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Involuntary Neurotoxicity, Criminal Responsibility and the Strange Case of David Garabedian

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Abstract

The legal concept of insanity spans more than a thousand years. Concurrently, our understanding of neuropathology has continuously evolved. Psychological knowledge has resulted in reformulations of the insanity defence, and spawned the defences of diminished capacity and diminished responsibility, offshoots of the insanity defence. As the study of neurobiology flourishes, evidence is multiplying that important aspects of behaviour can be affected via involuntary exposure to neuro-modulating substances with wide-ranging results, from severe psychiatric disturbances to murderous rage. But the inherent complexities of defining insanity are compounded by new insights into the workings of the human brain. This research uses the research methodologies of both historical and doctrinal legal research. By looking back at the first attempt at an involuntary neurotoxic damage defence, this paper investigates the manner in which contemporary neuroscientific findings interface with the traditional jurisprudence of criminal non-responsibility and provides a guidepost for legal, scholarly, and forensic practitioners.

Keywords: insanity defence, involuntary neurotoxic damage, criminal jurisprudence, forensic psychology, criminal non-responsibility, criminal culpability, diminished capacity, diminished responsibility

Introduction

On a March afternoon in 1983, David Garabedian, a mild-mannered community college student and part-time employee of the Old Fox Lawn Care Company in Massachusetts, arrived at the home of Eileen Muldoon to spray her lawn with insecticides. He knocked on the door and received no response, so he surveyed the front lawn and calculated a cost estimate. What happened next would reverberate throughout the jurisprudential ages, pivoting the legendary *M'Naughten* on its axis and forever altering the foundational premises of centuries-old *mens rea*. In fact, Mrs. Muldoon was home that day, an altercation ensued, and Garabedian, a model citizen who had never shown violent tendencies in the past, suddenly dislodged nearby stones and bludgeoned Mrs. Muldoon to death (Commonwealth v. Garabedian, 1988).

Garabedian was charged with first-degree murder, and, based on the facts, it looked like an open-and-shut case. What possible defence could he have? But Garabedian's lawyer entered a plea of insanity. The unusual defence – the "involuntary neurotoxic damage defence," as it came to be known – was predicated on chemical poisoning of the nervous system that impeded Garabedian's capacity to control his temper. It was the first such defence ever used in a criminal trial. As Garabedian formulated his unique defence based on prolonged exposure to allegedly mind-altering chemical agents, the ensuing trial called into question what role neurobiology plays in the intent to commit murder.

At Garabedian's trial, the defence argued that Garabedian was under the influence of "acetylcholinesterase inhibitors," a main component of the lawn-care chemicals he was using, and that, furthermore, involuntary exposure to the chemicals had profoundly affected Garabedian's ability to modulate an appropriate hostile response (Commonwealth v. Garabedian, 1988).

How could this possibly be? The defence's expert would explain. As the newfound study of neurobiology began flourishing, evidence was multiplying that important aspects of one's physiology and even behaviour can be affected via involuntary exposure to sublethal doses of neurotoxins, with results ranging from mild gastrointestinal discomfort to severe psychiatric disturbances, even violence. The defence produced a timeline that illustrated that, in the weeks leading up to the murder, Garabedian exhibited systemic symptoms of being under the influence of this neurotoxin. He had become unusually fatigued, irritable, tense, and impatient. On one occasion, he uncharacteristically argued with and struck his younger sister. He frequently experienced abdominal pain, diarrhoea, weight loss, and his skin itched. He salivated excessively and complained of headaches. Of importance, he frequently experienced urinary urgency. On the day of the murder, according to Garabedian, he felt a strong urge

to urinate, and, under the impression that no one was home, walked around the back of the house and proceeded to urinate on the lawn. Mrs. Muldoon emerged from her home unexpectedly and berated Garabedian. Minutes later, she was dead (Commonwealth v. Garabedian, 1988).

Since the Garabedian case, a vast body of scientific literature has emerged concerning the workings of the brain at the neurobiological level. This paper explores how these scientific findings may impact criminal non-responsibility. The paper employs an historical doctrinal analysis of the history of the insanity defence and the legal requirements for criminal non-responsibility. The paper also elucidates medical evidence linking neurochemicals with violent behaviour and re-examines our understanding of the role that neurobiology plays in criminal culpability. Finally, the paper employs doctrinal legal research to explore our understanding of what role the biochemical machinery that powers volition is playing in conviction. This paper investigates the manner in which contemporary neuroscientific findings interface with the jurisprudence of criminal non-responsibility and provides a guidepost for legal, scholarly, and forensic psychology practitioners.

Background

Neuroscientific Explanations for Aggression

Molecular Neurobiology of the Amygdala: Absence of Intent

It is humbling and frightening to consider that human rationality may depend on the normal functioning of tissue within our skulls, but neurobiologists have recently mapped out complex molecular mechanisms that mediate behaviour, including aggression. Just before the turn of the twentieth century, Dr. F.L. Goltz demonstrated that lesions of the forebrain transform a gentle animal into one that explodes into a rage and launches a vicious attack upon minimal provocation (Britannica, 2021; Dressler & Potter, 1991, pp.
250-256; Gamboa, 2020). It took another fifty years for scientists to identify the limbic system in the brain specifically as the area from which aggression emanates. More recently, scientists have further noted that violence that erupts from the limbic system often does so despite the absence of any intent on the part of the perpetrator (Dressler & Potter, 1991, pp. 250-256). Finally, molecular biologists have determined that acetylcholine is the neurotransmitter responsible for mediating brain functioning, including aggressive responses, in the limbic system (Kandel et al., 1995).

Acetylcholinesterase inhibitors have typically been used in chemical warfare. These "nerve gases" operate to leave the diaphragm stuck in the flexed position, such that the victim cannot exhale or draw more breath. This respiratory paralysis is lethal. However, researchers discovered that in much smaller doses, the chemical could have the effect of killing insects, but not humans. Therefore, the first lawn insecticides were diluted versions of nerve gas. Since acetylcholine is involved in the operation of muscle tissue in the intestine and bladder and the activation of the autonomic nervous system, exposure to lawn insecticides of this type was known to cause fatigue, loss of appetite, abdominal cramps, diarrhoea, excessive perspiration and salivation, and urinary urgency (Colovic et al., 2013; Dressler & Potter, 1991).

