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Reviewing the Fault Line -
*Monoamine Oxidase-A Genotype Evidence and
the Criminal Law*

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Abstract

Recent advances in behavioural genetics suggest that there is a significant genetic component associated with the risk of criminality. This paper focusses on the MAOA gene, a gene which has been linked to aggressive and antisocial behaviour, and analyses what role such genetic evidence should play in the criminal law. In particular, this paper will explore the role of genetic predisposition evidence during sentencing, and will reflect on some associated ethical concerns, and the dangers of misinterpretation. This research highlights that genetic predisposition evidence may be relevant in sentencing. However, there is a potential for the evidence to be construed as both an aggravating and mitigating factor.

This is a field in which we must proceed with care. Science has a huge potential to assist decision makers, improve the criminal process and allow justice to be done. However, the other side of the coin is misinterpretation and abuse.

Key words: Monoamine Oxidase A, Sentencing, Criminal Law, Behavioural Genetics, Bioethics.

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I Introduction

The use of genetic evidence in the courtroom has been marred by misunderstanding and controversy. In the 1960s, creative defence counsel argued that their clients' violent actions were a product of their genes.¹ These claims related to XYY syndrome, a condition where males carry an extra Y chromosome in their cells. Some papers had suggested that this genotype was correlated with aggressive behaviour. However, these findings have been largely discredited.² Indeed, the link between XYY and criminal behaviour has been described as founded upon "breathtakingly atrocious methodology".³

Despite the controversy, aggressive behaviour is deeply grounded in genetics. While aggression may be influenced by situational factors, there is a significant body of evidence showing that some people "have a disposition to behave aggressively."⁴ Findings from neuroimaging analysis have also "identified structural and functional differences in regions of the prefrontal cortex and areas of the limbic system in offenders compared to non-offenders."⁵ This supports the conclusion that there may be something physically different between some criminals and the rest of the population.

¹ Debra Wilson *Crime Genetics and Justice* (Edward Elgar Publishing, Northampton, 2015) at 77. For a discussion of the use of XYY evidence by lawyers see Richard Fox "The XYY Offender: A Modern Myth?" (1971) 62 *The Journal of Criminal Law, Criminology and Police Science* 59.

² Paul Appelbaum "The Double Helix Takes the Witness Stand: Behavioral and Neuropsychiatric Genetics in Court" (2014) 82 *Neuron* 946 at 946.

³ Debra Wilson, above n 1, at 80.

⁴ David Chester and others "Monoamine oxidase A (MAOA) genotype predicts greater aggression through impulsive reactivity to negative affect" (2015) 283 *Behav Brain Res* 97; Deborah Denno "Courts' Increasing Consideration of Behavioural Genetics Evidence in Criminal Cases: Results of a Longitudinal Study" (2011) *Mich St L Rev* 967 at 972, explaining that "at least one hundred studies indicate a tie between genetics and criminality".

⁵ KM Beaver, JC Barnes and BB Boutwell "The 2-Repeat Allele of the MAOA Gene Confers an Increased Risk for Shooting and Stabbing Behaviors" (2014) 85 *Psychiatry Q* 257 at 258.

In recent years, scientific studies have begun to draw associations between specific genes and aggressive or antisocial behaviour. One gene of note, Monoamine Oxidase A (MAOA), has been the subject of extensive scientific focus, and represents the “best-validated genetic basis of aggression and antisocial behavior”.⁶ Studies have established a solid association between a particular allele of the MAOA gene referred to as MAOA-L, and antisocial or aggressive behaviour. It is this allele which is the focus of this paper.

In the last decade, MAOA has featured in a few court cases, leading to considerable controversy. For example, in 2009 an Italian court gave Abdelmalek Bayout a discounted sentence on the basis that his genes constituted a mitigating factor. The same year, a jury in Tennessee, USA, acquitted Bradley Waldroup of first degree murder, after evidence of a genetic predisposition towards violence was introduced by defence counsel.

The use of this evidence in the courtroom, and its popular portrayal in the media has been the subject of stern criticism from some members of the scientific community. Popular discourse around MAOA has also been tainted by racist undertones.⁷ This paper seeks to cut through the controversy and explore the validity of arguments relating to the use of MAOA evidence, and the implications which behavioural genetics research casts upon notions of criminal responsibility. In order to introduce the relevant background, Part II of this paper outlines the basic principles governing criminal liability, defences and sentencing. Part III will build upon this background by explaining the underlying science, and recent developments in the field of behavioural genetics. Part IV explores how some courts have dealt with MAOA genotype evidence in the past. Part V will then analyse why MAOA genotype evidence should not be relevant at the liability stage. Part VI will discuss the practical implications relating to the use of MAOA genotype evidence in sentencing

⁶ Sean Godar and others “The role of monoamine oxidase A in aggression: Current translational developments and future challenges” (2016) 69 *Prog Neuropsychopharmacol Biol Psychiatry* 90 at 90. In similar terms Chester et al. note that the “low functioning allelic variants of the monoamine oxidase A (MAOA) gene have emerged as uniquely potent correlates with violence” see David Chester and others, above n 4.

⁷ Alondra Oubré “The Extreme Warrior gene: a reality check” (31 July 2014) *Scientia Salon* <<https://scientiasalon.wordpress.com/2014/07/31/the-extreme-warrior-gene-a-reality-check/>>.

and parole decisions, and its role as both an aggravating and mitigating factor. Part VII will finish by exploring some of the ethical concerns posed by the use of such evidence.

II Criminal Liability, Defences, and Sentencing

Before embarking on an analysis of the issues raised by MAOA genotype evidence it is necessary to outline some of the background principles relating to criminal liability and sentencing. An exploration of this background is necessary to properly understand and evaluate the arguments which will be presented in this paper.

One of most obvious issues which arises when one considers evidence suggesting a predisposition towards aggression, is that of free will and criminal liability. It seems intuitive that notions of fault should depend on an actor's ability to refrain from a prohibited action. Accordingly, we are less inclined to attribute fault and responsibility where evidence, such as predisposition evidence, casts doubt upon an actor's ability to act otherwise. Thus, this is an appropriate place to commence this discussion.

A The Legal Myth of Free Will

There has long been a divide between two schools of philosophical thought; those of "free will" and "determinism". Free will refers to the "unique ability of persons to exercise control over their conduct in the fullest manner necessary for moral responsibility" whereas determinism represents the view "that every event is causally necessitated by antecedent events".⁸

From a philosophical perspective, the idea of individual responsibility is largely incompatible with theories of absolute determinism. Notions of blame are based on the extent to which an actor is responsible for an event – in other words whether an outcome

⁸ Colin Gavaghan and Amir Bastani "Genes, blame and loss of control: is there a place in criminal law for a 'genetic defense'?"
<https://www.researchgate.net/publication/272520354_Genes_Blame_and_Loss_of_Control_is_there_a_Place_in_Criminal_Law_for_a_'Genetic_Defense'> at 6.

is “voluntarily and knowingly chosen” as opposed to being beyond one’s control.⁹ If our actions could be attributed to factors beyond our control, such as our genes, then at a philosophical level, this would undermine a system of responsibility founded upon notions of individual fault.

Recent developments in the field of behavioural genetics have the potential to reshape the contours of the determinism/free will debate. Science, by its very nature seeks to provide mechanistic explanations for natural, physical or chemical phenomena. Scientific theory proceeds on the basis that observations are logically a product of something else. As the behavioural sciences progress, it may become difficult to cling to theories of free will.

Within the criminal law, the concept of moral blameworthiness is also widely regarded as laying the foundation for the imposition of liability.¹⁰ As a starting point, the criminal law regards its subjects as rational beings with the ability to refrain from behaviour prohibited by the law. It is on this basis that the law apportions responsibility. As HLA Hart notes:¹¹

Man’s fate should depend on his choice and this is to foster the prime social virtue of self-restraint.

This idea of choice is fundamental. The criminal law is concerned with “choices that individuals make as expressed through action, and not simply with an individual’s actions or character.”¹² In recognition of the importance of choice, the criminal law adopts a concept of mens rea, which assumes an objective, and a subjective form. The subjective standard is concerned with the actual mental state of the offender, whereas the objective standard focuses on the defendant’s departure from reasonable standards of practice. As

⁹ At 7.

¹⁰ Debra Wilson, above n 1, at 151.

¹¹ HLA Hart *Punishment and Responsibility* (Oxford University Press, Oxford, 1968) at 183.

¹² Sana Halwani and Daniel Krupp “The Genetic Defence: The Impact of Genetics on the Concept of Criminal Responsibility” (2004) 12 Health LJ 35 at 37.

Halwani and Krupp explain:¹³

The most serious criminal offences - such as murder - require proof of subjective mens rea; that is, they require guilty knowledge in relation to the circumstances or consequences specified in the offence. These offences therefore require the trier of fact to make a finding about what the accused actually knew or intended.

Although objective standards reduce the criminal law's ability to tailor punishment to an actor's level of blameworthiness... These offences still encompass a certain degree of fault, that being a "marked departure" from the standard of the reasonable person.

Within this framework, there exist several defences, although the availability of specific defences depends on the jurisdiction. Excusatory defences accept "that while society does not necessarily approve of the individual's actions, it recognizes that there is a good reason why the person should not be considered culpable".¹⁴ Justificatory defences take the form of an acknowledgement that "the defendant acted out of necessity and acted reasonably".¹⁵ Defences usually relate to "factors affecting an individual's capacity to decide to violate a law".¹⁶ Indeed, under the common law, "complete impossibility of compliance with the law will almost always lead to a complete acquittal".¹⁷ For example, in *R v Bell*, Lord Goff noted that criminal liability would not attach to involuntary conduct, such as where a motorist crashes after being attacked by a swarm of bees, or due to a brake failure.¹⁸ Likewise, defendants who offend while sleepwalking are not liable for their actions.¹⁹

New Zealand criminal law provides for several defences, such as automatism and insanity. Both these defences recognise that the defendant was unable to refrain from breaking the law, either because their actions were involuntary in the case of automatism, or in the case

¹³ At 37.

¹⁴ Debra Wilson, above n 1, at 151.

¹⁵ At 151.

¹⁶ At 152.

¹⁷ Colin Gavaghan and Amir Bastani, above n 8, at 8.

¹⁸ *R v Bell* [1985] RTR 202 at 207.

¹⁹ *R v Burgess* [1991] 2 QB 92.

of insanity, because their “disease of the mind” rendered them incapable of “understanding the nature and quality of the act or omission” or “knowing that the act or omission was morally wrong”.²⁰ These two defences are regarded as full or exculpatory defences. If successfully raised, they will absolve the defendant from liability. Such defences “are necessary as an expression of the criminal justice system's respect for the individual as a choosing being.”²¹ Other defences such as self-defence, are founded on policy considerations, such as the right of a person to defend themselves.

However, within this framework:²²

Factors relating to a person's background, including biological or environmental factors, are generally regarded by the law as not being sufficiently causative to affect this capacity [to refrain from breaking the law] and are therefore not relevant in determining criminal responsibility.

For example, in *Pohlot*, the US Court of Appeals for the Third Circuit noted that:²³

Criminal responsibility must be judged at the level of the conscious. If a person thinks, plans and executes the plan at that level, the criminality of his act cannot be denied, wholly or partially, because, although he did not realize it, his conscious was influenced to think, to plan and to execute the plan by unconscious influences which were the product of his genes and his lifelong environment.

Thus, an individual who has full control of their mental faculties cannot argue that certain negative social or genetic influences operated at a subconscious level to guide his or her conduct. This view is a sensible one. If the subconscious influence argument were accepted, then every defendant could potentially raise the defence, and the criminal law would be deprived of any effect. At some deeper level, every person is a product of their genes and their past experiences. Criminal responsibility must therefore be determined at the level of

²⁰ Crimes Act 1961, s 23.

²¹ Sana Halwani and Daniel Krupp, above n 12, at 39.

²² Debra Wilson, above n 1, at 151.

²³ *US v Pohlot* 827 F2d 889.

the conscious actor, by examining the extent of one's criminal intent at the time of the commission of the offence. This is necessary to ensure that the objectives of punishment, namely deterrence and retribution²⁴ are achieved.

