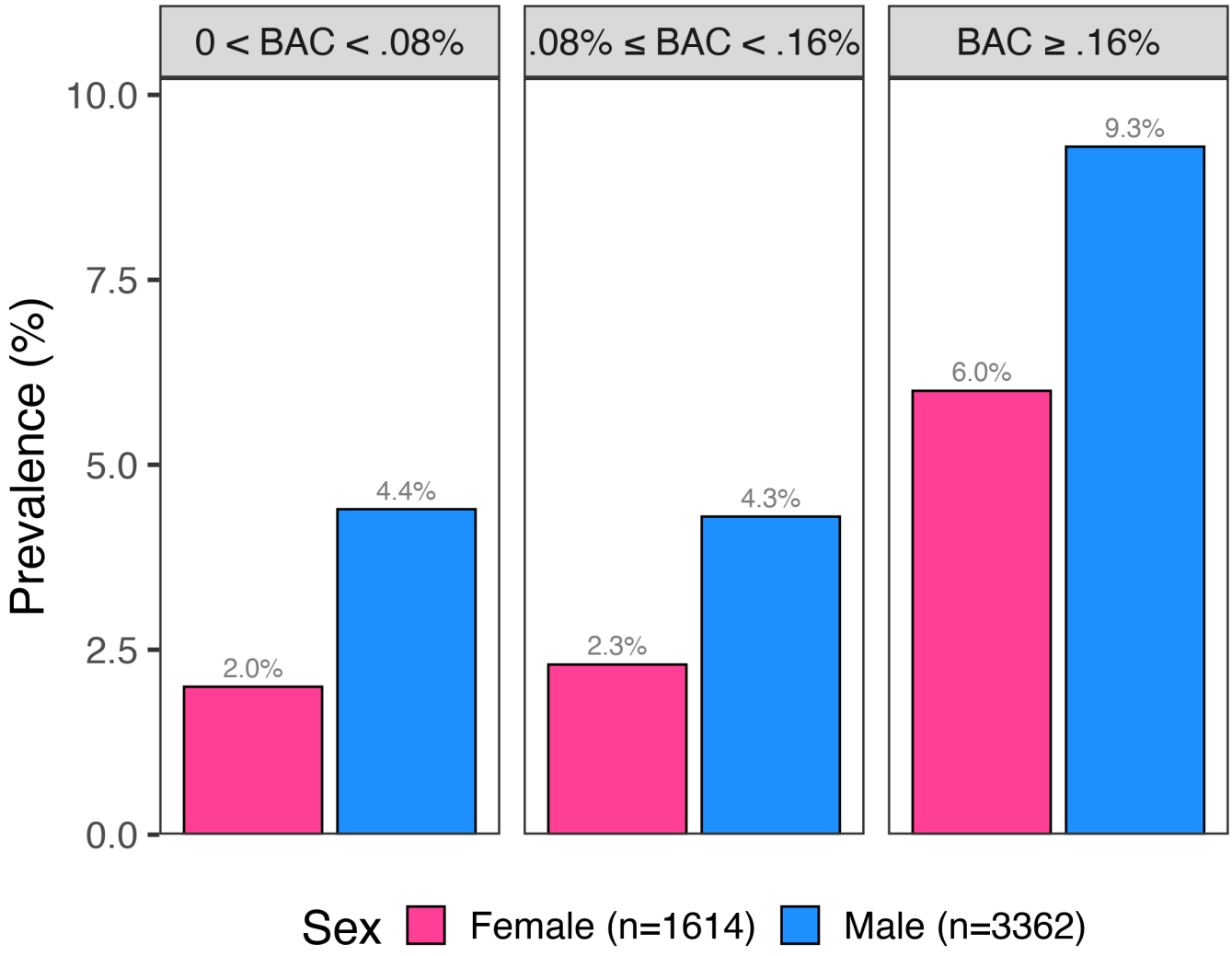

Figure 2. Prevalence of alcohol use among injured drivers in Canada, by sex

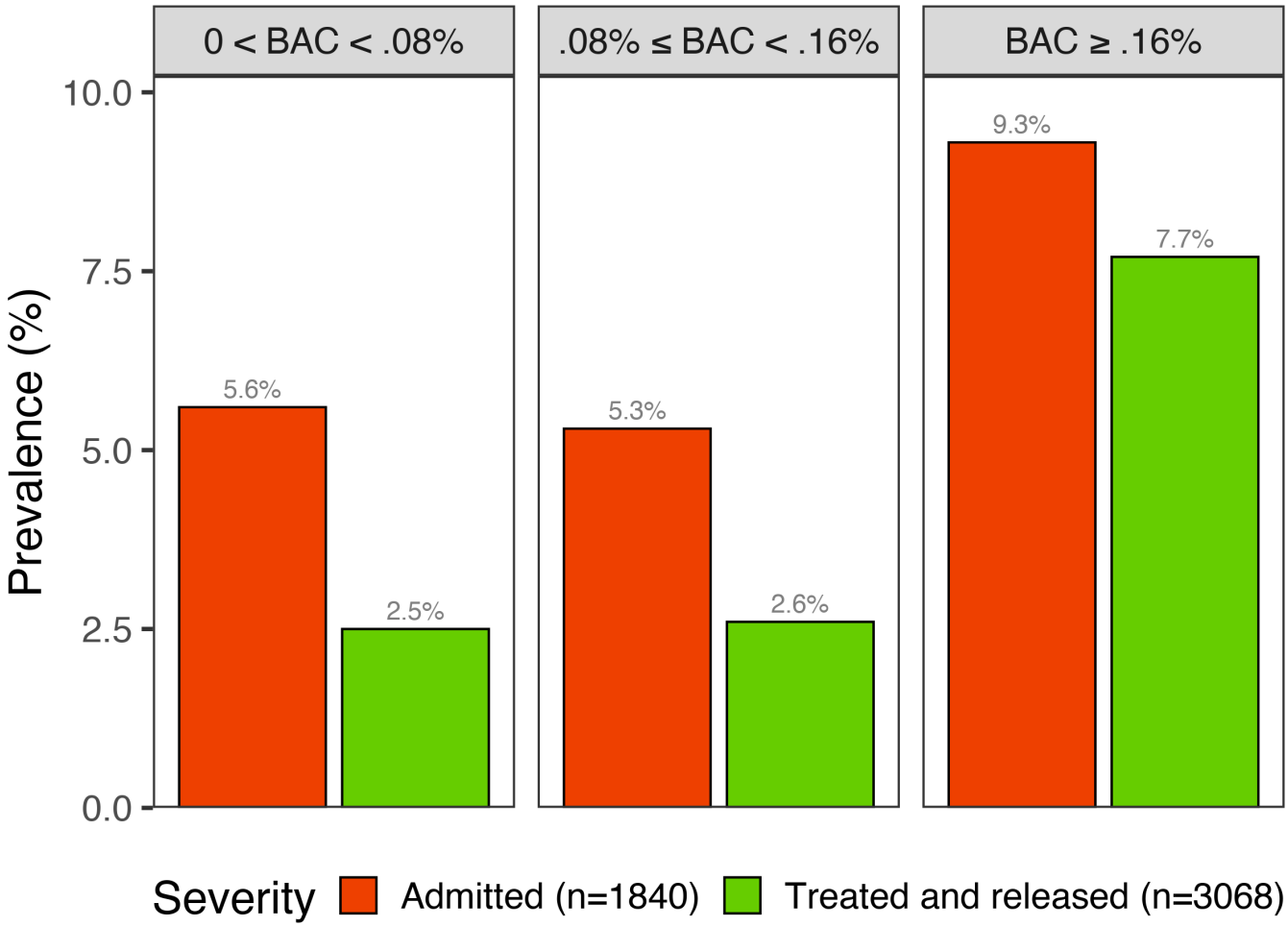

Figure 3. Prevalence of alcohol use among injured drivers in Canada, by disposition

