UNITED STATES DISTRICT COURT
DISTRICT OF MINNESOTA

IN RE NATIONAL HOCKEY PLAYERS' CONCUSSION INJURY LITIGATION The Document Relates to: ALL ACTIONS	MDL No. 14-2551 (SRN/JSM) DECLARATION OF THOMAS BLAINE HOSHIZAKI, PH.D

TABLE OF CONTENTS

I. QUALIFICATIONS

1. My name is Dr. Thomas Blaine Hoshizaki, and I am a Professor of Biomechanics at the School of Human Kinetics specializing in research involving head injuries from direct impacts to the head. I am primarily interested in head injury in sport and am considered an expert in research involving head injury reconstruction.

2. In 1978, I earned a Ph.D. in Exercise Physiology from the University of Illinois, Urbana, Champaign. I further earned a M.A. in Exercise Physiology from the University of South Alabama in 1975, and a Bachelor of Physical Education from the University of Calgary in 1974.

3. I am the founder and current Director of the Neurotrauma Impact Science Laboratory at the University of Ottawa, and have worked as a biomechanist, researcher, and professor of physical education for 38 years.

4. I have worked as a biomechanical consultant in the sporting goods industry from 2002 through 2004, was the Vice President of Research and Development for Canstar Sports Group, Inc. from 1989 through 1995, and worked an additional two years as a research consultant for Canstar and Nike, Inc. from 1995 through 1997.

5. I have published over 55 scientific articles in refereed journals and 165 presentations at scientific conferences involving head injuries in sport. I sit on ASTM, CSA, NOCSAE and ISO standards committees for helmet certification as well as a number of concussion committees.

6. In 2012, *The Globe and Mail* named me as one of the 50 Most Influential People in Canadian Sport due to my contributions to the research of head injuries in sport.

7. I have served as an expert witness on the topic of traumatic head injuries in the following cases: *Moore, et al. v. Bertuzzi, et al.*, 06-CV-306081 (2014 ONSC 1318) (for plaintiff Steve Moore); *Martin Szpiro v. Michel-Anthony*

Gigante, et al. (2014) (for defendant Michel-Anthony); *Albinati v. Bell* (2006) (for defendant Bell).

8. Attached as **Exhibit 1** is my most recent Curriculum Vitae, which includes a comprehensive list of my publications in the last ten years and a list of all civil cases in the last four years in which I testified as an expert at deposition or trial. Attached as **Exhibit 2** is a list of materials reviewed in preparing this declaration.

9. I have been retained as a consultant and potential expert witness by plaintiffs' counsel for the action presently pending in United States District Court for the District of Minnesota entitled *In re National Hockey League Players' Concussion Injury Litigation*, Case No. 14-2551. I bill at a rate of $400/hour for my services, and a total of $10,580.36 has been billed as of the date of this declaration.

II. SCOPE OF OPINIONS

10. I was asked to provide an opinion concerning brain trauma characteristics associated with head impacts experienced in the National Hockey League ("NHL"). Specifically, I was asked to opine on i) the biomechanical forces involved in a concussive or subconcussive blow, ii) the effects of concussive or subconcussive blows on the brain, iii) the types of blows to the head incurred by NHL players in an average season during four different decades, and iv) the level of maximum principle strain ("MPS") NHL players were exposed to during an average season. My opinion will be formed using a combination of the scientific literature involving head impacts and brain trauma in general and more specifically papers relating to head trauma in ice hockey, as well as through testing and video analysis.

III. SUMMARY OF OPINIONS

11. Concussive and subconcussive blows can cause damage to the brain through either linear (measured with g (9.8 m/s^2)) or rotational acceleration

(measured with radians per second^2), and most impacts to the head involve a combination of both accelerations. Measurement of the combined deformation forces on brain tissue, measured as MPS, most accurately reflects the biomechanical impact forces exerted on the brain. A MPS figure of 21% has been reported as representing a 50% likelihood of resulting in a diagnosed concussion for professional football players.

12. Yuen and colleagues (2009) used cell cultures to show that a 5% strain was the minimum level of injury required to induce a calcium influx and interestingly observed a significant increase in calcium when two sub-threshold injuries were repeated within 24 hrs. Immediately following a concussive or sub-concussive impact, acute changes described by indiscriminant presynaptic release of neurotransmitters that activate postsynaptic receptors, resulting in an efflux of potassium (K+), sodium (Na+) and calcium (Ca2+). This engages the Na+/K+ ATPase pumps and consumes ATP to re-establish the ionic balance which depletes the cellular energy stores. This initial response is followed by a prolonged glucose metabolic depression that can last up to ten days. Further, it is generally accepted that, following a concussive or sub-concussive impact, permanent cellular and subcellular changes occur in the axons of the brain in the form of permanent white matter cell loss which may increase the speed at which neuropathologies develop.

13. A total of 4630 head impacts were observed for 120 NHL games, 30 games from each of the 1986/87, 1995/96, 2003/04 and 2013/14 seasons. 680 impacts for 1986/87, 844 impacts for 1995/96, 1432 impacts for 2003/04 and 1674 impacts for 2013/14. The number of impacts per game for 1986/87 was 23, 1995/96 was 28, 2003/04 was 48, and for the 2013/14 season, 56 impacts as a conservative estimate. The most common impact involved head to glass impacts (40%) followed by punches to the head (23%), shoulder impacts (11%), elbow impacts (10%) with head to head impacts (8%), head to ice impacts (2%) and puck impacts to the head (0.7%).

14. The highest recorded MPS was for high velocity falls to the ice (79% MPS) this was followed by impacts to the boards (43% MPS), punches (41% MPS), elbow to the head (39% MPS), head to head and head to glass impacts (31% MPS) and shoulder to head impacts (27% MPS). The lowest MPS figure recorded was 9% MPS for a low velocity punch to the head in the 1986/87 season. The results of my video analysis and lab recreation indicated that the average NHL player would experience an impact greater than 9% MPS once (1.19) a game for 1986/87, more than once (1.48) for 1995/96, over twice a game (2.51) for 2003/04, and nearly three times per game (2.95) for 2013/14.

IV. THE BIOMECHANICS OF HEAD IMPACTS

A. Detecting and Diagnosing Concussion

15. Concussions by definition are described by a wide range of signs and symptoms including, but not limited to, decreased cognitive function, balance, vision issues, and inappropriate behavior. Head impact events resulting in concussive injuries are varied and not easily described by simple event dynamics. Concussions have been known in the medical community and scientific literature for centuries. While the term "concussion" was primarily used to describe the loss of consciousness following a direct impact to the head, subsequent research involving head injuries often identified the amount of force to create a skull fracture as representative of concussion. What followed involved describing the relationship between inter-cranial pressure and loss of consciousness and ultimately the search for an algorithm employing dynamic response variables to predict the risk and severity of a concussion.

16. More recently, finite element models of the brain and skull were employed to calculate MPS, von Mises stress, as well as a host of other mechanical variables reflecting the effect of the impact on brain tissue.