In this sense, the free will/determinism dichotomy is not strictly relevant in the criminal law. The criminal law bypasses this debate, and it does so in order to maintain the effectiveness of the criminal sanction. Herbert Packer explains why:²⁵

The idea of free will in relation to conduct is not, in the legal system, a statement of fact, but rather a value preference having very little to do with the metaphysics of determinism or free will... Very simply, the law treats man's conduct as autonomous and willed, not because it is, but because it is desirable to proceed as if it were.

Therefore, evidence of a disadvantaged background cannot be used to negate criminal liability. However, such evidence is open to consideration at the sentencing stage as a mitigating factor, as is any other evidence which reflects upon the defendant's culpability. The next section of this paper will explore how this sentencing process operates, and the role which mitigating and aggravating evidence plays.

B Principles of Sentencing

A fundamental principle of sentencing is that a sentence should be proportionate to the gravity of the offence, and the culpability of the offender. As LeBel J explained in the Canadian Supreme Court decision of *R v Ipeelee*:²⁶

Proportionality is the *sine qua non* of a just sanction. First, the principle ensures that a

²⁴ Hart explains that the criminal law should serve the ideals of both retribution and deterrence: "even if we regard the overall purpose of punishment as that of protecting society by deterring persons from committing crimes and insist that the penalties we inflict be adapted to this end, we can in perfect consistency and with good reason insist that these punishments be applied only to those who have broken a law and to whom no excusing conditions apply." HLA Hart "Legal Responsibility and Excuses" in ML Corrado (ed) *Justification and Excuse in the Criminal Law: A Collection of Essays* (Garland, New York, 1994) at 31.

²⁵ Herbert Packer *The Limits of the Criminal Sanction* (Stanford University Press, Stanford, 1968) at 74-75.

²⁶ *R v Ipeelee* [2012] 1 RCS at [37].

sentence reflects the gravity of the offence. This is closely tied to the objective of denunciation. It promotes justice for victims and ensures public confidence in the justice system. As Wilson J. expressed...

“It is basic to any theory of punishment that the sentence imposed bear some relationship to the offence; it must be a “fit” sentence proportionate to the seriousness of the offence. Only if this is so can the public be satisfied that the offender “deserved” the punishment he received and feel a confidence in the fairness and rationality of the system.”

Second, the principle of proportionality ensures that a sentence does not exceed what is appropriate, given the moral blameworthiness of the offender. In this sense, the principle serves a limiting or restraining function and ensures justice for the offender.

The consideration of mitigating and aggravating factors in sentencing is essential to ensure that the sentence given is proportionate to the offender’s culpability. Such considerations, along with appropriate regard for the harm done by the offending are fundamental. Proportionality in sentencing is essential to promote public confidence in the criminal system, and to ensure justice for the defendant.

The New Zealand sentencing system is based on the traditional common law discretionary model. Within this framework, the legislature prescribes the maximum penalty for an offence, and the types of sentences available, but otherwise leaves the judiciary with a wide discretion to tailor the sentence to the crime.²⁷ The judiciary can also seek guidance from appellate decisions, presentence reports and submissions from counsel.²⁸

Since 2002, the Sentencing Act has imposed additional constraints upon the exercise of judicial discretion during the sentencing process. The Act also provides much more comprehensive guidance.²⁹

²⁷ Warren Young and Andrea King “Sentencing Practice and Guidance in New Zealand” (2010) 22 Fed Sentencing Report 254 at 254.

²⁸ At 254.

²⁹ At 255.

In practice, New Zealand courts apply a two-stage approach when deciding on the appropriate length of a sentence of imprisonment. Firstly, the court looks at the offence and determines the appropriate starting point which “[reflects] the gravity of the offending conduct.” Then, the court looks at the circumstances of the offender and adjusts the sentence to give effect to any relevant mitigating or aggravating factors.³⁰ The structure of this analysis helps ensure that the sentence given is proportionate to the offender’s culpability and the severity of the offence.

Common examples of mitigating factors include youth,³¹ evidence of childhood maltreatment,³² or a timely guilty plea.³³ Classic aggravating factors include the use of a weapon,³⁴ cruelty,³⁵ or if the “offender was abusing a position of trust or authority in relation to the victim”.³⁶

C Framework for Analysis

Before evaluating claims based on MAOA genotype evidence, it is necessary to consider the different stages at which such evidence might be relevant in the criminal law. For the purposes of this paper, these stages are: (1) the question of mens rea, (2) in the context of a defence of insanity or automatism, (3) as a mitigating factor during sentencing, or (4) in the context of parole risk assessment. However, in order to properly analyse the relevance and utility of MAOA genotype evidence it is necessary to explore the underlying science. A discussion about genetics, without reference to the actual science would be quite fruitless and divorced from reality.

³⁰ *Hessell v R* [2010] NZSC 135 at [16]; *R v Taueki* [2005] 3 NZLR 372 (CA).

³¹ Sentencing Act 2002, s 9(2)(a); *Churchward v R* [2011] NZCA 531, (2011) 25 CRNZ 446 at [77].

³² *Pitceathly v R* [2010] NZCA 95 at [27]; *R v DJH* [2015] NZHC 1786 CRI-2014-070-001585 at [25]-[26].

³³ Sentencing Act 2002, s 9(2)(b).

³⁴ Section 9(1)(a).

³⁵ Section 9(1)(e).

³⁶ Section 9(1)(f).

III MAOA-L and Behavioural Genetics

A The MAOA Gene

The MAOA gene is found on the X-chromosome and codes for the enzyme monoamine oxidase A (MAO-A).³⁷ This enzyme has a vital role in the degradation of neurotransmitters such as serotonin, noradrenaline, and dopamine.³⁸ These neurotransmitters are involved in aggression, emotion and cognition.³⁹ Many studies support the role of MAOA in aggression. The first paper to highlight this link was published by Brunner et al. in the early 1990's.

Figure 1: Enzyme Structure of Human Monoamine Oxidase A⁴⁰



³⁷ For an overview of gene expression see Jonathan Kaplan “Misinformation, Misrepresentation, and Misuse of Human Behavioural Genetics Research” (2006) 68 Law Contemporary Problems 47.

³⁸ Sean Godar and others, above n 6, at 90.

³⁹ Dolores Garcia-Arocena “The Genetics of Violent Behaviour” (7 December 2015) Jackson Laboratory <<https://www.jax.org/news-and-insights/jax-blog/2015/december/the-genetics-of-violent-behavior>>.

⁴⁰ Luigi De Colibus and others “Three-dimensional structure of human monoamine oxidase A (MAO A): Relation to the structures of rat MAO A and human MAO B” (2005) 102 PNAS 12684.

B Brunner's Syndrome

In 1993, Brunner et al. published a paper describing a rare X-linked recessive disorder within an extended Dutch family. This disorder, later termed Brunner's syndrome, represents a functional knock-out of the gene, which means that affected men completely lack MAO-A.⁴¹ The syndrome involves a nonsense point mutation in the MAOA gene – a mutation leading to a complete loss of expression of the enzyme. In the family studied, male members with Brunner's syndrome, displayed “mild mental retardation; predisposition to aggressive outbursts, especially in response to frustration, anger and fear; and violent impulsive behaviour.”⁴² Indeed, within this family:⁴³

one man had raped his sister and while subsequently serving time in a mental institution had attacked his warden with a pitchfork. Another had tried to run his boss over with a car after being told that his work was not acceptable. A further two were arsonists.

Interestingly, further experiments conducted with MAOA knockout mice showed that mice afflicted by this mutation displayed similar symptoms to those characterised by Brunner's syndrome.⁴⁴

However, far from providing a simple genetic explanation for aggressive behaviour, the results of the study were of limited application. Brunner's syndrome is exceptionally rare.⁴⁵ Despite this, the publication of the research drew widespread attention from the scientific community. In the years that followed, the MAOA gene was the target of much scientific focus. But as has often been the case in biology, further research, rather than providing a clear answer, has unearthed deeper complexities.

⁴¹ Hans Brunner and others “Abnormal behaviour associated with a point mutation in the structural gene for monoamine oxidase A” (1993) 262 *Science* 578.

⁴² Joshua Buckholtz and Andreas Meyer-Lindenberg “MAOA and the neurogenic architecture of human aggression” (2008) 31(3) *Trends Neurosci* 120.

⁴³ Debra Wilson, above n 1, at 85.

⁴⁴ Sean Godar and others, above n 6, at 92.

⁴⁵ Mairi Levitt “Crime Genes” (last accessed 4 August, 2017) *The Biologist*, Royal Society of Biology <<https://thebiologist.rsb.org.uk/biologist-features/158-biologist/features/903-crime-genes>>.

C *MAOA-L and MAOA-H Alleles*

Besides the loss of function mutation associated with Brunner's syndrome, studies have discovered multiple alleles of the MAOA gene. As Mathew Baum explains:⁴⁶

In the gene's promoter, a region that controls transcription and expression efficiency, there are a variable number of nucleotide tandem repeats (VNTR)... While the VNTRs range from 1 to 5 repeats, genes with three or four repeats are most common. Approximately 30% of the alleles (gene copies) in the general population contain three repeats while approximately 65% contain 4. Interestingly, in vitro studies indicate that the variant with three repeats is expressed significantly less efficiently than the variant with four repeats, although there is conflicting data about whether this is also true in humans.

This distinction is important because a robust association has been reported between alleles associated with low enzyme activity (MAOA-L) and antisocial or violent activity.⁴⁷ In contrast, the alleles which are associated with higher enzyme activity (MAOA-H) have not been associated with aggressive behaviour. Typically, the 2 and 3 repeat alleles (VNTR-2 and VNTR-3) are grouped into the MAOA-L category whereas VNTR-3.5, VNTR-4 and VNTR-5 form the MAOA-H category.⁴⁸ However, it is sometimes unclear in papers whether the term MAOA-L refers to both 2 and 3 repeat alleles, or just the 3 repeat variant.⁴⁹

The matter is more complex when it comes to women. As Mairi Levitt explains:⁵⁰

⁴⁶ Matthew Baum "The Monoamine Oxidase A (MAOA) Genetic Predisposition to Impulsive Violence: Is It Relevant to Criminal Trials?" (2013) 6 *Neuroethics* 287 at 291.

⁴⁷ Sean Godar and others, above n 6.

⁴⁸ KM Beaver, JC Barnes and BB Boutwell, above n 5, at 258. The 5-repeat allele has also been described as falling within the MAOA-L category, see Macià Buades-Rotger and David Gallardo-Pujol "The role of the *monoamine oxidase A* gene in moderating the response to adversity and associated antisocial behavior: a review" (2014) 7 *Psychol Res Behav Manag* 185 at 187.

⁴⁹ See Alondra Oubré, above n 7.

⁵⁰ Mairi Levitt, above n 45; also see Macià Buades-Rotger and David Gallardo-Pujol, above n 48, at 187. Buades-Rotger and Gallardo-Pujol, at 192, also suggest that "[f]emales' heterozygosis may help to compensate [for] the detrimental effects of the *MAOA-L* allele through developmental deactivation of the X chromosome that carries the low-activity allele."

[F]emales have two copies of the X chromosome, they don't neatly divide into low and high groups. Nearly half have a low-high MAOA combination and it is not known which allele is active in an individual. Only 12% of women had the low-low variant.

D The Caspi Study

The MAOA-L allele, or alleles have been the subject of much investigation. One ground-breaking discovery was made by Caspi et al. in 2002. The researchers observed that male carriers of the MAOA-L variant who had *also* experienced childhood maltreatment were statistically more likely to display antisocial behaviours and be convicted of violent crimes.⁵¹ This was an example of a *gene x environment* interaction, where the risk attributed to the combination of environmental and genetic factors exceeded the additive risk of the individual factors.⁵²

Notably, men with MAOA-L and a history of severe maltreatment comprised 12% of the sample population, but accounted for 44% of the violent convictions.⁵³ The risk presented by the gene environment interaction was held to be comparable to that posed by “the major risk factors associated with cardiovascular disease”.⁵⁴ Indeed “85 per cent of those with [l]ow MAOA who were severely maltreated developed antisocial behaviour.”⁵⁵ In contrast, among males with high MAOA activity, “maltreatment did not confer significant risk for violent conviction.”⁵⁶

⁵¹ Avshalom Caspi and others “Role of genotype in the cycle of violence in maltreated children” (2002) 297 Science 851.

