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# Determinism v. Free Will & Genetic Evidence of Addiction in Plea Bargaining and Sentence Mitigation: Conversion of Incarceration to Probation and Rehabilitation Based on Genetic Addiction Risk Severity (GARS) Test

Kenneth Blum

Paul Mullen

Richard Green

# ARTICLE

# DETERMINISM V. FREE WILL & GENETIC EVIDENCE OF ADDICTION IN PLEA BARGAINING AND SENTENCE MITIGATION: CONVERSION OF INCARCERATION TO PROBATION AND REHABILITATION BASED ON GENETIC ADDICTION RISK SEVERITY (GARS) TEST

**KENNETH BLUM, PAUL M. MULLEN, & RICHARD GREEN**[*]



[*] Kenneth Blum, Division of Addiction Research & Education, Center for Exercise, Sports & Mental Health, Western University of the Health Sciences, Pomona CA, USA; Department of Molecular Biology, Adelson School of Medicine, Ariel University, Ariel, Israel. Paul Mullen, The University of Southampton, Southampton, UK (LL.M., 2020); The University of Maine School of Law, Portland, ME (JD, 2019). Richard Green, Precision Translational Medicine, LLC., San Antonio, TX, USA. The authors also thank David Baron, Raymond Brewer, John B. Floyd, Thomas Simpatico, David Smith, and A. Kenison Roy III for their invaluable contributions to the research and writing of this article.

ABSTRACT

***Objectiv*e:** The legal system presumes free will and imputes criminal responsibility, but also allows for the uncontrollable influence of determinism by providing exculpatory defenses or by mitigating resulting punishment.[1] This concept is considered an area of change that requires thoughtful consideration in the absence of legal precedent. Behavioral genetic evidence has been used in criminal defense with mixed results.[2] The real-world case presented in this Article (and several others previously pled in Bexar County) show quite clearly that judges and prosecutors are open to genetic evidence of alcoholic propensity in favoring treatment over incarceration for felony Driving While Intoxicated (DWI) offenders with clinically diagnosed Alcohol Use Disorder (AUD). ***Method:*** This case study is of a thirty-five-year-old male (AG) in sustained remission from active alcohol use with five DWI convictions and a previous three-year incarceration for a past DWI conviction. AG completed intensive out-patient treatment for AUD and was under continuing care and supervision for AUD. The legal brief presented to the court indicated a genetically induced hypodopaminergic, that is, a dopamine deficiency, dysfunction

1. *See* Murray L. Schwartz, Book Review, 21 STAN. L. REV. 1277, 1279–82 (1969) (reviewing HERBERT L. PACKER, THE LIMITS OF THE CRIMINAL SANCTION (1968)) (outlining the rationale, process, and limits of the legal system).

2. *Compare* Nicholas Scurich & Paul S. Appelbaum, *Behavioural Genetics in Criminal Court*, 1 NATURE HUM. BEHAV. 772, 772 (2017) (finding the introduction of genetic evidence in violent criminal cases "is ineffective at reducing judgments of culpability and punishment, and therefore its use in the legal process is likely to diminish."), *with* Daniela Guillen Gonzalez, et al., *Neuroscientific and Genetic Evidence in Criminal Cases: A Double-Edged Sword in Germany but Not in the United States?*, 10 FRONT. PSYCHOLOGY 2343(2019), https://www.frontiersin.org/articles/10.3389/fpsyg.2019.02343/full [https://perma.cc/3KW5-V9FX] (positing—based on a sample of several hundred surveyed law students—that American judges would find neurobiological and genetic explanations of psychopathy mitigating, but German judges would not), *and* Deborah W. Denno, *Courts' Increasing Consideration of Behavioral Genetics Evidence in Criminal Cases: Results of a Longitudinal Study*, 2011 MICH. ST. L. REV. 967 (2011) (asserting—based on review of eighty-one criminal cases from 1994–2011—" behavioral genetics evidence has no decipherable impact on a defendant's case or, at most, it becomes an effective tool along with a range of other kinds of variables in rendering a defendant ineligible for the death penalty.").

based on the defendant's genetic profile. ***Result:*** The prosecution and the presiding judge reviewed the brief and the evidence of AUD treatment, and in lieu of a custodial prison sentence, proposed ongoing treatment and monitoring. As a result of the brief and discovery, the adjudication was for five years mandated standard probation, fines, community service, monitoring, and ten nights in the county jail on work release. ***Conclusion*:** This new, consequential, and innovative legal precedent utilized genetic information to abrogate incarceration and accept rehabilitation in the face of genetic determinism.

***Public Significance Statement*:** We believe we have found precedential evidence in criminal court cases to persuade courts in choosing rehabilitation instead incarceration for DWI recidivism, specifically, probation, treatment for Substance Use Disorder (SUD) or AUD, and continued monitoring of treatment, such as rehabilitation, as an alternative to traditional sentencing.

The determining factors for eligibility for alternative sentencing are substantially based on a newly patented Genetic Addiction Risk Score (GARS) test. This is the first case study to objectively point to genetically-based "determinism" rather than "free will" in determining sentencing for a DWI offender.

*Keywords*: Judicial System, Genetic Addiction Risk Score (GARS), Reward Deficiency Syndrome (RDS), Substance Use Disorder (SUD), Alcohol Use Disorder (AUD), hypodopaminergia, Driving While Intoxicated (DWI)

*"We used to think that our fate was in the stars. Now we know, in large measure, that our fate is in our genes."*

—James Watson[3]

## I. INTRODUCTION

Today, genetic technology has evolved and is used every day in the American legal system. Thirty years ago, using DNA evidence to determine innocence or guilt was practically unheard of. Now, following the completion of the Human Genome Project, new defense plans hold the promise of a defense based on DNA linked to a medically accepted disorder that implies genetic culpability. At the same time, civil courts, prosecutors, and defenders rely on DNA as a rock-solid, indisputable source of evidence.[4]

Legal defenses based on genetic culpability for psychiatric disorders like alcohol use disorders (AUD) are practical replacements for the often unsuccessful insanity defense.[5] Modern "genetic theory" posits that genes and environment determine human behavior, with genetic factors being "the first stage of the causal sequence."[6] The primary argument against rehabilitation is the notion that, for example, rehabilitation of a convicted predator is not possible.[7] However, therapeutic models show significant improvement, reasonable recovery rates from AUD, and better clinical outcomes for individuals with addiction risk polymorphisms and compulsions correlated with those markers.[8]

3. Leon Jaroff, *The Gene Hunt*, TIME, March 20, 1989 at 62, 67.

4. *See* Brooke G. Malcom, *Convictions Predicated on DNA Evidence Alone: How Reliable Evidence Became Infallible*, 38 CUMB. L. REV. 313, 315 (2007) ("A major concern. . . is whether the significance of DNA has been overestimated by courts and jurors.").

5. Dawinder S. Sidhu, *Criminal Law x Addiction*, 99 N.C. L. Rev. 1083, 1108–10 (2021).

6. David C. Rowe & D. Wayne Osgood, *Heredity and Sociological Theories of Delinquency: A Reconsideration*, 49 AM. SOCIO. REV. 526, 527 (1984).

7. *See* David Lebowitz, *Proper Subjects for Medical Treatment - Addiction, Prison-Based Drug Treatment, and the Eighth Amendment*, 14 DEPAUL J. HEALTH CARE L. 271, 284 (2012) (("[A]ddicts are neither mechanistically coerced from within to behave in a certain way nor divested of their reasoning capabilities in a manner that prevents them from responding to rational incentives." (citing Stephen J. Morse, *Addiction, Genetics, and Criminal Responsibility*, 69 L. & CONTEMP. PROBS. 165, 176 (2006))).

8. *See generally* Mark P. McGovern & Kathleen M. Carroll, *Evidence-Based Practices for Substance Use Disorders*, 26 PSYCHIATRIC CLINICS N. AM. 991, 991 (2013) (concluding behavioral treatment can be an effective option for addiction-based disorders).

### A. *Nature vs. Nurture: Excuse or Explanation*

Classification of criminality in the past used everything from race to physical features and body structure.[9] However, since the discovery of DNA evidence, genetic predisposition towards crime has become a predominant factor in the classification of criminality. Early advocates of "biological criminology" were, for a time, overshadowed by proponents of environmental determinism who viewed socially deviant behavior as the result of the molding effects of environmental forces rather than as a physiological function.[10] Today, the environmental impact of social and cultural influences on human behavior coupled with the recent developments in genetics and related fields have prompted reconsideration by criminologists of some forms of antisocial behavior as manifestations of physiological dysfunction.[11] Lawrence Taylor has called this being born to crime.[12]

The genesis of psychiatric genetics occurred in 1990.[13] Research and development in this field led to the formula, Genetics + Environment = Phenome (G+E=P). Along these lines, the modern "genetic theory"[14] posits that genetic and environmental variation produce behavior to which genotype gives an initial direction to development. The genetic factors are "the first stage of the causal sequence" that determines human behavior.[15]

Accordingly, by definition, a crime is punishable if it includes certain behaviors in certain circumstances in which the designated mental state is "criminal intent."[16] Whether particular conduct is unacceptable or appropriate behavior depends on communally constructed norms and

9. Daniel Goleman, *New Storm Brews on Whether Crime Has Roots in Genes*, N.Y. TIMES, Sept. 15, 1992, at C1.

10. Diana Fishbein, *Biological Perspectives in Criminology*, 28 CRIMINOLOGY 27, 29 (1990).

11. *Id.* at 29–30.

12. *See generally* Simon Dinitz, Book Review, 14 CONTEMP. SOCIO. 715, (1985) (reviewing LAWRENCE TAYLOR, BORN TO CRIME: THE GENETIC CAUSES OF CRIMINAL BEHAVIOUR) (describing Taylor's analysis and approach in emphasizing the role of genetic determinism in behaviour).

13. *See generally* K. Blum et al., *Allelic Association of Human Dopamine D2 Receptor Gene in Alcoholism*, 263 JAMA 2055, 2055 (1990) (reporting "the first allelic association of the Dopamine $D_2$ receptor gene in alcoholism").

14. *See generally* K. Blum & H. Topel, *Opioid Peptides and Alcoholism: Genetic Deficiency and Chemical Management.*, 1 FUNCT NEUROL 71 (1986).

