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The Uneasy Case for Marijuana as Chemical Impairment Under a Science-Based Jurisprudence of Dangerousness

Andrea Roth*

As the marijuana legalization movement advances, states face a jurisprudential dilemma in addressing the burgeoning public health issue of “drugged driving.” Zero-tolerance laws targeting drivers with any illegal drugs in their systems, currently justified under a “jurisprudence of prohibition” based on the blameworthiness of the drug itself, are no longer a good fit due to legalization. Instead, states have attempted to treat marijuana like alcohol by importing drunk driving’s “jurisprudence of dangerousness” through enactment of per se driving under the influence of (DUI) marijuana laws redefining DUI as driving with a certain quantifiable amount of THC, marijuana’s main psychoactive compound, in one’s blood. These laws are legitimate, legislators claim, because they are analogous to per se .08 percent blood-alcohol concentration (BAC) impairment laws. What lawmakers have forgotten, and what legal scholars have largely neglected, is the buried and colorful history of drunk driving’s jurisprudence of dangerousness and the scientific framework for proving the link between specific BACs and crash risk established by the country’s first “traffic czar,” William Haddon Jr. Under this framework—which focuses first and foremost on fatal single-car crashes and case-control studies with a randomly selected control

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group—the illegitimacy of the new wave of DUI marijuana laws is painfully obvious. In fact, the few single-car crash and case-control studies that have been conducted have found no relationship between THC blood levels and an increased relative crash risk. Properly understood, the history of drunk driving jurisprudence offers what is still the only valid scientific framework for criminalizing chemical impairment.

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INTRODUCTION

If you can't measure it, measure it anyway.

- University of Chicago economist Frank Knight

In 1931, Prohibition was on its last legs, Henry Ford’s assembly lines had churned out over ten million Model Ts and were on to the Model A, and the new federal system of numbered highways was five years old.¹ The specter of drunk driving had begun to invade the nation’s psyche, and law enforcement and health officials scrambled to address the issue by urging states to criminalize driving “while impaired” by or “driving under the influence” of (DUI) alcohol, which jurors were told meant driving unsafely as a result of the effects of alcohol. But jurors did not always believe uncorroborated police testimony about a suspect’s drunkenness. Moreover, the medical community was concerned that much of the dangerous impairment causing fatal crashes did not manifest in obvious drunkenness, and might not even be capable of detection through means other than difficult-to-perform blood tests.² The criminal law, as an instrument of protecting the nation from a burgeoning public health crisis, was failing.

Help would arrive in the form of the “Drunk-O-Meter,” a balloon-like device that could measure a motorist’s blood-alcohol concentration (BAC) simply by capturing one’s breath. On New Year’s Eve 1938, in the streets of Indianapolis, the Drunk-O-Meter made its public debut, and the jurisprudence of dangerousness undergirding drunk driving laws entered the scientific age. Not only could the Drunk-O-Meter offer corroboration of police testimony of DUI, it also allowed researchers, through experiments and studies of crashes, to explore the relationship between particular BACs and a driver’s level of impairment. The machine could do so because of the uniquely predictable and uniform properties of ethyl alcohol, which allowed scientists to easily infer

1. See generally DAN MCNICHOL, *THE ROADS THAT BUILT AMERICA: THE INCREDIBLE STORY OF THE U.S. INTERSTATE SYSTEM* (2006) (discussing the increase in number of motor vehicles sold and development of the interstate highway system).

2. See discussion *infra* Part I.

proximity of use, precise level of alcohol intoxication in the brain, and level of driver impairment from one's BAC. Still, this initial wave of experimentation lacked the scientific rigor typical of epidemiological research of public health problems.

Luckily for drunk driving jurisprudence, the country's first "traffic czar" in the late 1960s, the bow-tie-wearing "workaholic government bureaucrat" William Haddon Jr.,³ was an epidemiologist. Haddon transformed the science of alcohol and traffic safety by explaining the need to test BACs of drivers in single-car fatal crashes, conduct case-control studies comparing BACs of fatal crash victims with those of randomly stopped drivers in similar circumstances, and conduct realistic on-road and simulated BAC-specific driver impairment studies. Bathed in the scientific ethos Haddon established, researchers conducted meticulous studies with thousands of motorists that showed precise increased relative crash risks at precise BACs. For example, these studies revealed a precipitous rise in crash risk at .10 and .15 percent BAC, and a lesser but still troubling increased relative crash risk at .08 percent.⁴ At the prodding of federal officials, states would eventually adopt these BACs as presumptive proof of impairment, and later—after DUI enforcement took a punitive, law-and-order turn in the 1980s—would redefine the very crime of DUI in terms of BAC.

The history of drunk driving's science-based jurisprudence of dangerousness should be compulsory reading for today's policymakers because it created a valid model—indeed, the only valid model we have—for using science to define criminally dangerous chemical impairment. It is true that DUI laws have occasionally veered close to a jurisprudence of prohibition—banning driving while drunk because drunkenness itself is morally blameworthy—in the form of "zero-tolerance" laws for minors and calls to lower the adult BAC even further from .08 percent to .05 percent. Even so, the legitimacy of these special (and not uncontroversial) laws has always turned on the strength of not only their prohibitionist logic, but also their link to increased relative crash risk, as judged through Haddon's established framework.

Unfortunately, Haddon's lessons appear to be lost on policymakers attempting to resolve the next perceived public health crisis being addressed through the criminal law: drugged driving. As the marijuana legalization movement advances, states face a jurisprudential dilemma. Before legalization, a number of states had passed "zero-tolerance" laws banning driving with *any* amount of any *illicit* drug in one's system. These laws were justified under a jurisprudence of prohibition; the state could legitimately criminalize driving under the influence because it considered the drug use itself morally blameworthy. But in those states that have wholly or partially legalized

3. BARRON H. LERNER, ONE FOR THE ROAD: DRUNK DRIVING SINCE 1900, at 38 (2011).

4. See discussion *infra* Part I.A.4.

marijuana, prohibition is no longer a good fit as a theory of punishment. Voters have instead chosen to treat marijuana like alcohol.

In line with marijuana legalization advocates' own analogies to alcohol, law enforcement and policymakers in several states have reasoned that the way to criminalize DUI marijuana is simply to import the DUI alcohol model. But to these officials, who are often not aware of the history of DUI alcohol science, adopting the DUI alcohol model has meant simply choosing some numerical definition of chemical impairment by marijuana as analogous as possible to alcohol's .08 percent. Six states have done just that, by picking thresholds such as one, two, or five nanograms per milliliter of tetrahydrocannabinol (THC)—the main psychoactive compound in marijuana—in the blood, and redefining the crime of DUI as driving with a THC level over that threshold. These officials have made their case by invoking the DUI alcohol analogy and, in a political twist, legalization advocates have themselves acquiesced as a means of winning over hesitant voters and law enforcement groups in their fight for legalization. The White House has now urged all states to adopt such laws, and a bill is pending in Congress to force states to do just that.

Remarkably, a surge of new criminal laws, to be enforced on the nation's highways, has been unleashed without their sponsors ever having to articulate a legitimate theory of punishment. Haddon's established framework of BAC-specific single-car crash studies, BAC-specific case-control studies, and realistic and BAC-specific impairment studies bears no resemblance to the rushed and unscientific process that produced per se DUI marijuana laws. The well-acknowledged truth is that there is no known relationship between THC blood levels and increased relative crash risk documented by single-crash or classic case-control studies, and no known relationship between a driver's THC blood level and his level of driving impairment. To the extent single-car and case-control crash studies do exist, they suggest, if anything, that drivers with only THC in their blood are not causing a disproportionate number of fatal crashes.

In short, once Haddon's established scientific framework for defining dangerous impairment is understood as a mandatory hurdle for any legitimate chemical impairment law, the illegitimacy of per se DUI marijuana laws under a jurisprudence of dangerousness becomes painfully obvious. Scientific validity is not a light switch that policymakers can turn on and off when convenient; it is the very premise of any legitimate chemical impairment law, like alcohol's .08 percent, that criminalizes having a measurable amount of a drug in one's body while performing a potentially dangerous activity.

Acknowledging the illegitimacy of this new wave of DUI marijuana laws is critical to any future attempt to use science as a jurisprudential tool in addressing drugged driving. If not distracted by a zealous quest to have an enforceable per se limit as soon as possible, officials would be newly motivated

to fund the type of research that could satisfy Haddon's established framework. For example, researchers could focus on better and quicker data collection after single-car fatal crashes. They could focus on finding a less invasive, more mobile, and more accurate means of detecting the proximity of use, level of intoxication, and level of driving impairment, which would allow more robust random sampling of case-control drivers. And they could focus on developing better training and validation studies for drug recognition experts, as well as more funding for dashboard and body-worn cameras, which could be used to corroborate and study physical manifestations of chemical impairment. Alternatively, they would be newly motivated to consider and develop a regime of swift but certain nonpenal, regulatory consequences that might end up being more effective than criminal DUI marijuana enforcement.

Part I of this Article dusts off the buried and colorful history of the evolution of American drunk driving laws, explaining the road to .08 percent, the choice DUI lawmakers have faced between a jurisprudence of prohibition and a jurisprudence of dangerousness, and the establishment of Haddon's scientific framework for defining dangerous chemical impairment. Part II explores the history of drugged driving laws, the prohibitionist approach of zero-tolerance drugged driving laws, and the political compromise resulting in per se THC laws to combat DUI marijuana, and explains why these per se laws are illegitimate under a science-based jurisprudence of dangerousness. Part III briefly describes a suggested course for future drugged driving research and jurisprudence. The Article concludes with parting thoughts on the lessons of the DUI story as a case study revealing technology's potentially distorting effects on criminal jurisprudence in the scientific age.

I.

ESTABLISHING THE FRAMEWORK FOR A SCIENCE-BASED JURISPRUDENCE OF DANGEROUSNESS: THE STORY OF DUI ALCOHOL

A. The Road to .08 Percent: Breath Machines and the Study of Crash Risk

While drunk driving is often overlooked in legal academia⁵ as a small, quirky, and gritty corner of the law involving only socially privileged defendants, this description is not entirely true and, even where true, only emphasizes why the field is in need of study. In fact, DUI is one of the most

5. To be sure, sociologists, criminologists, and historians have published social and cultural histories of DUI, which have discussed the scientific and penal theories underlying drunk driving laws and their effectiveness as a tool of social control. *See, e.g.*, JAMES B. JACOBS, DRUNK DRIVING: AN AMERICAN DILEMMA (1989); LERNER, *supra* note 3; H. LAURENCE ROSS, CONFRONTING DRUNK DRIVING: SOCIAL POLICY FOR SAVING LIVES (1992); SOCIAL CONTROL OF THE DRINKING DRIVER (Michael D. Laurence, John R. Snortum & Franklin E. Zimring eds., 1988) [hereinafter LAURENCE ET AL. 1988].

frequently committed crimes in the country,⁶ involves “some of the most complex [cases] in the criminal justice system,”⁷ and is the training grounds for America’s public criminal trial lawyers.⁸ It is also a public health issue of tremendous social importance: according to federal officials, alcohol-related crashes cost the United States more than \$37 billion annually, and in 2010 alone, more than ten thousand people on American roads—one every fifty-one minutes—died in such crashes.⁹ Over the history of the nation, this carnage has “exceed[ed] the death toll of all our wars.”¹⁰

Frank Zimring has suggested that “the student of legal policy can examine drunk driving as an example of exceptionally fast change in the criminal law.”¹¹ Compared with the arc of homicide law, the century-long arc of DUI law is quite short. The DUI story therefore offers the luxury of a documented account of how legal models can change in response to our perceived need to, and perceived ability to, measure factors that may be relevant to determining criminality. DUI law is in many ways a success story, but both its bright and dark moments offer critical lessons that explain why the nation’s most recent attempt to invoke criminal law to ensure public safety, through per se DUI marijuana laws, has no legitimate penal purpose.

1. The New Crime of DUI and the Need for an “Impairment” Machine

People have been drinking and driving in this country since the invention of the automobile, and various states have prohibited DUI by statute or common law since at least the beginning of the twentieth century.¹² But there was nothing inevitable about using the criminal law as the primary means of addressing drunk driving; indeed, “there is . . . an awkward fit between drunk

6. See FED. BUREAU OF INVESTIGATION, UNIFORM CRIME REPORT: CRIME IN THE UNITED STATES, 2012, at 1 (Fall 2013), available at <http://www.fbi.gov/about-us/cjis/ucr/crime-in-the-u.s/2012/crime-in-the-u.s.-2012/persons-arrested/arrestmain.pdf>.

7. NAT’L TRAFFIC LAW CTR., CHALLENGES AND DEFENSES II: CLAIMS AND RESPONSES TO COMMON CHALLENGES AND DEFENSES IN DRIVING WHILE IMPAIRED CASES v (2013).

8. Numerous prosecutors and public defenders with whom I have spoken stated that they were trained primarily on DUI cases. The large number of DUI-specific training manuals for prosecutors also evinces the profession’s recognition that such cases are both ubiquitous and entrusted to new lawyers. See, e.g., *National Traffic Law Center*, NAT’L DIST. ATT’YS ASS’N, http://www.ndaa.org/ntlc_home.html (last visited Apr. 6, 2015).

9. See *Impaired Driving*, NAT’L HIGHWAY TRAFFIC SAFETY ADMIN. (NHTSA), <http://www.nhtsa.gov/Impaired> (last visited Apr. 6, 2015).

10. *Perez v. Campbell*, 402 U.S. 637, 657 (1971) (Blackmun, J., concurring in part and dissenting in part).

11. Franklin E. Zimring, *Foreword to* JAMES B. JACOBS, DRUNK DRIVING, *supra* note 5, at x.

12. COMM. ON PUB. WORKS, 90TH CONG. 2D. SESS., 1968 ALCOHOL AND HIGHWAY SAFETY REPORT 100 (1968) [hereinafter 1968 REPORT]; JACOBS, *supra* note 5, at 57. Pennsylvania, for example, passed its first “driving while intoxicated” statute—without defining “intoxicated”—in 1909. See Robert J. Scheffer, *Under the Influence of Alcohol Three Hours After Driving: The Constitutionality of the (a)(5) Amendment to Pennsylvania’s DUI Statute*, 100 DICK. L. REV. 441, 444 (1996). Drunk driving was not an offense at common law unless it was a “nuisance.” 1968 REPORT, *supra* note 12, at 100 n.1, 139. See generally LERNER, *supra* note 3.

driving and criminal law.”¹³ DUI is unique in that it is an inchoate traffic offense. Unlike traffic laws that prohibit running a red light or crossing the center line, DUI criminalizes a physiological condition that *might* cause one to engage in these risky behaviors. While other conditions that can potentially cause traffic harm—such as sleepy, distracted, or angry driving—are targeted by aggressive public education campaigns, states generally do not criminally punish them.

The traditional justification for invoking the criminal law against “drinking drivers,” even before they have run a red light or caused a crash, has been that their driving is “more dangerous, by an order of magnitude, than other forms of impaired driving.”¹⁴ The drunk driver, it is said, is a “ticking bomb” punished for “creating a significant risk of injury or death to fellow road users.”¹⁵ The statutory phrases “intoxication,” “impairment,” and “under the influence” are not medical terms,¹⁶ but rather legal terms, intended to capture the blameworthy level of alcohol intoxication at which a driver is so potentially dangerous to others as to be justifiably labeled a criminal.

That threshold has always been understood to turn on the extent to which a person’s intoxication is likely to cause harm through a crash. In California, jurors are instructed that, to find a defendant guilty of driving “under the influence,” they must find that “his or her mental or physical abilities are so impaired that he or she is no longer able to drive a vehicle with the caution of a sober person, using ordinary care, under similar circumstances.”¹⁷ If “impairment” meant any deviation from stone-cold sobriety, then any drinking driver would be guilty; as the California Highway Patrol Chief declared in 1934, “[n]o man who has been drinking is a safe driver”¹⁸ But DUI alcohol laws, at least for adults, have never been justified on the argument that any drinking whatsoever is dangerous or otherwise morally blameworthy. Such a jurisprudence of prohibition would be politically untenable. A 1940 paper in the *Journal of the American Medical Association (AMA)* noted that while “[t]here can be little doubt that the operation of motor vehicles would be safer if the drivers were prohibited from consuming any alcohol whatever,”

13. JACOBS, *supra* note 5, at 63.

14. *Id.* at 59.

15. *Id.*

16. Even if the legal community were inclined to impute such a medical definition, none existed at the time the laws were created. *See, e.g.*, Herman A. Heise, *Alcohol and Automobile Accidents*, 103 JAMA 739, 741 (1934) (“No definite amount of alcohol in body fluids has been officially designated above which a person is intoxicated and below which he is sober.”).

17. Cal. Crim. Jury Instr. 2110 (2014 ed.).

18. *More Drunk Drivers*, L.A. TIMES, Mar. 15, 1934, at A4; *see also* LERNER *supra* note 3, at 25 (noting wide acceptance by scientists that small amounts of alcohol interfered with cognitive functioning); H. LAURENCE ROSS, DETERRING THE DRINKING DRIVER: LEGAL POLICY AND SOCIAL CONTROL 2 (1984) [hereinafter ROSS (1984)] (noting studies showing that even at very low BACs, driving ability is still “noticeably affected”) (citing Christensen, Foster, & Glad 1978); Henry Newman & Edwin Fletcher, *The Effect of Alcohol on Driving Skill*, 115 JAMA 1600 (1940).

Prohibition “demonstrated the inability to enforce legislation so generally unpopular Public sentiment is definitely against the *drunken* driver but not against the *drinking* driver.”¹⁹ In short, only *dangerous* drinking and driving by adults can legitimately be criminalized in this country.

The specter of the “drunken driver”—the dangerous drinking driver—arose largely because of the massive increase in fatal car crashes in the first few decades of the twentieth century,²⁰ which were caused by the mass production of cars and the end of Prohibition.²¹ Faced with anecdotal evidence suggesting that drivers who were clearly drunk—those showing obvious signs of intoxication—were overrepresented among those in fatal crashes, as well as newspaper accounts of highly publicized highway deaths at the hands of these so-called “killer-drunk[s],”²² several state legislatures began a serious push to get such drivers off the roads.²³ Thus, this period’s “classical” DUI laws targeted what the community had adjudged “clearly blameworthy conduct,” and the period’s “[p]enalties and procedures were drawn from the general criminal law and seemed to be appropriate to the behavior in question.”²⁴

From a law enforcement perspective, there were two key problems with this dangerousness-based “impairment” regime. The first was that jury acquittal rates in drunk driving cases were relatively high.²⁵ This tendency to acquit rose not simply from disagreement over whether certain signs of drunkenness suggested impairment. The Book of Proverbs’ description of the effects of too much wine—“woe, . . . sorrow, . . . contentions, . . . babbling, . . . wounds without cause, . . . [and] redness of eyes”²⁶—bears a striking resemblance to the factors police relied on in the early days of DUI prosecutions to prove

19. Newman & Fletcher, *supra* note 18, at 1602 (emphasis added).

20. LERNER, *supra* note 3, at 15–16. Even before the advent of cars, anecdotal data about the drunkenness of public transport operators causing accidents was well publicized. *Id.*; see also Franklin E. Zimring, *Law, Society, and the Drinking Driver: Some Concluding Reflections*, in LAURENCE ET AL. 1988, *supra* note 5, at 381 (“The American public always believed that drinking, or at least elevated levels of intoxication, increased the risk of automobile crashes.”).

21. Annual car sales went from eight thousand in 1900 to eight million in 1920. LERNER, *supra* note 3, at 15. Los Angeles officials soon after Prohibition claimed there was a “vast increase in drunk driving since repeal.” *Id.* at 21 (quoting *More Drunk Drivers*, L.A. TIMES, Mar. 15, 1934, at A4).

22. See ROSS, *supra* note 5, at 22 (citing Gusfield 1981). Criminologists appear to be in agreement that this socially constructed term is highly misleading and obfuscates the complexity of drinking-driving behavior. See, e.g., Alan C. Donelson, *The Alcohol-Crash Problem*, in LAURENCE ET AL. 1988, *supra* note 5, at 32 (noting that the “killer drunk” became “mythologized, obscuring a more complicated reality, which features diverse patterns and outcomes of drinking-driving behavior as well as diverse types of people who engage in that behavior”).

23. See, e.g., Uniform Act Regulating Traffic on Highways § 47, 1935 Ill. Laws 1247, 1258 (codified as amended at 625 ILL. COMP. STAT. 5/11-501 (2014)).

24. ROSS (1984), *supra* note 18, at 1.

25. See, e.g., HARRY KALVEN, JR. & HANS ZEISEL, *THE AMERICAN JURY* 67, 71, 295 (1966) (comparing judge and jury acquittal rates for drunken driving).

26. *Proverbs* 23:29 (King James).

impairment,²⁷ and in cases where obvious signs of drunkenness were captured on film at the stationhouse, prosecutions were generally successful.²⁸ Rather, the problem was that, in cases where the only evidence of impairment was a police officer's uncorroborated testimony of his observations of a driver, it was the "suspect's word against an officer's," and finders of fact—even judges—were "loath to convict."²⁹

The second problem was the suspicion of many police and public health officials that drivers might be unsafe to drive even when they exhibited no outward signs of obvious drunkenness.³⁰ Law enforcement attempted to address this problem by giving a battery of "field sobriety tests" (FST) to suspects stopped for erratic driving or otherwise suspected of being drunk.³¹ The thought was that if a suspect both failed to pass the tests and exhibited erratic driving, then they were unsafe to drive as a result of alcohol even if not obviously drunk. But early FSTs—such as forcing a suspect to say "Methodist Episcopal" several times fast³²—were notoriously subjective and unscientific, and were viewed as difficult to pass even by sober people. On the other hand, FSTs appeared to generate a fair number of false negatives. A study in Los Angeles County in the 1920s showed that a substantial number of drivers stopped for unsafe driving passed FSTs with flying colors but still had moderate levels of alcohol concentration in their urine.³³ While urine testing was discounted as "grossly inaccurate" at the time,³⁴ the study at least suggested that some drinking and potentially unsafe drivers were going undetected with then-current methods of enforcement.³⁵

27. See, e.g., R.N. Harger, *Some Practical Aspects of Chemical Tests for Intoxication*, 35 J. CRIM. L. & CRIMINOLOGY 202, 203 (1944) (noting that the "usually accepted signs" of impairment are "the odor of the breath, abnormal speech, clumsiness of movement, and evidence of 'stimulation,' or belligerent or otherwise abnormal behavior).

28. In cases after the dawn of breath testing where a suspect refused a breath or blood test, and thus where it was the "suspect's word against an officer's" and where "most judges [were] loath to convict," police in the early 1960s began to fight back "in a score of cities by making each suspect the star of a two-minute movie, frequently in sound and color." C.P. Gilmore, *How the Camera Catches Crooks*, POPULAR SCI., June 1962, at 194. Suspects "almost always plead guilty when shown movies of how they looked when they were brought in." *Id.* at 52–53.

29. Gilmore, *supra* note 28, at 194.

30. See, e.g., C.W. Muehlberger, Ph.D., Letter to the Editor, 115 JAMA 2198 (1940) ("The public associates the term 'drunk' with a stage of alcoholic intoxication characterized by staggering gait and confused or slurred speech. . . . Such standards, while entirely suitable for purposes of hospital classification, are hardly adequate for diagnosis of the influence of alcohol on motorists.").

31. See, e.g., LERNER, *supra* note 3, at 24; Harger, *supra* note 27, at 203.

32. LERNER, *supra* note 3, at 24; Harger, *supra* note 27, at 203.

33. Emil Bogen, *The Diagnosis of Drunkenness—A Quantitative Study of Acute Alcoholic Intoxication*, 26 CAL. & W. MED. 778, 779 (1927), available at <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1655515/?page=5>; *id.* at 778 ("[A] person may be under the influence of alcohol to an extent that seriously affects his . . . driving . . . without presenting the entire common syndrome of drunkenness.").

34. Lester D. Ellerbrook & Chester B. VanGaasbeek, *The Reliability of Chemical Tests for Alcoholic Intoxication*, 122 JAMA 996, 998 (1943).

35. Bogen, *supra* note 33, at 778.

Thus, law enforcement had two strong incentives to find a mechanical means of measuring chemical impairment from alcohol. First, to better corroborate imprecise or less than credible police officer testimony, they needed a more reliable and objective means of proving impairment. Second, they needed a way of detecting levels of impairment below obvious drunkenness, to prove that such levels were, indeed, causing carnage that would inspire public outrage and merit criminal punishment.

It was not a foregone conclusion that science could even offer an accurate, mechanical way to identify drivers with alcohol concentrations rendering them unsafe to drive. The most direct way to measure the effects of ethyl alcohol on the brain would have been to sample brain tissue, which was possible in fatal cases but “with living subjects . . . would not be very popular.”³⁶ Researchers also considered spinal fluid a possibility until they realized not only how difficult it was to obtain, but that the alcohol concentration in the lumbar region (where fluid samples were typically taken) was generally much lower than the brain’s alcohol concentration.³⁷ Urine testing was highly inaccurate, and saliva, while easy to obtain, was too likely to contain “residual” mouth alcohol that could overestimate brain alcohol concentration.³⁸ The Cleveland Police Department in the 1930s developed an “alcohol test” consisting of a “stereoscope” and two small pictures that appeared as separate images to a sober person but appeared to an “even slightly intoxicated [person]” to be merged; apparently, some traffic-court judges accepted the evidence “as proof of intoxication.”³⁹ Yet the stereoscope could at most detect the presence, not the precise concentration, of alcohol in a person’s system, and thus was of limited use.

The good news was that the unique properties of ethyl alcohol offered the possibility of estimating brain alcohol concentration through blood and breath testing. Alcohol is, of course, a drug that results in central nervous system effects—including euphoria, depression, and disorientation—similar to many other legal and illegal drugs.⁴⁰ But its “pharmacokinetic” properties (how it is processed by the body) and “pharmacodynamic” properties (how it acts on the body)⁴¹ are quite unusual.⁴² The substance is much less potent than most other

36. Harger, *supra* note 27, at 205.

37. Richard R. Burgee, *A Study of Chemical Tests for Alcoholic Intoxication*, 17 MD. L. REV. 193, 197 (1957).

38. *Id.*

39. *Eye Test Shows Up Topsy Drivers*, POPULAR SCI., July 1936, at 22.

40. See generally 5 DAVID L. FAIGMAN ET AL., MODERN SCIENTIFIC EVIDENCE: THE LAW AND SCIENCE OF EXPERT TESTIMONY § 41:20 (2012–2013 ed. 2012) (discussing the similarities between the pharmacological properties of alcohol and narcotic drugs such as morphine).

41. See generally Donelson, *supra* note 22, at 4 (explaining the difference between the two major branches of pharmacology: pharmacokinetics and pharmacodynamics).

42. AM. PROSECUTORS RES. INST., ALCOHOL TOXICOLOGY FOR PROSECUTORS: TARGETING HARDCORE IMPAIRED DRIVERS 5 (July 2003) [hereinafter APRI (2003)], available at http://www.ndaa.org/pdf/toxicology_final.pdf.

drugs, meaning that high concentrations must be present in the body for its effects to take hold. In turn, such high concentrations of alcohol “affect[] all organ and biochemical systems of the body.”⁴³ It is both water and fat soluble, meaning that it equilibrates rapidly between blood and brain and “produces no active metabolites.”⁴⁴ Alcohol also quickly dissipates from the body almost entirely through metabolism.⁴⁵ The level of alcohol in one’s bodily fluids at any given point is proportional to rates of absorption, distribution, and elimination that are similar, though not identical, among humans.⁴⁶ And because of alcohol’s complete water solubility and volatility at body temperature, the level of alcohol in one’s breath from the depths of one’s lungs (deep-lung air or “end-expiratory” breath) is in a generally constant and measurable ratio with the level of alcohol in one’s arterial blood.⁴⁷ In turn, one’s BAC is generally proportional, in a linear fashion, to the intensity of the effect on the user’s central nervous system.⁴⁸

Alcohol’s distinctive qualities enabled Swedish physician Erik M.P. Widmark to develop an equation—still used in prosecutions today when a defendant refuses blood or breath testing—linking one’s BAC and one’s body weight, amount of alcohol consumed, time of last consumption, and alcohol elimination rate.⁴⁹ Widmark also developed in 1922 a means of blood testing for alcohol concentration.⁵⁰ However, blood tests were hard to administer in time to be useful for DUI prosecution, especially in rural areas. Moreover,

43. FAIGMAN ET AL., *supra* note 40, at § 41:20.

44. Gary M. Reisfield et al., *The Mirage of Impairing Drug Concentration Thresholds: A Rationale for Zero Tolerance Per Se Driving Under the Influence of Drugs Laws*, 36 J. ANALYTICAL TOXICOLOGY 353, 353 (2012).