⁵² For an explanation of the gene x environment interaction see Matthew Baum, above n 46, at 290.

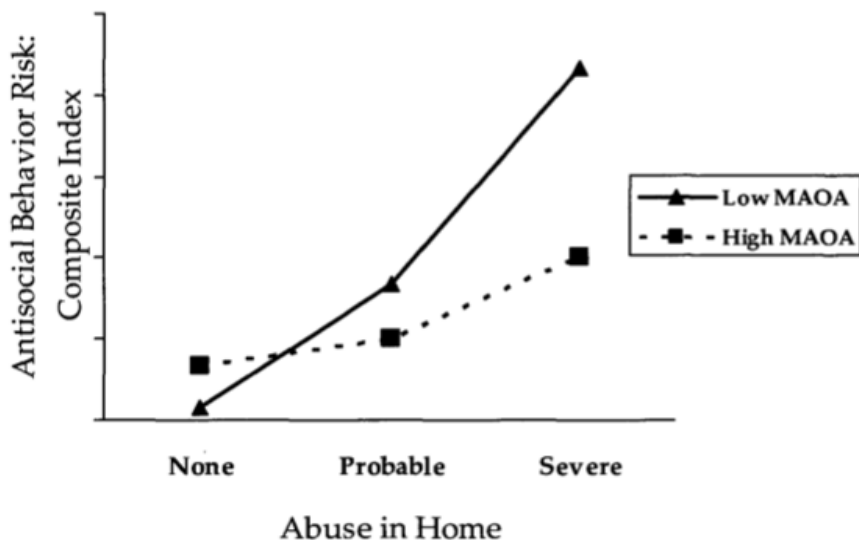
⁵³ Avshalom Caspi and others, above n 51, at 851-854.

⁵⁴ At 851-854.

⁵⁵ At 851-854.

⁵⁶ At 851-854.

Figure 2: Results of the Caspi Study



Intriguingly, studies of rhesus monkey populations have revealed similar trends. One paper noted that: “[c]arriers of low-activity alleles reared in small groups or without mothers were found to exhibit higher aggression and other anxiety-related maladaptive behaviors.”⁵⁷

While the results in the Caspi paper have been confirmed by several researchers, others have published inconsistent results.⁵⁸ However, the majority of studies (31 out of 37) “present statistically significant evidence for the interaction between the *MAOA* gene and environmental adversity measures.”⁵⁹

E The Tiihonen Study

Another finding of great interest comes from a recent study of a Finnish prison population. In this publication, the authors found a correlation between MAOA-L and violent crime, independent of any history of childhood maltreatment. The study also discussed another

⁵⁷ GM Karere and others “What is an “adverse” environment? Interactions of rearing experiences and MAOA genotype in rhesus monkeys” (2009) 65 *Biol Psychiatry* 770; TK Newman and others “Monoamine oxidase A gene promoter variation and rearing experience influences aggressive behavior in rhesus monkeys” (2005) 57 *Biol Psychiatry* 167.

⁵⁸ For a discussion of the different studies see Sean Godar and others, above n 6.

⁵⁹ Macià Buades-Rotger and David Gallardo-Pujol, above n 48, at 188.

gene, CDH13, but a discussion of this has been omitted since the focus of this paper is on MAOA. In *Molecular Psychiatry*, the authors explained that:⁶⁰

Our results, from over 500 offenders, showed a strong main effect for this genotype, but maltreatment did not modify the risk in any way...

A majority of all severe violent crimes in Finland are committed under the influence of alcohol or amphetamine, both of which induce a transient increase in the dopamine levels in the brain. Therefore, it is logical to assume that a low dopamine metabolism rate due to low-activity MAOA genotype may result in higher level of aggression during alcohol or stimulant intoxication.

The low-activity MAOA genotype also affects the metabolism of serotonin and serotonin signaling within the corticolimbic circuitry, leading to increased impulsive aggression.

The authors of this study also explored the legal and ethical ramifications of their research:⁶¹

[O]nly the actual mental capability (phenotype) of the offender matters when punishment or legal responsibility is considered, and the putative risk factors per se (such as genotype) have no legal role in the resulting judgment... it is essential to emphasize that the sensitivity and specificity of the genotype findings are much too low for any screening purposes, either for primary or secondary prevention of violent offending.

Professor Tiihonen was subsequently reported in the *Independent Newspaper* as saying that:⁶²

[I]f these two genes did not exist, there might be between five and 10 per cent less violent crime in Finland, but we cannot be sure of what the mechanism is that causes this...

⁶⁰ J Tiihonen and others "Genetic background of extreme violent behavior" (2015) 20 *Mol Psychiatry* 786.

⁶¹ J Tiihonen and others.

⁶² Steve Connor "Two genes found linked to tendency for violent crime" *The Independent* (online ed, London, 28 October 2014).

We've observed two genes that have a relatively big effect on violent behaviour but there are possibly tens or hundreds of other genes that have a smaller effect. This is why a 'test for criminality' is not possible from this study.

This paper is very interesting for a number of reasons. While Tiihonen et al. state that putative risk factors have no legal role in judgment; this runs contrary to the practice of the courts. While it is true that the actual mental state of the offender is the cornerstone of criminal liability, risk factors for offending are frequently accepted as mitigating evidence by sentencing judges in New Zealand. This study also raises the intriguing possibility of a relationship between alcohol and MAOA-L, and raises questions relating to genetic screening.

F The VNTR-2 Allele

The most prevalent allele in the MAOA-L category is the VNTR-3 allele. However, the much rarer⁶³ VNTR-2 allele has also been associated with aggressive behaviour. One recent study has revealed that “African-American males who carry the 2-repeat allele are significantly more likely than all other genotypes to engage in shooting and stabbing behaviors and to report having multiple shooting and stabbing victims.”⁶⁴

Some writers have “speculate[d] that *MAOA-2R* might account for — or at least play a significant role in — the relatively higher rates of violent crime in African Americans.” The 2-repeat variant has even been referred to as the “extreme warrior gene”.⁶⁵ The 2-repeat allele has also been associated with an even lower degree of transcriptional efficiency than the 3-repeat allele.⁶⁶ This seems to be yet another piece in an increasingly complex puzzle.

⁶³ It has been reported that this allele is found in “5.5% of African American men, 0.9% of Caucasian men, and 0.00067% of Asian men”. See Alondra Oubré, above n 7.

⁶⁴ KM Beaver, JC Barnes and BB Boutwell, above n 5.

⁶⁵ Alondra Oubré, above n 7.

⁶⁶ Sean Godar and others, above n 6.

G What Conclusions Can One Draw from the Evidence?

The media portrayal of behavioural genetics is rife with misunderstanding. The MAOA gene has frequently been referred to in the popular media as the “warrior gene”. However, such a term is highly misleading.⁶⁷ Possessing the MAOA-L genotype does not condemn an individual to a life of crime and aggression. Buckholtz and Meyer-Lindenberg explain that “considered independently from [environmental] factors, inheritance of the MAOA-L allele is completely compatible with psychiatric health.”⁶⁸

However, studies, such as the replicated publication by Caspi et al. have suggested that MAOA-L in conjunction with other environmental factors is associated with antisocial or criminal behaviour in men. The evidence points to this particular *gene x environment* interaction as a risk factor for aggressive or antisocial behaviour.

Some scientists, wary of the dangers of misinterpretation, have presented a very circumspect view of the evidence. For example, Forzana et al. have noted that:⁶⁹

It is crucial to avoid simplistic causal relations between genetic variants associated with violence or aggression and actual violent or aggressive behaviour. Whereas some people showing more aggressive or violent behaviour might have these particular gene variants, others will have the same variants despite being perfectly law-abiding citizens. It should be clear that there is no such thing as a ‘criminal gene’.

There is no scientific support to declare that gene variants, *claimed to predispose to aggression*, would make the carriers incapable of repressing an aggressive behaviour and thus unable to choose appropriate socially acceptable behaviours.

Jonathan Kaplan, in 2006, seemed cautiously prepared to accept the evidence as suggesting something more than a mere association. However, Kaplan was also in tune with the

⁶⁷ Ed Yong “Dangerous DNA: The truth about the ‘warrior gene’” (7 April 2010) New Scientist <<https://www.newscientist.com/article/mg20627557-300-dangerous-dna-the-truth-about-the-warrior-gene/>>.

⁶⁸ Joshua Buckholtz and Andreas Meyer-Lindenberg, above n 42.

⁶⁹ F Forzano and others “Italian appeal court: a genetic predisposition to commit murder” (2010) 18 Eur J Hum Genet 519. (emphasis added)

pressing need to dispel any notions of genetic determinism from his writing:⁷⁰

Research into the relationship between variation in the MAOA promoter regions and antisocial behaviors can reasonably claim to have gone beyond generating mere statistical associations... [It is] hardly a stretch to suspect that the associations found involve causally salient differences at the level of the developmental pathways.

But even if [the results of the Caspi study] do turn out to be typical, it is unclear what, if any, policy implications they might have... low-MAOA activity, even when coupled with growing up in a violent household, does not guarantee that a violent or antisocial adult will result.

In recent years, scientists and commentators have tried to shed some more light upon the underlying mechanisms. Mathew Baum, has suggested that some people might have genetic susceptibilities to adverse environments.⁷¹ After reviewing the scientific literature, he explains that while there are no born criminals, certain individuals “given the right combination of gene and environment are more vulnerable to act on violent impulses.”⁷² Again, Baum is careful to acknowledge that the “current scientific evidence is insufficient to argue that MAOA even when combined with severe abuse leads to legally involuntary action.”⁷³ On this point the science seems to be conclusive.

Scientists from the University of Kentucky have hypothesised “that disrupted serotonergic systems predispose individuals towards aggressive behavior by increasing impulsive

⁷⁰ Jonathan Kaplan, above n 37, at 71.

⁷¹ Matthew Baum, above n 46, at 293.

⁷² At 293. Such a claim is supported by experimental data which suggests that individuals with the MAOA-L allele might respond differently to stress. For example, “Eisenberger et al found that carriers of the low-activity MAOA allele displayed greater activity in the anterior cingulate cortex as a response to experimentally induced social exclusion, as compared to MAOA-H and MAOA-H/L participants. This evidence suggested that carriers of the MAOA-L variant would experience greater distress when confronted with adverse conditions.” See Macià Buades-Rotger and David Gallardo-Pujol, above n 48, at 194.

⁷³ Matthew Baum, above n 46, at 293.

reactivity to negative affect.”⁷⁴ They explain that MAOA-L “may not be associated with aggression per se but with reactive, retaliatory aggression to provocative situations.”⁷⁵ These ideas seem entirely credible, the enzyme MAO-A is involved in the degradation of key neurotransmitters which are involved in aggression, emotion and cognition. One can imagine how decreased MAO-A levels might translate to a perturbation in neurotransmitter levels, with complex neurological consequences.

Godar et al. in 2016, conducted a comprehensive review of the scientific literature relating to MAOA, and concluded that the evidence:⁷⁶

has unequivocally shown that male carriers of low-activity alleles of this gene are predisposed to a negative bias in the interpretation of social stimuli, which results in a greater propensity for aggressive and impulsive reactions to provocation and stress.

From reviewing the literature, it is safe to say that there is absolutely no evidence of a “warrior-gene” in the sense of a gene which destines individuals to a life of crime. However, there is a convincing case for the proposition that certain genetic and environmental influences predispose individuals to respond aggressively to stimuli. In particular, the combination of the MAOA-L genotype with childhood maltreatment seems to be implicated as a high-risk factor for aggressive behaviour.

The conclusion seems to be that “the *MAOA* gene may predispose toward reactive aggression after provocation.”⁷⁷ This raises intriguing criminal law implications. There also appears to be some evidence that MAOA-L can be a risk factor for aggressive behaviour, independently of any history of childhood maltreatment, but the evidence for this proposition is weaker.

⁷⁴ David Chester and others, above n 4.

⁷⁵ At 98.

⁷⁶ Sean Godar and others, above n 6, at 94.