15. Rowe & Osgood, *supra* note 6.

16. *Mens Rea*, BLACK'S LAW DICTIONARY (11th ed. 2019) ("The state of mind that the prosecution, to secure a conviction, must prove that a defendant had when committing a crime . . . . Mens rea is the second of two essential elements of every crime at common law . . . .").

beliefs. This view is not a dialogue on moral relativism, but rather a perspective on the social construct. The fundamental idea is that what one society may designate as a "crime" another society may not. By their very nature, then, notions of crime, because of increased social subjectivity, are based on a reconsideration of the evolving knowledge of human behavior. An act is characterized as "criminal" if that act is deserving of punishment.[17] There are four underlying objectives that support socially imposed punishment. First, vengeance—the meting out of institutionalized retribution.[18] Second, incapacitation—the removal of offenders from society to prevent future harm.[19] Third, specific and general deterrence. Specific deterrence prevents the individual offender from executing future criminal acts,[20] while general deterrence instills fear of similar penalties to discourage others from committing such acts.[21] Fourth, rehabilitation—through identification, education, and discipline, the justice system attempts to benefit the subject and society by reforming the offender.[22]

Under Anglo-American criminal law, the basis of excuses is a "causal theory."[23] That is, "when an agent is caused to act by a factor outside his control, he is excused; only those acts not caused by some factor external to his will are unexcused."[24] Understanding responsibility, or the relative strength of free will against the external forces of causation, is crucial in

17. *Crime*, BLACK'S LAW DICTIONARY (11th ed. 2019) ("An act that the law makes punishable.").

18. *Vengeance*, BLACK'S LAW DICTIONARY (11th ed. 2019) ("Punishment inflicted as a deserved penalty, esp. by the person wronged, in the name of justice; retributive punishment.").

19. Anthony Bottoms & Andrew von Hirsch, *The Crime-Preventive Impact of Penal Sanctions*, *in* THE OXFORD HANDBOOK OF EMPIRICAL LEGAL RESEARCH 96, 113–14 (Peter Cane & Herbert M. Kritzer eds., 2010) ("Incapacitation is the idea of simple restraint: rendering a convicted offender incapable, for a period of time, of offending again . . . . [O]bstacles are interposed to impede the person from carrying out whatever criminal inclinations he or she may have. Usually the obstacles are prison walls, but other incapacitative techniques are possible—such as exile or house arrest."); *see also Incapacitation*, BLACK'S LAW DICTIONARY (11th ed. 2019) ("The action of disabling or depriving of legal capacity.").

20. *Deterrence*, BLACK'S LAW DICTIONARY (11th ed. 2019) ("specific deterrence. (1951) A goal of a specific conviction and sentence to dissuade the offender from committing crimes in the future.")

21. *Id.* ("A goal of criminal law generally, or of a specific conviction and sentence, to discourage people from committing crimes.")

22. *Rehabilitation*, BLACK'S LAW DICTIONARY (11th ed. 2019) ("The process of seeking to improve a criminal's character and outlook so that he or she can function in society without committing other crimes . . . .")

23. *See* Michael S. Moore, *Causation and the Excuses*, 73 CAL. L. REV. 1091, 1105 (1985) (describing how Anglo-American jurisprudence gives rise to the causal theory).

24. *Id.* at 1091.

determining a standard for liability. Further explanations of the differences between "free will" and ultimate determinism are the subject of much debate. According to the tenets of Western philosophy, individual development is dependent upon the uniquely human "ability to exercise free choice."[25] Modern science challenges this proposition by providing support for the definition of an individual according to predetermined genetic characteristics referred to as Reward Deficiency Syndrome (RDS).[26] The tension is between these two conflicting perspectives of human behavior: free will and determinism.

Often the outcomes of legal proceedings are thought of in terms of only two possibilities: guilty or not guilty. Most jurists, however, recognize varying degrees of guilt exist. Once innocence has been ruled out through a guilty verdict or plea, there are degrees of punishment depending on escalating factors. For instance, sentencing and charges are increased for drunk driving in Texas when: (1) an open-container of alcohol is in the possession of the individual at the time of the incident;[27] (2) the individual's blood alcohol level (BAC) is 0.15 or higher;[28] (3) a minor is in the car;[29] (4) the individual has one or more previous convictions for a DWI;[30] and (5) an accident caused serious bodily injury or death.[31] However, some factors not accounted for in the penal code, may have a mitigating effect on sentencing. These factors include lack of previous criminal history, age of the defendant, and employment history. In Texas, intoxication is not available as an affirmative defense under the "voluntary intoxication" laws.[32]

25. Rochelle Cooper Dreyfuss & Dorothy Nelkin, *The Jurisprudence of Genetics*, 45 VAND. L. REV. 313, 317 (1992) (citing GARY WATSON, *FREE AGENCY*, *reprinted in* AGENCY AND ANSWERABILITY: SELECTED ESSAYS 337, 337–38 (2004); SUSAN WOLF, FREEDOM WITHIN REASON 3–4 (1990)).

26. *Id.* at 318; *see* Kenneth Blum et al., *Reward Deficiency Syndrome (RDS) Surprisingly is Evolutionary and Found Everywhere: Is it "Blowin' in the Wind"?*, 12 J. PERSONALIZED MED. 1, 2 (2022) ("While it is not as yet in the DSM, RDS refers to the breakdown of reward neurotransmission and the destructive behaviors initiated by the combination of environmental (epigenetic) influences and DNA-based neurotransmission deficits that interfere with the usual achievement of the satisfaction of human physiological
drives (food, water, sex).").

27. TEX. PENAL CODE ANN. § 49.04(c) (West 2011).

28. *Id.* at § 49.04(d).

29. *Id.* at § 49.045.

30. *Id.* at § 49.09.

31. *Id.* at §§ 49.07, 49.08.

32. *Id.* at § 8.04(a).

Before the novel defense or mitigation proposal discussed here, addiction disorders had no real precedent as a defense. Cases were (unsuccessfully) fought on the grounds of mitigation of culpability via chemically induced blackout[33], or on the basis of insanity. Although the proposition that addiction is purely a choice—the sum of moral weakness in an individual—is mostly considered a relic of a bygone era, "demonstrating that Anglo-American criminal law is most consistent with [that] position . . . ."[34]

Viewing addiction from the genetic perspective helps frame it as something ingrained in the subject—that is, it is something inherent and not chosen. Persons with the genetic markers consistent with addiction might have the compulsions correlated with those markers. As Morse[35] described it, "[t]he concept of compulsion or something like it is crucial to the no-choice model because without it an addiction is just a very bad habit that is difficult to break."[36]

The mistake, often tended by myopic thinking, is that determinism based on our genes is "hard-wired" at birth and is a permanent phenomenon unaffected by environmental events.[37] While it is correct that genetics have predictive value in determining human behavior,[38] and effectively negate free will, in some cases, one's environment can overcome the essentialism of our DNA code. Specifically, on the one hand, Kevin Blum's research suggests the dopaminergic system, and in particular the dopamine D2 receptor, has been profoundly implicated in reward mechanisms in the mesolimbic circuitry of the brain.[39] Dysfunction of the D2 dopamine receptors leads to an aberrant substance—alcohol, drug, tobacco, and

33. *See* Mark R. Pressman & David S. Caudill, *Alcohol-Induced Blackout as a Criminal Defense or Mitigating Factor: An Evidence-Based Review and Admissibility as Scientific Evidence*, 58 J. FORENSIC SCIS. 932, 932, 939 ("Amnesia is a common claim of criminal defendants, and alcohol-related amnesia is reported by 19–80% of criminal defendants." (footnotes omitted)).

34. Stephen J. Morse, *The Science of Addiction and Criminal Law*, 25 HARV. REV. PSYCHIATRY 261, 261 (2017).

35. Stephen J. Morse is a professor at the University of Pennsylvania in the areas of law and psychology. *Stephan J. Morris*, U. PENN. CAREY L. SCH., https://www.law.upenn.edu/faculty/smorse/ [https://perma.cc/QU7P-ZS9H]. He is well versed in the areas of individual responsibility and agency. *Id.*

36. Morse, *supra* note 34, at 934.

37. *Id.* at 261 (footnotes omitted) ("Some, especially those who believe that addiction is a chronic and relapsing brain disease or neurologic disorder, think that seeking and using are solely or almost solely signs of a disease and that addicts have little choice about whether to seek and use.").

38. *See generally* Kenneth Blum et al., *The D2 Dopamine Receptor Gene as a Predictor of Compulsive Disease: Bayes' Theorem*, 10 FUNCT NEUROL 37 (1995).

39. *Id.*

food—seeking behavior.[40] Decades of research indicate that genetics play an important role in vulnerability to severe substance seeking behavior.[41] Utilizing the Bayes Theorem, Blum proposed that at birth, the predictive value (PV) of carrying variants of the D2 dopamine receptor gene are important common genetic determinants in predicting compulsive disease with a PV of seventy-four percent.[42]

In contrast, Caruso found that exercise could significantly overcome the role of polymorphisms of the fat mass and obesity associated (FTO) gene in producing excess fat cells in humans at birth.[43] Dopamine mutations and RDS lie at the heart of impaired decision making in addiction.[44] It may seem counterintuitive, but SUD is, among other things, a disease of choice. Poor choice or decision making is often the problem with repeat DWI offenders who, when intoxicated, choose to operate vehicles—a choice they would not make if their judgment were not impaired.

In today's judicial system, our legal apparatus operates according to an "as if" theory. This approach accepts the truth of determinism yet adopts an "as if" view of human freedom. In other words, society should design institutions "as if" human action was not determined. Proponents of this scheme recognize that although determinism may be the first postulate of science to choose free action as the first postulate of legal and moral thought, the philosophy remains subject to challenges because it ignores predispositions, e.g., reward gene variations (polymorphisms). Along these lines, Tikkanen offers a thought-provoking report that found carriers of the MAOA-H (high activity) allele have a high risk for committing severe, recidivistic impulsive violent crimes after exposure to heavy drinking and Childhood Physical Abuse (CPA).[45]

In essence, under the "as if" theory, the legal system attempts to reconcile the two paradigms by working out a form of "rough justice," which is arguable at best and requires out-of-the box thinking. The system presumes free will and imputes criminal responsibility to accomplish this, but also

40. *Id.*

41. *Id.*

42. *Id.*

43. V. Caruso et al., *The Beneficial Effects of Early Short-Term Exercise in the Offspring of Obese Mothers are Accompanied by Alterations in the Hypothalamic Gene Expression of Appetite Regulators and FTO (Fat Mass and Obesity Associated) Gene*, 25 J. NEUROENDOCRINOLOGY 742, 749–50 (2013).

44. *See generally*, Blum *supra* note 38.

