Here's the thing that will be invisible!

January 1, 2021

LawyerFirst LawyerLast Esquire

SampleFirmName

123 Address St.

CityVille, StateLand 12345

Tel: (012) 345-6789

RE: *Pl1FirstName Pl1LastName; Pl2FirstName Pl1LastName; Third One Third Name et al. v SampleCaseDefendantName et al., Case No: CaseNoSample, SampleCourtName*

Date of Crash: January 1, 2020

Date of Birth: *Pl1FirstName Pl1LastName:* January 1, 1999 [20 years old at time of crash]

*Pl2FirstName Pl1LastName:* January 2, 1990 [29 years old at time of crash]

*Third One Third Name:* January 4, 1994 [25 years old at time of crash]

Dear Mr. LawyerLast,

I am in receipt of your correspondence regarding the above-named action. I have reviewed the documentation accompanying your correspondence including medical records, information regarding the subject crash, litigation documents, and other materials, including the January 1, 2021, report from the defendant's crash reconstruction and biomechanical expert, Dr. ExpertFirst ExpertLast.

The purpose of this report is to provide an analysis of the causal relationship between the subject rear impact collision and Ms. and Mr. Pl1LastName and Mx. Third Name’s subsequently diagnosed disk injuries and need for treatment.

*My qualifications to provide opinions concerning the matters herein, particularly on issues of the causal relationship between trauma and injury, are as follows:*

I am Professor and Chair of Forensic and Legal Medicine with the Faculty of Forensic and Legal Medicine of the Royal College of Physicians (UK), and a consultant in the fields of forensic medicine and forensic epidemiology. I am credentialed as a Fellow of the Royal College of Pathologists (UK), Fellow of the Faculty of Forensic and Legal Medicine (FFLM) of the Royal College of Physicians (UK) and member of the British Association in Forensic Medicine. I hold the following relevant academic degrees and certifications: a Doctor of Medicine degree (Med.Dr.) from Umeå University, a Doctor of Philosophy (Ph.D.) in public health/epidemiology from Oregon State University, a Master of Public Health (MPH) in epidemiology and biostatistics, also from Oregon State University, a master’s degree in forensic medical sciences (MScFMS) with the Academy of Forensic Medical Sciences in the United Kingdom, i.a. In addition to my degreed education, I have completed a 2-year post-doctoral fellowship in forensic pathology at Umeå University in Sweden and hold a Diploma of Legal Medicine (DLM) with the FFLM. I am also a fellow of both the American Academy of Forensic Sciences and the American College of Epidemiology. I am a Fulbright Fellow and held a 3-year roster appointment (2017-20) with the United States Department of State as a Fulbright Specialist in the field of forensic medicine. I serve as tenured Associate Professor of Forensic Medicine at Maastricht University and a joint Clinical Professor of Psychiatry and Public Health and Preventative Medicine at Oregon Health and Science University School of Medicine, where I have taught courses for the past 24 years in forensic medicine, forensic epidemiology, and injury epidemiology. From 2005-2017 I held an appointment as an Adjunct Professor of Forensic Medicine and Epidemiology at the Institute of Forensic Medicine, Faculty of Health Sciences, Aarhus University, Aarhus, Denmark, and am a recent (2020-21) visiting professor at University of Indonesia in the Faculty of Medicine.

I have been a crash reconstructionist since 1996 and have had ACTAR accreditation (the Accreditation Commission on Traffic Accident Reconstruction) since 2005. Over the past >25 years I have participated in the reconstruction of more than 3,000 crashes, including more than 300 fatalities. From 1999 through 2007 I served as a vehicular homicide investigator for law enforcement (consultant to the state medical examiner and special deputy sheriff), and I am a former affiliate medical examiner with the Allegheny County Medical Examiner’s office.

I am a member of the American Society of Biomechanics and have more than 60 scientific publications pertaining to injury biomechanics, including a book for the Society of Automotive Engineering and taught injury biomechanics in a faculty peer-reviewed course at OHSU for 15 years. I have served as a consultant on injury biomechanics to state and federal government.

I am an associate editor of the Journal of Forensic and Legal Medicine and serve or have served as an associate editor or editorial board member of 14 additional scientific peer-reviewed journals. I have published approximately 230 scientific papers, abstracts, book chapters and books on topics that include traffic crash injuries, crash reconstruction, injury causation and injury biomechanics, including the text for Elsevier, Forensic Epidemiology: Principles and Practice (2016). My publications have been cited by other authors more than 4,700 times.

I have provided testimony in more than 400 civil and criminal trials in state and Federal courts throughout the United States, Canada, and Australia. Please see my CV for further details.