⁷⁷ Macià Buades-Rotger and David Gallardo-Pujol, above n 48, at 196.

IV MAOA-L and the Courts

This next section will move on from an analysis of the science and examine how MAOA genotype evidence has been used in the courtroom. The narrative in the courts was initially one of profound scepticism, but some cases illustrate that decision makers are warming to the idea of accepting genotype evidence which points to an inclination towards aggressive behaviour. However, as this section will explore, issues are raised by the way in which MAOA evidence has been considered. A discussion of some key decisions is thus necessary to build a solid platform for subsequent analysis.

A Mobley v The State (1995)

In 1995, counsel for Stephen Mobley, a convict on “death row”, made a motion for funds so that Mobley could be tested to see if he possessed some form of genetic disorder which might act to mitigate his culpability, and thus allow him to escape the death penalty. This request was in part inspired by Mobley’s serious personality disorder and the family’s extensive history of violent and aggressive behaviour.⁷⁸ This motion was also based on recent developments in the field of behavioural genetics, such as the Brunner study.⁷⁹ The Supreme Court of Georgia however, rejected the request, explaining that:⁸⁰

[T]he theory behind the request for funds will not have reached a scientific stage of verifiable certainty in the near future and that Mobley could not show that such a stage will ever be reached.

This is an understandable conclusion, at the time there was limited scientific evidence available. Nevertheless, the Mobley case was significant since it represented the first attempt to use MAOA genotype evidence. While the argument was unsuccessful here, this case would have been a clear inspiration for creative defence counsel in future cases, such as that of *State v Waldroup*.

⁷⁸ Debra Wilson, above n 1, at 131.

⁷⁹ At 131.

⁸⁰ *Mobley v State* 455 SE 2d 61.

B State v Waldroup (2009)

In 2006, Bradley Waldroup brutally murdered Leslie Bradshaw, and severely injured his wife Penny. The incident was no petty altercation, Ms Bradshaw was gunned down, while Penny Waldroup was first shot in the back, then beaten with a shovel and hacked at in the head with a machete.⁸¹ Miraculously, Penny survived after fleeing the house and escaping in a police car.⁸² Waldroup was even reported to have told his kids to “come tell your mama goodbye”.⁸³ He later admitted to the acts, acknowledging that “I snapped... I’m not proud of none of it.”⁸⁴

Facing a difficult task, his defence counsel had to innovate, managing to persuade the trial judge to accept evidence showing that Bradley Waldroup had a “high risk” gene for the development of aggressive tendencies. Genetic testing had revealed that Mr Waldroup had a low expression form of MAOA⁸⁵ The argument constructed was that Waldroup’s genetic makeup, combined with a history of childhood abuse, should act to mitigate his responsibility.⁸⁶ It seems that defence counsel drew on the work of Caspi et al. to construct a plea for sympathy founded on genotype evidence.

After only 11 hours of deliberation, Bradley Waldroup was acquitted of the charge of murder, the jury instead pronouncing him guilty of voluntary manslaughter, attempted second-degree murder, and aggravated kidnapping.⁸⁷ Subsequent interviews with members of the jury revealed that they were influenced by this genetic evidence. One juror later

⁸¹ *State v Waldroup* E2010.01906.CCA.R3.CD (Tenn. Crim. App. 2011). Available from: <http://tncourts.gov/sites/default/files/waldroupremandopn.pdf>.

⁸² *State v Waldroup*.

⁸³ Barbara Hagerty “Can Your Genes Make You Murder?” (1 July, 2010) NPR <<http://www.npr.org/templates/story/story.php?storyId=128043329>>.

⁸⁴ Barbara Hagerty.

⁸⁵ Eloise Gibson “A Dunedin Study discovery saved an American killer from the death penalty” (21 March, 2017) The Listener <<http://www.noted.co.nz/currently/science/a-dunedin-study-discovery-saved-an-american-killer-from-the-death-penalty/>>; Barbara Hagerty, above n 83.

⁸⁶ Barbara Hagerty, above n 83.

⁸⁷ Deborah Denno, above n 4, at footnote 13.

noted in a newspaper interview that:⁸⁸

Evidently, it's just something that doesn't tick right. ... Some people without this would react totally different than he would. A diagnosis is a diagnosis, it's there. A bad gene is a bad gene.

The finding of voluntary manslaughter does not seem to have been supported by the facts. Under Tennessee law voluntary manslaughter is defined as:⁸⁹

The intentional or knowing killing of another in a state of passion produced by adequate provocation sufficient to lead a reasonable person to act in an irrational manner.

No evidence of provocation was raised at trial.⁹⁰ Therefore, the only logical explanation is that the jury were so swayed by the genetic evidence, that they considered Mr. Waldroup's culpability to only warrant a lesser charge, regardless of the content of the statutory provisions.⁹¹ This case illustrates that the use of genotype evidence in the hands of a skilful advocate is a powerful tool, but one which can produce questionable results. The case raises serious issues that genotype evidence may be misinterpreted to the extent that it leads to a miscarriage of justice.

Opposing counsel and the judge certainly did not hesitate to make their thoughts known on this result. The prosecutor described the admission of the genotype evidence as "smoke and mirrors".⁹² Judge Carroll Ross candidly advised Waldroup against appealing, noting that he "might not be as fortunate with a jury next time."⁹³ Waldroup was sentenced to 32 years in prison.⁹⁴

⁸⁸ Barbara Hagerty; Debra Wilson, above n 1, at 117-118.

⁸⁹ Debra Wilson, above n 1, at 118.

⁹⁰ At 118.

⁹¹ At 118.

⁹² Barbara Hagerty, above n 83.

⁹³ Debra Wilson, above n 1, at 119.

⁹⁴ Barbara Hagerty, above n 83.

C The Use of MAOA Genotype Evidence in Italy

MAOA-L genotype evidence has also been successfully raised as a mitigating factor in two Italian murder trials. In 2009, Abdelmalek Bayout received a year discount on his murder sentence after evidence was introduced that he had a “higher risk version” of MAOA.⁹⁵ Judge Reinotti noted that Bayout’s genes “would make him particularly aggressive in stressful situations”.⁹⁶ No evidence of childhood maltreatment was relied upon in this case, other than the cultural shock of moving to Italy from Algeria.⁹⁷ This decision has proved controversial, and has even been described as setting a “dangerous precedent”.⁹⁸ For example Forzano et al. were quick to note that:⁹⁹

[T]he possibility of using genetic variants to evaluate the actual mental capacity of a person at a given time is far from being established... [an individual] should be judged on the basis of his actual condition and mental capacity... not on the basis of probability interpretation.

In another Italian case, the murder trial of Stefania Albertani, Judge Luisa Lo Gatto reduced her sentence “from life to 20 years, after ruling that neuroimaging and genetic tests proved the partial mental illness of the defendant.”¹⁰⁰ Interestingly, “[t]he brain scans showed that she had alterations in the grey matter of two brain regions: the anterior cingulate gyrus, which is involved in inhibiting behavior, and the insula, which is linked to aggression.”¹⁰¹ Evidence was also presented regarding Stefania’s low MAOA condition.¹⁰² This case is notable not only for its acceptance of genotype evidence, but also for accepting brain scan evidence as a mitigating factor.

⁹⁵ Emiliano Feresin “Lighter sentence for murderer with ‘bad genes’” (30 October 2009) Nature <<http://www.nature.com/news/2009/091030/full/news.2009.1050.html>>.

⁹⁶ Emiliano Feresin.

⁹⁷ Debra Wilson, above n 1, at 142-143.

⁹⁸ F Forzano and others, above n 69.

⁹⁹ F Forzano and others.

¹⁰⁰ Emiliano Feresin, above n 95.

¹⁰¹ Debra Wilson, above n 1, at 143.

¹⁰² Debra Wilson, above n 1, at 143.

These legal developments raise several pressing questions. Should we accept evidence of a defendant's genotype in the context of the criminal law? And if so, to what extent should it be relevant to criminal proceedings? The next two sections of this paper will attempt to provide some answers to these questions.

V MAOA-L and Criminal Liability

A MAOA-L Evidence and the Question of Criminal Intent

A defendant might be able to point to evidence which shows that their MAOA genotype and their social background are risk factors for aggression. However, this evidence does not suggest that they would be incapable of exercising rational thought or physical control over their actions. As discussed in Part II, this is probably the standard that would have to be satisfied for the evidence to be relevant to the question of criminal intent. Therefore, MAOA genotype evidence should not be relevant to a determination of mens rea.

A mere tendency to act in a given manner does not deprive one of the ability to act otherwise.¹⁰³ A person predisposed towards behaving violently could still be capable of understanding the wrongfulness of their actions, and could still have the ability to act otherwise. These factors are not mutually exclusive. As Paul Applebaum notes:¹⁰⁴

[M]any defendants experience pressures to commit criminal acts—from peer encouragement to the disinhibiting effect of intoxicating substances—but in general we expect them to resist the urge to act illegally or suffer the consequences.

A mere predisposition, which does not deprive a defendant of their mental capacity, would probably be viewed as analogous to these factors. Moreover, MAOA-L has been generally linked to impulsive aggressive behaviour.¹⁰⁵ This renders the evidence particularly ineffective in situations where a defendant is charged with a premeditated offence. As

¹⁰³ Paul Appelbaum, above n 2, at 946.

¹⁰⁴ At 948.

¹⁰⁵ See David Chester and others, above n 4.

Gavaghan and Bastani put it: “terrorist bombers should probably look elsewhere.”¹⁰⁶

B MAOA-L Evidence and Excusatory Defences

Since MAOA-L genotype evidence does not support claims that someone would be unable to control their actions, in the sense of being physically unable to do so, this effectively rules out the possibility of using such evidence to support a defence of automatism.

The same issue arises in the context of a defence of insanity. In New Zealand, the requirements for the defence of insanity are enacted in the Crimes Act 1961, s23, which provides that:

No person shall be convicted of an offence by reason of an act done or omitted by him when labouring under natural imbecility or disease of the mind to such an extent as to render him incapable -

- (a) Of understanding the nature and quality of the act or omission; or
- (b) Of knowing that the act or omission was morally wrong, having regard to the commonly accepted standards of right and wrong.

A ‘disease of the mind’ has been defined as “a term which defies precise definition and which can comprehend mental derangement in the widest sense.”¹⁰⁷ The key element is that the ‘disease of the mind’ has an internal, rather than an external origin.¹⁰⁸ Given that a ‘disease of the mind’ is a legal term, it is conceivable that a genetic predisposition towards aggression could be brought within the scope of this broad definition. However, the fundamental issue here, is that a person’s MAOA-L genotype does not tell us anything about their ability to understand the nature and quality of their acts or omissions, or the distinction between right and wrong.

Additionally, if an accused sought to found a defence on the basis of the Caspi study,

¹⁰⁶ Colin Gavaghan and Amir Bastani, above n 8, at 13.

¹⁰⁷ *R v Cottle* [1958] NZLR 999 (CA) at 1011.

¹⁰⁸ *Police v Bannin* [1991] 2 NZLR 237 (HC) at 242.

relating to a gene x environment interaction, then the environmental component would be considered *external*. This might cause some difficulty with the overall condition being characterised as a ‘disease of the mind’ since this term is defined as something which is internal rather than external.¹⁰⁹ This legal distinction might lead judges to consider genetic predisposition evidence in isolation from environmental influences, or focus on what might be the primary cause of the “disease of the mind”. Drawing such distinctions is not desirable. The way we behave is dictated by a complex interaction between genetic and behavioural influences. It does not make much sense to divorce the genetic component associated with a behaviour from the environmental component.

Indeed, creating this internal/external distinction only serves to reinforce misconceptions around the role of genetics in human behaviour. The distinction is out of touch with modern science. At any rate, it seems relatively clear that MAOA genotype evidence should not be relevant in a determination of criminal liability.

VI MAOA-L Genotype Evidence at Sentencing

A MAOA-L Genotype Evidence at Sentencing

While MAOA genotype evidence is unlikely to be used to negate criminal responsibility or support defences of insanity or automatism, we might still have some sympathy for a defendant if there is evidence which suggests that they are predisposed towards aggressive behaviour - in the sense that they carry a gene which is a known risk factor for the development of aggressive tendencies. Thus, there is still a potential for MAOA genotype evidence to be relevant during sentencing.