The Unusual Case of David Garabedian: The Molecular Mechanism of Intent

David Garabedian, charged with first-degree murder, pled temporary insanity. He claimed that the "acetylcholinesterase inhibitor," concentrated in the lawn-care product that he used frequently, affected his nervous system in such a way as to extremely limit his ability to control his temper (Commonwealth v. Garabedian, 503 N.E.2d 1290, (1988). The case was constructed by Dr. Peter Spencer, a toxicologist and professor of neuroscience at the Albert Einstein College of Medicine in New York, and Dr. David Bear, a neuroscientist at the University of Massachusetts. Acetylcholinesterase, the doctors stated, is responsible for limiting the action of acetylcholine, a neurotransmitter found in high concentrations in neuromuscular junctions. In the presence of an inhibitor enzyme, acetylcholinesterase is prevented from terminating the action of acetylcholine. Therefore, acetylcholine remains active within the tiny spaces between nerve and muscle, maintaining the flexed position in muscle tissues. The neurotransmitter is also found in the section of the brain that mediates fear and aggression (Kandel, Schwartz & Jessell, 1995). The main question in this case was whether prolonged activation of acetylcholine within the aggression centres in the brain causes uncontrollable changes in one's behaviour and actions.

Dr. Bear and his colleague, Dr. Orin Devinsky, Chairman of Neurology at New York University Hospital, believed that there is a causal connection between exposure to this enzyme and increased aggression (Dressler & Potter, 1991). But while the explanation posited by these learned medical scientists seemed entirely plausible, there was no way to prove it. And the crucial fact that so many others had been exposed to pesticides and had not committed murder remained.

Observed Link between Acetylcholinesterase Inhibitor and Aggression

Dursban

Critics of Garabedian's defence pointed to the fact that others in contact with Dursban, the pesticide used by Old Fox Lawn Care, did not commit murder, and cited this as proof that Dursban must be safe. The United States government did not agree. Shortly after Garabedian's murder conviction, the chemical Dursban underwent a "silent ban" in the U.S. That is to say, the government did not renew registrations of Dursban, therefore gradually phasing out the use of the chemical. The reason cited was the potential for nervous system damage (Asprion, 2021; Browner, 2000). Synthetic pyrethroids, which are more toxic than Dursban but break down faster, replaced traditional Ach inhibitors such as Dursban. Today, these pesticides are applied in such minuscule quantities that they would only have an effect if one had the body mass of a mosquito.

Our knowledge of sensory input, aging, language, and learning is constantly being modified. Recent literature on neurophysiology reveals a startling glimpse of the sweeping changes taking place in the field of cognitive neuroscience. Acetylcholine plays a crucial role in aggressive responses (Bear & Connors, 2020). But what role does exposure to Ach inhibitor play in modifying an aggressive response? At this point in time, without an autopsy, this information was impossible to ascertain. But the invention of advanced neuroimaging devices such as SPECT would shed new light on this case (Amen, Stubbefield, Carmicheal, & Thisted, 1996; Ozyurt, Yilmazlar, Tamgaç & Kaplan, 1997). SPECT images of those exposed to pesticides have decisively shown that exposure manifests as long-term damage to the (left) parietal lobe (Ozyurt, Yilmazlar, Tamgaç & Kaplan, 1997). Interestingly, this lobe houses the limbic system, including the amygdala, the hippocampus, and the hypothalamus. Still, the long-term effects could not conclusively be measured without the involvement of a large number of subjects.

Sarin

On March 20, 1995, a terrorist group released sarin gas, an acetylcholinesterase inhibitor used as a chemical warfare agent, in a Tokyo subway during rush hour. The sarin was released on three separate subway lines when terrorists, before fleeing the trains, used umbrellas to puncture lunch-boxes and other containers that held the gas. More than 5,500 people were injured and 11 were killed (Minami, Hui, Wang, Katsumata, Inagaki, Li, Inuzuka, Mashiko, Yamamoto, Ootsuka, Boulet, & Clement, 1998). Treating physicians recognized agitation and irritability among the injured, an effect of sarin exposure. Many were diagnosed with post-traumatic stress disorder ("PTSD"). A small percentage required ongoing psychiatric treatment secondary to psychological symptoms including agitation, irritability, anxiety, and PTSD. Notable symptoms of those diagnosed with PTSD included angry outbursts and hypervigilance (Yokoyama K, Araki S, Murata K, Nishikitani M, Okumura T, Ishimatsu S, Takasu, 1998). However, a study performed on sarin attack victims six months later resulted in several unique findings. Scientists discovered that the chronic neurologic sequelae manifested as psychological symptoms were directly related to dysfunctional acetylcholinesterase activity and that this symptomatology had been diagnosed as PTSD (Yokoyama K, Araki S, Murata K, Nishikitani M, Okumura T, Ishimatsu S, Takasu, 1998). Researchers noted that sarin, the acetylcholinesterase inhibitor, possessed a neurotoxic action that lasted beyond the turnover period of the neurotoxin itself, in addition to an inhibitory action of the acetylcholine in the brain. Clearly, acetylcholinesterase inhibitors exert a much more detrimental effect on the brain than anyone had ever thought, and the effects were regular in studies with participants numbering in the hundreds and even thousands. Approximately one-third of all subjects in every study published regarding the sarin attack exhibited some type of heightened aggressive response requiring ongoing clinical treatment (Yokoyama K, Araki S, Murata K, Nishikitani M, Okumura T, Ishimatsu S, Takasu, 1998).

Donepezil

Acetylcholine has been implicated as the neurotransmitter responsible for Alzheimer's disease. The drug Donepezil, which is a reversible cholinesterase inhibitor, has been newly developed to counter the effects of this disease by increasing the availability of "intrasynaptic" acetylcholine in the brain. In essence, the molecular mechanism of the drug is similar to that of Dursban, sarin, and other acetylcholinesterase inhibitors. Early in this drug's history, the *American Journal of Psychiatry* printed a warning issued by Dr. W.P. Bouman and Dr. G. Pinner of Nottingham, England. Under the heading *Violent Behavior Associated with Donepezil*, Dr.'s Bouman and Pinner described a 76-year-old man with Alzheimer's whose medical history was otherwise completely unremarkable. He started a regimen of Donepezil, and five days later, began beating his wife and was discovered to be holding her hostage at knifepoint. Physical and laboratory findings, including CT scans, revealed no abnormalities aside from generalized atrophy. The acetylcholinesterase inhibitor was discontinued and he was administered haloperidol, a psychotropic medication. Within a few days, his behaviour returned to normal. When contacted by these physicians, the drug company that manufactures the acetylcholinesterase inhibitor revealed that five percent of patients taking Donepezil develop agitation, and *one percent develop actual physical aggression*.