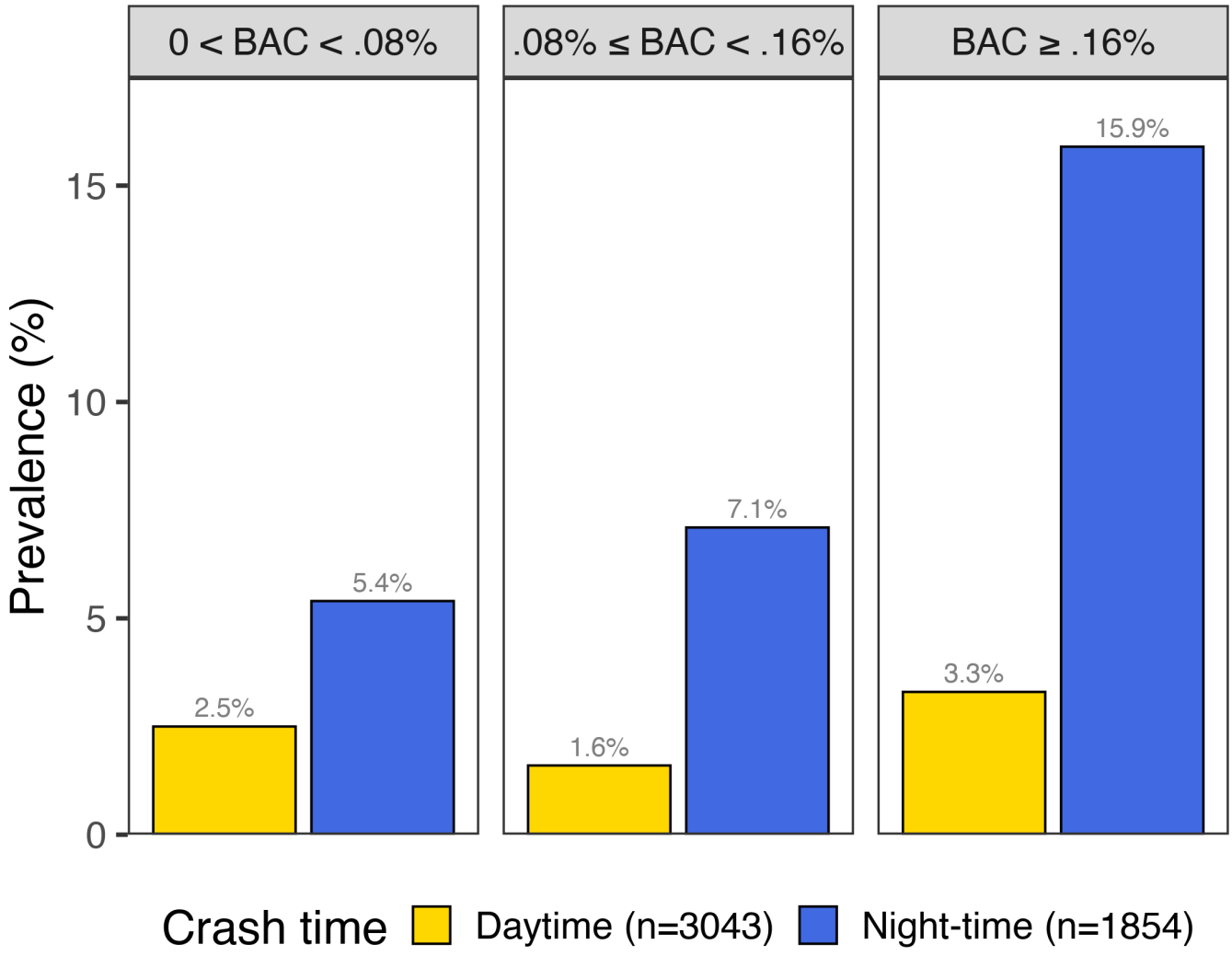

Figure 4. Prevalence of alcohol use among injured drivers in Canada, by time of crash

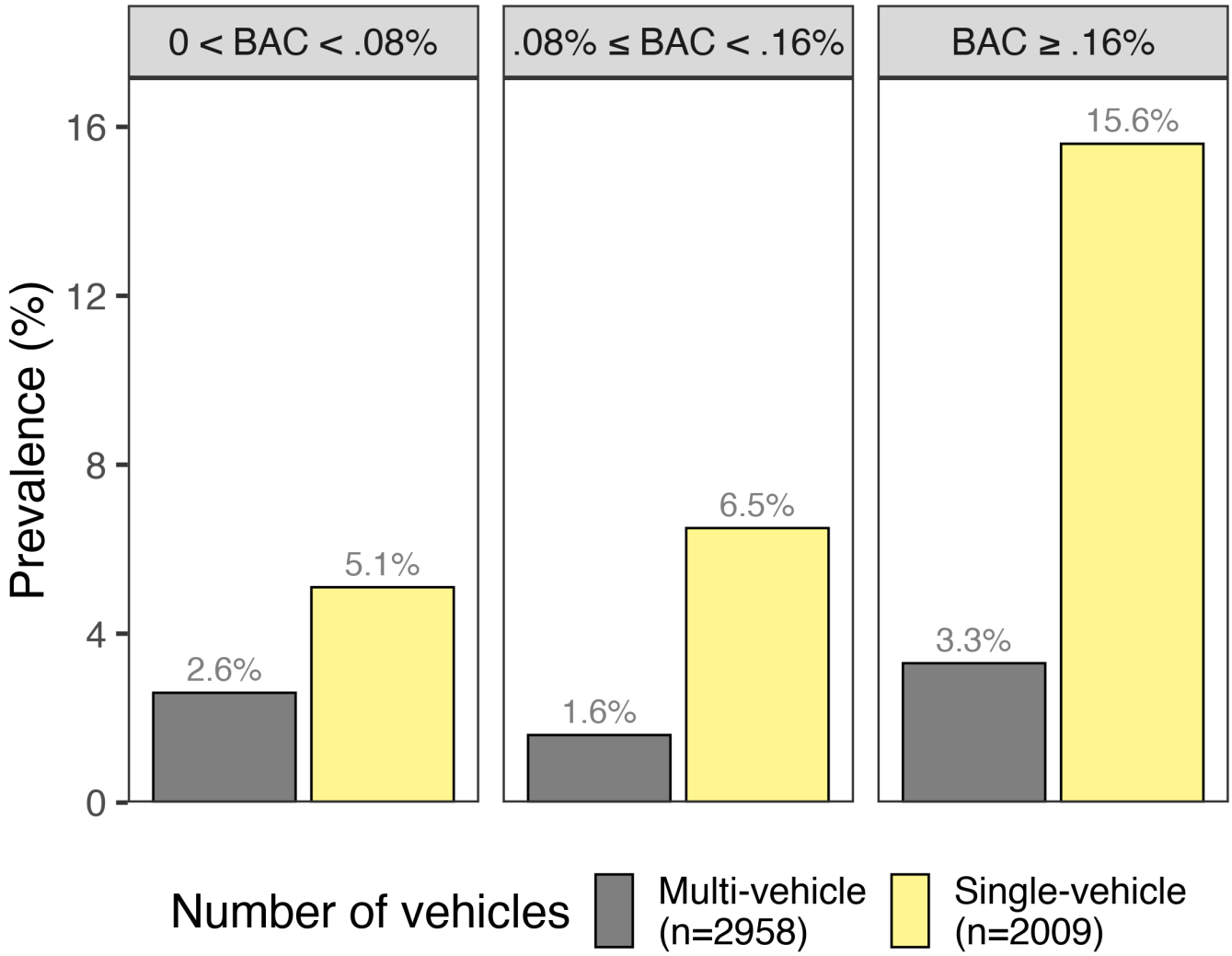

Figure 5. Prevalence of alcohol use among injured drivers in Canada, by number of vehicles involved in the crash

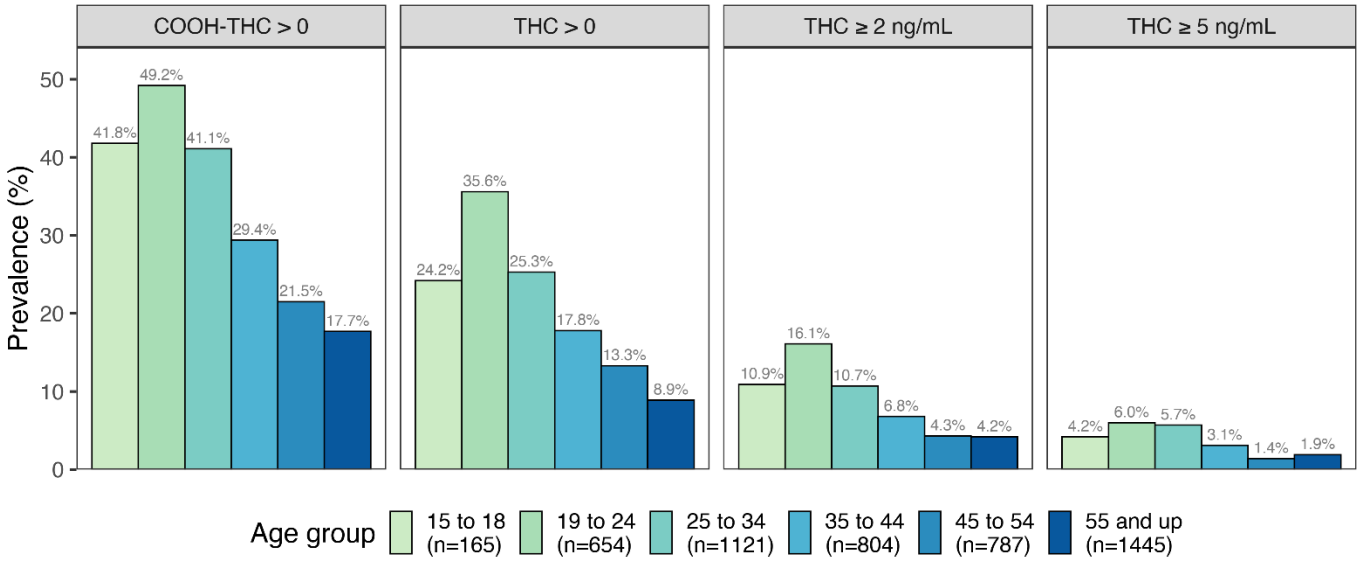

Figure 6. Prevalence of cannabinoids among injured drivers in Canada, by age group