45. Roope Tikkanen et al., *MAOA Alters the Effects of Heavy Drinking and Childhood Physical Abuse on Risk for Severe Impulsive Acts of Violence Among Alcoholic Violent Offenders*, 34 ALCOHOLISM: CLINICAL AND EXPERIMENTAL RSCH. 853, 857 (2010).

allows for the uncontrollable influence of determinism by providing exculpatory defenses or by mitigating resulting punishment.[46] It is this latter concept that we, as neuroscientists, geneticists, and clinicians, consider the area of change that requires deeper thinking in the absence of legal precedent. Courts have responded with varying degrees of receptivity to scientific evidence suggesting a causal link between human behavior and predetermined biological factors. A genetic defense claim implies impairment of free will, much like the known defenses of insanity or diminished capacity. However, the judicial system has been relatively consistent in alignment with defenses based on insanity and diminished mental capacity. In Texas, it is challenging and rare to use an "insanity" or "diminished capacity" defense successfully.[47]

The logic behind the insanity defense is obvious; put simply, "the insanity defense accepts that most people act under free-will but allows leeway for a person who is incapable of making decisions based on acceptable moral and legal standards."[48] The standard test, adopted by most American jurisdictions, comes from an English case called *M'Naughten*.[49] The *M'Naughten* rule states:

> In all cases of this kind the jurors ought to be told that every man is presumed to be sane, and to possess a sufficient degree of reason to be responsible for his crimes, until the contrary be proved to their satisfaction: and that to establish a defence on the ground of insanity, it must be clearly proved that at the time of committing the act the party accused was laboring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing, or as not to know that what he was doing was wrong.[50]

46. Schwartz, *supra* note 1, at 1278–79.

47. *E.g.*, Reyna v. State, 116 S.W.3d 362, 365–68 (Tex. App.—El Paso 2003, no pet.) (determining from the great weight of the evidence that the defendant successfully pled insanity based on expert testimony regarding his polysubstance abuse diagnosis and evidence showing he suffered from hallucinations). *But see* Afzal v. State, 559 S.W.3d 204, 208 n.4, 215 (Tex. App.—Texarkana 2018, pet. ref'd) ("One who voluntarily and illegally ingests a substance should do so at the risk of whatever mental disturbances flow from that voluntary act, regardless that they may not fit within the common understanding of being 'under the influence.'").

48. Michelle Prejean, *Texas Law Made this Mad Woman Sane*, 42 Hous. L. Rev. 1487, 1490–91 (2006) (citing Finger v. State, 27 P.3d 66, 71 (Nev. 2001).

49. M'Naghten's Case (1843) 8 Eng. Rep. 718 (H.L).

50. *Id.* at 719.

Texas case law also defines *M'Naughten* as meaning that the actor must have been "in an extreme delusional state[] that caused [him or her] to misperceive the very nature of [his or her] acts, or to believe that in acting, [he or she was] obeying rather than violating the laws of society."[51]

Insanity is an affirmative defense the defendant must prove, and the myriad of judicial hurdles can render this approach functionally ineffective. It is our position that the ability to withstand the powers of, for example, alcoholism (and alcoholism's effect on decisions), is not causally linked to "free will." America would not be facing the self-destruction of approximately 22,000,000 of its citizens battling substance use disorders if it were that simple.[52] It is well-established that severe alcoholism can lead to violent crimes affecting millions.[53] The legal system causally links an enormous percentage of violent crime and motor death with DWI, e.g., intoxication assault and intoxication manslaughter.[54] Over the last several years, "recovery-oriented policies have aimed to expand social supports for recovery and to improve access to [substance use disorder] treatment within the criminal justice system."[55] The Affordable Care Act substantially modified "access to substance abuse treatment by mandating that health insurance include services for substance use disorders" comparable to coverage for medical and surgical treatments.[56] This new approach is not merely a "war on drugs," instead, what it seems to be is an approach with

51. Rubio v. State, 241 S.W.3d 1, 13 (Tex. Crim. App. 2007) (citing Ellen Byers, *Mentally Ill Criminal Offenders and the Strict Liability Effect: Is There Hope for a Just Jurisprudence in an Era of Responsibility/Consequences Talk?*, 57 ARK. L. REV. 447, 480 (2004)).

52. *Key Substance Use and Mental Health Indicators in the United States: Results from the 2019 National Survey on Drug Use and Health*, SUBSTANCE ABUSE & MENTAL HEALTH SERVS. ADMIN. 5 (2019).

53. Lawerence A. Greenfeld, *Alcohol and Crime: An Analysis of National Data on the Prevalence of Alcohol Involvement in Crime*, DEP'T OF JUSTICE at v–vii (1998) (providing various statistics on the role of alcohol in crime).

54. *Id.*

55. Ariela O. Karasov & Michael J. Ostacher, *Alcohol and the law*, 125 HANDBOOK OF CLINICAL NEUROLOGY 649 (2014); *see also Facing Addition in America: The Surgeon General's Report on Alcohol, Drugs, and Health*, DEP'T OF HEALTH & HUMAN SERVS., 4-39–4-40 (2016) (describing the elements and benefits of the continuum of treatment for substance use disorders); Tex. Gov't Code Ann. § 493.009 (granting the Texas Department of Criminal Justice, with the Texas Department of State Health Services, the power to create and operate the state's Substance Abuse Treatment Program).

56. Karasov & Ostacher, *supra* note 55, at 653; *see also* Amanda J. Abrams et al., *The Affordable Care Act: Transformation of Substance Use Disorder Treatment*, 107 AM. J. PUB. HEALTH 31, 31 (2017) ("[T]he ACA extends the 2008 Mental Health Parity and Addiction Equity Act, which requires that insurers cover SUD treatment in a no more restrictive way than medical and surgical services. Federal parity rules now apply to all private plans including those offered on state exchanges and Medicaid expansion programs.").

"increasing emphasis on evidence-based policy development that approaches alcohol use disorders with hope for treatment and prevention."[57] Some judges and prosecutors are embracing the idea of treatment over incarceration. However, generally speaking, relief from criminal responsibility, or having responsibility mitigated, will require defendants to rebut the presumption of free will, offering proof that there are forces that negated their ability to choose or rationally execute their actions.

### B. *The Gene Violence Debate in Modern Times*

There is now an explicable connection between the overlap of nature and nurture. However, in the past, many debated the classic psychology question of nature vs. nurture with behaviors, habits, and preferences explained as a matter of genetic disposition or as learned from the environment. This type of debate exists because it is difficult to discern when one ends and the other begins. As genetic mapping becomes more explicit regarding abhorrent behavior like domestic violence, the victims and perpetrators can, in the therapeutic context, use this new data to analyze, understand, and hopefully treat and prevent these acts of horrific violence.[58]

In looking for the reason for this type of negative behavior, the nature theory is the proposition based on heredity, where specific genes spark the behavior.[59] Genetic association studies support this theory and, in combination with nurture theory, the way that particular genes frequently correlate with similar violent behavior in reaction to stressful environments.[60] These ideas may explain the reason, despite being raised in a violent environment, some individuals can become healthy, productive

57. Karasov & Ostacher, *supra* note 55, at 655.

58. *See generally* Jari Tiihonen, et al. *Genetic Background of Extreme Violent Behavior,* 20 MOLECULAR PSYCHIATRY 786, 786 (2015) (highlighting two more genes associated with violent crime).

59. *Cf. id.* (reporting that persons with two specific genes are more likely to recommit violent crime).

60. *See* Kent W. Nilsson et al., *Gene–Environment Interaction of Monoamine Oxidase A in Relation to Antisocial Behaviour: Current and Future Directions*, 125 J. NEURAL TRANSMISSION 1601, 1601–02 (2018), ("[I]ndividuals may vary in their ability to cope with stressful experiences and environments depending on their genetic make-up, a phenomenon commonly referred to as gene–environment interaction (G×E). . . . Heterogeneous neurobiological, psychological and behavioural components constitute aggressive behaviour. The association between cognition, emotion and aggression is well-known, and neural circuitries such as the serotonergic system have been shown to play a key role in regulating aggressive behaviour." (citations omitted)).

members of society, while others repeat abusive behavior throughout their own lives.

Human traits are complex, affected by a multitude of environmental and genetic factors and the subsequent interactions among them.[61] "However, previous gene-environment interaction (G×E) studies have typically focused on one or only a few genetic variants at a time."[62] For example, Liu developed a gene map that helped explain aggressive and violent behaviors in delinquent youth involving 403 genes and 39 Single-Nucleotide Polymorphisms (SNPs).[63] It is noteworthy that Aslund looked for a genetic predictor of adolescent delinquency.[64] They investigated a possible interaction between a functional polymorphism in the MAOA gene promoter (MAOA-VNTR) and childhood maltreatment.[65] They found that boys with a short variant and girls with one or two long variants of the polymorphism showed a higher risk for delinquency when exposed to maltreatment.[66] Also, Armstrong found that "[t]he low expressing allele of the MAOA-uVNTR genotype (MAOAL) interacted with abuse to predict self-reports of less serious criminal and delinquent behavior and had a direct association with serious criminal activity."[67] This finding suggests the importance of dopaminergic function in childhood maltreatment.[68] Another remarkable discovery by Vaske showed that violently victimized offenders are more likely to carry the DRD2 (A1) risk allele than non-victimized offenders.[69] In terms of adolescent delinquency, anger, and

61. Hexuan Liu et al., *Gene by Social-Environment Interaction for Youth Delinquency and Violence: Thirty-Nine Aggression-Related Genes*, 93 SOC. FORCES 881, 881 (2015).

62. *Id.*; *see generally* Nilsson, et al., *supra* note 60 (focusing on the enzyme monoamine oxidase A encoded by the MAOA gene); Tiihonen et al., *supra* note 58 (analyzing the MAOA or CDH13 genes and their relation to violence).

63. *See generally* Hexuan Liu et al., *supra* note 61 (depicting the trend toward examining the relationship between multiple gemes at a time).

64. *See generally* C. Åslund et al., *Maltreatment, MAOA, and Delinquency: Sex Differences in Gene–Environment Interaction in a Large Population-Based Cohort of Adolescents*, 41 BEHAV. GENETICS 262, 262 (2011) ("The present study investigated a possible interaction between a functional polymorphism in the MAOA gene promoter (MAOA-VNTR) and childhood maltreatment in the prediction of adolescent male and female delinquency.").

65. *Id.*

66. *Id.* at 266–67.

67. Todd A. Armstrong et al., *Monoamine Oxidase A Genotype, Childhood Adversity, and Criminal Behavior in an Incarcerated Sample*, 24 PSYCHIATRIC GENETICS 164, 164 (2014)

68. *Id.* at 169–70.

