"That Accident Really Set Off My MS!"

Does Trauma Cause or Worsen Multiple Sclerosis?

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Ms. Higgins, a middle-aged woman, fell as she exited an elevator which allegedly failed to properly level as it reached the appropriate floor. She did not sustain any cuts or abrasions but asserted that a dormant multiple sclerosis condition was aggravated, and within days of the event, symptoms appeared which progressed to where she could no longer walk without the assistance of a walker.³ Suit was filed against the elevator company and a battle among the experts ensued as to whether trauma can cause or aggravate multiple sclerosis, a scenario that has become all too familiar in courts around the country.⁴

I. Introduction

Multiple sclerosis (MS) is a debilitating neurological process⁵ in which an abnormal response of the body’s immune system is directed against the central nervous system.⁶ As noted in Hunt v. Secretary of Health and Human Services,⁷ the cause of the condition remains unknown but it is believed to be an autoimmune disease in which a

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⁴ See id.

⁵ See Multiple Sclerosis, LA JOLLA INST. FOR IMMUNOLOGY, https://www.lji.org/faculty-research/research-areas/diseases/multiple-sclerosis?gclid=Cj0KCQjwwb3rBRDrARIsALR3XcZyzaC9-mznXvQ1SUJG18_s22CxC8Vg52P1LgFVDPeLYt2IGQ5DoAaAl--EALw_wcB (last visited Oct. 27, 2019).


person’s immune system attacks its own tissues. Therefore, it is little wonder that the question as to whether trauma can trigger or exacerbate MS is so hotly debated in both medical and legal circles.

A number of theories exist as to the cause of MS, but none have been definitively proven. Likewise, a number of risk factors such as distance from the equator, smoking, vitamin D levels, and viral infections have been implicated in the development of the disease. Added to this mix, is the possibility that trauma, especially to the head, could be a predisposing factor for developing or worsening MS. Since the disease is one of the most common causes of non-traumatic disability among young adults and traumatic brain injury (TBI) is one of the leading causes of mortality and morbidity in children and young adults, correlative evidence tying these two conditions together could either be a boon or headache for the legal profession, depending upon counsel’s view. This article will explore both the medical and legal considerations of such a possible connection.

II. Medical Discussion

A. Epidemiology

In the United States, MS affects approximately 400,000 people, with the disease being three times more prevalent in women than men. Symptoms usually first occur when the person is in their twenties and can first appear up to fifty years of age. Among ethnic races, MS is more prevalent in Caucasians than in African American or Hispanic groups. The disease prevalence increases the greater the distance from the equator in either hemisphere. Thus, in the United States, MS is more prevalent in Minnesota than it is in Florida.

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11 See Hersh & Fox, supra note 9.
13 See Piyameth Dilokthornsakul et al., Multiple Sclerosis Prevalence in the United States Commercially Insured Population, 86 NEUROLOGY 1014, 1014-21 (2016) (providing background information on population with Multiple Sclerosis).
15 See id.
16 See Mateen, supra note 10, at 177.
17 See id. (noting Multiple Sclerosis is considered more prevalent in higher-income countries).
B. Pathophysiology

Some basic neuroanatomy is required to understand the mechanism of the disease. At the microscopic level, the main functional cell type of the nervous system is a neuron, which works as a microprocessor. The role of a neuron is to receive, generate, and transmit information within the vast network known as the nervous system. Input to the neuron is collected on cellular processes called dendrites and on the neuron cell body, which also houses the metabolic machinery of the neuron. The output from the neuron is by a single long process called an axon. The axon connects to other neurons via a specialized contact termed a synapse. In this way, neurons communicate with each other. Many of the axons in the nervous system are individually wrapped with an insulating material called myelin, which is produced by a specialized cell type of glial cell, called an oligodendroglia. These wrapped axons are called myelinated axons. The purpose of the myelin is to increase the conduction velocity along the axon, allowing greater speed of communication. Within the brain and spinal cord, collectively termed the central nervous system, to the naked eye the masses of myelinated axons bundled together appear white, and consequently these regions are called white matter. Conversely, in regions where there is little myelin but a higher concentration of dendrites, cell bodies and unmyelinated axons, the area appears gray; hence, the term gray matter.

With MS, the body's immune defenses go haywire and attack the myelin covering of the axons in an inflammatory form of "friendly fire." This results in MS categorized as an autoimmune inflammatory disease resulting in demyelination (damage to the myelin) within the central nervous system.

The reason for the inflammation is not known, but some type of autoimmune response against an antigen in the central nervous system is suspected. Because the bundled groups of axons in the white matter cannot function properly due to the myelin damage, the result is neurological symptoms such as numbness, imbalance, weakness, visual loss, or speech difficulties. The damage can also cause brain atrophy, cognitive impairment, limited mobility, and shortened life expectancy. The types of symptoms resulting from this inflammatory attack depend upon what part of the brain or spinal cord is affected. For example, if the visual system, such as the optic nerve, is attacked, the person experiences visual dimming or loss. If the motor pathways are targeted, the person experiences weakness or paralysis to that part of the body controlled by that pathway.

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19 See Jack E. Hubbard & Samuel D. Hodge, Jr., Head Trauma and Brain Injury for Lawyers 21-65 (Am. Bar Ass’n Book Publishing 2016).
20 See Amos v. Biogen IDEC, Inc., 249 F. Supp. 3d 690, 693 (W.D.N.Y. 2017). MS is an "autoimmune disease in which white blood cells enter the central nervous system and attack myelin, a fatty substance that surrounds nerve cells and assists in the transmission of signals to and from the brain." Id. “MS gradually destroys myelin (a process known as ‘demyelination’), resulting in nerve damage throughout the brain and spinal cord.” Id.
21 See Hersh & Fox, supra note 9.
22 See id. (suggesting the role of certain lifestyle factors in this process).
23 See Amos, 249 F. Supp. 3d at 693.
C. Types of MS

It is necessary to understand the types of MS and their associated clinical course to understand the possible impact of trauma upon a person with the malady. As noted in Zurndorfer v. Unum Life Insurance Co. of America, MS manifests itself in many different ways with four main types of MS being recognized: 1) relapsing-remitting MS (RRMS), 2) secondary progressive MS (SPMS), 3) primary progressive MS (PPMS), and progressive relapsing MS (PRMS).24

The relapsing-remitting MS (RRMS) type is the most common form25 that is present in 85% of patients with the disease.26 In RRMS, the person experiences a neurological worsening such as balance problems, visual loss, or numbness, that lasts anywhere from a few days to weeks (relapse) then improves (remit).27 During the relapse phase, acute inflammation within the central nervous system occurs resulting in myelin damage, and with remission, the damage is repaired by the body’s own mechanisms. In some cases, the remission may be complete with no neurological sequelae, but often the recovery is incomplete with minimal to moderate residual symptoms. On an average, individuals with RRMS will experience a relapse once every one to two years.28 Neurological symptoms associated with a relapse usually develop over hours to days, then worsen over several weeks, and subside during remission, either completely or incompletely, over several weeks to a month or two.29

The secondary progressive MS (SPMS) form represents a worsening progression of the RRMS type.30 In this type, a person with RRMS progressively worsens following the relapsing-remitting phase.31 Within ten years of diagnosis, this progression occurs in about 50% of patients with RRMS.32 Although poorly understood,33 SPMS is thought to

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25 See W.C. v. Sec’y of HHS, 100 Fed. Cl. 440 (2011), aff’d,704 F.3d 1352 (Fed. Cir. 2013) (“The most common type of multiple sclerosis . . . is known as relapsing remitting multiple sclerosis. People with relapsing remitting multiple sclerosis usually have approximately one relapse of multiple sclerosis per year. Like the cause of the onset of multiple sclerosis, the cause of relapses of multiple sclerosis is not known”). In W.C., a National Vaccine Injury Compensation Program petitioner alleged that his 2004 influenza vaccine caused his MS or, in the alternative significantly aggravated his pre-existing, asymptomatic MS. Id. at 443. The petitioner was denied compensation. Id. at 461.
26 Brian D. Weinshenker, Natural History of Multiple Sclerosis, 36 ANN. NEUROLOGY, S6, S7 (1994).
27 Id. (noting that the majority of patients who start with RRMS develop progressive MS).
28 See Mateen, supra note 10, at 177.
29 See id. (stating physical activity is commonly considered to combat disability in MS).
30 See Krieger & Solomon, supra note 24, at S8.
31 See SGS-92 -X003 v. United States, 118 Fed. Cl. 492, 510 (2014). As one medical expert explained, “when people begin to worsen between attacks without an ability to say they had some worsening with subsequent improvement, if they’re worsening between attacks for at least six months, this is evidence of secondary progressive multiple sclerosis.”
represent progressive damage not only to just the myelin, but also irreversibly to the neuronal axons themselves, leading to neuronal loss and gradual disability. This progressive form usually develops about ten to fifteen years after onset of the RRMS type.