45. Donelson, *supra* note 22, at 5. A very small amount is excreted in breath, urine, and sweat. *Id.*

46. *Id.*

47. See generally FAIGMAN ET AL., *supra* note 40, at § 41:32, :38, :40. When a person drinks ethyl alcohol, it travels from the stomach, to the intestines, to the blood, and to all bodily tissues, including air pockets deep within one’s lungs called “alveoli,” which are surrounded by blood-rich membranes. Some portion of the alcohol in one’s blood evaporates into the alveoli. As you exhale, your Breath Alcohol Concentration (BrAC) starts at its lowest level and rises until it reaches its peak as the deep air from the alveoli is finally expelled. According to “Henry’s Law,” at a constant 34 degrees Celsius (the average temperature of human breath when it leaves the mouth), one’s BrAC is in a constant ratio with one’s BAC. This ratio differs for each person, but is close to 2100 to 1. Thus, the number that a breath test reports as the BAC is not actually the BAC, but a calculated estimate of the BAC. *Id.*

48. *Id.* § 41:22. The effects are more pronounced when one’s BAC is rising rather than falling. *Id.*; see also Donelson, *supra* note 22, at 6–7 (noting that “the greater the amount of alcohol present in the body, the greater the effects of alcohol” and discussing the results of a study that showed that 86 percent of participants were judged intoxicated at BACs between 151 and 200 mg percent, while only 34 percent were judged intoxicated at BACs between 51 and 100 mg percent).

49. See CLARKE’S ANALYTICAL FORENSIC TOXICOLOGY 320–21 (Adam Negrusz & Gail Cooper eds., 2d ed. 2013); see also APRI (2003), *supra* note 42, at 16–17.

50. See, e.g., Rune Andréasson & A. Wayne Jones, *The Life and Work of Erik M.P. Widmark*, 17 AM. J. FORENSIC MED. & PATHOLOGY 177 (1996), available at http://journals.lww.com/amjforensicmedicine/Fulltext/1996/09000/The_Life_and_Work_of_Erik_M_P_Widmark.1.aspx#.

blood tests required the aid of a medical professional, and most physicians were hesitant to cooperate with police because BAC tests were not within the traditional doctor-patient relationship and, thus, not covered by medical malpractice insurance.⁵¹

Researchers thus focused their efforts on breath testing. A 1927 *Popular Science* article hailed the invention of an early balloon-like device by W.D. McNally, a Chicago “coroner’s chemist,” by declaring that it could “tell infallibly whether a person has taken a single drink” (leading the authors to lament that “[a] drinking man doesn’t stand a chance these days”).⁵² While the test was significant in that it did not require an invasive blood draw, it too proved only the presence, rather than the concentration, of alcohol in the body.

Finally, in 1931, Dr. Rolla Harger, a biochemist at Indiana University and police consultant, invented the “Drunk-O-Meter”—the first machine that did not require invasive testing, offered an estimate of BAC, and could be used by police during roadside traffic stops.⁵³ The person being tested blew air into a balloon, and the air was then released into a chemical solution (see Table 1).⁵⁴ In the presence of alcohol, the chemical solution changed color. The greater the color change, the more alcohol in one’s breath.⁵⁵ The suspect’s *blood* alcohol concentration (BAC) could then be estimated from the *breath* alcohol concentration (BrAC) through a simple, scientifically accepted conversion ratio.⁵⁶ Fresh from the laboratory, the Drunk-O-Meter made its public debut in 1938 on New Year’s Eve, on the streets of Indianapolis.⁵⁷ By 1944, competitors had developed the “Intoximeter” and “Alco-Meter,” similar machines that also used chemical solutions and balloons to test BrAC.⁵⁸

51. See 1968 REPORT, *supra* note 12, at 105.

52. *Tests a Tippler’s Breath*, POPULAR SCI., Aug. 1927, at 56.

53. See LERNER, *supra* note 3, at 24.

54. *Id.*

55. *Id.*

56. See *supra* note 47.

57. See *How Police Nab Drunk Drivers: From Drunkometer to Breathalyzer*, 90.9WBUR (Dec. 31, 2012), hereandnow.wbur.org/2012/12/31/breathalyzer-history.

58. AM. PROSECUTORS RES. INST., BREATH TESTING FOR PROSECUTORS: TARGETING HARDCORE IMPAIRED DRIVERS 11 (2004) [hereinafter BREATH TESTING FOR PROSECUTORS].



TABLE 1. A demonstration of Harger's "Drunk-O-Meter" in 1950.

(Reprinted with permission from the Associated Press)

In cases of obvious drunkenness, Drunk-O-Meter results gave law enforcement what they had hoped for: corroboration of police testimony and, thus, more convictions. A suspect's claim that he was not obviously drunk—contradicting officer testimony—was much less believable when the state introduced test results showing that the suspect blew a .20 percent BAC, which an expert could explain was likely the equivalent of drinking several alcoholic beverages in a short time. The public, all too familiar with the risks of driving while obviously drunk, had little trouble convicting defendants with mechanical corroboration in the more egregious cases.⁵⁹

Anticipating that drunk drivers could now be proven guilty with certainty, the press celebrated the Drunk-O-Meter as nothing short of an impairment machine. A scholar writing in 1953 heralded the Drunk-O-Meter as fulfilling the "age-old dream of man" to have the sort of proof "whereby a situation is fed into a device and out rolls the correct adjudication."⁶⁰ One journalist described the machine as a "scientific wonder[] that measure[s] the immeasurable."⁶¹ A 1955 *Popular Science* article excitedly observed that "[t]he hunches and walk-a-straight-line methods of old-time police work are being replaced by scientific devices to tell the cops just how drunk a driver is by analyzing a whiff of his breath."⁶² Numerous other newspaper accounts

59. See, e.g., Richard L. Holcomb, *Alcohol in Relation to Traffic Accidents*, 111 JAMA 1076, 1077 (1938) (noting that "prosecutions based on the tests have been unusually successful").

60. Dillard S. Gardner, *Breath-Tests for Alcohol: A Sampling Study of Mechanical Evidence*, 31 TEX. L. REV. 289 (1953).

61. David Le Roi, *How Drunk is Drunk? Science Has the Answer*, TIMES PICTORIAL, Sept. 30, 1950, at 9.

62. Joseph Dorlaque, *New Machines Get Drunks Off the Road*, POPULAR SCI., Oct. 1955, at 166.

described the new gadget with awe.⁶³ Courts, too, were impressed. One court expressed relief that “[t]he prosecution need no longer rely solely upon” FSTs now that “scientific methods” could “determine exactly the extent to which a suspect is ‘under the influence of intoxicating liquor.’”⁶⁴ Indeed, some courts, citing the availability of the Drunk-O-Meter, found police testimony alone legally insufficient for DUI conviction.⁶⁵ In short, a “steadily growing respect” was building for the Drunk-O-Meter’s “superiority over human testimony.”⁶⁶

Yet in cases where a suspect was not obviously drunk, the Drunk-O-Meter’s results did not make the jury’s job simpler, absent some sense of what a particular BAC meant in terms of safe driving. In choosing which drivers to morally condemn, the public appeared no more interested in criminalizing low levels of intoxication—at least not based solely on a loss of efficiency in certain driving skills—than they were before the Drunk-O-Meter. Indeed, large swaths of the public drove at low levels of intoxication and surely would have experienced no small amount of cognitive dissonance in widening the criminal net to include anyone whose driving was affected by alcohol.⁶⁷ As one doctor wrote in a letter to the AMA, “Drunkenness is distinctly a social and legal condition While chemical tests can prove that a man is intoxicated they cannot prove that a man is drunk.”⁶⁸ Without proof that driving with, say, a .15 percent BAC actually caused an unacceptable number of road accidents, the public was unmoved by this mechanical wonder in cases with ambiguous non-machine evidence of morally blameworthy impairment.⁶⁹

2. *Early Attempts to Equate BAC with Dangerousness*

As I explain in this Section, while courts and commentators continued to praise the Drunk-O-Meter as an impairment machine, certain public health officials appeared to recognize that the only meaningful way to show that certain BACs equated to criminal impairment was not simply to quantify BACs, but to determine the extent to which certain BACs caused or posed a high risk of causing a car crash.

63. See, e.g., James Doherty, *Drunkometer Demonstrated At N.U. School: Prosecutors View Tests by Inventor*, CHI. TRIB., Aug. 17, 1950, at A13 (celebrating Harger’s training of 100 young prosecutors in a “special course on criminology”).

64. *McKay v. State*, 235 S.W.2d 173, 174 (Tex. Crim. App. 1950) (quoting a 1950 Northwestern study).

65. See, e.g., *State v. Matchok*, 82 A.2d 444, 446 (N.J. Super. A.D. 1951) (holding that doctor’s testimony about smell of alcohol on defendant’s breath was insufficient proof of DUI, particularly “in view of modern scientific advances” for quantitatively testing intoxication and the doctor’s unfamiliarity with such modern tests).

66. Dorlaque, *supra* note 62, at 166.

67. See LERNER, *supra* note 3, at 3–4.

68. Dr. W.C. Woodward, Abstract of Discussion, *Alcohol and Automobile Accidents*, 103 JAMA 741, 741 (1934).

69. See LERNER, *supra* note 3, at 5. See generally KALVEN & ZEISEL, *supra* note 25.

Yet any attempt to identify the cause of a crash as being the act of driving with a certain BAC was challenging for four primary reasons. First, the cause of a crash, and the assignment of fault for the crash, might have consisted of a complex web of factors difficult to untangle. Second, there was little reliable data on non-fatal crashes for much of the twentieth century, in part because many were unreported. And fatal crashes, while recorded, might have had a disproportionate number of drunk drivers because of the circumstances under which they tended to occur—at night, with male drivers, and involving only a single car.⁷⁰ Third, the fact that a driver at a certain BAC was several times more likely to be in a crash did not by itself prove that this level of intoxication *caused* the crash. Rather, some other factor—such as trauma or psychopathy—could have caused both the drinking and the crash.⁷¹ Finally, the fact that a certain BAC caused an increase in the *relative risk* of a crash, compared with a sober driver, did not suggest that the *absolute risk* of a crash at that BAC was particularly high.⁷²

Another central challenge in proving a causal link between BAC and crash risk based on crash statistics alone was the absence of a baseline or “case-control.” Even if 20 percent of drivers in fatal car crashes had a BAC of .15 percent or higher, that fact alone would not suggest a causal link between .15 percent and increased relative crash risk if, for example, the number of drivers at .15 percent BAC *not* involved in fatal car crashes under similar times and conditions was also 20 percent. To suggest causation, the number of drivers involved in fatal crashes involving a certain BAC would have to be disproportionately high compared to the number of drivers with that BAC, under the same circumstances, *not* involved in fatal crashes. While the advent of roadside blood and breath alcohol testing made such a case-control study possible, no scientist had yet attempted one as of the early 1930s.

These challenges were on full display in the early attempts by scientists in the 1930s to show a causal connection between alcohol and crashes. Dr. Herman Heise of Milwaukee had noticed during his time as a military doctor performing autopsies that many of the soldiers who were involved in fatal car crashes while rushing home to the base to meet curfew had alcohol in their systems.⁷³ He hypothesized that “it is not primarily the obvious ‘drunk’ who constitutes a major road menace but the man I have termed the ‘drinking driver’”⁷⁴ In turn, Heise recognized that “the problem of controlling the drinking driver and pedestrian [was] far from being solved . . . due, in part, to

70. JACOBS, *supra* note 5, at 31.

71. *Id.* at 39.

72. *See, e.g.*, Donelson, *supra* note 22, at 20 (explaining that “increased risk” does not necessarily correspond with “very likely” because of the distinction between relative risk—when drivers with illegal BACs have an increased risk of accident compared to the average sober driver—and absolute risk—the actual probability of a serious crash).

73. LERNER, *supra* note 3, at 25.

74. Heise, *supra* note 16, at 740.

the fact that no accurate statistics [were] available regarding the relationship of alcohol to automobile accidents.”⁷⁵

Heise published a celebrated paper in 1934 first confirming, through laboratory tests involving subjects given alcohol, that a loss of efficiency in motor skills occurred at BACs as low as .02 percent.⁷⁶ Heise also confirmed Emil Bogen’s previous results that many subjects who passed FSTs, and who were all below .10 percent BAC, nonetheless suffered decreased ability to avoid obstacles in the road and increased reaction times.⁷⁷ Heise tested drivers’ skills on actual public roads in Uniontown, New York, where the mayor had closed down a part of the town and let the doctor’s subjects “drive back and forth to [the doctor’s] heart’s content.”⁷⁸ Heise then analyzed 119 consecutive car accidents involving injury or death in Uniontown and noticed that, among the accidents, those involving a drinking driver were more than twice as likely than other accidents to involve more than one injury or death.⁷⁹ Dr. Harger, the Drunk-O-Meter’s inventor, praised Heise’s work as confirmation that “low blood alcohol figures . . . may really be a menace to the public.”⁸⁰

Yet Heise’s study, while an important first step, did not reveal the increased risk of being in an accident at any given BAC. At most, it suggested that, where a driver’s BAC is at some moderately high (but not precisely identified) level, the risk of *injury* from a crash is twice as high as in a crash where no alcohol is involved.

In 1938, Richard Holcomb, a researcher at the Northwestern Traffic Safety Institute, conducted the first attempt at a case-control study showing the increased probability of being in a crash as a function of BAC. As Holcomb noted, breath tests for the first time “ma[d]e possible research into the relation of alcohol to accidents.”⁸¹ With the cooperation of local police, Holcomb recorded the BACs of drivers involved in crashes in Evanston, Illinois, over a twelve-month period, as well as the BACs of other drivers on the same roads during that same time period.⁸² Holcomb understood the critical importance of

75. *Id.* at 739.

76. *Id.*

77. *Id.* at 739–40. Similar studies, showing an effect on motor skills and judgment at low BACs, were also conducted in Europe in the 1930s. LERNER, *supra* note 3, at 26.

78. Dr. Herman A. Heise, Abstract of Discussion, *Alcohol and Automobile Accidents*, 103 JAMA 741, 741 (1934). Heise put himself at risk in conducting this research. “[A] truck swerved across the path of the test car, narrowly avoiding a collision. When I said to the driver, who . . . [was] about 0.13 per cent, ‘Say, wasn’t that a narrow escape from that truck?’ he looked at me with an expressionless face, saying, ‘What truck?’ After this incident, less dangerous experiments . . . were instituted.” Herman A. Heise, Letter to the Editor, 115 JAMA 2200, 2200 (1940).

79. Heise, *supra* note 16, at 740.

80. Rolla N. Harger, Abstract of Discussion, *Alcohol and Automobile Accidents*, 103 JAMA 741, 741 (1934).

81. Holcomb, *supra* note 59, at 1077.

82. *Id.*

having a baseline group against which to compare the prevalence of alcohol use among drivers in accidents:

[I]f it was found that 46 percent of drivers involved in personal injury accidents had been drinking and . . . 46 percent of all drivers had been drinking, the alcohol consumed would seem to have no bearing on accidents. However, if only 12 percent of all drivers had been drinking and yet 46 [percent] of the drivers involved in accidents had been drinking, it would appear that the drinking drivers were suffering more than their share of mishaps, and drinking would seem to be a causal factor in accidents.⁸³

Holcomb secured the permission of randomly stopped drivers to be tested by asking them innocuous questions such as “[a]re you bothered by headlight glare?” for “the purpose of gaining [the driver’s] cooperation and to allow him to get over the strangeness of the situation.”⁸⁴ Then assistants, dressed in “physicians’ white smocks,” approached the driver “for the dual purpose of making a favorable impression on the driver and of making themselves readily visible to other traffic”⁸⁵ Remarkably, of 1,750 drivers stopped, only twenty-four refused to be tested.⁸⁶

Holcomb found that a driver with a BAC of .15 percent was thirty-three times more likely to be involved in a crash than a sober driver.⁸⁷ He also found that almost 25 percent of drivers involved in crashes had a BAC of over .10 percent, compared to less than 3 percent of the control group.⁸⁸ In contrast, he found that the ratio of drinkers in the accident group to drinkers in the control group reached around one-to-one at BACs of .05–.06 percent or lower.⁸⁹ Notably, a full 12 percent of the control group had been drinking to some extent.⁹⁰ While it was only one study, Holcomb’s results were highly influential, no doubt because it was clearly the most salient study thus far analyzing the question of what level of intoxication should be deemed “impairment” for purposes of American criminal law.

3. NSC/AMA Criteria and the Need for a Better Impairment Machine

Based in large part on Holcomb’s study,⁹¹ the newly formed Committee on Tests for Intoxication of the National Safety Council (NSC), along with the AMA, published a joint report in 1939 that set forth what it viewed as

83. *Id.*

84. *Id.* at 1078.

85. *Id.*

86. *Id.* at 1077–78.

87. *Id.* at 1081.

88. *Id.* at 1082 tbl. 12.

89. *Id.* at 1081–82.

90. *Id.* at 1078.

91. See Ellerbrook & VanGaasbeek, *supra* note 34, at 998 (noting that the Committee’s chosen thresholds were created after developing crash risk data “calculated from the data of Holcomb”).

appropriate criteria for determining driver impairment based on BAC for purposes of DUI laws.⁹² The report suggested that there be no DUI prosecution at BACs below .05 percent, that there should always be a DUI prosecution at BACs above .15 percent, that BACs above .15 percent should be presumptive evidence of impairment, and that a driver with a BAC between .05 and .15 percent should be prosecuted only if the circumstances “give definite confirmation of such influence.”⁹³

The NSC/AMA criteria were not simply scientific assessments of the increased risk of accident at different BACs; that was what Holcomb had done. Rather, the report’s recommendations were value judgments, based on the increased crash risk associated with various BACs, about what level of risk should be considered impairment for purposes of triggering criminal liability. The committee’s decision to place the exoneration threshold at .05 percent rather than a lower number reflected a desire to “vindicate[] the . . . temperate driver.”⁹⁴ “Temperate driver” was, of course, not a scientific term, but a socially constructed category of drinking drivers whose level of intoxication was, in the committee’s view, too low to warrant criminal punishment. And while many on the committee wished to set an upper limit of .11 percent rather than the “extremely conservative” .15 percent, others were concerned that .11 percent would subject to criminal liability at least “a few tolerant persons” who could hold their liquor better than most.⁹⁵

Those in favor of a lower threshold pointed to the decisions of Norway and other Scandinavian countries in the mid-1930s to criminalize not only driving while impaired, but also the act of simply driving with a BAC over a certain threshold, sometimes as low as .02 percent.⁹⁶ Historians and criminologists have described this “Scandinavian model” as resulting from those countries’ dire drunk driving problems, “a politically powerful and moralistic temperance movement that was willing to blame almost any social ill on alcohol,”⁹⁷ and a lesser focus on individualism and civil liberties in their crime policies.⁹⁸

In the end, the committee chose .15 percent, knowing that the data suggested that those between .10 and .15 percent were over ten times more likely to cause a crash.⁹⁹ In their judgment, the increased risk associated with .10 percent did not unambiguously merit criminal punishment, at least not in a

92. See HERMAN A. HEISE ET AL., REPORT OF THE COMMITTEE TO STUDY PROBLEMS OF MOTOR VEHICLE ACCIDENTS (1939 & 1942).

93. LERNER, *supra* note 3, at 28 (citing HERMAN A. HEISE ET AL., REPORT OF THE COMMITTEE TO STUDY PROBLEMS OF MOTOR VEHICLE ACCIDENTS (1939 & 1942)).

94. *Id.* at 29.

95. *Id.*

96. See, e.g., ROSS (1984), *supra* note 18, at 22–24.

97. *Id.* at 22.

98. See, e.g., *id.* at 22–24.

99. *Id.* at 28.

nation whose citizens were still reeling from the collapse of Prohibition and had a laissez-faire attitude toward alcohol.¹⁰⁰ And while it is not clear why the NSC/AMA would have special skills in making such value judgments, their criteria were highly influential on DUI policy for decades thereafter. In 1939 alone, Indiana and Ohio adopted the chart wholesale in their new DUI laws.¹⁰¹ Several other states, as well as the Uniform Vehicle Code, followed suit in the next few years.¹⁰²

Even as the Drunk-O-Meter's readings were being used to study accident rates and thereby set national crime policy, scientists and law enforcement officials were acknowledging that the device was not quite the impairment machine the public believed it to be. Dr. Holcomb noted up front in his 1938 study that the Drunk-O-Meter was "somewhat less accurate than urinalysis."¹⁰³ Later, the NSC itself acknowledged that the Drunk-O-Meter offered only a "rough measure" of BAC and "require[d] a chemist's delicate balance" for an accurate reading.¹⁰⁴ By the 1940s, some studies began to question whether BAC was even a good proxy for impairment,¹⁰⁵ with one trio of Canadian studies concluding that BAC, at least at low levels, was not a reliable indicator of how alcohol affects the brain.¹⁰⁶ Based on these and other studies, the Michigan Supreme Court in 1949 held that the Drunk-O-Meter did not pass muster under the *Frye v. United States* "general acceptance" test for admissibility of novel scientific evidence.¹⁰⁷ The court analogized the test's "continuous series of errors" to "a slot machine,"¹⁰⁸ and noted that because of its questionable reliability and the "elaborate exposition" necessary to explain its inner workings, its admission might result, like the polygraph, "in a trial of the [machine] rather than the issues in the case."¹⁰⁹

100. LERNER, *supra* note 3, at 30.

101. ROSS (1984), *supra* note 18, at xv (citing Indiana Acts 1939, ch. 48, § 52).

102. *See, e.g.*, McKay v. State, 235 S.W.2d 173, 175 (Tex. Crim. App. 1950) (citing the study and the presumptions suggested therein with approval).

103. Holcomb, *supra* note 59, at 1077.

104. Dorlaque, *supra* note 62, at 167.

105. *See, e.g.*, *Time a Factor in Drunkenness, Laboratory Tests Reveal*, POPULAR SCI., Aug. 1942, at 207 (noting a Stanford Medical School study showing that while an initial drink caused some impairment, performance "returned to normal" afterwards "despite small doses which kept the [BAC] . . . constant").

106. Charles U. Letourneau, *Chemical Tests in Alcoholic Intoxication*, 28 CAN. B. REV. 858, 871 (1950); I.M. Rabinowitch, *Medicolegal Aspects of Chemical Tests of Alcoholic Intoxication*, 26 CAN. B. REV. 1437, 1439 (1948); I.M. Rabinowitch, *Medicolegal Aspects of Chemical Tests of Alcoholic Intoxication*, 39 J. CRIM. L. & CRIMINOLOGY 225, 234 (1948).

107. *People v. Morse*, 38 N.W.2d 322, 324 (Mich. 1949) (citing *Frye v. United States*, 298 F. 1013 (1923)).

108. *Id.* (quoting a doctor sworn in for the defense).

109. *Id.* (quoting *State v. Bohner*, 246 N.W. 314, 317 (Wis. 1933) (internal quotation marks omitted)); *see also* *State v. Hunter*, 68 A.2d 274, 276-77 (N.J. Super. Ct. App. Div. 1949) (granting new trial based on a Drunk-O-Meter expert's failure to use the right type of druggist's scale to measure the weight of absorbed vapor).

Notwithstanding the misgivings of some courts and scientists about the reliability of the Drunk-O-Meter, Dr. Harger defended its use in the absence of a better alternative to addressing the drunk driving crisis. In a rebuttal to the Canadian study, he insisted that “the disposition of the daily crop of drunken driving cases cannot await absolute perfection in the field of chemical tests.”¹¹⁰ The Supreme Court itself expressed similar sentiments. In holding in 1957 that involuntary blood testing of a DUI suspect did not violate due process,¹¹¹ the Court relied in no small part on the perceived necessity of such scientific tests, however imperfect or invasive, in addressing the harm caused by drunk driving:

Modern community living requires modern scientific methods of crime detection lest the public go unprotected. The increasing slaughter on our highways, most of which should be avoidable, now reaches the astounding figures only heard of on the battlefield. The States, through safety measures, modern scientific methods, and strict enforcement of traffic laws, are using all reasonable means to make automobile driving less dangerous. As against the right of an individual . . . must be set the interests of society in the scientific determination of intoxication, one of the great causes of the mortal hazards of the road.¹¹²

Luckily for Dr. Harger, absolute perfection—or at least a step closer to it—arrived before the scientific conflict over the Drunk-O-Meter’s reliability had a chance to affect the legitimacy of Holcomb’s crash studies or the NSC/AMA’s criminal policy recommendations. In 1954, Robert Borkenstein, a former Indiana police officer with only a high school education, invented the “Breathalyzer” while on a two-week vacation.¹¹³ The machine boasted standardized reagents and reaction times, as well as a “colorimetry” system that offered more precision than the Drunk-O-Meter (see Table 2).¹¹⁴ The breathalyzer’s portability allowed people to use it at cocktail parties for informal experimentation; at one scientific conference in 1956, attendees watched a colleague become visibly drunk at a .08 percent BAC.¹¹⁵ Because early models forced users to manually set a baseline, however, the machine’s detractors labeled it the “Dial-a-Drunk” machine, out of a concern that police officers could theoretically manipulate it to indicate impairment in a suspect.¹¹⁶ A few courts refused to admit Breathalyzer readings until the machine’s reliability became more established, likening it to the “push button justice” of

110. R.N. Harger, *Medicolegal Aspects of Chemical Tests of Alcoholic Intoxication: Comments on Dr. I.M. Rabinowitch’s Paper*, 39 J. CRIM. L. & CRIMINOLOGY 402, 410 (1948).

111. *Breithaupt v. Abram*, 352 U.S. 432, 437 (1957).

112. *Id.* at 439.

113. LERNER, *supra* note 3, at 48–49.

114. BREATH TESTING FOR PROSECUTORS, *supra* note 58.

115. LERNER, *supra* note 3, at 49.

116. BREATH TESTING FOR PROSECUTORS, *supra* note 58, at 11.

another recent invention used in traffic prosecutions, the radar gun.¹¹⁷ Still, by the mid-1950s, public and judicial opinion toward breath machines reached a sort of repose. Conviction rates increased, and the public began to perceive breath-test results as “practically impossible to deny in court.”¹¹⁸



TABLE 2. Robert Borkestein training police officers on the use of the Breathalyzer in 1969. (Reprinted with permission from Indiana University Archives)

Even as courts and the public began to accept Breathalyzer results as reliable proof of BAC, jurors continued to acquit in drunk driving cases in larger numbers than in trials for other crimes.¹¹⁹ Perhaps this was a function of a new wave of scientific challenges at trial by the well-organized DUI defense bar, or of jury nullification in protest of sentences that the public viewed as too punitive,¹²⁰ or even an unintended effect of the NSC’s setting the presumptive threshold at .15 percent.¹²¹ Or, as public health officials would later claim, perhaps the public continued to believe that moderate levels of intoxication were not morally blameworthy.¹²² As one historian has described public opinion at the time, there was still a sense that a moderate level of intoxication suggested the driver was merely a “social drinker,” a term with a “positive

117. *People v. Seger*, 314 N.Y.S.2d 240, 245 (J. Ct. 1970) (quoting *People v. Offermann*, 125 N.Y.S.2d 179, 185 (Sup. Ct. 1953) (internal quotation marks omitted)).

118. Dorlaque, *supra* note 62, at 270; *see also* Editorial, *Chemical Tests and the Drunken Automobile Driver*, 154 JAMA 1279 (1958) (reporting conviction rates above 95 percent in Detroit, Los Angeles, Milwaukee, Minneapolis, and Chicago).

119. *See generally* KALVEN & ZEISEL, *supra* note 25.

120. This was the overwhelmingly prevalent theory of judges interviewed by Kalven and Zeisel in DUI cases, who noted that jurors could “put themselves in [the] defendant’s position.” *Id.* at 266 (quoting a judge’s comments in a drunk driving case).

121. LERNER, *supra* note 3, at 51 (arguing that .15 percent essentially became the “default value for DUI prosecutions”).