The point of introducing such evidence at the sentencing stage, from the perspective of the defendant, would be to illustrate that they were less able to exercise restraint over their violent impulses than an ordinary member of society, and therefore should be less responsible for the consequences of their actions. Provided there is sufficient scientific consensus, such evidence could be admissible at sentencing in New Zealand. MAOA genotype evidence could constitute a “particular circumstance of the offender” within the

¹⁰⁹ Debra Wilson, above n 1, at 123.

meaning of s8(h), Sentencing Act 2002.¹¹⁰ As the Canadian Supreme Court acknowledged in *R v Ipeelee*, “systemic and background factors may bear on the culpability of the offender, to the extent that they shed light on his or her level of moral blameworthiness.”¹¹¹ MAOA genotype evidence could be viewed as a background factor relevant to the question of moral blameworthiness.

For an argument of mitigation to be successful, the sentencing judge would have to be satisfied that the evidence supported the conclusion that the defendant was more vulnerable to act on impulses or was less able to exercise self-restraint. As the discussion in Part III illustrates, this is a viable interpretation of the scientific data. And as the discussion in Part IV illustrates, some overseas courts have accepted this argument.

In the New Zealand context, courts have accepted that evidence of foetal alcohol spectrum disorder (FASD), a condition which, among other things, is associated with impulsive behaviour, can be a mitigating factor. For example, in *Pomare v R* it was accepted that Mr Pomare’s FASD condition “generally increased [his] vulnerability to impulsive criminal behaviour” suggesting that his culpability was diminished.¹¹² By analogy, similar reasoning could justify the acceptance of MAOA genotype evidence during sentencing.

B The Problem of Probabilities? What Constitutes a Mitigating Factor?

There is a significant further issue with MAOA genotype evidence which warrants discussion. At best, genotype evidence only indicates that an individual will be predisposed towards behaving aggressively. For example, an individual with the MAOA-L genotype, even combined with a severely abusive childhood, will not necessarily behave

¹¹⁰ Section 8(h), Sentencing Act 2002, provides that the courts: “must take into account any particular circumstances of the offender that mean that a sentence or other means of dealing with the offender that would otherwise be appropriate would, in the particular instance, be disproportionately severe.”

¹¹¹ *R v Ipeelee*, above n 26, at [73].

¹¹² *Pomare v R* [2017] NZCA 155 at [9]-[10]. Also see *Edri v R* [2013] NZCA 264 at [18]. FASD has also been recognised as a potent mitigating factor in Australia and Canada. See *R v Charlie* [2012] YKTC 5 and *LCM v Western Australia* [2016] WASCA 164.

aggressively, or be an aggressive person.

There is an important distinction to be made between genotype and phenotype. Genotype refers to one's genes whereas phenotype refers to the observed properties of an individual. The two are associated, since the genotype influences the phenotype, but the link between the two is complex.¹¹³ In the case of MAOA and aggression, knowledge of the genotype (MAOA-L) does not tell us the phenotype of an individual. Human behaviour is a highly complex phenomenon, and the effects of one gene upon a behaviour tend to be very small.

The question for the courts, is whether they would be prepared to accept as a mitigating factor, a genotype, which is associated with an increased likelihood of aggressive behaviour.

In the Waldroup case, the jury seemed to have accepted the mitigating nature of the evidence. As discussed in Part IV, the conclusion to acquit on the charge of first degree murder seems to have been influenced by the genotype evidence presented. However, this conclusion might have been founded upon a misunderstanding of the science. The jury might have incorrectly interpreted the evidence as suggesting that Waldroup was "destined" to act the way he did. Comments such as "a bad gene is a bad gene" lend weight to the view that the jury (or at least that individual juror) was influenced by simplistic assumptions. The tenor of this statement that "a bad gene is a bad gene" is one of genetic determinism. As discussed, it can be misleading to describe the MAOA-L allele as simply "bad". A vast number of individuals carry the MAOA-L allele and do not commit crimes. The scientific reality is much more nuanced.

However, risk factors such as genotype evidence, can in principle be used as mitigating evidence. For example, the circumstances of one's upbringing could be considered as mitigating evidence.¹¹⁴ Yet most children from backgrounds of maltreatment do not go on to commit violent offences. Indeed, Avshalom Caspi et al. explain that while "maltreatment increases the risk of later criminality by about 50%, most maltreated children do not

¹¹³ See Jonathan Kaplan, above n 37, at footnote 1.

¹¹⁴ For example, see *R v DJH*, above n 32, at [25]-[26].

become delinquents or criminals.”¹¹⁵ Accepting childhood maltreatment as a mitigating factor would be relatively uncontentious, yet it represents a similar idea to referring to genotype evidence during sentencing.

A similar argument has been made in relation to the disadvantaged circumstances faced by indigenous Canadians. In *Ipeelee*, the Supreme Court of Canada noted that: “it would be extremely difficult for an Aboriginal offender to ever establish a direct causal link between his circumstances and his offending. The interconnections are simply too complex.”¹¹⁶ Yet in the view of the Court, this lack of a direct causal link did not preclude evidence of a disadvantaged background from being considered at sentencing, provided that the evidence had some bearing upon the defendant’s culpability.¹¹⁷ This standard of *some bearing upon culpability* allows for the consideration of risk factors during sentencing, such as genotype evidence which predisposes an individual to respond aggressively when placed in stressful situations.

However, not all risk factors for criminality can constitute mitigating evidence. For example, Steve Jones notes that “90% of all murders are committed by people with a Y chromosome — males. Should we always give males a shorter sentence?”¹¹⁸ The answer is surely “no”. Fatal to such an argument is the obvious and crucial point that most men are not murderers.¹¹⁹ Likewise, a corollary of accepting the male gender as a mitigating factor, is accepting the female gender as an aggravating factor. Thus, it seems that an individual’s increased likelihood of acting violently or criminally can be a mitigating factor in some circumstances, but not in others.

What then makes a risk factor for criminality a mitigating factor? One view is that MAOA genotype evidence, as a genetic risk factor for criminality, is analogous to the Y chromosome. However, at a practical level, arguments founded upon a gene x environment

¹¹⁵ See Avshalom Caspi and others, above n 51, at 851.

¹¹⁶ *R v Ipeelee*, above n 26, at [83].

¹¹⁷ At [83].

¹¹⁸ Per Steve Jones, in Emiliano Feresin, above n 95.

¹¹⁹ The same argument can of course be made in the context of genotype evidence.

interaction (or an “obscure” gene) associated with aggression may be more persuasive. The judge or jury will likely have little knowledge of the subject matter, and thus might be more open to considering how the “condition” might mitigate the defendant’s culpability. Arguments founded upon gender would not be viewed so sympathetically. The judge or juror’s personal experiences will clearly refute the idea that gender could be an excusatory factor. It is also not practical to treat the male gender as a mitigating factor since it applies to the majority of violent offenders.

There are difficult distinctions to be made. However, given that the courts accept evidence of childhood maltreatment as mitigating evidence, it only takes a small step to accept genotype evidence which might explain how the defendant was particularly susceptible to the effects of negative early childhood experiences. Construed in such a manner, the genetic evidence might seem more palatable to a sentencing judge.

The next part of this paper moves on from this debate and considers the practical implications of referring to MAOA-L genotype evidence during sentencing.

C Pragmatic Benefits of Genotype Evidence During Sentencing

Genetic evidence based on MAOA-L has generally been adduced to support claims of leniency during sentencing.¹²⁰ While these cases have been few and far between, genetic evidence has a certain special allure. In contrast to evidence of childhood maltreatment which is often viewed as unconnected to the defendant’s crime, and mental health evidence, which may be perceived as “fake”,¹²¹ evidence of a genetic nature offers something with *a more concrete semblance*. As Brett Walker notes:¹²²

In the case of mental health issues, the lack of tangible evidence of mental disorders often leads the jury to conclude that the defendant is merely malingering, or faking, the symptoms

¹²⁰ For example, see the Bayout and Albertani cases referred to in Part IV. Also see Paul Appelbaum, above n 2, at 947.

¹²¹ See Brett Walker “When the Facts and the Law Are Against You, Argue the Genes?: A Pragmatic Analysis of Genotyping Mitigation Defenses for Psychopathic Defendants in Death Penalty Cases” (2013) 90 Wash U L Rev 1779 at 1790.

¹²² At 1790.

to avoid the... penalty. Other jurors may simply discount the psychological expert as being a hired gun whose testimony is biased towards the defendant who is paying the bill.

In contrast, genetic evidence may play into popular beliefs that some people are born evil. It offers a tangible and credible explanation for behaviour, which may be so heinous and offensive that it would otherwise defy logic and reason.

Moreover, psychological evidence is not as useful for building a narrative of sympathy. Where an offender's aggressive behaviour is attributed in part to a genetic predisposition, this can create the impression that the offender is less at fault, because they were less able to resist their aggressive impulses. The idea is that because someone cannot choose their genes, they should be less responsible if their genes lead to aggressive behaviour. Of course, a person with the MAOA-L genotype may not be aggressive, but the idea that it could somehow be "the fault of the genes" is nevertheless, a very powerful one.

Conversely, attributing aggressive behaviour to the offender's personality (without suggesting any genetic basis) merely serves to reinforce an existing negative perception of the offender. This argument is akin to saying "my client has no other excuse; he is just a bad person and is really aggressive". Such an argument would not generate much sympathy.

D The CSI Effect

There are other reasons why genetic evidence may appeal to defence counsel. One of these reasons might be due to what has been described as the "CSI effect". The CSI effect refers "to a perception that due to the popularity of the television programme, jurors expect to be provided with scientific evidence as part of the prosecution case and give undue weight to this."¹²³ Far from representing some abstract hypothesis, this theory has acquired empirical support. For example, a study by Shelton has illustrated that jurors demand and expect scientific evidence.¹²⁴ The Shelton study however, attributed this demand to "the result of

¹²³ Debra Wilson, above n 1, at 146.

¹²⁴ DE Shelton "A study of juror expectations and demands concerning scientific evidence: Does the CSI-effect exist?" (2006) 9(2) Vand J Ent & Tech L 331.

broader changes in popular culture related to advancements in both technology and information distribution” and not due to juror television preferences.¹²⁵ Moreover, it was noted that DNA evidence possesses a “special aura of credibility”.¹²⁶ Given this research, it is possible that MAOA-L genotype evidence may play into such demands and expectations. From a lay person’s perspective, genetic evidence can be viewed as authoritative and undisputable. This might be due to misconceptions acquired from television crime shows, in which DNA identification evidence is portrayed as being the decisive proof of a suspect’s guilt. Or it might simply be attributable to popular views that science is objective, authoritative and conclusive.

However, genetic evidence is not always “objective, authoritative and conclusive” – indeed as the discussion in Part III illustrates, uncertainty is inherent in behavioural genetics. Indeed, while there is a tendency for science to be viewed as objective and factual, uncertainty may in fact be the mark of an objective expert opinion, in the sense that the expert is “resisting the temptation to present probability as truth.”¹²⁷ However, reality and perception are two different things. There is a danger that those from non-scientific backgrounds will place undue weight on genotype evidence and view it as determinative of an individual’s behaviour. The use of statistics adds further risk of misinterpretation. Indeed, the Court of Appeal has raised the concern that “[a]lthough the maxim “lies, damn lies and statistics” is well known, there is a natural human tendency for people to be impressed by large numbers.”¹²⁸

E MAOA-L Evidence... The Knife that Cuts Both Ways

As the above discussion illustrates, genetic predisposition evidence has the potential to be a potent weapon in the arsenal of a defence lawyer. However, it is a weapon that must be wielded with care. Such evidence is open to multiple interpretations, and not all of them

¹²⁵ At 368.

¹²⁶ At 357.

¹²⁷ Yvette Tinsley “Science in the Criminal Courts: Tool in Service, Challenge to Legal Authority or Indispensable Ally?” (2013) 25(4) NZULR 844 at 847.