Primary progressive MS (PPMS) has no relapsing-remitting phase but progressively worsens to disability. Occurring in 10-15% of MS cases, those with PPMS tend to be older and respond poorly to treatments for the condition.

Progressive relapsing MS (PRMS) also leads to disability, but is characterized by multiple relapses throughout the course of the disease; full recovery may or may not result after each relapse.

Therefore, the course and prognosis of an individual with MS is highly variable and individual. Within the scope of the disease, affected individuals can have just several mild relapses, and then remain neurologically stable for the rest of their life, or they may rapidly progress to complete disability over several years’ time.

D. Diagnosis

Multiple sclerosis was first described more than 150 years ago and named for the many areas of sclerosis, or scarring, seen on pathological examination of the brain and spinal cord. Given the length of time that has passed since MS was initially recognized as its own disease, one would assume that specific scientific advancements such as laboratory or imaging findings would have been developed by now to effectively diagnose the disease. However, as of this writing, no clinical, laboratory, or imaging findings are specific for MS. As stated by Hersh and Fox, “the diagnosis ultimately is a clinical decision based on weighing the factors that support the diagnosis against those that fail to support it or point to the possibility of an alternative diagnosis.” The diagnosis of MS, then, requires consideration of a person’s clinical course (e.g. a...

33 See E. Ann Yeh & Robert J. Fox, Demyelinating Lesions and Progressive MS, 93 NEUROLOGY 283, 283-84 (Aug. 2019) (discussing MS disease progression and clinical ramifications of associating disease progression with tissue injury or spinal cord lesions).
34 Id.
35 Id.
36 See Krieger & Solomon, supra note 24, at S8.
39 See Krieger & Solomon, supra note 24, at S8.
40 See Cottrell, supra note 38, at 638.
41 See id. at 637.
42 See Helmar C. Lehmann et al., 150th Anniversary of Clinical Description of Multiple Sclerosis: Leopold Ordenstein’s Legacy, 90 NEUROLOGY 22 (May 2018) (examining the first medical thesis defining the clinical features of multiple sclerosis).
43 See Hersh & Fox, supra note 9.
44 Id.
relapsing-remitting pattern), MRI (magnetic resonance imaging) scan findings of characteristic white matter lesions, and results from a cerebrospinal fluid analysis for an abnormal protein termed oligoclonal bands. These pieces of the puzzle have been codified into the gold standard for MS diagnosis known as the *McDonald criteria*. First introduced in 2001, the McDonald criteria have undergone a number of revisions, with the latest one published in 2017.

Critical to the implementation of the *McDonald criteria* is the demonstration of dissemination of lesions in time (DIT) and space (DIS). That is, a person diagnosed with MS needs to have had at least two neurological events either identified clinically or by the MRI scan separated by time (DIT) and/or two or more lesions in different locations (DIS). A third critical element is the exclusion of other alternative diagnoses which could explain the person’s symptoms.

Strict adherence to these criteria is critical not only for the diagnosis of MS but also to prevent misdiagnosis. Studies have shown that 30–67% of patients referred to MS subspecialty centers were found not to have MS, but rather another condition. Common problems misdiagnosed as MS are migraines, fibromyalgia, vascular events, and functional (psychological) causes. The main reason for this misdiagnosis is inappropriate or faulty use of the *McDonald criteria* by treating physicians, especially on the over-reliance of MRI abnormalities with nonspecific white matter lesions.

Counsel must remember this point when handling a multiple sclerosis case.

E. Treatment

The court in *Cury v. Colonial Life Insurance Company of America* pointed out the unfortunate problem with MS by stating: “There is no cure for multiple sclerosis, and the disease usually follows a slow, progressive course marked by a history of exacerbations and remissions.” Pharmacological management strategies are directed to both treating acute relapses and decreasing disease activity and disability progression.

An acute relapse is due to an inflammatory reaction, so corticosteroids have been found to be effective in decreasing the relapse duration as well as long term

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45 See W. Ian McDonald et al., *Recommended Diagnostic Criteria for Multiple Sclerosis: Guidelines from The International Panel on The Diagnosis of Multiple Sclerosis*, 50 ANNALS NEUROLOGY 121, 121-27 (2001) (stating new diagnostic criteria for MS).

46 See Alan J. Thompson et al., *Diagnosis of Multiple Sclerosis: 2017 Revisions of the McDonald Criteria*, 17 LANCET NEUROLOGY 162, 173 (2018) (updating the McDonald criteria for diagnosing MS).

47 See Krieger & Solomon, supra note 24.

48 See Andrew J. Solomon et al., *Misdiagnosis of Multiple Sclerosis; Impact of The 2017 McDonald Criteria On Clinical Practice*, 92 NEUROLOGY 26, 26-33 (2019) (arguing that atypical clinical presentations require further evaluation and monitoring before immediate MS diagnosis).


50 See Andrew J. Solomon et al., *The Contemporary Spectrum of Multiple Sclerosis Misdiagnosis: A Multicenter Study*, 87 NEUROLOGY 1393, 1393-99 (2016) (finding misdiagnosis of MS leads to unnecessary and harmful risks to patients).


52 See Hersh & Fox, supra note 9.
A typical approach is to give intravenous high dose (1,000 mg) of methylprednisolone daily for three to five days followed by a gradual taper.\textsuperscript{54} A proactive way to prevent relapses has been developed through the use of disease-modifying therapies (DMT) for relapsing MS.\textsuperscript{55} While not a cure for the disease, these therapies have been demonstrated to reduce the relapse rate and slow the time to onset of disability. As stated by Hersh and Fox, “they target the immune dysfunction in MS and resultant neural tissue damage with the goal of preventing or at least reducing the long-term risk of clinically significant disability.”\textsuperscript{56} A number of disease-modifying therapies have been developed, working by different mechanisms and administered in different ways including oral pills, injectable forms, and infusion therapies. As remarkable as these treatments are, they are considered preventive but not restorative.\textsuperscript{57} That is, once neuronal damage and loss occurs, the neurological deficits remain and are irreversible. For this reason, early treatment is “key since it offers the greatest chance of preventing or delaying tissue injury and long-term disability.”\textsuperscript{58} On the other hand, this pressure to make an early diagnosis to start DMT often leads to misdiagnosis of the disease.\textsuperscript{59}

F. Does Trauma Cause or Worsen MS?

The issue at hand is whether there is credible medical evidence that trauma either precipitates MS or causes a relapse/progression. It is understandable that given the variability in expression of the disease, such a question has become controversial. For example, in 2000 an issue of Archives of Neurology featured a section titled Controversies in Neurology with the topic being trauma and multiple sclerosis.\textsuperscript{60}

As stated by Mumford, “we have to accept that neurologists are divided on this issue. Some well-known names in the field of multiple sclerosis have very strongly held, almost polarized, views.”\textsuperscript{61} Further, just the question “does trauma cause multiple sclerosis?” is considered simplistic for it must be expanded to include type and severity of trauma, pre-existing tendencies, timing of MS onset relative to the trauma, and even the nature of the cause of MS.\textsuperscript{62} These and other variables make it difficult to make apple-to-apple comparisons among the various studies.

Two types of investigations are accepted forms of medical research in attempting to find a causal link between MS and trauma; they are termed the cohort

\textsuperscript{53} See Brownlee, supra note 49, at 15.
\textsuperscript{54} See Hersh & Fox, supra note 9.
\textsuperscript{55} See id.
\textsuperscript{56} See id.
\textsuperscript{57} See id.
\textsuperscript{58} See id.
\textsuperscript{60} See Vladimir Hachinski, Trauma and Multiple Sclerosis, 57 Arch. Neurology 1078 (2000).
\textsuperscript{61} Colin Mumford, Can Trauma Provoke Multiple Sclerosis?, 2 Pract. Neurology 36, 39 (2002) (disscussing several United Kingdom cases alleging MS resulting from trauma).
\textsuperscript{62} See id. at 37.
In the cohort type of study, the goal is to prospectively show that the incidence of MS onset or relapse attacks is greater in patients following a traumatic injury than in a group of MS patients who have not experienced an injury. A limitation of this type of study is the source of the data; that is, is the injury self-reported or based upon the medical records? Also, had the individuals been screened for MS prior to the trauma?

In the case-controlled type of investigation, which is less direct, the attempt is to demonstrate that a previous traumatic injury was more common in patients with MS than in the control group. Limiting factors with this type of study include the reliance on the subject’s recall of trauma without verification by independent, factual sources. Also, since the time of MS onset is often uncertain, it is difficult to establish the claim that the trauma occurred prior to development of the disease. Even the definition of what constitutes a traumatic injury is problematic. Finally, as with the cohort type of study, what is a reasonable time between the MS onset/relapse and the trauma—three months, six months, or a year? Most researchers consider a year interval to be at the upper limit of acceptance. Within this context of uncertainty, criticisms have been hurled at some studies by others who claim that they are not statistically relevant or valid, especially studies that report negative results. From a medical-legal standpoint, a consensus opinion is necessary “since persons with MS continue to seek compensation for injuries which they feel may have caused their disease, adding to debate on this topic.”