The doctors reasoned that "although a causal relationship between the violent incident and Donepezil cannot be proven, a temporal relationship between the commencement of Donepezil and the occurrence of behavioural disturbance in ... a patient with no previous history of violence, warrants caution with the prescription of this drug" (Bouman & Pinner, 1998). Other physicians also noted behavioural complications with this drug (Wengel, Roccaforte, Burke, Bayer, McNeilly, Knop, 1998). And more recently, mania and psychotic disturbance were also noted in individuals taking this acetylcholinesterase inhibitor (Benazzi, 1999).

The reversible acetylcholinesterase inhibitor Donepezil remained in use for the clinical treatment of early signs of dementia, despite reports of adverse side effects including abnormal cardiac rhythm, insomnia, vomiting, and muscle cramps. The most recent studies on this drug have sought to understand the extent to which these side effects are actually caused by the acetylcholinesterase inhibitor and not just naturally existing within the clinical population. One such study, an elegant 2020 study using zebrafish, sought to analyse the potential adverse effects of Donepezil on short-term memory and behaviour of normal zebrafish. Adult zebrafish were exposed to the drug, which apparently caused a slight improvement in the short-term memory of the zebrafish but concurrently induced a significant elevation in aggressiveness. Overall, researchers concluded that chronic exposure to the acetylcholinesterase inhibitor was capable of severely inducing adverse side effects upon a normal species, zebrafish, in a dose-dependent manner. Researchers further concluded that these unexpected adverse effects as to behavioural alteration should be carefully addressed in future studies (Audira, Ngoc Anh, Ngoc Hieu, Malhotra, Siregar, Villalobos, Villaflores, Ger, Huang, Chen, Hsiao, 2020).

Clearly, neuroscientific research has shone a brighter light upon the molecular mechanism of aggression via the metabolism of acetylcholine within the amygdala. Neuroimaging, behavioural neurology, and molecular neurobiological analysis have all converged to indicate that when the breakdown of acetylcholine is attenuated via the introduction of an enzymatic substance, aggression can and does result. But where does this new understanding fit within our traditional legal maxims? And should it fit anywhere at all? Do traditional notions of guilt and intent readily encompass neurobiological differences? Are juries equipped to draw the demarcation of where free will ends and neurobiology begins?

Historical Doctrinal Analysis

Jurisprudence of Criminal Culpability

Law assumes free will. The essential underpinning of free will is that individuals possess a consciousness from which rational thought can be derived. The very concept of law presupposes that an individual possesses sufficient rationality, self-control, and self-regulation to be able to conform his or her conduct to the law (Daly, 2015).

We now know that the human brain can malfunction, even temporarily, due to conditions over which a person has no control, and this malfunction can impair a person's capacity to know what he is doing and to remember afterward what he has done. Such knowledge has spawned the defences of diminished capacity and criminal non-responsibility, offshoots of the insanity defence. The intricacies of defining insanity are complicated by new insights into the inner workings of the human brain. When questions concerning brain compromise are brought to bear upon a criminal trial, how is responsibility to be gauged in these situations?

Understanding Legal Insanity: From Ancient Greece and Rome to Modern Day

Modern clinicians, jurists, and legal scholars sceptical of the insanity defence must be aware that there is no time in the recorded history of Western law when there has not been an insanity defence. Principles of the modern insanity defence are present as early as the homicide laws of the ancient Greek legislator Draco and within Rome's Twelve Tables (Robbins, 1990). The legal concept of insanity has been stalwartly set within the chapters of the history of jurisprudence from the very outset, and, of course, this is so out of necessity. Early thinkers recognized that there must be faculties within the human mind capable of contemplating the evil act. The Romans called it *mens rea*, literally Latin for a "guilty mind," for only those who could intend and comprehend the wrongdoing were deserving of punishment (Platt & Diamond, 1966). This concept is very roughly translated in modern legal systems to requisite intent, described in various ways in common law and spelled out statutorily in criminal codes. By the 16th century, the insanity defence had found its way into an English legal treatise as follows:

> "If a madman or a natural fool, or a lunatic in the time of his lunacy, or a child who apparently hath no knowledge of good nor evil do kill a man, this is no felonious act…for they cannot be said to have any understanding of will" (Lambarde, 1581).

Echoing the Romans, the English jurisprudence set forth that even in the presence of *actus reus*, or "guilty act," there could be no crime without *mens rea* (Simon, & Gold, 2004).

Throughout the Middle Ages and into the 17th century, treatises made frequent reference to the concept of "good and evil," likening insanity to a childlike quality marked by an infantile grasp of morality (Platt & Diamond, 1966). However, *Rex. v. Arnold* (1723) is the benchmark case most commonly cited as the historical beginning of the insanity defence in English common law. Arnold had attempted to shoot and kill a nobleman, but missed. He stood trial for murder, as the jurisprudential thinking at that time was that a defendant should not be spared the death penalty simply because they were a bad shot—an attempt on another's life was considered the same as if one had succeeded. Various testimonies depicted Arnold as an odd man. His sister testified that, as a child, Arnold was prone to burning down the house after a disagreement. His barber testified that he would off-handedly entice the barber to "cut my throat" by leaning backward during grooming and exposing his neck. Arnold himself believed that the nobleman was sending imps to disturb his sleep.

Upon the conclusion of the trial, Judge Robert Tracy instructed the jury "not to fret" about the need to employ the defence of insanity, citing the history of the insanity defence from Roman times to the current British common law understandings of insanity. Judge Tracy articulated for the jury what came to be known as the "wild beast" test of insanity:

> It is not every kind of frantic humour, or something unaccountable in man's behaviour, that points him out to be such a man as is exempted from punishment; it must be a man that is totally deprived of his understanding and memory, and doth not know what he is doing, no more than an infant, than a brute or wild beast; such a one is never the object of punishment (Moran, 1985).