¹²⁸ *R v Aymes* [2005] 2 NZLR 376 (CA) at [134].

are defendant friendly. In another context, a judge could easily infer that a defendant is more dangerous and less susceptible to rehabilitation. Thus, the same evidence can viably act as both a mitigating and aggravating factor. Moreover, given the possibility of reaching two contrary conclusions from the same evidence, the interpretation drawn could well depend on the overall direction in which the decision maker was heading. In other words, the evidence might be construed to support the decision maker's conclusion. For example, a lenient judge might view the evidence as further establishing the defendant's diminished culpability, whereas a fearful judge, or a judge with a more punitive inclination, might be further swayed in the opposite direction. In practice, the use of genetic predisposition evidence could accentuate disparate outcomes in sentencing.

The Waldroup, Bayout and Albertani cases create the impression that MAOA-L evidence has a mitigating character.¹²⁹ However, other judges have taken a different view. For example, in *Landrigan v Schriro*, the US Court of Appeals for the Ninth Circuit noted that:¹³⁰

[It was] highly doubtful that the sentencing court would have been moved by information that Landrigan was a remorseless, violent killer because he was genetically programmed to be violent, as shown by the fact that he comes from a family of violent people, who are killers also... [A]lthough Landrigan's new evidence can be called mitigating in some slight sense, it would also have shown the court that it could anticipate that he would continue to be violent... As the Arizona Supreme Court so aptly put it when dealing with one of Landrigan's other claims, "[i]n his comments, defendant not only failed to show remorse or offer mitigating evidence, but he flaunted his menacing behavior." On this record, assuring the court that genetics made him the way he is could not have been very helpful.

Interestingly, a survey in the United States has revealed that judges would be inclined to give a lesser sentence, by a year, where a hypothetical defendant's psychopathy could be attributed to a genetic basis.¹³¹ However, the results of a survey of the general United States

¹²⁹ See the discussion in Part IV.

¹³⁰ *Landrigan v Schriro* 441 F 3d 638 (9th Cir. 2006) at 1228–29.

¹³¹ Lisa Aspinwall, Teneille Brown and James Tabery "The Double-Edged Sword: Does Biomechanism Increase or Decrease Judges' Sentencing of Psychopaths?" (2012) 337 Science 846.

population suggest that individuals do not assign lesser sentences after being informed that the defendant had a genetic predisposition towards aggressive behaviour.¹³² What the respondents did note, was a greater fear towards the defendant.¹³³ Increased fear has the potential to manifest itself in the form of a more punitive sentence, or a sentence with a greater focus on incapacitation. Thus, the use of genetic evidence at sentencing by defence counsel could backfire.

It is understandable that such evidence might have been adduced in cases such as that of Mr. Waldroup. Waldroup was facing the death penalty if found guilty of first degree murder. Defence counsel desperately needed something to distract the jury from the atrocities of his crimes. However, one should expect counsel to be more circumspect in other contexts. The introduction of genetic predisposition evidence may be too great a risk in cases where less is at stake. Indeed, Deborah Denno has written that the 33 United States “behavioural genetics” cases between 2007-2011 all involved a murder conviction, in which, with one exception, the behavioural genetics evidence was relied upon as a mitigating factor to avoid the death penalty.¹³⁴

1 An analogy to neuro-disability and traumatic brain injury: the case of P v R

In determining the potential for MAOA genotype evidence to be construed as both an aggravating and mitigating factor, a useful analogy can be drawn to cases of traumatic brain injury. One such case is the New Zealand case of *P v R*.¹³⁵ This case involved a defendant with a traumatic brain injury, and no arguments were made in relation to genetic predisposition evidence. Nevertheless, the defendant’s mental disability is analogous to an alleged predisposition towards aggressive behaviour supported by MAOA genotype evidence, or some other form of genetic predisposition evidence. Thus, by analogy, the

¹³² Paul Appelbaum and Nicholas Scurich “Impact of Behavioral Genetic Evidence on the Adjudication of Criminal Behavior” (2014) 42 J Am Acad Psychiatry Law 91 at 91.

¹³³ At 91.

¹³⁴ Deborah Denno, above n 4, at 973.

¹³⁵ *R v DP* [2015] NZHC 1796. On appeal the case is referred to as *P v R* (see footnote 142).

case highlights the potential for genetic predisposition evidence to act as both a mitigating and aggravating factor during sentencing.

The facts of the case were simple, but brutal. P and R (the co-defendant) decided to rob a dairy owned by the Kumar family. In the process of robbing the store, P stabbed Mr Kumar with a knife, killing him. P was only 13 years old. Following a trial by jury, P was found guilty of manslaughter. In the High Court, Lang J gave a sentence of six years' imprisonment with a minimum non-parole period of three years and three months.¹³⁶

By all accounts, P had led an extremely unfortunate childhood. He showed symptoms of foetal alcohol spectrum disorder and had also developed a traumatic brain injury following a collision with a vehicle, for which he was not properly treated.¹³⁷ His home was used as a place for dealing drugs, and he was reported to have abused both alcohol and other substances.¹³⁸

In the High Court, Lang J viewed P's brain injury as implying "a risk that a similar situation could occur if P was placed in a stressful context in the future"¹³⁹ and thus justifying a longer minimum non-parole period:¹⁴⁰

The material that is before me makes it clear that your head injury makes you vulnerable in times of stress or complexity to act impulsively or instinctively. Your present offending is proof of that... The protection of our society and indeed your own protection, in my view, can only be met by assuring that you are in a safe and secure environment for the next few years.

Lang J essentially viewed the traumatic brain injury as an aggravating factor on the basis

¹³⁶ At [33].

¹³⁷ At [11] - [12].

¹³⁸ At [13].

¹³⁹ Nessa Lynch "Case Note: The Sentencing of Vulnerability: P v R [2016] NZCLR at 106.

¹⁴⁰ *R v DP*, above n 135, at [35].

that it made P a more dangerous offender, and increased his likelihood of future offending.¹⁴¹ This justified a longer sentence in order to protect society.

The case was appealed to the Court of Appeal. One of the grounds of appeal was whether the 20% discount due to P's youth and brain injury was appropriate.¹⁴² The Court of Appeal held that this discount was insufficient, and instead weighed the evidence as justifying a 40% discount.¹⁴³ The court substituted a sentence of 4 and a half years' imprisonment.¹⁴⁴ In contrast to Lang J, the Court of Appeal seems to have viewed the traumatic brain injury evidence as having greater mitigating character, quoting with approval a passage on the effects of the brain injury:¹⁴⁵

In my opinion, although knowing right from wrong, [P] was significantly reduced in his capacity to choose right from wrong, due to his lasting brain injury impairments. He could not use his knowledge normally to control his actions on the day and in the situation in the dairy. He had less control than another person his age would have had in the same circumstances due to his brain damage.

These different interpretations are not surprising, both views are viable. Importantly for this discussion, just as evidence of a mental disability can be construed as both rendering the defendant more dangerous and simultaneously less culpable, the same can be said of other evidence which highlights a predisposition towards aggressive or impulsive behaviour (such as genetic evidence).

2 Genetic predisposition evidence and the principles of sentencing

Another way to consider the effects which genetic predisposition evidence might have upon sentencing is by examining the evidence in relation to the principles and purposes of sentencing. Section 7 of the Sentencing Act 2002 provides a description of the purposes of

¹⁴¹ Nessa Lynch, above n 139, at 107.

¹⁴² *P (CA479/2015) v R* [2016] NZCA 128 at [1].

¹⁴³ At [45].

¹⁴⁴ At [54].

¹⁴⁵ At [44].

sentencing an offender. The section provides that the purposes of sentencing are:

- (a) to hold the offender accountable for harm done to the victim and the community by the offending; or
- (b) to promote in the offender a sense of responsibility for, and an acknowledgment of, that harm; or
- (c) to provide for the interests of the victim of the offence; or
- (d) to provide reparation for harm done by the offending; or
- (e) to denounce the conduct in which the offender was involved; or
- (f) to deter the offender or other persons from committing the same or a similar offence;
or
- (g) to protect the community from the offender; or
- (h) to assist in the offender's rehabilitation and reintegration; or
- (i) a combination of 2 or more of the purposes in paragraphs (a) to (h).

The three most relevant sentencing principles in the context of MAOA-L genotype evidence are rehabilitation, deterrence and the protection of the community (incapacitation).

(a) Rehabilitation

If an offender is viewed as genetically predisposed towards aggressive behaviour, then sentences aimed at rehabilitation might be perceived as offering less utility. Predisposed offenders might be seen as beyond help. Such an inference would weigh in favour of an imprisonment sentence, rather than one aimed at rehabilitation.

Matters of perception aside, an analysis of the offender's rehabilitation prospects should be largely based on the results of psychological analyses, and what they reveal about the actual mental state and aggressive nature of the defendant. As explained in Part III, the genotype of an offender does not tell the whole story. In the case of aggression, knowledge of one gene will not tell us an individual's phenotype. Human behaviour is far too complex. It is also important to recognise that environmental factors go a long way towards influencing behaviour. Judges should not be too hasty to conclude that an offender is

beyond help. Human behaviour is an incredibly complex phenomenon that is currently beyond prediction. We must be very cautious about ascribing too much weight to genetic risk factors, since there is a lot more that goes into shaping human behaviour, than one gene.

(b) Deterrence

If evidence of an aggressive predisposition is considered, a sentence based on individual deterrence may seem less appropriate. An offender that is prone to behaving in an aggressive manner will not be perceived as particularly receptive to the ideologies represented by deterrence theory. Deterrence theory relies on the presence of rational actors, who will decide that the offending is not worth the punishment. However, the same argument that deterrence is ineffective could be made in the context of most violent offenders, few of whom would be considered rational. Therefore, it is unclear how different an analysis of the value of deterrence would be in this context.

It is possible for deterrence to have some value at the level of the wider population, although it is worth noting that the effectiveness of general deterrence theory has been the subject of much critique. It is the certainty of punishment, rather than its potential gravity which is the main deterrent to offending.¹⁴⁶

Overall, it seems that an analysis of the value of deterrence in the case of an offender genetically predisposed towards aggression should be no different to that of any other aggressive offender who commits irrational acts of violence. Imposing a harsher punishment may not be very effective in terms of deterring the individual from future violence. However, the sentence may have some value in terms of sending a message to society that criminal behaviour is not tolerated, and will be punished accordingly.

¹⁴⁶ Daniel Nagin "Deterrence in the Twenty-First Century" (2013) 42 *Crime & Justice* 199.

(c) Incapacitation

The incapacitation of offenders genetically predisposed towards violence, or indeed violent offenders in general, would be consistent with the principle of the protection of the community. The focus of incapacitation theories of punishment is on the risk of reoffending and the potential harm to the community, which would be perceived as particularly high in a criminal genetically predisposed towards violence. Moreover, notions of individual blameworthiness would not be as relevant within this framework since this analysis focuses on the safety of the community. Thus, it would be less of a concern that a genetically predisposed individual was less able to restrain their violent impulses, and was less at fault. Viewed through this lens, evidence highlighting a predisposition towards aggression would be highly detrimental to the defendant's case.

Overall, this analysis indicates that MAOA-L genotype evidence has the potential to be detrimental to the defendant during sentencing.

3 Hypothetical defendants with knowledge of their MAOA-L condition

There is another potential layer of complexity. As discussed in Part III, a recent and intriguing hypothesis from the Tiihonen study is that “a low dopamine metabolism rate due to low-activity MAOA genotype may result in higher level of aggression *during alcohol or stimulant intoxication.*”¹⁴⁷

If a *firm and compelling* link is established between MAOA-L and heightened levels of aggression during intoxication, then we might imagine a hypothetical situation where a defendant with knowledge of their MAOA-L genotype, becomes intoxicated, and in the process, commits a violent offence. These circumstances could be viewed as aggravating due to the defendant's failure to take precautions against a known risk. This situation would be analogous to that of a defendant with a history of alcohol fuelled violence, who consumes alcohol and offends while intoxicated. In such a circumstance, the extent of the defendant's culpability would logically depend on the extent of the risk presented by the

¹⁴⁷ J Tiihonen and others, above n 60. (emphasis added)

predisposition evidence, the extent of the defendant's knowledge of this risk, and the extent of the preventive measures taken.