These limitations and controversies require the question of trauma and MS be approached from a pro and a con standpoint. Those who support a causal link between trauma and MS can find support for their position dating back to when MS was first described in the late 1800s, and many have considered a traumatic injury a risk factor for the disease since then. The studies throughout the early 1900s, however, examining this relationship consisted of only a few patients in each study with poor statistical data handling. What is considered the first controlled study of the effects of trauma on MS was reported by McAlpine and Compston in 1952.
of 250 patients with MS and 250 control subjects, the investigators found that 14.4% of patients had a history of trauma within three months preceding the onset of their MS compared with only 5.2% of the control subjects, leading them to conclude that the trauma was a factor in development of MS in those patients. However, this study has been criticized for lack of details about the actual data and data analysis description.74

A significant factor in relating trauma to MS is a determination of what actually constitutes trauma and, further, how does the trauma worsen MS. In a prospective study of 170 MS patients and a control group of 134 individuals followed for over eight years, the researchers identified 1,407 instances of peripheral body trauma which was sorted into various categories including dental procedures, minor surgery, major surgery, fractures, electrical injury, sprains, burns, various abrasions/lacerations/contusions, and minor head injuries.75 Interestingly, the MS patients had two to three times more incidences of trauma than did the control subjects. Despite this greater rate of occurrence, there was no correlation between the number of these various forms of trauma and MS exacerbations and there was no correlation between the frequency of trauma and progression of disability from MS.76

Advocates of the trauma-MS correlation agree that injury to more peripheral parts of the body and minor head trauma are not factors in MS worsening. Rather, they conclude that only significant trauma to the brain and/or spinal cord (collectively termed the central nervous system) can set into motion the mechanism for the onset or worsening of MS.77 The likely scenario for this correlation is that trauma to the central nervous system damages the neural tissue by breaking down the blood-brain barrier, exposing the various damaged cellular fragments to the body’s immune system.78 The immune system then perceives these neuronal cellular fragments as foreign antigens, setting in motion the destructive immune mechanisms against the myelin and axons.

Even critics of the trauma-MS correlation state that with this scenario “…it seems reasonable to conclude that a causal relationship between trauma and either the onset or the exacerbation of MS is biologically possible.”79 In a 2014 meta-analysis of 1,362 individual studies in medical literature examining physical trauma as a risk factor for the subsequent diagnosis of MS, researchers found 36 case-control studies that met their inclusion criteria for review.80 Their conclusion was that there was a “statistically significant association between premorbid head trauma and the risk for developing MS. More specifically, those with premorbid head trauma were significantly more likely to be diagnosed with MS in comparison to those controls of similar age and sex who had not

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74 See Goodin, supra note 63, at 1740 (introducing critiques on the first controlled study).
75 William A. Sibley et al., A Prospective Study of Physical Trauma and Multiple Sclerosis, 54 J. NEUROLOGY, NEUROSURGERY, & PSYCHIATRY 584, 584-85 (1991) (tracking the health of MS patients).
76 See id. at 585.
77 Charles Poser, Trauma to the Central Nervous System May Result in Formation or Enlargement of Multiple Sclerosis Plaques, 57 ARCH. NEUROLOGY 1074, 1074-77 (2000) (arguing that only certain types of trauma can initiate symptoms of MS).
78 See id. at 1074.
79 See Goodin, supra note 63, at 1738.
sustained head trauma.” However, despite this positive finding, the authors caution that “this in no way suggests or demonstrates causality, in that epidemiological studies can only provide etiological clues at best.” Interestingly, in the same study, they also reviewed four cohort studies and found no association of statistical significance between head trauma and the subsequent diagnosis of MS.

In 2015, a large cohort study focused on the possibility of MS development after a spinal cord injury. In this investigation, 11,913 subjects with a history of spinal cord injury were compared with 59,565 matched subjects without a history of such injury and were followed over several years. The researchers found that spinal cord injured patients had a more than eight-times higher risk of developing MS compared to those without spinal cord injuries.

A 2017 Swedish study of concussion in children and adolescents with MS development reviewed the medical records of 7,292 individuals with MS and 72,920 patients without the disease, evaluating how many in the MS group had a previous concussion. They also evaluated the incidence of fractured limbs to determine if simple physical trauma was a factor as well. The researchers determined that in the childhood age group (0-10 years old), there was no correlation between either concussions or broken bones and subsequent development of MS in that group. However, in the adolescent group (11-20 years old), a single concussion resulted in a small but statistically significant risk of developing MS while two concussions more than doubled the risk of later developing MS with no risk of the group with just a limb bone fracture. In discussing the results, the researchers consider the possibility that those injured may have had undiagnosed MS, leading to difficulty with mobility, coordination and balance, making it more likely that they experienced head trauma (reverse causation). However, the lack of MS correlation with the limb fracture group tends to dispel this possibility.

Those who oppose a trauma-MS correlation point to their own statistical studies and rationale. Neither head nor spinal cord injury are listed in an extensive statistical review article on risk factors that are associated with the onset of relapsing-remitting or primary progressive MS. Another way to statistically look at this question is to compare the gender differences between the two problems. MS occurs three times

81 See id. at 22.
82 Id.
83 See id. at 21.
85 See id. at 655.
86 See id. at 657.
87 See Scott Montgomery et al., Concussion in Adolescence and Risk of Multiple Sclerosis, 82 ANNALS NEUROLOGY 554, 554-61 (emphasizing the severity of head injuries in pediatric patients).
88 See id. at 556.
89 See id. at 559.
90 See generally Kyla McKay et al., Risk Factors Associated with the Onset of Relapsing-Remitting and Primary Progressive Multiple Sclerosis: A Systematic Review, 10 BIOMED. RES. INT’L 1155, 1155-66 (2015) (arguing for further research on primary progressive MS).
more often in women than it does in men. However, overall, and in most age groups, head trauma mortality and morbidity is higher in men than in women. For example, a study on the incidence of hospital-treated traumatic brain injury in Norway, reported that the male:female ratio was almost two to one and in the 20-29 year age group, the individuals most likely to develop MS, the males were three times more likely to have head injury than females. From a strictly statistical standpoint, if trauma were a significant risk factor, the incidence and prevalence of MS should be higher in the male than the female population, which is not the case. In a Danish cohort study, 95,111 men and 55,757 women hospitalized for cerebral concussion, confusion, or skull fracture under age 55, were followed for the development of MS after the year following the head injury. The researchers found no significant increase in that population beyond what would be expected, concluding that “head injury of any severity does not affect the risk of acquiring MS later in life.”

Several in depth analytical reviews of the literature have concluded that there is no relationship between trauma and MS. For instance, in 1999, a report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology, concluded that “[o]n the basis of strong and generally consistent Class II evidence,” (provided by one or more well-designed clinical studies such as case control and cohort studies) “any posited association of trauma, especially head trauma, with more than a small effect on either MS onset or MS exacerbation is excluded. Moreover, the preponderance of the Class II evidence supports no association between physical trauma and either MS onset or MS exacerbation.” In their mixed conclusions, Lunny and colleagues found that “in the meta-analysis of cohort studies, pooled results did not support a statistical association between head trauma and the later diagnosis of MS.” In their systematic review and meta-analysis of the literature, Warren and co-workers determined that “these findings support the conclusion that there is no association between traumatic injury and multiple sclerosis onset.” However, they qualify their conclusion by stating that their study “cannot rule out such an association, largely because of limitations in the studies conducted.” Other researchers consider that any relationship between trauma and MS is explained either by coincidence or by the physical/psychological effects of trauma rather than a direct neuropathological causation.