1. Causes of Concussion in NHL Hockey

17. Brain trauma associated with injury in hockey include skull fractures, intracranial bleeds, subdural bleed, persistent concussions, transient concussions, and subconcussive injuries. Skull fractures, intracranial bleeds, and subdural bleeds are effectively diagnosed and for the most part are well managed in ice hockey using rules of play and certified equipment. Concussive injuries are diagnosed using signs and symptoms requiring specialized skills and treatment, and have not been historically managed properly in ice hockey.[1]

18. Brain trauma in ice hockey is the result of a variety of events, including body to body collisions, falls to the ice, falls to the boards, collisions with the glass, punches, stick to head impacts, and impacts from pucks. All of these examples of head trauma are represented in ice hockey at the NHL level. Head trauma in this declaration will primarily describe events that create levels of brain tissue trauma associated with changes to the molecular structure of neuronal cells, neuronal cell damage, and changes to the semipermeable membrane upsetting the ionic balance of the cell presenting as concussions.

19. As discussed below, brain trauma in ice hockey has historically been largely defined by diagnosed concussions and described as resulting from a variety of impact events, including shoulder to head, elbow to head, punches to the head, head to ice, head to the boards and glass and pucks to the head. Body checking,

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[1] C.A. Emery & W.H. Meeuwisse, *Injury Rates, Risk Factors and Mechanisms of Injury in Minor Hockey*, 34 AM. J. SPORTS MED. 12, 1960-69 (2006). *See also* S.G. Gerberich et al., *An epidemiological study of high school ice hockey injuries*, 3 CHILDS NERV. SYST. 2, 59-64 (1987); M.G. Hutchison, *Concussion in the National Hockey League (NHL): The video analysis project*, UNIV. OF TORONTO (Jan. 9, 2012), https://tspace.library.utoronto.ca/handle/1807/31786; M. Hutchison & P. Comper, *Concussions in National Hockey League (NHL) players: 5-year video analysis*, 47 BR. J. SPORTS MED. 5 (2013); A.C. Bain & D.F. Meaney, *Tissue-level thresholds for axonal damage in an experimental model of central nervous system white matter injury*, 122 J. BIOMECH. ENG. 6, 615-22 (2000).

defined as a defensive skill designed to separate a player from the puck using the hip or body as a point of contact, was reported as the primary cause of concussion in ice hockey.[2] While players are taught to body check without making contact to the opponent's head, 68% of concussions occurring in the NHL between 2006 and 2009 were attributed to body-to-head impacts.[3] The analysis also revealed that the shoulder (68%) and elbow (23%) were the body parts that came most often into contact with an opponent's head, resulting in a concussion. Benson *et al.*[4] reported that player to player contact represented 49% of reported concussions, 34% attributed to contact with the glass, boards or net, leaving only 5% representing head to ice contact. Hutchison *et al.*[5] reported body contact represented 88% of concussive injuries, fighting 8%, and falling to the ice 7% for NHL hockey players.

20. Specific to NHL hockey, Rousseau *et al.*[6] reported no significant differences between peak linear acceleration for concussions when compared to non-concussions from shoulder impacts in hockey. However, he reported significantly greater peak rotational acceleration for concussions when compared to non-concussions from shoulder impacts in hockey. Impacts using an extended elbow resulted in significantly higher peak linear and angular acceleration when

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2 *Id.*

3 Hutchison, *supra* n. 1.

4 B.W. Benson et al., *A prospective study of concussions among National Hockey League players during regular season games: the NHL-NHLPA Concussion Program*, 183 CAN. MED. ASS'N. J. 8, 905-11 (2011).

5 M.G. Hutchison et al., *A systematic video analysis of National Hockey League (NHL) concussions, part I: who, when, where and what?*, 49 BR. J. SPORTS MED. 8, 547-51 (2015).

6 P. Rousseau & T.B. Hoshizaki, *Defining the effective impact mass of elbow and shoulder strikes in ice hockey*, 14 SPORTS BIOMECH. 1, 57-67 (2015).

comparing impacts resulting in concussion with those that did not result in concussions.

21. This research reported several important findings – the compliance or softness describing the shoulder and elbow impacts in ice hockey was high, resulting in substantially longer impact durations and lower peak linear and angular accelerations resulted in concussive impacts. The longer the acceleration event, the lower peak magnitude required to create dangerous MPS values. The reported differences for MPS (finite element modeling) was 18 - 30 % MPS for diagnosed concussion impacts, and 15 - 25% MPS for non-concussed (undiagnosed) or subconcussive impacts. This, in part, was event-specific, with the highest values for shoulder impacts and the lowest for tucked in elbow impacts to the head. The head impacts that were not diagnosed as concussions nevertheless reported high levels of strain on the brain.

2. Using Maximum Principle Strain to Identify Concussion

22. Several variables have been used to measure brain tissue trauma resulting from a head impact, with MPS in the grey matter being the most commonly used metric. A number of authors have reported a MPS of between 19 - 26 % corresponding to a 50% risk of diagnosed concussion.[7] Concussive

[7] S. Kleiven, *Predictors for traumatic brain injuries evaluated through accident reconstructions*, 51 STAPP CAR CRASH J., 81-114 (2007). *See also* L. Zhang et al., *A proposed injury threshold for mild traumatic brain injury*, 126 J. BIOMECH. ENG. 2, 226-36 (2004); R.A. Oeur et al., *A comparison of head dynamic response and brain tissue stress and strain using accident reconstructions for concussion, concussion with persistent postconcussive symptoms, and subdural hematoma*, 123 J. NEUROSURG. 2, 415-22 (2015); D.C. Viano et al., *Concussion in professional football: comparison with boxing head impacts—part 10*, 57 NEUROSURGY 6, 1154-72 (2005); D.A. Patton et al., *Injury data from unhelmeted football head impacts evaluated against critical strain tolerance curves*, 226 J. SPORTS ENG. AND TECH. 3-4, 177-84 (2012).

impacts reconstructed by Rousseau have reported MPS figures as low as 14% for diagnosed concussions involving body collisions in hockey.[8]

23. Rousseau's results are similar to MPS values reported in the literature for concussions experienced in other sports. These results are consistent with anatomical studies by Galbrath *et al.* who reported that axons experiencing greater than 12% MPS for a period of 14 ms lost function temporarily, but impacts over 20% MPS resulted in axons permanently failing to return to their resting potential.[9] At levels over 25% strain, the axon failed structurally.[10] Bain and Meaney reported functional strain thresholds of 13%, 18%, and 28% as conservative, liberal, and optimal thresholds, respectively, for axonal structural damage (guinea pig optic nerve).[11] Research employing finite element models to reconstruct concussive impacts have reported between 19 to 26% MPS as 50% risk for concussions.[12]

24. Also important is the fact that there may be a risk in relying solely on the presence of symptoms as a way of identifying the point at which brain trauma becomes dangerous and risks to head injury are present, since relying on symptoms alone leads to underrepresentation of risks. Yuen *et al.* used cell cultures to show that a 5% strain was the minimum level of injury required to induce a destructive calcium influx (as explored *infra* Section IV.B) and interestingly observed a significant increase in calcium when two sub-threshold

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[8] P. Rousseau, *Analysis of concussion metrics of real-world concussive and non-injurious elbow and shoulder to head collisions in ice hockey*, DISS. UNIVERSITÉ D'OTTAWA/UNIV. OF OTTAWA, (2014).

[9] J.A. Galbraith et al., *Mechanical and electrical responses of the squid giant axon to simple elongation*, 115 J. BIOMECH. ENG. 1, 13-22 (1993).

[10] *Id.*

[11] Bain & Meaney, *supra* n.1.

[12] *Infra* n.35.

injuries were repeated within 24 hrs.[13] Thus, even comparatively low levels of MPS are important to analysis of the effects of head impacts and subsequent injury risk.