122. *See, e.g.*, 1968 REPORT, *supra* note 12, at 90, 103 (noting that only 10 percent of the public “felt that there was something bad or inherently stupid about the *drinking* driver” and that jurors would often acquit if the defendant was not a hard core drinker).

connotation, suggesting that such an individual was both sociable and harmless.”¹²³

4. *Haddon’s Science-Based Agenda for Using BACs to Target Dangerous Driving*

In response to this lingering juror ambivalence, public officials in the 1950s and early 1960s did not call for the removal of the question of impairment from the jury’s consideration. The NSC could have easily done so, of course, by recommending that states adopt so-called per se laws. Rather than leaving the issue of impairment for the jury to decide on the basis of the driver’s BAC and other circumstances, a per se law actually redefines impairment in terms of BAC. In a per se prosecution, the question for the jury is not whether the state proved that the driver was impaired, but simply whether the state proved the requisite BAC based on the breath test result. While every state eventually adopted per se laws, such punitive measures did not occur in most states until well after 1980 and in some states, not until 2004.¹²⁴

Instead, the period from the 1940s through the 1970s was defined by a paradigm of treating drunk driving as a public health problem, under a so-called epidemiological model rather than a law-and-order model. With the growth of the automobile culture and development of the interstate highway system, car crashes became one of the most serious health crises in the United States. By the 1960s, roughly 50,000 Americans were killed annually in car crashes alone.¹²⁵ If jurors—or the public, the jury writ large—looked at these statistics and still did not see low levels of intoxication in drivers as a menace, then it was incumbent upon officials and scientists both to confirm the risk through careful study and to educate the public about the risk. Any other disease would be treated similarly, after all. In the words of one official, the focus of DUI laws was first and foremost to prevent the “thousands of unnecessary deaths” from the disease of “alcoholism” itself.¹²⁶

The seeds of this public health movement had been sown shortly after the Drunk-O-Meter was invented. The 1930s and 1940s saw the founding of the Research Council on Problems of Alcohol (1937), the Yale School of Alcohol Studies (1943), the National Council for Education on Alcoholism (1944), Alcoholics Anonymous (1935), and the NSC’s Committee on Tests for Intoxication (later the Committee on Alcohol and Other Drugs, or CAOD) (1936).¹²⁷ Holcomb’s famous 1938 study was, after all, commissioned by a traffic safety institute in Chicago.¹²⁸

123. LERNER, *supra* note 3, at 23.

124. *See id.* at 145.

125. *Id.* at 53.

126. *Id.* at 19 (quoting National Council for Education on Alcoholism President Mary Mann).

127. *Id.* at 19, 22.

128. *Id.* at 26.

The person typically credited with conceptualizing the drunk driving problem as a post-war public health emergency in the 1950s and 1960s, and with ensuring that proper scientific studies undergirded public policy on drunk driving, was a Harvard-educated, “workaholic government bureaucrat” in New York by the name of William Haddon Jr. (Table 3).¹²⁹ Unlike law enforcement types such as Borkestein and Holcomb, Haddon came from the world of epidemiology.¹³⁰ Haddon noticed that, up to the late 1950s, most of the data about car crashes—other than Holcomb’s study, which was done using very early breath-testing models—was anecdotal.¹³¹ Even in the influential 1957 treatise *Chemical Tests and the Law* by Robert Donigan of the Traffic Institute at Northwestern, the author only cited studies showing that a high percentage of drivers involved in fatal crashes had been drinking.¹³² Haddon understood what Holcomb had known before him: that without an estimate of the baseline percentage of the other drivers on the road who had been drinking, such statistics by themselves were insufficient to establish that alcohol *caused* accidents. And even if alcohol did cause accidents, existing studies left unclear the relationship between specific BACs and increased risk.

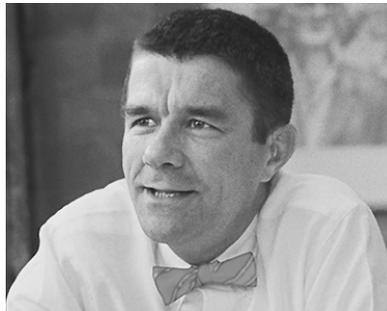


TABLE 3. William Haddon, Jr.

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Haddon’s first move was to study the role played by alcohol in fatal single-car crashes in Westchester County, New York, from 1949 to 1957.¹³³ When multiple cars are involved in a crash, it is often difficult to determine which car caused the crash, and what precise behavior or condition caused the crash. Less ambiguity exists in a single-car crash; the lone driver’s impairment

129. *Id.* at 38.

130. *Id.* at 53–54.

131. *Id.* at 54.

132. ROBERT L. DONIGAN, *CHEMICAL TESTS AND THE LAW* 1, 173–74 (1957); *see also* LERNER, *supra* note 3, at 55 (citing a 1956 Delaware study finding that 59 percent of crash-related deaths involved alcohol, and a Cleveland study showing that 40–60 percent of crash victims had alcohol in their blood).

133. William Haddon Jr. & Victoria A. Bradess, *Alcohol in the Single Vehicle Fatal Accident: Experience of Westchester County, New York*, 169 *JAMA* 1587 (1959).

is much more likely to have been a causal factor.¹³⁴ A full half (forty-one) of the eighty-three fatally injured drivers in single-car crashes during the period, whose BAC had been tested within four hours of their crash, had a BAC of .15 percent or more. Another 20 percent (seventeen) had a BAC between .05 and .14 percent, and only three of eighty-three drivers had non-zero BACs below .05 percent.¹³⁵ The study was the first since the 1930s to provide a scientific basis for the claim that certain BACs were actually a frequent cause of car crashes.¹³⁶

Haddon next performed a classic case-control study of fatal crashes in New York City in 1962.¹³⁷ He compared the BACs of New York drivers killed in nighttime car accidents with a control group of drivers at the same time and location who were not involved in accidents.¹³⁸ By focusing on nighttime crashes, Haddon focused on the period involving the richest data in terms of alcohol and crash risk; that is, when both the percentage of drinking drivers on the road and the percentage of drivers involved in crashes are highest.¹³⁹ Haddon found that 26 percent of the control group had a BAC over zero, and none had a BAC at or over .25 percent.¹⁴⁰ In contrast, 73 percent of those killed had alcohol in their system, and over 50 percent had BACs at or over .25 percent.¹⁴¹ While these studies did not definitively connect low levels of

134. See, e.g., Kenneth S. Opiela et al., *Driving After Dark*, 66 PUB. ROADS 2 (2003), available at <https://www.fhwa.dot.gov/publications/publicroads/03jan/05.cfm> (noting that drunk driving rates and fatal accident rates are both highest at night); Paul L. Zador, *Alcohol-Related Relative Risk of Fatal Driver Injuries in Relation to Driver Age and Sex*, 52 J. STUD. ALCOHOL 302, 303 (1991) (“The relative crash risk for drivers fatally injured in single-vehicle crashes provides a good measure of the true contribution of alcohol to increased risk of involvement in the serious crash.”).

135. Haddon & Bradess, *supra* note 133, at 1589 (explaining that twenty-two drivers tested negative, therefore leaving three drivers who had BACs between 0.01 and 0.05 percent). Most (63 percent) of the crashes occurred at night. *Id.*

136. Haddon also conducted a pedestrian study in 1961. While it did not directly shed light on the relationship between BAC and crash risk, it confirmed that a case-control study could show that the same BACs that generated risk in driving also generated risk in walking. LERNER, *supra* note 3, at 54. While a group of researchers had previously published a study on pedestrian deaths and alcohol in 1941, the study merely counted the percentage of pedestrians in fatal car crashes who tested at various BACs, and included no control group. See Thomas A. Gonzales & Alexander O. Gettler, *Alcohol and the Pedestrian in Traffic Accidents*, 117 JAMA 1523 (1941). Haddon, in contrast, compared fifty pedestrians who died in car crashes at particular locations with a control group of two hundred pedestrians who had walked safely through the same locations at the same time. LERNER, *supra* note 3, at 54. While only 33 percent of the control group had been drinking, 74 percent of those killed had been drinking. *Id.*

137. James R. McCarroll & William Haddon Jr., *A Controlled Study of Fatal Automobile Accidents in New York City*, 15 J. CHRON. DISEASES 811 (1962).

138. *Id.* at 811–12.

139. Haddon did not make these advantages of nighttime study explicit to his readers, but they are evident from later research. See, e.g., Michael D. Keall et al., *The Contribution of Alcohol to Night Time Crash Risk and Other Risks of Night Driving*, 37 ACCIDENT ANALYSIS & PREVENTION 816, 816–17 (2005).

140. *Id.* at 824.

141. *Id.*

intoxication to a specific increase in crash risk, they laid the groundwork by showing that alcohol was a causal factor in crashes.

As Haddon became convinced that a small percentage of drivers were causing a large percentage of highway deaths, he sought to end the use of the term “accident” altogether and instead referred to alcohol-related crashes as a preventable disease.¹⁴² Based on Holcomb’s study and his own studies up to that point, Haddon also successfully urged Governor Rockefeller’s administration in 1960 to push to make driving with a BAC of .10 percent or higher an infraction under New York state law.¹⁴³ That same year, the CAOD finally lowered its suggested presumptive threshold for DUI from .15 percent to .10 percent, and the Uniform Vehicle Code followed suit two years later.¹⁴⁴

Still, by 1964, only one state other than New York had actually changed its laws to incorporate the change from .15 percent to .10 percent.¹⁴⁵ And while Haddon had raised the scientific bar for crash risk studies significantly, there were still few if any studies, besides Holcomb’s, showing the precise level of increased risk of accident at BACs lower than .15 percent.

Robert Borkenstein himself would finally fill that gap with his famous Grand Rapids Study.¹⁴⁶ Borkenstein took the 9,353 drivers involved in car crashes in the city of Grand Rapids, Michigan for a twelve-month period in 1962–63 and compared them to a control group of 8,008 drivers who had driven at the same times and locations without being involved in a crash.¹⁴⁷ Borkenstein found that drivers with BACs less than .04 percent were no more likely than sober drivers to be in crashes and that, surprisingly, such a low but non-zero BAC actually lowered one’s chances of being in a crash.¹⁴⁸ But he also found that those with a BAC of .08 percent were nearly twice as likely to be in a crash than similarly situated sober drivers; those with a BAC of .10 percent were nearly six times as likely; and those at .15 percent were over ten times more likely.¹⁴⁹ Finally, Borkenstein found that injury from a crash increased with the driver’s BAC level, starting at .08 percent.¹⁵⁰

By 1965, Haddon, Borkenstein, and others had firmly established the scientific basis for labeling drunk driving a public health emergency. Reacting

142. See LERNER, *supra* note 3, at 55.

143. *Id.* at 58.

144. *Id.* at 59.

145. *Id.*

146. ROBERT F. BORKENSTEIN ET AL., THE ROLE OF THE DRINKING DRIVER IN TRAFFIC ACCIDENTS (1964).

147. *Id.*

148. *Id.* at 213. CAOD would later explain in a 1988 report that this so-called “Grand Rapids Dip,” was simply an “artifact resulting from different mixes of infrequent, moderately frequent, and very frequent drinkers at various [BACs].” NAT’L SAFETY COUNCIL, *Policy Statement on Impairment at Low Alcohol Concentrations*, in A HISTORY OF THE COMMITTEE ON ALCOHOL AND OTHER DRUGS (CAOD), app. at 114.

149. BORKENSTEIN ET AL., *supra* note 146, at 213.

150. *Id.* at 176–77.

to the new urgency of the issue, President Johnson created the Department of Transportation (DOT) in 1966 and named Haddon the country's first "traffic czar,"¹⁵¹ heading up what would eventually be known as the National Highway Traffic Safety Administration (NHTSA). The DOT began to require state officials to record driver BACs in fatal crashes, setting the stage for further study of dose-related crash risk.¹⁵²

In addition to influencing federal policy, the Grand Rapids Study convinced several more states to adopt, with strong public support, statutory rebuttable presumptions of impairment for BACs over .15 percent.¹⁵³ By 1969, all but seven states had done so.¹⁵⁴ Many states even lowered their presumptive limit from .15 percent to .10 percent in response to CAOD's recommendation to do so.¹⁵⁵ Soon, federal laws would link highway funds to the willingness of states both to lower their presumptive threshold and implement so-called implied consent laws, which required stopped motorists to submit to breath testing or have their failure to submit used against them as evidence of their impairment.¹⁵⁶ By the 1960s, a majority of Americans expressed support for implied consent laws,¹⁵⁷ and by 1973, every state had adopted one.¹⁵⁸

Still, by 1968, only Nebraska had taken the next step and passed a per se law criminalizing the mere act of driving with a BAC over a certain threshold.¹⁵⁹ The remaining states, as well as the Uniform Vehicle Code, still left it to the jury to ultimately decide whether the defendant had been impaired, even at levels as high as .15 percent and above.¹⁶⁰ Moreover, acquittal rates in cases involving BACs below states' presumptive thresholds were stubbornly high.¹⁶¹ Haddon, for one, blamed these high acquittal rates not on a legitimate disagreement among local communities as to whether driving with a dangerously high BAC was morally unacceptable, but on simple public ignorance.¹⁶²

151. LERNER, *supra* note 3, at 60.

152. *Id.* at 61.

153. See 1968 REPORT, *supra* note 12, at 91; Andrew Gore, *Know Your Limit: How Legislatures Have Gone Overboard with Per Se Drunk Driving Laws and How Men Pay the Price*, 16 WM. & MARY J. WOMEN & L. 423, 427 (2010).

154. William Haddon, *The Shocking Facts About Drinking and Driving*, POPULAR SCI., May 1969, at 81.

155. See *id.*

156. LERNER, *supra* note 3, at 61.

157. 1968 REPORT, *supra* note 12, at 88.

158. BREATH TESTING FOR PROSECUTORS, *supra* note 58, at 5.

159. See 1968 REPORT, *supra* note 12, at 122.

160. *Id.* at 101, 122. There was some variation among states in whether the prohibition extended to all alcohol, to merely "alcoholic liquor," or more broadly to "intoxicants." Most states eventually adopted a broad definition prohibiting driving "under the influence" of an intoxicating substance. *Id.* at 102.

161. See LERNER, *supra* note 3, at 31.

162. See generally 1968 REPORT, *supra* note 12, at 88–99 (discussing "public opinion" about drinking drivers); see also *id.* at 122 (discussing the need to "restrain[]" "overly sympathetic juries" through per se laws).

Haddon's newly formed DOT submitted a strongly worded 1968 report to Congress that became a "seminal document in the emergence of drunk driving as an American social problem."¹⁶³ It catalogued the few existing studies concluding that drivers experience impairment at BACs lower than those "at which they outwardly appear to be mildly affected or intoxicated."¹⁶⁴ Yet, as Haddon noted, states often chose not to prosecute DUIs, even those supported by chemical test results, because juries so often acquitted.¹⁶⁵ Although a majority of Americans in 1968 believed that penalties for drunk driving were too lenient,¹⁶⁶ only 10 percent "felt that there was something bad or inherently stupid about the drinking driver."¹⁶⁷ Even when jurors thought it likely that the defendant had been driving while intoxicated, they would often acquit absent evidence that the defendant was a "hard core" or "problem" drinker.¹⁶⁸ Some people still believed that low concentrations of alcohol actually "sharpen[ed]" driving ability.¹⁶⁹

In his zeal to prove to Congress the enormity of the problem, Haddon played somewhat fast and loose in his description of existing studies. Lumping together the data from several small regional studies of fatal crashes, for example, Haddon declared that "almost half of the drivers were found to have [BACs] of .10 or greater,"¹⁷⁰ although he had rightfully criticized others for drawing causal inferences based simply on number of crashes involving alcohol. Other claims made in the report, such as that "alcohol has been found to be the largest single factor leading to fatal crashes," were unsupported by citation and simply inaccurate.¹⁷¹ In a follow-up report to Congress ten years later, the DOT was much more modest in its claims, concluding that "it is impossible to state conclusively that impairment of the ability to perform critical driving tasks by alcohol has caused any given fraction of crashes involving alcoholics or problem drinkers."¹⁷²

Whatever his reasons for embellishing the data in this way, Haddon used these dramatic claims to urge Congress to incentivize states to lower the presumption of impairment to .10 percent or, better yet, to pass per se laws

163. JACOBS, *supra* note 5, at 27.

164. 1968 REPORT, *supra* note 12, at 45.

165. *Id.* at 103.

166. *Id.* at 88.

167. *Id.* at 90 (emphasis added).

168. *See id.* at 103. A study cited by *Popular Science* in 1958 noted that while the public disapproved of drunk driving, "indignation was reserved for flagrant, not technical, violations— . . . not just one or two drinks." *What Do Motorists Really Believe About Safety?*, POPULAR SCI., Oct. 1958, at 20.

169. Haddon, *supra* note 154, at 81.

170. 1968 REPORT, *supra* note 12, at 14.

171. JACOBS, *supra* note 5, at 205 n.2 (quoting 1968 REPORT, *supra* note 12, at 11).

172. RALPH K. JONES & KENT B. JOSCELYN, HIGHWAY SAFETY RES. INST., ALCOHOL AND HIGHWAY SAFETY 1978: A REVIEW OF THE STATE OF KNOWLEDGE 41-42 (1978) (report prepared for the DOT).

defining impairment directly in terms of BAC.¹⁷³ The United Kingdom had passed a similar law in 1967,¹⁷⁴ and Haddon noted its effectiveness in increasing the conviction rate:

Such a [per se] statute eliminates the concepts of intoxication or impairment of driving ability altogether. Once the prohibited [BAC] has been competently put into evidence, the defense lawyer may only attack the correctness of the evidence, and should seldom succeed where the system employed has been carefully established. Overly sympathetic juries can be restrained by strict instructions to convict if the laboratory evidence is found to be valid. Trials can be shorter, saving the resources of police who would otherwise act as witnesses, and of the courts themselves. Experience under the English [per se .08] statute has shown a significantly higher rate both of guilty pleas and of convictions after a full trial.¹⁷⁵

Though few states moved immediately to a per se regime as a result of Haddon's report, its vehemence would lend legitimacy years later to arguments for per se laws and for lowering the limit to .08 percent, when the political climate was more conducive to passing such laws. In essence, it changed the national conversation on drunk driving and low-level intoxication. Not surprisingly, CAOD in 1971 recommended lowering the presumptive BAC for impairment from .10 percent to .08 percent.¹⁷⁶ In 1975, it recommended that states redefine their presumptions to be triggered not only by a .08 percent *blood*-alcohol concentration, but also by a .08 percent *breath*-alcohol concentration. This latter shift eliminated the relevance of defendants' frequent assertions that their own bodily BAC/BrAC ratio—the “partition ratio”—was different from the one programmed into the Breathalyzer, and thus that a .08 percent test result, while perhaps proving a BrAC of .08 percent, did not prove a BAC of .08 percent.¹⁷⁷

Haddon and his approach also legitimized, in the name of public health, shaming drunk driving apologists. The Licensed Beverage Industries (LBI), a trade group representing the liquor industry, ran a controversial magazine ad in 1970 that included a chart of the number of drinks one could imbibe and still “drive safely,” based on body weight.¹⁷⁸ The NSC and AMA complained to the LBI about the ad, noting that while following the chart might allow you to remain slightly under most states' legal intoxication limit of .10 percent BAC, that level still left one impaired and unsafe to drive.¹⁷⁹ Four months after the ad ran, the DOT sent a letter to LBI noting the difference between being “safe

173. 1968 REPORT, *supra* note 12, at 121–22.

174. *Id.* at 106 (citing Road Safety Act of 1967).

175. *Id.* at 122.

176. BREATH TESTING FOR PROSECUTORS, *supra* note 58, at 5.

177. *See* *People v. Vangelder*, 312 P.3d 1045, 1057–58 (Cal. 2013).

178. LERNER, *supra* note 3, at 66.

179. *Id.* at 66–67.

from breaking the law” and “safe from having an automobile crash.”¹⁸⁰ While states and jurors might not be ready to declare BACs lower than .10 percent to be criminal, the DOT warned that no one should be declaring such levels of intoxication “safe” for driving.¹⁸¹

5. *The Punitive Turn in the 1980s and the Final Push Toward .08 Percent*

Even with the prodding of the DOT, most states before 1980 were in no hurry to pass per se laws, turn DUI offenses into felonies,¹⁸² or lower their BAC thresholds to .08 percent. Public support for such laws was not universal, and legislators’ own views of the social harm from low-level impairment, and the desirability of increasing police power over motorists, were varied. Moreover, the scientific community and criminologists were divided at the time as to whether the increased crash risk from .08 percent BAC was sufficiently scientifically proven, morally blameworthy, or capable of deterring would-be impaired drivers.¹⁸³

In 1980, with the formation of the politically formidable groups Mothers Against Drunk Driving (MADD) and Remove Intoxicated Drivers (RID), the public conversation about DUI—as with other drug crimes—began to shift away from public health toward a law-and-order approach.¹⁸⁴ MADD originally focused primarily on fighting local practices granting lenient sentences and plea deals, giving a voice to victims in the criminal process, and lowering the legal BAC below .15 percent in those states that still had that standard.¹⁸⁵ In making its case, most if not all of the anecdotes MADD highlighted involved obviously intoxicated drivers who received very light sentences even after causing serious injury or death.¹⁸⁶

MADD’s national presence and influence culminated in the creation of new federal and state apparatuses to combat drunk driving. By 1984, Ronald Reagan had created a Presidential Commission on Drunk Driving and signed federal legislation tying highway funds to states’ willingness to lower their legal limits to .10 percent BAC, impose stiffer penalties on those convicted, and raise the legal drinking age from eighteen to twenty-one.¹⁸⁷ Between 1981

180. *Id.* at 67.

181. *Id.*

182. See 1968 REPORT, *supra* note 12, at 103 (noting that generally all DUI offenses as of 1968 were misdemeanors).

183. See, e.g., Gore, *supra* note 153, at 428; U.S. GEN. ACCOUNTING OFF., GAO/RCED-99-179, HIGHWAY SAFETY: EFFECTIVENESS OF STATE .08 BLOOD ALCOHOL LAWS 2 (1999), available at <http://www.gao.gov/archive/1999/rc99179.pdf>.

184. See LERNER, *supra* note 3, at 75–78. See generally MICHELLE ALEXANDER, THE NEW JIM CROW: MASS INCARCERATION IN THE AGE OF COLORBLINDNESS (2010).

185. See, e.g., LERNER, *supra* note 3, at 77.

186. See, e.g., *id.* at 73–74.

187. *Id.* at 89–90.

and 1986, 729 new state DUI laws were passed.¹⁸⁸ While some officials from the Haddon public-health-model era did not fully approve of the wave of punitive legislation, historian Barron Lerner has noted that the new laws were “congruent with Reagan-era efforts to fix social issues through the criminal justice system.”¹⁸⁹

In 1986, prominent sociologist J. Laurence Ross and law professor Graham Hughes took on MADD’s claim that a significant decline in crash fatalities from 1980 to 1985 was due to law-and-order drunk driving reform efforts.¹⁹⁰ There seemed to be no dispute that the number of deaths attributed to drunk driving dropped significantly in the wake of many of the policies MADD and RID helped to pass.¹⁹¹ But these reforms were varied: public education on the dangers of drunk driving, cultural shifts away from the romanticization of alcohol use, fewer lenient plea deals, more jail time, swifter and more certain penalties, more frequent administrative sanctions such as license revocation, banning happy hours, and nonalcoholic sporting events and graduations.¹⁹² Ross and Hughes argued that the decline could be due to these and other causes, such as “reduced speed limit, safer cars, better roads with better lighting, [and] improvements in medical techniques and in the delivery of emergency medical services.”¹⁹³

At the same time, the public’s attention turned away from drunk driving, as the AIDS epidemic and other new social problems emerged.¹⁹⁴ It would take “one last push” by a government official, then-surgeon general C. Everett Koop, to remind the public that drunk driving was a critical public health issue.¹⁹⁵ Koop held a workshop at the Mayflower Hotel in Washington, D.C. in December 1988, at which he argued for a shift to .08 percent. The workshop itself was a reaction to the tragic night of May 14, 1988, when a driver with a BAC of .24 percent was driving on the wrong side of an interstate in Carrollton, Kentucky, and crashed into a school bus, which then burst into flames killing 27 children coming home from a church trip.¹⁹⁶

Still, by 1994, only twelve states had reduced their limit to .08 percent, and the beverage industry fought Koop’s plan. In an ironic twist, the industry

188. *Id.* at 89.

189. *Id.* at 90–91; see also Craig Reinerman, *The Social Construction of an Alcohol Problem: The Case of Mothers Against Drunk Drivers and Social Control in the 1980s*, 17 *THEORY & SOC’Y* 91, 92 (1988) (noting that MADD’s agenda fit the “social-control strategies of the Reagan administration and a renascent right”).

190. See H. Laurence Ross & Graham Hughes, *Drunk Driving: What Not To Do*, *NATION*, Dec. 13, 1986, at 663.

191. See, e.g., LERNER, *supra* note 3, at 91 (noting a reported 32 percent drop from 1980 to 1985 in deaths attributed to drunk driving).

192. *Id.* at 91–92.

193. Ross & Hughes, *supra* note 190, at 664.

194. LERNER, *supra* note 3, at 115.

195. *Id.*

196. *Id.* at 125.

made two MADD founders, Cindi Lamb and Candy Lightner, its spokeswomen.¹⁹⁷ Lightner argued publicly that the vast majority of drunk driving offenders had BACs well over .08 percent, including the driver that killed her daughter, and that the push to lower limits to .08 percent from .10 percent would “dilut[e] law enforcement efforts” against “truly dangerous drivers.”¹⁹⁸ Lightner also argued that lowering speed limits would save more lives than lowering BACs.¹⁹⁹ The American Beverage Institute (ABI) went further, calling the push for .08 percent “neoprohibitionism,” and warning that calls for a .05 percent limit could not be far behind.²⁰⁰ An editorial in the *Washington Times* declared the push for .08 percent a “prohibitionist jihad driven by hysteria, not medical reality.”²⁰¹

Academics also expressed skepticism. Drawing on the earlier work of Gusfield and Ross, several criminologists from the late 1980s to the early 2000s argued that criminalizing BACs under .10 percent made little sense both in light of the limits of law enforcement and goals of the criminal law. Some argued that criminalizing driving at low BACs could only be justified on deterrence grounds, not moral grounds, given the modest increase in crash risk; and that even if the data showed that punitive DUI laws were effective, deterrence alone was an insufficient basis for invoking the criminal law.²⁰² Others argued that the increased risk of a crash based on a BAC of .08 percent was simply not high enough to warrant punishment, given that the “overwhelming majority of impaired drivers caused no harm.”²⁰³ Law professor James Jacobs, in his seminal 1989 book on drunk driving, argued that focusing attention on “light drinkers” would “undermin[e] the effort to identify and isolate drunk driving as a major problem.”²⁰⁴ Others argued that per se laws set at low BACs gave insufficient notice to potential offenders, that driving after moderate drinking was a commonplace and morally accepted activity that therefore should not be criminalized, and that per se laws arbitrarily set a numerical line that did not correlate with actual impairment.²⁰⁵

Amid this debate came yet another tragedy that would revive the political viability of reducing the legal limit to .08 percent: Princess Diana’s death in

197. *Id.* at 126.

198. *Id.* at 128; see also Doug Bandow, *Targeting the Most Dangerous Drunk Drivers*, WASH. TIMES, Jan. 28, 1994, at A23; Katherine Griffin, *No Longer MADD*, SAN FRANCISCO CHRON., Aug. 7, 1994, at 6, 7; Connie Koenenn, *The Company She Keeps*, L.A. TIMES, Jan. 26, 1994, available at http://articles.latimes.com/1994-01-26/news/vw-15591_1_candy-lightner.

199. LERNER, *supra* note 3, at 128.

200. *Id.* at 136.

201. *Power MADD*, WASH. TIMES, Mar. 6, 2000, at A16.