91 See Gooch, supra note 14, at 479-84.
92 See Coronado, supra note 12, at 87.
94 See Goodin, supra note 63, at 1738.
95 See Claudia Pfleger et al., Head Injury Is Not a Risk Factor for Multiple Sclerosis: A Prospective Cohort Study, 15 MULTIPLE SCLEROSIS, 294, 294 (2009) (showing head injury does not affect risk of developing MS in cohort of 50,868 subjects).
96 See id.
97 See id.
98 See Goodin, supra note 63, at 1744.
99 See Lunny, supra note 80, at 20.
100 See Warren, supra note 64, at 168.
101 Id. at 174.
102 See id.
III. Legal Discussion

The casual connection between trauma and MS is another cog in the ever-growing list of controversies that plague the legal system especially in a compensation context. There is no clear-cut answer to the question and parties on both sides can find support for their positions. As noted in Neurology, “[t]rauma and multiple sclerosis is a topic that smolders and flares. As soon as a semblance of consensus settles upon the literature, a court case ignites the debate anew.”103

A search of the legal publications shows that very little has been written on the subject. The Attorneys Medical Advisor references MS as a condition that is thought to be connected to a traumatic injury but the journal further provides that “the connection to trauma has been postulated, but not proven.”104 As noted previously in this article, some studies assert that trauma might be a risk factor.105

Needless to say, several websites maintained by lawyers attempt to establish a link between trauma and MS. For instance, one discusses a study in which researchers determined that patients with a traumatic brain injury had a higher rate of MS during a 6-year period than the comparison group. This led the scientists to conclude that those who sustain a traumatic brain injury “are in fact at a higher risk of multiple sclerosis over a 6-year follow-up period.”106 This post was repopulated in the National Law Review.107 The National Multiple Sclerosis Society indicates that while a 1999 review found no relationship between MS and trauma, some recent writings have provided contradicting evidence on the topic. This includes case-controlled studies that report a statistically significant link between physical trauma and the risk of developing MS.108

It should not come as a surprise that these divergent views on causation have resulted in conflicting court decisions on whether trauma can cause or aggravate MS. As acknowledged in Lennon v. Norfolk, the casual link between trauma and MS is still being debated in the medical community.109 The court, therefore, held that while the plaintiff’s expert could state that the injury may have caused demyelination, the

103 See Hachinski supra note 60.
104 See Lee Russ et al., 4 ATTORNEYS MEDICAL ADVISOR § 33.19 (1994).
physician was prohibited from claiming that the trauma caused the MS. On the other hand, a jury awarded a musician $2 million based upon the allegation that a car accident triggered his dormant MS into an active disease.

A. Steps to Undertake

Whether MS first surfaces in a case following trauma or is an aggravation of a pre-existing condition, it is critical to obtain the claimant’s medical records. Counsel must ascertain the relationship to when the MS symptoms first appeared and whether the person was asymptomatic leading up to the incident. In this regard, counsel for the claimant should make an attempt to demonstrate that the client had a fruitful and productive life prior to the trauma and that there was a close temporal time frame between the trauma and onset of MS symptoms. Prior appearance of MS symptoms, however, can be fatal if the attorney is trying to demonstrate that the trauma initiated the MS diagnosis, so a diligent search of the medical records is critical.

Defense counsel should file a motion in limine as an attempt to exclude expert testimony that tries to link the MS to the incident. Much research shows that the cause of MS remains unknown and that any attempt to link the diagnosis or aggravation of a pre-existing condition to trauma may be speculative. The defense will also want to comb the medical records for any indication of symptoms that could have led to an earlier diagnosis or a flare up of the malady before the trauma. In this regard, there is much literature that can be used to disprove the assertion that there is a link between MS and trauma. As a corollary, people with MS find it difficult to maintain balance and strength so that these frailties can cause a higher rate of falls and injuries; they are also at risk for injuries to bones, and the soft tissues of the body. Defense counsel handling a slip and fall should be cognizant of this fact and examine the medical records for signs of this disease as the real cause of the accident.

In a workers’ compensation context, the cases appear to favor the granting of benefits even though the connection between trauma and MS may be tenuous. This is due to the long-standing policy of the courts to provide a liberal construction of the

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110 See id. at 1154-55.
111 See Jury Finds That Car Accident Triggered Multiple Sclerosis, 9 NO. 4 VERDICTS, SETTLEMENTS & TACTICS, Apr. 1989, at 118.
113 Id.
114 Id.
116 See generally Kane v. Ford Motor Co., 477 N.E.2d 662, 668 (Ohio Ct. App. 1984) (awarding benefits despite employer seeking to offer evidence disputing link between trauma and MS); see also Stella v. Mancuso, 7 A.D.2d 673, 674 (N.Y. App. Div. 1958) (granting benefits due to causal link between trauma and MS although expert testimony was divided); see also Johnson v. Industrial Com’n of Utah, 660 P.2d 244, 245 (Utah 1983) (remanding where benefits were not awarded for further fact-finding regarding link between trauma and MS).
workers compensation laws to provide benefits. As the cases demonstrate, causation does not have to be unqualified and unequivocal. There is no similar tendency to favor the awarding of damages in a third-party liability case, so the defense might be more successful in challenging causation in this context.

A Westlaw search reveals that 231 cases have addressed multiple sclerosis and trauma. The following is a discussion of some of the court decisions that have arisen involving MS and trauma in different contexts.

B. Cases Finding a Connection Between Multiple Sclerosis and Trauma

As early as 1958, a New York court found a link between trauma and multiple sclerosis. In Stella v. Mancuso, an employee was involved in three separate accidents over a short time frame. Following the last incident, the plaintiff started to experience headaches, dizzy spells and difficulty walking. He was eventually sent to the hospital and diagnosed with multiple sclerosis. This condition was described by the court as “a disease of the central nervous system, a disease marked by sclerosis (hardening) occurring in patches throughout the brain or spinal cord, or both. It is accompanied by weakness, incoordination, jerking movements of the legs, and especially the arms.”

A claim was advanced for worker’s compensation and the Board determined that the condition was causally related to the last accident. This decision was appealed on the basis that the record did not contain sufficient evidence to establish a link between the disease and trauma. The record demonstrated that all of the medical experts agreed that the cause of MS is unknown and that trauma does not cause the condition. The discrepancy in the testimony dealt with whether trauma can aggravate or precipitate the


118 See e.g., Hall v. Baxter Healthcare Corp., 947 F. Supp. 1387, 1387 (D. Or. 1996). See also Sheldon Margulies, Proving Specific Causation Under Daubert, For the Defense (2002). Margulies, a neurologist and member of the Maryland Bar, wrote:

Only after the plaintiff has shown that the agent in question can cause his or her illness (or pathophysiology) can the finder of fact consider specific causation. If scientific studies have already refuted general causation, the courts will not allow specific causation testimony, because it is illogical, and therefore unscientific, to consider the agent as the cause of the plaintiff’s illness if that agent has been found not to cause that illness.

Id.

119 This is based upon a Westlaw search of the phrase “multiple sclerosis and trauma” conducted on December 19, 2019. The search yielded 115 “reported” cases and 116 “unreported” cases.


121 See id. at 170.

122 See id.

123 See id.

124 See id.

125 See id. at 170-71.

126 See Stella, 7 A.D.2d at 170-71.

127 See id. at 171.
The defense claimed that the medical conclusions offered by the plaintiff’s physicians were speculative and that the trial judge should not have referred the matter to an impartial expert. The appellate court disagreed and ruled that there was substantial evidence to support a causal link. The testimony of the independent doctor, when read in conjunction with the testimony of the plaintiff’s expert, was sufficient to establish a causal link in that the trauma may have been the precipitating cause.

_Kane v. Ford Motor Company_ involves an employee who maintained that her pre-existing multiple sclerosis was substantially aggravated by an injury sustained at work. The evidence revealed that the worker started to display marked symptoms of MS within a few months of the incident. The employer asserted that the testimony of the employee’s expert was improper since it was premised upon unfounded medical theories concerning the development of multiple sclerosis. The defense, however, was denied the right to reference a study that disputed any causal link between trauma and MS. On appeal, the court stated that the denial was proper because Ohio does not recognize the learned treatise rule and the defense was still allowed to attack the studies relied upon by the employee’s expert. That witness stated that multiple sclerosis is caused by a virus which attacks the myelin sheath of the nervous system, and it can stay hidden in the patient until sometime between the ages of twenty and forty when unmasked either without warning or by a factor in the environment including trauma. In this case, all of the experts agreed the scientific literature recognized a connection between trauma and multiple sclerosis. In finding in favor of the worker, the court noted that the employee showed no symptoms of MS before the incident and then began experiencing symptoms within one week which then worsened over time. The injury, therefore, unmasked the disease and this was enough to establish the connection between the injury and MS.