25. Fundamentally, the comparatively heightened level of precision in MPS figures lends itself most effectively to the study of concussion frequency. Unfortunately, much of the study of concussion frequency in the NHL, by both the NHL and independent researchers, relies primarily on the reporting of concussions based on the presence of reported symptoms.

3. Reported Numbers of Concussions in the NHL

26. While concussions defined by signs and symptoms are a reported injury in professional ice hockey and can be used to represent a general estimate of the level of head trauma, they are underreported in professional sports generally, including ice hockey.[14] Athletes who have been concussed often do not recognize the signs or suffer delayed onset of symptoms, or, if they do recognize the signs, for a variety of reasons they choose not to report concussions. Therefore, the rate of ***reported*** concussions is considered to be well below the rate of ***actual*** concussion rates in professional sports.

27. Given this limitation, Benson *et al.* reported 559 concussions over seven years (1997-2004) in the NHL, averaging 80 concussions per season, resulting in an estimated 1.8 concussions per 1000 player-hours.[15] Hutchison *et al.* reported 197 medically diagnosed concussions for regular season NHL games,

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[13] T. Yuen et al., *Sodium channelopathy induced by mild axonal trauma worsens outcome after a repeat injury,* 87 J. NEUROSCI. RES. 16, 3620-25 (2009).

[14] P.S. Echlin et al., *A prospective study of physician-observed concussions during junior ice hockey: implications for incidence rates*, 29 NEUROSURG. FOCUS 5 (2010).

[15] Benson et al., *supra* n.4.

or 56 concussions per season, in the period of 2006-2010.[16] Donaldson *et al.* reported 44 concussions and 24 suspected concussions for the 2009-2010 NHL season, 65 concussions and 42 suspected concussions for the 2010-11 NHL season, and 84 concussions and 36 suspected concussions for the 2010-11 NHL season.[17] They reported that the majority of concussions were the result of legal plays. The most common penalty assessed for a concussion event was for fighting.[18] For comparison, Nathanson *et al.* reported 6.61 concussions per 1000 athletic exposures in the 2012 and 2013 NFL seasons.[19]

28. Data documenting diagnosed concussion injuries provided by the NHL for this case listed an average of 108 concussions for each year over the 2006-07 (80), 2007-08 (85), 2008-09 (81), 2009-10 (102), 2010-11 (148), 2011-12 (137), and 2013-14 (123) seasons for a total of 756 diagnosed concussions.[20] [redacted]

29. Combining all documented NHL reported concussions provides the following figures: 1997-98 (56), 1998-99 (88), 1999-2000 (66), 2000-01 (109),

[16] M.G. Hutchison et al., *A systematic video analysis of National Hockey League (NHL) concussions, part 1: who, when, where and what?*, BR. J. SPORTS MED. (2013).

[17] L. Donaldson et al., *Bodychecking Rules and Concussion in Elite Hockey*, 8 PLOS ONE 7 (2013).

[18] However, it is reported that fighting only accounts for a small percentage of reported concussions in the NHL.

[19] J.T. Nathanson et al., *Concussion Incidence in Professional Football: Position-Specific Analysis With Use of a Novel Metric*, 27 ORTHOP. J. SPORTS MED. 4, (2016).

[20] This excludes the shortened season 2012-13.

[21] NHL-2502169.

2001-02 (96), 2002-03 (72), and 2003-04 (72);[22] 2006-07 (80), 2007-08 (85), 2008-09 (81), 2009-10 (102), 2010-11 (148), 2011-12 (137), and 2013-14 (123) averages to 93.9 concussions per season over 14 seasons. Each of the 30 teams in the NHL would average a reported 3.1 concussions each year with this data.

30. Delany *et al.*[23] investigated the level of unreported concussions of university varsity athletes to be as high as 78.3%, with football and hockey athletes the least likely to report a suspected concussion. It is logically expected that athletes with more at risk of losing their place on a team would be more likely to not report a suspected concussion. Among the reasons for failing to report a concussion, the reason "did not feel the concussion was serious/severe and felt you could still continue to play with little danger to yourself" was listed most commonly (55/92) as a cause for not seeking medical attention for a presumed concussion.[24] This was also reported by Benson *et al.*[25] as the possible reason for lower than expected concussions in the NHL: "Possible explanations for this finding include the following: more conservative management or return-to-play decisions by physicians; increase in the NHL in the severity of concussions (i.e., longer time to complete resolution of post-concussion symptoms); failure by players to report symptoms for fear of being held out of games; reporting by players of only severe symptoms; use by NHL team physicians of higher thresholds for diagnosis of concussion; or increasing use of neuropsychological

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[22] Benson et al., *supra* n.4.

[23] J.S. Delaney et al., *Why University Athletes Choose Not to Reveal Their Concussion Symptoms During a Practice or Game*, 25 CLIN. J. SPORT MED. 2, 113-25 (2015).

[24] *Id.*; J.K. Register-Mihalik et al., *Knowledge, attitude, and concussion-reporting behaviors among high school athletes: a preliminary study*, 48 J. ATHL. TRAIN. 5, 645-53 (2013); Benson et al., *supra* n.4.

[25] Benson et al., *supra* n.4.

testing results before making return-to-play decisions" for NHL ice hockey players.

B. Cellular and Subcellular Changes Following a Concussive or Subconcussive Impact

31. Relying on observable symptoms as a way to diagnose and monitor concussion recovery may not be prudent, as axonal degeneration and functional impairment could be present and remain despite cognitive recovery.[26] Therefore, current methods of defining and diagnosing concussions through the presence of symptoms are likely providing an incomplete representation of brain trauma exposure, and risk to recurrent injury. Further, impacts that do not present with signs and symptoms for concussion may be contributing to trauma induced protein changes ("TIPC"), with the potential to lead to structural injury and degeneration of neurons.[27]

32. A little over a decade ago a similar neuropathology was observed upon autopsy of a retired National Football League player,[28] and is now widely known as chronic traumatic encephalopathy, or CTE.[29] CTE resulting from TIPC has now been observed in, among other sports, boxing, BMX cycling, American football, ice hockey and rugby. Those who participate in contact sports or

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[26] J.A. Creed et al., *Concussive brain trauma in the mouse results in acute cognitive and sustained impairment of axonal function*, 28 J. NEUROTRAUMA 4, 547-63 (2011).

[27] B.I. Omalu et al., *Chronic traumatic encephalopathy in a National Football League player*, 57 NEUROSURGERY 1, 128-34 (2005); A.C. McKee et al., *Chronic traumatic encephalopathy in athletes: progressive tauopathy after repetitive head injury*, 68 J. NEUROPATHOL. EXP. NEUROL. 7, 709-35 (2009); A.C. McKee et al., *The spectrum of disease in chronic traumatic encephalopathy*, 136 BRAIN 1, 43-64 (2013).

[28] Omalu et al., *supra* n.27.

[29] McKee et al., *supra* n.27.

activities where head trauma has a repetitive nature are at risk of developing CTE or other neuropathologies.[30]

33. Brain tissue trauma resulting from a concussive or subconcussive impact is followed by acute changes, described by indiscriminate presynaptic release of neurotransmitters that activate postsynaptic receptors and resulting in an efflux of potassium (K+), sodium (Na+) and calcium (Ca2+). This engages the Na+/K+ ATPase pumps and consumes ATP to re-establish the ionic balance, which depletes the cellular energy stores.[31] This initial response is followed by a prolonged glucose metabolic depression that can last up to ten days.