69. Jamie Vaske et al., *A Dopamine Gene (DRD2) Distinguishes Between Offenders Who Have and Have Not Been Violently Victimized*, 55 INT'L J. OFFENDER THERAPY AND COMPAR. CRIMINOLOGY 251, 259 (2010).

thrill-seeking, the impact of the environment in the presence of a risk genotype seems to be crucial. Dmitrieva found that social context plays an essential role in explaining the gender-specific phenotypic expression of the DRD4 gene.[70] Individuals with the 4/4 genotype were compared to the 7-repeat allele (7R).[71] Males had significantly higher delinquency, short temper, and thrill-seeking than females and higher exposure of males to psychosocial risk factors.[72] When the model included parental monitoring of youths' activities and youth exposure to violence, the 7R × gender interaction was no longer significant.[73] This result points to the notion that an individual is not doomed to 'bad behavior,' but rather, while set up to fail at birth, loving and caring parents may help.[74]

Moreover, the positive epigenetic impact of nurture may overcome genetic insults. There is evidence that religiosity prevents relapse[75] and may impact adolescent delinquency despite carrying a risk allele like DAT1 or DRD2.[76] In the prediction of adolescent delinquency, Beaver uncovered an interaction between gene X environment and the A-1 allele of DRD2 and religiosity.[77]

Legally speaking, attorneys are often hesitant to entertain any connection between domestic violence and heredity. They often desist because these explanations tend to deflect some responsibility from the aggressor and can hamper prosecutors in their capacity as victim advocates. The concept of accountability necessitates that those who commit a harmful behavior be held responsible for their actions. A genetic cause for an individuals'

70. Julia Dmitrieva et al., *Gender-Specific Expression of the DRD4 Gene on Adolescent Delinquency, Anger and Thrill Seeking*, 6 SOC. COGNITIVE & AFFECTIVE NEUROSCIENCE 82, 87 (2011).

71. *Id.* at 85.

72. *Id.* at 86–87.

73. *Id.* at 86.

74. *Id.* at 87.

75. *See generally* Stephen J. Schoenthaler et al., *NIDA-Drug Addiction Treatment Outcome Study (DATOS) Relapse as a Function of Spirituality/Religiosity*, 1 J. REWARD DEFICIENCY SYNDROME 36 (2015) (discussing the impact of spirituality on remission from abused drugs).

76. *See generally Id.* at 36 (reporting "regular spiritual practice, particularly weekly attendance at the religious services of their choice is associated with significantly higher remission"); *see* Guang Guo et al., *Contributions of the DAT1 and DRD2 Genes to Serious and Violent Delinquency Among Adolescents and Young Adults*, 121 HUMAN GENETICS 125, 127 (2007) ("The initial findings of the association concerning both DAT1 and DRD2 do not seem to vary by Add Health Waves or age, suggesting that the genotype effects may be relatively constant or the trajectories of delinquency across genotypes are likely to be parallel over adolescence and young adulthood.").

77. Kevin M. Beaver et al., *A Gene X Environment Interaction Between DRD2 and Religiosity in the Prediction of Adolescent Delinquent Involvement in a Sample of Males*, 55 BIODEMOGRAPHY & SOC. BIOLOGY 71, 79 (2009).

behavior calls the level of that individual's responsibility into question. The crucial question elicited by this notion is our primary focus—do people make decisions based on genetics and not free will? Consider this oversimplified example: a genetically short person is at fault for causing a car accident because they could not see from the same vantage point as a taller person. Silly, yes, and obviously dissimilar to our present conversation, *but are factors outside of our control not relevant to understanding the cause of actions and behaviors*? We are not trying to eliminate perpetrator responsibility, but to provide an advantageous in-depth understanding of the behavioral etiology and potential for positive expectations of scientifically validated rehabilitation.

Although a tendency to violence might be an identifiable genetic risk, exposure to violent acts that occur at a young age and imprint the child increase the likelihood of repeating the violence, especially without any positive epigenetic impact such as a nurturing parent or mentor.[78] This nurture concept crosses the line to behavior that can be learned by exposure to certain environments. Some remarkable research about heredity, environment, and violence includes a compelling study from Colorado State University and the Sam Houston State University College of Criminal Justice.[79] The study revealed that 82% of adult children who experienced partner violence growing up were involved in at least minor partner violence themselves.[80] Based on approximately 1,600 families, 92% of parents admitted to being involved in minor partner violence at least once, with 68% admitting to committing acts of violent intimate partner violence at least

78. Michael J. Shanahan et al., *Helping Relationships and Genetic Propensities: A Combinatoric Study of DRD2, Mentoring, and Educational Continuation*, 10 TWIN RSCH. AND HUM. GENETICS 285, 285, 296 (2007).

79. *See generally* Kelly E. Knight, et al., *Generational Cycles of Intimate Partner Violence in the US: A Research Brief*, SAM HOUSTON STATE UNIVERSITY (2013), http://dev.cjcenter.org/_files/cvi/Generation%20Cycles%20IPVforweb.pdf [https://perma.cc/TP78-ZA7L] (describing a brief study on intergenerational intimate partner violence); Molly B. Kenny, *Domestic Violence Study Finds Partner Abuse Is Generational*, https://www.mollybkenny.com/library/domestic-violence-study-abuse-travels-through-family-generations.cfm [https://perma.cc/CAX7-ZP3P] ("Last month, researchers from the Sam Houston State University College of Criminal Justice and Colorado State University revealed the results of their domestic abuse study.").

80. Knight, et al., *supra* note 79, at 2 ("Showing more stability across generations, 81.7% of offspring respondents also reporting perpetrating minor IPV."); Kenny, *supra* note 79 ("Specifically, 80 percent of study participants who were involved with intimate partner violence had adult children who were involved in a domestic violence incident.").

once.[81] Additionally, "93.4% of the parents and 78.8% of their adult children reported experiencing minor victimization from an intimate partner."[82] Very few families, 14%, were able to discontinue the cycle of violence between generations.[83]

These findings highlight the impact exposure to violence can have on children growing up and the cycle of domestic violence it perpetrates. These facts, coupled with epigenetic evidence, provide a framework for the probation system to recognize that abuse behaviors have been adequately shown in animal models to continue for up to F2 generations. Many personality traits might be enhanced or suppressed when children learn by mirroring the behavior of their parent;[84] an example is when a son witnesses his father hit his mother.

Generational domestic violence has the potential to affect many lives.[85] Studies and science can agree on one thing: this behavior is not healthy, and many young impressionable kids exposed to it engage in the harmful repetition of behavior they witness. The key to comprehending the unfortunate reality is that those exposed to it should be aware of these findings so that hopefully, with more education and understanding, they can avoid repeating negative behaviors.

### C. *Genetic Imbalance Theory of Crime Causation*

*People v. Yukl*[86] echoed the judicial conclusion that a genetic imbalance theory of crime causation was not yet sufficiently established or accepted to warrant admitting evidence of a biological affliction.[87] Rather than stating that the evidence failed to meet any specific test of legal insanity, however, the court in *Yukl* held the scientific theory simply failed to meet the

81. Knight, et al., *supra* note 79, at 2 ("Across all interviews, 92.1% of parents reported perpetrating minor IPV at least once."); Kenny, *supra* note 79 ("A staggering 92 percent of respondents admitted that they had committed at least one act of domestic violence, ranging from pushing and threats to their partner, to punching or threatening to kill their spouse.").

82. Knight, et al., *supra* note 79, at 2.

83. *Id.*

84. *See, e.g.*, Benoit Labonté et al., *Genome-wide Epigenetic Regulation by Early-Life Trauma*, 69 ARCHIVES GEN. PSYCHIATRY 722, 722 (2012) ("Early studies have shown that variations in the quality of postnatal parent-offspring interactions directly alter intracellular signals that regulate epigenetic states, with sustained effects on gene transcription.").

85. *See, e.g.*, Thomas J.H. Chen et al., *Are Dopaminergic Genes Involved in a Predisposition to Pathological Aggression?*, 65 MED. HYPOTHESES 703, 704–05 (2005) ("[W]e are convinced that the likelihood of either having a disorder or carrying a vulnerability gene for any psychiatric disorder is quite common.").

86. People v. Yukl, 372 N.Y.S.2d 313 (Sup. Ct. 1975).

87. *Id.* at 320.

threshold evidentiary test of admissibility.[88] Importantly, the court suggested that future research efforts might lead to the admissibility of genetic theory.[89] Although, at that time, no 'exact biological mechanism' or causal connection had been identified to show a relationship between genetic composition and deviant behavior, the Court surmised: "The answers to these problems are currently being sought by scientists and their solution will assist immeasurably in providing a firmer footing for the incorporation of chromosome abnormality . . . ."[90] In *Yukl*, the defendant, charged with murder, requested the appointment of a cytogeneticist to conduct chromosomal tests.[91] The defense sought to determine whether he possessed the XYY complement.[92] We agree with the conclusion of the court whereby, although the Court recognized the established existence of the XYY genetic phenomenon, it determined that "the sampling, thus far, has been inadequate and inconclusive," and reflected a "built-in bias" because of the institutionalized subject populations and lack of proper control group data.[93] Moreover, we further agree with the court's conclusion that "[s]cientists and legal commentators appear to be in agreement that further study is required to confirm the initial findings and to concretely establish a causal connection between one's genetic complement and a predisposition toward violent criminal conduct."[94] It is noteworthy that the court proposed a qualifying test:

> [A]n insanity defense based on chromosome abnormality should be possible only if one establishes with a high degree of medical certainty an etiological relationship between the defendant's mental capacity and the genetic syndrome. Further, the genetic imbalance must have so affected the thought processes as to interfere substantially with the defendant's cognitive capacity or with his ability to understand or appreciate the basic moral code of his society.[95]

88. *Id.* at 319–20.

89. *Id.* at 318 & n.5 (citing Kenneth J. Burke, *The XYY Syndrome: Genetics Behavior and the Law*, 46 DEN. L.J. 261 (1969); John Money et al., *Impulse, Aggression and Sexuality in the XYY Syndrome*, 44 St. John's L. Rev. 220 (1969)).

90. *Id.* at 319–20.

91. *Id.* at 317.

92. *Id.*

93. *Id.* at 318.

94. *Id.* at 318 & n.5 (citing Burke, *supra* note 89; Money, *supra* note 89).

95. *Id.* at 319.