4 MAOA-L evidence and preventive detention

In order to develop a more complete analysis of the potential effects of genetic predisposition evidence during sentencing it is necessary to examine the New Zealand preventive detention system. In New Zealand, the courts have the capacity to impose preventive detention sentences “to protect the community from those who pose a significant and ongoing risk to the safety of its members”.¹⁴⁸ A sentence of preventive detention allows a prisoner to be imprisoned for an indeterminate period. It is the most punitive weapon in the arsenal of a sentencing judge. Section 87 authorises the court to impose indefinite prison sentences so long as the offender is convicted of a qualifying sexual or violent offence.¹⁴⁹

One of the risks of presenting genetic predisposition evidence as defence counsel, is that such evidence could be construed by a judge as lending support to a decision to impose a sentence of preventive detention, either for that offence, or for a future offence. When considering whether to impose a sentence of preventive detention the court must take into account:¹⁵⁰

- (a) any pattern of serious offending disclosed by the offender's history; and
- (b) the seriousness of the harm to the community caused by the offending; and
- (c) information indicating a tendency to commit serious offences in future; and
- (d) the absence of, or failure of, efforts by the offender to address the cause or causes of the offending; and
- (e) the principle that a lengthy determinate sentence is preferable if this provides adequate protection for society.

Genetic predisposition evidence could constitute “information indicating a tendency to

¹⁴⁸ Sentencing Act 2002, s 87.

¹⁴⁹ Section 87(5).

¹⁵⁰ Section 87.

commit serious offences”. Furthermore, such an offender might be perceived as unable to address the underlying causes of the offending, which would reflect negatively upon them. It may not necessarily be true that an offender cannot address their aggressive behaviour, MAOA-L genotype evidence is merely a risk factor for the development of aggressive behaviour. However, there has been a tendency in the popular media for genetic evidence to be construed in a simple, deterministic manner. There is always a risk that the courts will interpret the evidence in light of such portrayals. Genetic evidence might thus be highly detrimental.

It is also worth considering that the preventive detention system raises human rights concerns “that a person’s liberty is being curtailed due to an assessment that a person may commit an act in the future.”¹⁵¹ In the context of genetic predisposition evidence, the human rights concerns associated with preventive detention may be further exacerbated since the risk factors used in the assessment are beyond the offender’s ability to control, and beyond his or her capacity to assume responsibility for.

5 MAOA-L evidence and parole decisions – assessments of future dangerousness

In addition to the preventive detention dimension, there is also the issue that evidence presented during sentencing by defence counsel might later be relied upon at parole hearings as evidence that the offender poses a harm to the community. Section 7(1), Parole Act 2002 provides that:

When making decisions about, or in any way relating to, the release of an offender, the paramount consideration for the Board in every case is the safety of the community.

Evidence of a predisposition towards aggressive behaviour could be a factor for the Board to consider in determining whether the release of the offender would be a danger to the community, and thus should not happen. There are also concerns that the Board might place too much emphasis on such evidence. Given that MAOA-L genotype evidence is only a risk factor, and not is determinative, it would be best if the Board consulted geneticists to

¹⁵¹ Susan Glazebrook “Risky Business: Predicting Recidivism” (2010) 17 *Psychiatr Psychol Law* 88 at 90.

ensure that the predictive value of the evidence is not overestimated.

Concerns have been raised that assessors may overestimate the risk which an offender poses. This is because assessors will be aware that the consequences of false negatives are much more observable than the consequences of false positives. Indeed, the consequences of a false positive may never be observed.¹⁵² A false negative in this context would be where an offender is deemed to be at low risk, is released on parole, and then offends. In contrast, a false positive would occur where an offender is *mistakenly* considered to be at high risk of reoffending, and is not released. In such a situation, it would be difficult to detect the mistake in the assessment. Given this reality, there is a danger that the risk associated with genotype evidence may be overestimated, to the detriment of the defendant.

6 Which way will the knife cut?

At best, genetic predisposition evidence is open to manipulation by counsel on both sides. At worst, it could cause the defendant to be perceived as a peril to community and beyond redemption. Such findings might inform risk prediction decisions by parole boards or judges considering a sentence of preventive detention. Moreover, an analysis of the principles of sentencing highlights the potential for MAOA-L genotype evidence to be construed as an aggravating factor. Subject to situations such as the Waldroup case, where the defendant's options lay somewhere between death and a long time in jail, the admission of genetic predisposition evidence by defence counsel is a tactic fraught with danger, and is probably not worth the risk.

F Dangers of Misinterpretation

In addition to the potential for MAOA-L genotype evidence to be construed as both an aggravating and a mitigating factor, there are grave risks of misinterpretation.

As with any matter in law, the way an argument is constructed, and language used, will influence the reception it receives. One suggestion is that the MAOA-L allele could be

¹⁵² Armon Tamatea, Nick Lascelles and Devon Polaschek "Criminal Justice Psychology in Aotearoa New Zealand: Issues for Practitioners" *Professional Practice of Psychology in Aotearoa New Zealand* 451 at 465.

portrayed as a ‘susceptibility gene’.¹⁵³ This suggestion is founded upon an interpretation of the Caspi study, which indicates that MAOA-L carriers may be more susceptible to the negative effects conferred by a childhood of maltreatment and abuse. Rather than placing the emphasis on the criminal character of a defendant, this conception of MAOA-L further characterises the defendant as a victim of factors beyond his or her control. This language may be particularly appealing for counsel seeking to highlight the vulnerability and diminished culpability of a defendant.

Conversely, the gene could be branded as a “criminal gene” or a “violence gene”. Such conceptions might be used to reinforce perceptions of the criminal nature of a defendant, and thus might be useful for counsel seeking to highlight the aggravating nature of the evidence, and the danger posed by the defendant to the community.

While practically useful, such simplistic language is potentially misleading. Indeed, the utility of such phrases lies in their capacity to mislead. Where genetic predisposition evidence is raised, it is important that decision makers are made aware of the underlying science, so that they are not swayed by gross oversimplifications.

There are serious risks that genetic evidence, and the statistics used to convey a particular message, will be misinterpreted. Most people have “a strong tendency to assume they understand how the world works in far more detail than they really do”.¹⁵⁴ In this highly complex field, this may lead to jurors or judges drawing incorrect, and simplistic assumptions. Indeed, cases such as that of Bradley Waldroup illustrate the potential for jury members to be overly swayed by their misunderstanding of genetic evidence.

¹⁵³ Jay Belsky “The nature (and nurture?) of plasticity in early human development” (2009) 4(4) *Perspecti Psychol Sci* 345.

¹⁵⁴ Frank Keil “Getting to the Truth: Grounding Incomplete Knowledge” (2008) 73 *Brook L Rev* 1035 at 1038.

G An Illustration of the Potential for the Misinterpretation of Genetics

In order to illustrate this point, it is useful to explore a brief example relating to the XYY syndrome. In the case of *People v Yukl*, the court explained that:¹⁵⁵

[A] court in Australia has accepted the XYY syndrome as part of a valid insanity defense. Laurence E. Hannett was charged with murder and was acquitted after a psychiatrist testified that every cell in his body was abnormal.¹⁵⁶

Such a statement illustrates the comprehension problems that can arise when discussing genetic evidence. Technically, this statement is entirely accurate, every cell in the defendant's body would have had an extra Y chromosome. At a chromosomal level, his cells would all have been highly abnormal. However, this statement is painfully open to misinterpretation. To a layperson this language could easily be interpreted as meaning that something was seriously wrong with the defendant, so seriously wrong that he might have been incapable of deciphering right from wrong. This is not a claim which can be substantiated by evidence of an XYY condition.¹⁵⁷ Quite to the contrary, XYY individuals can lead entirely normal lives.¹⁵⁸ Great care must be taken with how scientific evidence is presented in court so that the risk of misinterpretation is minimised.

¹⁵⁵ *People v Yukl* 83 Misc 2d 364, 372 NYS 2d 313.

¹⁵⁶ It seems there is some confusion about the defendant's name and the weight which was given to the evidence of his XYY condition. For example, Richard Fox notes that: "The closing months of 1968 saw further newspaper attention devoted to the XYY defect in the report[] of the trial[] of *Lawrence Edward Hannell* in Melbourne, Australia..." (emphasis added). The author also explains that "[the XYY condition] did not significantly affect the jury's determination that he was not guilty on the ground of insanity since, on any interpretation of his conduct, Hannell was clearly legally insane..." see Richard Fox, above n 1, at 60.

¹⁵⁷ There may have been further substance to the explanation, but I am using this example to illustrate what could happen, not necessarily what did happen.

¹⁵⁸ Bernet et al. note that "Men with an XYY karyotype were once thought to be more aggressive and violent, but this theory was largely disproved by larger, population-based studies. However, the presence of the extra Y chromosome may confer some greater risk for antisocial behavior because of an association with learning problems and low intelligence." See William Bernet and others "Bad Nature, Bad Nurture, and Testimony regarding MAOA and SLC6A4 Genotyping at Murder Trials" (2007) 52(6) *Journal of Forensic Sciences* 1362 at 1363.

H Some Words of Caution

Building on this point, in relation to the characterisation of MAOA, Debra Wilson notes that “[i]t is important that whatever term is ultimately chosen, it accurately reflects what the studies are describing.”¹⁵⁹ Behavioural genetics is a highly complex field, and simplistic labels only increase the chance of misunderstandings. If expert witnesses are compelled to discuss genetic evidence in court, they must take great care with the way such evidence is presented. This concern is particularly real given the common misconceptions of the “warrior gene” which have been broadcasted by the popular media. Likewise, if counsel properly understand the underlying science, it would be unethical for them to create arguments which are misleading and unsubstantiated.

Most importantly, the courts should be wary about the use of genotype evidence, especially where there is a lack of scientific consensus. Scientists should also remember their responsibility to acknowledge contrary views, and present the evidence in an objective manner. This might seem rather obvious, but there are serious concerns that expert witnesses might be driven by subconscious bias in the delivery of their testimony.¹⁶⁰

If an expert delivers their testimony in a biased manner, it may be difficult for a judge, who lacks the appropriate scientific background, to evaluate their testimony.¹⁶¹ As Walter Lippmann, famously noted:¹⁶²

If [the expert’s] advice is followed, and he is wrong, the consequences may be incalculable... [for] except on a few subjects where our knowledge is great, we cannot choose between true and false accounts.

¹⁵⁹ Debra Wilson, above n 1, at 105.

¹⁶⁰ For example, Tinsley notes that: “a scientist who has extensively researched the fallibility of human memory is likely to feel a natural sympathy for defence arguments of misidentification” see Yvette Tinsley, above n 127, at 851.

¹⁶¹ At 850.

¹⁶² Walter Lippmann *Public Opinion* (New York, 1922).

VII Ethical Issues

This final section will explore some of the ethical issues which may arise as our knowledge and acceptance of genetics evolves. This point of this section is not to provide a comprehensive analysis, but simply to highlight some potential issues.

A Genetic Screening of New-borns

Given the remarkable progress which genetics has made in the last century, we can confidently expect science to continue to deliver insights into the genetic makeup of aggressive offenders. As the link between risk factors and aggressive behaviour strengthens there may be a powerful case to answer for the mandatory genetic screening of crime suspects, and indeed, the general population.¹⁶³

If courts accept MAOA-L genotype evidence, or any other genetic predisposition evidence as part of offender risk assessment, then this would support claims for the genetic screening of offenders. A claim could be made that such screening should be mandatory, since it would improve the accuracy of the algorithms currently used for reoffending risk prediction such as ROC*ROI. Thus, genetic information might conceivably be used to reduce crime.¹⁶⁴ Genetic effects are often individually minor, and screening for one gene may not be that useful. However, screening for multiple genes and risk factors associated with a behaviour could “significantly improve risk management.”¹⁶⁵

There is also the potential for population wide genetic screening. New Zealand currently has a system of new-born genetic screening in place. This process is not compulsory and requires the informed consent of the new-born’s parents. Currently, 28 different conditions

¹⁶³ The potential for screening is of course not limited to genetic testing. In the future, neuroimaging techniques or psychological analyses could also be used during early childhood to provide an assessment of risk.