Nearly a century later, a man named James Hadfield, a soldier who had previously sustained a serious head injury in battle by being struck by the handle of a sword, fired a pistol at the King of England, George III, as he entered the royal box theatre in Drury Lane (*R. v. Hadfield*). The shot narrowly missed the king, and Hadfield was charged with treason (Moran, 1985).

Hadfield, whose brain compromise was obvious to jurors at trial, suffered from delusions and believed that murdering the King would trigger the return of the Messiah. He was represented by one of the most distinguished trial lawyers in the United Kingdom at that time, Thomas Erskine (Allen, 2021; Stryker, 1949). Erskine mounted an extraordinary defence, one that would forever alter the course of criminal legal jurisprudence. In the two-pronged defence, Erskine first convinced the jury, by summarizing the history of the legal concept, that the insanity defence was perfectly acceptable and appropriate. He spoke of *non compos mentis* (not sane) and other terminology from the Twelve Tablets. But it was in the second prong of his ingenious defence that his brilliance gleamed. Erskine sought to both convince the jury that legal insanity was the appropriate defence under these circumstances while simultaneously convincing the jury that the criterion for legal insanity at the time was deficient. The "wild beast" criterion required the defendant to have no more rational power than an infant or brute. And yet, Hadfield had carefully planned his crime. Erskine, therefore, urged jurors to accept insanity as a defence but to reject the "wild beast" criterion. He employed the services of an expert in criminal insanity who testified that delusions were currently understood to be the hallmark of insanity. Erskine then argued to the jury that, if the delusional content of Hadfield's thought was true, then jurors must contemplate how the law should treat this defendant. In this case, Hadfield, however compromised his brain's thought process was, believed that he was fulfilling Divine commandments, and therefore, no court should convict a man for doing what is nothing less than the will of God. In essence, Erskine was successful in establishing a different standard for legal insanity, one based on a more contemporary scientific understanding of insanity at that time. Hadfield was acquitted with this defence. Until this point, individuals found not-guilty-by-reason-of-insanity were released from the courtroom. However, given the political climate at the time – wherein there was decided reluctance to grant freedom to one who had just made an attempt on the life of the King – the Criminal Lunatics Act 1800 (9 & 40 Geo 3 c 94) was hastily passed by Parliament. The new legal understanding of insanity was accompanied by newly designed facilities, which meant that Hadfield was sent to a place reserved for those deemed criminally insane. The case set the stage for a new conceptualization of legal insanity (Allen, 2021; Moran, 1985; Stryker, 1949).

A half-century later, in 1843, the definition of insanity was broadened as a result of the trial of Daniel M'Naghten. M'Naghten, convinced that a member of the Tory party was targeting him for persecution, shot at the Tory prime minister and hit the prime minister's secretary by mistake. At trial, M'Naghten was found not guilty by reason of insanity. The House of Lords formulated the so-called "M'Naghten rule," which would apply at trials where the defence of insanity was being raised. Specifically, the rule put forth that, to successfully lay out a defence of insanity, "it must be clearly proved that at the time of committing the act, the party accused was labouring under such a defective reason from disease of the mind as not to know the nature and quality of the act he was doing, or if he did know it, that he did not know that he was doing wrong" (R v. Daniel M'Naghten, 1843). Fuelled by an aversion to the insanity defence, public outrage followed M'Naghten's acquittal (Kadish, 1999).

This M'Naghten definition of insanity, while it has endured, has also perpetuated conflict about what constitutes a "mental defect." The American Law Institute's Model Penal Code refers to "a mental disease or defect

which results in a lack of substantial capacity to appreciate the criminality of one's conduct or to conform one's conduct to the requirements of the law." Other criteria for legal insanity in common law have focused upon the notion of impulsivity.

Contemporary Doctrinal Legal Research

Evolution of Diminished Capacity and Diminished Responsibility

The Problem with M'Naghten

The earliest American scholarly commentaries on criminal law referenced the "good and evil" test of legal insanity that permeated historical English jurisprudence, and, later, the "right and wrong" concept that pre-dated *M'Naghten*. The responsibility assigned to defendants deemed legally insane in American law during the nineteenth century was determined to be consistent with the traditional principles of English common law and bolstered by concepts and emerging expertise of medical jurisprudence (Buchanan, 2000). Isaac Ray, a forward-thinking forensic psychiatrist, had postulated that such a test as "right and wrong" was incompatible with current psychological knowledge of human behaviour. He adjudged that the American legal system's "right and wrong" test of responsibility was based on the presupposed notion that "the insane mind is not entirely deprived of [the] ... power of moral discernment, but on many subjects is perfectly rational and displays the exercise of a sound and well balanced mind" (Ray, 1839, p. 29). Dr. Ray's theories exerted little influence prior to the English Hadfield case. The U.S. courts embraced the Hadfield decision, and M'Naghten in 1843. The concept of the defendant's appreciation of right and wrong was maintained as the traditional test of insanity, until New Hampshire, influenced by Dr. Isaac Ray's contemporary thinking, decided *State v. Pike* in 1869. Here, the court rejected the traditional "right and wrong" test and instead formulated a test which excused from responsibility the defendant whose actions were "the offspring or product of mental disease" (*State v. Pike*, 49 N.H. 399 (1869)). In other words, legal insanity hinged, not on one's ability to discern right from wrong, but whether the individual's actions stemmed directly from some pathology. This judgment represented a vast departure from hundreds of years of jurisprudential thinking. In Washington D.C. in 1954 in a case called *Durham*, New Hampshire's progressive stance on pathology was relied upon in the formulation of the so-called Durham Rule, or product test (*Durham v. United States*, 214 F.2d 862 (D.C. Cir. 1954).

Defences in the United States related to mental defect have morphed and continuously changed. There are four tests of legal insanity: 1) the M'Naghten test, 2) the irresistible-impulse test, 3) the Durham or product rule, and 4) the newest on the scene, Model Penal Code (MPC) test, viewed broadly as a compromise among the other three tests. Yet, despite multiple attempts to change the M'Naghten rule, this "right and wrong" test continues to endure, and remains the most widely accepted test of responsibility. *Durham* was overturned on the basis that the test removed decision-making capacity from the jury and put it into the hands of paid experts (*U.S. v. Brawner*, 471 F.2d 969 (1972). The Model Penal Code was issued as a guide by the American Law Institute and defines insanity as whether "at the time of such conduct as a result of mental disease or defect he lacks substantial capacity either to appreciate the criminality of his conduct or to conform his conduct to the requirements of the law." Thus, the MPC, which has been picking up steam, considers the age-old "right and wrong" concept, the "disease or defect" criterion of *M'Naghten*, and the irresistible impulse concept altogether. The majority of American jurisdictions cling *to M'Naghten*; Massachusetts, where the Garbedian case occurred, follows the MPC (Commonwealth vs. James N. McHoul, 1967).

