National Transportation Safety Board
Office of Research and Engineering
Washington, DC 20594

HWY23FH010

MEDICAL

Specialist's Factual Report

March 12, 2024

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A. CRASH

Location: Woodlawn, Maryland
Date: March 22, 2023
Time: About 12:36 PM local time

B. MEDICAL SPECIALIST

Specialist: Turan Kayagil, MD, FACEP
National Transportation Safety Board
Washington, DC

C. DETAILS OF THE INVESTIGATION

1.0 Purpose

This investigation was performed to evaluate the surviving, non-commercial, 54-year-old female driver of the 2017 Acura TLX (driver) for potentially impairing substances and potentially impairing medical conditions.[1]

2.0 Methods

Records from the driver's law enforcement post-crash toxicological testing were reviewed, as were records from the driver's post-crash hospital care. At the request of the National Transportation Safety Board (NTSB), the Federal Aviation Administration (FAA) Forensic Sciences Laboratory performed toxicological testing of a blood specimen remaining from the driver's law enforcement post-crash toxicological testing; records from this FAA toxicological testing were reviewed.

D. FACTUAL INFORMATION

1.0 Post-Crash Hospital Records

According to emergency medical services (EMS) records contained in reviewed hospital records, EMS arrived on scene as the driver was being extricated from her vehicle. The responding EMS provider documented that the driver reported that she had a history of seizures and had experienced a seizure prior to the crash. The EMS provider documented that bystanders and first responders had not seen the driver unconscious, and that the driver did not appear postictal upon EMS arrival.

[1] The 2017 Volkswagen Jetta driver was not included in this medical investigation, as NTSB investigators determined that the Jetta driver was not administered post-crash toxicological testing by law enforcement and did not receive post-crash medical care.

The driver was transported to a hospital and was admitted for management of her injuries. She was alert and oriented upon her assessment by the hospital trauma team. She reported to her hospital providers that she had experienced a seizure while driving which had caused her crash. She initially stated that she had been taking seizure medication as prescribed but later stated that she had not taken her seizure medication for two days due to vomiting. The pharmacy from which the driver reported receiving seizure medication had no record of such medication. The driver's outpatient pain management physician stated that the driver had stopped taking seizure medication years ago.

An in-hospital neurology consultation was obtained for evaluation of the driver's seizure history. The consulting neurology provider noted that the driver gave multiple versions of her history. Based on asking the driver and reviewing her chart, the neurology provider documented that the driver had a history of possible seizure disorder diagnosed before 2012 for which she had been on and off a common seizure medication at various doses. Her episodes reportedly involved blacking out, without convulsions, occurring more frequently at night. Sometimes she had other nocturnal symptoms. The symptoms were much more frequent in 2012. At that time, she had a stay in an epilepsy monitoring unit, which captured multiple episodes without corresponding electroencephalogram (EEG) changes, consistent with non-epileptic "seizures" – additionally, she reported multiple episodes during the final night of recording but had been asleep during the time she stated they had occurred. Despite limited evidence to support an epilepsy diagnosis, the driver had been intermittently followed by a neurologist and prescribed seizure medication. The last time she took seizure medication was around May 2022; she did not fill it at all in the year before the crash. She told the hospital neurology provider that 1 year earlier her neurologist stated the driver's seizures were well enough controlled that she could stop medication. The driver told the hospital neurology provider that the driver's episodes started coming back over the past few months and she had intended to reach out to her neurologist but had not yet done so. The hospital neurology provider was unable to locate records of any additional seizure workup to confirm an epilepsy diagnosis since 2012.

With respect to the crash, the driver told the hospital neurology provider that all the driver remembered was feeling like her normal self then blacking out and waking up in the hospital hours later.