34. Trauma induced damage to neurons are primarily associated with rotational accelerations creating shear strains and damaging the white matter of the brain.[32] Finally changes in the molecular structure of cell protein (tau)[33] and

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[30] K.F. Bieniek et al., *Chronic traumatic encephalopathy pathology in a neurodegenerative disorders brain bank*, 130 ACTA. NEUROPATHOL. 6, 877-89 (2014). *See also* A.Kondo et al., *Antibody against early driver of neurodegeneration cis P-tau blocks brain injury and tauopathy*, 523 NATURE 7561, 431-36 (2015).

[31] C.C. Giza, D.A. Hovda, *The neurometabolic cascade of concussion*, 36 J. ATHL. TRAIN 3, 228-35 (2001); P. Mayumi et al., *The pathophysiology of traumatic brain injury at a glance*, 6 DIS MODEL MECH. 6, 1307-15 (2013).

[32] J.J. Bazarian et al., *Subject Specific Changes in Brain White Matter on DTI after Sports-Related Concussion*, 30 MAGNETIC RESONANCE IMAGING 2, 171-80 (2012); J.J. Bazarian et al., *Persistent, Long-term Cerebral White Matter Changes after Sports-Related Repetitive Head Impacts*, 9 PLOS ONE 4 (2014); V.A. Cubon et al., *A diffusion tensor imaging study on the white matter skeleton in individuals with sports-related concussion*, 28 J NEUROTRAUMA 2 (2011); I.K. Koerte, et al., *White Matter Integrity in the Brains of Professional Soccer Players Without a Symptomatic Concussion*, 308 JAMA 18, 1859-61 (2012); T.W. McAllister et al., *Effect of head impacts on diffusivity measures in a cohort of collegiate contact sport athletes*, 8 NEUROLOGY 1, 63-69 (2014).

[33] A. Kondo et al., *Antibody against early driver of neurodegeneration cis P-tau blocks brain injury and tauopathy*, 523 NATURE 7651 (2015); S. Neselius et al., *CSF – Biomarkers in Olympic Boxing: Diagnosis and effects of repetitive head trauma*, 7 PLOS ONE 4 (2012).

damage to the neurofilament structures of the cell resulting from brain tissue trauma have been well documented.[34]

35. It should be noted that injury to brain cells that result in cell death are not replaced, and instead the brain is required to compensate for the decreased function in order to perform its required functions. Individuals possess a certain level of "reserve" cells in excess of the minimum required to function normally, but as cell damage accumulates, at some point the brain is unable to fully compensate for the loss of reserve cells and damaged neurons and mental performance is compromised over time, typically in the form of various neuropathologies such as CTE, Parkinson's disease, early-onset dementia, or ALS.[35]

36. Changes involving white matter structures indicating diffuse axonal injury in the brains of Canadian University competitive hockey players over a season of hockey has been reported by Koerte *et al*.[36] Of note, despite the changes to white matter structure, none of the 25 athletes reported any symptoms associated with concussions for the season studied.

37. In a study comparing American college varsity football players (n=20) with varsity swimming (n=20) athletes for serum neurofilament light polypeptide ("NFLP") the levels of NFLP were higher for starters when compared to the non-starters in varsity football over the course of a season.[37] When

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[34] Neselius et al., *supra* n.33; J.M. Oliver et al., *A longitudinal study examining the effects of a season of American football on lipids and lipoproteins*, 14 LIPIDS IN HEALTH AND DISEASE 1 (2015).

[35] L. Moretti et al., *Cognitive decline in older adults with a history of traumatic brain injury*, 11 LANCET NEUROL. 12 (2012).

[36] Koerte, et al., *supra* n. 32.

[37] S. Neselius et al., *Monitoring concussion in a knocked-out boxer by CSF biomarker analysis*, 23 KNEE SURGERY, SPORTS TRAUMATOLOGY, ARTHROSCOPY 9, 2536-39 (2015); S. Neselius et al., *CSF-biomarkers in Olympic boxing:*

compared to the controls (varsity swimmers), both the starters and the non-starters had elevated levels of serum NFLP. Elevated levels of serum NFLP are indicative of axonal injury as a result of head impacts. These results are consistent with changes in functional magnetic resonance imaging (fMRI), diffuse-tensor imaging (DTI-MRI), and cerebral spinal fluid (CSF) and blood levels of tau protein and NFLP that reflect axonal injury resulting from brain trauma.

38. In a subsequent study using biomarker methodologies, Oliver *et al.* compared serum levels of NFLP in American football players at four measurement points over the course of one season.[38] Starting athletes experienced significant increases in serum NFLP levels throughout the season compared with both non-starters and controls, where changes coincided with an increase in intensity and hours of contact. This supports a relationship between frequency and intensity of exposure, and heightened NFLP.

C. Long-Term Consequences of Head Impacts

39. Using only reported symptoms to characterize risks of CTE or other neuropathologies has critical limitations in measuring overall trauma exposure, particularly with the association between trauma exposure and neurodegeneration attributed to repetitive TIPC. Research involving trauma exposure has proposed that, in addition to the magnitude of the impact, the frequency of the trauma and time interval between impacts to the head may interact to create risks of chronic neurodegeneration.[39]

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diagnosis and effects of repetitive head trauma, 7 PLOS ONE 4 (2012); Neselius, *supra* n. 33.

[38] J.M. Oliver et al., *Effect of Docosahexaenoic Acid on a Biomarker of Head Trauma in American Football*, 48 MED SCI SPORTS EXERC. 6, 974-82 (2016).

[39] Koerte, et al., *supra* n. 32; McKee et al., *supra* n.27.; Bazarian et al. (2014), *supra* n. 32; S.L. Banks et al., *Impulsiveness in professional fighters*, 26 J. NEUROPSYCHIATRY CLI. NEUROSCI. 1, 44-50 (2014); Oliver et al., *supra* n. 34; P.H. Montenigro et al., *Cumulative Head Impact Exposure Predicts Later-Life*

40. Neurological disorders are closely associated with activities involving direct impacts to the head. Under particular conditions, unstable proteins miss-fold or unfold preventing them from forming into their biochemically functional native form. These toxic conformations have the ability to form aggregates creating neurofibrillary tangles and neurofil threads leading to neuronal cell degeneration.[40]

41. There is considerable evidence that shows patients who have suffered a single moderate to severe traumatic brain injury are at an increased risk of developing cognitive impairments from progressive and psychiatric disorders.[41] Neurological consequences of repetitive head impacts date back over a century, and were famously discussed in 1928 when Martland described these risks and neuropsychiatric sequelae from chronic exposure to repeat brain trauma.[42] He termed this 'punch drunk' as a way to describe the chronic symptoms that were being demonstrated by boxers who had sustained multiple hits to the head. A number of published reports on the risks of repeat impact within boxing followed.[43]

Depression, Apathy, Executive Dysfunction, and Cognitive Impairment in Former High School and College Football Players, J. OF NEUROTRAUMA (2016).

[40] M. Stefani, C.M. Dobson, *Protein aggregation and aggregate toxicity: new insights into protein folding, misfolding diseases and biological evolution*, 81 J. Mol. Med. 11, 678-99 (2003); McKee et al., *supra* n. 27.

[41] B.E. Masel, D.S. DeWitt, *Traumatic brain injury: a disease process, not an event*, 27 J. NEUROTRAUMA 8, 1529-40 (2010).