At the time of the crash,

***Injury Causation Analysis***

A crash-related injury causation analysis for a specific individual is performed by assessing the risk of injury from the collision and comparing it to the probability that the injuries or conditions would have been present at the same point in time if the collision had not occurred. The process is referred to as a "3-step" injury causation method in which improbable alternative causes are ruled out and the single most likely cause is identified. The analysis is accomplished via the application of crash reconstruction, biomechanical, medical, and epidemiologic (risk assessment) principles.[[1]](#footnote-1),[[2]](#footnote-2) This 3-step methodology has been extensively described in the peer-reviewed literature, been deemed generally accepted by Courts in the United States, and has been adopted as part of case law in the U.S.[[3]](#footnote-3),[[4]](#footnote-4) See the Appendix at the end of this report for more information.

The three fundamental elements or steps of an injury causation analysis are as follows: Whether the injury mechanism had the potential to cause the injury in question (aka general causation);

The degree of temporal proximity between the injury mechanism and the onset of the symptoms reasonably indicating the presence of the injury; and

Whether there is a more likely alternative explanation for the occurrence of the symptoms at the same point in time (aka differential etiology).

As applied to the facts in the subject case, these 3 steps are as follows:

*Reconstruction of the crash*

*Injury biomechanics*

The driver’s-side impact would have resulted in the PlCarMake’s occupants to initially move to the left and somewhat forward at 10 mph, until Ms. and Mr. Pl1LastName and Mx. Third Name struck the center console and passenger door with their lower extremities and were restrained by their seatbelts which would have caused their heads to continue to accelerate left and forward and create high bending loads on the joints and disks of their necks, combined with sudden muscular protective forces which would have produced high levels of compression on the spinal disks in the neck and back.[[5]](#footnote-5) Per their description, Ms. and Mr. Pl1LastName and Mx. Third Name then rebounded back to their right and struck the passenger side door and window. This all would have taken less time than it takes to blink an eye (around 250 msecs).

Although a 10 mph side impact collision does not sound like a particularly severe crash, such collisions have the potential to produce relatively violent occupant movements. This fact is illustrated in the photographic stills on the following pages, which show the sequential occupant kinematics of a healthy crash test volunteer undergoing a 3.1 mph far-side impact delta V, with forces that are around 40% of the subject crash.[[6]](#footnote-6)



Figure 1: The bullet vehicle is approaching at 7.5 mph.



Figure 3: The target vehicle is starting to rotate, and the center of mass of the vehicle is accelerated to 3.1 mph (this is the delta V). The occupant begins to move to the right, relative to the vehicle interior.



Figure 4: The occupant’s head and torso flexes violently to the right as the vehicle continues to rotate from under her.



Figure 5: Inertial forces on the occupant’s head and torso continue to cause flexion to the right as the vehicle continues to rotate, with the impact to final frame above occurring over approximately 1/5th of a second. The subject experiences compression, rotation, and shear in the low back and neck.

*Discussion*

The types of spinal injuries that Ms. and Mr. Pl1LastName and Mx. Third Name was diagnosed with (primarily symptomatic disk derangements and associated sequelae) are highly consistent with the injury mechanism of the crash. Traumatic loading of the spine that results in axial (up and down) compression, particularly in combination with the other load types occurring with the subject collision, has the potential to damage the peripheral disk annulus, which surrounds and holds in the disk nucleus. Women in their early 3rd decade, like Ms. and Mr. Pl1LastName and Mx. Third Name (who was 12 at the time of the crash) typically have d age-related degenerative changes of the disks of the spine, a fact that makes the collision more likely to have "converted" at least some asymptomatic disk degeneration to a symptomatic state, rather than being the sole cause of all of the pathology identified in the post-crash imaging.

The symptoms of spinal disk injury may, in some cases, be instantly recognizable after a traffic crash because of the sudden onset of radiculopathy, but recent research has demonstrated that only about 1 in 17 cervical disk injuries are recognized as such in the ED after a crash.[[7]](#footnote-7) By far, the majority (94%) of what are later determined to be spinal disk injuries are initially diagnosed as in the ED as spinal strains.

Although the subject crash was no "bumper tap" it is well established in biomechanics, medicine, and epidemiology that an excessive level of force is not required to cause symptomatic injury to a spinal disk with any degree of degeneration, and that in most cases, the diagnostic imaging of the disk will not reveal whether related symptoms are of a traumatic origin or not, in the absence of fracture.[[8]](#footnote-8) Traumatic disk injuries have been described in the peer-reviewed literature as resulting from low to moderate force events, including minimal or no damage traffic crashes, roller coaster rides, and even more mild forces such as sneezing.[[9]](#footnote-9)-[[10]](#footnote-10)[[11]](#footnote-11)[[12]](#footnote-12)[[13]](#footnote-13)[[14]](#footnote-14)[[15]](#footnote-15) It is accurate to state, both from a biomechanical and epidemiological perspective, that there is no established or generally accepted lower force threshold at which it can be said that an acute intervertebral disk injury in any part of the spine cannot occur, and that the load threshold at which individual’s disk may injured is only known after the injury has occurred, and the external load has been estimated. it is impossible to estimate the additional compression due to internal forces, however, and the precise load associated with a temporally proximate spinal disk injury is often impossible to accurately determine after the fact.