¹⁶⁴ For an explanation of ROC*ROI see Department of Corrections “Risk of Reconviction” <http://www.corrections.govt.nz/resources/research_and_statistics/risk-of-reconviction.html>

¹⁶⁵ Macià Buades-Rotger and David Gallardo-Pujol, above n 48, at 195.

are tested for and the compliance rate with this procedure is reported to be 99%.¹⁶⁶ It is not much of a stretch to consider the inclusion of tests for aggression risk factors such as MAOA genotype. Indeed, some scientists have expressly viewed this as a possibility in the future. In response to the Tiihonen study (see Part III), Dr von Schantz, told the Independent Newspaper that:¹⁶⁷

findings such as these may make it possible, in [the] future, to screen people with vulnerable backgrounds and identify those who are at greater risk of becoming offenders, so that they can get appropriate help before they commit any serious violent crimes.

This proposition is not that far-fetched. In the future, population wide screening might be economically viable. Given the incredible technological progress since the start of the Human Genome Project, complete genomes can now be sequenced for relatively affordable prices. The original Human Genome Project was estimated to cost around \$2.7 billion.¹⁶⁸ By 2006, estimates put the cost of sequencing a complete genome at a leisurely \$14-25 million.¹⁶⁹ Remarkably, a genome can now be sequenced for around \$1000.¹⁷⁰ Moreover, a complete genome sequence is not necessary to test for a particular gene. Currently, MAOA genotype tests can be conducted for a mere \$250.¹⁷¹ This might still be too expensive for wide spread application; however, if a number of competitors begin providing the service then we can reasonably expect prices to fall. Indeed, as the above

¹⁶⁶ Debra Wilson, above n 1, at 190.

¹⁶⁷ Steve Connor, above n 62.

¹⁶⁸ “[This] number represents the total U.S. funding for a wide range of scientific activities under the HGP's umbrella beyond human genome sequencing, including technology development, physical and genetic mapping, model organism genome mapping and sequencing, bioethics research, and program management.” See National Human Genome Research Institute “The Cost of Sequencing a Human Genome” (6 July, 2016) <<https://www.genome.gov/27565109/the-cost-of-sequencing-a-human-genome/>>.

¹⁶⁹ National Human Genome Research Institute.

¹⁷⁰ National Human Genome Research Institute.

¹⁷¹ Debra Wilson, above n 1, at 201.

discussion illustrates, technological improvements in this area are rapidly driving prices down.

B Screening for MAOA-L, the Benefits and the Problems

If the science reaches a sufficient level of certainty about the relationship between genetics and aggressive behaviour, then there may be a strong case for genetic screening. As mentioned, genetic evidence could be used to enhance the performance of risk prediction algorithms. However, the benefits may not just be societal. Members of the population who are made aware of their increased likelihood of behaving aggressively at an early age might be better placed to seek some form of intervention to help manage any issues which they may face. Knowledge of a predisposition towards aggression or antisocial behaviour might provide the necessary impetus for an individual to seek help, and take steps to reduce the risk of harmful future behaviour. On this basis, genetic screening could be construed as a significant advantage, not only from society's perspective, but from that of the individual. However, there are serious issues.

1 Those most in need of treatment will be the least able to access it

This is very much a hypothetical discussion, and there is no knowing what the science of tomorrow will reveal. However, one of the main risk factors for aggression in the literature is the *MAOA-L genotype x childhood maltreatment interaction*. This raises issues from a screening and treatment perspective. Children growing up in an environment of abuse and neglect are much less likely to have access to support to manage their "condition", and thus genetic screening at birth for MAOA-L might be futile. Those most at risk of offending will be the least able to access support.

In contrast, those children able to receive help may be less likely to need it. For example, a finding from the Caspi study was that "[c]hildren who grew up in households rated as likely nonabusive had the same low risk of becoming violent adults, regardless of their level of MAOA."¹⁷² Of course, the science may establish strong genetic risk factors which

¹⁷² Jonathan Kaplan, above n 37, at 69.

are independent of any history of childhood maltreatment. There seems to be evidence of this in the Tiihonen study.¹⁷³ Nevertheless, this is an issue worth considering.

But there is a further concern that this discussion around genetics may have missed a fundamental point. Whatever one's MAOA genotype, childhood maltreatment is a *major* risk factor for criminal behaviour.¹⁷⁴ It makes sense for this issue to be addressed first, rather than pursuing the more contentious links between genetics and aggression. Indeed, endeavours aimed at preventing childhood abuse would have other highly beneficial consequences for children, most obviously the avoidance of abusive childhoods. This seems like a better use of society's limited resources.

One idea might be to target special initiatives towards improving the family environments of individuals known to have the MAOA-L allele, and thus preventing childhood maltreatment in the case of children who may be the most susceptible towards the negative effects of abusive childhoods. However, this does not seem to be a feasible distinction. All children should have the right to a safe family environment, and support initiatives should not discriminate between families on the basis of genetic differences.

2 Labelling effects, stigma and the self-fulfilling genetic prophesy

If compulsory screening were conducted, then designating certain individuals as "at risk" might have self-fulfilling prophecies. One interesting example comes from a study showing that "[a] person who believes she is an alcoholic, when informed a particular drink contains alcohol, will consume more of the drink than the average person, even if there is no alcohol in the drink."¹⁷⁵ Such self-fulfilling prophecies might also feature in the context of a known predisposition towards aggression. It seems quite possible that individuals who view themselves as predisposed towards violence, might be less inclined to seek help to try and manage any aggressive tendencies which they may have, or attempt to exercise self-

¹⁷³ See the discussion in Part III.

¹⁷⁴ Avshalom Caspi and others, above n 51, at 851-854.

¹⁷⁵ Allison Morse "Searching for the Holy Grail: The Human Genome Project and Its Implications" (1998-99) 13 J Law & Health 219 at 242.

restraint. It has also been argued that “the belief that a negative event is genetically mandated results in decreased self-esteem and feelings of hopelessness and depression.”¹⁷⁶

Besides these negative impacts, there is also the potential for an “at risk” individual to be actively discriminated against. For example, employers, or insurance companies might request genotype information,¹⁷⁷ and indeed might place undue emphasis on the results of genetic screening. The use of genotype evidence in such a way would be particularly problematic given that the evidence examined in this paper is only suggestive of a risk of aggressive behaviour. Access to such information could thus be unfairly prejudicial and might lead to certain individuals with no history of violent behaviour, and who may never offend, being perceived as aggressive offenders, and being shunned by society. These concerns are truly worrying. In fact, such problems could manifest themselves regardless of their scientific validity. Stigma and discrimination are largely grounded in matters of perception, not scientific fact.¹⁷⁸ Fundamentally misguided perceptions and prejudices have the potential to cause serious harm. Genetic screening opens the door to a whole new world of discrimination.

3 The issue of consent

Another crucial issue with screening initiatives is that many families may not want their children to be screened. This is entirely understandable given the potential labelling and stigmatising effects associated with being designated as an “at risk” individual. While genetic screening at birth in New Zealand is voluntary and has a high participation rate, this is probably because knowing the results of such analyses will be largely positive for new-borns and their families. If the new-born has a genetic condition which is treatable, then it is good to know this at an early stage so that an optimal treatment strategy can be devised. Parents, however may be very reluctant to have their child tested if a “positive result” is perceived as jeopardising their child’s future. Therefore, participation in such

¹⁷⁶ Robert Stone “The Cloudy Crystal Ball: Genetics, Child Abuse and the Perils of Predicting Behavior” (2003) 56 Vand L Rev 1557 at 1566.

¹⁷⁷ At 1567.

¹⁷⁸ At 1566.

screening procedures may be quite low. Naturally, this would make the procedure much less effective in terms of providing nationwide data for use in risk assessment.

4 Privacy concerns

Once genetic evidence is obtained from an individual, that individual has little control over the access to and distribution of the information. In the case of genetic information obtained at birth, an individual will have not consented to the taking of such information, and may not even know of its existence. It may be tempting for police and lawyers to access and exploit information in genetic databases, especially if a stronger link is established between aggression and specific genetic markers. In a world where accurate risk predictions depend on genetic evidence, an individual's privacy rights may pale in comparison to the predictive utility of the evidence.

Of course, DNA from suspects is used all the time in the criminal justice system. However, privacy is less of an issue where DNA evidence is used in a conventional sense, in order to identify a suspect. However, if DNA evidence is used by police, without consent, to identify particular traits in a suspect, such as a predisposition towards violence, then this raises much more serious privacy concerns. Indeed, even without a system of genetic screening at birth, police may be able to extract this knowledge from their own offender DNA databases, or from samples obtained at the crime scene.

The genes we have constitute highly personal information which one may not wish to share with the world. While environmental influences undoubtedly play a huge role in shaping our futures, the role of genetics cannot be ignored. At a fundamental level, our DNA provides the basis for who we are, and the thought of police tapping into such information to gain hidden insights into our deepest and darkest secrets is a concerning one. In a hypothetical future society, the police could come to understand things about an individual which even they would not know about themselves.

Overall, genetic screening and the construction of population risk databases raises massive ethical concerns. These issues may seriously jeopardise the possibility of using genetic

information for the purposes of screening, when and if, the science progresses to such a level.

VIII Conclusion

Recent developments in the field of behavioural genetics have sparked a renewed interest in the role of genetic evidence in the criminal law. While there are many risk factors for aggressive behaviour, one highly promising candidate is the low expression allele of the MAOA gene. Early attempts to use MAOA-L genotype evidence in the courts have been shrouded in controversy, and indeed have been tainted by failed historical experiments to draw links between XYY syndrome and aggressive behaviour. Scientists have also been very careful to reject theories of genetic determinism. There is no evidence of a “warrior gene” in the sense of a gene which differentiates criminals from law abiding citizens. While conditions such as the rare Brunner’s syndrome or severe mental disabilities may make an individual much more likely to behave violently or irrationally, references to genetic determinism should be avoided.

However, there is a growing body of evidence identifying the MAOA-L allele, or the MAOA-L allele in conjunction with a background of childhood maltreatment, as a significant risk factor for aggressive behaviour. While such evidence should not be relevant to questions of criminal liability, it could be relevant during sentencing. MAOA genotype evidence, as a risk factor for aggressive behaviour, can be viewed as analogous to accepted risk factors. The appropriate weight given to such evidence is another question altogether, and it is a decision which may require careful scrutiny. It is also important to remember that the evidence points to MAOA-L being a risk factor for acts of impulsive aggression committed by men, and not for premeditated crimes.

In terms of how such evidence might be construed, MAOA-L genotype evidence can be interpreted as both an aggravating and mitigating factor. In one sense, it can act as a marker for the future dangerousness of the defendant. In another sense, it can be utilised to attract the sympathy of the court. Moreover, even if MAOA-L genotype evidence is accepted at

first instance as a mitigating factor, the admission of such evidence could come back to bite the defendant in the context of risk assessment by the parole board. Thus, the utility of such evidence is highly situational. Indeed, there is good reason to suspect that in the New Zealand context MAOA-L evidence may be construed as highlighting the future dangerousness of a defendant.

This particular issue is not confined to the interpretation of genetic predisposition evidence. Indeed, evidence of neurological disability such as that associated with foetal alcohol spectrum disorder, or with a traumatic brain injury can give rise to the same interpretive issues. One way to remedy any concerns about inconsistency in sentencing might be for the legislature to introduce further legislative guidance into the Sentencing Act, stipulating whether the conditions have an aggravating or mitigating nature.

Additionally, the implications of genetic research are not confined to the courtroom. Our progress in understanding MAOA, and other risk markers for aggression, raises strong ethical concerns. As science continues to advance, there may be a temptation for governments to introduce genetic screening tests at birth aimed at identifying particular genes associated with aggressive behaviour. In the not so distant future we may have to ask ourselves whether society's right to prevent crime and maintain social order should take precedence over the individual's right to prevent an inquiry into their genetic makeup.

Finally, it is appropriate to end this discussion with a word of caution; genetic evidence is highly open to misinterpretation. We must proceed with care in the legal system and ensure that decisions are not founded upon erroneous science. Genetics may give us new insights into the complexities of human behaviour. However, scientific progress also opens the door to a world of misinterpretation and injustice. It is impossible to accurately assess that which one cannot understand, and this is a field in which much is not understood.

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