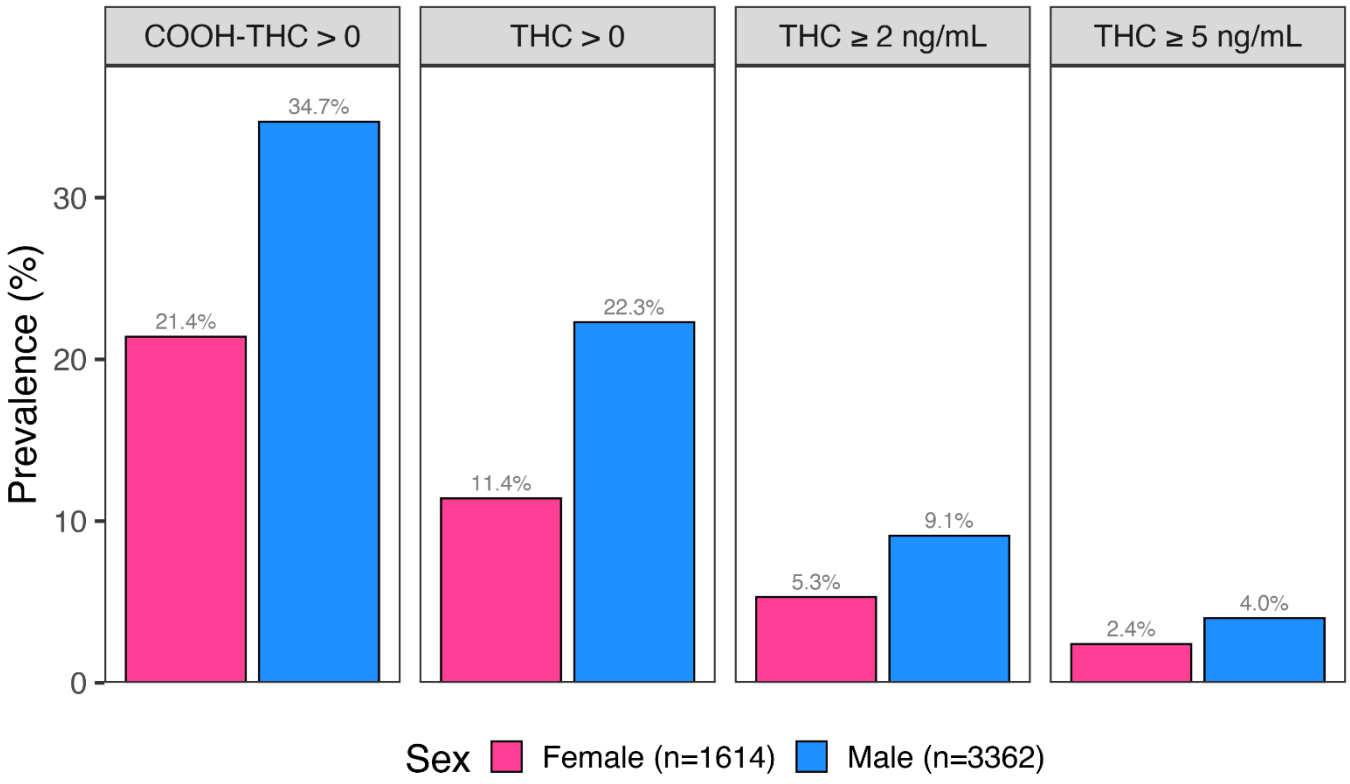

Figure 7. Prevalence of cannabinoids among injured drivers in Canada, by sex

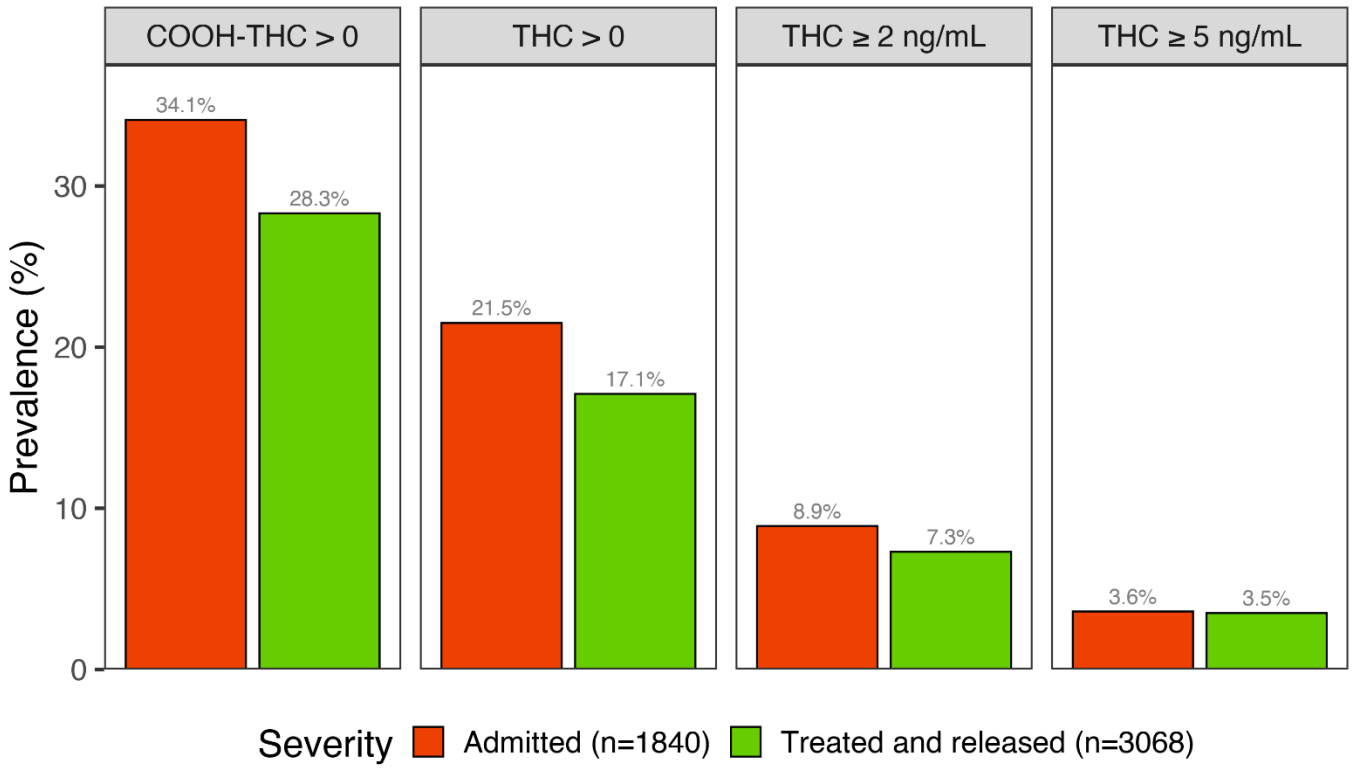

Figure 8. Prevalence of cannabinoids among injured drivers in Canada, by disposition

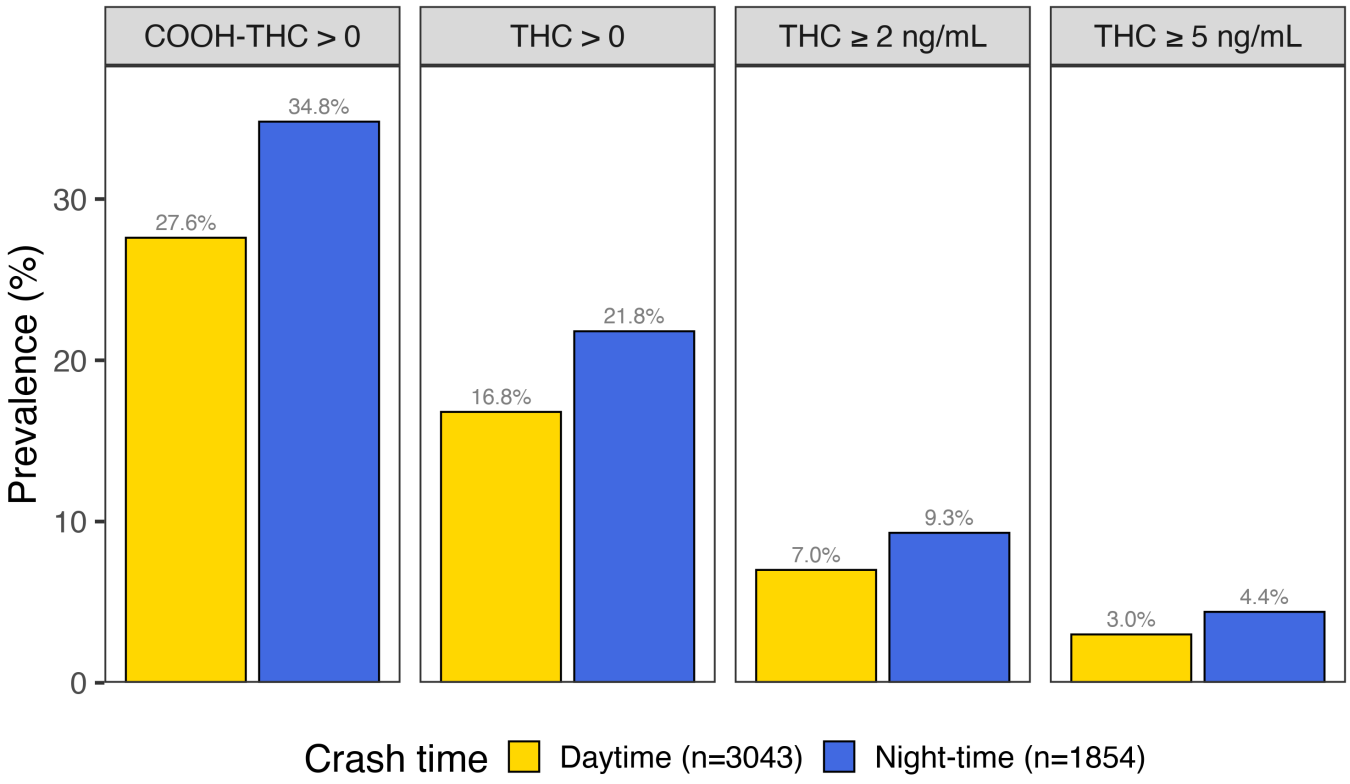

Figure 9. Prevalence of cannabinoids among injured drivers in Canada, by time of crash