[42] H.S. Martland, *Punch drunk*, 91 Journal of the American Medical Association 15 (1928).

[43] J.A. Millspaugh, *Dementia pugilistica*, 35 U.S. Naval Med. Bull. 297, 297-303 (1937); W. Brandenburg, J. Hallervorden, *Dementia pugilistica with anatomical findings*, VIRCHOWS ARCHIV 325, 680-709 (1954); A.H. Roberts, *Brain damage in boxers*, London, Pitman Publishing (1969); J.A. Corsellis *et al., The aftermath of boxing*, 3 Psychol Med. 3 (1973); M.F. Mendez, *The neuropsychiatric aspects of boxing*, 25 INT. J. PSYCHIATRY MED. 3, 249-62 (1995).

42. Further, Kondo *et al.* have shown similar durations of elevated levels of pathogenic *cis* phosphorylated tau protein in a repeat trauma model to one of a single severe traumatic event.[44] What this indicates is that lower magnitude brain trauma, if repetitive, may show similar pathological outcomes to one higher magnitude traumatic event, suggesting there may be a similar neurological risk associated with the cumulative effect of trauma to neural tissues.[45]

43. A relationship between head trauma and long-term disability has been documented with the level of trauma and injury investigated. Research reporting MPS levels as low as 5-15% are associated with functional impairment of signal transmission and reserve cell loss, even in the absence of structural damage.[46] Reconstructions of head impacts with no reported symptoms experienced by American football lineman during game play show 20-30g head accelerations with strain values of 9-11%.[47] As these athletes are experiencing conceivable estimates of upwards of 1000 head impacts over one season, the cumulative effect of trauma-induced protein changes is important and must be considered.[48]

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[44] Kondo et al., *supra* n. 33.

[45] *Id.*

[46] Yuen et al., *supra* n. 13; Bain et al., *supra* n. 1; K. Zanetti et al., *Identifying risk profiles for three player positions in American football using physical and finite element modeling reconstructions*, PROCEEDINGS OF IRCOBI (2013); B.S. Elkin, B. Morrison 3rd, *Region-specific tolerance criteria for the living brain*, 51 STAPP CAR CRASH J, 127-38 (2007); A. Singh et al., *A new model of traumatic axonal injury to determine the effects of strain and displacement rates*, 50 STAPP CAR CRASH JOURNAL, 601-623 (2006).

[47] Zanetti et al., *supra* n. 47.

[48] Bazarian et al., *supra* n. 32; B. Schnebel et al., *In vivo study of head impacts in football: a comparison of National Collegiate Athletic Association Division I versus high school impacts*, 60 NEUROSURGERY 3, 490–95 (2007); J.J. Crisco et al., *Frequency and location of head impact exposures in individual collegiate*

44. Considering that one who has sustained a concussive injury becomes more susceptible to repeat injury, and that subsequent injuries are often more severe than prior injuries, it may be postulated that when the brain is initially injured and in a state of energy crises, a subsequent lower magnitude impact could potentially have a similar injurious effect, accompanied by varying degrees of cell death.[49] If a second impact is experienced during the window of vulnerability – that of decreased cerebral metabolic rate of glucose – the degree of depressive state (a decrease in cell metabolism) – increases and prolongs the duration of acute symptom recovery.[50]

45. As CTE and other neuropathologies are often not evident until many years post-trauma, recent research has focused, in large part, on ways to identify axonal damage immediately following trauma, in the absence of cognitive signs and symptoms. The physiological response to trauma may be detectable within blood and CSF in the form of elevated levels of biological markers that, over time, may lead to an accumulation of protein tangles and neurofil aggregates.[51] Elevated levels of NFLP and total-tau (T-tau) have been measured in athletes participating in contact sports such as boxing and ice hockey. These observations were made in the blood and CSF of athletes sustaining head impacts resulting in concussion, as well as those not presenting with symptoms. Interestingly, axonal biomarkers remained elevated for longer durations in knocked-out boxers versus

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football players, 45 J. ATHLETIC TRAIN. 6, 549-59 (2010); J.J. Crisco et al., *Head impact exposure in collegiate football players*, 44 J. BIOMECH. 15, 2673-78 (2011).

[49] K.M. Guskiewicz et al., *Cumulative effects associated with recurrent concussion in collegiate football players*, 290 *JAMA* 19, 2549-55 (2003); Giza et al., *supra* n. 31.

[50] *Id.*

[51] A. Petzold, et al., *Glial and axonal body fluid biomarkers are related to infarct volume, severity, and outcome*, 17 J. STROKE CEREBROVASC. DIS. 4, 196-203 (2008); N. Shahani, R. Brandt, *Functions and malfunctions of the tau proteins*, 59 CELL MOL. LIFE SCI. 10, 1668-80 (2002).

both non-knocked-out boxers and concussed ice hockey players.[52] These findings support the theory that trauma frequency and/or time interval contribute to neuronal damage and that damage can be detected in the absence of concussive symptoms, especially where subconcussive impacts are frequent.

46. Similar reports have also supported this relationship using diffusion tensor imaging (DTI) and functional magnetic resonance imaging (fMRI) measures following asymptomatic head trauma.[53] Significant relationships have been reported between neurophysiological changes detected by fMRI and the number of head hits experienced.[54] Moreover, changes in DTI measures worsened in athletes who experienced a higher number of impacts exceeding an estimated rotational acceleration threshold.[55]

47. Characterized as a tauopathy disease, research on how CTE manifests itself has postulated a similar mechanism to a number of other

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[52] Neselius S, Brisby H, Theodorsson A, Blennow K, Zetterberg H, Marcusson, *CSF – Biomarkers in Olympic Boxing: Diagnosis and effects of repetitive head trauma*, 7 PLOS ONE 4 (2012); S. Neselius et al., *Monitoring concussion in a knocked-out boxer by CSF biomarker analysis*, 23 KNEE SURG., SPORTS TRAUMATOL., ARTHROSC. 9 (2014); P. Shahim, et al., *Blood biomarkers for brain injury in concussed professional ice hockey players*, 71 JAMA NEUROLOGY 6, 684-92 (2014); H. Zetterberg, et al., *[Guidelines for brain concussion in sports--how are they to be applied in boxing?]*, 104 LAKARTIDNINGEN 22, 1715 (2006).

[53] V.A. Cubon, et al., *A diffusion tensor imaging study on the white matter skeleton in individuals with sports-related concussion*, 28 J NEUROTRAUMA 2, 189-201 (2011); Bazarian et al., *supra* n. 32; E.L. Breedlove, et al., *Biomechanical correlates of symptomatic and asymptomatic neurophysiological impairment in high school football*, 45 J. OF BIOMECHANICS 7, 1265–72 (2012); Koerte et al., *supra* n. 32.; T.M. Talavage et al., *Functionally-detected cognitive impairment in high school football players without clinically-diagnosed concussion*, 31 J. NEUROTRAUMA 4, 327-38 (2014).

[54] Breedlove et al., *supra* n. 55.