The driver was started on seizure medication as a precaution during her hospitalization. No seizure episodes were documented in the hospital, although the driver reported multiple episodes that she thought could be seizures, including symptoms that she described as ongoing while being examined by a neurology provider (the consulting neurologist documented that the symptoms were not consistent with a seizure). The consulting neurologist concluded that it was very unclear if the driver had a seizure disorder or not, and that some of her symptoms

sounded like they might be sleep-disorder related. The neurologist recommended outpatient follow up for additional testing, with no further workup needed during the driver's hospital stay. The driver was prescribed seizure medication upon discharge as a precaution, and was instructed not to drive until cleared by a physician.

No significant natural disease was identified on hospital laboratory testing or hospital imaging (including computed tomography of the head) performed to evaluate the driver's injuries. The driver's reported medical history included chronic pain treated with medications. A pain management consultation note documented that the driver held a medical marijuana card and routinely smoked marijuana three times per day.

2.0 Toxicology Testing

2.1 Hospital Toxicology Testing

A hospital drug screen of a blood specimen collected from the driver at 1:49 PM on the crash date was negative for ethanol, benzodiazepines, barbiturates, and tricyclic antidepressants.

A hospital drug screen of a urine specimen collected from the driver at 6:54 PM on the crash date was presumptively positive (screened positive without a secondary confirmation test) for cannabinoids. The urine drug screen was also presumptively positive for substances that had been administered to the driver during her post-crash medical care. No other screened-for substances were detected.[2]

2.2 Law Enforcement Toxicology Testing

The Maryland State Police Forensic Sciences Division performed toxicology testing of blood collected from the driver at 5:12 PM on the crash date. No alcohol was detected. The blood specimen was reported to be positive for tetrahydrocannabinol (THC) and the THC metabolites hydroxy-THC and carboxy-THC.[3]

[2] Screened-for substances were amphetamines, barbiturates, benzodiazepines, cannabinoids, cocaine metabolite, fentanyl, methadone, opiates, oxycodone/oxymorphone, and phencyclidine.

[3] According to the Maryland State Police toxicology reports, blood was tested for alcohol and for the following drugs or drug groups: amphetamine, cocaine, opiates (codeine, morphine, hydrocodone, hydromorphone, oxycodone, oxymorphone), barbiturates (butalbital, phenobarbital), benzodiazepines (nordiazepam, diazepam, oxazepam, lorazepam, temazepam, clonazepam, alprazolam), methamphetamine, methadone, cannabinoids, phencyclidine (PCP), zolpidem, buprenorphine, and fentanyl.

2.3 FAA Toxicology Testing

At the request of the NTSB, the FAA Forensic Sciences Laboratory performed toxicological testing of blood remaining from the testing performed by the Maryland State Police. Due to limited specimen quantity, the NTSB requested that the FAA Forensic Sciences Laboratory not perform confirmation testing for substances that were administered to the driver during her post-crash medical care before the blood specimen was collected, as determined by the NTSB upon review of the driver's post-crash hospital records. Only confirmed results were reported.

The FAA Forensic Sciences Laboratory toxicology testing detected delta-9-THC at 5.5 ng/mL, 11-hydroxy-THC at 3.5 ng/mL, and carboxy-delta-9-THC at 73.5 ng/mL. Cyclobenzaprine was detected at a low level.[4] Norcyclobenzaprine (an active metabolite of cyclobenzaprine) was detected at 1 ng/mL.

2.4 Descriptions of Detected Substances

Delta-9-THC is the primary psychoactive chemical (cannabinoid) in marijuana and other products derived from the cannabis plant.[5] 11-hydroxy-THC is a psychoactive metabolite of Delta-9-THC.[6] Carboxy-delta-9-THC is a non-psychoactive metabolite of delta-9-THC.

Cannabis may be smoked, vaped, or ingested recreationally by users seeking mind-altering effects. Psychoactive effects of cannabis vary depending on the user, dose, and route of administration. Cannabis has a potential to adversely affect driving performance by slowing reaction time, worsening control of lane position and following, and impairing sustained attention, route planning, decision making, and risk assessment.[7,8] In experimental driving studies, control of lateral position within a lane (a measure of drug-induced driving impairment) has been shown to be modestly

[4] The cyclobenzaprine result was reported as "detected," indicating that its concentration was significantly below the medication's typical therapeutic range.