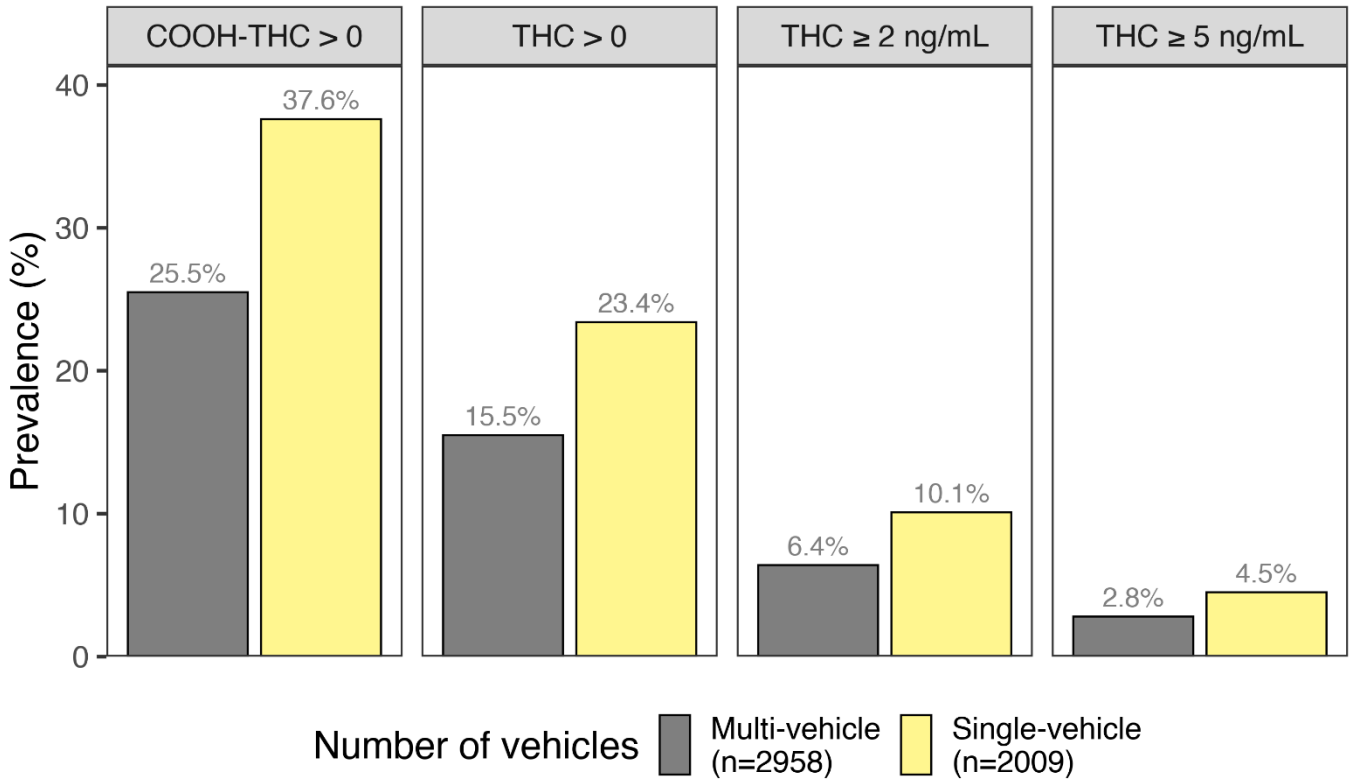

Figure 10. Prevalence of cannabinoids among injured drivers in Canada, by number of vehicles involved in the crash

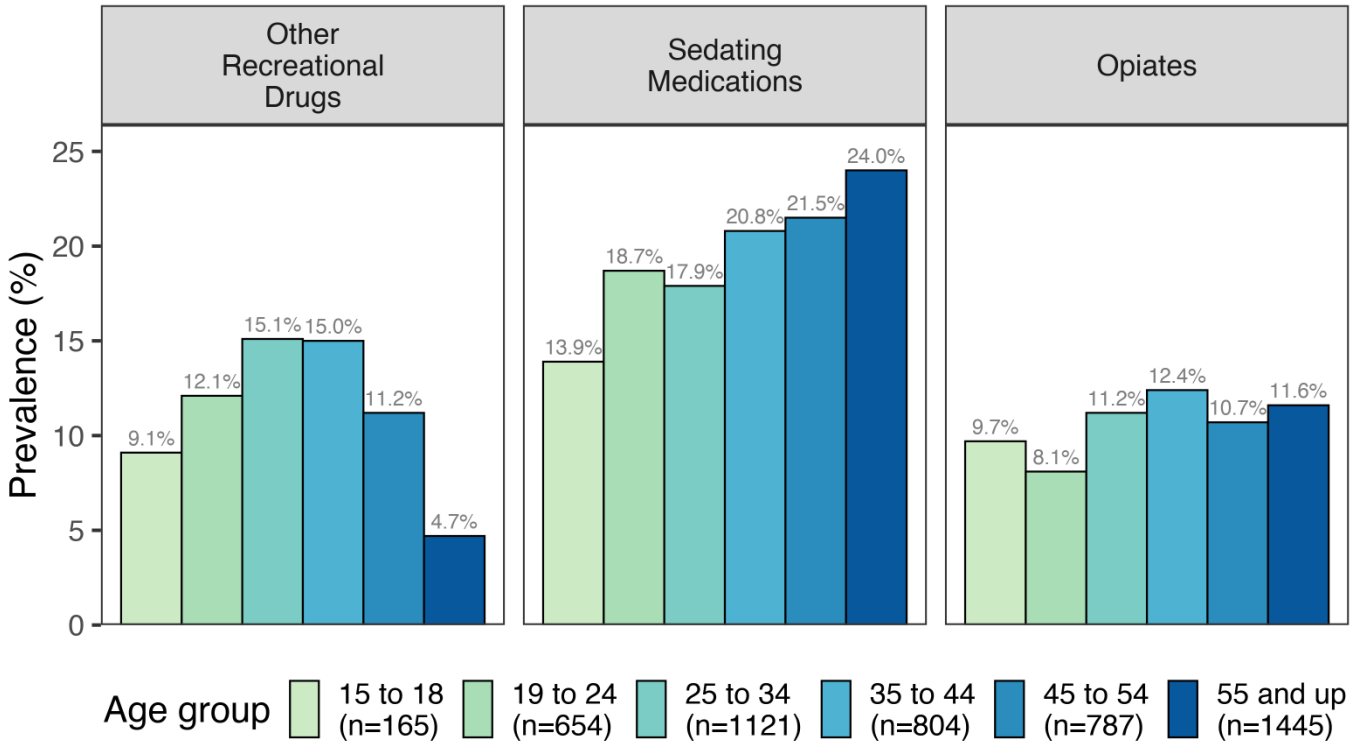

Figure 11. Prevalence of other recreational drugs, medications, and opiates among injured drivers in Canada, by age group