[55] Bazarian et al., *supra* n. 32.

neurodegenerative diseases.[56] It has been proposed that the accumulation of disrupted protein in CTE cases may result as the brain's ability to clear waste from the brain parenchyma is overwhelmed and/or compromised, ultimately leading to cell degeneration and death. Further, as levels of tau protein remain elevated within a chronic state and accumulation of perivascular tau takes place, blood-brain barrier dysfunction results, potentially exacerbating pathology.[57] This is supported by consistent findings indicating the role of the length of an athletic career and the overall amount of exposure to head trauma as significant contributors to behavioral and cognitive abnormalities.[58]

48. A variety of mechanisms and types of trauma-induced brain injuries exist, and what we presently define as a concussion injury represents a level of trauma eliciting functional disturbances in the absence of gross structural damage. However, impacts of lower magnitudes of neuronal tissue trauma, as evidenced through structural change to the white matter and changes in cellular proteins, are putting athletes at long-term risk of neurological disorders. The molecular changes caused by trauma are the precursor to suffering neurocognitive deficits, consistent with neurological degenerative diseases later in life. This is amplified by trauma that causes physiological response. Knowing this, brain trauma risk assessment cannot be limited to concussion and severe traumatic injuries, since

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[56] J.J. Iliff, et al., *Cerebral Arterial Pulsation Drives Paravascular CSF–Interstitial Fluid Exchange in the Murine Brain*, 33 J. NEUROSCI. 46, 18190-99 (2013).

[57] L.J. Blair, et al., *Tau depletion prevents progressive blood-brain barrier damage in a mouse model of tauopathy*, 3 ACTA NEUROPATHOLOGICA COMMS. 1 (2015).

[58] McKee et al., *supra* n.27; Banks et al., *supra* n.39; C. Bernick, et al., *Predicting Low Cognitive Function in Professional Fighters*, 28 BRAIN INJURY 5-6, (2014); J. M. Stamm et al., *Age at First Exposure to Football Is Associated with Altered Corpus Callosum White Matter Microstructure in Former Professional Football Players*, 32 J. OF NEUROTRAUMA 22, 1-9 (2015); Montenigro et. al., *supra* n.39.

they are not the only cause of long-term brain degeneration; TIPC should also be considered and biomarkers should be tracked when attempting to evaluate the brain-injury risk associated with a given sport. Consideration of cumulative brain trauma exposure is warranted when dealing with the long-term brain health of athletes and sports participants.

D. Video Analysis and Lab Recreation of NHL Head Impacts

49. I have been asked to determine the number of head impacts suffered by NHL players in a typical game, and season, and the corresponding MPS figures associated with the head impacts. This analysis was performed for four different decades, from the 1980s through to the present, to assess changes or trends of MPS exposure.

50. Each of the four decades is represented in the analyses presented below; 30 games each for 1986-87, 1995-96, 2003-04 and 2013-14 seasons, with 15 games obtained from the first half of each season, and 15 from the second half of each season for a total of 120 games.[59] Each team was included at least once for the analysis and in some cases a team would have been analyzed more than two times, depending on the match-ups. The analyzed games are listed for each season below.

51. It should be mentioned that the video provided was limited to game coverage and not intended to document all head impacts that occur during a game, as many of the players are not in constant view of the camera.[60] Additionally, players typically participated in as many as four practices each week, which would inevitably produce additional head impacts that would not be recognized.

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[59] *See* **Exhibit 3**.

[60] A Post & T.B. Hoshizaki, *Rotational acceleration, brain tissue strain, and the relationship to concussion,* 137 J. OF BIOMECHANICAL ENGINEERING 3 (2015); Rousseau et al., *supra* n.6; Oeur et al., *supra* n.7.

52. A team of video analyzers was trained to identify head impacts from hockey game video provided by Plaintiffs' counsel, who were provided the video footage from the NHL. The video analyzers were trained to classify each head impact as either low (2.0 – 4.0 m/s), medium (4.0 - 7.0 m/s) or high (>7.0 m/s) velocity impact by being shown videos and discussing them with people who were experienced in this area, until a consensus was reached for the assignment of the level of energy. A second researcher then reviewed the video analysis data and if anomalies were found, the video was reanalyzed.

53. Each head impact was identified as either a head to ice, head to shoulder, head to elbow, head to head, head to boards, head punch, or puck impact to head. This provided a frequency count for head impacts for each game, for each type of impact, and an estimated corresponding MPS. An exemplar event for each impact event, for each impact velocity impact, and for each decade was chosen for impact reconstruction.[61] These exemplar impacts were chosen based on the clarity of the event on the video, and the calibration tools in the frame.

54. A video for each mechanism (boards, elbow, glass, head-to-head, fall on ice, shoulder, puck and punch) and each velocity level (low, medium, high) was chosen, when possible, for a maximum of 24 reconstructions per season. Videos were not analyzed only when no impact of a mechanism and corresponding velocity level were reported throughout a season, or no adequate video of the event could be found.

55. Inclusion criteria for videos were: markings of known distance visible on the ice, the exact moment of impact was visible, the impact location was visible, the mechanism of impact was clearly established, and only one impact occurred, with exception to punches in a fight. The impact location for the exemplar videos were chosen according to the most commonly reported location

[61] Rousseau et al., *supra* n.6.

per mechanism of impact and energy level. The videos chosen for the reconstruction were those videos that best represented the events leading up to the impact, after viewing multiple videos for the mechanism and the impact energy level being analyzed; i.e. the calculated velocity fell within the category of energy level being analyzed, the head was accelerated following the impact and the other inclusion criteria for videos were met.

56. When multiple videos met all of the aforementioned criteria, the video closest to the average calculated impact velocity was chosen. The impact velocity was calculated using the equation v=d/t using Kinovea (version 0.8.20) to calculate the distance prior to the impact. The video was rewound by a few frames prior to the impact and the distance was measured from this point. The number of frames varied between 1 and 5 for most video impacts depending on the quality of the video and the presence of markings on the video, with the exception of puck impacts, which required a slightly different method. The number of frames prior to the impact was used to determine the time from the moment for which the distance was calculated. Every video was captured at 25 frames per second, which equates to 0.04 second per frame.[62] The horizontal velocity was then calculated for collisions with boards, glass, elbow, shoulders, punches and pucks, as it is the only velocity that can be controlled for these types of reconstructions. In the case of falls to the ice, only the vertical velocity was calculated for the same reason. The event was then reconstructed in the laboratory using appropriate equipment to obtain the six dynamic response values for each impact. The dynamic data was then inputted into a finite element model (UCDFEM) to obtain the MPS for each event.[63]

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[62] *Id.*

[63] A. Post et al., *Evaluation of the protective capacity of baseball helmets for concussive impacts*, 19 COMPUT. METHODS BIOMECH. BIOMED. ENGIN. 4, 366-75 (2016); A. Post et al., *Determination of high-risk impact sites on a Hybrid III*

57. The total number of head impacts documented for the 30 games are presented for each type of event and for each year in *Table 1*. The impacts were categorized as low (2.0 - 4.0 m/s), medium (4.0 - 7.0 m/s) or high (>7.0 m/s) velocity impacts in *Tables 2-4*. This categorization was an approximation in order to more appropriately describe the various levels of velocity of impacts.

58. 4630 impacts were recorded as head impacts resulting from the boards, elbows, glass, head to head, ice, shoulder, puck and punches for the 120 game set. The most common impact involved head to glass recording 1868 impacts over 120 games, followed by punches to the head (1067), shoulder to head (495) and elbow to the head (485). Of these 4,630 impacts, 2813 impacts were low velocity impacts, 1584 impacts represented medium velocity impacts, and 228 represented impacts at high velocity. The impact totals for the 1986-87 season (30 games) was 680 impacts, for 1995-96 there were 844 impacts, for 2003-04 there were 1,432 impacts, and for 2013-14 there were 1,674 impacts. While the impacts per game the NHL reported for the 2005/06 through 2011/12 seasons (32-45 hits/game) did not involve the same years, the reported hits per game (*Table 10*) were consistent with the numbers reported in this research.