Diminished Responsibility, a Partial Defence

History of "Excuse" Defences to Homicide

The defence of diminished responsibility, a partial defence to murder that requires the defendant to have acted with a particular state of mind, differs from the defence of insanity, a complete affirmative defence. A successful insanity defence, in most jurisdictions, manifests as an acquittal on the grounds of insanity. This defence historically emerged from Scottish common law for purposes of, rather than preventing conviction, reducing the punishment due to temporary or partial insanity (Kinloch, 1795, p. 94). The diminished responsibility concept emerged from a habit of 19th-century Scottish juries of returning a verdict of guilty with an accompanying recommendation of a downgraded sentence. Juries had contemplated the defendant's particular circumstances and presentation that mitigated sentencing while not fully absolving the person of responsibility. The doctrine of diminished responsibility, therefore, grew from a body of common law that stated that certain presentations of mental limitations could have the effect of reducing the sentence for one culpable for the homicide. The case of Sir Archibald Gordon Kinloch of 1795 is one of the earliest examples of the Scottish diminished responsibility concept. Sir Archibald Kinloch killed his brother, and was thought to be temporarily insane due to mistrust in the accounts of his father's will (believing he was owed £200) and thinking that his brother, the victim, had tried to poison him the day before the murder. Kinloch was found guilty, but his death sentence was reduced to life in

prison. He was soon released into the care of a physician (Kinloch, 1795).

Centuries later, Scottish courts placed limitations on diminished responsibility. The Scottish High Court in *HM Advocate v Savage* (1923) instructed the jury as to specific criteria as follows:

> It is very difficult to put it in a phrase, but it has been put in this way: that there must be aberration or weakness of mind; that there must be some form of mental unsoundness; that there must be a state of mind which is bordering on, though not amounting to, insanity; that there must be a mind so affected that responsibility is diminished from full responsibility to partial responsibility. In other words, the prisoner in question must be only partially accountable for his actions. And I think one can see running through the cases that there is implied ... that there must be some form of mental disease. (HM Advocate v Savage, 1923, p. 51)

Diminished responsibility was further elucidated and broadened in recent cases. In *Galbraith v HM Advocate,* the criteria of *Savage* were thought to be aggregate, but the defendant need not possess them all. There must be some semblance of mental defect not necessarily bordering on insanity that undermined the defendant's capacity to either make determinations regarding or control his behaviour, or else substantially impaired this capacity. Excluded from consideration were defendants who had consumed drugs or alcohol, and psychopathic personalities (*Galbraith v HM Advocate*, 2002; Hodelet & Darjee, 2005*).* This particular defence gained wide acceptance in U.S. but varies considerably across jurisdictions.

Akin to diminished responsibility is the concept of diminished capacity. While these two concepts are often used interchangeably, there are, nonetheless, subtle distinctions. Diminished capacity focuses wholly on the *mens rea* element and asserts that, although he is not legally insane, the defendant was unable to formulate the requisite intent for the crime charged; however, he may have met the *mens rea* element of a lesser charge, such as manslaughter (Arnella, 1977).

Partial Defences in the Commonwealth of Massachusetts

While the Commonwealth of Massachusetts explicitly rejects diminished capacity as a specific defence as elucidated in *Commonwealth v. Parker* (1995), the defence is *implicitly* recognized and accepted. Defendants of a murder charge in Massachusetts may produce expert testimony on the issue of whether defendant's mental processes precluded the defendant from being able to deliberately premeditate (*Commonwealth v. Gould*, 1980), and whether the defendant acted with extreme atrocity or cruelty (*Commonwealth v. Baldwin*, 1997). Moreover, the evidence presented with regard to defendant's mental capacity is relevant to the question of whether the crime of murder was committed at all (*Commonwealth v. Grey*, 1987), including issues of intent and knowledge (*Commonwealth v. Sires*, 1992). In essence, the defence of diminished capacity is available not to fully exculpate the defendant, but to reduce the charge of first-degree murder. In the Garabedian case, the defendant, in order to be guilty, must have committed the crime of murder with premeditation, malice, aforethought, and extreme atrocity or cruelty (Commonwealth v. Garabedian, 1988). If not, the *mens rea* element is not established and the defendant cannot be said to be guilty of first-degree murder. He may, instead, be said to be guilty of a lesser charge, such as second-degree murder or manslaughter.

The diminished capacity defence is predicated on the notion that the mentally defective defendant could not have formed the necessary intent to commit the crime. In contrast to the diminished capacity defence, the diminished responsibility test is not a *mens rea* defence. Rather, the jury includes the defendant's alleged mental defect in the culpability equation. The defendant may be said to be less blameworthy, and this is reflected in sentencing. While the two defences are clearly distinct, their definitions are typically blurred in court proceedings (Brakel & Brooks, 2001). The defendant, David Garabedian, may also have been able to assert a diminished responsibility defence in this Massachusetts jurisdiction (*Commonwealth vs. James N. McHoul,* 1967). The defence of "not guilty by reason of lack of criminal responsibility," which is essentially an insanity defence predicated on defective mental capacity, is available in Massachusetts (*Commonwealth v. Plant,* 1994).