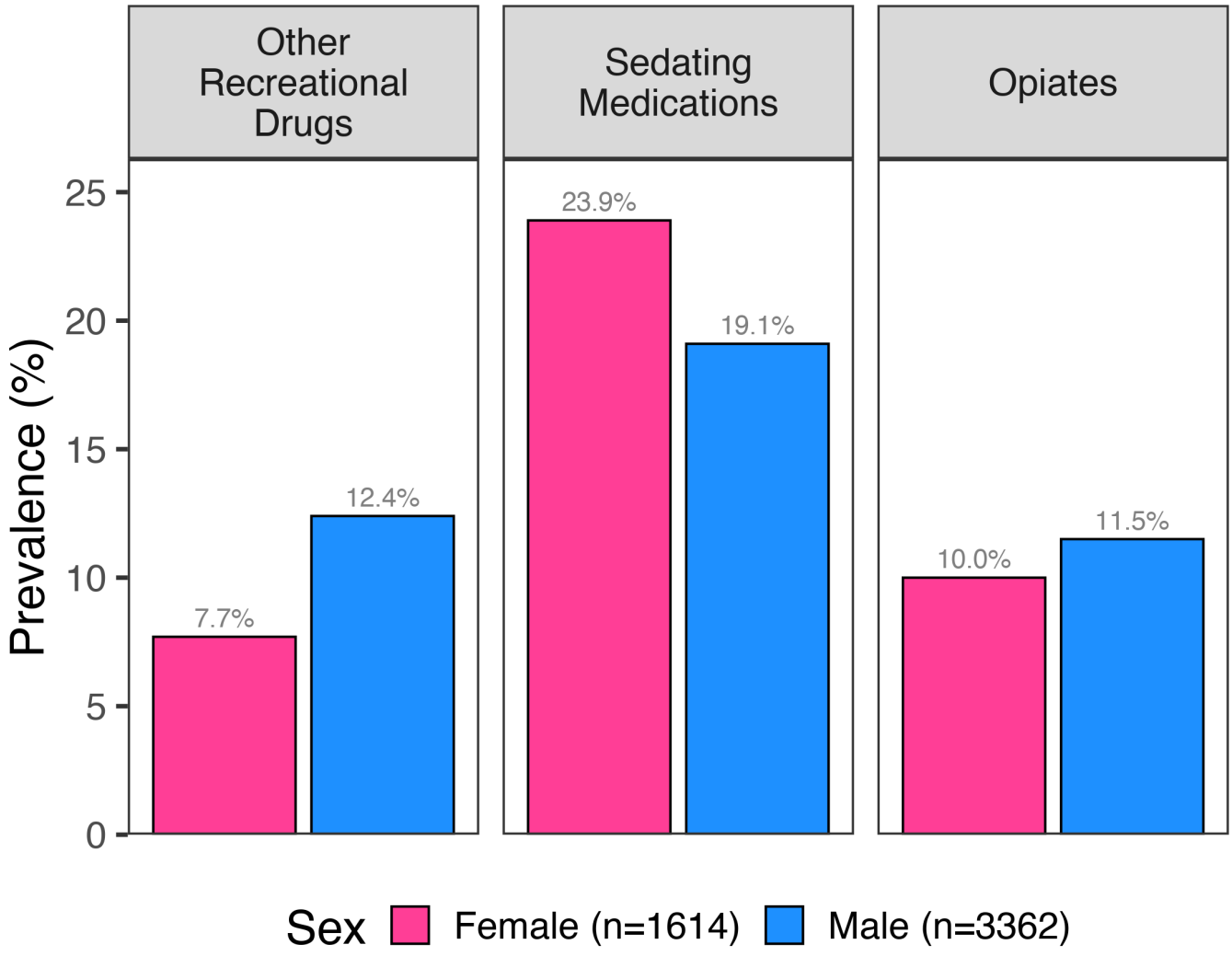

Figure 12. Prevalence of other recreational drugs, medications, and opiates among injured drivers in Canada, by sex

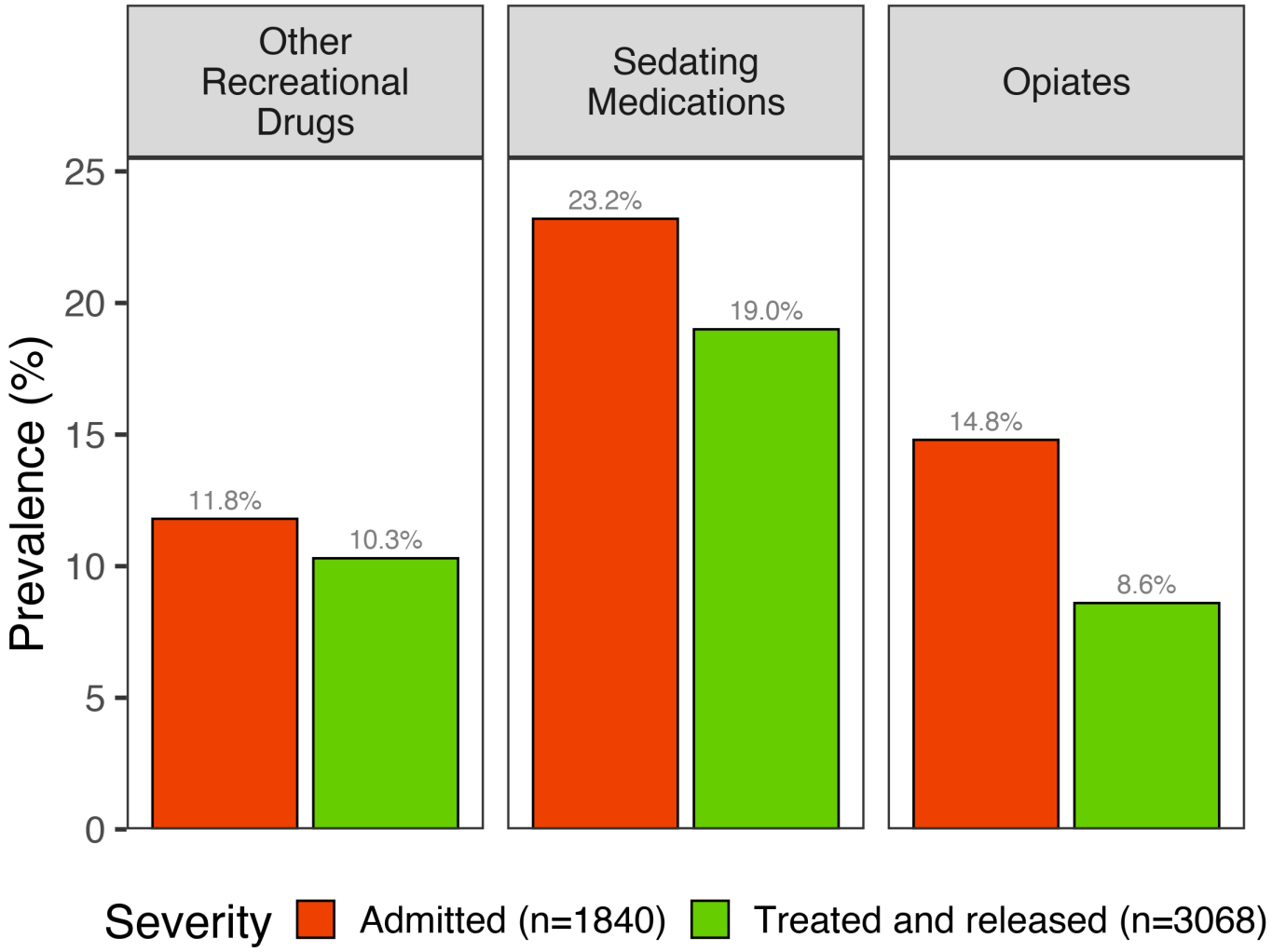

Figure 13. Prevalence of other recreational drugs, medications, and opiates among injured drivers in Canada, by disposition

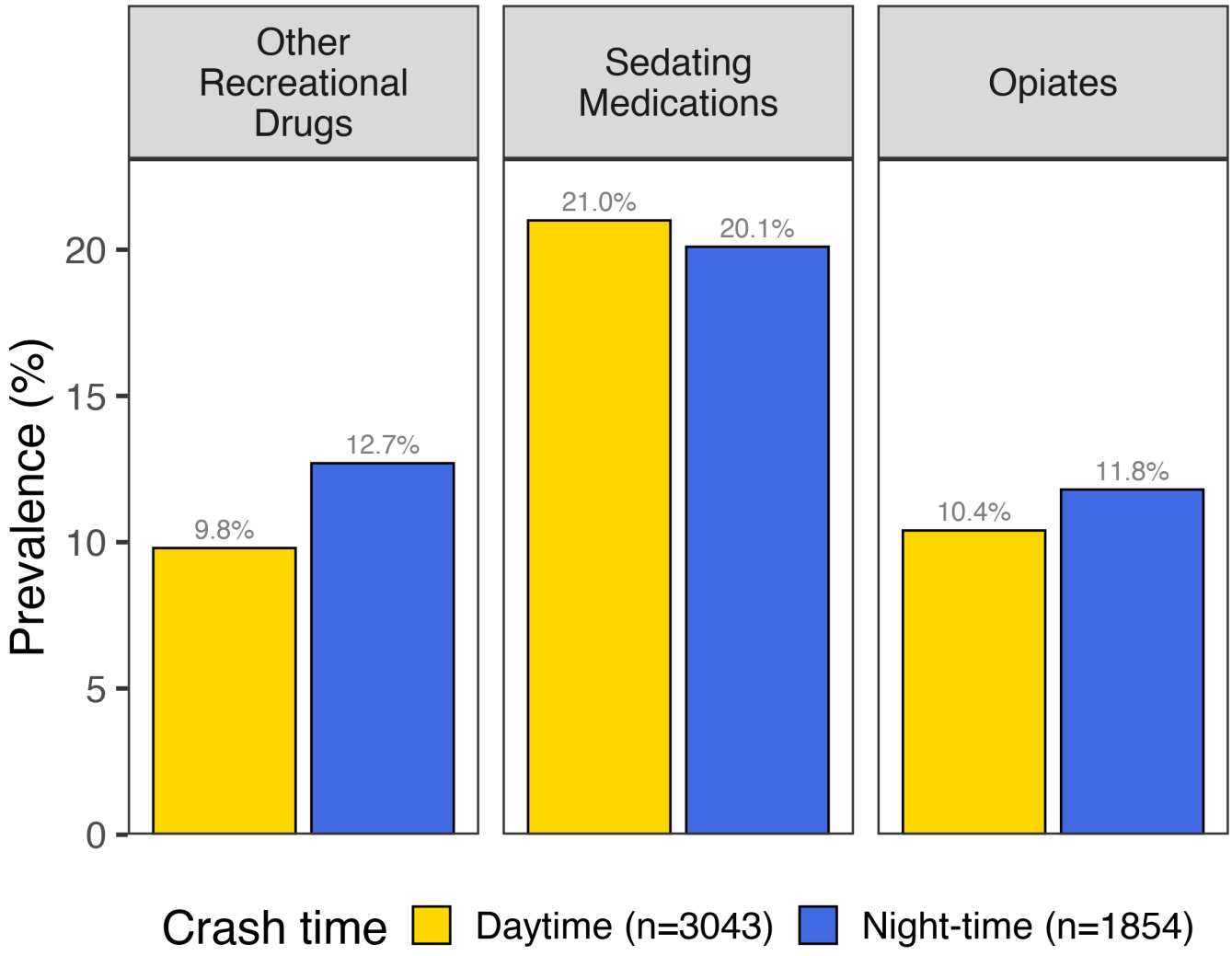

Figure 14. Prevalence of other recreational drugs, medications, and opiates among injured drivers in Canada, by time of crash