59. The MPS figures associated with each of the reconstructed exemplar impacts are located in *Table 5*, with the associated mean in *Table 6*. The highest recorded maximum principle strain was for high velocity fall to the ice (79% MPS), falls into the boards (43% MPS), and punches (41% MPS), which was followed by elbow to the head (33% MPS), impacts with the glass and head to head (31% MPS) and shoulder to the head (29% MPS) and finally puck to head (20 %). The MPS for the medium velocity impacts were the highest for head to ice impacts (40% MPS), head to boards (31% MPS) with the rest of the events ranging between 17 to 21% MPS. The reconstructed head impacts for the low

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headform by finite element analysis, J. OF SPORTS ENGINEER. AND TECH. (2014); Rousseau et al., *supra* n.6.

velocity impacts all resulted in MPS between 10 and 13% with the exception of head impacts to the boards (18% MPS) and head impacts to the ice (24% MPS). Most notably, ***all*** of the reconstructed head impacts significantly exceeded the reported minimum threshold (between 5% and 8%) associated with white matter damage and increases to tau and NFLP in CSF, with all impacts greater than 10% MPS. Thus, the impact magnitude measured in MPS and frequency, reported as head impacts per game, reflect brain tissue trauma associated with white matter damage, tau protein damage, and cell structure damage.

60. Further, the 30 NHL teams begin their season with rookie camp, training camps, practices, and exhibition games (8-9 games) followed by 80 to 82 season games and then potentially four, best-of-seven playoff series (28). The average team will play as few as 90 games and potentially 118 games in a season. Thus, calculating MPS and total impact figures to an entire season of play (and a per game basis) involves arithmetic calculations to convert 30-game figures to an entire season, lasting between 90 and 118 games.[64] These figures for a 90 game season are reported in *Table 7*.

61. Finally, *Table 8* details the number of impacts each player of a team of 19 would, on average, experience at each velocity level for each game. Considering impact frequency and brain tissue strain (MPS) level, the data in *Table 8* would indicate that, on average, each NHL player would experience an impact of greater than 9% MPS once (1.19) a game for 1986/87, more than once (1.48) for 1995/96, twice a game (2.51) for 2003/04, and more than twice a game (2.95) for 2013/14.

62. These per game, per player MPS figures must be expressed in light of the fact that the brain trauma resulting from impacts is not distributed identically amongst all players. Benson *et al.* undertook an analysis of all reported

[64] As previously mentioned, this does not account for the additional exposure associated with the up-to 130 practices players would participate in each season.

concussions during regular season games for the 1997-98 to 2003-04 seasons in the NHL.[65] He reported 559 physician reported concussions with 162 for centermen (30.5%), 167 for defensemen (31.4%), 179 for wingers (33.6%), and 24 for goalies (4.5%). He discussed the implication of the distribution of concussion based on the make up of a line – one center, two wingers and two defensemen – which assumes there is an equal distribution of lines (four total). It should be noted, NHL teams primarily play the game with nineteen players: four centers, eight wingers, six defense and one goalie.

63. Considering the distribution of concussion by player position reported by Benson, the following risk of a player receiving a head impact above the minimum threshold to cause white matter change, per player per game, were calculated and described in *Table 9*. The lowest number of impacts above this minimum threshold (9% MPS) included 0.95 impacts per game for wingers, followed by goalies at 1.02 impacts per game in the 1986/87 and 1.18 impacts per game for wingers followed by goalies at 1.26 impacts per game for the 1995/96 seasons. The highest number of impacts per player per game was consistently reported for center men 4.25 impacts in 2013/14 and 3.64 impacts per game in 2003/2004.

64. Variance in risk is influenced by playing time, style of play, position, physical size, and coaching strategy. For example, a goalie is much more likely to receive a puck to the head than be hit into boards than a player in the wing position. Likewise, an enforcer would likely receive many more punches to the head than other players. Ultimately, however, the conservative nature of the film review and its inability to capture head impacts that occur off-camera make these impacts-per-player results conservative, and the data set is sufficiently rigorous such that I believe, to a reasonable degree of scientific certainty, that the impacts-per-player figures are an accurate representation of an average player's

[65] *Supra* n.4.

actual exposure to head blows in the NHL. Further, the position-based risk, as discussed in Benson's study, revealed that although the risk for head impacts was not identical for all positions, the levels of head impact exposure did not materially vary from the minimum threshold of one impact per game.

65. Injury to the brain tissue is a function of frequency and magnitude resulting in damage to neuronal molecules (tau) cellular structures (NFLP), neuronal damage (white matter) and concussions (metabolic). It is my opinion, based on my analysis, that professional hockey players experience a significant number of impacts from a variety of events that create significant levels of brain tissue strain and its associated injuries to brain tissue. Based on the frequency of head impacts recorded and recreated, it is my opinion that an average NHL player has likely received a head impact in each game sufficient to cause permanent injury to brain tissue. The published literature describing head trauma in ice hockey, in combination with the number of impacts, level of impacts, and associated tissue trauma contribute to my opinion that NHL hockey players experience a significant level of brain trauma contributing to an increased risk of brain damage.

Video Analysis and Lab

	Events for 30 games					Average/year for 30 games
Events	1986/87	1995/96	2003/04	2013/14	Total/ 120	
Boards	22	31	66	98	217	54.25
Elbow	89	106	126	164	485	121
Glass	182	334	637	715	1868	467
Head/Head	20	37	94	208	359	90
Ice	26	19	37	26	108	27
Shoulder	40	70	145	240	495	124
Puck	9	4	8	10	31	7.75
Punch	292	243	319	213	1067	267
total	680	844	1432	1674	4630	1157

Hits/game	22.7	28	47.7	55.8	38.6	

Table 1. Total impact count for 30 games for each of the representative years.

	Low velocity events/30 games					Average
Events	1986/87	1995/96	2003/04	2013/14	Total/120	
Boards	13	17	35	47	112	28
Elbow	58	87	66	103	314	78.5
Glass	133	249	355	348	1085	271.25
Head/Head	10	34	74	164	282	70.5
Ice	10	10	11	11	42	10.5
Shoulder	19	50	83	138	290	72.5
Puck	4	0	0	2	6	1.5
Punch	159	212	161	150	682	170.5
total	406	659	785	963	2813	703.25
Hits/game	13.5	22.0	26.2	32	23.4	

Table 2. Impact count for 30 games for each of the representative years for low velocity impacts.

	Medium velocity events/30 games					Average
Events	1986/87	1995/96	2003/04	2013/14	Total/120	
Boards	9	9	31	45	94	23.5
Elbow	28	18	51	54	151	37.75
Glass	47	78	263	323	711	177.75
Head/Head	10	2	17	40	69	17.25
Ice	16	9	12	14	51	12.75
Shoulder	15	20	48	94	177	44.25
Puck	2	3	2	7	14	3.5
Punch	125	26	107	59	317	79.25
Total	252	165	531	636	1584	396
Hits/game	8.4	5.5	17.7	21.2	13.2	

Table 3. Impact count for 30 games for each of the representative years for medium velocity impacts.