[5] United States Drug Enforcement Administration. Marijuana. Drug Fact Sheets. https://www.dea.gov/factsheets/marijuana. Updated March 30, 2023. Accessed March 12, 2024.

[6] More precisely, 11-hydroxy-delta-9-THC is a psychoactive metabolite of delta-9-THC. 11-hydroxy-delta-9-THC is one of the substances that the FAA Forensic Sciences Laboratory reports as 11-hydroxy-THC.

[7] Couper FJ, Logan BK. Drugs and Human Performance Fact Sheets. National Highway Traffic Safety Administration. DOT HS 809 725. April 2014 (Revised). https://www.nhtsa.gov/sites/nhtsa.dot.gov/files/809725-drugshumanperformfs.pdf. Accessed March 12, 2024.

[8] Compton RP. Marijuana-Impaired Driving: A Report to Congress. National Highway Traffic Safety Administration. DOT HS 812 440. July 2017. https://www.nhtsa.gov/sites/nhtsa.dot.gov/files/documents/812440-marijuana-impaired-driving-report-to-congress.pdf. Accessed March 12, 2024.

worsened by acute cannabis effects.[9,10,11] After using cannabis, some drivers may attempt to compensate for impairment by driving more slowly and cautiously, but also may not accurately judge whether they are impaired.[8,12,13] Epidemiological studies indicate that acute cannabis intoxication likely moderately increases motor vehicle crash risk; while some meta-analyses estimated an approximate doubling of crash risk, a more recent meta-analysis revised the estimated relative crash risk to about 1.3.[11,14-17] Cannabis might affect sleep and drowsiness; however, the relationship between cannabis use and sleep is complex and incompletely understood.[18-21] The effect of cannabis use on seizure risk is also incompletely understood.[22]

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[9] Arkell TR, Vinckenbosch F, Kevin RC, Theunissen EL, McGregor IS, Ramaekers JG. Effect of cannabidiol and Δ9-tetrahydrocannabinol on driving performance: a randomized clinical trial. *JAMA*. 2020;324(21):2177-2186. doi:10.1001/jama.2020.21218.

[10] McCartney D, Arkell TR, Irwin C, McGregor IS. Determining the magnitude and duration of acute Δ9-tetrahydrocannabinol (Δ9-THC)-induced driving and cognitive impairment: a systematic and meta-analytic review. *Neurosci Biobehav Rev*. 2021;126:175-193. doi:10.1016/j.neubiorev.2021.01.003.

[11] Simmons SM, Caird JK, Sterzer F, Asbridge M. The effects of cannabis and alcohol on driving performance and driver behaviour: a systematic review and meta-analysis. *Addiction*. 2022;117(7):1843-1856. doi:10.1111/add.15770.

[12] Brooks-Russell A, Brown T, Friedman K, et al. Simulated driving performance among daily and occasional cannabis users. *Accid Anal Prev*. 2021;160:106326. doi:10.1016/j.aap.2021.106326.

[13] Marcotte TD, Umlauf A, Grelotti DJ, et al. Driving performance and cannabis users' perception of safety: a randomized clinical trial. *JAMA Psychiatry*. 2022;79(3):201-209. doi:10.1001/jamapsychiatry.2021.4037.

[14] Li MC, Brady JE, DiMaggio CJ, Lusardi AR, Tzong KY, Li G. Marijuana use and motor vehicle crashes. *Epidemiol Rev*. 2012;34(1):65-72. doi:10.1093/epirev/mxr017.

[15] 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:e536. doi:10.1136/bmj.e536.