Commonwealth v. Garabedian

The Trial

At trial, evidence and testimony painted a picture of Garabedian as a clean-cut, hard-working young Armenian immigrant who other Old Fox customers had characterized as a "polite gentleman." And yet, when reprimanded by Mrs. Muldoon, a bizarre and inexplicable scenario had unfolded. Garabedian suddenly tussled with her from atop a garden ledge, choked her, the pair of them toppling to the ground as he then reached for a jacket string with which to strangle her just before lobbing three massive stones and rendering her disfigured and dead. The prosecution produced two key witnesses, psychiatrists who addressed the "mental disease or defect," or *M'Naghten* component of the MPC test as stated in *McHoul*. The physicians rendered opinions as to Garabedian's psychological condition at the time of the murder. These witnesses established their opinions through a forensic examination and evaluation

of the defendant, a review of his medical and psychological records, and interviews with the employees of Old Fox Lawn Care and other witnesses in the case. Meanwhile, the defence offered testimony of two experts, Dr. Peter S. Spencer, a neurotoxicologist and founder of the Centre for Research on Occupational and Environmental Toxicology, and Dr. David M. Bear, a psychiatrist and expert in the neuropsychiatry of aggression. They testified that exposure to organophosphate compounds, or acetylcholinesterase inhibitors, might lead Garabedian to select aggressive responding upon minimal provocation. The jury was instructed as to the defendant's defence of involuntary chemical intoxication and its bearing on the issues of extreme atrocity or cruelty and deliberate premeditation. However, the jury did not receive an instruction on voluntary manslaughter as a lesser included offense. Garabedian's defence failed, and he, at age twenty-one, was sentenced to life in prison without possibility of parole (*Commonwealth v Garabedian*, 1988).

Conviction on Appeal

Garabedian's conviction of first-degree murder predicated on "deliberately premeditated malice aforethought and extreme atrocity and cruelty" was upheld. Judges relied upon facts in the case that Garabedian used progressively more force in the encounter in reaching their conclusions that the *mens rea* element of the crime was met. While Garabedian asserted a defence of diminished responsibility according to McHoul, judges reasoned that the defendant's actions were deliberate and not impulsive or spontaneous. Furthermore, the judges ruled that Garabedian was not entitled to a jury instruction of involuntary manslaughter due to the involuntary intoxication; the jury had considered said intoxication in deliberating requisite *mens rea* and rejected it (Commonwealth v Garabedian, 1988).

The Dissent

Judges Liacos and O'Connor indicated in their opinions that something was clearly amiss in Garabedian's conviction. How could a twenty-one-year-old man with no prior criminal record or violent propensity suddenly become so enraged? They recognized that Garabedian arrived to inspect Muldoon's lawn, without a weapon and without "a vengeful purpose" (Commonwealth v Garabedian, 1988). The events that followed could only be described, in their opinion, as "senseless conduct." Indeed, the "thrust of the evidence" indicated "spontaneity rather than premeditation," in that although "there is an 'irreducible doubt in all the circumstances whether the defendant consciously formed a purpose ... to do [the victim] mortal injury; if the defendant did, it is still probable that the resolve lasted for only 'a fleeting period of time.' The dissenting judges cited case after case in which the appeals court had exercised its discretion because "justice required the entry of a verdict of a lesser degree of guilt." And in a solemn footnote, the dissenters noted that

> although, as the court implicitly acknowledges, there was substantial evidence that the defendant's conduct was explainable in no other way than that involuntary chemical intoxication caused the defendant to behave in an abberrational manner, I need not rely on this aspect of the case to reach the conclusion that the defendant should have the verdict of guilt reduced to murder in the second degree" (Commonwealth v Garabedian, 1988).

Application of Defences in Light of Current Understanding of Acetylcholinesterase Inhibitor

By analysing the history of the insanity defence and its offshoots, the partial defences, we note that the common law historically reflects the view of psychology deemed most accurate by advancing knowledge. The Romans' view of insanity was quite simplistic. English common law evolved from a notion of insanity as child- or beast-like to one of a "disease" state or defectiveness. Eventually, certain clinical hallmarks of psychopathology, such as delusions or interruptions in reality contact, were deemed worthy of exculpability. And most recently, impulse control, housed in the frontal lobes of the human brain, has been the focus of consideration. In essence, the concept of *mens rea* at any given time reflects our level of understanding of brain compromise.

At the time of Garabedian's case, the concept of volition being subsumed by the inner workings of the human brain at the neurobiological level was relatively new. But, increasingly, scientists have been able to identify the links between neurophysiology and violence. Acetylcholinesterase is part of an enzymatic cascade, a messaging system arranged in an ever-increasing succession of stages. The shut-off valve is the acetylcholinesterase inhibitor. Without the shut-off valve, the cascade of enzymatic messengers just keeps flowing. Parts of the body dependent upon the shut-off become "stuck" in the on position. Sarin, the nerve agent used in bio-warfare, leaves the diaphragm left in the "on" position because sarin, an acetylcholinesterase inhibitor, inhibits the breakdown of the acetylcholine. Victims cannot draw breath and therefore suffocate. Acetylcholine, we now understand, plays a critical role in the amygdala, the seat of aggression and various drives in human behaviour. Exposure to the acetylcholinesterase inhibitor leaves portions of this brain region stuck in the "on" position. Once an aggressive response is mustered and triggers the acetylcholine enzymatic cascade, the aggressive response cannot be modulated because the inhibiting enzyme stops the breakdown of the acetylcholine enzyme. Clearly, the court in

Garabedian did not definitively dispose of the issue of Ach neurotoxicity affecting one's ability to formulate a requisite intent or conform one's behaviour to the law. The argument that Garabedian used progressively more force in the encounter does not discount the involuntary neurotoxic exposure argument. The crucial issue is whether or not Garabedian may be said to be less blameworthy given the particular circumstances. This is true because otherwise blame might be assigned to one who is not necessarily deserving of retribution.

In terms of an insanity defence, the defence that fits the case most appropriately is the Irresistible Impulse Test. The facts and circumstances fail to completely align with *M'Naghten* because, regardless of whether the neurotoxic damage could be characterized as a "disease or defect," David Garabedian most certainly knew that what he was doing was wrong. Garabedian possessed the mental capacity immediately preceding the violent episode to check to make sure no one was home before carefully walking around to the back, and he took measures to shield detection of his urinating on Mrs. Muldoon's lawn. In other words, in the moments immediately preceding the murder, he commanded planning and judgment capacity adequate to conform his behaviour to socially acceptable behaviour and to recognize when his behaviour would be adjudged to fall short of these expectations. However, once Mrs. Muldoon became aware that he was urinating on her lawn and she was offended, the situation very quickly escalated. Garabedian's emotional and aggression centre, the amygdala, was aroused and the neurotoxin on-board hindered the shut-off valve. In essence, neuroscientists would explain, given our understanding of the neurobiology of aggression and the role that this neurotoxin plays, Garabedian could assert a defence by excuse and not be held criminally liable because he could not control those actions even if he knew them to be wrong. And for this reason, Irresistible Impulse is where a defence in the context of involuntary neurotoxic damage nestles most comfortably.