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128 See id.
129 See id.
130 See id.
131 See id.
132 See _Kane v. Ford Motor Company_, 477 N.E.2d 662 (Ohio App. 1984). See also _Cooney v. Terminex_, 94 N.J.A.R.2d (WCC) 16 (N.J. Adm.) (holding that plaintiff’s condition was exacerbated by injury suffered while working).
133 See _Kane_, 477 N.E.2d at 666-67.
134 See _Kane_ at 664.
135 See id. at 664-65 (noting appellant’s experts could use study in forming opinion on causal link between MS and trauma).
136 Id. at 664. The "learned treatise" is an exception to the hearsay rule, which allows a statement contained in a treatise, periodical, or pamphlet if (a) the statement is called to the attention of an expert witness on cross-examination or relied on by the expert on direct examination; and (b) the publication is established as a reliable authority by the expert’s admission or testimony, by another expert’s testimony, or by judicial notice. Fed. R. Evid. 803(18).
137 See _Kane_, 477 N.E.2d at 665.
138 See id. at 666 (indicating all testifying witnesses, including witness of appellant, agreed multiple sclerosis is latent disease).
139 See id. at 667 (finding plaintiff proved prima facie case entitled to recovery).
140 See id.
McSwain v. Chicago Transit Authority provides an example of the difficulty to diagnosis MS soon after a traumatic event.\textsuperscript{141} The lower court found there was a causal relationship between MS and trauma and awarded $150,000.\textsuperscript{142} On appeal, the judges upheld the award and noted that multiple sclerosis is a disease of the myelin; the coating around the cells of the nervous system which “insulates, nourishes and conducts impulses along the axons of the nervous system.”\textsuperscript{143} Its cause remains unknown but doctors believe that MS originates from a virus which is present in the body before the age fifteen and which is activated or exacerbated upon some triggering event such as trauma.\textsuperscript{144} The court stated that prior to the accident, the plaintiff, a 41-year-old woman, had been in good health and did not exhibit any signs of MS.\textsuperscript{145} The accident in question occurred when the elevated train in which the plaintiff was a passenger was hit in the rear by another train causing her to strike a metal bar on the seat in front of her.\textsuperscript{146} She was admitted to the hospital several days later for treatment of traumatic radiculitis.\textsuperscript{147} The pain was so severe that is was only moderately controlled by medication.\textsuperscript{148} She also complained of weakness on her right side.\textsuperscript{149} After her discharge, the plaintiff returned to work as a nurse for three weeks until she fell at home, broke her arm and never returned to work again, as she developed dizziness and pain in multiple parts of her body.\textsuperscript{150} Nine months after the accident, she complained of double vision and asserted that her neck always hurt, that her right extremities were weak, and that she had radiating pain from her neck and shoulder on the right side.\textsuperscript{151} The doctor determined at this time that she had paresthesia and hyposthenia related to the incident but that something more than the trauma was involved.\textsuperscript{152} Twenty-eight months after the train accident, she was involved in a motor vehicle collision and was admitted to the hospital with an aggravation of her symptoms.\textsuperscript{153} The examining doctor determined that she was experiencing some type of neurological event aside from the accident but did not believe that she had multiple sclerosis, a condition which he did not treat as part of his practice.\textsuperscript{154} Following discharge, she was again hospitalized after her neurological symptoms intensified and it was determined that she had MS.\textsuperscript{155}

\textsuperscript{142} See id. at 1264.
\textsuperscript{143} See id. at 1265, 1278; see also Hersh & Fox, supra note 9 (describing changing disease pathology in early, progressive and late stages of disease).
\textsuperscript{144} See McSwain, 362 N.E.2d at 1265 (indicating injury to neck, fever, excessive fatigue, infections, emotional distress or other factors trigger MS).
\textsuperscript{145} See id. at 1266 (demonstrating plaintiff worked continuously as licensed practical nurse).
\textsuperscript{146} See id. (testifying she had lump in her throat, difficulty swallowing, gagging feeling, and tight chest).
\textsuperscript{147} See id. at 1267. Radiculitis, when severe, is a permanent condition which involves the nerves coming out between the cervical vertebrae and joining up with the nerves over the shoulder. \textit{Id.}
\textsuperscript{148} See id. Neck pain was severe with active and passive movement of plaintiff’s head, resistance aggravated the pain; muscle spasticity was measured at the top degree of severity or intensity of pain; movement of plaintiff’s right arm was restricted and painful. \textit{Id.}
\textsuperscript{149} \textit{Id.}
\textsuperscript{150} McSwain, 362 N.E.2d at 1269.
\textsuperscript{151} \textit{Id.} at 1268-69.
\textsuperscript{152} \textit{Id.} at 1269.
\textsuperscript{153} \textit{Id.} at 1270.
\textsuperscript{154} \textit{Id.} at 1270-71.
\textsuperscript{155} \textit{Id.} at 1271.
At trial, the diagnosing physician was asked if the plaintiff’s MS was related to the trauma of the train accident and he responded: “I think that it could well be.” Another physician opined that trauma does not cause or exacerbate MS, but an insult to the cervical area can provoke an element of the disease and that an external force may bring forth a symptom complex of multiple sclerosis. A third physician noted that no one knows the cause of multiple sclerosis but an aggravation can be triggered “by trauma, fatigue, infections, emotional outbursts and worry.” He went on to say that before a diagnosis of MS can be made, it is reasonable to see such findings as nystagmus, Babinski signs, reflex changes, and vision difficulties within weeks to months after the trauma before one can exhaust the connection of trauma to the disease. The court also reported that trauma can trigger the symptoms of MS and that one of the most difficult issues with multiple sclerosis has to do with the diagnosis: “It takes a very trained observer making a very careful study to really be able to make a diagnosis of multiple sclerosis when it does not manifest itself in the classic form, when it is not in its most advanced stages.”

The defendant asserted on appeal that the plaintiff did not satisfy her burden of proof concerning the connection between MS and trauma. The appellate court ruled that this was a question of fact which had already been decided by the lower court. Therefore, the verdict was upheld.

National Castings Division of Midland-Ross Corporation v. The Industrial Commission involves a worker’s compensation claim in which there were dueling experts as to whether trauma can cause or aggravate MS. This case dealt with an employee with pre-existing MS who was hit in the face with industrial sand resulting in the loss of vision in the left eye. The plaintiff’s ophthalmologist noted that the origins of multiple sclerosis are not known but the condition can affect assorted nerves which can cause blindness. He also felt that any trauma could precipitate the disease or activate a dormant condition. On cross-examination, the eye doctor admitted that because the origins of MS are not known, there was a chance that the employee’s optic nerve could atrophy without a traumatic incident.

The defendant’s expert countered that while a traumatic event might exacerbate multiple sclerosis, the amount of trauma would have to be similar to a blow to the spinal cord or head and could not be caused from emotional stress such as that which might occur in a patient with a foreign substance in his eye. Another expert for the defense confirmed that the cause of multiple sclerosis is unknown, and opined that there was no causal connection between the employee’s eye problem and the incident at work:

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156 McSwain, 362 N.E.2d at 1273.
157 Id. at 1274-75.
158 Id. at 1276.
159 Id.
160 Id.
161 Id. at 1264-65.
162 McSwain, 362 N.E.2d at 1278.
164 Id. at 331.
165 Id.
166 Id. at 331.
167 See id.
168 See id. at 332.
“explaining that emotional shock and trauma could not aggravate a dormant condition of multiple sclerosis and that trauma was not generally accepted as an etiology of this disease.”

This testimony was then countered by a neurologist called by the claimant who noted that there could be a causal relationship between the employee’s blindness and his job because latent multiple sclerosis can be made worse by any type of trauma, which in some cases may actually precipitate symptoms of multiple sclerosis.

In awarding worker’s compensation benefits, the court found that the origin of multiple sclerosis remains unknown. However, three of the doctors who testified found that trauma could accelerate latent multiple sclerosis even though they disagreed as to the amount of trauma which would be necessary. When there is incomplete medical knowledge about an illness, the court is able to conclude that the medical testimony relating to causation does not have to be unqualified and unequivocal.

_Nardozzi v. Piotrowski_ involved a matter in which the defense attempted to affirmatively use a plaintiff’s previous diagnosed multiple sclerosis condition to mitigate the damages from a car accident. The plaintiff was in a collision when the defendant ran a red light causing injuries to the plaintiff’s neck and back. The defense asserted that her symptoms were caused by Nardozzi’s MS and not related to the accident. Prior to the event, Nardozzi had some symptoms of MS and underwent foraminotomy post-accident to relieve herniated disc symptoms. The jury returned with a $700,000 verdict that was apportioned 65% to the plaintiff and 35% to the defendant.

C. Cases Finding No Connection Between Multiple Sclerosis and Trauma

In 1943, the court was asked to find a connection between MS and a traumatic accident and refused to make that association. The case, _Zanski v. Yellow Cab_, dealt with a worker’s compensation claim involving a 32-year-old cab driver who sustained non-life threatening injuries in a November 18, 1937 motor vehicle accident, which he alleged caused his MS. After the accident he returned to work on November 30, 1937 and drove cabs until June 18, 1938, when he quit to work as a furrier during the summer season. Six months later, he returned to his job driving a cab after the employer’s

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170 See id.
171 See id. at 333.
172 See id. at 332.
173 See id. at 333.
175 See Rosenhouse & Lunn, supra note 174 at §4.10.
176 See id.
177 See id.
178 Id.
180 See id. The driver’s injuries included a left hip contusion, muscular sprain and low back tenderness, but no bon involvement or injuries to the head, spine or central nervous system. Id.
181 See id.
physician cleared him for employment, finding that he had no disability.\textsuperscript{182} He then worked for almost another year until he quit on October 14, 1939, but asserted that he never fully recovered from his motor vehicle accident injuries.\textsuperscript{183} Several months before he quit, he started walking unsteadily and his difficulties increased.\textsuperscript{184} After quitting, the driver was diagnosed with advanced multiple sclerosis, which he claimed was caused by his work-related injury.\textsuperscript{185} A number of physicians testified that the etiology of the disease is not definitively known and remains exclusively in the realm of speculation.\textsuperscript{186} The expert testimony, relying on 1940s medical knowledge, indicated that MS is typically characterized by periods of remission and exacerbation and there may be either rapid onset of symptoms or slow development.\textsuperscript{187} The sole witness to testify on behalf of the plaintiff failed to claim that an accident can cause the disease, but maintained that trauma can cause an aggravation of the claimant’s predisposition to the disease resulting in disability.\textsuperscript{188} Four other physicians, however, testified that there is no scientific proof that there is a relationship between trauma and MS.\textsuperscript{189} Therefore, the court concluded that the evidence established that the plaintiff’s MS was not caused by the accident, “since the evidence of [a] traumatic aggravation of the disease [was] purely speculative and conjectural.”\textsuperscript{190}