[16] Rogeberg O, Elvik R. The effects of cannabis intoxication on motor vehicle collision revisited and revised. *Addiction*. 2016;111(8):1348-1359. doi:10.1111/add.13347.

[17] Rogeberg O, Elvik R, White M. Correction to: 'The effects of cannabis intoxication on motor vehicle collision revisited and revised' (2016). *Addiction*. 2018;113(5):967-969. doi:10.1111/add.14140.

[18] Gates PJ, Albertella L, Copeland J. The effects of cannabinoid administration on sleep: a systematic review of human studies. *Sleep Med Rev*. 2014;18(6):477-487. doi:10.1016/j.smrv.2014.02.005.

[19] Kaul M, Zee PC, Sahni AS. Effects of cannabinoids on sleep and their therapeutic potential for sleep disorders. *Neurotherapeutics*. 2021;18(1):217-227. doi:10.1007/s13311-021-01013-w.

[20] Kolla BP, Hayes L, Cox C, Eatwell L, Deyo-Svendsen M, Mansukhani MP. The effects of cannabinoids on sleep. *J Prim Care Community Health*. 2022;13:21501319221081277. doi:10.1177/21501319221081277.

[21] Lavender I, McGregor IS, Suraev A, Grunstein RR, Hoyos CM. Cannabinoids, insomnia, and other sleep disorders. *Chest*. 2022;162(2):452-465. doi:10.1016/j.chest.2022.04.151.

[22] Kaczor EE, Greene K, Zacharia J, Tormoehlen L, Neavyn M, Carreiro S. The potential proconvulsant effects of cannabis: a scoping review. *J Med Toxicol*. 2022;18(3):223-234. doi:10.1007/s13181-022-00886-3. Erratum in: J

Acute impairing effects of cannabis typically last at least 1-2 hours after use, with return to baseline within about 3-7 hours, although some residual effects may persist longer, and the duration of acute effects may be increased at higher cannabis doses or when cannabis is used orally.[7,10] Frequent cannabis users often develop some tolerance to effects of cannabis, but often adjust their usage upward to compensate. Driving impairment from acute cannabis intoxication is not simply predictable from tolerance.[12,13]

When cannabis is smoked, the blood delta-9-THC concentration peaks rapidly (this peak is delayed and flattened when cannabis is consumed orally). Subsequent decline in the blood delta-9-THC concentration is initially hastened by diffusion of delta-9-THC into fatty tissues. An occasional cannabis user's blood delta-9-THC concentration may fall to a very low or undetectable level within hours after smoking, whereas a frequent cannabis user's blood delta-9-THC concentration may remain detectable for days or weeks of abstinence, while delta-9-THC sequestered in fatty tissues redistributes into blood. [7,23 – 26] During this time, as the blood delta-9-THC concentration gradually trends downward due to drug elimination, it might transiently fluctuate upwards due to other physiological factors.[27]

Forensic interpretation of a blood concentration of delta-9-THC can be challenging. The complex pharmacokinetics of delta-9-THC prevent reliable back-extrapolation from a measured concentration to a concentration at a given earlier time. More fundamentally, a person's instantaneous blood concentration of delta-9-THC does not directly predict that person's impairment.[7,8] Thus, attempts at predicting impairment from measured concentrations of delta-9-THC and its metabolites often focus on estimating whether the time of last cannabis use was

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Med Toxicol. 2022 Apr 21; Erratum in: J Med Toxicol. 2023 Jan;19(1):54-60.

[23] Huestis MA. Human cannabinoid pharmacokinetics. *Chem Biodivers*. 2007 Aug;4(8):1770-804. doi: 10.1002/cbdv.200790152.

[24] Bergamaschi MM, Karschner EL, Goodwin RS, et al. Impact of prolonged cannabinoid excretion in chronic daily cannabis smokers' blood on per se drugged driving laws. *Clin Chem*. 2013;59(3):519-526. doi:10.1373/clinchem.2012.195503.