Based on the preceding discussion there was ample and biomechanically appropriate force exerted on Ms. and Mr. Pl1LastName and Mx. Third Name’s body in the subject collision to have caused their medically documented injuries, and associated need for evaluation and treatment, including their spinal pain management procedures and cervical spine surgery.

**Temporal relationship between the crash and symptoms indicative of injury**

The second step of the injury causation analysis is the assessment of the timing between the trauma and the onset of symptoms indicative of injury. The hallmark of injury is that "you know it when it happens;" *i.e.* the causal relationship between the trauma and onset of the symptoms indicative of the injury is usually close enough in time that it is easy to recognize when the injury was incurred. There are some injuries that exhibit a delayed onset of symptoms, including injuries to intervertebral disks, which can first manifest with identical symptoms to a simple spinal strain. Other injuries are always immediately apparent, such as bony fractures. Some injuries are considered "distracting" from others because the pain they generate distracts attention from other, less painful injuries. There are still other injuries that can "mask" pain from nearby parts of the body through complicated pain accommodation mechanisms that are mediated at the spinal cord and in the brain. The assessment of the timing of the onset of symptoms reasonably attributed to an injury can sometimes be nuanced and complicated, and sometimes requires expert assessment.

Ms. Pl1LastName experienced the onset of symptoms indicative of acute spinal injury immediately after the crash, and he was diagnosed with neck and back injuries directly by 3 days following the crash, including the presentation of lower extremity radicular symptoms, an ominous early sign of injury to the intervertebral disks in the spine. The symptoms and diagnoses progressively evolved and worsened over the weeks and months following the crash and demonstrate a well-documented contiguous chain of causation linking the subject crash and their low back and cervical spinal injuries, and associated need for treatment.

Based on this history, there was a strong temporal relationship between the subject collision and Ms. and Mr. Pl1LastName and Mx. Third Name’s first development of symptoms indicative of spinal injury.

**Alternative explanations**

This last step of the injury causation analysis specific to the individual involves the assessment of the probability of the same symptoms, injuries, diagnoses, and need for treatment occurring at the same point in time, but in the absence of the investigated crash.

This part of the analysis is accomplished in 2 ways; first and most obviously, any competing contemporaneous traumatic cause of injury must be ruled out. There is, however, no such history for Ms. or Mr. Pl1LastName or Mx. Third Name, and the only source of trauma apparent in my review of materials that is temporally proximate to their post-crash injuries is the 01/01/2020 rear impact crash.

The second part of the analysis requires an assessment of the probability that Ms. or Mr. Pl1LastName or Mx. Third Name would have developed the symptoms and diagnoses of spine injury at the same point in time had they not been initially injured in the 01/01/2020 rear impact crash. As a generally healthy 12-year-old man, Mr. SAMPLE-P1-LN was at a negligible annual risk of spontaneously developing chronically painful and potentially surgical cervical or lumbar spinal disk derangements; less than 1 in 2,000 is indicated by epidemiologic study and national hospital data.[[16]](#footnote-16) The chance that they were going to develop the onset of chronic neck or low back pain attributable to symptomatic disk derangements on the same day as the crash by pure coincidence is obviously much smaller; less than 1 in 730,000 (the annual risk divided by 365 days). In comparison the risk of acute spine injury in the subject crash was more than 50%, and the risk of a spinal disk injury was likely no less than 1 in 25 in the general population. Based on this comparison of risk during the timeframe of interest, the crash is the most likely cause of Mr. SAMPLE-P1-LN and Mr. SAMPLE-P2-LN’s chronic spinal injuries by >99%.

A relatively common medicolegal question is whether persisting symptoms of back or neck pain after a traffic crash are truly related to the crash, or more probably due to other factors incidental to an acute crash-related injury to the spine. Most typically, the "other factors" that are raised in a medicolegal setting are the presence of pre-existing degenerative changes in the spine, or pre-crash history of low level or sporadic symptoms.