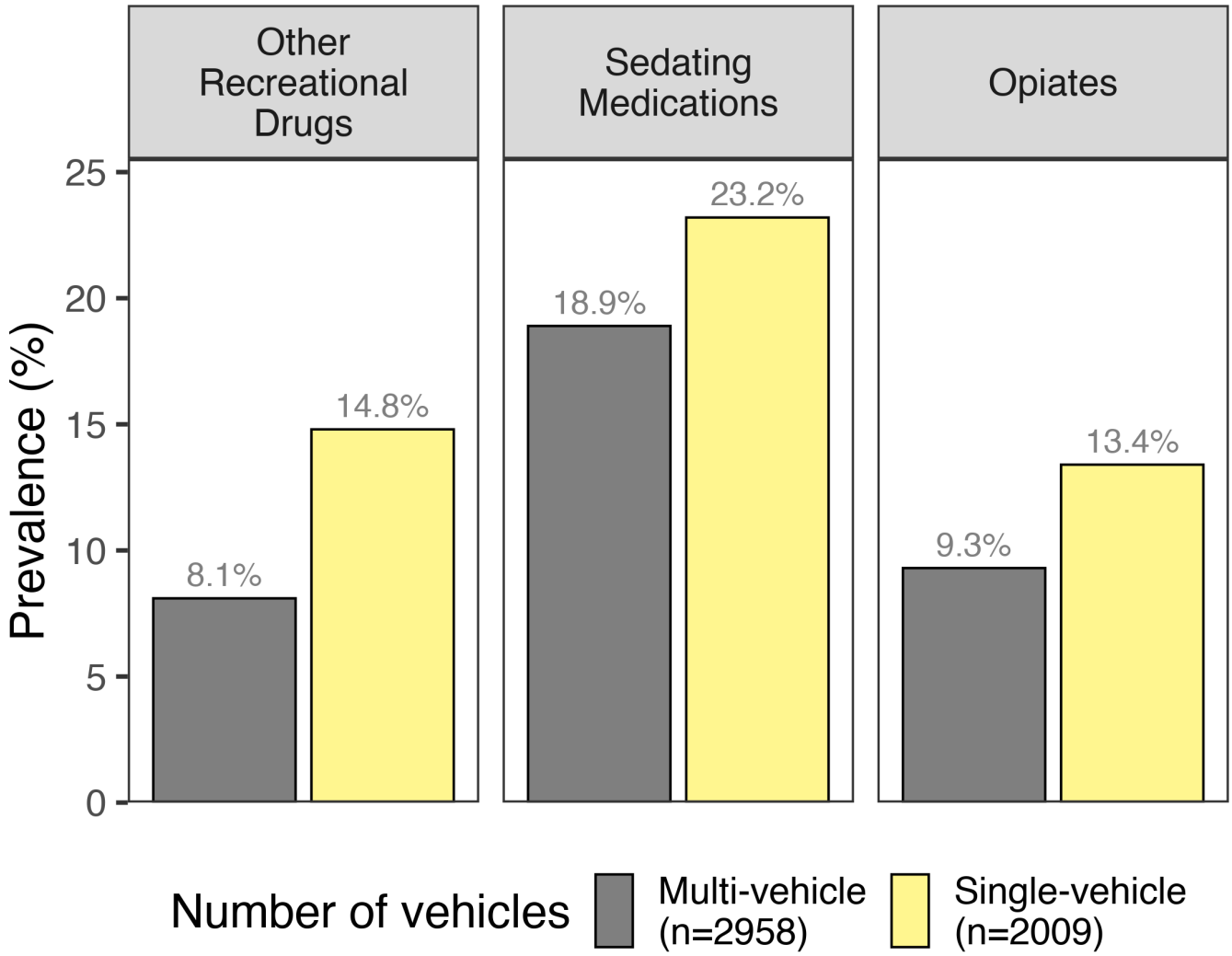

Figure 15: Prevalence of other recreational drugs, medications, and opiates among injured drivers in Canada, by number of vehicles involved in the crash.

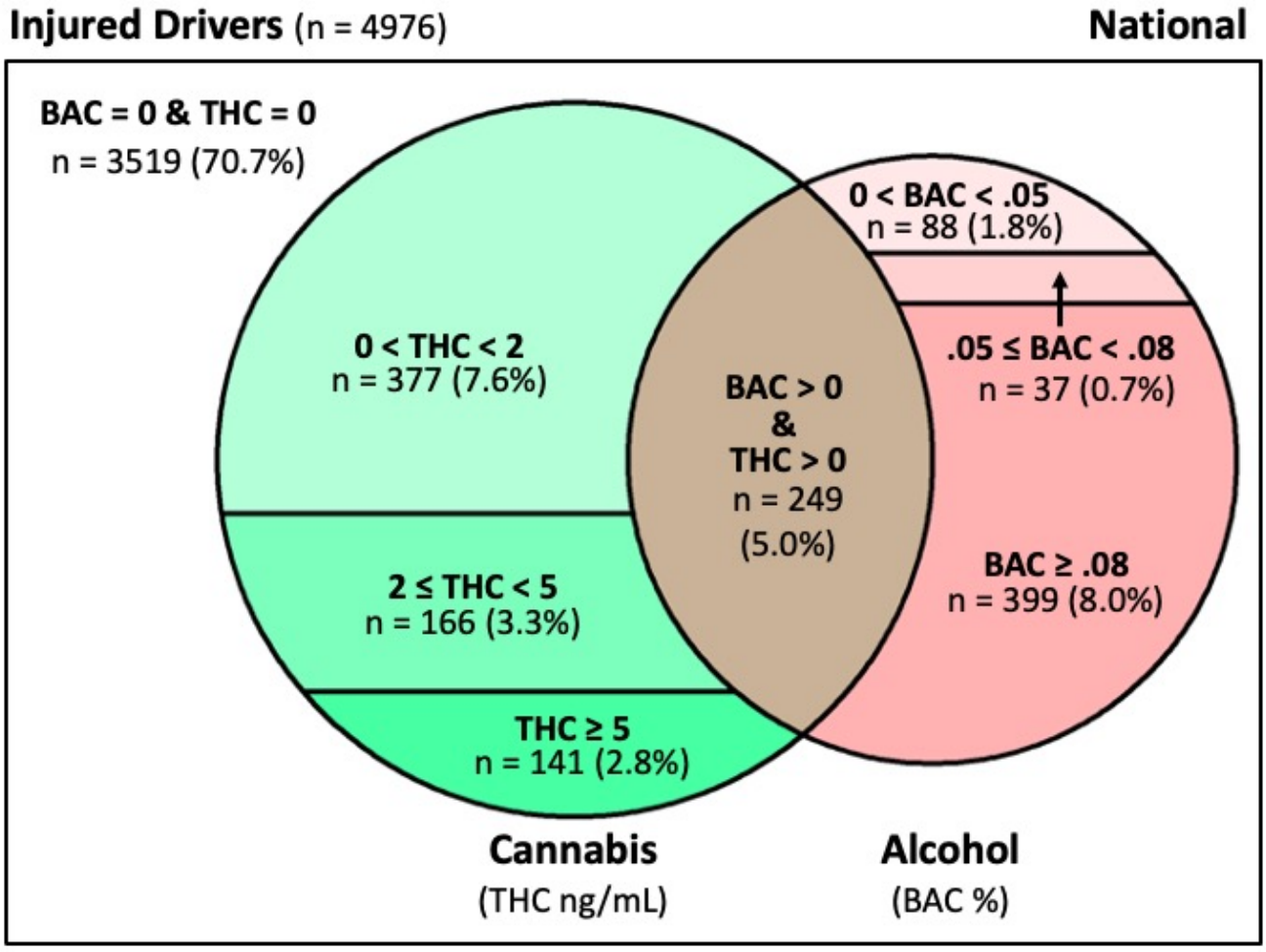

Figure 16. Use of alcohol, cannabis or both among injured drivers in Canada. Area in brown represents 249 drivers who tested positive for both alcohol and THC. Majority of THC positive drivers (in green) had relatively low levels compared to most of the alcohol positive drivers (in red) who had BAC above the 0.08% legal limit.