[25] Spindle TR, Cone EJ, Schlienz NJ, et al. Acute pharmacokinetic profile of smoked and vaporized cannabis in human blood and oral fluid. *J Anal Toxicol*. 2019;43(4):233-258. doi:10.1093/jat/bky104.

[26] Vandrey R, Herrmann ES, Mitchell JM, et al. Pharmacokinetic profile of oral cannabis in humans: blood and oral fluid disposition and relation to pharmacodynamic outcomes. *J Anal Toxicol*. 2017;41(2):83-99. doi:10.1093/jat/bkx012.

[27] Peng YW, Desapriya E, Chan H, Brubacher J. Residual blood THC levels in frequent cannabis users after over four hours of abstinence: a systematic review. *Drug Alcohol Depend*. 2020;216:108177. doi:10.1016/j.drugalcdep.2020.108177.

recent enough to indicate probability of related acute psychoactive effects.[7,28,29] Because an occasional cannabis smoker's blood delta-9-THC concentration typically will decline below 5 ng/mL within a few hours of smoking, concentrations above this value sometimes are interpreted to indicate recent cannabis use. However, a frequent cannabis user's blood delta-9-THC concentration may remain elevated above 5 ng/mL for longer periods of abstinence, beyond the period of acute impairing effects, and sometimes as long as days.[27,30,31] Peak delta-9-THC concentrations after cannabis use may be much higher, but typically undergo rapid early decline, often before forensic blood specimens can be collected.[32]

Marijuana is a Schedule I controlled substance under federal law.[5] Maryland state law permits medical use of cannabis for certain qualifying conditions, including chronic pain, subject to restrictions. As of July 1, 2023, recreational use of cannabis by individuals of age 21 years or older is also permitted in Maryland, subject to restrictions. Driving under the influence of cannabis is illegal in Maryland regardless of the reason for cannabis use.[33]

Cyclobenzaprine is a prescription medication that acts on the central nervous system to produce muscle-relaxing effects, for relief of musculoskeletal pain. The drug commonly causes drowsiness, and typically carries a warning that it may impair mental and/or physical abilities required for performance of hazardous tasks, such as operating machinery or driving a motor vehicle.[34,35] The elimination half-life of

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[28] Huestis MA, Henningfield JE, Cone EJ. Blood cannabinoids. II. Models for the prediction of time of marijuana exposure from plasma concentrations of delta 9-tetrahydrocannabinol (THC) and 11-nor-9-carboxy-delta 9-tetrahydrocannabinol (THCCOOH). *J Anal Toxicol*. 1992;16(5):283-290. doi:10.1093/jat/16.5.283.

[29] Kosnett MJ, Ma M, Dooley G, et al. Blood cannabinoid molar metabolite ratios are superior to blood THC as an indicator of recent cannabis smoking. *Clin Toxicol (Phila)*. 2023;61(5):355-362. doi:10.1080/15563650.2023.2214697.

[30] Karschner EL, Schwilke EW, Lowe RH, et al. Implications of plasma delta 9-tetrahydrocannabinol, 11-hydroxy-THC, and 11-nor-9-carboxy-THC concentrations in chronic cannabis smokers. *J Anal Toxicol*. 2009;33(8):469-477. doi:10.1093/jat/33.8.469.

[31] Odell MS, Frei MY, Gerostamoulos D, Chu M, Lubman DI. Residual cannabis levels in blood, urine and oral fluid following heavy cannabis use. *Forensic Sci Int*. 2015;249:173-180. doi:10.1016/j.forsciint.2015.01.026.

[32] Jones AW, Holmgren A, Kugelberg FC. Driving under the influence of cannabis: a 10-year study of age and gender differences in the concentrations of tetrahydrocannabinol in blood. *Addiction*. 2008;103(3):452-461. doi:10.1111/j.1360-0443.2007.02091.x.