The answer to this question is now decided science. Along with my research colleagues, I have recently published 2 analyses and systematic reviews of nearly 10,000 articles regarding the long-term effects of traffic crash-related injury to the neck and low back and associated chronic pain. , Systematic reviews are considered the most reliable and strongest form of scientific evidence (i.e. Level I evidence), as they consist of a comprehensive synthesis of world literature on a topic. The result of the analyses indicate that when men like Mr. SAMPLE-P1-LN and men like Mr. SAMPLE-P2-LN have persisting neck or back pain after an acute crash-related injury, *more often than not* (>50% of the time) the symptoms are due to the crash, versus all other causes, regardless of pre-crash medical history.

The preceding opinions were given as reasonable medical, and scientific probabilities. I reserve the right to amend any of my opinions should new information come to light.

Very truly yours,



Michael D. Freeman, MedDr, PhD, MScFMS, MPH, FRCPath, FFFLM, FACE, DLM

**David Jenkins Memorial Professor and Chair in Forensic and Legal Medicine**  
Faculty of Forensic and Legal Medicine, Royal College of Physicians (London, UK)  
  
**Associate Professor of Forensic Medicine,**  
Care and Primary Healthcare Research Institute, Faculty of Health, Medicine, and Life Sciences, Maastricht University, Maastricht, Netherlands  
  
**Clinical Professor of Forensic Psychiatry**  
Department of Psychiatry, School of Medicine, Oregon Health & Science University  
  
Fellow, Royal College of Pathologists (UK)  
Fellow, Faculty of Forensic and Legal Medicine, Royal College of Physicians (London, UK)  
Fellow, American College of Epidemiology  
Member, American Society of Biomechanics

1. Melia P et al. Development of the INFERENCE (INtegration of Forensic Epidemiology and the Rigorous EvaluatioN of Causation Elements) approach to causal inference in forensic medicine. Int J Environ Res Public Health 2020;17:8353; doi:10.3390/ijerph17228353. [↑](#footnote-ref-1)
2. Freeman MD. A practicable and systematic approach to medicolegal causation. Orthopedics 2018;41(2):70-2. [↑](#footnote-ref-2)
3. Freeman MD, Centeno CJ, Kohles SS. A systematic approach to clinical determinations of causation in symptomatic spinal disc injury following motor vehicle crash trauma. PM R 2009;1(10):951-6. [↑](#footnote-ref-3)
4. Etherton v. Owner Insurance Company. U.S. District Court of Appeals, 10th Circuit. Case No. 14-1164. [↑](#footnote-ref-4)
5. Adams M et al. Biomechanics of back pain. London, UK, Churchill Livingstone, 2012. [↑](#footnote-ref-5)
6. I was involved with the experimental protocols associated with the crash testing depicted in the proceding stills and can authenticate the results. [↑](#footnote-ref-6)
7. Freeman MD, Leith WM. Estimating the number of traffic crash-related cervical spine injuries in the United States; an analysis and comparison of national crash and hospital data. Accident Analysis and Prevention 2020: doi:https://doi.org/10.1016/j.aap.2020.105571. [↑](#footnote-ref-7)
8. Fardon et al. Lumbar disc nomenclature: version 2.0: Recommendations of the combined task forces of the North American Spine Society, the American Society of Spine Radiology and the American Society of Neuroradiology. Spine J. 2014;14(11):2525-45. [↑](#footnote-ref-8)
9. Giuliano et al. The use of flexion and extension MR in the evaluation of cervical spine trauma: initial experience in 100 trauma patients compared with 100 normal subjects. Emerg Radiol. 2002;9(5):249-53. [↑](#footnote-ref-9)
10. Freeman et al. Significant spinal injury resulting from low-level accelerations: A case series of roller coaster injuries. Arch Phys Med Rehab 2005;86:2126-30. [↑](#footnote-ref-10)
11. Lutz et al. CT myelography of a fragment of a lumbar disk sequestered posterior to the thecal sac. Am J Neuroradiol 1990;11(3):610-1. [↑](#footnote-ref-11)
12. Sadanand et al. Sudden quadriplegia after acute cervical disc herniation. Can J Neurol Sci 2005;32(3):356-8. [↑](#footnote-ref-12)
13. Pappas et al. Outcome analysis in 654 surgically treated lumbar disc herniations. Neurosurgery 1992;30(6):862–6. [↑](#footnote-ref-13)
14. Smith J. An analysis of 72 real world impacts - an initial investigation into injury and complaint factors. SAE Technical Paper 1999-01-0640. [↑](#footnote-ref-14)
15. Freeman MD. Medicolegal causation analysis of a lumbar spine fracture following a low speed rear impact traffic crash. J Case Rep Prac 2015; 3(2): 23-9. [↑](#footnote-ref-15)
16. Nolet et al. Exposure to a Motor Vehicle Collision and the Risk of Future Neck Pain: A Systematic Review and Meta-Analysis. PM R. 2019 Apr 25. doi: 10.1002/pmrj.12173. [↑](#footnote-ref-16)