202. See, e.g., James D. Stuart, *Deterrence, Desert, and Drunk Driving*, 3 PUB. AFF. Q. 105, 109 (1989).

203. LERNER, *supra* note 3, at 129.

204. JACOBS, *supra* note 5, at 43.

205. See, e.g., Douglas N. Husak, *Is Drunk Driving a Serious Offense?*, 23 PHIL. & PUB. AFF. 52, 71–73 (1994).

1997 in Paris during a high-speed car chase with paparazzi.²⁰⁶ Notably, though, Diana's driver's BAC was .2275 percent.²⁰⁷ Nevertheless, MADD and numerous state legislators seized upon the episode to push the .08 percent issue once again; one bill introduced in New York to lower the limit from .10 percent to .08 percent was even dubbed the "Diana bill."²⁰⁸ In contrast, the general counsel of the ABI countered that Diana "was killed by an alcohol abuser, not a social drinker."²⁰⁹

Nevertheless, in 2000, the momentum for the switch to .08 percent was enough to convince President Clinton to sign a bill withholding highway funds from the thirty-one states that had not yet adopted a .08 percent standard.²¹⁰ Meanwhile, an influential meta-study by NHTSA and the CDC in 2001 found a 7 percent decrease in traffic deaths in states with .08 percent, and a 3.8 to 24 percent decrease in fatal crashes in states with zero-tolerance laws for minors.²¹¹ By 2004, three years after this report, all fifty states and the District of Columbia had per se offenses set at 0.08 percent BAC.²¹²

*B. Haddon's Legacy: A Compulsory Scientific Framework for Proving
Criminally Dangerous Impairment*

The Haddon-era DUI alcohol model established how to determine, through valid science, the relationship between impairment and dangerousness. The scientific framework underlying per se DUI alcohol laws was—and continues to be—the standard against which to judge drug impairment as a fatality risk. Those who hope to use the criminal law as an instrument of public safety by setting a scientific standard for impairment measured by the body's drug levels must do so in a certain way to claim any validity. That way is through systematic study of single-car fatal crashes, particularly at night, and case-control studies comparing drug levels of fatal crash victims with those of a group of randomly selected drivers under similar circumstances. These studies are the "smoking guns" for linking levels of intoxication to dangerousness. Without them, the venerable .08 percent standard would be illegitimate under a jurisprudence of dangerousness.

A casual observer might be tempted to argue that even DUI alcohol laws have veered away from Haddon's framework, citing (1) the adoption of .08 percent even amid scientific controversy, coupled with recent calls from public

206. LERNER, *supra* note 3, at 133.

207. *Id.* at 133–34 (citing Robert Davis, *French Struggle: Drinking and Driving*, USA TODAY, Sept. 3, 1997, at 3A).

208. *Id.* at 134.

209. Rick Berman, *MADD Doesn't Differentiate Between Drunks and Social Drinkers*, WASH. TIMES, Sept. 26, 1997, at A18.

210. LERNER, *supra* note 3, at 145.

211. *Id.* at 146.

212. *Id.* at 145.

officials to lower the limit even further to .05 percent, and (2) zero-tolerance DUI laws for minors. Even these examples, however, fit within the framework.

1. *The Controversy Over .08 Percent and Calls to Lower the Limit to .05 Percent*

While .08 percent was controversial, as evidenced by high DUI acquittal rates that linger to this day,²¹³ its advocates purported to rely on the established framework in justifying the law under a science-based jurisprudence of dangerousness. Holcomb's 1938 paper had suggested that BACs higher than .06 percent increased crash risk and, while the paper was problematic, the Grand Rapids Study confirmed this result. As Borkenstein himself wrote in a follow-up paper in 1974, "[t]he probability of accident involvement increases rapidly at BACs over .08 percent."²¹⁴ And while the Grand Rapids Study was essentially the last case-control study of crash risk before the wave of .08 percent laws were passed in 2004, two subsequent, large-scale case-control studies by Blomberg et al. (2005) and Krüger and Vollrath (2005) have confirmed that significant relative risk increases begin at .08 percent.²¹⁵ These authors, like Borkenstein and Haddon, took care to explain the need for a true control group that mirrors the driver group in every way other than BAC, including season, direction of travel, day of week, time of day, and precise location.²¹⁶

A casual observer might also mistake ongoing governmental efforts to lower the legal limit to .05 percent BAC (based primarily on impairment studies rather than crash risk studies) as evidence that Haddon's framework is not the only way to justify impairment laws on dangerousness grounds. On the contrary, the lack of data showing a significant increase in relative crash risk at .05 percent is surely the reason that every recommendation for a .05 percent limit has been rejected out of hand.

213. See generally R.J. CINQUEGRANA, REPORT TO THE SUPREME JUDICIAL COURT 6–7, 55 (Oct. 2012) (observing that jury acquittal rates were nearly 60 percent, and judicial acquittal rates in some counties were over 80 percent); Rebecca Snyder Bromley, *Jury Leniency in Driving and Driving Cases: Has It Changed? 1958 versus 1993*, 20 LAW & PSYCHOL. REV. 27, at 42 tbl. 1 (1996) (finding jury acquittal rates in Colorado of 39 percent and judge acquittal rates of 27 percent).

214. Robert F. Borkenstein et al., *The Role of the Drinking Driver in Traffic Accidents (The Grand Rapids Study)*, 11 BLUTALKOHOL 1 (1974).

215. RICHARD D. BLOMBERG ET AL., CRASH RISK OF ALCOHOL INVOLVED DRIVING: A CASE-CONTROL STUDY 6–7, 28–38, 70 (Sept. 2005), available at <http://www.dunlapandassociatesinc.com/crashriskofalcoholinvolveddriving.pdf> (noting the lack of case-control studies since Grand Rapids, publishing a study based on 1997–99 crash data (2,871 crashes) and data from thousands of randomly stopped drivers at similar times, days, and locations in California and Florida, and concluding that drivers at .08 percent were 2.69 times more likely than sober drivers to be in an accident); H.P. Krüger & M. Vollrath, *The Alcohol-Related Accident Risk in Germany: Procedure, Methods, and Results*, 36 ACCIDENT ANALYSIS & PREVENTION 125, 131 (2004) (finding an OR of 2.8 for BACs from .05–.079 and an OR of 15.1 for BACs from .08–.159).

216. See BLOMBERG ET AL., *supra* note 215, at 7.

The first such unsuccessful attempt to lower the limit to .05 percent was in 1958. The Symposium on Alcohol and Road Traffic declared that year that a BAC of .05 percent “definitely impair[s] the driving ability of some individuals.”²¹⁷ The NSC and AMA soon endorsed the statement, and at least two state legislators introduced bills to lower the legal limit to .05 percent.²¹⁸ Yet these efforts went nowhere, perhaps because policymakers had not yet forgotten Emil Bogen’s lesson that impairment studies alone are an inaccurate indicator of relative crash risk.

The issue was briefly resurrected in the late 1980s, when the NSC and CAOD declared that driving impairment begins for “some individuals” below .05 percent, based on several impairment studies (but no crash studies).²¹⁹ Yet even MADD did not react to this new data by recommending that states adopt .05 percent as a new legal limit.²²⁰ And local attempts to set such a limit were unsuccessful; when Washington, D.C., authorized local police in 2005 to arrest anyone with a BAC over .03 percent, the city council soon rescinded the policy in response to protests from restaurant owners and the public.²²¹

The most recent call for a .05 percent limit, in a 2013 National Transportation Safety Board (NTSB) report, has also been met with hostility.²²² The report relied primarily on on-road and simulator studies showing that “several types of performance are affected by BAC levels as low as .01”²²³ and that “significant cognitive decrements” occur at .048 percent.²²⁴ The only crash study cited in the report that estimated increased crash risk specifically at .05 percent²²⁵ showed an increase in “relative risk” of 1.38,²²⁶ meaning that a driver at .05 percent was 1.38 times more likely to be in a crash than a sober driver.²²⁷

217. LERNER, *supra* note 3, at 52.

218. *Id.*

219. NAT’L SAFETY COUNCIL, *supra* note 148, app. at 112.

220. See Laura Dean-Mooney, Letter, *Don’t Be Mad at MADD*, WASH. TIMES, May 4, 2009, at A20.

221. LERNER, *supra* note 3, at 144.

222. Bart Jansen, *Make DUI Limit 0.05 Percent Blood-Alcohol Level, NTSB Says*, USA TODAY (May 14, 2013, 1:48 PM), available at <http://www.usatoday.com/story/news/nation/2013/05/14/national-transportation-safety-board-drunken-driving/2158107/>.

223. NAT’L TRANSP. SAFETY BOARD, NTSB/SR-13/01, REACHING ZERO: ACTIONS TO ELIMINATE ALCOHOL-IMPAIRED DRIVING 18 (2013).

224. *Id.*

225. However, the report also cited the Grand Rapids Study as showing an unmeasured “increased risk of crashes beginning at a BAC of 0.04,” and a study showing that BACs between .05 and .079 correlated with a three to seventeen times greater risk of crash. *Id.* at 20 (citing Paul L. Zador et al., *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*, 61 J. STUD. ALCOHOL 387–95 (2000)).

226. *Id.* at 20–21 (citing R.P. Compton et al., *Crash Risk of Alcohol-Impaired Driving*, 16 INT’L CONF. ON ALCOHOL, DRUGS & TRAFFIC SAFETY PROC. (2002)).

227. See, e.g., *What Researchers Mean by . . . Absolute and Relative Risk*, INST. FOR WORK & HEALTH, <http://www.iwh.on.ca/wrmb/absolute-and-relative-risk> (last visited Apr. 6, 2015). “Relative risk” of crash—the ratio of the risk of crash at a certain BAC or THC level compared to a control group of sober drivers under similar conditions—is different from “absolute risk,” the probability of

While the report labels this number a “significant[] increase[],”²²⁸ policymakers, industry, and the public appear to disagree. The Governors Highway Safety Association formally opposed the recommendation,²²⁹ and a USA Today editorial expressed concern that “lowering the legal limit would turn a lot of responsible social drinkers into criminals.”²³⁰ The ABI predictably derided the recommendation as “ludicrous.”²³¹

Any argument that a .05 percent criminal DUI law would be “ludicrous” rests on two hidden premises. First, that the law would be part of a jurisprudence of dangerousness rather than prohibition. If the penal purpose of a .05 percent law were to enforce a prohibition on alcohol, such as Saudi Arabia’s zero-tolerance DUI alcohol law,²³² then the failure to link .05 percent with significantly increased crash risk would not be an issue, and the law would be legitimately related to its penal purpose. Thus, enforcing a .05 percent limit for underage drinkers would presumably meet with little resistance. The second hidden premise suggests that under a jurisprudence of dangerousness, the relative crash risk associated with .05 percent is not significant enough to merit criminal punishment. It is true that a variety of other driving behaviors also modestly increase the relative risk of crash, such as driving with a cell phone or hands free device, which by one study is the equivalent of driving at .08 percent in terms of relative increased crash risk;²³³ driving 80 miles per hour in a 65 mile per hour zone;²³⁴ driving with a hangover;²³⁵ and driving on little sleep.²³⁶ While one might argue that driving under these conditions also merits

being involved in a crash at a certain BAC or THC level. *See id.* A driver at a .15 percent BAC might have a high “relative risk” of crash compared to a sober driver—say, a relative risk of 10, or ten times more likely than a sober driver to crash — but might still have only a modest “absolute risk” of crashing—say, 10 percent—based on that level of impairment.

228. NAT’L TRANSP. SAFETY BOARD, *supra* note 223, at 21.

229. Jansen, *supra* note 222.

230. Editorial, *Don’t Lower Blood Alcohol Content to .05%: Our View*, USA TODAY (May 30, 2013, 8:36 PM), available at <http://www.usatoday.com/story/opinion/2013/05/30/drunken-driving-blood-alcohol-content-editorials-debates/2374199/>.

231. Jansen, *supra* note 222.

232. *See (c) World Health Organization*, WHO [\(http://apps.who.int/gho/athena/data/GHO/SA_0000001520.html?profile=ztable&filter=COUNTRY:*;BACGROUP:*\)](http://apps.who.int/gho/athena/data/GHO/SA_0000001520.html?profile=ztable&filter=COUNTRY:*;BACGROUP:*(last visited Apr. 6, 2015)) (last visited Apr. 6, 2015) (World Health Organization database of all countries’ BAC limits; over 100 are .05 percent or below).

233. A 1997 study found that drivers using cell phones, even if using a hands-free device, were four times more likely to crash than other drivers. Donald A. Redelmeier & Robert J. Tibshirani, *Association Between Cellular-Telephone Calls and Motor Vehicle Collisions*, 336 NEW ENG. J. MED. 453, 456 (1997).

234. *See, e.g.,* Letty Aarts & Ingrid van Schagen, *Driving Speed and the Risk of Road Crashes: A Review*, 38 ACCIDENT ANALYSIS & PREVENTION 215, 220–22 (2006).

235. “*If You Drink, Don’t Drive*” *Motto Now Applies to Hangovers As Well*, 250 JAMA 1657, 1657 (1983) (describing a study indicating that a hangover may “diminish driving ability by as much as 20 percent”).

236. *See, e.g.,* James M. Lyznicki et al., *Sleepiness, Driving, and Motor Vehicle Crashes*, 279 JAMA 1908, 1909 (1998) (citing studies claiming that sleepiness is the “principal causative factor in about 100,000 police-reported crashes each year in the United States”).

criminal punishment, the American justice system treats them as administrative offenses at worst.

While a few other countries claim to have a dangerousness-based .05 percent limit, such countries appear to be operating under inaccurate assumptions about crash risk or have a different moral conception of the level of risk deserving punishment. For example, Norway's conspicuous embrace of a .05 percent limit as early as 1936 rested on a "loose foundation," in the words of DUI research pioneer Johannes Andenaes.²³⁷ In the debate preceding the law's adoption, members of Parliament made sweeping claims that a "deplorably large part" of car accidents were "caused by the driver being under the influence" or occurred "because of drunkenness."²³⁸ As Andenaes notes, "it seems that none of the speakers had any systematic material on which to base their statements."²³⁹ Nonetheless, the law remains, substantially unchanged, eighty years later.

Given this state of affairs, one might reasonably speculate that the true motivation for the recent push for a .05 percent limit is not so much a concern with the marginal increase in relative crash risk at .05 percent, but rather to ride the penal momentum that led to the changes from .15 percent to .10 percent to .08 percent, in an effort to secure more convictions through ever more conviction-friendly bounds of criminality.

2. *Zero-Tolerance Laws for Minors*

A casual observer might also invoke zero-tolerance DUI laws for minors—those under the drinking age of twenty-one—as evidence that Haddon's framework is not the only way to justify a dangerousness-based impairment law. On the contrary, the legitimacy of Haddon's framework helps to explain the awkward evolution of, and uneasy public reaction to, such laws. To be sure, crash data at least modestly supports a zero-tolerance regime; minors driving at non-zero (even if low) BACs do have a moderately increased relative crash risk.²⁴⁰ Perhaps Haddon would agree that the regulatory state, if not the criminal law, should prohibit minors from drinking and driving to any extent. But the fact that the increase is not significant is surely relevant to the controversy surrounding zero-tolerance criminal laws and their minimal enforcement by police.²⁴¹ In the end, one might call these laws *sui generis*, reflecting both a partial jurisprudence of prohibition and an intuitive

237. Johannes Andenaes, *The Effects of Scandinavia's Drinking-and-Driving Laws*, 6 SCANDINAVIAN STUD. CRIMINOLOGY 35, 48 (1978).

238. *Id.* at 47–48 (emphasis added).

239. *Id.* at 48.

240. See discussion *infra* at nn.244–46, 250, 254–56.

241. See, e.g., Susan A. Ferguson, *Comment on "Low BAC Policies: Results and Mechanisms,"* TRANSP. RES., Sept. 2001, available at <http://www.iihs.org/frontend/iihs/documents/masterfiledocs.ashx?id=1714>.

assumption—modestly supported by crash data—that the unique combination of minority status and non-zero (even if low) BAC is presumptively dangerous.

The short history of these laws—less than forty years—makes clear the unique and precarious justification underlying their adoption. Before the 1970s, most states had a drinking age of twenty-one and no special DUI laws for minors.²⁴² Between 1970 and 1976, twenty-nine states lowered their drinking age to eighteen, presumably in response to the Twenty-Sixth Amendment lowering voting age to eighteen.²⁴³ Upon seeing a subsequent increase in alcohol-related fatal crashes in eighteen to twenty-one-year olds, some states in the 1980s reversed course and reimposed a drinking age of twenty-one.²⁴⁴ As a result of this mish-mash of state drinking-age laws, young people between ages eighteen and twenty-one in states with an age of majority of twenty-one had an incentive to drive to neighboring states with a drinking age of eighteen to legally purchase alcohol, often leading them to drive home intoxicated.

While the research on youth and relative crash risk was nascent in the early 1980s, researchers were already aware that drinking drivers under the age of twenty-one were overrepresented among fatal car crash victims, even though the incidence of drinking among randomly stopped young drivers was lower than among adult drivers.²⁴⁵ The 1964 Grand Rapids Study had found no predictable relationship between increased crash risk and age groups at varying BACs, but it did find that the youngest and oldest drivers were the only groups with an increased relative crash risk at BACs lower than .05 percent.²⁴⁶ In addition, a study in 1983 comparing randomly stopped nighttime drivers with fatally injured nighttime drivers showed that minors' higher relative crash risk occurred at all BAC levels.²⁴⁷ One clear reason for this disparity, independent of minors' proclivity for drinking and driving, was simply their relative lack of driving skill and their tendency to drive during particularly hazardous times of day.²⁴⁸

242. Many states have had a minimum drinking age of twenty-one since the repeal of Prohibition. See Robert B. Voas et al., *Assessing the Effectiveness of Minimum Legal Drinking Age and Zero Tolerance Laws in the United States*, 35 ACCIDENT ANALYSIS & PREVENTION 579, 580 (2003).

243. JACOBS, *supra* note 5, at 174.

244. *Id.*

245. See, e.g., *id.* at 47–48 (citing ALEXANDER C. WAGENAAR, ALCOHOL, YOUNG DRIVERS, AND TRAFFIC ACCIDENTS (1983); R. FARRIS ET AL., DOT-HS-801-826, A COMPARISON OF ALCOHOL INVOLVEMENT IN EXPOSED AND INJURED DRIVERS: PHASES I AND II (1976); M.W. Perrine, *The Vermont Driver Profile: A Psychometric Approach to Early Identification of Potential High-Risk Drinking Drivers*, in ALCOHOL, DRUGS, & TRAFFIC SAFETY (S. Israelstam & S. Lambert eds., 1975)).

246. See Borkenstein et al., *supra* note 214, at 143–54.

247. D.R. Mayhew & H.M. Simpson, *Alcohol, Age, and Risk of Road Accident Involvement*, 9 INT'L CONF. ON ALCOHOL, DRUGS & TRAFFIC SAFETY PROC. 937 (1983).

248. See JACOBS, *supra* note 5, at 177 (citing Wagenaar (1983) and Zylman (1973)); see also Tracy L. Cameron, *Drinking and Driving Among American Youth: Beliefs and Behaviors*, 10 DRUG & ALCOHOL DEPENDENCE 1 (1982) (noting that young drivers were more likely than adults to drive at night).

The increasing number of crash fatalities among young people, whatever its cause, put the problem of underage drinking and driving on the map and justified the federalization of alcohol laws to remove the incentive for minors to drive across state lines to buy alcohol. Congress enacted the National Minimum Drinking Age Amendment in 1984 to pressure states, through the withholding of highway funds, to raise their drinking age to at least twenty-one.²⁴⁹ Ultimately, then, raising the drinking age nationwide to twenty-one was based not on a moral judgment against those under twenty-one ingesting alcohol, but on the safety issues caused by lack of uniformity among states. Still, the subsequent wave of zero-tolerance DUI laws for those under twenty-one was at least partially prohibitionist, intended to deter alcohol use by those just beginning to learn to drive.²⁵⁰

At the same time, zero-tolerance laws were also modestly supported by crash studies showing that minors had higher relative crash risks at low BACs than adults.²⁵¹ But the increased risk even for minors at low BACs was not particularly significant, and the push for zero-tolerance laws may not have succeeded without MADD adding them to its agenda by the early 1990s and deploying dramatic anecdotes of underage crash victims.²⁵² Between 1983 and 1990, whether as a result of minimum drinking age, crash risk studies, MADD's influence, or the convergence of these factors, ten states passed new laws setting special low or zero BAC limits for minors.²⁵³

Beginning in the 1990s, a new wave of research on age, BAC, and fatal crash risk emerged. This research relied on the Insurance Institute for Highway Safety's (IIHS) 1986 nationwide roadside breath-testing survey, the first of its kind since 1973.²⁵⁴ One 1991 study compared the 1986 survey data with federal Fatality Analysis Reporting System (FARS) data from single-car crashes in 1985 and 1986 and found that male drivers under age twenty-one had relative crash rates at least three times those of adult male drivers for every

249. See National Minimum Drinking Age Amendment, Pub. Law 98-363, 98 Stat. 435 (codified as amended at 23 U.S.C. § 158 (2012)).

250. Officials were keenly aware of the advantages of "phasing" the legal driving and drinking ages so they would not coincide, to ensure that minors did not learn to drink and drive at the same time. As Frank Zimring has noted, drinking and driving are two activities that probably should not begin at the same time in a young person's life. FRANK ZIMRING, *THE CHANGING LEGAL WORLD OF ADOLESCENCE* 108-10 (1982).

251. A 1986 study compared fatal crash data with roadside surveys and confirmed that minors' crash risk rose at a higher rate beginning at lower BACs. See D.R. Mayhew et al., *Youth, Alcohol and Relative Risk of Crash Involvement*, 18 ACCIDENT ANALYSIS & PREVENTION 273, 279-81 (1986).

252. See LERNER, *supra* note 3, at 123-24.

253. See Ralph Hingson et al., *Lower Legal Blood Alcohol Limits for Young Drivers*, 109 PUB. HEALTH REP. 738, 739 (1994) (noting two states with BAC limit at .05 percent, one state with .04 percent, three states with .02 percent, and four states with zero).

254. The NHTSA conducted the first such nationwide roadside survey in 1973. See NHTSA, *PILOT TEST OF NEW ROADSIDE SURVEY METHODOLOGY FOR IMPAIRED DRIVING 6* (2007), available at <http://www.nhtsa.gov/people/injury/research/pub/HS810704/index.html>. The IIHS conducted the second in 1986. *Id.* The NHTSA and IIHS jointly funded the third in 1996. *Id.*

BAC level.²⁵⁵ Males aged sixteen to twenty were 5.8 times more likely to crash than male adult drivers at non-zero BACs under .02 percent; 5.5 times more likely at BACs between .02 and .04 percent, and 12.3 times more likely at BACs between .05 and .09 percent.²⁵⁶ The studies also further cemented the link between age and significant increases in crash risk overall. A 1994 study examining FARS data from the early 1990s showed that, while 17 percent of all fatal single-vehicle nighttime crashes “involved” alcohol, the percentage rose to 52 percent when limited to drivers aged fifteen to twenty.²⁵⁷

Influenced by this wave of research, and building on the momentum begun by MADD, numerous other states in the early 1990s passed zero-tolerance laws. By 1994, a majority of states had enacted them.²⁵⁸ By 2004, all fifty states and the District of Columbia had done so, abandoning former policies that were indifferent to age as well as those that responded moderately to the indication of higher crash risk with lower BAC limits for minors.²⁵⁹

Recent crash risk studies that question the significance of minors’ increased crash risk at low BACs have rekindled the debate over zero-tolerance laws’ legitimacy and effectiveness.²⁶⁰ While NHTSA materials frequently claim that over 30 percent of youth fatalities in crashes are “related” to or “involve” alcohol,²⁶¹ this statistic does not show that drivers under twenty-one with low BACs are more likely to crash than a sober driver or an adult driver at the same BAC. Indeed, of drivers aged fifteen to twenty years old killed in crashes in 2007, only 5 percent had a BAC under .08 percent, which may well be less than or equal to the percentage of randomly selected young drivers at that BAC range.²⁶²

255. Zador, *supra* note 134.

256. *Id.* at 306.

257. Hingson et al., *supra* note 253, at 741–43.

258. *See id.* at 739 (listing numerous states and passage dates).

259. *See Youth Impaired Driving Manual for Sheriffs: Background*, NHTSA, <http://www.nhtsa.gov/people/injury/alcohol/Sherrifs%20Web/background.html> (last visited Apr. 8, 2015).

260. A comprehensive 2005 NHTSA study found no difference in crash risk between adults and minors at BACs less than .12 percent. *See, e.g.,* BLOMBERG ET AL., *supra* note 215, at 79. A subset of the same researchers in 2007 reran this data and concluded that there was, indeed, a statistically significant difference in crash risk between adult and minor drivers at BACs lower than .08 percent. *See* R.C. Peck et al., *Improved Methods for Estimating Relative Crash Risk in a Case-Control Study of Blood Alcohol Levels*, 18 INT’L CONF. ON ALCOHOL, DRUGS, & TRAFFIC SAFETY, available at <http://icadts2007.org/print/101relcrashrisk.pdf>.

261. *See, e.g.,* NHTSA, *Alcohol-Related Fatal Crash Rates for Youth Reach Historic Low: Strategies for Combating Juvenile DUI*, 35 ANNALS EMERGENCY MED. 192, 192 (2000).

262. *See* NHTSA, DOT HS 811 218, *Fatal Crashes Involving Young Drivers*, TRAFFIC SAFETY FACTS RES. NOTE 4 (2009), available at <http://www-nrd.nhtsa.dot.gov/Pubs/811218.pdf> (finding that in 2007, 31 percent of drivers aged fifteen to twenty that were killed in car crashes had BACs of .01 percent or greater and 26 percent had BACs of .08 percent or greater). It is, for the record, no greater than the percentage of the overall driving population for that year—5 percent—in that BAC range. *Id.* Even the fact that such laws have arguably reduced youth driving fatalities does not show that minors are highly dangerous drivers—worthy of criminal punishment—at non-zero but very low BACs. Reductions in fatalities following passage of zero-tolerance laws might show that such laws effectively

Now, zero-tolerance laws are not widely enforced,²⁶³ perhaps because of a realization that a twenty-year-old driving at a .01 percent BAC, while breaking the underage drinking law, does not deserve a stigmatizing DUI conviction. Moreover, the data on the extent to which such laws reduce fatal crashes is inconsistent.²⁶⁴ On one theory, zero-tolerance laws might even be counterproductive; although they create a strong disincentive to take a first drink, they might actually negate any incentive to stop drinking before one's BAC rises even further.²⁶⁵ At the very least, such laws clearly represent the high-water mark with respect to the level of chemical that impairment communities are willing to call "criminal."²⁶⁶

The lesson from the DUI alcohol story is not that a DUI jurisprudence of prohibition is impossible or inherently illegitimate, nor that lawmakers cannot criminalize, through a jurisprudence of dangerousness, behavior that causes only marginal increases in crash risk. The lesson is that if a chemical impairment law's legitimacy is based on a jurisprudence of dangerousness, then the law has to live up to that penal purpose. To do so, the law must satisfy the established Haddon-era framework—still the only valid framework the law has—for scientifically proving the link between a certain measurable level of the drug's concentration in the body and dangerousness. Once science reveals this relationship, the community is still faced with the secondary decision of what level of risk should be criminal. Sometimes politics influences the decision to criminalize a certain level of risk in a way that renders bad policy,

deter drinking and driving at dangerously high BACs, but they do not suggest that low BACs cause crashes. Surely, for example, a zero-tolerance law for adults would also decrease fatalities, but would not prove that a .01 percent BAC in an adult is worthy of criminal punishment.

263. See, e.g., Ferguson, *supra* note 241, at 141.

264. Compare NHTSA, ZERO TOLERANCE FOR YOUTH: FOUR STATES' EXPERIENCE WITH ZERO TOLERANCE LAWS (2000), available at <http://www.nhtsa.gov/About+NHTSA/Traffic+Techs/current/Zero+Tolerance+For+Youth+Four+States%27+Experience+With+Zero+Tolerance+Laws> (finding reductions in single vehicle nighttime injury crashes involving drivers under twenty-one as a result of zero tolerance laws), and Voas et al., *supra* note 242, at 585 (finding that zero-tolerance laws, in conjunction with minimum drinking age, reduced alcohol-positive involvement in fatal crashes), with Darren Grant, *Dead on Arrival: Zero Tolerance Laws Don't Work*, 48 ECON. INQUIRY 756 (2010) (using federal FARS data from 1988–2000 and finding no material effect of such laws on fatality rates or distribution of BACs among drivers in fatal accidents).