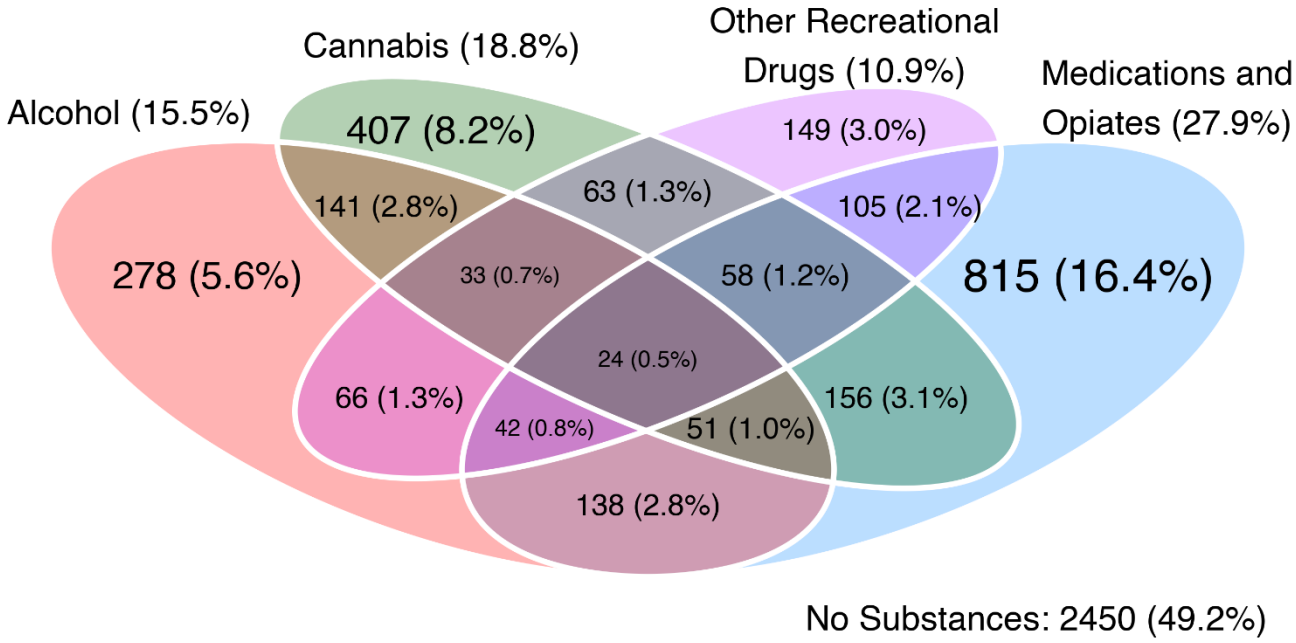

Figure 17. Polysubstance use among injured drivers in Canada. The overlapping ellipses represent the numbers of drivers testing positive for various combinations of impairing substances. The cannabis ellipse counts drivers who tested positive for THC. For example, 141 drivers tested positive for alcohol and cannabis only.

References

1. Hunault CC, Bocker KB, Stellato RK, Kenemans JL, de Vries I, Meulenbelt J. Acute subjective effects after smoking joints containing up to 69 mg Delta9-tetrahydrocannabinol in recreational users: a randomized, crossover clinical trial. Psychopharmacology (Berl). 2014;231(24):4723-33.

2. Karschner EL, Swortwood MJ, Hirvonen J, Goodwin RS, Bosker WM, Ramaekers JG, et al. Extended plasma cannabinoid excretion in chronic frequent cannabis smokers during sustained abstinence and correlation with psychomotor performance. Drug Testing & Analysis. 2016;8(7):682-9.

3. Smith HW, Popham RE. Blood alcohol levels in relation to driving. Can Med Assoc J. 1951;65(4):325-8.

4. Borkenstein RF, Crowther RF, Shumate RP, Ziel WB, Zylman R. The role of the drinking driver in traffic accidents (the Grand Rapids Study). Blutalcohol. 1974;11 (Suppl):7-13.

5. Zador PL, Krawchuk SA, Voas RB. Alcohol-related relative risk of driver fatalities and driver involvement in fatal crashes in relation to driver age and gender: an update using 1996 data. J Stud Alcohol. 2000;61(3):387-95.

6. Blomberg RD, Peck RC, H. M, Burns M, Fiorentino D, editors. The Long Beach/Fort Lauderdale Relative Risk Study. International Council on Alcohol, Drugs, and Traffic Safety (ICADTS); 2007; Seattle, WA.

7. Tippetts AS, Voas RB, Fell JC, Nichols JL, Tippetts AS, Voas RB, et al. A meta-analysis of .08 BAC laws in 19 jurisdictions in the United States. Accid Anal Prev. 2005;37(1):149-61.

8. Tay R. The effectiveness of enforcement and publicity campaigns on serious crashes involving young male drivers: Are drink driving and speeding similar? Accident Analysis & Prevention. 2005;37(5):922-9.

9. Tay R. Drink driving enforcement and publicity campaigns: are the policy recommendations sensitive to model specification? Accid Anal Prev. 2005;37(2):259-66.

10. Rothschild ML, Mastin B, Miller TW. Reducing alcohol-impaired driving crashes through the use of social marketing. Accid Anal Prev. 2006;38(6):1218-30.

11. Asbridge. The criminalization of impaired driving in Canada: Assessing the deterrent impact of Canada's first per se law. J Stud Alcohol. 2004;65(4):450.

12. Simpson H, Singhal D, Vanlaar W, Mayhew D. The road safety monitor: Drugs and driving. Ottawa, Ontario, Canada: Traffic Injury Research Foundation; 2006.

13. Ialomiteanu AR, Hamilton HAA, E.M., Mann RE. Substance Use, Mental Health and Well Being Among Ontario Adults. Toronto, Ontario: Centre for Addictions and Mental Health; 2016.

14. Beasley E, Beirness D. Drug use by fatally injured drivers in Canada. (2000 - 2008). Ottawa, ON: Canadian Centre on Substance Abuse. 2011.

15. Beasley E, Beirness D. Drug and Alcohol use among Drivers - the 2010 BC Roadside Survey. BC Injury Prevention Conference; Vancouver2010.

16. Beirness D, Beasley E, McClafferty K. Alcohol and drug use among drivers in Ontario: Findings from the 2014 roadside survey Toronto, Ontario: Ontario Ministry of Transportation; 2015.

17. Solomon R, Clarizio M. Total Crash Deaths Involving Alcohol and/or Drugs in Canada, by Jurisdiction: 2012. London, Ontario: MADD Canada; 2016.

18. Brubacher JR, Chan H, Martz W, Schreiber W, Asbridge M, Eppler J, et al. Prevalence of alcohol and drug use in injured British Columbia drivers. BMJ Open. 2016;6(3).

19. Hartman RL, Brown TL, Milavetz G, Spurgin A, Pierce RS, Gorelick DA, et al. Cannabis effects on driving lateral control with and without alcohol. Drug Alcohol Depend. 2015;154:25-37.

20. Downey LA, King R, Papafotiou K, Swann P, Ogden E, Boorman M, et al. The effects of cannabis and alcohol on simulated driving: Influences of dose and experience. Accid Anal Prev. 2013;50:879-86.

21. Dubois S, Mullen N, Weaver B, Bédard M. The combined effects of alcohol and cannabis on driving: Impact on crash risk. Forensic Sci Int. 2015;248:94-100.

22. Ronen A, Gershon P, Drobiner H, Rabinovich A, Bar-Hamburger R, Mechoulam R, et al. Effects of THC on driving performance, physiological state and subjective feelings relative to alcohol. Accid Anal Prev. 2008;40(3):926-34.

23. Grotenhermen F, Leson G, Berghaus G, Drummer OH, Krüger HP, Longo M, et al. Developing Science-Based Per Se Limits for Driving under the Influence of Cannabis (DUIC): Findings and Recommendations by an Expert Panel. Report International Association for Cannabis as Medicine.; 2005.

24. Sewell RA, Poling J, Sofuoglu M, Sewell RA, Poling J, Sofuoglu M. The effect of cannabis compared with alcohol on driving. Am J Addict. 2009;18(3):185-93.

25. Ramaekers JG, Berghaus G, van Laar M, Drummer OH. Dose related risk of motor vehicle crashes after cannabis use. Drug Alcohol Depend. 2004;73(2):109-19.

26. Berghaus G, Scheer N, Schmidt P, editors. Effects of Cannabis on Psychomotor Skills and Driving Performance - a Metaanalysis of Experimental Studies. International Council on Alcohol Drugs and Traffic Safety (ICADTS); 1995; Adelaide, Australia.

27. Grotenhermen F, Leson G, Berghaus G, Drummer OH, Kruger HP, Longo M, et al. Developing limits for driving under cannabis. Addiction. 2007;102(12):1910-7.

28. Berghaus G, Sticht G, Grellner W, Lenz D, Naumann T, Wiesenmuller S. Meta-analysis of empirical studies concerning the effects of medicines and illegal drugs including pharmacokinetics on safe driving. 2010.

29. Vindenes V, Jordbru D, Knapskog A-B, Kvan E, Mathisrud G, Slordal L, et al. Impairment based legislative limits for driving under the influence of non-alcohol drugs in Norway. Forensic Sci Int. 2012;219(1-3):1-11.