In \textit{Carter v. Flintrol, Inc.}, the plaintiff slipped at work and hit her head.\textsuperscript{191} The plaintiff employee testified that she was relatively healthy prior to the fall, as she had experienced no balance issues and had not sought medical treatment in several years.\textsuperscript{192} After the fall, the plaintiff exhibited back pain and balance issues, prompting her to miss four days of work.\textsuperscript{193} Upon her return to work, plaintiff’s balance issues continued and she started having vision issues and became nauseated.\textsuperscript{194} She was eventually diagnosed with MS and was unable to work.\textsuperscript{195} A claim was advanced for worker’s compensation.\textsuperscript{196} The medical testimony was conflicting.\textsuperscript{197} Her treating doctor noted that evidence exists in medical literature supporting the principle that multiple sclerosis may be caused or aggravated by trauma but he failed to present a personal opinion as to a connection in this case.\textsuperscript{198} Another doctor stated that the claimant had a subdural hematoma and secondarily MS, while a third physician testified, “I do not feel that the

\begin{footnotesize}
\begin{enumerate}
\item Id.
\item Id.
\item See id. at 342.
\item See \textit{Zanski}, 143 Neb. at 342.
\item Id.
\item Id.
\item Id.
\item Id. at 343.
\item Id. The physicians admitted that there is some authority that trauma and MS may have “relativity in case of serious, deep injury to the physical structure of the back of the head involving the brain, spinal cord or central nervous system, seat of pathology.” Id. at 344.
\item Id.
\item Id.
\item Id.
\item Id. at 318-19.
\item Id. at 318.
\item See \textit{Carter}, 19 Ark App. at 318-20.
\item Id.
\end{enumerate}
\end{footnotesize}
multiple sclerosis has any relation whatsoever to her injury and appears to be just
coincidental.” 199 The administrative law judge concluded that a causal connection did
exist. 200 The Commissioner upheld this determination, but reversed the finding that
there was a causal relationship between the injury and the resulting MS. 201 On appeal,
the court noted that the treating doctor failed to testify there was a causal connection
between the injury and MS. 202 He provided no opinion on the topic after reading that
literature. 203 In the face of the other medical testimony, the court was unable to find
that the claimant had met her burden of proof with regard to the multiple sclerosis, so
her claim for benefits and disability attributable to the MS was denied. 204

D. Evidentiary Rulings

The first question with admissibility deals with whether a medical expert must
be called to testify that the plaintiff’s MS was aggravated by an accident. That issue was
addressed in Fladmo v. Sprague. 205 The plaintiff was involved in a motor vehicle accident
and claimed that the incident aggravated her pre-existing MS condition. 206 The
defendant maintained that the trial court incorrectly allowed evidence to be presented
concerning Fladmo’s subjective complaints in ascertaining causation and that medical
testimony on this issue needed to be presented. 207 The court found that whether the
plaintiff’s multiple sclerosis was aggravated by the accident is not within common
knowledge, so that medical testimony is necessary. 208

The defendants asserted that no physician “stated that the aggravation of
plaintiff’s multiple sclerosis was more probably than not caused by the accident.” 209
Nevertheless, the court found that the medical experts plainly testified that it is not easy
to establish a direct correlation between the ailments caused by accident and the
symptoms caused by her condition, but they agreed that symptoms of MS can be made
worse by trauma. 210 In this case, the plaintiff was doing well and was improving with
increased strength, balance and coordination. Following the accident, she had a decline

199 Id.
200 Id. at 318.
201 Id.
202 Id. During the doctor’s testimony, he merely advised the court that “there is evidence in the
medical literature supporting the idea that multiple sclerosis may be triggered or aggravated by
203 Id. at 319-21.
204 Id.
2013).
206 Id.
207 Id. The trial court stated that if the medical evidence did not clearly establish the burden of
proof of causation, then then court could consider a witnesses’ subjective complaints of pain to
find the burden satisfied, pending it is credible, consistent and corroborated. Id.
208 Id. at *2. In order to successfully recover damages for injuries caused by another, the plaintiff
has the burden of proving causation by a preponderance of the evidence; the burden must be
met with the entirety of the evidence (direct and circumstantial), and shows that the fact or
causation is more probable than not. Id.
209 Id. at *3.
210 Id. (explaining plaintiff’s condition since approximately 1997 to present).
In health and an increase in symptoms. In upholding the award in favor of the plaintiff, the court noted that the trial judge weighed the medical testimony, the medical records, and the testimony of the plaintiff and concluded that causation was established more probably than not. Even if the subjective complaints of the plaintiff were not considered, the medical evidence was enough for the claimant to meet her burden of proof as to causation.

Lennon v. Norfolk and Western Railway Company involved a motion to exclude the testimony of the plaintiff’s treating neurologist that an incident at work may have caused her multiple sclerosis. The facts show that the plaintiff fell at work and hit his head. He soon started complaining of severe headaches but a CT Scan was negative. The plaintiff returned to his job with no further complaints, but one year later, he was suspended because of an allegation that he falsified his overtime pay requests. Several days later, he visited his primary care physician alleging headaches, memory problems, and feeling “spaced out.” Lennon was referred to a neurologist and the diagnosis of MS was made. The plaintiff claimed that the MS was caused by his injury and that he could no longer work because of the disease. Suit was filed under the Federal Employers’ Liability Act and the railroad asserted that there is no reliable scientific evidence that an injury to the head could proximately cause MS. The court noted that “whether physical trauma may trigger or precipitate MS has been a topic of much debate in the medical community since the latter part of the nineteenth century. That debate rages today.” While recent studies have not found a link between trauma and MS, there is some opposition to this view. While the overwhelming weight of the evidence suggests that there has been no conclusive showing demonstrating a link between trauma and the onset or exacerbation of MS, that might have been enough under the old Frye standard of general acceptability to allow the evidence. However, Daubert mandates a further analysis. In this case, the plaintiff’s expert is a neurologist who has conducted no research on the association between trauma and MS and his opinion that a head injury may precipitate MS is premised upon his review of the medical literature which was very selective and lop-sided. The point that other patients with MS may have had trauma prior to the disease's inception by itself does not

211 Sprague, 2013 WL 6506258, at *3
212 Id. at *5.
213 Id.
215 Id. at 1145.
216 Id. at 1146.
217 Id.
218 Id.
219 Id.
220 Lennon, 123 F. Supp. 2d. at 1146.
221 Id. at 1148.
222 Id.
223 Id. at 1151. See Frye v. United States, 293 F. 1013, 1014 (D.C. Cir. 1923) (establishing standard for expert testimony as “general acceptance” in the “field in which it belongs”).
224 Lennon, 123 F. Supp. 2d. at 1151. See Daubert v. Merrell Dow Pharm., Inc., 509 U.S. 579, 588 (1993). The court established a new standard that expert testimony is admissible if it will “assist the jury in comprehending the evidence and determining issues of fact”. Id.
225 Lennon, 123 F. Supp. 2d. at 1151.
carry the day.\textsuperscript{226} Therefore, the defendant’s motion to exclude was granted because there was no competent evidence establishing a relationship between the fall and the development of multiple sclerosis.\textsuperscript{227}

Thomas v. Poole reached the opposite result. This case involved a motor vehicle accident in which the plaintiff suffered an alleged aggravation of his pre-existing multiple sclerosis.\textsuperscript{228} A motion in limine was filed by the defense to bar the plaintiff’s expert from discussing any type of casual connection between the claimant’s MS and the trauma.\textsuperscript{229} It was asserted that because such testimony could not fulfill the requirements of the Daubert standard of admissibility, any mention of the plaintiff’s MS would be irrelevant and prejudicial.\textsuperscript{230} The court noted that the defendant’s motion puts it “squarely in the middle of a complex, and often heated debate within the medical world.”\textsuperscript{231} The court went on to provide an excellent history of the controversy with citations to various medical literature concerning a possible causal link between trauma and MS.\textsuperscript{232} The court, however, was able to sidestep the issue of causation because plaintiff’s treating physician distanced himself from having to say that there was a link between the disease and trauma so he was precluded from testifying in this regard.\textsuperscript{233} Nevertheless, the physician could talk about how an MS patient might experience discomfort from his injuries that would be different from a person who did not suffer from the malady.\textsuperscript{234} This kind of opinion did not discuss the etiology of MS but instead dealt with the symptoms of MS, and would be allowed.\textsuperscript{235} Thus, the defendant’s motion to exclude all references to the plaintiff’s MS was denied.\textsuperscript{236}