265. Grant, *supra* note 264, at 769.

266. Zero-tolerance or low-BAC laws for truck drivers and operators of special equipment, see, e.g., CAL. VEH. CODE § 23152(d) (2014) ("It is unlawful for a person who has 0.04 percent or more, by weight, of alcohol in his or her blood to drive a commercial motor vehicle . . ."), similarly target dangerous driving through what is, in essence, a presumption of an unacceptable magnitude risk. See, e.g., FED. AVIATION ADMIN., U.S. DEP'T OF TRANSP., DRUGS AND ALCOHOL IN CIVIL AVIATION ACCIDENT PILOT FATALITIES FROM 2004–2008, at 6 (2011), available at http://www.faa.gov/data_research/research/med_humanfacs/oamtechreports/2010s/media/201113.pdf (noting the FAA BAC limit of 0.04).

as was arguably the case with zero-tolerance laws and the push for a .05 percent limit.

In the context of per se DUI marijuana laws, as the next Part explores, lawmakers have passed statutes that are the product of political compromise with no clear penal purpose. Although ostensibly modeled after DUI alcohol's jurisprudence of dangerousness, these per se regimes cannot pass muster under Haddon's framework and are therefore illegitimate.

II.

PUNISHMENT WITHOUT PURPOSE: THE ILLEGITIMACY OF CRIMINAL PER SE DUI MARIJUANA LAWS

A. The Rise of Drugged Driving Laws and Recognition of the Arbitrariness of Per Se Limits

1. The Origins of Drugged Driving Laws

As with DUI alcohol laws, DUI drug (DUID) laws had their origins in a jurisprudence of dangerousness. Partly influenced by a burgeoning federal regime of drug taxation in the years before and during Prohibition,²⁶⁷ many of the first states to criminalize DUI explicitly included driving under the influence of drugs and narcotics along with alcohol.²⁶⁸ Laws against DUI marijuana were not prohibitionist; most states did not criminalize the drug until the 1930s, largely in response to the 1932 Uniform State Narcotic Act and the urging of the new Federal Bureau of Narcotics director, H.J. Anslinger.²⁶⁹

By 1937, every state had a law prohibiting the sale and possession of the drug,²⁷⁰ but federal officials' attempt to create a marijuana "crisis" in the eyes of the public was "largely unsuccessful."²⁷¹ Although every state had a drugged driving law by the 1960s,²⁷² there was no large political movement pushing to institute highly punitive drug laws or zero-tolerance drugged driving laws until decades later. This was true even though prosecution of drugged driving was

267. See generally John F. Galliher & Allyn Walker, *The Puzzle of the Social Origins of the Marijuana Tax Act of 1937*, 24 SOC. PROBS. 367, 367–69 (1977) (chronicling federal taxation on drugs leading up to 1937).

268. See, e.g., *Black v. State*, 130 S.E. 591, 592 (Ga. Ct. App. 1925) ("[I]t is . . . an indictable offense for any person, 'while under the influence of intoxicating liquors or drugs,' to operate any motor vehicle upon any highway of this state, regardless of the rate of speed." (quoting The Act of 1921, 1921 Ga. Laws 255)).

269. See Galliher & Walker, *supra* note 267, at 367–69 (noting that states had criminalized marijuana years before the Tax Act).

270. See *id.* at 367.

271. See *id.* at 371 (noting the lack of success of the Federal Bureau of Narcotic's educational campaign aimed to convince the public of the evils of marijuana).

272. New York was one of the last states to enact a drugged driving statute in 1966. See *People v. Litto*, 822 N.Y.S.2d 130, 130–31 (App. Div. 2006) ("[I]n 1966, the [New York] Legislature added Vehicle and Traffic Law § 1192(4), making it a misdemeanor to operate a motor vehicle while impaired by the use of a drug." (internal quotation marks omitted)).

from the start just as, if not more, difficult to prosecute than drunk driving. The government typically proved DUI drug charges, like DUI alcohol charges, through police testimony that the driver was acting impaired.²⁷³ Although reliable acquittal rate data from the early days of drugged driving prosecutions appears elusive, the problems with proving DUI alcohol charges to juries were surely only magnified in drugged driving cases; unlike drivers severely impaired by alcohol, who nearly always showed outward signs of drunkenness, drivers experiencing even acute drug intoxication sometimes showed more “subtle symptoms.”²⁷⁴

The solution to this dilemma in the DUI alcohol context had come from the invention of breath testing devices, which led both to the use of BAC results to corroborate police testimony and to the ability to set presumptive (and later per se) BAC thresholds based on dosage-specific crash risk data. The law enforcement and scientific communities appeared to understand from the beginning, however, that the drugged driving problem could not be addressed in the way that drunk driving had been. Noted toxicologist Bryan Finkle worried at a 1974 conference that the DUI alcohol model would simply be adopted wholesale by lawmakers in the drugged driving context without sufficient thought to the complexities of the latter:

Surely the jig is up for the Drinking Driver. For the past forty years he has . . . had his behavior, his body fluids and his very breath analyzed by the most pedantic techniques of science, until . . . [r]esearchers . . . made “Drunk Driver” and “Problem Driver” household clichés. But what of the “Drugged Driver?”

Is there a danger that, by logical progression and without relevant knowledge, a specter is being created and legislated for the Drugged Driver? Unidentified, and cloaked in a veil of fear by a generation familiar with drug-abuse, it is natural to assume that drugs play a role in the current highway carnage. Millions of law-abiding citizens drive under the influence of drugs every day; but whether this is a significant factor in the total picture of traffic safety and requires priority attention is an important but unresolved question.²⁷⁵

Finkle’s concern was premature. William Haddon Jr. had apparently cast a long shadow; respect for the undisputable logic of his epidemiological approach remained even after the punitive turn of criminal law in the 1980s. As

273. See NHTSA, *DRUG PER SE LAWS: A REVIEW OF THEIR USE IN STATES 1* (2010), available at http://www.nhtsa.gov/staticfiles/nti/impaird_driving/pdf/811317.pdf [hereinafter NHTSA Report (2010)].

274. Barry Meier, *New Test for a Highway Peril, the Drugged Driver*, N.Y. TIMES, Sept. 25, 1989, at B7.

275. Bryan S. Finkle, “Will the Real Drugged Driver Please Stand Up?” *An Analytical Toxicology Assessment of Drugs and Driving*, 6 INT’L CONF. ON ALCOHOL, DRUGS, & TRAFFIC SAFETY PROC. 607 (1974).

the AMA acknowledged in 1985, “relatively little would be gained by a per se approach [to drugged driving laws] based on arbitrary data.”²⁷⁶

2. *Marijuana’s Profound Differences from Alcohol in Terms of Ability to Identify Proximity of Use, Psychoactive Influence, and Driving Impairment from Specific Blood Levels*

To understand why the AMA would so dramatically declare that research on drugged driving crash risk was no more than “arbitrary data,” an exploration of the stark differences between alcohol—an “unusually simple drug”²⁷⁷—and other drugs is in order. I focus here on marijuana because the partial or full legalization of marijuana in several states has, as explained in Part II.D, recently motivated state officials to pass per se marijuana laws. Three primary factors make the study of marijuana’s relationship to crash risk more problematic than the study of alcohol and crash risk: the difficulty in identifying proximity of use, the wide variance in dose-related psychoactive influence, and the limited ability to study the effect of specific THC blood levels on driving.

Proximity of use. One unique property of alcohol, not shared by marijuana, is the manner in which its concentration in the blood so uniformly and predictably reflects proximity of use. Ethyl alcohol is the only psychoactive compound in the alcohol we drink.²⁷⁸ Ethyl alcohol is both fat soluble and entirely water soluble, creates no long-lived metabolites, and quickly dissipates from the body.²⁷⁹ One’s BAC has a predictable relationship with rates of absorption, distribution, and elimination that are similar among humans.²⁸⁰ Because of these qualities, breath test results can indicate with relatively high precision how long ago a person drank alcohol and in what quantity.²⁸¹

The problem with marijuana’s detectability is, one might say, that it is an embarrassment of riches; as explained below, it can be detected quickly, effectively, and for a long time. These characteristics confound the ability to infer recency or extent of use from blood levels, which in turn makes the inference between consumption and dangerousness difficult to draw. Cannabis, the plant with marijuana as a derivative,²⁸² has over 421 chemicals, and when one smokes it, the human body breaks down more than two thousand

276. *Drug Concentrations and Driving Impairment*, 254 JAMA 2618, 2620 (1985).

277. Reisfield et al., *supra* note 44, at 353.

278. See FAIGMAN et al., *supra* note 40, § 41:4, 20, 58.

279. See *id.* § 41:4, 40, 60.

280. See *id.* § 41:32, 40, 43, 62.

281. See *id.* § 41:40, 43.

282. Marijuana is a derivative of the plant *Cannabis sativa*. See Emma Puighermanal et al., *Cellular and Intracellular Mechanisms Involved in the Cognitive Impairment of Cannabinoids*, 367 PHIL. TRANSACTIONS ROYAL SOC. B: BIOLOGICAL SCI. 3254, 3254 (2012).

compounds.²⁸³ The most psychoactive of these compounds is delta9-tetrahydrocannabinol (THC), on which most studies of the effects of marijuana impairment focus.²⁸⁴ THC from smoked cannabis is “detectable in plasma within seconds after the first puff,” with peak plasma concentration generally happening within three to ten minutes.²⁸⁵ But THC also produces two additional compounds when it is metabolized in the liver: a psychoactive compound, 11-OH-THC, and a non-impairing, inactive compound, THC-COOH.²⁸⁶

After cannabis consumption, THC and its metabolites may remain in the body for a time period that varies among humans. While THC and its metabolites reach their *peak* concentrations within three to ninety minutes, and the metabolites are excreted from the body through feces and urine,²⁸⁷ the compounds linger in low levels in the body for much longer. Even THC itself can be found for several hours or even days after consumption, depending on the frequency and amount of usage; a 2014 study from Norway concluded that THC can remain in the saliva of frequent cannabis users for over eight days.²⁸⁸ THC’s metabolites linger even longer, and can be found in one’s blood for days, weeks, or even over a month in chronic users.²⁸⁹ For example, the level and timing of concentrations change not only with tolerance, but with ingestion method. For those who orally ingest marijuana by swallowing an extract or applying a tincture to the bottom of the tongue (practices used by most medical

283. Priyamvada Sharma et al., *Chemistry, Metabolism, and Toxicology of Cannabis: Clinical Implications*, 7 IRAN J. PSYCHIATRY 149, 149 (2012).

284. *Id.* at 150.

285. *Id.* at 151. When one smokes marijuana, THC itself quickly reaches a high concentration in the blood, where approximately 90 percent of the THC circulates in plasma and the rest in red blood cells, which together comprise “whole blood.” FRANJO GROTENHERMEN ET AL., DEVELOPING SCIENCE-BASED PER SE LIMITS FOR DRIVING UNDER THE INFLUENCE OF CANNABIS (DUIC) 6 (2005), available at <http://www.canorml.org/healthfacts/DUICreport.2005.pdf>.

286. *Id.*

287. 11-OH-THC often reaches its peak concentration a few minutes after THC’s peak. *See, e.g.*, Louis Lemberger et al., *Comparative Pharmacology of Delta9-Tetrahydrocannabinol and Its Metabolite, 11-OH-Delta9-Tetrahydrocannabinol*, 52 J. CLINICAL INVESTIGATION 2411, 2416 (1973) (finding that participants reached a peak “psychologic high and symptom sign score” within two to three minutes after intravenous administration of 11-OH-THC, but around fifteen to thirty minutes after intravenous injection of THC). *Id.* at 2412 tbl. 1. The inactive compound THC-COOH reaches its peak much later; in one study, the average among users was eighty-one minutes after ingestion. Sharma et al., *supra* note 283, at 152.

288. H.T. Andås et al., *Detection Time for THC in Oral Fluid After Frequent Cannabis Smoking*, 36 THERAPEUTIC DRUG MONITORING 808, 809 (2014), available at <http://www.ncbi.nlm.nih.gov/pubmed/24819969>.

289. *See, e.g.*, *How long does cannabis stay in the body after smoking*, NAT’L HEALTH SERV., <http://www.nhs.uk/chq/Pages/2287.aspx?CategoryID=53> (last visited Dec. 30, 2013); Sharma et al., *supra* note 283, at 152 (noting that the half-life of THC for infrequent users is 1.3 days and for frequent users is five to thirteen days); Amy Berning & Dereece D. Smither, NHTSA, DOT HS 812 072, *Understanding the Limitations of Drug Test Information, Reporting, and Testing Practices in Fatal Crashes*, TRAFFIC SAFETY FACTS RES. NOTE 1 (2014), available at <http://www-nrd.nhtsa.dot.gov/Pubs/812072.pdf> (noting that “traces of cannabinoids can be detected in blood samples weeks after use”).

marijuana patients), the efficiency of THC's effect decreases compared to smoking.²⁹⁰ Because THC (unlike alcohol) is "barely soluble in water, the body absorbs only a small fraction of the available [THC] when it is swallowed," causing THC blood levels and impairment effect to be delayed.²⁹¹ In sum, marijuana's unpredictable qualities render nearly impossible any determination from THC blood levels alone whether a person is actually under the influence of marijuana at the time of driving or has simply ingested marijuana in the somewhat recent past.

Dose-related psychoactive influence. A second distinction between the measurability of impairment from alcohol and from marijuana is that the properties of alcohol render its dose-related psychoactive effects predictable and uniform among humans. Ethyl alcohol's fat and water solubility, as well as its effectiveness at only high concentrations, cause it to affect the entire body and to "equilibrate[] readily" between levels in the brain, blood, and other tissue.²⁹² Its water solubility and volatility at body temperature also render BAC and BrAC levels constant and proportional.²⁹³ These properties together make BAC highly correlated to the level of alcohol intoxication in the brain, and therefore to alcohol's psychoactive effects.²⁹⁴

THC's psychoactive effect, in contrast, is much more complex and more disconnected to its levels in bodily fluids. When a person smokes marijuana, highly fat soluble THC gets rapidly absorbed through the lungs and distributed quickly into lung tissue, fat tissue, the liver, and the spleen.²⁹⁵ But because it is "barely soluble in water," it does not—like alcohol—reach a uniform concentration throughout bodily tissues at a rate similar among humans.²⁹⁶ Thus, unlike brain alcohol intoxication and BAC, the level of THC in the brain is not predictable from the level of THC in the blood. Further, while THC's active metabolite, 11-OH-THC, also has some psychoactive effect, though much smaller than THC (though it might be responsible for users feeling the "munchies"), it is just as unconnected from levels in bodily fluids.²⁹⁷ THC's inactive compound, THC-COOH, has no psychoactive effect, regardless of its level in bodily fluids.²⁹⁸

290. ALISON MACK & JANET JOY, MARIJUANA AS MEDICINE? THE SCIENCE BEYOND THE CONTROVERSY 11–12 (2001).

291. *Id.*

292. *See Drug Concentrations and Driving Impairment*, 254 JAMA 2618, 2619 (1985).

293. *See supra* text accompanying note 47.

294. *See Drug Concentrations and Driving Impairment, supra* note 292, at 2619.

295. Sharma et al., *supra* note 283, at 151.

296. MACK & JOY, *supra* note 290, at 12.

297. *See, e.g.,* Louis Lemberger et al., *Comparative Pharmacology of Delta9-Tetrahydrocannabinol and Its Metabolite, 11-OH-Delta9-Tetrahydrocannabinol*, 52 J. CLINICAL INVESTIGATION 2411 (1973).

298. *See, e.g., State ex rel. Montgomery v. Harris*, 322 P.3d 160, 160 (Ariz. 2014) (explaining that "Carboxy-THC," or THC-COOH, is a "non-impairing metabolite of Cannabis").

Moreover, while the pharmacodynamics of alcohol are decently well understood, scientists are not in agreement on exactly how and why THC affects the brain.²⁹⁹ We do know that THC interacts with specific cannabinoid receptors in the brain that are in regions associated with “cognition, memory, reward, anxiety, pain sensory perception, motor co-ordination and endocrine function.”³⁰⁰ We also know that THC can affect a range of other targets in diffuse and unpredictable ways, including opioid receptors, enzyme systems, hormones, and neurotransmitters.³⁰¹ But unlike with ethyl alcohol, the extent of THC’s effects is a function of several variables that interact in complex ways, including dosage and tolerance. And with respect to dosage, because of THC’s pharmacokinetic qualities, there is simply no predictable or uniform connection between specific blood levels of THC and its metabolites and these psychoactive effects.³⁰² Thus, even if the first factor were resolved, and scientists could straightforwardly infer proximity of use from THC blood levels, marijuana’s unpredictable properties render nearly impossible any inference about the likely psychoactive effect on the brain of a specific THC blood level.

Impairment of ability to drive safely. Because BAC, unlike THC blood level, corresponds so predictably to proximity of use, brain concentration, and level and type of psychoactive effect, BAC’s effects on driving can be well studied and generalized to the adult population. Specifically, alcohol’s qualities, along with the non-invasiveness of breath testing and legality of its use, allow for study of its impairing effects on driving through both controlled simulations at precise BAC levels and study of dosage-specific fatal crash statistics. At the 1983 National Institute on Drug Abuse conference addressing the potential aptness of the DUI alcohol model for studying DUI drugs, the somewhat pessimistic consensus was that ethanol is an “unusually good model for studying the effects of a drug on driving performance.”³⁰³ More recently, the NHTSA in a 2010 report on drugged driving acknowledged that it was only from “the epidemiological evidence of the increased risk of crash involvement associated with increased driver BACs” that “the rationale was developed for setting presumptive and then per se levels defining the alcohol-impaired driving offense.”³⁰⁴ And in a November 2014 publication, two NHTSA researchers acknowledged that “[c]urrent knowledge about the effects of drugs other than alcohol on driving performance is insufficient to make judgments about connections between drug use, driving performance, and crash risk.”³⁰⁵

299. See Sharma et al., *supra* note 283, at 150.

300. *Id.*

301. *Id.*

302. See, e.g., *id.* at 152; MACK & JOY, *supra* note 290, at 12.

303. See *Drug Concentrations and Driving Impairment*, *supra* note 292, at 2619.

304. NHTSA REPORT (2010), *supra* note 273, at 4.

305. Berning & Smither, *supra* note 289, at 1 (citing R. COMPTON ET AL., DOT HS 811 268, DRUG-IMPAIRED DRIVING: UNDERSTANDING THE PROBLEM AND WAYS TO REDUCE IT (2009)).

Moreover, the very same factors that render alcohol so conducive to study significantly limit the ability to study the effect of specific THC blood levels on the safety of one's driving. Controlled driving experiments under realistic conditions are difficult both because marijuana and other drugs are illegal in most states (and thus cannot be administered to subjects with the same ease), and because specific levels of THC in the blood have wildly different levels of psychoactive effect depending on the person and the circumstances. As the NHTSA website acknowledges, "[i]t is difficult to establish a relationship between a person's THC blood or plasma concentration and performance impairing effects."³⁰⁶ Indeed, researchers have found that chronic cannabis users develop a tolerance to many of the psychomotor effects of THC,³⁰⁷ including those specifically related to driving tasks.³⁰⁸ Researchers have also noted that the impairing effects of certain drugs might be confused with the impairing effects of *withdrawal* from those drugs.³⁰⁹ In short, as a recent consensus report on drugged driving concluded, "[i]nterpreting the meaning of either drug/metabolite concentration in a single biological specimen with reference to impaired driver performance is therefore an extremely difficult task for a scientist and even more difficult for a prosecutor."³¹⁰

B. The Prohibitionist Approach: The Use of Zero-Tolerance DUI Drug Laws Before the Legalization Movement

As the 1980s drew to a close, the war on drugs was in full force, causing the unresolved problem of drugged driving to come to the fore.³¹¹ Beginning in

306. *Cannabis/Marijuana Fact Sheet*, NHTSA, <http://www.nhtsa.gov/people/injury/research/job185drugs/cannabis.htm> (last visited Apr. 9, 2015) (emphasis added).

307. See, e.g., Brett C. Ginsburg et al., *Blood Levels Do Not Predict Behavioral or Physiological Effects of Delta-9-Tetrahydrocannabinol in Rhesus Monkeys with Different Patterns of Exposure*, 139 DRUG & ALCOHOL DEPENDENCE 1, 2 (2014); D.M. Schwoppe et al., *Psychomotor Performance, Subjective and Physiological Effects and Whole Blood Delta-9-Tetrahydrocannabinol Concentrations in Heavy, Chronic Cannabis Smokers Following Acute Smoked Cannabis*, 36 J. ANALYTICAL TOXICOLOGY 405, 409–11 (2012) (finding no significant effect in heavy users with respect to tracking error and reaction time, and a "minimal" effect on divided-attention tasks).

308. See, e.g., W.M. Bosker et al., *Medicinal Delta-9-Tetrahydrocannabinol (Dronabinol) Impairs On-the-Road Driving Performance of Occasional and Heavy Cannabis Users But Is Not Detected in Standard Field Sobriety Tests*, 107 ADDICTION 1837, 1837 (2012).

309. See P.M. Marzuk et al., *Prevalence of Recent Cocaine Use Among Motor Vehicle Fatalities in New York City*, 263 J.A.M.A. 250, 255 (1990) (noting that cocaine withdrawal may be responsible for increased accident risk); see also M. Haney et al., *Marijuana Withdrawal in Humans: Effects of Oral THC or Divalproex*, 29 NEUROPSYCHOPHARMACOLOGY 158, 167 (2004) (noting symptoms of marijuana withdrawal: irritability, "muscle pain, chills, decreased food intake, and decreased self-reported sleep quantity").

310. THE WALSH GROUP, *THE FEASIBILITY OF PER SE DRUGGED DRIVING LEGISLATION* (2002), available at <http://stopdruggeddriving.org/pdfs/FeasibilityofPerSeLawsConsensusReport.pdf>; see also Ginsburg et al., *supra* note 307, at 7 ("The relationship between blood levels of THC and behavioral impairment differ from the relationship for ethanol.").

311. Recent government survey results reveal that millions of teenagers and adults drive under the influence of illicit drugs. U.S. DEP'T OF HEALTH & HUMAN SERVS., *RESULTS FROM THE 2006 NATIONAL SURVEY ON DRUG USE AND HEALTH: NATIONAL FINDINGS 2* (2007) (noting that 10.2

1990, twelve states³¹² chose to address the problem through a new DUI jurisprudence of prohibition, banning driving with *any* illicit drug in one's body. Under a prohibitionist logic, these laws made sense: if having an illicit drug in one's system is itself morally blameworthy, then driving while having the drug in one's system is morally blameworthy, even without proof of any impairing effect. In 1990, marijuana was not only illegal in all fifty states, but a "Schedule I" drug deemed to have "no medicinal value and high potential for abuse."³¹³ Indeed, in most states, it still is today.³¹⁴ Criminal punishment of driving with any amount of an illicit drug in one's body therefore had a legitimate penal purpose, regardless of the inability to show any link to impairment.

Government officials were open about their embrace of this new penal theory for DUI *illicit* drugs, as well as about their retention of the traditional focus on dangerousness for *legal* drugs. The NHTSA described the central premise of zero-tolerance laws as being that "the use of the drug is illegal, not that a specific concentration equates to impairment."³¹⁵ When an Arizona man recently challenged his conviction under that state's zero-tolerance law because he had only the inactive THC metabolite (THC-COOH) in his body, the State argued that the law simply did not target dangerousness; rather, its purpose was to target any trace of an illicit drug in the driver's body through a "per se prohibition" and a "flat ban on driving with *any* proscribed drugs in one's

million people aged twelve and older reported driving under the influence of an illicit drug in the previous year).

312. See 1990 Ariz. Sess. Laws (codified as amended at ARIZ. REV. STAT. ANN. § 28-1381) (effective June 28, 1990; zero tolerance for any metabolite); 75 Del. Laws 2006, ch. 397, § 15 (codified as amended at 21 DEL. CODE ANN. tit. 21, § 4177 (2014)) (effective July 10, 2007); 2001 Ga. Laws 208 (codified as amended at GA. CODE ANN. § 40-6-391 (2014)) (effective July 1, 2001); 1997 Ill. Laws 90-400 (codified as amended at 625 ILL. COMP. STAT. 5/11-501 (2014)) (effective Aug. 15, 1997; excepting medical marijuana use from zero-tolerance provision); 2001 Ind. Acts. 175 (codified as amended at IND. CODE ANN. § 9-30-5-1 (2015)) (effective July 1, 2001); 1998 Iowa Acts ch. 1138 (codified as amended at IOWA CODE ANN. § 321J.2 (2015)) (effective July 1, 1998; "any amount of a controlled substance"); 2003 Mich. Pub. Acts No. 61 (codified at MICH. COMP. LAWS § 257.625) (effective Sept. 30, 2003; zero-tolerance for controlled substances); 2006 Minn. Laws c. 260, art. 2, § 2 (codified as amended at MINN. STAT. ANN. § 169A.20 (2009)) (effective Aug. 1, 2006; excepting cannabis); 2005 R.I. Pub. Law 2005, ch. 443, § 1 (codified as amended at R.I. GEN. LAWS § 21-28.6-7 (2014)) (effective Jan. 3, 2006); 1994 Utah Laws (codified as amended at UT. CODE ANN. § 41-6a-517 (West 2014)) (effective May 2, 1994); 2005 Va. Acts c. 845 (codified as amended at VA. CODE ANN. § 18.2-266 (2014)) (approved Mar. 26, 2005; imposing zero-tolerance for marijuana and some drugs, with low numerical thresholds for cocaine, methamphetamine, PCP, and Ecstasy); 2003 Wis. Sess. Laws. a. 30, 97 (codified as amended at WIS. STAT. ANN. § 346.63 (effective Dec. 19, 2003)).

313. Shaunacy Ferro, *Why It's So Hard for Scientists To Study Medical Marijuana*, POPULAR SCI. (Apr. 18, 2013), <http://www.popsci.com/science/article/2013-04/why-its-so-hard-scientists-study-pot>.

314. See, e.g., Editorial, *High Time for Advancing Marijuana Research*, 17 NATURE NEUROSCIENCE 481 (2014).

315. NHTSA, PRIORITIES AND STRATEGIES FOR IMPROVING THE INVESTIGATION, USE OF TOXICOLOGY RESULTS, AND PROSECUTION OF DRUG-IMPAIRED DRIVING CASES: FINDINGS AND RECOMMENDATIONS 12 (2004).

system.”³¹⁶ One researcher arguing for a zero-tolerance approach to DUI marijuana reasoned that it “sends a clear and concise message to those who use illicit drugs such as cannabis that this behaviour will not be tolerated, especially in connection with driving.”³¹⁷ Moreover, states with such laws created exceptions for prescription drugs, which were to be governed by a subjective impairment standard requiring a showing that the driver was actually under the influence of the drug.³¹⁸ In short, officials’ retention of a subjective impairment requirement for legal drugs reflected a jurisprudence of dangerousness in line with the Haddon framework.

To be sure, this new prohibitionist approach had as its ultimate goal not simply to deter drug use, but to get more convictions in cases involving what officials saw as dangerous, but difficult to detect, drugged driving.³¹⁹ But a prohibitionist approach is an awkward fit if the justification for the law is the dangerousness of the drug’s impairing effects, rather than simply the immorality of using the drug. Presumably, the reason these states chose a prohibitionist approach to justifying a law that had dangerous driving as its ultimate *raison d’être* was that they viewed the alternatives as either ineffective or impossible. Directly criminalizing dangerous drugged driving through a subjective impairment standard had proven ineffective in securing high conviction rates, and enactment of a non-zero per se limit, akin to .08 percent BAC, that was causally linked to increased relative crash risk had proven scientifically elusive.

Adopting a prohibitionist approach to solve a dangerousness problem may have been a bad fit in terms of penal legitimacy, but it at least made convictions easier. When asked to explain the impetus for a congressional bill introduced in 2004 that would incentivize states to pass zero-tolerance DUI laws for illicit drugs, John Walters, then the director of the White House Office of National Drug Control Policy, argued that “a simple clear guideline . . . is needed to

316. See, e.g., State’s Response to Petition for Review of Special Action Decision of the Court of Appeals at 10, *State ex rel. Montgomery v. Harris*, 322 P.3d 160 (Ariz. 2013) (No. CV–13–0056–PR) (quoting *State v. Phillips*, 873 P.2d 706, 710 (Ariz. Ct. App. 1994)); see also *id.* at 11 (arguing that the legislature had determined “that there is no level of illicit drug use which can be acceptably combined with driving a vehicle”) (quoting *State v. Phillips*, 873 P.2d 706, 710 (Ariz. Ct. App. 1994)).