[33] This and additional information is available from the Maryland Cannabis Administration website at https://mmcc.maryland.gov/ (Accessed March 7, 2023).

[34] National Institutes of Health National Library of Medicine. Fexmid. DailyMed. https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=b97e2c9e-130c-4543-b3c5-531079bf2005. Updated March 13, 2019. Accessed March 8, 2023.

[35] Caron J, Kaye R, Wessel T, Halseth A, Kay G. An assessment of the centrally acting muscle relaxant tolperisone on driving ability and cognitive effects compared to placebo and cyclobenzaprine. *J Clin Pharm Ther*.

cyclobenzaprine in plasma is roughly 9-40 hours.[36] Norcyclobenzaprine is an active metabolite of cyclobenzaprine with a longer half-life of elimination.[37]

E. SUMMARY OF MEDICAL FACTS

The 54-year-old female driver of the 2017 Acura TLX (driver) was hospitalized for her injuries following the crash. She reported to responding emergency medical services providers and hospital providers that she had a history of seizures and had experienced a seizure while driving which had caused her crash. While in the hospital, she received a neurology consultation for her reported seizure history. She told the hospital neurology provider that all the driver remembered was feeling like her normal self then blacking out and waking up in the hospital hours later.

The consulting neurologist concluded that it was very unclear if the driver had a seizure disorder or not. The driver had undergone a previous epilepsy monitoring unit stay in 2012 that was consistent with non-epileptic episodes, without evidence of seizures. She also reported some nocturnal symptoms that the neurologist thought sounded like they might be sleep-disorder related. The neurologist recommended outpatient follow up for additional testing.

No significant natural disease was identified on hospital laboratory testing or hospital imaging performed to evaluate the driver's injuries. The driver's reported medical history included chronic pain treated with medications. A pain management consultation note documented that the driver held a medical marijuana card and routinely smoked marijuana three times per day.

A hospital drug screen of a blood specimen collected from the driver at 1:49 PM on the crash date was negative for ethanol, benzodiazepines, barbiturates, and tricyclic antidepressants. A hospital drug screen of a urine specimen collected from the driver at 6:54 PM on the crash date was presumptively positive for cannabinoids, plus substances that had been administered after the crash.

At the request of the National Transportation Safety Board (NTSB), the Federal Aviation Administration (FAA) Forensic Sciences Laboratory performed toxicological testing of blood remaining from testing performed by the Maryland State Police. This blood was collected at 5:12 PM on the crash date. The FAA Forensic Sciences

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2020;45(4):774-782. doi:10.1111/jcpt.13165.

[36] Schulz M, Schmoldt A, Andresen-Streichert H, Iwersen-Bergmann S. Revisited: therapeutic and toxic blood concentrations of more than 1,100 drugs and other xenobiotics. *Crit Care*. 2020;24(1):195. doi:10.1186/s13054-020-02915-5.

[37] Sullivan GM, Gendreau RM, Gendreau J, et al. Randomized clinical trial of bedtime sublingual cyclobenzaprine (TNX-102 SL) in military-related PTSD and the role of sleep quality in treatment response. *Psychiatry Res*. 2021;301:113974. doi:10.1016/j.psychres.2021.113974.

Laboratory toxicology testing detected the cannabinoid delta-9-THC, which is the primary psychoactive substance in cannabis, at 5.5 ng/mL, along with its metabolites 11-hydroxy-THC at 3.5 ng/mL and carboxy-delta-9-THC at 73.5 ng/mL. These results were generally consistent with qualitative results reported by the Maryland State Police. Additionally, FAA testing detected the centrally acting muscle relaxant cyclobenzaprine at a low level, and its active metabolite norcyclobenzaprine at 1 ng/mL. Only confirmed substances were reported; at the direction of the NTSB, the FAA Forensic Sciences Laboratory did not perform confirmation testing for substances that had been administered after the crash.

Submitted by:

Turan Kayagil, MD, FACEP
Medical Officer