The Durham, or Product Rule somewhat fits the involuntary neurotoxic damage defence scenario, especially in "clean cases like Garabedian's, where the neurotoxin was the only exogenous substance involved and the defendant's background was otherwise devoid of criminal propensity. According to the Durham Rule, the criminal act must have been the "product" of the defendant's mental disease or defect. As the dissenting opinion highlights, there is no evidence at bar to explain the defendant's aberrant behaviour other than the involuntary neurotoxic damage preventing him from modulating his aggressive response. In other words, the violent rage exhibited by Garabedian was a product of his inability to shut off the cascading enzymes that control aggressive responding.

The Model Penal Code test, which is a combination of *M'Naghten* and the Irresistible Impulse test and is available in Massachusetts, is problematic from a defence perspective primarily because of the *M'Naghten* component. The defence will need to produce testimony as to multiple criteria of the rule and certainly at the outset establish by expert testimony whether the nature and degree of the disability incurred by the neurotoxin are sufficient to establish a mental disease or defect, as *M'Naghten* progeny have defined those terms. Additional MPC criteria are that the individual lacked the capacity to appreciate the wrongfulness of the act or to conform to the requirement of the law.

Considering that scientific evidence shows exposure to acetylcholinesterase inhibitor causes violent behaviour, Garabedian may have been able to assert a diminished capacity defence. This defence is controversial. Some jurisdictions have forcibly opposed such defences (Brakel, S.J. & Brooks, A.D. (2001). Indeed, courts who have rejected the diminished capacity defences have reasoned that the "unrestrained application of the diminished capacity doctrine would have a profound impact upon both the separate defence of insanity and the statutory scheme which governs claims of irresponsibility" (Bethea v. United States, 1976). Moreover, the well-being of those in the community could be jeopardized by the application of psychiatric evidence to the threshold of intent (Brakel & Brooks 2001). Other courts have destroyed the defence by characterizing psychiatry as "esoteric and largely unproved," and deemed the admission of psychiatric evidence as "intolerable" in *mens rea* (Brakel & Brooks 2001).

As discussed earlier, while the diminished capacity defence is directed toward the *mens rea* or requisite intent component of the conviction phase, diminished responsibility exerts its impact typically upon the sentence that the defendant receives. The two are closely associated; this close association is between, on one hand, liability to blame or punishment for one's actions, and, on the other hand, the capacity to conform conduct to the law (Sparks, 1964). With newfound knowledge as to the mechanism of neuromodulation of aggression, Garabedian may have been able to successfully assert a diminished responsibility defence, as was alluded to in the strong dissenting opinion. The dissenters repeatedly refer to their "33E" power, which states as follows:

> Upon such consideration the court may, if satisfied that the verdict was against the law or the weight of the evidence, or because of newly discovered evidence, or for any other reason that justice may require (a) order a new trial or (b) direct the entry of a verdict of a lesser degree of guilt, and remand the case to the superior court for the imposition of sentence (Mass. Gen. Laws Ch. 278 § 33E).

The dissenting opinion recites multiple cases in which 33E was invoked and the jury verdict overturned due to

circumstances such as the age of the victim, exemplary community involvement and prior conduct, and no history of violence. In essence, the *mens rea* and *actus reus* components may have been technically met, but the defendant was deemed nonetheless only partially blameworthy.

As for the medico-legal aspects of this case venturing further away from psychiatry and decidedly toward neuroscience, justice demands that our age-old understanding of legal insanity be re-examined. Historically, legal insanity has been likened to a type of psychosis and criteria have included a recognized, diagnosable "disease or defect" component. Later instantiations have added behavioural components to the cognitive "right and wrong test." The pithiest questions that the criminal justice system must answer in a case like Garabedian's in order that justice be served and fairness of punishment be determined is whether the defendant possessed some neuropsychological abnormality in some identifiable clinical sense during the commission of the crime and, if so, whether, in that mental state, he could stop himself from ommitting the crime.

Contemporary Cases

While presentation of neuroscientific evidence has grown steadily over the years and recently skyrocketed (Farahany, 2016), involuntary neurotoxic damage defences based on prolonged exposure to acetylcholinesterase inhibitors alone have typically failed. This is partly because the defendant's case is usually confounded by additional voluntary exposure to other substances, including drugs and alcohol. For example, just a few years after *Garabedian*, the defendant in *Sette* was charged with a stabbing death and attempted murder. Noting the *Garabedian* case and a New Jersey statute, at trial he asserted the affirmative defences of involuntary and pathological intoxication (State v. Sette, 1992).

Sette claimed that he was *prevented from knowing the nature and quality of his conduct* by a combination of cocaine, marijuana and Co-Tylenol, which he voluntarily ingested, *and* by a build-up of toxic pesticides in his body acquired during several years as a landscape worker. Sette, like Garabedian, had been employed in the gardening and landscaping business, where he was routinely exposed to pesticides, herbicides, and chemicals including the bug killer Sevon. The levels and concentration of his exposure to these chemicals was disputed. The State maintained that the crimes were motivated by revenge because of sexual rejection and noted that, in addition to his involuntary exposure to pesticides, he often used cocaine on weekends (State v. Sette, 1992).

Sette was convicted, and on appeal, the court stipulated that a defendant charged with murder and attempted murder could show that he did not understand the nature and quality of his criminal acts. Essentially, the court applied the M'Naghten standard. Under the M'Naghten standard of irresponsibility, the court reasoned that a defendant would have no criminal responsibility only if that condition was induced solely by his involuntary occupational pesticide exposure which defendant had no reason to anticipate would induce a state of insanity. The court further reasoned that intoxication from drug use has the same legal consequences as intoxication from alcohol. Voluntary drug use which prevents the formation of purposeful or knowing conduct negates the crime of murder, but not the crime of reckless manslaughter (State v. Sette, 1992).