317. Alan W. Jones et al., *Driving Under the Influence of Cannabis: A 10-Year Study of Age and Gender Differences in the Concentrations of Tetrahydrocannabinol in Blood*, 103 ADDICTION 452, 459 (2008).

318. See, e.g., ARIZ. ST. SENATE, FACT SHEET, H.B. 2433, DUI: OMNIBUS (1990) (providing a prescription drug exception to the new zero-tolerance DUI drug law, and noting that the subjective impairment standard would apply); 21 DEL. CODE ANN. tit. 21, § 4177(b)(3)(b) (2014) (prescription drug exception); R.I. GEN. LAWS § 21-28.6-7 (2014) (medical marijuana exception). A 2002 Consensus Report of criminal justice actors recommended that “[a] model [zero-tolerance] law should include . . . [p]rovisions to stipulate that legal prescription use of a drug is an affirmative defense to a DUI charge.” WALSH GROUP, *supra* note 310, at 2.

319. See, e.g., NHTSA REPORT (2010), *supra* note 273, at 5 (“In most of these per se States, the compelling argument for adoption of the drug per se statute was that, prior to the laws, a driver was far less likely to be prosecuted.”).

combat drugged driving.”³²⁰ As one legal scholar sympathetic to such laws has argued, “[a] zero tolerance law is the easiest standard to prove” because it does not turn on “the impact the drug has on one’s ability to drive.”³²¹ A sponsor of a recent zero-tolerance bill in Illinois similarly acknowledged that a driver could be “sober as a judge, because they smoked [marijuana], or -- or consumed it twenty days ago,” but that “it’s DUI . . . [t]hat’s the law.”³²² The sponsor explained that unlike alcohol, a context in which “.08 has been generally accepted as enough to indicate an impaired driver,” marijuana has no accepted test for driving impairment.³²³

The scientific community also candidly acknowledged that the zero-tolerance approach was a way to bypass the need for science to back up a dangerousness-based DUI law. One physician suggested in a 2012 article in the *Journal of Analytical Toxicology* that the adoption of zero-tolerance laws were necessary precisely because the ability to determine impairment based on non-zero numerical thresholds was a “mirage,” and that “there is abundant scientific evidence demonstrating that” finding drug concentrations analogous to the .08 percent BAC level in terms of indicating unsafe levels of intoxication “is unachievable.”³²⁴ Another researcher noted that zero-tolerance DUI drug laws “avoid[] the need for a reliable science-based correlation between drug concentration and level of impairment and facilitate[] enforcement.”³²⁵ Until it was possible to justify a per se limit through science and a focus on dangerousness, a prohibitionist stance would have to do.

The shift toward a jurisprudence of prohibition was a clever and effective move for DUI in the context of illicit drugs. The illegality of the drug itself made the laws both legitimate in terms of penal purpose and politically feasible. If certain cultural and political shifts had not been on the near horizon, the country might have followed the urging of the NHTSA and the White

320. Donna Leinwand, *Growing Danger: Drugged Driving*, USA TODAY, Oct. 21, 2004 (citing John Walters, former director of the White House Office of National Drug Control Policy); see also Drug Impaired Driving Enforcement Act of 2004, H.R. 3922, 108th Cong. (2004); Mark Hansen, *Drugged State*, A.B.A. J. (Dec. 29, 2005, 11:44 AM), available at http://www.abajournal.com/magazine/article/drugged_state (noting critics’ argument that the laws “are based not on science but on convenience,” and that “[s]ince scientists can’t agree on what level of drug or drug metabolite constitutes impairment,” politicians “often simply choose to set the standard at zero”).

321. Tina W. Cafaro, *Slipping Through the Cracks: Why Can’t We Stop Drugged Driving?*, 32 W. NEW ENG. L. REV. 33, 45 (2010).

322. *Transcript*, 2013 S., 98th Sess. (Ill. 2013) (statement of Sen. William Haine).

323. *Id.*

324. Reisfield, *supra* note 44, at 353; see also Jones et al., *supra* note 317, at 459 (arguing that because “[s]cientists find it virtually impossible to agree” on a THC concentration that leads to impairment, zero-tolerance laws are the most “pragmatic” solution).

325. F. Grotenhermen et al., *Developing Limits for Driving Under Cannabis*, 102 ADDICTION 1910, 1911 (2007).

House to create a nationwide zero-tolerance drugged driving regime.³²⁶ What would throw state lawmakers for a loop, and send them scrambling to find a quick fix, was the marijuana legalization movement.

C. *The Illegitimacy of Per Se THC Thresholds*

1. *Legalization and the Rise of Per Se Marijuana Laws: Political Compromise Without a Penal Purpose*

The recently successful movements in many states to decriminalize or legalize some combination of medical and recreational marijuana, beginning with California's medical marijuana law in 1996 and involving most recently the decisions of Colorado, Washington, Alaska, and Oregon to fully legalize recreational marijuana,³²⁷ has created quite a public health and jurisprudential dilemma for state officials. On the public health end, the drugged driving problem seems to have become worse. Just as the end of Prohibition gave the drunk driving problem new urgency, the legalization of marijuana seems to have increased the number of drivers on the road who have recently ingested marijuana.³²⁸ Meanwhile, the country is also facing a surge in driving by users of other legal prescription drugs and narcotics; while "drunken-driving deaths are dropping[,] . . . prescriptions for narcotic painkillers, anti-anxiety medications, sleep aids and other powerful drugs are rampant."³²⁹

326. In 2002, the NHTSA recommended that states pass per se drugged driving laws (which, at the time, meant zero-tolerance laws). Fox Butterfield, *Many, Undetected, Use Drugs and Then Drive, Report Says*, N.Y. TIMES, Nov. 15, 2002, https://www.google.com/url?sa=t&rct=j&q=&esrc=s&source=web&cd=1&cad=rja&uact=8&ved=0CB8QFjAA&url=http%3A%2F%2Fwww.nytimes.com%2F2002%2F11%2F15%2Fus%2Fmany-undetected-use-drugs-and-then-drive-report-says.html&ei=l-8mVbalH5TpoATi34CICg&usq=AFQjCNEOcuJOn_I6FoS2AGBZ7hAoZF4byA&sig2=IyHlmqG Xy-3sn82aODS_A&bvm=bv.90491159,d.cGU. As of 2012, the White House itself has urged all states to adopt "per se ('zero tolerance') laws." OFF. OF NAT'L DRUG CONTROL POLICY, EXEC. OFF. OF THE PRESIDENT, WORKING TO REDUCE DRUGGED DRIVING AND PROTECT PUBLIC HEALTH AND SAFETY 2 (Apr. 2012), available at https://www.whitehouse.gov/sites/default/files/page/files/drugged_driving_fact_sheet_4-6-12.pdf.

327. The legal age for marijuana consumption in the states that have legalized marijuana is generally 21. See, e.g., ALASKA STAT. § 17.38.010 (2014); COLO. REV. STAT. § 18-18-406 (2014); WASH. REV. CODE. § 69.50.401 (2015); *Recreational Marijuana: Frequently Asked Questions*, OREGON.GOV, http://www.oregon.gov/olcc/marijuana/Pages/Frequently-Asked-Questions.aspx#Personal_Use (last viewed Apr. 9, 2015) (noting that the legal age for marijuana in Oregon beginning July 1, 2015, will be twenty-one).

328. See, e.g., Gene Johnson, *More Washington State Drivers Testing Positive for Marijuana*, HUFFINGTON POST (Nov. 21, 2013, 2:50 PM), http://www.huffingtonpost.com/2013/11/21/washington-state-marijuana_n_4318516.html. Washington's per se 5 ng/mL law was passed primarily to "allay fears that legalizing pot would lead to more impaired drivers on the roads." Gabriel Spitzer, *Will Legal Marijuana Make Our Roads More Dangerous?*, KPLU NEWS (Oct. 8, 2012), www.kplu.org/post/will-legal-marijuana-make-our-roads-more-dangerous.

329. Abby Goodnough & Katie Zezima, *Drivers on Prescription Drugs: Dangerous, but Hard to Convict*, N.Y. TIMES, July 24, 2010, at A1.

On the jurisprudential side of things, state officials have also faced a new predicament in envisioning how to criminalize DUI marijuana. The prohibitionist, zero-tolerance approach no longer works where marijuana is legal, just as it does not work for alcohol. If the use of marijuana itself is no longer deemed worthy of criminal punishment, then driving while using marijuana cannot be criminalized solely on the basis of the immorality of the drug itself. But the alternative—subjective impairment laws focused on dangerous driving—is similarly unsatisfying to states. Drugged driving prosecutions in states without zero-tolerance laws had perhaps been made slightly easier since the 1970s with the development of “Drug Recognition Expert” training programs for police officers³³⁰ and dashboard cameras that could corroborate police testimony in cases of clear outward signs of drug impairment. But such efforts pale in comparison to the power that per se BAC limits gave law enforcement to secure drunk driving convictions.

Faced with the prospect of marijuana being as legal as alcohol, law enforcement officials in some states have claimed they need a non-zero numerical threshold, like .08 percent, to successfully combat DUI marijuana.³³¹ And the way officials and advocacy organizations in these states have sold the concept of such numerical limits to the public has simply been to analogize marijuana to alcohol. In arguing for a per se THC limit in Colorado, District Attorney Thomas Raynes testified before the state legislature that such per se standards are “already used” in the DUI alcohol context.³³² And, a co-sponsor reiterated that drugged driving law needed an “analogous limit” to DUI and added, for good measure, jurors “really like to see that chemical test.”³³³ Federal officials have used similar logic. The Limiting Unsafe Cannabis-Impaired Driving Act (LUCID), a bill introduced in March 2014 by Representative Jared Polis of Colorado, would require states that allow the use of recreational or medicinal marijuana to enact per se THC limits for DUI marijuana.³³⁴ In attempting to justify the Act, Polis explained that because states that legalize marijuana have chosen to “follow the will of their citizens

330. See NHTSA Report (2010), *supra* note 273, at 3.

331. See, e.g., *Hearing on Montana H.B. 168*, 2013 Leg. 63rd Leg. Reg. Sess. (Mont. 2013), at 1:32 (statement of Rep. Moore) (noting that Montana police struggle with DUI drug prosecutions because they do not have a “specified level,” akin to “BAC,” to measure impairment).

332. *Statement of Thomas Raynes in the H. Judiciary Comm.*, 2013 Reg. Sess., at 1:22 (Colo. Feb. 26, 2013), available at <http://www.leg.state.co.us/clics/clics2013A/cslFrontPages.nsf/Audio?OpenPage>.

333. See, e.g., *Statement of Rep. Waller in the H. Judiciary Comm.*, 2013 Reg. Sess., at 17:30; 25:28; 25:42 (Colo. 2013), available at <http://www.leg.state.co.us/clics/clics2013A/cslFrontPages.nsf/Audio?OpenPage>; *Hearing on Montana H.B. 168*, 2013 Leg. 63rd Leg. Reg. Sess., at 1:32 (Mont. 2013) (statement of Rep. Moore).

334. Matt Ferner, *Stoned Driving Standards Could Get a Push from Congress*, HUFFINGTON POST (Mar. 7, 2014, 7:24 PM), http://www.huffingtonpost.com/2014/03/07/marijuana-high-driving_n_4914645.html; see also Lucid Act, H.R. 4179, 113th Cong. (2014).

and implement regulations to treat marijuana like alcohol,” it clearly follows that these states’ traffic laws should do the same.³³⁵

Some advocates of legalization appear to have been willing to give law enforcement a number—any number—in exchange for the holy grail of treating DUI marijuana like DUI alcohol.³³⁶ In Washington State, a grand compromise between legalization advocates and law enforcement led to a ballot measure that linked legalization to the enactment of a per se THC threshold for DUI marijuana of 5 nanograms per milliliter of whole blood (ng/mL).³³⁷ When asked by an audience member at a panel on the new legalization law “why the initiative’s authors had seen the need for a crackdown on drugged driving after years of marijuana use in society,” the Seattle city attorney “said the language was at least, in part, a political calculation.”³³⁸ Another participant in the negotiations, interviewed by National Public Radio, said the Washington law was “a deal-sweetener for hesitant voters.”³³⁹ A similar compromise by all accounts occurred in Colorado; the bill setting per se DUI marijuana limits in that state failed three times until the fourth successful attempt, which coincided with the passage of Amendment 64, legalizing recreational marijuana use.³⁴⁰ While a California per se THC-limit bill was rejected, its “registered support[ers]” included eight law enforcement associations.³⁴¹ At least two of these laws allow an affirmative defense for drivers with a legal prescription for marijuana.³⁴² This exception further confuses the laws’ penal purpose and adds to the evidence that they are the result of quick political compromise rather than a deliberate focus on either the immorality of drug use or the dangerousness of drug intoxication while driving.

335. Ferner, *supra* note 334 (internal quotation marks omitted).

336. One pro-legalization website complains that “the push for *5ng/ml per se DUID* is simply a case of lazy policy makers applying alcohol standards to marijuana,” but in a fit of self-awareness, adds that “[m]aybe our constant drumbeat of ‘treat marijuana like alcohol’ is partially to blame.” See *No Direct Correlation Between Driving Impairment and THC Concentration*, THE RUSS BELVILLE SHOW (Mar. 14, 2013), <http://radicalruss.com/no-direct-correlation-between-driving-impairment-and-thc-concentration/>.

337. The drugged driving law and legalization law were passed as part of the same ballot initiative. See *Marijuana: Legalization & Regulation*, Initiative Measure No. 502, 63rd Leg., 2013 Reg. Sess. (Wash. 2012) (codified as amended at WASH. REV. CODE. § 69.50.401 (2015)).

338. Kirk Johnson, *Marijuana Referendum Divides Both Sides*, N.Y. TIMES, Oct. 14, 2012, at A18.

339. See Martin Kaste, *Pot’s Legal in Washington State, But Don’t Drive High*, NPR (Dec. 5, 2012, 2:23 AM), <http://www.npr.org/2012/12/05/166531388/pots-legal-in-washington-state-but-dont-drive-high/>.

340. See Matt Ferner, *Too Stoned To Drive? Marijuana DUI Bill Passes Colorado House*, HUFFINGTON POST (Mar. 3, 2015, 7:24 PM), http://www.huffingtonpost.com/2013/04/02/too-stoned-to-drive-marij_n_3001016.html.

341. See GABRIEL CASWELL, BILL ANALYSIS FOR THE CAL. STATE ASSEMB. COMM. ON PUB. SAFETY, A.B. 2500, 2013–2014 Reg. Sess. (2014).

342. See, e.g., NEV. REV. STAT. § 484C.110 (2014) (medical marijuana exception). California’s rejected per se 2 ng/mL bill also had a medical marijuana exception. See A.B. 2500, 2013–2014 Assemb., 2013–2014 Reg. Sess. (Cal. 2014).

The tactic of analogizing to DUI, picking a number, and offering to support legalization in exchange for that number has been decently successful thus far in convincing voters and legislators to agree to *per se* THC limits for DUI marijuana. While the LUCID Act is still pending in committee, five states have passed laws prohibiting driving with a certain non-zero threshold of cannabinoids such as THC and, in some states, their metabolites in one's "whole blood":³⁴³ Montana (5 ng/mL),³⁴⁴ Pennsylvania (1 ng/mL),³⁴⁵ Ohio (2 ng/mL),³⁴⁶ Nevada (2 ng/mL),³⁴⁷ and Washington (5 ng/mL).³⁴⁸ In addition, in Colorado, the presence of THC/blood levels above 5 ng/mL "gives rise to a permissible inference that the defendant was under the influence."³⁴⁹ The California legislature has been the only state body thus far to entertain and reject a bill that would have imposed a 2 ng/mL threshold.³⁵⁰ Alaska and Oregon are notable exceptions to the trend; both states legalized recreational marijuana by public initiative in 2014,³⁵¹ and yet neither has imposed a *per se* THC limit for DUI purposes. But these legalization laws are still in their implementation and public comment phase, and new DUI marijuana regulations will follow.³⁵²

343. These thresholds measure the concentration of THC in *whole blood*, not plasma.

344. MONT. CODE ANN. § 61-8-411(1)(a).

345. 75 PA. CONSOL. STAT. ANN. § 3802(d)(1); 41 Pa. Bulletin 2295 (Apr. 30, 2011) (noting that minimum threshold for *per se* DUI marijuana under § 3802(d)(1) is 1 ng/mL), *available at* <http://www.pabulletin.com/secure/data/vol41/41-18/738.html>.

346. OHIO REV. CODE ANN. § 4511.19(A)(1)(j)(vii).

347. NEV. REV. STAT. § 484.397(3)(g),(h).

348. *See, e.g.*, WASH. REV. CODE § 46.61.502(1)(b) (2015) ("A person is guilty of driving while under the influence of . . . marijuana . . . if the person drives a vehicle . . . [a]nd the person has, within two hours after driving, a THC concentration of 5.00 or higher as shown by analysis of the person's blood.")

349. COLO. REV. STAT. § 42-4-1301(6)(IV) (2015) ("If at such time the driver's blood contained five nanograms or more of delta 9-tetrahydrocannabinol per milliliter in whole blood, as shown by analysis of the defendant's blood, such fact gives rise to a permissible inference that the defendant was under the influence of one or more drugs.")

350. *See Marijuana DUI Law Killed in California Legislature*, HUFFINGTON POST (May 5, 2014, 10:37 AM), [http://www.huffingtonpost.com/the-marijuana- dui-law-killed-_b_5257807.html](http://www.huffingtonpost.com/the-marijuana- DUI-law-killed-_b_5257807.html).

351. *See* Control, Regulation, and Taxation of Marijuana and Industrial Hemp Act, Measure 91 (Or. 2014) (enacted in scattered sections of 2015 Or. Laws Ch. 1), *available at* <http://www.oregon.gov/olcc/marijuana/Documents/Measure91.pdf>; An Act to Tax and Regulate the Production, Sale, and Use of Marijuana, Ballot Measure 2 (Alaska 2014) (codified in scattered sections of ALASKA STAT. § 11.71 (2015)), *available at* <http://www.elections.alaska.gov/doc/bml/BM2-13PSUM-ballot-language.pdf>.

352. *See, e.g.*, Research Brief from Chuck Burnham, Legislative Analyst, to State Senator Lesil McGuire, at 6-8, (Nov. 28, 2014), *available at* https://www.alaskasenate.org/2014/files/5614/1763/1020/Leg_Research_Report_on_Marijuana_Legalization.pdf (noting, in response to a request from State Senator Lesil McGuire, the pros and cons of *per se* THC limits and other states' approaches to DUI marijuana); Suzanna Caldwell, *After Warnings of Delay, Walker Says Pot Regulations on Schedule*, ALASKA DISPATCH NEWS (Dec. 30, 2014), <http://www.adn.com/article/20141230/after-warning-possible-delay-walker-says-pot-regulations-schedule>; Noelle Crombie, *Oregon Liquor Control Commission Asks Public for Input on Recreational Marijuana*, OREGONIAN (Jan. 6, 2015, 11:15 AM), http://www.oregonlive.com/marijuana/index.ssf/2015/01/oregon_liquor_control_commissi_2.html;

In sum, a number of states have successfully passed criminal laws imposing per se THC limits for drivers without ever articulating a penal theory for their legitimacy beyond simply wanting a number like .08 percent to better combat drugged driving. If lawmakers *were* forced to articulate a penal theory that justified these laws, they would be stuck. The jurisprudence of prohibition offers no comfort in a jurisdiction where marijuana use is not itself deemed criminal. And lawmakers have made only minimal efforts in attempting to explain why these non-zero limits, such as 5 ng/mL, might correspond to dangerous driving in the general population. They are, it would seem, ignorant of how .08 percent was justified through a jurisprudence of dangerousness in the DUI alcohol context: through careful scientific study of the relationship between BAC levels and impairment and between particular BAC levels and fatal crash risk—most especially the “smoking gun” of single-car, nighttime fatal crashes.

The public seems to understand that something is amiss. Press stories abound detailing stories of medical marijuana patients who wake up in the morning with THC levels higher than those set under the new DUI marijuana laws in their state.³⁵³ Marijuana advocates clearly suspect that the limits are arbitrary in their relationship to dangerous driving, but seem to believe either that the tradeoff was worth the risk or that the per se limits will eventually be changed to something more rational. The executive director of the National Organization for the Reform of Marijuana Laws, in commenting on Washington’s initiative, said that “[e]verybody anticipates if this passes, within a year or so afterwards it will be fixed”³⁵⁴

Yet even against the backdrop of this growing public intuition about the disconnect between per se limits and driving impairment, there seems to be little to no discussion of why the DUI alcohol analogy is a bad one, and what, precisely, is missing from the body of scientific research on marijuana impairment. That is, there is no discussion of how the DUI alcohol limits themselves were reached over decades of epidemiological research and what body of scientific evidence would have to be developed in the marijuana context to develop a rational per se regime akin to .08 percent under Haddon’s compulsory framework. The next Section details how the current science on marijuana as chemical impairment fares under this framework and how that science, if anything, undermines the hypothesis that THC blood levels are predictably related to increased relative crash risk.

353. See, e.g., *Here’s Where You Can Get Arrested for DUI Weeks After Smoking Marijuana*, HUFFINGTON POST (updated Jan. 23, 2014, 1:38 AM), http://www.huffingtonpost.com/2013/12/31/marijuana-dui_n_4520129.html.

354. Johnson, *supra* note 328 (quoting Kevin Sabet).

2. *The Scientific Invalidity of Per Se DUI Marijuana Limits Under Haddon's Established Framework*

To find a precise BAC level that corresponded to a morally blameworthy level of dangerous drinking and driving, Heise, Holcomb, Borkenstein, Haddon, Blomberg et al., and others knew where to look: BACs of drivers killed in single-car crashes, and case-control studies comparing BAC levels of drivers in fatal crashes with BACs of randomly stopped drivers under exactly the same conditions (aside from BAC itself). Researchers also measured, through actual driving experiments involving subjects at various BACs, the level of deterioration of various driving-related skills. They had the luxury of being able to conduct such studies because of the unique pharmacokinetic and pharmacodynamic properties of alcohol. Applying this DUI alcohol framework to the marijuana context, it is clear that the THC blood limits chosen by states have no scientific basis if their purpose is to target dangerous driving. On the contrary, the science that does exist strongly suggests that these levels do not correspond with dangerous driving impairment. This is not to say that driving while stoned is safe; it is only to say that, as of this writing, THC blood levels cannot legitimately be used to define chemical impairment under a science-based jurisprudence of dangerousness.

a. *Single-Car Fatal Crashes*

In studying alcohol, William Haddon had looked first to the ultimate smoking gun of crash causation: single-car fatal crashes, in which culpability could most readily be inferred from the involvement of alcohol. He found that fully half of fatally injured drivers in eighty-three single-car crashes, most of which occurred at night, had a BAC of .15 percent or more, another 20 percent had a BAC between .05 and .14 percent, and only three of eighty-three drivers had a non-zero BAC lower than .05 percent.³⁵⁵ Haddon took measures to remove drivers from the study who had not been tested shortly after the crash, so that the BAC results would better reflect the driver's BAC at the time of the crash.³⁵⁶

With respect to marijuana, no research team has yet conducted a single-car crash study that limits the sample to drivers tested within a certain period after an accident, focuses on THC only rather than in combination with other drugs, and distinguishes between low, moderate, and high levels of THC in the blood.³⁵⁷ Indeed, according to the NHTSA itself, "sound epidemiologic risk

355. See *supra* text accompanying notes 133–135.

356. See Haddon & Braddess, *supra* note 133, at 1588.

357. Likewise, no THC study has yet focused—as Haddon did—on nighttime crashes. Nighttime crashes in the alcohol context were a particularly rich source of data because both alcohol use and crashes were at their apex at night. See discussion *supra* note 139. Marijuana use may be less concentrated in the evening hours than alcohol use, although some studies suggest that alcohol-related nighttime crash rates have declined in states with medical marijuana laws in large part because

studies have not been conducted” for DUI marijuana or other drugged driving.³⁵⁸ Period. This is in part because “[f]ortunately for traffic safety but unfortunately for epidemiological research[,] DUI [cannabis] is far less common” than DUI alcohol.³⁵⁹

Strikingly, the four studies that do offer data on THC and single-car fatal crashes suggest that THC-only users do *not* cause a disproportionate number of crashes. The most comprehensive study thus far has been Romano and Voas (2011), which looked at over 44,000 single-car fatal crashes listed in FARS from 1998-2009 and found that only 6 percent of drivers tested positive for THC or THC plus alcohol (the authors did not further separate the two).³⁶⁰ Six percent of drivers also tested positive for stimulants.³⁶¹ Citing the fact that a full 7.6 percent of drivers in the 2007 NHTSA roadside survey tested positive for marijuana while only 1.9 percent tested positive for stimulants, Romano and Voas concluded that this data “seems to suggest that stimulants may be a larger contributor to crash risk than cannabinoids.”³⁶² Indeed, from a comparison of the FARS and NHTSA data alone, it appears that marijuana users are underrepresented, not overrepresented, among single-car crash victims.

The second single-car-crash study, Poulsen et al. (2014), found that of 460 single-car crashes in New Zealand from 2004 to 2009, only thirty-five, or 7.6 percent, of the drivers tested positive for any amount of THC.³⁶³ They concluded that there was “no apparent increased risk” of a fatal crash with “blood THC concentrations” above 5 ng/mL.³⁶⁴

marijuana use has substituted for alcohol use. See, e.g., Maia Szalavitz, *Why Medical Marijuana Laws Reduce Traffic Deaths*, TIME (Dec. 2, 2011), <http://healthland.time.com/2011/12/02/why-medical-marijuana-laws-reduce-traffic-deaths/>.

358. NHTSA Report (2010), *supra* note 273, at 3. Nor have “scientific studies to demonstrate the[] effectiveness” of per se DUID laws been conducted. *Id.* at 5.

359. Grotenhermen et al., *supra* note 325, at 1912.

360. Eduardo Romano & Robert B. Voas, *Drug and Alcohol Involvement in Four Types of Fatal Crashes*, J. STUD. ALCOHOL & DRUGS, July 2011, at 567, 571.

361. *Id.* at 571.

362. *Id.*; see also NHTSA, 2007 NATIONAL ROADSIDE SURVEY OF ALCOHOL AND DRUG USE BY DRIVERS: DRUG RESULTS 81 (2007), available at <http://www.nhtsa.gov/Driving+Safety/Research+&+Evaluation/2007+National+Roadside+Survey+of+Alcohol+and+Drug+Use+by+Drivers> (noting percentages of marijuana use only, broken down by region, gender, etc.; drivers in the Northeast had a 9.5 percent THC-only rate).

363. Helen Poulsen et al., *The Culpability of Drivers Killed in New Zealand Road Crashes and Their Use of Alcohol and Other Drugs*, 67 ACCIDENT ANALYSIS & PREVENTION 119, 122 tbl. 1 (2014). The study did not break down THC blood levels except to note that, of 1,046 drivers involved in any crash (single or multiple car) during the period, ninety-six tested positive for THC only, and of those, only thirty tested over 5 ng/mL. The study made no further distinctions between levels above 5 ng/mL. *Id.*

364. *Id.* at 126. Even if a full half of the 7.6 percent THC-only drivers had low blood levels, such a percentage (3.8 percent) would be around the percentage of drivers in Haddon’s study at non-zero BACs below .05 percent. Moreover, the Poulsen data likely overstates the crash risk of low THC blood levels because of the delay in testing blood samples for THC after fatal accidents. See Grotenhermen et al., *supra* note 325, at 1913 (“Delayed sample collection causes a decrease in THC

In a similar vein, the third such study, Gjerde et al. (2011)³⁶⁵ found that of the sixty-eight drivers killed in single-car accidents in southeastern Norway from 2003 to 2008,³⁶⁶ not one driver tested positive for THC only.³⁶⁷ Gjerde concluded there was no statistically significant association “between the use of cannabis only and fatal road accidents.”³⁶⁸

In the fourth and final study analyzing single-car crash data, Marie Longo et al. (2000) found that in 522 single-car *non-fatal-injury* crashes in South Australia from 1995-96, in which well over 90 percent of all drivers were found culpable, only 11 drivers—or 2 percent—tested positive for THC only, and only 7 drivers tested positive for both THC and alcohol.³⁶⁹ Compared to Haddon’s data justifying a .15 percent presumption of impairment based on *nearly half* of drivers killed in single-car crashes being over .15 percent,³⁷⁰ such low numbers for THC are striking.