In Carney v. Dhillon, the defense filed a motion in limine to exclude testimony of two physicians concerning the causal relationship between physical trauma and the exacerbation of multiple sclerosis.\textsuperscript{237} The court denied the motion and found that the

\begin{itemize}
\item \textsuperscript{226} Id. at 1152.
\item \textsuperscript{227} Id. at 1160.
\item \textsuperscript{228} Thomas v. Poole, 2000 U.S. Dist. LEXIS 1529, at *2 (E. Dist. La. Feb. 11, 2000).
\item \textsuperscript{229} Id. at *10.
\item \textsuperscript{230} See id.
\item \textsuperscript{231} Id. at *11.
\item \textsuperscript{232} See Thomas, 2000 U.S. Dist. LEXIS, at *11-15 (discussing differing medical opinions on link between trauma and MS). Court recognized that many medical experts cannot conclude whether there is or is not a link between trauma and MS. Id. at 11. “‘Despite the long history of this idea, the proposed causal link between [physical trauma or psychological stress] and MS has yet to be established or refuted conclusively.’” (quoting D.S. Goodin et al., The Relationship of MS to Physical Trauma and Psychological Stress: Report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology, 52 Neurology 1737 (1999)). Id. Court also observed some medical opinions that criticize the studies such as Goodin and their experimental methods. Id. at 13-14. “Dr. Poser argued that researchers…unjustifiably ‘assume that MS patients are not only genetically and immunologically identical, but that their disease has reached the same extent of clinical and pathologic involvement, and a similar degree of activity at the time of trauma.’” Id. (quoting Charles M. Poser, The Role of Trauma in Pathogenesis of Multiple Sclerosis: A Review, 96 Clinical Neurology and Neurosurgery 103 (1994)). Id. at 14.
\item \textsuperscript{233} See Thomas, 2000 U.S. Dist. LEXIS, at *15.
\item \textsuperscript{234} See id. at *17.
\item \textsuperscript{235} Id. at *18.
\item \textsuperscript{236} Id.
\item \textsuperscript{237} See Motion to Exclude Testimony 1:18-22, Carney v. Dhillon, (No. 03-2-23783-3SEA), 2004 WL 3584981.
\end{itemize}
relationship between trauma and MS is not new so that a Frye review is improper. The challenge by the defense goes to the weight of the evidence and not its admissibility.

The court rejected the allegation that trauma triggered a dormant MS condition in Higgins v. Delta Elevator Services. This matter involved an insurance adjuster who fell while exiting an elevator. Six years earlier, she had been diagnosed with MS but that condition was asymptomatic. A few days after the incident, her multiple sclerosis flared up to the point that she had to use a cane to ambulate and was confined to a wheelchair at the time of trial. The defense produced two neurologists who stated that MS cannot be exacerbated by trauma. The plaintiff’s physician was unable to prove that his exacerbation theory was generally accepted in the medical community.

The defendant filed a motion in limine to exclude the testimony of the plaintiff’s expert on the ground that the exacerbation theory was not reliable or valid. The trial judge allowed the doctor to testify and instructed the jury that they “must determine whether the reasoning or methodology underlying the testimony is scientifically valid and applicable to the facts at issue,” an instruction upheld on appeal. The defendant successfully rebutted the “anecdotal” evidence of an exacerbation by introducing two studies that demonstrated that there was no significant relationship between trauma and an exacerbation of MS.

E. Discovery

Keleman v. Quinton Fitness Equipment, Inc. involved a discovery dispute pertaining to the plaintiff’s pre-existing multiple sclerosis condition. Keleman claimed that a treadmill was improperly designed causing her to sustain a fractured ankle and aggravation of a prior back problem. The defense, however, wanted to explore her pre-existing MS condition and she objected. The court found that her MS was

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238 See Order on Defendant’s Motion to Exclude Testimony 2:14-17, Carney v. Dhillon (No. 03-2-23783-3SEA), 2004 WL 5064683. See also Frye v. United States, 293 F. 1013 (D.C. Cir. 1923).
239 See Order on Defendant’s Motion to Exclude Testimony at 2:18-19, Carney v. Dhillon (No. 03-2-23783-3SEA), 2004 WL 5064683.
243 Id.
244 Id.
245 Id.
247 See id. at 644-45. The judge also instructed jurors to “look at specific factors brought out in the testimony as being probative of the reliability of Higgins’s expert medical opinion testimony.” Id.
248 See Higgins, supra note 242, at 11:32 (noting that the jury ultimately found that the defendant was not negligent).
250 Id. at 185.
251 Id. at 186.
relevant to the issue of causation because it may have had a bearing on her inability to walk and her neurological deficits may have played a part in her fall.\textsuperscript{252}

F. Other Countries

The courts in the United States are not the only jurisdictions that have struggled with the question of whether MS can be caused by or aggravated by trauma. It seems to be an evidentiary controversy around the world. For instance, Canada tackled the issue in \textit{Taylor v. Liong}.\textsuperscript{253} This matter arose out of a car accident and a hearing was held to determine the admissibility of the evidence as to whether mild head trauma or whiplash could be linked to the onset of MS symptoms.\textsuperscript{254} In this regard, the court noted that it felt like it was being asked to determine a scientific rather than a legal question.\textsuperscript{255} While it appreciated the ultimate issue of causation, the court wrote that the general issue, whether trauma can set off MS, “dominates the ultimate or specific issue to such an extent that it must be proved on a balance of probabilities to engage even a remote possibility of a connection between the accident and the plaintiff’s ensuing symptomatic MS.”\textsuperscript{256} According to the plaintiff’s theory of the case, it was maintained that MS symptoms can be caused by trauma in less than 5% of all cases.\textsuperscript{257} Therefore, the court framed the issue as not one of general scientific theory but whether the plaintiff can demonstrate a connection between the onset of her MS symptoms and the accident.\textsuperscript{258} The court further noted that it could not find any studies or analysis relied upon by the claimant to create a logical linchpin for proof of the causal connection being made.\textsuperscript{259} Therefore, the court concluded that the evidence did not support proof of a causal relationship between mild trauma such as whiplash and MS exacerbation.\textsuperscript{260}

The high court in the United Kingdom took up the issue in \textit{Perry v. The Post Office}.\textsuperscript{261} This matter involved a worker who tripped over a mail bag, hurt her back, and hit her head.\textsuperscript{262} The parties agreed that her injuries were not significant.\textsuperscript{263} The facts show that she saw her primary care physician and remained out of work for five weeks.\textsuperscript{264} When she returned to the job, her back pain worsened and she developed a feeling of heaviness in the legs.\textsuperscript{265} Perry was sent to a specialist who made the diagnosis

\begin{itemize}
\item \textsuperscript{252} \textit{Id.}
\item \textsuperscript{253} \textit{Taylor v. Liong}, 2008 BCSC 242 (Can.).
\item \textsuperscript{254} \textit{Id.} at para. 1.
\item \textsuperscript{255} \textit{Id.} at para. 10.
\item \textsuperscript{256} \textit{Id.} at para. 24.
\item \textsuperscript{257} \textit{See id.} at para. 26 (reasoning illogical conclusion plaintiff’s MS symptoms induced by trauma).
\item \textsuperscript{258} \textit{See id.} at para. 27.
\item \textsuperscript{259} \textit{See Taylor}, 2008 BCSC 242, at para. 121 (maintaining awareness of rarity of “logical linchpin for proof of the causal relationship being espoused”).
\item \textsuperscript{260} \textit{See id.} at para. 123.
\item \textsuperscript{261} \textit{See Mark Corcoran, An Unhappy Coincidence Between Multiple Sclerosis and Trauma}, 359 THE LANCET 726 (2002) (explaining case finding woman’s MS diagnosis after work accident coincidence, not result of trauma).
\item \textsuperscript{262} \textit{See id.}
\item \textsuperscript{263} \textit{See id.} (noting worker’s injuries included slight tenderness in her lower thoracic and lumbar areas).
\item \textsuperscript{264} \textit{See id.} (noting worker’s physician diagnosed muscle strain/spasm and prescribed analgesia).
\item \textsuperscript{265} \textit{See Corcoran, supra note 261} (noting the worker returned for three days and these symptoms occurred during this period).
\end{itemize}
of multiple sclerosis and her physical condition continued to deteriorate.\textsuperscript{266} The issue before the court was what injuries the plaintiff sustained and whether physical trauma can cause the onset of MS or be a causal factor at all.\textsuperscript{267} Each side called a neurologist who shared an opposite view.\textsuperscript{268} The plaintiff’s expert maintained that trauma, such as a flexion/extension injury, could result in focal breaches in the blood-brain barrier thereby causing the release of substances into the central nervous system causing demyelination within three months of trauma.\textsuperscript{269} The expert for the employer asserted that MS occurs as the result of more subtle changes involving the movement of T lymphocytes across the blood-brain barrier and that there is no epidemiological evidence to support a connection between MS and trauma.\textsuperscript{270} This expert concluded that the presentation of symptoms so soon after the trauma in this case was a coincidence.\textsuperscript{271} The court was very critical of the plaintiff’s expert and found that he had exaggerated his argument which did not hold up upon closer examination.\textsuperscript{272} Therefore, the court found that there was no reliable evidence presented to bolster the plaintiff’s argument.\textsuperscript{273} While the court may have been impressed by evidence presented by another witness for the plaintiff on causation, none was presented.\textsuperscript{274} The court ended up finding in favor of the defense because of the inadequate evidence to support the plaintiff’s position.\textsuperscript{275}