30. Desrosiers NA, Ramaekers JG, Chauchard E, Gorelick DA, Huestis MA. Smoked cannabis' psychomotor and neurocognitive effects in occasional and frequent smokers. J Anal Toxicol. 2015;39(4):251-61.

31. Broyd SJ, van Hell HH, Beale C, Yucel M, Solowij N. Acute and Chronic Effects of Cannabinoids on Human Cognition-A Systematic Review. Biol Psychiatry. 2016;79(7):557-67.

32. Colizzi M, Bhattacharyya S. Cannabis use and the development of tolerance: a systematic review of human evidence. Neurosci Biobehav Rev. 2018;93:1-25.

33. Soar K, Mason C, Potton A, Dawkins L. Neuropsychological effects associated with recreational cocaine use. Psychopharmacology (Berl). 2012;222(4):633-43.

34. Ramaekers JG, Kuypers KPC, Bosker WM, Brookhuis KA, Veldstra JA, Simons R, et al. Effects of stimulant drugs on actual and simulated driving: perspectives from four experimental studies conducted as part of the DRUID research consortium. Psychopharmacology (Berl). 2012;222(3):413-8.

35. Wittmann M, Leland DS, Churan J, Paulus MP. Impaired time perception and motor timing in stimulant-dependent subjects. Drug Alcohol Depend. 2007;90(2-3):183-92.

36. Fillmore MT, Rush CR, Hays L. Acute effects of oral cocaine on inhibitory control of behavior in humans. Drug Alcohol Depend. 2002;67(2):157-67.

37. Rush CR, Baker RW, Wright K. Acute physiological and behavioral effects of oral cocaine in humans: a dose-response analysis. Drug Alcohol Depend. 1999;55(1-2):1-12.

38. Foltin RW, Fischman MW, Pippen PA, Kelly TH. Behavioral effects of cocaine alone and in combination with ethanol or marijuana in humans. Drug Alcohol Depend. 1993;32(2):93-106.

39. Smith JL, Mattick RP, Jamadar SD, Iredale JM. Deficits in behavioural inhibition in substance abuse and addiction: a meta-analysis. Drug Alcohol Depend. 2014;145:1-33.

40. Spronk DB, van Wel JH, Ramaekers JG, Verkes RJ. Characterizing the cognitive effects of cocaine: a comprehensive review. Neurosci Biobehav Rev. 2013;37(8):1838-59.

41. Gustavsen I, Hjelmeland K, Bernard JP, Morland J. Psychomotor performance after intake of zopiclone compared with intake of ethanol: a randomized, controlled, double-blinded trial. J Clin Psychopharmacol. 2011;31(4):481-8.

42. Conen S, Theunissen EL, Van Oers ACM, Valiente R, Ramaekers JG. Acute and subchronic effects of bilastine (20 and 40 mg) and hydroxyzine (50 mg) on actual driving performance in healthy volunteers. J Psychopharmacol. 2011;25(11):1517-23.

43. Zacny JP, Lichtor SA. Within-subject comparison of the psychopharmacological profiles of oral oxycodone and oral morphine in non-drug-abusing volunteers. Psychopharmacology (Berl). 2008;196(1):105-16.

44. Leufkens TR, Vermeeren A, Smink BE, van Ruitenbeek P, Ramaekers JG, Leufkens TRM, et al. Cognitive, psychomotor and actual driving performance in healthy volunteers after immediate and extended release formulations of alprazolam 1 mg. Psychopharmacology (Berl). 2007;191(4):951-9.

45. Wingen M, Ramaekers JG, Schmitt JA, Wingen M, Ramaekers JG, Schmitt JAJ. Driving impairment in depressed patients receiving long-term antidepressant treatment. Psychopharmacology (Berl). 2006;188(1):84-91.

46. Verster JC, Veldhuijzen DS, Patat A, Olivier B, Volkerts ER. Hypnotics and driving safety: meta-analyses of randomized controlled trials applying the on-the-road driving test. Current Drug Safety. 2006;1(1):63-71.

47. Wingen M, Bothmer J, Langer S, Ramaekers JG. Actual driving performance and psychomotor function in healthy subjects after acute and subchronic treatment with escitalopram, mirtazapine, and placebo: a crossover trial. J Clin Psychiatry. 2005;66(4):436-43.

48. Bramness JG, Skurtveit S, Morland J. Testing for benzodiazepine inebriation--relationship between benzodiazepine concentration and simple clinical tests for impairment in a sample of drugged drivers. Eur J Clin Pharmacol. 2003;59(8-9):593-601.

49. Hill JL, Zacny JP. Comparing the subjective, psychomotor, and physiological effects of intravenous hydromorphone and morphine in healthy volunteers. Psychopharmacology (Berl). 2000;152(1):31-9.

50. Hanks GW, O'Neill WM, Simpson P, Wesnes K. The cognitive and psychomotor effects of opioid analgesics. II. A randomized controlled trial of single doses of morphine, lorazepam and placebo in healthy subjects. Eur J Clin Pharmacol. 1995;48(6):455-60.

51. Volz HP, Sturm Y. Antidepressant drugs and psychomotor performance. A review. Neuropsychobiology. 1995;31(3):146-55.

52. Asbridge M, Hayden JA, Cartwright JL. Acute cannabis consumption and motor vehicle collision risk: systematic review of observational studies and meta-analysis. BMJ. 2012;344.

53. Li MC, Brady JE, DiMaggio CJ, Lusardi AR, Tzong KY, Li G. Marijuana use and motor vehicle crashes. Epidemiol Rev. 2012;34:65-72.

54. Rogeberg O, Elvik R. The effects of cannabis intoxication on motor vehicle collision revisited and revised. Addiction. 2016;111(8):1348-59.

55. Drug use and road safety: a policy brief. Geneva, Switzerland: World Health Organization; 2016.

56. Elvik R. Risk of road accident associated with the use of drugs: A systematic review and meta-analysis of evidence from epidemiological studies. Accid Anal Prev. 2013;60(0):254-67.

57. Hels T, Lyckegaard A, Simonsen KW, Steentoft A, Bernhoft IM. Risk of severe driver injury by driving with psychoactive substances. Accid Anal Prev. 2013;59:346-56.

58. Compton RP, Berning A. Drug and Alcohol Crash Risk. National Highway Traffic Safety Administration. Washington, D.C.; 2015. Report No.: DOT HS 812 117.

59. Beirness DJ, Beasley EE. A roadside survey of alcohol and drug use among drivers in British Columbia. Traffic injury prevention. 2010;11(3):215-21.

60. Beirness D, Swann P, Logan BK. Drugs and driving: Detection and deterrence. Paris, France: International transport forum; 2010.

61. Beirness D, Beasley E. An evaluation of immediate roadside prohibitions for drinking drivers in British Columbia: Findings from roadside surveys. Traffic Inj Prev. 2014;15(3):228-33.

62. Brunet B, Hauet T, Hebrard W, Papet Y, Mauco G, Mura P. Postmortem redistribution of THC in the pig. Int J Legal Med. 2010;124(6):543-9.

63. Holland MG, Schwope DM, Stoppacher R, Gillen SB, Huestis MA. Postmortem redistribution of Delta9-tetrahydrocannabinol (THC), 11-hydroxy-THC (11-OH-THC), and 11-nor-9-carboxy-THC (THCCOOH). Forensic Sci Int. 2011;212(1-3):247-51.

64. Lemos NP, Ingle EA. Cannabinoids in postmortem toxicology. J Anal Toxicol. 2011;35(7):394-401.

65. Pounder DJ, Jones GR. Post-mortem drug redistribution--a toxicological nightmare. Forensic Sci Int. 1990;45(3):253-63.

66. Yarema MC, Becker CE. Key concepts in postmortem drug redistribution. Clin Toxicol (Phila). 2005;43(4):235-41.

67. Brubacher JR, Chan H, Erdelyi S, Asbridge M, Mann RE, Purssell RA, et al. Police documentation of drug use in injured drivers: Implications for monitoring and preventing drug-impaired driving. Accid Anal Prev. 2018.

68. Mann RE, Stoduto G, Ialomiteanu A, Asbridge M, Smart RG, Wickens CM. Self-reported collision risk associated with cannabis use and driving after cannabis use among Ontario adults. Traffic injury prevention. 2010;11(2):115-22.

69. Brubacher JR, Chan H, Erdelyi S, Macdonald S, Asbridge M, Mann RE, et al. Cannabis use as a risk factor for causing motor vehicle crashes: a prospective study. Addiction. 2019.

70. Blomberg RD, Peck RC, Moskowitz H, Burns M, Fiorentino D. The Long Beach/Fort Lauderdale relative risk study. Journal of Safety Research. 2009;40(4):285-92.

71. Peck RC, Gebers MA, Voas RB, Romano E. The relationship between blood alcohol concentration (BAC), age, and crash risk. Journal of Safety Research. 2008;39(3):311-9.

72. Schulze H, Schumacher M, Urmeew R, Alvarez J, Bernhoft IM, de Gier H, et al. Driving under the influence of drugs, alcohol and medicines in Europe—findings from the DRUID project. Lisbon, Portugal: European Monitoring Centre for Drugs and Drug Addiction; 2012.