Nonetheless, public officials and the media have taken to making sweeping claims about drugged driving causality. The Institute for Behavioral Health claimed in 2007 without citation that “20 percent of crashes are *caused by* drugged driving.”³⁷¹ An NBC News reporter wrote that “new research” shows a causal link between marijuana and crashes because the percentage of drivers in fatal accidents who tested positive for marijuana has increased three-fold in the last eleven years.³⁷² One legal scholar recently wrote that “drug-impaired drivers are *causing* accidents and deaths on the roadways in increasing numbers, and the statistics are hard to ignore.”³⁷³ The National Institute on Drug Abuse’s website, in answer to the question, “How Often Does Drugged Driving Cause Accidents?,” simply cites a 2009 NHTSA study

concentration, artificially inflates the calculated accident risk for a given THC concentration and blurs the differences between THC concentration classes.”)

365. Hallvard Gjerde et al., *Alcohol, Psychoactive Drugs and Fatal Road Traffic Accidents in Norway: A Case-Control Study*, 43 ACCIDENT ANALYSIS & PREVENTION 1197 (2011).

366. Thirty-nine of the sixty-eight drove in the period from 4:00 p.m. to 4:00 a.m. *Id.* at 1200.

367. *Id.* at 1199, 1201 tbl. 4.

368. *Id.* at 1202. Gjerde noticed an association with fatal crashes and drivers with a combination of THC and other drugs or alcohol, but did not break down the combination further into blood levels of each drug. *Id.*

369. Marie C. Longo et al., *The Prevalence of Alcohol, Cannabinoids, Benzodiazepines and Stimulants Amongst Injured Drivers and Their Role in Driver Culpability, Part II: The Relationship Between Drug Prevalence and Drug Concentration, and Driver Culpability*, 32 ACCIDENT ANALYSIS & PREVENTION 623, 626 & tbl. 2 (2000). Longo et al. studied 2,500 injured drivers overall, but separated out single-car crashes from multiple-car crashes and gave separate analyses of each in terms of drug use prevalence. The number of drivers in each mutually exclusive drug category adds up to n=522. *Id.* at 626 tbl. 2.

370. See Haddon & Bradess, *supra* note 133, at 1589.

371. INST. OF BEHAVIOR & HEALTH, IBH PUBLIC POLICY STATEMENT REGARDING DRUGGED DRIVERS, available at http://www.druggeddriving.org/pdfs/druggeddriving_policy-statement.pdf (emphasis added) (last viewed Apr. 9, 2015).

372. Bill Briggs, *Pot Fuels Surge in Drugged Driving Deaths*, NBC NEWS (Feb. 15, 2014, 10:58 AM), <http://www.nbcnews.com/health/health-news/pot-fuels-surge-drugged-driving-deaths-n22991>.

373. Cafaro, *supra* note 321, at 35 (emphasis added).

showing that 18 percent of fatally injured drivers tested positive for at least one drug.³⁷⁴ To the question “What drugs contribute to accidents?” the site answers that THC is, after alcohol, “the substance most commonly found in the blood of impaired drivers, fatally injured drivers, and motor vehicle crash victims” and that “4 to 14 percent of drivers who sustained injury or died in traffic accidents tested positive for THC.”³⁷⁵

These claims are scientifically irresponsible. One of the central lessons of the Haddon era is that “the presence of a drug (medication) in a fatally injured driver is not conclusive proof the drug was associated with the accident.”³⁷⁶ For example, one would naturally expect, as the percentage of THC-positive drivers grows, that the percentage of THC-positive drivers in fatal accidents will grow regardless of whether THC use increases crash risk. More importantly, such claims invariably aggregate all drugs together, or fail to differentiate THC-only use from polydrug use. Every indication from existing single-car crash studies (and, it turns out, from case-control studies involving randomly selected drivers) strongly suggests that THC blood levels alone have no relationship with increased crash risk, much less THC at levels at or below 5 ng/mL. The NHTSA itself has recently underscored this point in a 2015 case-control study that “did not find an increase in population-based crash risk associated with THC use” based on drivers’ blood levels.³⁷⁷

Even the percentage of THC-positive drivers in total (single and multiple vehicle) fatal crashes is exceedingly low, suggesting—given the widespread use of marijuana—a lack of causality. In a recent Arizona Department of Public Safety review, fewer than 1 percent (three of 335) of suspected impaired drivers involved in crashes involving injury or death in 2012 tested positive only for marijuana.³⁷⁸ While the review was not limited to single-car crashes, those fatal crashes suspected to have involved impaired drivers are more likely than an average crash to involve culpability. Similar results came from a 2014 analysis of FARS data from 1993–2010, which included 986,173 drivers who

374. *DrugFacts: Drugged Driving*, NAT’L INST. ON DRUG ABUSE (Dec. 2014), <http://www.drugabuse.gov/publications/drugfacts/drugged-driving>.

375. *Id.*

376. Virginia Cowart & Peter Kandela, *Prescription Drugs and Driving Performance*, 254 *JAMA* 15, 20 (1985); see also José Pulido et al., *Association Between Cannabis and Cocaine Use, Traffic Injuries and Use of Protective Devices*, 21 *EUR. J. PUB. HEALTH* 753, 753–54 (2011) (surveying Spanish drivers about their involvement in non-fatal car crashes as well as drug use, and noting that while such surveys may help solve certain methodological problems in other studies, they “cannot provide causal evidence”).

377. See U.S. DEP’T OF TRANSP., NHTSA, *Drug and Alcohol Crash Risk Study: Executive Summary*, Feb. 2015, available at <http://www.nhtsa.gov/About+NHTSA/Press+Releases/2015/nhtsa-releases-2-impaired-driving-studies-02-2015>.

378. Ray Stern, *Marijuana by Itself Not a Significant Factor in Fatal and Injury Crashes in 2012, DPS Data Shows*, PHOENIX NEW TIMES (May 17, 2013, 10:00 PM), http://blogs.phoenixnewtimes.com/valleyfever/2013/05/marijuana_not_a_significant_fa.php. Only another nineteen tested positive for THC and alcohol or other drugs; 285 tested positive only for alcohol. *Id.*

were involved in fatal accidents, 287,907 of whom were tested for drugs.³⁷⁹ Of those, 998, or .3 percent, tested positive only for THC and alcohol, and fewer than half of those, approximately 450 people or .13 percent, tested positive only for THC.³⁸⁰ Drummer et al. (2004) found that, of 3,398 fatally injured drivers in three Australian states during various periods in the 1990s, only fifty-eight (1.7 percent) tested positive only for THC, with only nine testing at less than 5 ng/mL. The study did not further distinguish blood level.³⁸¹ Khiabani et al. (2006) found that of approximately thirty thousand drivers pulled over for suspected DUI in Norway from 1997–99, fewer than 2 percent (589) tested positive only for THC or its metabolites.³⁸² And del Rio and Alvarez (2000) found that of 285 fatally injured drivers in Spain in 1994–96, zero tested positive only for THC, while over 60 percent tested positive for drugs or alcohol.³⁸³

b. Case-Control Studies Comparing THC Levels of Drivers in Crashes to Randomly Stopped Drivers Under Similar Conditions

Working within Haddon’s established framework, Borkenstein et al. and Blomberg et al. conducted classic case-control studies comparing BACs of drivers involved in fatal crashes with BACs of randomly stopped drivers under nearly identical conditions. As Haddon established, such carefully crafted case-control studies, along with single-car fatal crash studies, are the gold standard of epidemiological research on the relationship between chemical impairment and relative crash risk. As the NHTSA itself recently noted with respect to drugged driving in particular, “‘roadside studies’ would be needed to collect blood samples from motorists (not involved in accidents) who are exposed to situations similar to those of fatally injured drivers” before inferring crash causality from a driver’s drug use.³⁸⁴

Yet such case-control studies are harder to perform with respect to marijuana than alcohol. It is more difficult to persuade randomly stopped drivers to submit to a blood test for THC than to a breath test for BAC, and

379. Fernando A. Wilson et al., *Fatal Crashes from Drivers Testing Positive for Drugs in the U.S., 1993–2010*, 129 PUB. HEALTH REP. 342 (2014).

380. *Id.*

381. Olaf H. Drummer et al., *The Involvement of Drugs in Drivers of Motor Vehicles Killed in Australian Road Traffic Crashes*, 36 ACCIDENT ANALYSIS & PREVENTION 239 (2004).

382. Hassan Z. Khiabani et al., *Relationship Between THC Concentration in Blood and Impairment in Apprehended Drivers*, 7 TRAFFIC INJURY PREVENTION 111, 112 (2006).

383. M. Carmen del Rio & F. Javier Alvarez, *Presence of Illegal Drugs in Drivers Involved in Fatal Road Traffic Accidents in Spain*, 57 DRUG & ALCOHOL DEPENDENCE 77, 179 (2000). In a later study, del Rio et al. (2002) found that of 5,745 fatally injured drivers in Spain from 1991–2000, while over 50 percent tested positive for a substance, 2.2 percent (127) tested positive for THC. The authors did not report how many tested positive for THC alone, but noted that cocaine was the post prevalent drug, with fifty-four drivers testing positive for cocaine alone. M. Carmen del Rio et al., *Alcohol, Illicit Drugs and Medicinal Drugs in Fatally Injured Drivers in Spain Between 1991 and 2000*, 127 FORENSIC SCI. INT’L 63, 66 (2002).

384. Cowart & Kandela, *supra* note 376, at 20.

other tests for THC—such as saliva and urine—are currently less accurate than blood and may underestimate THC blood level, potentially leading to artificially high crash risk estimates for low THC levels.³⁸⁵ The sample sizes are also much smaller, given the lower incidence of THC use than alcohol use, limiting statistical significance. In addition, the delayed timing of sampling after a crash versus a roadside control test might both overestimate the relative crash risk associated with low levels of THC and underestimate the relative crash risk associated with THC overall. More generally, the complexity of THC's effect on the body makes drawing inferences about level-specific dangerousness significantly more difficult compared to alcohol.

That said, none of the nine case-control studies conducted thus far on DUI marijuana with randomly stopped drivers has found a relationship between THC blood levels alone and increased crash risk, much less THC at levels as low as 5 ng/mL. The most comprehensive thus far is Romano et al. (2014), which compared drivers from FARS fatal crash data from 2006–08 with drivers stopped as part of the NHTSA's 2007 roadside survey.³⁸⁶ The study found no increased crash risk associated with marijuana use itself, in either the THC-only group or the THC-plus-alcohol group.³⁸⁷

A Columbia University study by Li et al. (2013) compared 737 fatal crashes in the United States in 2007 with the NHTSA's 2007 roadside survey of 7,719 drivers.³⁸⁸ While the authors found that 9.8 percent of drivers were THC-positive as compared to only 5.6 percent of the randomly surveyed group, they did not separate those positive only for THC from polydrug users nor did they differentiate blood levels.³⁸⁹

The Norway study by Gjerde et al. (2011), compared tests of drivers in fatal crashes from 2003–08 to oral samples from randomly stopped drivers in the same area from 2005–06.³⁹⁰ After adjusting for “confounding factor[s],” Gjerde found that the THC-only group was even less likely to be in a crash than sober drivers, albeit at a statistically insignificant level.³⁹¹

A 2011 European study comparing roadside survey results (oral and blood samples) in fifteen countries with drug and alcohol hospital tests on seriously or fatally injured drivers in crashes in a subset of those countries found widely

385. See Garry Milman et al., *Oral Fluid and Plasma Cannabinoid Ratios After Around-the-Clock Controlled Oral Delta-9-Tetrahydrocannabinol Administration*, 57 CLINICAL CHEM. 1597, 1603 (2011).

386. Eduardo Romano et al., *Drugs and Alcohol: Their Relative Crash Risk*, J. STUD. ALCOHOL & DRUGS, Jan. 2014, at 56.

387. *Id.* at 62.

388. Guohua Li et al., *Drug Use and Fatal Motor Vehicle Crashes: A Case-Control Study*, 60 ACCIDENT ANALYSIS & PREVENTION 205 (2013).

389. *Id.* at 208.

390. Gjerde et al., *supra* note 365, at 1197–98. The authors collected oral samples, rather than blood samples, in order to ensure that they had plenty of controls since people are pretty reticent to give blood samples. *Id.* at 1201.

391. *Id.* at 1201 tbl. 3.

variable rates of THC use in both the overall driver population (zero to 6 percent) and injured or killed driver population (zero to slightly over 2 percent).³⁹² The relative risks it calculated for THC varied widely, and it did not separate THC-only cases from polydrug cases.³⁹³ The authors' conclusion, stated with caution because of the design limits, was that "the relative risk for getting seriously injured and of getting killed while positive for cannabis were not significantly above 1,"³⁹⁴ meaning that the risk of crash at positive THC levels was not significantly higher than the risk of crash while sober.

Woratanarat et al. (2009) compared drug tests of two hundred drivers admitted to a hospital in Bangkok, Thailand, for road traffic injuries with 849 controls, with four or five controls taken from a randomly stopped driver at a gas station within one kilometer of each crash site.³⁹⁵ The authors found only four THC-positive crash victims and twenty THC-positive controls and made no causality claims (the "odds ratio" (OR), meaning the ratio of the odds of getting in a crash if THC positive to the odds of getting in a crash if THC negative, was below 1).³⁹⁶

Mathijssen and Houwing (2005) compared drug levels in 184 seriously injured drivers in a Dutch hospital and 3,799 randomly stopped drivers in the hospital's catchment area in the same week and found that while 3.9 percent of the control group tested positive only for THC, only 3.4 percent of the crash victims did.³⁹⁷

Movig et al. (2004) compared 110 seriously injured drivers and 816 randomly stopped motorists in the same Dutch region and found "no

392. Inger M. Bernhoft, DRUID PROJECT, RESULTS FROM EPIDEMIOLOGICAL RESEARCH – PREVALENCE, RISK, AND CHARACTERISTICS OF IMPAIRED DRIVERS (2011), available at http://www.druid-project.eu/Druid/EN/deliverables-list/downloads/Deliverable_2_4_1.pdf?__blob=publicationFile.

393. *Id.* at 6 fig. 6, 10, 12 fig. 14, 21 fig. 26.

394. *Id.* at 22. The author also took data from a previous culpability study in France from 2001–03 (not a case-control study with random drivers) to determine the relative risk of a *fatal* crash. For THC-positive drivers, the authors reported an "adjusted" relative risk for cannabis of 1.89. *Id.* at 26 fig. 33. The author did not differentiate blood levels above 5 ng/mL. *Id.* Curiously, the author included THC's inactive metabolite in counting THC-positive injured or killed drivers, but not in counting THC-positive controls, thus presumably artificially elevating the relative risk for THC. *See id.* at 5, 12 fig. 14 (noting that THC-COOH was not included in roadside survey data because it is not detectable in oral fluid, but in a later chart, including THC-COOH in calculating the percentage of THC-positive injured drivers).

395. Patarawan Woratanarat et al., *Alcohol, Illicit, and Non-Illicit Psychoactive Drug Use and Road Traffic Injury in Thailand: A Case-Control Study*, 41 ACCIDENT ANALYSIS & PREVENTION 651 (2009).

396. *Id.* at 655 (finding an adjusted OR of .78 for cannabis only).

397. RENÉ MATHIJSSSEN & SJOERD HOUWING, INST. FOR ROAD SAFETY RES., THE PREVALENCE AND RELATIVE RISK OF DRINK AND DRUG DRIVING IN THE NETHERLANDS; A CASE-CONTROL STUDY IN THE TILBURG POLICE DISTRICT 5, 19 (2005), available at <http://www.swov.nl/rapport/r-2005-09.pdf>. Curiously, the authors' calculated OR for cannabis only, based on 3.4 percent of cases and 3.9 percent of controls testing positive for cannabis only, was 1.45. *Id.* at 26. Because the percentage of controls was *higher* than the percentage of cases, the OR should presumably be less than 1. For purposes of the chart at Figure 5, *infra*, I use the 1.45 figure.

association” between cannabis and increased crash risk.³⁹⁸ Also in 2004, Blows et al. compared the 571 drivers injured or killed in Auckland, New Zealand, area crashes in 1998–99 with 588 randomly stopped drivers in the area and found that “acute” marijuana use (defined as ingesting within three hours of driving) had no association with increased crash risk.³⁹⁹

Finally, Dussault et al. (2002) compared blood or urine tests from 354 fatally injured drivers in Quebec from 1999–2001 and urine tests of 5,931 randomly stopped drivers in August 1999 and August 2000 at similar times of day, proportionately, to the fatal crashes.⁴⁰⁰ They found that 19.5 percent of the fatally injured drivers and 6.7 percent of the randomly stopped drivers tested positive for THC and/or some other drug.⁴⁰¹ But they did not state what percentage of these drivers also tested positive for alcohol or specify the drivers’ blood levels.⁴⁰² Moreover, they tested not just for THC but for the inactive compound THC-COOH, and had less than a 50 percent voluntary participation rate among controls, suggesting a selection bias that would significantly inflate relative risk estimates.⁴⁰³

c. “Culpability” Studies

Because of the difficulty in conducting THC crash-risk studies involving randomly selected, similarly situated drivers as controls, researchers have instead generally settled for “culpability” analyses.⁴⁰⁴ A culpability study looks at a group of drivers involved in crashes, and divides them into two groups—those who are “culpable” for their crash and those who are not. Researchers use the non-culpable group as a control and compare the percentage of drivers with various qualities—such as testing positive for THC—within the two groups. If THC-positive drivers are overrepresented in the culpable group, the logic goes,

398. K.L.L. Movig et al., *Psychoactive Substance Use and the Risk of Motor Vehicle Accidents*, 36 ACCIDENT ANALYSIS & PREVENTION 631, 633 (2004).

399. Stephanie Blows et al., *Marijuana Use and Car Crash Injury*, 100 ADDICTION 605, 607 (2005).

400. C. Dussault et al., *The Contribution of Alcohol and Other Drugs Among Fatally Injured Drivers in Quebec: Some Preliminary Results*, in ROAD TRAFFIC & PSYCHOACTIVE SUBSTANCES PROC., at 215 (2004), available at <https://wcd.coe.int/com.instranet.InstraServlet?command=com.instranet.CmdBlobGet&InstranetImage=889972&SecMode=1&DocId=1279432&Usage=2#page=215>.

401. *Id.* at 218–19.

402. *See id.*

403. *Id.* at 215. The authors did report that drivers using “cannabis alone” had an OR of 2.2 for crash risk and OR of 1.2 for culpability, whereas “cannabis + alcohol” had a stunningly high OR of 80.5 and OR of 2.5 for culpability. *Id.* at 221 tbl. 1. But the “cannabis + alcohol” category included only BACS above .08 percent, *id.*, even though the categories purported to cover “all cannabis cases,” suggesting that the “cannabis only” category included cases with cannabis and BACs between zero and .08 percent.

404. *See, e.g.*, J.G. Ramaekers et al., *Dose Related Risk of Motor Vehicle Crashes After Cannabis Use*, 73 DRUG & ALCOHOL DEPENDENCE 109, 110 (2004) (“Epidemiologists have tried to overcome the lack of normative data from the general driving population by analyzing the culpability index of drivers involved in traffic accidents.”).

one can infer causality and calculate an OR expressing the increased odds of being in a crash due to a driver's THC use. Traffic safety researchers now generally determine "culpability" of a driver based on eight "mitigating" factors suggested by Olaf Drummer in 1994: "the condition of the road, the condition of the vehicle, general driving conditions, the type of crash, witness observations, road law obedience, the difficulty of the task involved and the level of fatigue."⁴⁰⁵ The driver is assigned a "score" for each factor based on the level of mitigation ("four" being the worst possible state of a condition and therefore the most mitigating).⁴⁰⁶

There are significant potential problems with this approach, when compared to case-control studies with randomly selected drivers under identical conditions that formed the basis for .08 percent BAC laws. First, the scores are somewhat subjective, based on the researcher's (or, in some studies, the reporting police officer's) own assessment of the circumstances of a crash.⁴⁰⁷ Second, as with other THC studies, there are so few drivers with only THC in their system that inferences in favor of causation from THC by itself are difficult to draw. One reason may be that THC users do not drive as much as alcohol users; another may be the major overlap between high-risk demographics and cannabis-user demographics: young, male, with a high drunk-driving incidence.⁴⁰⁸ Third, as with other THC studies, the generalizable inferences that can be drawn from culpability studies are limited by the numerous factors that affect the psychoactive qualities of cannabinoids.⁴⁰⁹ For these and other reasons, one researcher at the Harvard School of Public Health recently concluded that culpability studies generally can only be said to accurately measure whether someone crashed without an "identified external cause" or whether an individual crashed in "good" driving conditions.⁴¹⁰ On the other hand, culpability studies have been criticized for *underestimating* crash risk attributable to THC, because the "baseline crash risk in non-culpable drivers is biased upwards,"⁴¹¹ and because some early culpability studies finding no association between THC and crash risk counted a positive test for the inactive metabolite THC-COOH as "positive" for THC.⁴¹²

405. Longo, *supra* note 369, at 625 (listing Drummer (1994)'s eight mitigating factor test).

406. *Id.*

407. Cf. Prachi Sanghavi, Author's Response, *Using Culpability Analysis to Infer Crash Risk Requires Unrealistic Assumptions*, 43 INT'L J. EPIDEMIOLOGY 272, 272 (2014) (using cell phone studies as an example).

408. See Rebecca L. Hartman & Marilyn A. Huestis, *Cannabis Effects on Driving Skills*, 59 CLINICAL CHEMISTRY 478, 479 (2013).

409. *Id.* at 478.

410. Prachi Sanghavi, *Commentary: Culpability Analysis Won't Help Us Understand Crash Risk Due To Cell Phones*, 42 INT'L J. EPIDEMIOLOGY, 267, 267-68 (2013).

411. Mark Asbridge et al., *Acute Cannabis Consumption and Motor Vehicle Collision Risk: Systematic Review of Observational Studies and Meta-Analysis*, 344 BMJ E536, at 4 (2012).

412. See, e.g., Ramaekers et al., *supra* note 404, at 110 (noting that Terhune (1992) tested for both THC and THC-COOH).

Of course, as Haddon recognized, the most probative culpability study of all is of single-car nighttime fatal crashes. With these crashes, we do not need an eight-factored test to infer culpability from the presence of alcohol or drugs in a driver's body. Researchers would have much more valuable and objective data simply from conducting more single-car crash studies than from culpability analyses, and such studies would not be prohibitively difficult, so long as drivers are tested shortly after crashes. Moreover, the fact that the single-car crash studies that have been conducted appear to strongly suggest a lack of causality should give lawmakers and researchers pause before inferring causality from the culpability studies that do exist.

In any event, the culpability studies conducted with respect to THC blood levels and fatal crashes have either not been relevant to, or have come to different conclusions about, whether THC alone is associated with increased crash risk. Some have found little to no association, or even a negative association. Poulsen (2014) reported curious results: an OR of 1.42 (a 42 percent increase in odds of crash over sober drivers) for THC levels below 2 ng/mL, a *lower* OR (a *lesser* odds of crashing than sober drivers) for levels between 2–5 ng/mL, and an OR of 1.61 for THC levels above 5 ng/mL.⁴¹³ Because of the small sample size, the confidence intervals ranged from .44 to 4.22 for various levels.⁴¹⁴ Longo (2000) used Drummer's technique and found, in a study of 2,500 non-fatally injured drivers in Australia from 1995–96, that drivers who tested positive only for THC were no more likely than drug-free drivers to have been "culpable" in a non-fatal crash.⁴¹⁵ Terhune et al. (1992) found that the "responsibility rates" of THC-only drivers were "actually lower than [those] of the drugfree drivers."⁴¹⁶ Other studies have found no association between THC and crash risk but have been discounted by researchers because of some unusual aspect of study design.⁴¹⁷

413. Poulsen, *supra* note 363, at 125 tbl. 5.

414. *Id.*

415. Longo, *supra* note 369, at 626. Others have criticized Longo's study for using a THC cut-off amount that is much too high (40 ng/mL) to capture low levels of THC in drivers. *See, e.g.*, P. Mura et al., *Comparison of the Prevalence of Alcohol, Cannabis and Other Drugs Between 900 Injured Drivers and 900 Control Subjects: Results of a French Collaborative Study*, 133 *FORENSIC SCI. INT'L* 79, 80 (2003).

416. K.W. TERHUNE ET AL., NHTSA, THE INCIDENCE AND ROLE OF DRUGS IN FATALLY INJURED DRIVERS 65 (1992), available at http://ntl.bts.gov/lib/26000/26600/26685/DOT_HS_808_065.pdf.

417. *See, e.g.*, Drummer et al., *supra* note 381, at 240 (reviewing previous culpability studies, including one, Lowenstein and Koziol-Mclain (2001), that found increased crash risk only for alcohol and alcohol in combination with drugs, but used only urine testing for THC; and Williams et al. (1985), which found no association "but the numbers of drivers were small"); Longo, *supra* note 369, at 624 (noting that Terhune found a decreased crash risk for THC alone, and that most previous studies had very small sample sizes for THC alone); *see also* Grotenhermen et al., *supra* note 325, at 17–20 (finding, in a meta-analysis of existing studies, no association between increased accident risk and cannabis blood levels below 10 ng/mL).

Other culpability studies have claimed a positive association between THC-only drivers and increased crash risk, but the inference of causality from each of them is questionable. The two most cited are Drummer (2004) and Laumon (2005). Of the 3,398 fatally injured drivers in Drummer's Australian study, just fifty-eight had only THC in their blood, and only nine had THC levels below 5 ng/mL; the median was 12 ng/mL.⁴¹⁸ With this small sample, Drummer calculated a .7 OR for THC concentrations below 5 ng/mL, and a 6.6 OR for the vast range between 5 and 100 ng/mL.⁴¹⁹ He acknowledged the possibility of confounding factors, the fact that the ORs did not "imply a similar increase in [relative] crash risk," and that "[n]either the size nor the statistical significance of the associations we observed can be used to directly infer causality."⁴²⁰ Other researchers have tried to extrapolate what a relative crash risk might be at THC levels of 6–20 ng/mL based on Drummer's data set and claim to have found a slightly higher chance of a crash (compared to a sober driver) at levels above 6 ng/mL.⁴²¹ But researchers seem in agreement that extrapolations based on such miniscule sample sizes are not a "statistically acceptable basis" for drawing conclusions about blood levels and crash risk.⁴²²

Laumon's culpability study on over 10,000 drivers involved in fatal crashes in France from 2001–03 suffered from similar flaws.⁴²³ The samples were not taken until three to four hours after the incident, and some drivers with low but non-zero BACs were listed as "null BAC," thus resulting in over reporting the number of "THC-only" cases.⁴²⁴ Moreover, the authors do not appear to have separated the THC-only cases from drivers with THC and other drugs in their system.⁴²⁵ A 2012 metastudy of nine case-control or culpability studies chosen for their use of blood-only samples and testing for THC and its active metabolite found that drivers with "acute cannabis consumption" were twice as likely as sober drivers to be in a fatal crash, but noted that "the studies in our review did not have enough data on [THC] concentration to examine dose-response effects."⁴²⁶

418. Drummer et al., *supra* note 381, at 244.

419. *Id.*

420. *Id.* at 245–46.

421. See Grotenhermen et al., *supra* note 325, at 1912.

422. See *id.* at 1912 fig. 1. The researchers, who shared their unpublished estimations in personal communications with Grotenhermen, estimated an OR of 2 at 8 ng/mL, up to an OR of nearly 7 at 13 ng/mL. *Id.*

423. See Bernard Laumon et al., *Cannabis Intoxication and Fatal Road Crashes in France: Population Based Case-Control Study*, 331 *BMJ* 1371 (2005).

424. See Grotenhermen et al., *supra* note 325, at 1913.

425. See Laumon et al., *supra* note 423, at 1375 (separating BAC-negative drivers out from THC-positive drivers, but apparently not those testing positive for other drugs).