The court concluded that the voluntary ingestion of drugs constituted a confounding variable in Sette's pathological intoxication defence. The court adamantly maintained that it could not allow consideration of a pathological intoxication defence when intoxication resulted from a combination of voluntary ingestion of illegal intoxicants, or of legal intoxicants from which a person should reasonably expect an adverse reaction, or both, and a pathological intoxication due to involuntary exposure to pesticides.

In order for defendant charged with murder and attempted murder to have his behaviour excused altogether through the defence of pathological intoxication, the jury would have to find that his intoxication resulted solely from his exposure to organophosphate in pesticides, independent of ingestion of illegal drugs. However, the judge formally instructed the jury that voluntary intoxication was relevant to disputing the purposeful and knowing mental state required for murder (State v. Sette, 1992).

In Sette's case, the court recognized that "the only real evidence ... of either involuntary or pathological herbicide intoxication demonstrated that any pathological frailty which afflicted defendant was insufficient by itself to inhibit his mental faculties but could only have prevented appreciation of his acts in combination with his voluntary ingestion of the illegal drugs, cocaine and marijuana, and the overdose of the legal drug, Co-Tylenol" (State v. Sette, 1992).

Thus, the court recognized involuntary exposure to organophosphates as a cognizable defence to the crime for murder given a different set of facts. The court in dicta stated that: "[i]f the defendant's system had been slowly poisoned by pesticides until he succumbed to a psychotic state under the intoxicating effects of organophosphate toxin alone, defendant could soundly rely on the pathological [or involuntary] intoxication defence" (State v. Sette, 1992). Such a clean case as Garabedian's toxic exposure remains elusive.

Conclusion

Understanding the precise effect of acetylcholinesterase inhibitors and other neurotoxic exposures upon behaviour has crucial societal and legal implications. The fact that we punish more severely for intentional acts than negligent acts stems from the notion that individuals are rational thinkers; generally speaking, we are all motivated by a system of socially acceptable beliefs. Adhering to this notion, we assign blame to those whose acts are motivated by beliefs not within the realm of acceptability (Lelling, 1993). In essence, assignments of blame are grounded in at least some minimal understanding of the science of the human mind. The foundation of society's comprehension of cognition and behaviour is empirical science. Some legal theorists have asked what would happen if the rapid progress of modern cognitive science were to "veer away" from the psychological theory upon which we base our morality and our law. If great leaps in scientific research are connected in "chain-link fashion to shifts in morality and then legality, is society willing to go wherever that research leads us?" (Lelling, 1993).

Continual modifications in neuroscientific theory draw scientists progressively further away from traditional psychological assumptions. The broadening neuroscientific perspective is forcing us to redefine psychology in neurophysiological terms, thus altering the traditional view of cognition and the social constructions that depend on it, such as law. At the time of Muldoon's murder, traditional neuropsychological theories were inadequate and biological psychiatry was in its infancy. However, as a result of heightened scrutiny, the molecular mechanisms underlying the neurobiological effects of pesticide exposure are much more clearly understood.

This heightened scientific recognition, coupled with increasing numbers of latent neurological manifestations, may result in an increasing number of neurotoxic claims. The notion of criminal responsibility represents a continuum. The realms of insanity and sanity are not sharply demarcated in either the scientific or legal domains. Despite the troubling questions raised by the neurotoxic damage defence, it may be a legitimate shield to maximum sentencing. As the defence is raised more frequently, the criminal justice system will be forced to respond.

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Appendix

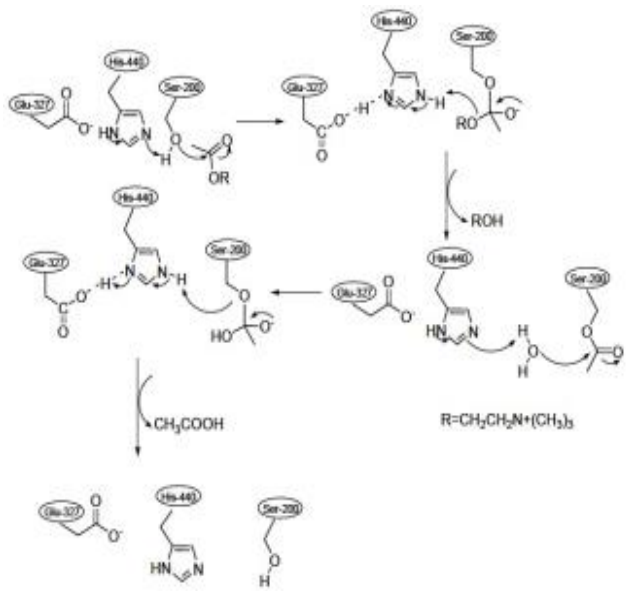

Figure 1. Hydrolysis of acetylcholine with acetylcholinesterase Image courtesy of Alinebloom under CC BY-SA 4.0 (Acetylcholinesterase Inhibitor)

Figure 2. James Hadfield Image courtesy of Etzagots under CC BY-SA 4.0 (James Hadfield)

Figure 3. Hadfield at Drury Lane Theater
Image courtesy of V & A Museum under CC BY-SA 3.0

Figure 4. Daniel M'Naughten
Image courtesy of the Wellcome Library under CC BY 4.0 (McNaughten)

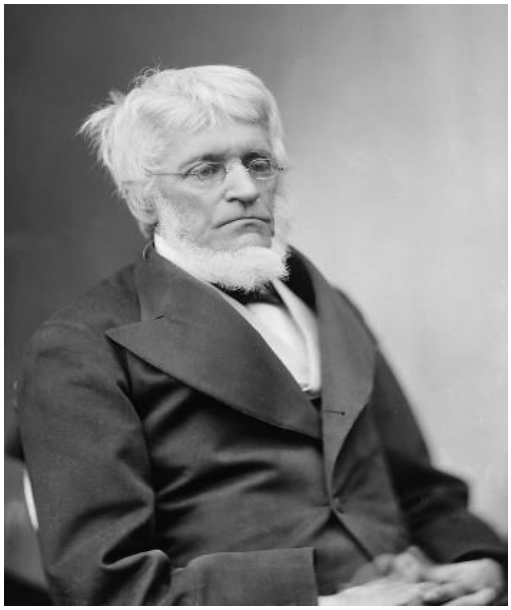

Figure 5. Forensic Psychiatrist Isaac Ray
Image courtesy of the United States Library of Congress under CC BY-SA 3.0 (Issac Ray)