D. *Alcoholism and Addiction: Can Genetics Be Used as a Defense?*

The aforementioned series of cases understandably did not result in the establishment of a successful genetic defense. Courts gave the impression that, absent convincing proof of causality, mere demonstration of a biological defect would not excuse criminal behavior.

Indeed, this is the very point of this Article—since the seminal findings of Blum were published and the association of dopaminergic gene variants with severe alcoholism as a direct biological link for predisposition, the entire field of "Psychiatric Genetics" was born.[96] Whereas relatively unexplored chromosomal aberrations like the XYY condition have been met with judicial skepticism, courts more readily have addressed hereditary afflictions such as alcoholism and chemical addiction as potentially relevant factors in identifying moral culpability and appropriate sentencing. Despite the Supreme Court's position that the "status" or condition of chemical addiction cannot be considered in and of itself a criminal offense,[97] courts have remained reluctant to completely absolve those whom the state has duly convicted. Evidence that an individual suffered from a biological abnormality is most often used to mitigate punishment for unlawful behavior without real genetic proof.

The Supreme Court, in 1962, in *Robinson v. California*,[98] held that the "status" of chemical addiction alone is not a crime.[99] A California statute made narcotic addiction a punishable offense for which "at any time before he reforms," the individual could be prosecuted even though he had never used or possessed narcotics in California, nor been guilty of any antisocial behavior within the State.[100] The Court struck down the statute on the

96. *See generally* Kenneth Blum et al., *Allelic Association of Human Dopamine D2 Receptor Gene in Alcoholism*, 263 JAMA 2055, 2055 (1990) (reporting "the first allelic association of the Dopamine $D_2$ receptor gene in alcoholism").

97. *See* Robinson v. California, 370 US 660, 667 (1962) ("In this Court counsel for the State recognized that narcotic addiction is an illness. Indeed, it is apparently an illness which may be contracted innocently or involuntarily. We hold that a state law which imprisons a person thus afflicted as a criminal, even though he has never touched any narcotic drug within the State or been guilty of any irregular behavior there, inflicts a cruel and unusual punishment in violation of the Fourteenth Amendment.").

98. Robinson v. California, 370 US 660 (1962).

99. *See id.* at 667 (asserting the involuntary nature of addiction makes punishment for it analogous to punishment for the common cold).

100. *Id.* at 666.

ground that, in violation of the Eighth and Fourteenth Amendments, it inflicted cruel and unusual punishment.[101]

According to the plaintiff, equating a mere physical condition with criminality would be as unjust as making mental illness or leprosy a criminal offense (which would be arguably violative of the Eighth Amendment's prohibition against cruel and unusual punishment).[102] *Robinson* declared that this age of enlightenment could not tolerate such barbarous action. Perhaps most importantly, the Court characterized chemical addiction as an illness or disease.[103] Some of the most stirring commentary in that case came from Justice Douglas's concurrence: "A prosecution for addiction, with its resulting stigma and irreparable damage to the good name of the accused, cannot be justified as a means of protecting society, where a civil commitment would do as well."[104]

These informative remarks and precedents are presented here as a background for the present plausibility of linking actual genetic data as a defense for wrongdoing or mitigation of punishment.

In *Powell v. Texas*,[105] the Court reaffirmed the legislative right to impose criminal sanctions to protect society from acts posing substantial health and safety hazards or offending moral and aesthetic sensibilities.[106] As long as the affirmative conduct of the individual endangered public welfare, criminal punishment would not be considered "cruel and unusual," regardless of the causal forces behind the act.[107] They further considered excusing "compulsive behavior" without any real proof, especially concerning biological effects such as genetics, as absurd.[108] Accordingly, Judge Black concluded, "The range of problems created would seem totally beyond our capacity to settle at all, much less to settle wisely, and even the attempt to define these terms and thus to impose constitutional and doctrinal rigidity seems absurd in an area where our understanding is even today so incomplete."[109]

Most importantly, holding the older state of scientific knowledge inadequate to raise a legitimate physiological defense, the *Powell* court, as in

101. *Id.* at 667.
102. *Id.* at 666.
103. *Id.*
104. *Id.* at 677 (Douglas, J., concurring).
105. Powell v. Texas, 392 U.S. 514, 546 (1968).
106. *Id.* at 532.
107. *Id.* at 532–33.
108. *Id.*
109. *Id.* at 546 (Black, J., concurring).

*Yukl*, intimated that more definite proof might lead to a more successful claim: "[I]n order to make out a constitutional defense, should one be recognized[,]" a person would have to display both a "loss of control" once he or she had begun to drink and an "inability to abstain from drinking in the first place."[110]

Indeed, this is now easily explained by recent data involving specific reward genes and relapse for alcohol and drugs or displaying both loss of control and inability to abstain.[111]

Along these lines, Dahlgren, and later Balldin concluded that there is an association between the TaqI A1 allele of the DRD2 gene and substantially increased relapse rates in alcoholics.[112] There is also data showing that the Val66Met (COMT) and Brain-Derived Neurotrophic Factor (BDNF) gene polymorphism were associated with a higher risk and earlier occurrence of relapse among patients treated for alcohol dependence.[113] Moreover, the dopamine receptor type 4 (DRD4 VNTR 48 bp), unlike type <7R, may have protective properties concerning short Average Time to Relapse [ATR].[114] Also, the effect of the combination "of polymorphisms in serotonin transporter and monoamine oxidase-A genes on the aetiopathogenesis of alcoholism investigated in a sample of 714 individuals" found an increased frequency of subjects having three "suspected" genotypes (5-HTTLPR-LL, STin2-1010, and MAO-A 3-repeat allele).[115] This result was found significantly among type-2 alcoholic patients.[116] There are also studies showing the association of polymorphisms in the DRD1 gene and high

110. *Id.* at 524–26.

111. Kenneth Blum & Mark S. Gold, *Neuro-chemical Activation of Brain Reward Meso-limbic Circuity is Associated With Relapse Prevention and Drug Hunger: A Hypothesis*, 76 MED. HYPOTHESES 576–584 (2011).

112. Jan Balldin et al., *Varför vissa återfaller i alkoholberoende. Relation finns till en genvariant i dopaminsystemet och till psykologi.*, 110 Lakartidningen 21-23 (2013) (Title translation: "Why some people relapse in alcohol dependence. There is a relation to a specific gene variant in the dopamine system and to psychology.");Angelica Dahlgren et al., *Do Alcohol-dependent Individuals with DRD2 A1 Allele Have an Increased Risk of Relapse? A Pilot Study*, 46 ALCOHOL AND ALCOHOLISM 509, 510–11 (2011).

113. Marcin Wojnar et al., *Association Between Val66Met Brain-Derived Neurotrophic Factor (BDNF) Gene Polymorphism and Post-Treatment Relapse in Alcohol Dependence*, 33 ALCOHOLISM: CLINICAL & EXPERIMENTAL RSCH. 693, 700 (2009).

114. A. O. Kibitov et al., *Duration of therapeutic remission alcohol dependence: a role of dopamine system genes polymorphism and family history density*, 115 Zhurnal nevrologii i psikhiatrii im. S.S. Korsakova 51-58 (2015).

115. Tatjana Bordukalo-Niksic et al., *Combination of polymorphic variants in serotonin transporter and monoamine oxidase-A genes may influence the risk for early-onset alcoholism*, 200 PSYCHIATRY RSCH. 1041, 1041 (2012).

116. *Id.*

sensation-seeking alcoholic men.[117] Comings eloquently showed an additive role of the DRD1 and DRD2 risk alleles loading onto several RDS behaviors, including alcoholism, gambling, and smoking behaviors.[118]

In 1999, scholar Maureen P. Coffey suggested "courts have previously rejected defenses based on biological predispositions on the grounds of insufficient evidence of affliction and inconclusive proof of causation."[119] Scientific "progress may overcome the former shortcoming, but the latter dilemma remains a point of speculation."[120] Coffey further suggested that "[i]n light of the gap between identifying actual genetic aberration and demonstrating an adequate causal connection, the legal system must determine how much weight, if any, to give each factor."[121]

Coffey proposes a reconsideration based on evidence:

> Regardless of whether courts or legislatures decide to consider evidence of biological abnormality as a legal excuse, as a mitigating factor during sentencing, or as having no negating effect on guilt, traditional concepts of individual responsibility and social justification must be restated in terms that reflect scientific reality.[122]

She continues,

> The model of free will must be reconsidered in light of increasing support for deterministic influences. If moral culpability no longer serves as the basis for penalizing an offender, society must recognize that social utility may be the more predominant concern. In any respect, evidence of 'genetic factors in crime' cannot be ignored. Although simplistic or reductionist theories must be discredited and avoided, society must address the ethical, social, and legal implications that accompany a greater understanding of the human body and mind.[123]

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117. F. Limosin et al., *Association Between Dopamine Receptor D1 Gene Dde I polymorphism and Sensation Seeking in Alcohol-Dependent Men*, 27 Alcoholism: Clinical & Experimental Research 1226-1228 (2003).

118. DE Comings et al., *Studies of the Potential Role of the Dopamine D1 Receptor Gene in Addictive Behaviors*, 2 MOLECULAR PSYCHIATRY 44, 54 (1997).

119. Maureen Coffey, *The Genetic Defense: Excuse or Explanation?*, 35 William & Mary Law Review 353, 399 (1993).

120. *Id.*

121. *Id.*

122. *Id.*

123. *Id.*

The court system no longer has the luxury of ignoring the role of genes in committing a crime. That is why this case study presents the results of genotyping for genetic risk for Alcohol Use Disorder (AUD) and its impact on penalization.

## II. METHODS AND MATERIALS

### A. *The Case*

The proband is a thirty-five year old (at the time of adjudication) male (AG) of Hispanic descent diagnosed with severe AUD. The patient signed an improved informed consent form approved by the IRB of the University of Vermont. AG adopted the addition of the GARS genetic test and the Precision Addiction Management platform, BSBA, LCDC, as standard practice. The subject underwent detailed assessments, including polymorphic information via the GARS test, and an extensive treatment and monitoring plan. The defense recommended the continuing treatment and monitoring plan to the prosecution as a condition of probation.