426. Asbridge et al., *supra* note 411, at 45. One additional culpability study, Bédard (2007), found an association between cannabis and increased crash risk. They looked to FARS data on fatally injured drivers from 1993 to 2003 (N=314,636), used citation for a driving infraction (like speeding) as a proxy for culpability, and found that cannabis-positive (and BAC-negative) drivers had a modestly higher OR of 1.29. The authors did not, however, separate THC-only cases from polydrug

The following graphs juxtapose Borkenstein and Blomberg et al.’s data on BAC and relative crash risk with the data from the eleven case-control and culpability studies on marijuana that separate THC from other drugs (those eight studies with straight lines offer only one relative risk estimate for THC without measuring or distinguishing precise blood levels):

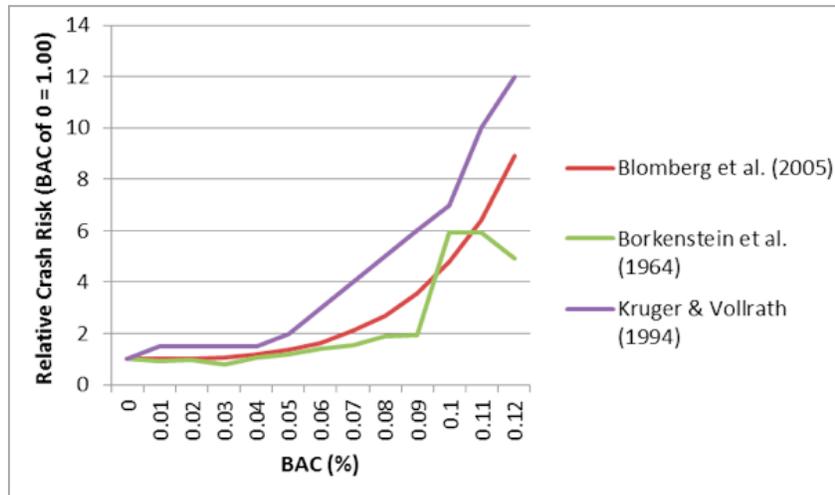


TABLE 4. BAC and relative crash risk, from Borkenstein et al., Krüger & Vollrath, and Blomberg et al. data⁴²⁷

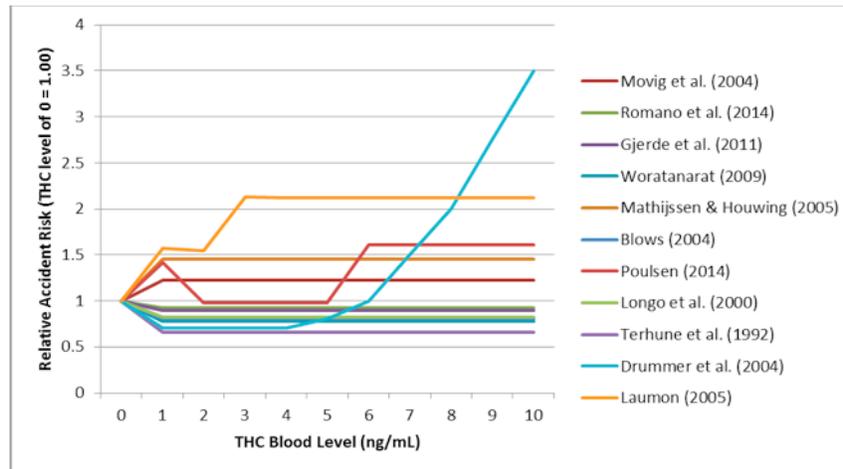


TABLE 5. THC blood levels and relative crash risk, from all case-control and culpability studies offering results for THC-only⁴²⁸

cases, or THC from its metabolites, and gave no blood level data. Michel Bédard et al., *The Impact of Cannabis on Driving*, 98 CAN. J. PUB. HEALTH 6, 8–10 (2007).

427. The data points are reproduced, with permission, from BLOMBERG ET AL., *supra* note 215, at xviii, tbl. ES-1.

The differences between the graphs are almost cartoonishly stark. The THC study results are inconsistent and generally not level-specific. Six of the studies indicate a *decrease* in crash risk at low levels of THC, compared to sober drivers.

And the only line showing any significant increase at levels higher than 6 ng/mL, Drummer et al. (2004), is an unpublished extrapolation from a study with a concededly insignificant sample size and an acknowledged possibility of confounding factors.⁴²⁹ In short, based on current studies, there is simply no established predictable or linear relationship between THC blood levels and relative risk of crash.

d. Simulated and On-Road Concentration-Specific Driver Impairment Studies

As the scientists studying alcohol recognized, the most probative evidence of dangerous impairment comes from epidemiological studies of crashes, not impairment studies. For one thing, impairment studies' "sensitivity to pharmacological drug effects" is much higher than that of crash risk studies; hence, while alcohol impairment studies show widespread impairment at BACs as low as .02 percent, crash risk does not predictably increase until at least .05 percent BAC.⁴³⁰ Still, impairment studies can help confirm and explain the causal link between drug use and crashes. Alcohol-related studies of impairment were particularly probative because subjects could ethically be given a wide range of doses of the drug and could drive in realistic conditions, because the dosage of alcohol corresponded predictably to level of BAC, and as BAC rose, impairment effect with respect to specific driving-related skills rose as well.

In contrast, THC impairment studies suffer from several inherent difficulties, mirroring the difficulties in establishing any predictable relationship between THC blood levels and crash risk. First, while there have been scores of studies of THC and cognitive and psychomotor impairment, nearly all have been dosage-related (the amount of THC given to the subject to ingest) rather than blood-level-related (the amount of THC the subject has in

428. Some studies expressed their results in terms of odds ratios; others in terms of relative risk. Others treat the two as interchangeable (the relative risk and odds ratio approach the same value when the absolute crash risk is low). I calculated the relative risk for THC-only from Terhune (1992) based on the reported percentage of culpable controls and culpable THC-only drivers (67.7 percent versus 57.9 percent) and the estimated relative risk equation used by Terhune listed at TERHUNE ET AL., *supra* note 416, at 78. To give the benefit of the doubt to the culpability studies, I have also included the attempts documented in Grotenhermen (2007) to extrapolate data from Drummer (2004) to levels from 6-20 ng/mL and have included the Laumon (2005) data even though it is not clear that the THC-only ORs do not also include polydrug use.

429. See, e.g., Grotenhermen et al., *supra* note 325, at 1912 & fig. 1.

430. J.G. Ramaekers et al., *Cognition and Motor Control as a Function of Delta 9-THC Concentration in Serum and Oral Fluid: Limits of Impairment*, 85 *DRUG & ALCOHOL DEPENDENCE* 114, 120-21 (2006).

her blood), and the dosages given to subjects have created widely varying blood levels.⁴³¹ Second, frequent cannabis users are systematically underrepresented in such studies, potentially underestimating tolerance effects.⁴³² Third, blood levels vary widely in the level of cognitive and psychomotor effect to which they correspond. Two comprehensive metastudies on THC and impairment concluded that the “relationship between THC [blood levels] and street driving performance is equivocal”⁴³³ and that “[t]he debate” over marijuana and impairment “is complicated by the temporal dissociation of THC concentrations from acute driving impairment.”⁴³⁴

Even within the inherent limits of dosage studies, the impairing effects vary greatly depending on the person and the skill involved. Some dosage studies show minimal to no impairment effect in most people at low to medium doses of THC.⁴³⁵ One study concluded that after “about three hours,” the impairment effects of even a “strong social dose” of 15–20 mg are “comparable to a BAC of less than .03 percent.”⁴³⁶ And while alcohol tends to affect “complex driving functions” (like anticipation of traffic or reaction time) the most and “automatic” functions (like tracking performance) least, THC tends to do the opposite.⁴³⁷ Thus, while “automatic” skills in one study were affected by dosages of, say, 6 milligrams of THC, more “complex” skills remained unaffected by dosages of up to 18 milligrams,⁴³⁸ an amount equal to about two sellable marijuana “edibles” in Colorado.⁴³⁹ One hypothesis for this difference

431. See, e.g., M.G. Lenne et al., *The Effects of Cannabis and Alcohol on Simulated Arterial Driving: Influences of Driving Experience and Task Demand*, 42 ACCIDENT ANALYSIS & PREVENTION 859, 861 (2010).

432. See, e.g., Khiabani et al., *supra* note 382, at 111 (noting that experimental THC impairment studies might underestimate tolerance effects because study subjects are almost always infrequent users).

433. Erin Kelly et al., *A Review of Drug Use and Driving: Epidemiology, Impairment, Risk Factors and Risk Perceptions*, 23 DRUG & ALCOHOL REV. 319, 326 (2004).

434. Hartman & Huestis, *supra* note 408, at 483.

435. See, e.g., C.T.J. Lamers & J.G. Ramaekers, *Visual Search and Urban City Driving Under the Influence of Marijuana and Alcohol*, 16 HUM. PSYCHOPHARMACOLOGY: CLINICAL & EXPERIMENTAL 393 (2001) (finding that doses of 100 µg/kg of THC had minimal effect on nighttime city driving).

436. GROTENHERMEN ET AL., *supra* note 285, at 6.

437. *Id.*; see also Anthony Liguori et al., *Separate and Combined Effects of Marijuana and Alcohol on Mood, Equilibrium and Simulated Driving*, 163 PSYCHOPHARMACOLOGY 399, 404 (2002) (noting no effect of THC dosages on brake latency, unlike alcohol); Annick Menetrey et al., *Assessment of Driving Capability Through the Use of Clinical and Psychomotor Tests in Relation to Blood Cannabinoids Levels Following Oral Administration of 20 mg Dronabinol or of a Cannabis Decoction Made with 20 or 60 mg Delta-9-THC*, 29 J. ANALYTICAL TOXICOLOGY 327, 336 (2005) (finding no effect on reaction time in driving test after 20 and 60 mg THC doses, but finding an effect on tracking performance).

438. GROTENHERMEN ET AL., *supra* note 285, at 6.

439. See, e.g., Keith Coffman, *Colorado's Edible Pot Facing Proposed Tighter Rules on Packaging, Potency*, WASH. POST, Aug. 3, 2014, http://www.washingtonpost.com/local/colorados-edible-pot-facing-proposed-tighter-rules-on-packaging-potency/2014/08/03/473643be-1b4d-11e4-ae54-0cfe1f974f8a_story.html.

is that, unlike drunk drivers' tendency to underestimate risk, THC-intoxicated drivers overcompensate for a known risk by slowing down or keeping distance.⁴⁴⁰

This difference between impairment of automatic and complex skills was also observed in the most realistic study to date, one that would make Herman Heise himself blush. Researchers gave subjects placebos or a mix of ethanol, orange juice, and Grand Marnier, followed by a marijuana joint sixty minutes later, and sent them off into urban traffic with a guide who had concurrent controls over the car.⁴⁴¹ They found that while the THC-alcohol mix caused significant impairment of certain skills, a seven to twenty-one milligram dosage of THC lacked any "appreciable effect[]" on anything but tracking performance, and a higher dosage "had an effect that only approached significance" on skills other than tracking.⁴⁴²

A handful of tests have looked to THC blood levels rather than simply dosage, but have not found consistent or significant impairment in most users. One metastudy in 1998 found that even at levels of 60–70 ng/mL of THC in blood serum (meaning about 30–35 ng/mL in whole blood), fewer than half of subjects experienced "significant" impairment in all categories of driving-related skills. While the percentage was higher at slightly lower serum levels, the relationship was not linear.⁴⁴³ Papafotiou et al. (2005) found that subjects who smoked a marijuana cigarette were significantly impaired eighty minutes (but, curiously, not thirty minutes) after ingestion, but found that "the *level of THC in the blood* does not provide an accurate and reliable indicator of whether driving performance is impaired."⁴⁴⁴ Laboratory tests, while not as probative as more realistic impairment studies, similarly indicate no predictable relationship between blood levels and impairment levels.⁴⁴⁵ Moreover, studies

440. See, e.g., Grotenhermen et al., *supra* note 325, at 1915.

441. See J.G. Ramaekers et al., *Marijuana, Alcohol, and Actual Driving Performance*, 15 HUMAN PSYCHOPHARMACOLOGY: CLINICAL & EXPERIMENTAL 551, 552 (2000). The joints had 100–200 µg/kg, which corresponds to 7–14 mg. Hartman & Huestis, *supra* note 408, at 487.

442. Ramaekers et al., *supra* note 441, at 554; see also H. Robbe, *Marijuana's Impairing Effects on Driving Are Moderate When Taken Alone But Severe When Combined with Alcohol*, 13 HUMAN PSYCHOPHARMACOLOGY: CLINICAL & EXPERIMENTAL S70, S77–S78 (1998) (finding that a 7–21 mg dose of THC had a "small" or "moderate" effect, mostly on road tracking, and concluding that "it is not possible to conclude anything about a driver's impairment on the basis of his/her plasma concentrations of THC" or its metabolites).

443. GROTENHERMEN ET AL., *supra* note 285, at 28 fig. 5.

444. K. Papafotiou, *The Relationship Between Performance on the Standardised Field Sobriety Tests, Driving Performance and the Level of Delta-9-Tetrahydrocannabinol (THC) in Blood*, 155 FORENSIC SCI. INT'L 172, 178 (2005) (emphasis added).

445. See, e.g., Ginsburg et al., *supra* note 307, at 7 (concluding that "the lack of impairment . . . at early time points after THC administration (when blood levels were highest) indicates that blood levels of THC may not be an appropriate instantaneous measure of impairment at any threshold" (emphasis added)); Grotenhermen et al., *supra* note 325, at 1913 (noting studies showing that while driving instructors rated subjects at .04 percent BAC as "impaired," police rated subjects who had consumed 7 mg of THC as "unimpaired" and subjects at .08 percent BAC as "more impaired" than the cannabis users); Khiabani et al., *supra* note 382, at 112 (finding that in a test of 589 THC-only drivers

have consistently shown that THC blood levels in chronic users can and often do remain constant, and over 5 ng/mL, for days or even weeks after last use.⁴⁴⁶

While these inconclusive results on impairment do not prove that THC-positive drivers are safe at all blood levels, at the very least they suggest that marijuana as chemical impairment, if it is to be scientifically proven at all, will have to be proven first and foremost through epidemiological studies of crashes.

III.

THE ROAD AHEAD: CRAFTING LEGITIMATE DRUGGED DRIVING LAWS UNDER A SCIENCE-BASED JURISPRUDENCE OF DANGEROUSNESS

Until lawmakers and the public understand the DUI alcohol story, they are doomed to deploy inapt analogies to it in ways that do violence to the scientific ethic of the Haddon era, the decades of research underlying the justification for the per se DUI alcohol regime, and the principles of just punishment. The first step on the road to a legitimate chemical impairment law for marijuana, then, will be to ensure that policy makers appreciate the difference between choosing a jurisprudence of prohibition and a jurisprudence of dangerousness, accept the fact that per se limits for legal drugs must be justified in terms of the scientifically demonstrated link to dangerous driving, and understand the significance of single-car and case-control crash risk studies in connecting specific drug concentrations to specific levels of risk.

The dire consequences of maintaining an illegitimate per se DUI marijuana regime are twofold. The first is, simply, injustice. Take, for example, the obviously sympathetic cases of medical marijuana patients who wake up in the morning at 10 ng/mL and will be unfairly labeled as criminals if they are stopped, tested, and prosecuted for DUI. But even putting these cases aside, drivers who smoke and drive—like drivers who drink and drive—are simply not worthy of criminal punishment under a jurisprudence of dangerousness unless they are actually dangerous. Whether one is a retributivist or utilitarian, punishment without purpose is immoral. The consequences of any DUI conviction are grave, affecting not only physical liberty but potentially

stopped for suspected drugged driving, most passed all physician-administered clinical tests for impairment, including 43 percent of drivers at greater than 10 ng/mL); Amy J. Porath-Waller & Douglas J. Beirness, *An Examination of the Validity of the Standardized Field Sobriety Test in Detecting Drug Impairment Using Data from the Drug Evaluation and Classification Program*, 15 TRAFFIC INJURY PREVENTION 125, 129 (2014) (citing studies showing no effect of cannabis on FSTs, including “walk and turn” (WAT) or horizontal gaze nystagmus (HGN) tests, except the “one leg stand”); Ramaekers et al., *supra* note 430, at 119 (finding that “linear relations” between THC serum levels and impairment levels existed but “were rather low” and that the “lack of a strong association seems to indicate that serum THC cannot be taken as an accurate predictor of the magnitude of performance impairment”).

446. See, e.g., M.M. Bergamaschi et al., *Impact of Prolonged Cannabinoid Excretion in Chronic Daily Cannabis Smokers' Blood on Per Se Drugged Driving Laws*, 59 CLINICAL CHEMISTRY 519 (2013); Ginsburg et al., *supra* note 307, at 2, 3.

opportunities for housing, voting, juror service, gun ownership, and employment.

One could imagine the cynical response that, even if a 5 ng/mL DUI marijuana law has no legitimate penal purpose, lawmakers must sometimes “ris[e] above principle”⁴⁴⁷ for the sake of tackling an otherwise intractable social ill like drugged driving. Even assuming the value of such an approach in the abstract, its logic in the marijuana context is as shortsighted as it is specious. First, the question whether dangerous driving from marijuana impairment is a prevalent problem to begin with is left unanswered by the current unscientific approach to studying the relationship between cannabis and crash risk. Second, the more our criminal law deviates from legitimate bases for punishment and the more the definition of criminality brings average citizens into the fold, the more jurors will acquit and the more the penal regime will appear both ineffective and illegitimate.

Most fundamentally, to the extent DUI marijuana is indeed a burgeoning public health crisis, settling for an unscientific approach hinders the search for real solutions to that crisis. In a world in which the only goal of state officials is to generate evidence of THC blood levels for successful DUI marijuana prosecutions, rather than to truly understand the connection between blood levels, impairment, and crash risk, critical research will never be done. For example, the future holds the possibility of improving research using a breath test for THC that might have a shorter window of detection and allow quicker sampling of stopped motorists,⁴⁴⁸ but such a test is still in its early experimental phase.⁴⁴⁹ Researchers are also working to develop tests to more accurately indicate timing and extent of drug use from saliva, sweat, and hair samples.⁴⁵⁰ Others have suggested the use of “pupillometry” and eye tracking⁴⁵¹ to better measure drug impairment, though such methods are currently limited by a

447. See Geoffrey C. Hazard, Jr., *Rising Above Principle*, 135 U. PA. L. REV. 153 (1986).

448. See HELEN N. TASHIMA & SCOTT V. MASTEN, CAL. DEP’T MOTOR VEHICLES, AN EVALUATION OF FACTORS ASSOCIATED WITH VARIATION IN DUI CONVICTION RATES AMONG CALIFORNIA COUNTIES X (2011), available at http://apps.dmv.ca.gov/about/profile/rd/r_d_report/Section_3/S3-235.pdf (“There are no devices available like hand-held alcohol breathalyzer devices to detect drugs.”); M.L. Chipman et al., *Being “At Fault” in Traffic Crashes: Does Alcohol, Cannabis, Cocaine, or Polydrug Abuse Make a Difference?*, 9 INJURY PREVENTION 343, 343 (2003) (noting that “[u]ntil a test of breath or saliva, analogous to the breath tests for alcohol, exists for other substances,” reliable case-control studies will be difficult to perform).

449. See Sarah K. Himes et al., *Cannabinoids in Exhaled Breath Following Controlled Administration of Smoked Cannabis*, 59 CLINICAL CHEMISTRY 1780 (2013).

450. NHTSA REPORT (2010), *supra* note 273, at 5; see also Ramaekers et al., *supra* note 430, at 118–19 (confirming a strong correlation between THC serum and oral fluid levels and that oral testing might hold promise for “easy-to-use, non-invasive roadside drug tests” in the future).

451. Wallace B. Pickworth & Rudy Murillo, *Pupillometry and Eye Tracking as Predictive Measures of Drug Abuse*, in PHARMACOKINETICS AND PHARMACODYNAMICS OF ABUSED DRUGS 127, 130, 140 (Steven B. Karch ed., 2007) (finding that “several classes of abused drugs,” including smoked marijuana, have “specific, dose-related effects” on “pupil size and measures of the light reflex”).

number of confounding factors.⁴⁵² Better roadside surveys could also be developed to give a valid baseline for case-control studies of THC levels and crash risk, such as the voluntary roadside swab program for suspected drugged drivers begun in 2014 by the Los Angeles City Attorney and Police Department.⁴⁵³ And drug impairment prosecutions could benefit from increased use of dashboard cameras and body-worn cameras (BWCs) in DUI patrol cars, to corroborate officer testimony and FSTs. Finally, quicker and more accurate data collection after fatal car crashes—particularly single-car crashes—will be critical to studying marijuana as chemical impairment through Haddon’s framework.⁴⁵⁴

Once better roadside surveys and better data collection after crashes are available, researchers will be able to conduct the type of case-control studies that Borkenstein accomplished in the Grand Rapids Study. Specifically, case-control studies in the future should have the following characteristics to adhere to the Haddon framework:

- Blood testing rather than oral fluid or urine testing, to avoid artificially low THC-blood estimates, until a suitably reliable alternative testing method is developed;
- Large enough sample sizes in both control and driver populations to capture a statistically significant group of THC-only drivers;
- Randomly selected control driver population subject to the same conditions (time of day, etc.) as the fatal/injured driver population;
- Testing of THC alone, without its two metabolites, and without combination with alcohol or other drugs;
- Differentiation of blood levels up to a high enough number to reveal relative crash risk at low, moderate, and high levels; and
- Adjustment for remaining confounding factors such as age and sex.

Settling for an unprincipled criminal law also blinds lawmakers to an additional option for addressing drugged driving beyond current criminal impairment laws: the regulatory state. If lawmakers truly believe that low levels of THC cause impairment worthy of regulation, but cannot prove through science that blood levels can be linked through crash risk data to

452. The “practical utility” in using pupil size and light reflex to detect recent ingestion of drugs is limited by substantial variability among persons, the “small and transient” effect of the drugs, the level of ambient light in the surrounding area at the time of measurement, and the effect of other physical conditions such as fatigue, disease, or legal drug use. *Id.* at 139.

453. Melissa Pamer, *LAPD Highlights New Roadside Swab Drug Test*, KTLA5 NEWS (Dec. 27, 2013, 1:52 PM), <http://ktla.com/2013/12/27/lapd-highlights-new-roadside-swab-drug-test/>.

454. See, e.g., Berning & Smither, *supra* note 289, at 3 (“As more complete data becomes available, FARS data on drug-involved driving will be strengthened.”).

morally blameworthy behavior, then regulatory law, rather than penal law, might provide a legitimate means to tackle the issue. After all, early reports from the medical profession on the hazards of drugged driving focused not on the need to criminalize or even regulate such behavior, but simply on the need to curb it through sound advice from drug users' physicians.⁴⁵⁵ For what it is worth, even some of the sponsors of per se bills appear to think that is what they are really trying to do. As one Colorado legislator curiously insisted in defending that state's permissive inference of criminal guilt at 5 ng/mL, "This is a regulatory bill, not a criminal justice bill."⁴⁵⁶ If that is indeed the purpose of such laws, then their consequences should be changed to match their justification.

CONCLUSION

Per se THC-limit DUI marijuana laws, as currently conceived, serve no legitimate penal purpose. They are not justified under a jurisprudence of prohibition because they do not seek to punish the immorality of possessing marijuana. In the growing number of states in this country where marijuana is legal or decriminalized, the jurisprudence of prohibition is simply unavailable as a justification for criminal punishment of the simple act of driving with some amount of marijuana in one's body.

Such laws are also not justified under a jurisprudence of dangerousness. As this Article has uncovered, what makes the emptiness of the dangerousness justification so obvious in the marijuana context is the detailed history of alcohol science and the drunk driving jurisprudence built on that science. Properly understood, this history—William Haddon's legacy—becomes the compulsory agenda for using the criminal law as an instrument of public safety. Instead of adhering to Haddon's framework, states with per se THC limits have focused solely on finding a number—any number—to analogize to DUI alcohol's .08 percent BAC limit, without first choosing a legitimate purpose for punishing drugged driving and finding a definition of the crime that serves that purpose. State officials have forgotten that the venerable .08 percent limit itself was the result of decades of careful single-car crash studies and case-control studies showing a predictable and significant relationship between BAC and crash risk. Even then, .08 percent was controversial and arguably on the edge of what the American public was willing to condemn through the criminal law. In the marijuana context, single-car crashes and case-control studies are rare and, to the extent they exist, suggest no predictable relationship between THC

455. See, e.g., Carlos J.G. Perry & Alan L. Morgenstern, Comm. on Medical Aspects of Automotive Safety & Council on Drugs, *Drugs and Driving*, 195 JAMA 376 (Jan. 31, 1966).

456. See Eli Stokols, *Lawmakers Finally Send Marijuana DUID Measure to Hickenlooper*, FOX31 DENVER NEWS (May 6, 2013, 1:23 PM), <http://kdvr.com/2013/05/06/monday-afternoon-floor-fight-looms-on-duid-bill/> (quoting Colorado State Senator Ted Harvey).

blood levels and crash risk. In short, the illegitimacy of per se THC limits as an attempted analog to .08 percent is not a close call.

Revealing the lack of science behind per se DUI marijuana laws is not only important in its own right, as a critique of an otherwise soon-to-be ubiquitous criminal regime in this country, but also as a case study revealing the perils of technology as a potentially distorting force in modern criminal law. Per se THC limits are prime examples of what James Scott described as “heroic simplification” of a social problem by the state.⁴⁵⁷ In the administrative context, this distortion has come in the form of “policies involving simple questions and answers that are easy to translate into code, even when strong substantive reasons favor a more nuanced approach.”⁴⁵⁸ In the abortion context, such simplification arguably came in the form of what many scholars describe as the “morally arbitrary” line of viability, a bright line borrowed from the scientific community that changed as technology improved and that the Court never seemed to treat as urgently in need of a connection to legal principle.⁴⁵⁹

Likewise, machines like the Drunk-O-Meter transform what was once a complex jury judgment about a driver’s “impairment” into a simple machine judgment about BAC. The availability of the machine uniquely allowed state officials to reduce the problem of impairment to an easily determinable element. As Jonathan Simon has suggested, simplicity in service to the state is especially problematic in criminal justice, given the ever-present possibility that “prosecutorial and judicial procedures have been smoothed or simplified to eliminate possible sources of resistance.”⁴⁶⁰ By focusing single-mindedly on making DUI marijuana convictions easier through machine results comparable to the Drunk-O-Meter and its progeny, rather than focusing on the legitimacy of punishment, public officials have “allowed technical knowledge, somewhat arbitrarily, to dictate the path of justice.”⁴⁶¹

This is not to say that drugged driving is not a public health problem nor that the criminal law has nothing to offer as a means of combating it. With better detection methods and more comprehensive data collection on crash-involved drivers and dose-specific drug prevalence in the overall driving population, it might be that we will find a scientific way to measure marijuana as chemical impairment. Or, at least, we might find a way to identify a

457. JAMES C. SCOTT, *SEEING LIKE A STATE: HOW CERTAIN SCHEMES TO IMPROVE THE HUMAN CONDITION HAVE FAILED* 8 (1998).

458. Danielle Keats Citron, *Technological Due Process*, 85 WASH. U. L. REV. 1249, 1255 (2008).

459. See, e.g., Randy Beck, Gonzales, Casey, and the Viability Rule, 103 NW. U. L. REV. 249 (2009) (discussing the “morally random results the [viability] rule produces”).

460. Jonathan Simon, *Uncommon Law: America’s Excessive Criminal Law & Our Common-Law Origins*, 143 DAEDALUS 62, 63 (2014).

461. Bernard E. Harcourt, *Against Prediction: Sentencing, Policing, and Punishing in an Actuarial Age* 3 (Chicago Pub. Law & Legal Theory Working Paper No. 94, 2005), available at http://papers.ssrn.com/sol3/papers.cfm?abstract_id=756945.

numerical threshold high enough to ensure no “false positives” but low enough to detect at least some intoxicated drivers who might be acquitted under a subjective impairment standard. If not marijuana, then we might be able to measure other illicit and prescription drugs that turn out to have a more predictable relationship between dosage or measurable amount in the body and risk of harm.

But if marijuana is truly impossible to measure in a way that allows for a coherent per se DUI law in the foreseeable future based on THC blood levels, then the answer should not be to settle for incoherency in our criminal law in the name of securing more convictions. The answer should be to settle for an imperfect subjective impairment standard for criminal DUI marijuana laws and to use numerical thresholds, if at all, to trigger nonpunitive regulatory sanctions. In any event, we should be guided by science in tackling the issue of drugged driving, whether our goal is to achieve justice, to promote public safety, or to have the audacity to try to do both.