Another case from England reported in \textit{The BMJ} (previously known as \textit{The British Medical Journal}) reached the opposite result and found a connection between trauma and MS.\textsuperscript{276} This matter involved a former police officer who was awarded $820,875 when it was determined that he developed MS following a whiplash injury in a motor vehicle accident.\textsuperscript{277} The plaintiff claimed that his symptoms of MS surfaced within 17 days of the accident that involved the overturning of his police van.\textsuperscript{278} His condition continued to worsen over time and he was forced to retire from his job three years after the accident.\textsuperscript{279} Expert witnesses were called from both Britain and the United States who were split on the question of whether trauma can cause the symptoms of multiple sclerosis.\textsuperscript{280} The plaintiff’s experts noted that they had seen cases in which patients had developed MS symptoms within weeks of the trauma.\textsuperscript{281} While

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\item \textsuperscript{266} See \textit{id.} (explaining that worker was diagnosed with MS fourteen months after seeing specialist).
\item \textsuperscript{267} See \textit{id.}
\item \textsuperscript{268} See \textit{id.} The two experts had special interest in MS and demyelinating diseases, but nearly polar opposite views on the presence of a causal link between trauma and MS. \textit{id.}
\item \textsuperscript{269} See \textit{id.} Both experts maintained that pathogenesis of MS includes a change to the regular functioning of the blood-brain barrier. \textit{id.}
\item \textsuperscript{270} See Corcoran, \textit{supra} note 261.
\item \textsuperscript{271} See \textit{id.}
\item \textsuperscript{272} See \textit{id.}
\item \textsuperscript{273} See \textit{id.} (discussing why court did not credit expert’s testimony).
\item \textsuperscript{274} See \textit{id.} (noting narrow nature of court’s holding).
\item \textsuperscript{275} \textit{id.}
\item \textsuperscript{276} Bryan Christie, \textit{Multiple Sclerosis Linked with Trauma in Court Case}, 313 \textit{The BMJ} 1228, 1228 (1996).
\item \textsuperscript{277} \textit{id.}
\item \textsuperscript{278} \textit{id.}
\item \textsuperscript{279} \textit{id.}
\item \textsuperscript{280} \textit{id.}
\item \textsuperscript{281} \textit{id.}
\end{itemize}
injuries would not cause MS by themselves, they testified that the trauma could bring on
the condition in a susceptible person.\textsuperscript{282}

The experts for the defense disagreed and opined that the relationship between
when the symptoms first appeared and the accident were coincidental.\textsuperscript{283} In fact, one
expert testified that epidemiological studies had failed to find any link, and that it was
generally accepted in the United States that no such connection existed.\textsuperscript{284} The court
found that there is controversy within the medical community about the relationship of
trauma and MS but felt that the dispute was not for it to resolve.\textsuperscript{285} In this case, the
court believed that the plaintiff satisfied his burden
of proof by demonstrating a causal
link between his flexion/extension injury and the development of MS symptoms in the
same area.\textsuperscript{286} Therefore, the court accepted the evidence that trauma can be a causal
factor in some cases.\textsuperscript{287} The court further wrote that it was particularly impressed with
the testimony of the plaintiff’s experts who testified that they had seen cases where they
had accepted the onset or recurrence of MS as the result of trauma, especially after a
whiplash injury.\textsuperscript{288}

Two cases from British Columbia, however, demonstrate how difficult it is to
establish a causal link between MS and trauma.\textsuperscript{289} In one decision, the court dismissed
an appeal filed by the plaintiff in which the trial judge determined that she had not
sustained brain injuries in two accidents.\textsuperscript{290} The judge noted that the plaintiff had been
diagnosed with MS of a remitting/relapsing form.\textsuperscript{291} Therefore, her neurological
symptoms could be explained on that basis and not as the result of the accidents.\textsuperscript{292}

The second matter, the aforementioned \textit{Taylor v. Liong}, involved whether
physical trauma caused the onset or exacerbation of MS.\textsuperscript{293} One British Columbia

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\item \textsuperscript{282} See Christie, \textit{supra} note 276, at 1228 (summarizing opinions of experts).
\item \textsuperscript{283} \textit{Id}.
\item \textsuperscript{284} \textit{Id}.
\item \textsuperscript{285} \textit{Id}.
\item \textsuperscript{286} \textit{Id}.
\item \textsuperscript{287} \textit{Id} (quoting Lord Dawson’s judgment). “‘I also accepted that the historical, anecdotal and
experimental evidence supported the proposition that trauma can be a causative factor in some
cases and in some circumstances.’” \textit{Id}. See also Dingley v. Chief Constable of Strathclyde Police
(2000) SC 77 (Scot.) (providing an overview of trial court judgment and affirming judgment).
\item \textsuperscript{288} See Christie, \textit{supra} note 276, at 1228 “‘More compelling… was the evidence of the medical
witness for Mr[.] Dingley who had all themselves seen cases where they had accepted that the
onset or recurrences of symptoms had been brought about by trauma, especially whiplash injury.
In my opinion, these circumstances are far too strong to be put down to mere chance.’” \textit{Id}.
\item \textsuperscript{289} See Erik Magraken, \textit{MS and Trauma – Difficult to Legally Link}, BC INJURY LAW BLOG (Mar. 23,
Columbia court cases involving link between MS and trauma).
\item \textsuperscript{290} See Roeske v. Brickwood Holdings Ltd., 2008 BCCA 88, para. 1-4 (Can.) (reviewing the trial
court’s reasoning and dismissing the appeal).
\item \textsuperscript{291} See \textit{id}. at para. 38 (asserting that no evidence was provided suggesting brain injuries can be
relapsing/remitting in nature).
\item \textsuperscript{292} See \textit{id} (acknowledging likelihood that MS caused the plaintiff’s neurological symptoms).
\item \textsuperscript{293} See Taylor v. Liong, 2008 BCSC 242, para. 4 (Can.). The case was heard to determine “the
substantive issue of whether the evidence before the Court proves on the balance of probabilities
that trauma (such as mild head trauma or whiplash) can or is capable of triggering or
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attorney who blogged about the decision noted that the competing medical evidence made the question difficult to resolve, and that because the judge reviewed the leading scientific evidence on the issue, the decision is a “must-read” for litigators that allege MS connected to trauma.\footnote{294} Upon conclusion of the case, the court dismissed the lawsuit because the plaintiff had not demonstrated on balance the probabilities that such a connection exists.\footnote{295} In support of this conclusion, the court noted that a “substantial majority of the relevant scientific community has rejected the notion of a causal connection based on developments in understanding the pathogenesis of the disease, epidemiological studies, [and] reanalysis of previous studies said to support the link.”\footnote{296}

In \textit{Taylor v. Liong}, the Supreme Court of British Columbia had to decide if it should admit expert testimony concerning whether multiple sclerosis was caused by an accident.\footnote{297} The court refused to allow the expert for the plaintiff to testify because his proposed opinion failed to meet the proper standards for reliability.\footnote{298}

\section*{IV. Conclusion}

Unfortunately, the medical answer as to whether or not trauma causes worsening of MS is uncertain at best, and too muddled and confusing. The problems in demonstrating such a relationship are many, starting with the nature and cause of the disease in the first place. Then, one must consider the variabilities of the disease as well as the difficulty in the statistical analyses of the studies demonstrating either a correlation or no correlation between trauma and MS, sometimes influenced by the bias of the researchers. As Mark Twain once quipped, “There are three kinds of lies: lies, damned lies and statistics.”\footnote{299} Until more definitive evidence is available and subspecialty consensus statements are made, the best approach is to “place more weight on the best epidemiological data currently available to us which have, up to this point, failed to demonstrate any link between trauma and the development or exacerbation of multiple sclerosis.”\footnote{300} Even those whose work supports the trauma-MS connection state that “more rigorous prospective studies, with high statistical power, are needed to convincingly establish an association between trauma and MS.”\footnote{301}

With this split in the medical community, it is little wonder that lawsuits trying to establish a link between MS and trauma are so contentious and become a battle of the
experts. Sufficient medical articles and judicial opinions exist to support either argument which is most unusual. An analysis of the reported cases also finds no pattern to guide counsel on the outcome of a particular claim and the credibility of the witnesses has a strong bearing on the ultimate determination.