### B. *Sample Collection and Processing*

Buccal cells were collected from the patient using a Sterile Copan 4N6FLOQ Swab (Regular Size Tip In 109MM Long Dry Tube with Active Drying System) from an established, minimally invasive collection kit provided by Geneus Health Laboratories in San Antonio, Texas.[124] The subject "collect[ed] cells from both cheeks by rubbing the swab at least 25 times on each side of his mouth, and [returning] the swab to the specimen tube."[125] The specimen tubes, labeled with a pre-defined bar-coded ID, were sent via currier to the Geneus Health laboratory for subsequent genotyping. For all sample processing steps, known DNA standards were included and verified, including non-template controls.[126]

Each selected risk polymorphism of the genes included within the GARS panel, shown in Table 1, had a known contribution (such as a

124. Kenneth Blum et al., *Biotechnical Development of Genetic Addiction Risk Score (GARS) and Selective Evidence for Inclusion of Polymorphic Allelic Risk in Substance Use Disorder (SUD)*, 6 J. SYS. & INTEGRATIVE NEUROSCIENCE 1, 4, 15 (2019) [hereinafter *Biotechnical Development of GARS*] https://www.oatext.com/pdf/JSIN-6-221.pdf [https://perma.cc/65WU-BQKG].

125. *Id.*

126. *Id.*

hypodopaminergic function within the brain) to Reward Deficiency Syndrome (RDS).[127]

**FIGURE 1:** FULL GARS PANEL[128]

| Gene | Polymorphism | Location | Risk Allele(s) |
|---|---|---|---|
| **Dopamine D1 Receptor DRD1** | rs4532 SNP | Chromosome 5 | A |
| **Dopamine D2 Receptor DRD2** | rs1800497 SNP | Chromosome 11 | A |
| **Dopamine D3 Receptor DRD3** | rs6280 SNP | Chromosome 3 | C |
| **Dopamine D4 Receptor DRD4** | rs1800955 SNP | Chromosome 11 | C |
| | 48 bases Repeat VNTR | Chromosome 11, Exon 3 | 7R,8R,9R,10R,11R |
| **Catechol-0-methyltransferase COMT** | rs4680 SNP | Chromosome 22 | G |
| **Mu-Opioid Receptor OPM1** | rs1799971 SNP | Chromosome 6 | G |
| **Dopamine Active Transporter DAT1** | 40 bases Repeat VNTR | Chromosome 5, Exon15 | 3R,4R,5R,6R,7R,8R |
| **Monoamine Oxidase A MAOA** | 30 bases Repeat VNTR | Chromosome X, Promoter | 3.5R, 4R |
| **Serotonin Transporter SLC6A4 (5HTTLPR)** | 43 bases Repeat INDEL/VNTR rs25531 | Chromosome 17 | LG, S |

127. *Id.* For details about the biotechnical methods used to identify the alleles used in the (GARS) test panel in Figure 1, *see* Kenneth Blum et al., *Genetic Addiction Risk Score (GARS™) as a Predictor of Substance Use Disorder: Identifying Predisposition Not Diagnosis*, CURRENT TRENDS MED. DIAGNOSIS METHODS, Sept. 11, 2018, https://www.gavinpublishers.com/assets/articles_pdf/1549618762article_pdf381191185.pdf [https://perma.cc/43RA-KR28].

128. *Biotechnical Development of Gars*, *supra* note 124, at Figure 4A–4C.

| | | | |
|---|---|---|---|
| **GABA(A) Receptor, Alpha 3 GABRB3** | CA Repeat DNR | Chromosome 15 (downstream) | 181 |

SNP=Single Nucleotide Polymorphism; VNTR=Variable Tandem Repeat; INDEL=Insertions and Deletions; DNR=Di-Nucleotide Repeat

DNA was isolated from a buccal sample. "For genotyping the single nucleotide polymorphisms, . . . commercially available or custom TaqMan RT-PCR assays (Thermo Fischer Scientific, Waltham, MA) were used."[129] Thermal cycling conditions were manufacturer recommended, "and genotypes were called using TaqMan Genotyper Software v1.3 (Life Technologies, Carlsbad, CA)."[130]

**FIGURE 1A:** SINGLE NUCLEOTIDE POLYMORPHISMS (SNPS)[131]

| Gene | Polymorphism | Variant Alleles | Risk Allele |
|---|---|---|---|
| **Dopamine D1 Receptor *DRD1*** | rs4532 | A/G | A |
| **Dopamine D2 Receptor *DRD2*** | rs1800497 | A/G (A1/A2) | A (A1) |
| **Dopamine D3 Receptor *DRD3*** | rs6280 | C/T | C |
| **Dopamine D4 Receptor *DRD4*** | rs1800955 | C/T | C |
| **Catechol-O-Methyltransferase *COMT*** | rs4680 | A/G (Met/Val) | G (Val) |
| **Mu-Opioid Receptor *OPRMI*** | rs1799971 | A/G (Asn/Asp) | G (Asp) |

129. *Id.*
130. *Id.*
131. *Id.* at Figure 4A.

**FIGURE 1B:** SIMPLE SEQUENCE REPEATS (VARIABLE NUMBER TANDEM REPEATS AND INSERTIONS/DELETIONS)[132]

| Gene | Polymorphism | Variant Alleles | Risk Alleles |
|---|---|---|---|
| **Dopamine D4 Receptor *DRD4*** | rs761010487 | 48bp repeat 2R-11R | ≥ 7R, long form |
| **Dopamine Active Transporter *DAT1*** | rs28363170 | 40p repeat 3R-11R | <9R |
| **Monoamine Oxidase A *MAOA*** | rs768062321 | 30bp repeat 2R-5R | 3.5R, 4R, 5R |
| **Serotonin Transporter *SLC6A4 (5-HTTLPR)*** | rs4795541, rs25531 | 43bp repeat, with SNP L/XL and S, G/A | S, LG |

**FIGURE 1C:** DINUCLEOTIDE REPEATS[133]

| Gene | Polymorphism | Variant Alleles | Risk Allele |
|---|---|---|---|
| **GABA(A) Receptor, Alpha-3 *GABRB3*** | Rs764926719 | CA dinucleotide repeat 171-201bp sized fragments | 181 |

132. *Id.* at Figure 4B
133. *Id.* at Figure 4C.

**FIGURE 2:** GARS SINGLE NUCLEOTIDE POLYMORPHISM ASSAY INFORMATION[134]

| | Gene & SNP | Context Sequence |
|---|---|---|
| **C____1011777_10** | *DRD1* rs4532 | TCTGATGACCCCTATTCCCTGCTT [G/A] GGAACTTGAGGGGTGTCAGAGCCCC |
| **C____7486676_10** | *DRD2, ANKK1* rs1800497 | CACAGCCATCCTCAAAGTGCTGGTC [A/G] AGGCAGGCGCCCAGCTGGACGTCCA |
| **C_____949770_10** | *DRD3* rs6280 | GCCCCACAGGTGTAGTTCAGGTGGC [C/T] ACTCAGCTGGCTCAGAGATGCCATA |
| **C____7470700_30** | *DRD4* rs1800955 | GGGCAGGGGAGCGGGCGTGGAGGG [C/T] GCGCACGAGGTCGAGGCGAGTCCGC |
| **C___25746809_50** | *COMT* rs4680 | CCAGCGGATGGTGGATTTCGCTGGC [A/G] TGAAGGACAAGGTGTGCATGCCTGA |
| **C___8950074_1_** | *OPRM1* rs1799971 | GGTCAACTTGTCCCACTTAGATGGC [A/G] ACCTGTCCGACCCATGCGGTCCGAA |

134. *Id.* at Figure 5.

**FIGURE 3:** GARS REPEATS PRIMER DETAILS[135]

| Primer | Sequence (5' to 3') | 5' Label | Reaction (nM) |
|---|---|---|---|
| **AMELO-F**<br>**AMEL0-R** | CCC TGG GCT CTG TAA AGA ATA GTG<br>ATC AGA GCT TAA ACT GGG AAG CTG | NED<br>- | 150 |
| **MAO-F**<br>**MAO-R** | ACA GCC TGA CCG TGG AGA AG<br>GAA CGG ACG CTC CAT TCG GA | NED<br>- | 120 |
| **DAT-F**<br>**DAT-R** | TGT GGT GTA GGG AAC GGC CTG AG<br>CTT CCT GGA GGT CAC GGC TCA AGG | 6FAM<br>- | 120 |
| **DRD4-F**<br>**DRD4-R** | GCT CAT GCT CTA CTG GGC<br>CTG CGG GTC TGC GGT GGA GTC TGG | VIC<br>- | 480 |
| **GABRA-F**<br>**GABRA-R** | CTC TTG TTC CTG TTG CTT TCA ATA CAC<br>CAC TGT GCT AGT AGA TTC AGC TC | NED<br>- | 120 |
| **HTTLPR-F**<br>**HTTLPR-R** | ATG CCA GCA CCT AAC CCC TAA TGT<br>GAG GGA CTG AGC TGG ACA ACC AC | PET<br>- | 120 |

135. *Id.* at Figure 6.

**FIGURE 4:** CLINICAL EVALUATION AND ASSESSMENT PROCESSES

| Substance Abuse Disorder Evaluations | | |
|---|---|---|
| Clinical Interview with Proband | Clinical Consult with Addiction Medicine Physician and Addiction Counseling Treatment Team | ReCAPS (Recovery Capital Measurement) |
| ASAM CONTIUUM (software algorithm) | COMPRIS (formerly McRISC) | WHOQOL-BREF (Quality of Life Measurement-World Health Organization) |
| BAM (Basic Addiction Monitor) | Assessment of 12-Step Program Involvement Inventory | RCQ-TV (Readiness to Change Questionnaire) |
| WAI (Working Alliance Inventory) | CSS-5 (Commitment to Sobriety Scale) | DERS (Difficulties in Emotional Regulation Scale)-18 |
| EQ (Emotional Quotient Score) | 16 PF (Personality Factors) | Dark Triad |
| MDQ (Mood Disorder Questionnaire) | PHQ-9 (Patient Health Questionnaire-9 Depression Screening) | GAD-7 (General Anxiety Questionnaire) |
| NPI (Narcissistic Personality Inventory) | THQ (Trauma History Questionnaire) | ACE (Adverse Childhood Experiences) |
| Resilience Questionnaire | | |

Other than substance use disorder evaluations, the subject was evaluated for the disorders associated with his mutations through interviews and examination.

**FIGURE 5:** PROBAND'S GENE POLYMORPHISMS

| The Proband's Gene Polymorphisms Linked to RDS Behaviors |
|---|
| The G allele of the dopamine COMT—ADHD, Oppositional Defiant (subject showed some traits, but did not meet the diagnostic criteria), Pathological Aggression, Panic Disorder, Anxiety, OCD, and Internet Gaming Addiction |
| **The A allele of the DRD1 receptor gene—Novelty Seeking issues such as:**<br>**1. Exploratory Excitability**<br>**2. Impulsiveness**<br>**3. Extravagance**<br>**4. Disorderliness** |
| The 4R of MAOA gene-Harm Avoidance (excessive worrying; pessimism; shyness; and being fearful, doubtful, and easily fatigued), ADHD, and Novelty Seeking |
| The C allele or 7R allele of the DRD4 receptor gene—ADHD, Novelty Seeking, Conduct Disorder, Hypersexuality, and Pathological Aggression |

## III. TREATMENT

The subject had four prior DWI convictions, as well as previous incarcerations—state prison and a state prison SUD treatment facility, each one-year stints consecutively. On the advice of his attorney, the subject entered treatment for AUD before adjudication of this charge.

While awaiting adjudication for more than a year, AG completed an eight-week Intensive Outpatient Program (IOP) for AUD, followed by continuing care. The IOP treatment consisted of nine hours of counseling per week.

**FIGURE 6:** IOP TREATMENT

| IOP Treatments the Subject Received While Awaiting Adjudication | | |
|---|---|---|
| Attended Group Process counseling sessions and Chemical Education classes three times a week | Attended once weekly Individual Counseling sessions, or Marriage Counseling sessions with a licensed counselor | The subject's wife infrequently engaged in one weekly Family Chemical Education class and one weekly Family Process group for eight weeks |
| Attended 2–3 Alcoholics Anonymous meetings per week | Worked with an Alcoholics Anonymous sponsor and completed AA steps 1–3 | Additionally, assessment by an addiction medicine physician confirmed the diagnosis of Alcohol Use Disorder, Severe |

The physician determined that Naltrexone therapy, in the form of either a subcutaneous implant or oral ingestion (50 mg QID), would be a viable option. The physician and legal team concurred that serial Naltrexone implants could be presented to the prosecution as a condition of probation for relapse risk mitigation. However, the subject declined to undertake Naltrexone therapy. Under the care of his addictionologist, he instead agreed to the GARS genetic test. The patient continued weekly one-hour group process and weekly individual therapy sessions after completion of IOP. In addition, twelve-step meeting attendance and engagement were maintained.

AG struggled with denial, marriage issues, trauma, and his family's continued use of alcohol. He reported a significant history of childhood physical abuse, specifically a physically abusive, alcoholic father. Family members substantiated this report. The subject's denial of his AUD revolved around his pathological drive to not "be like his father." AG's wife continued to use alcohol in the home and even reported providing alcohol for the couple's teenage child and the child's friends. Family counseling sessions addressed the prominence of the use of alcohol in the subject's extended family interactions and culture.

During IOP treatment, the subject pled guilty to his fifth DWI arrest, and a district (criminal court) judge scheduled a sentencing hearing. The proband was facing a probable five-year prison sentence.

## IV. RESULTS

The GARS test results were critical in addressing the subject's denial and mitigating his sentence. The results were reviewed with the subject to address his denial of having AUD. The results of the genetic testing showed a total of six alleles: DRD1- rs4532 (A/A), 2 alleles; DRD4 - rs761010487 (7R/7R), 2 alleles; COMT- rs4680 (A/G), 1 allele; MAOA - rs768062321 (4R), 1 allele.[136]

**FIGURE 7:** GARS TEST RESULTS

| **Single Nucleotide Polymorphisms (SNPs)** | | | | |
|---|---|---|---|---|
| **Gene** | **Identifiers** | **Risk Allele** | **Patient Results** | **Risk Allele Count** |
| COMT | rs4680 (Val158Met) | G | A/G | 1 |
| DRD1 | rs4532 | A | A/A | 2 |
| DRD2 | rs1800497 (Taq1A) | A | G/G | 0 |
| DRD3 | rs6280 | C | T/T | 0 |
| DRD4 | rs1800955 | C | T/T | 0 |
| OPRM1 | rs1799971 | G | A/A | 0 |
| **Variable Tandem Number Repeats & Insertion/Deletions** | | | | |
| **Gene** | **Identifiers** | **Risk Allele** | **Patient Results** | **Risk Allele Count** |
| DAT1 | rs28363170 | < than 9 repeats | 9R/10R | 0 |
| 5-HTT-LINKED | rs4795541 | S, LG | LA/LA | 0 |
| MAOA | rs768062321 (chrX*) | 3.5R, 4R | 4R | 1 |
| DRD4 | rs761010487 | ≥ 7 repeats | 7R/7R | 2 |
| **Dinucleotide Repeat** | | | | |
| **Gene** | **Identifiers** | **Risk Allele** | **Patient Results** | **Risk Allele Count** |
| GABRB3 | rs764926719 | 181 | 185/193 | 0 |

136. *See infra* Figure 7.

The Vincere Program and New Resources Medical Arts developed a legal brief from these evaluations and assessments which outlined findings: a genetically induced dopamine dysfunction, clinical diagnoses (Alcohol Use Disorder (Severe in Early Remission) and Reactive Attachment Disorder), and extensive long-term treatment and accountability recommendations. As mentioned earlier, Sequential Naltrexone Implant therapy for the duration of probation was offered and subsequently declined as an optional condition of probation.

With the assistance of the Vincere staff, the subject assembled a binder—including more than 300 pages of discovery—documenting his treatment and evaluations. The prosecution was given the binder and the information included therein the week before the sentencing hearing.

In Court, on the morning of the sentencing hearing, the prosecution presented a plea agreement to the defense. The plea deal mandated the subject to five years' probation (with ten nights in jail as a term of such probation), and continued treatment counseling for SUD and trauma issues until released by his therapist. Typical conditions of probation, such as the use of an ignition interlock in-car breathalyzer and moderate fines, were also imposed. The defendant eagerly accepted the plea offer.

This outcome in Bexar County Court is highly unusual—possibly unprecedented. Usually, defendants of DWI 4 or higher receive a minimum two-year prison sentence.[137] In fact, prison sentences of five years or more are common in these cases.[138]

A subsequent legal proceeding granted AG an occupational driver's license because he owns a commercial vehicle repair service. He continued for more than a year after adjudication to have family stressors and persisted in struggling, at times, with denial. Nevertheless, he remained sober and continues to stay sober at the time of this writing. He continues to attend AA, weekly process groups, twice-monthly individual counseling sessions, and has completed two and a half years of probation. AG volunteers with a national non-profit group that works to prevent drunk driving and often addresses groups on behalf of the organization as a speaker. He has had no positive urine tests, interlock violations, nor probation violations.

137. Committing and receiving a conviction for a fourth DWI offense, would ordinarily, under Texas's enhanced-penalty statute and the third-degree-felony statute, bring a penalty of two-to-ten years imprisonment. Tex. Penal Code §§ 49.09, 12.34.

138. *Id.* In fact, upon receiving a fourth DWI offense, the unpublished Texas appellate opinion in *Lewis v State of Texas,* affirmed a penalty of sixteen years of imprisonment. Lewis v. State, No. 01-15-00778-CR, 2016 WL 5400498 (Tex. App.—Hous. [1st Dist.] Sept. 27, 2016).

## V. DISCUSSION

There are some valuable clinical benefits related to the utilization of the GARS test. By reviewing the results of the GARS test and the impact of a DRD1 mutation, the subject was able to make significant strides towards breaking through his denial. The GARS test led to a high level of engagement in treatment and preparation for the legal case. Also, and of critical importance, the GARS test was a mitigating factor in sentencing. The GARS test results clearly illustrated the biologic factors involved in the defendant's AUD to the court.

The GARS test results indicated a need for pro-dopaminergic supplementation. It was recommended the subject begin taking the precision supplement, Equigen™, to help overcome dopamine deficits due to documented genetic predispositions leading to the reward deficiency and overall lack of well-being. The subject's compliance with this recommendation is unknown. Crucial to this discussion was the presence of the DRD1 mutation. This clinical finding is associated with "binge drinking." The binge drinking type of AUD is challenging to treat due to the fact that those afflicted appear at times to have control over their intake of alcohol. Excessive alcohol intake in and of itself is a serious but preventable public health problem in the United States and worldwide. "Alcohol and other substance use disorders occur co-morbidly with more generalized reward deficiency disorders, characterized by a reduction in dopamine signaling within the reward pathway, and classically associated with increased impulsivity, risk-taking, and subsequent drug-seeking behavior."[139] Increasing dopamine availability with nutrigenomic technologies, and thus restoring dopamine homeostasis in the mesocorticolimbic system,[140] could reduce the motivation to seek and consume alcohol. Recently, the Blum group, in conjunction with Gondre-Lewis's group, treated animals with KB220Z, also known as pro-

139. Naimesh Solanki et al., *Administration of a Putative Pro-Dopamine Regulator, a Neuronutrient, Mitigates Alcohol Intake in Alcohol-Preferring Rats*, BEHAV BRAIN RES., May 15, 2020, https://www.sciencedirect.com/science/article/abs/pii/S0166432819318169 [https://perma.cc/5J76-F2KW].

140. The mesocorticolimbic—comprised of two central dopaminergic pathways: the mesolimbic and mesocortical—has "been implicated as key circuits that are disrupted in addictive behaviors." Vani Pariyadath et al., *Neuroscience for Addiction Medicine: From Prevention to Rehabilitation—Methods and Interventions*, *in* 224 PROGRESS IN BRAIN RESEARCH 155, 155 (Hamed Ekhtiari & Martin P. Paulus eds., 2016) (internal citation omitted).

dopaminergic neuro-nutrient, designed to augment dopamine signaling.[141] Along these lines, Solanki administered KB220Z "to genetically alcohol-preferring (P) adult male and female rats by oral gavage (PO), intraperitoneally (IP), or subcutaneously (SQ) for four consecutive days at a 3.4 mL/Kg rat equivalent dose and compared such findings to saline (SQ, IP) or water (PO) controls."[142] After treatment, "lever pressing and consumption of 10% ethanol or control 3% sucrose during operant responding were assessed using a drinking in the dark multiple scheduled access [(DIDMSA)] binge drinking protocol."[143]

Locomotor and elevated zero maze (EZM)[144] activity and DRD2 mRNA expression via in situ hybridization assessed independently following four days of the SQ regimen of KB220Z markedly and immediately reduced binge drinking of 10% ethanol in both male and female rats.[145] There was no effect of SQ KB220Z on 3% sucrose drinking, whereas PO administration took at least three days to decrease lever pressing for ethanol in both male and female rats.[146] Elevated activity in the open field decreased significantly, and time spent in the open arm of the EZM decreased moderately.[147] The regimen of SQ KB220Z did not impact the number of DRD2 punctae in neurons of the NAc,[148] but the NAc shell expressed more DRD2 mRNA/cell than NAc core independent of KB220Z.[149]

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141. *See generally* Kenneth Blum et al., *Researching Mitigation of Alcohol Binge Drinking in Polydrug Abuse: KCNK13 and RASGRF2 Gene(s) Risk Polymorphisms Coupled with Genetic Addiction Risk Severity (GARS) Guiding Precision Pro-Dopamine Regulation*, 12 J. PERSONALIZED MED. (2022).

142. Solanski, *supra* note 139.

143. *Id.*.

144. Laura B. Tucker & Joseph B. McCabe, *Behavior of Male and Female C57BL/6J Mice Is More Consistent with Repeated Trials in the Elevated Zero Maze than in the Elevated Plus Maze*, 11 FRONT. BEHAV. NEUROSCI. 1, 1 (2017) ("[E]levated zero maze (EZM) [is a] behavioral test[] that [is] widely employed to assess anxiety-like behaviors in rats and mice following experimental manipulations, or to test the effects of pharmacological agents. Both tests are based on approach/avoidance conflict, with rodents perceived as 'less anxious' being more willing to explore the brighter, open and elevated regions of the apparatus as opposed to remaining in the darkened and enclosed regions.").

145. Solanski, *supra* note 139.

146. *Id.*

147. *Id.*

148. Zhao Li et al., Abstract, *Cell-Type-Specific Afferent Innervation of the Nucleus Accumbens Core and Shell*, 12 FRONT. NEUROANAT. 1, 1 (2018) ("The nucleus accumbens (NAc) is [] implicated in reward processing and drug addiction, as well as in numerous neurological and psychiatric disorders[.]").

149. Rutsuko Ito & Anja Hayen, *Opposing Roles of Nucleus Accumbens Core and Shell Dopamine in the Modulation of Limbic Information Processing*, 31 J. OF NEUROSCI. 6001, 6001 (2011) ("The NAc itself is differentiated into at least two anatomically and functionally distinct regions, the core and the shell,

The importance of these confirming animal results is that they provide significant evidence of effective treatment of binge drinking by the administration of a neuro nutrient pro-dopamine regulator. In AG's case, the gene specific KB220Z formulation is Equigen. Apropos the question as to whether treatment of genetic determinism in human binge drinking is equal to the animal experiment with KB220? Blum's group has recently studied this question with positive outcomes utilizing this known "Precision Behavioral Management."[150] The subject's compliance with Equigen is being monitored and is the subject of additional research.

Based on AG's GARS score having the COMT and MAOA high activity alleles, catabolism of both low serotonin and dopamine in the synapse and mitochondria is abnormal.[151] AG also has two copies of the DRD4 equal or greater than 7 R that can result in sensation seeking,[152] along with other destructive behaviors as discussed earlier in this paper.

## VI. LIMITATIONS & FUTURE PERSPECTIVES

While we are proposing "genetically induced at birth determinism" compared to free will, we are cognizant of the epigenetic or environmental aspects of this novel modality in terms of adjudication of multiple DWIs and subsequent incarceration, not rehabilitation. We are presenting only one case study as a precedent for how evidence of "determinism" and not "free will" in genetic defense of addiction and subsequent conversion of incarceration to probation and rehabilitation (sentencing mitigation), based on the Genetic Addiction Risk Score (GARS). Candidly, we are not accepting this harmful behavior. Based on AG's GARS, he has a moderately increased risk for AUD, but a high increased risk for other drugs such as opioids. These phenotypes, as expressed in this proband, can be

with different but overlapping patterns of limbic connectivity. Thus, while the NAc shell receives converging limbic inputs from the BLA and ventral subiculum, the major output region of the hippocampus (HPC), the NAc core receives inputs from the BLA and parahippocampal regions.") (internal citation omitted).

150. *See generally* Kenneth Blum et al., *A Review of DNA Risk Alleles to Determine Epigenetic Repair of mRNA Expression to Prove Therapeutic Effectiveness in Reward Deficiency Syndrome (RDS): Embracing "Precision Behavioral Management"*, 14 PSYCHO. RES. BEHAV. MANAG. 2115 (2021).

151. Kenneth Blum et al., *Manipulation of Catechol-O-Methyl-Transferase (COMT) Activity to Influence the Attenuation of Substance Seeking Behavior, a Subtype of Reward Deficiency Syndrome (RDS), is Dependent upon Gene Polymorphisms: A Hypothesis*, 69 Med. Hypotheses 1054, 1054 (2007).

152. Ernest P. Noble et al., *D2 and D4 Dopamine Receptor Polymorphisms and Personality*, 81 Am. J. Med. Genetics 257, 257 (1998) ("Boys with the DRD4 7 repeat (7R) allele also had a significantly higher Novelty Seeking score than those without this allele.").

characterized by utilizing GARS, and dopamine homeostasis achieved, as discussed earlier, via "Precision Behavioral/Addiction Management" customization of neuronutrient supplementation based on the GARS test result, along with many behavioral interventions. The pro-dopamine nutrigenomic is therapeutic per se, and its longitudinal impact on cases such as AG, is the subject of planned, systematic assessment of outcomes that will provide an evidence-based medical necessity for the incorporation of the GARS test with the KB220Z PBM. Finally, it is prudent to note that currently, there are at least thirty-eight published studies in both animals and humans showing robust positive outcomes, including "[decreased] AMA [(Against Medical Advice)] rate, attenuation of craving behavior, reward system activation including BOLD [(Blood Oxygen Level Dependent)] dopamine signaling, relapse prevention, as well as reduction in stress, anger, and aggressive behaviors,"[153] even DUIs.

The legally or socially minded reader will be curious to know what fruit this bears for individuals charged with AUD / SUD related offenses, especially in Texas.

Texas is one of only nine states in the country that does not have a lookback period. A lookback period "is the length of time that a drunk driving offense remains on a driver's record . . . [and] is the timeframe used to determine whether previous offenses can be taken into consideration."[154] In Texas, a DWI arrest allows prosecutors to consider every DWI conviction in an individual's life, even if they are decades old. As mentioned previously, many factors can escalate the potential prison time a client is facing.[155] First and foremost is the number of previous DWI convictions.[156] Additionally, causing significant injury or permanent disfigurement (intoxication assault), or having a minor in the vehicle at the time both raise the charge to a felony and influence sentencing.[157] Blood Alcohol Concentration (BAC) over 0.15 at the time of the offense is also an

153. *See generally* Kenneth Blum et al., *Pro-Dopamine Regulator (KB220) A Fifty Year Sojourn to Combat Reward Deficiency Syndrome (RDS): Evidence Based Bibliography (Annotated)*, 1 CPQ NEUROLOGY & PSYCH. 2 (2018) (concluding, based on various published studies, KB220 "shows promise in the addiction and pain space") https://www.cientperiodique.com/journal/fulltext/CPQNP/1/2/13 [https://perma.cc/UJX5-RNJU].

154. *State Law: DUI Look-Back Periods*, FOUND. FOR ADVANCING ALCOHOL RESP., https://www.responsibility.org/alcohol-statistics/state-map/issue/dui-look-back-periods/ [https://perma.cc/DJ4R-QKG2].

155. *See supra* notes 27–31.

156. TEX. PENAL CODE ANN. § 49.09 (West 2011).

157. *Id.* at §§ 49.07, 49.045.

escalator, raising the charge to a Class A misdemeanor.[158] In Texas, as in many states, there are prisoners with arrests and circumstances similar to this defendant. Those prisoners will live decades, maybe even the rest of their lives, in prison.

Beyond the patient described herein, sixteen individuals managed by AG's treatment provider have entered into treatment rather than prison, and another eight cases are awaiting adjudication. The sixteen adjudicated cases were facing up to a cumulative total of 198.75 years (1,741,050 hours) in prison or state jail (low-security prison) in Texas. Since the sample size (n=20) is small, more conservative nonparametric paired sample tests were conducted in order to detect the statistical significance of jail time difference before and after GARS testing. The (pseudo) median jail time saved after GARS is 10.5 years (91,980.2 hours). The two-sided Wilcoxon signed rank test with continuity correction detected a very strong statistical significance with p-value = 8.953x10^-5 along with the 95% confidence interval of (6.5 years, 15.0 years) or equivalently, (56,940.2 hours, 131,400.5 hours). The one-sided Wilcoxon signed rank test with continuity correction also detected a very strong statistical significance of jail time saving with p-value = 4.477x10^-5 along with the 95% confidence interval of at least 8.5 years or, equivalently, 74,460.4 hours. Cumulatively, in other words, *these sixteen patients received .0008213434—less than one ten-thousandth—of the time they were facing.* This team is actively tracking these patients and continuing to develop this unpublished research for future peer review.

## VII. CONCLUSION

We present a case of a presently abstinent (2.5 years), thirty-five year old (at time of adjudication) alcoholic male (AG) of Hispanic descent who has five DWI convictions on his record, as well as a previous incarceration of two years for DWI. AG has undergone and continues to be engaged in out-patient SUD treatment. He entered treatment before adjudication and was mandated by the court to continue treatment to assist in maintaining sobriety. Treatment included the administration of the GARS test for genetic addiction risk. AG was facing a probable five-year sentence for his fifth DWI conviction in Bexar County, Texas (San Antonio). However, based on the genetic risk results showing a total of six SOMETHING alleles, a brief developed indicated a genetically induced dopamine dysfunction, hypodopaminergia. The presiding judge adjudicated AG to be

158. *Id.* at § 49.04(d).

mandated to 5 years' probation and required to continue treatment and monitoring for rehabilitation—an exceptionally rare legal outcome for this type of offense. Most often, the fifth DWI arrest leads to a custodial prison sentence for the offender.

We are cognizant that probands could use the relative idea of "determinism" vs. "free–will" as an excuse to use alcohol, but this is both unacceptable and unlikely. Defendants involved in a court proceeding for DWI have good reason to seek treatment for their genetically determined severe AUD. Here, the GARS test result and the individualized long-term treatment influenced by the GARS test results was a mitigating factor in sentencing. To our knowledge, this is a noteworthy legal precedent that utilizes genetic information to advocate for rehabilitation instead of incarceration in SUD cases, especially for individuals with multiple DWI convictions. Courts fundamentally want to help people—society as a whole, victims, and accused offenders. The use of the GARS test to identify issues and plan for the rehabilitation of accused offenders gives courts a valuable tool in their adjudicatory repertoire and advances their ability to resolve cases effectively.