	High velocity events/30 games					Average
Events	1986/87	1995/96	2003/04	2013/14	Total/120	
Boards	0	5	0	6	11	2.75

Elbow	3	1	9	7	20	5
Glass	2	7	19	44	72	18
Head/Head	0	1	3	4	8	2
Ice	0	0	2	1	3	0.75
Shoulder	6	0	14	8	28	7
Puck	3	1	6	8	18	4.5
Punch	8	5	51	4	68	27.2
total	22	20	104	82	228	57
Hits/game	.73	.67	3.5	2.7	1.9	

Table 4. Impact count for 30 games for each of the representative years for high velocity impacts.

EVENT	Impact Velocity	SEASON (MPS level (standard deviation))				Mean	SD
		1986-1987	1995-1996	2003-2004	2013-2014		
Elbow to head	Low	0.10 (0.01)	0.14 (0.004)	0.10 (0.01)	0.10 (0.01)	0.12	0.02
	Med	0.15 (0.01)	0.20 (0.02)	0.19 (0.01)	0.29 (0.004)	0.21	0.06
	High	0.36 (0.03)	0.18 (0.01)	0.39 (0.03)	0.39 (0.03)	0.33	0.09
Shoulder to head	Low	0.13 (0.02)	0.09 (0.002)	0.08 (0.01)	0.18 (0.02)	0.12	0.04
	Med	0.19 (0.01)	0.20 (0.03)	0.15 (0.01)	0.16 (0.01)	0.17	0.02
	High	0.28 (0.01)	N/A	0.31 (0.02)	0.27 (0.002)	0.29	0.02
Head to head	Low	0.09 (0.02)	0.11 (0.003)	0.14 (0.03)	0.11 (0.02)	0.11	0.03
	Med	0.18 (0.01)	0.19 (0.01)	0.18 (0.004)	0.18 (0.02)	0.18	0.01

	High	N/A	0.31 (0.01)	0.31 (0.01)	N/A	0.31	0.13
Punch to head	Low	0.10 (0.02)	0.09 (0.001)	0.14 (0.01)	0.08 (0.01)	0.1	0.03
	Med	0.23 (0.02)	0.20 (0.02)	0.28 (0.02)	0.10 (0.01)	0.2	0.07
	High	0.41 (0.02)	N/A	0.39 (0.17)	0.43 (0.03)	0.41	0.07
Puck to head	Low	N/A	N/A	N/A	N/A	N/A	N/A
	Med	N/A	N/A	N/A	N/A	N/A	N/A
	High	0.26 (0.01)	0.19 (0.02)	0.18 (0.04)	0.16 (0.004)	0.2	0.04
Glass	Low	0.11 (0.003)	0.15 (0.02)	0.13 (0.004)	0.14 (0.01)	0.13	0.02
	Med	0.16 (0.005)	0.16 (0.03)	0.18 (0.01)	0.23 (0.03)	0.18	0.04
	High	0.26 (0.02)	0.30 (0.02)	0.29 (0.003)	0.37 (0.02)	0.31	0.04
Boards	Low	0.18 (0.02)	0.19 (0.01)	0.12 (0.01)	0.22 (0.01)	0.18	0.04
	Med	0.27 (0.02)	N/A	0.28 (0.01)	0.36 (0.01)	0.31	0.05
	High	N/A	0.43 (0.02)	N/A	N/A	0.43	0.02
Ice	Low	0.23 (0.01)	0.23 (0.005)	0.25 (0.03)	0.26 (0.02)	0.24	0.02
	Med	0.56 (0.04)	0.40 (0.01)	0.23 (0.01)	0.41 (0.01)	0.4	0.12
	High	N/A	N/A	0.79 (0.04)	N/A	0.79	0.04

Table 5. Maximum principle strain values for exemplar impacts for each event, each velocity level and each year.

	Impact velocity					
	Low		Medium		High	
Season	MPS	Impacts/game	MPS	Impacts/game	MPS	Impacts/game
1986/87	0.13	13.5	0.21	8.4	0.31	0.73

1995/96	0.14	22	0.23	5.5	0.28	0.67
2003/04	0.14	26	0.21	17.7	0.38	3.5
2013/14	0.16	32	0.25	21	0.32	2.7
Mean	0.14	23.375	0.22	13.15	0.32	1.9

Table 6. Mean maximum principle strain values and impacts per game for each velocity level and each year.

	1986/87		1995/96		2003/04		2013/14	
Impact Velocity	30 games	Per 90 game season	30 games	Per 90 game season	30 games	Per 90 game season	30 games	Per 90 game season
Low	406	1218	659	1977	785	2355	963	2889
Medium	252	756	165	495	531	1593	636	1908
High	22	66	20	60	104	312	82	246
Total	680	2040	844	2532	1432	4296	1674	5022

Table 7. Estimated impact count per 90 games for each velocity level and each year.

	1986/87			1995/96			2003/04			2013/14			MPS
Impact Velocity	30 games	Per game	Impacts /player	30 games	Per game	Impacts /player	30 games	Per game	Impacts /player	30 games	Per game	Impacts /player	
Low	406	13.5	0.71	659	22	1.16	785	26	1.37	963	32	1.68	0.23
Medium	252	8.4	0.44	165	5.5	0.29	531	17.7	0.93	636	21	1.11	0.39
High	22	0.73	0.04	20	0.67	0.0.04	104	3.5	0.18	82	2.7	0.14	0.56
Total	680	22.7	1.19	844	28	1.48	1432	47.7	2.51	1674	56	2.95	

Table 8. Estimated impact count per game and per player (19 players) for each velocity level and each year.

		1986/87	1995/96	

		Hits	Hits/game (90)	Hits/player (19)	Hits	Hits/game (90)	Hits/player (19)	
Total	% of risk/position	2040	22.7	1.19	2532	28.1	1.48	
Centers (4)	30.5	622.2	6.9	1.73	772.3	8.6	2.15	
Wingers (8)	33.6	685.4	7.6	0.95	850.8	9.5	1.18	
Defense (6)	31.4	640.6	7.1	1.87	795.	8.8	1.47	
Goalies (1)	4.5	91.8	1.02	1.02	113.9	1.3	1.26	
	Benson *et al.* (2011)							

		2003/04			2013/14			
		Hits	Hits/game (90)	Hits/player (19)	Hits	Hits/game (90)	Hits/player (19)	
Total	% of risk/position	4296	47.7	2.51	5022	55.8	2.94	
Centers (4)	30.5	1310	14.6	3.64	1532	17.0	4.25	
Wingers (8)	33.6	1443	16.0	2.00	1687	18.7	2.34	
Defense (6)	31.4	1349	15.0	2.50	1577	17.5	2.92	
Goalies (1)	4.5	193	2.1	2.05	226	2.5	2.51	
	Benson et al. (2011)							

Table 9. Estimated impact count per game and per player (19 players) for each player position and each year.

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Season	Total # of Hits	Hits Per Game
2005-2006	39,305	32
2006-2007	44,006	36
2007-2008	46,736	38
2008-2009	51,557	42
2009-2010	54,214	44
2010-2011	55,911	45
2011-2012	55,445*	45

Month	HITS/GM	GAMES	HITS
OCT	42.686	318	6787
NOV	43.101	396	8534
DEC	44.157	420	9273
JAN	47.945	362	8678
FEB	47.085	402	9464

Table 10. NHL report from video analysis estimating hits per game.

V. CONCLUSION

I declare under penalty of perjury that the foregoing is true and correct.

Executed on: Nov. 27, 2016

Thomas Blaine Hoshizaki, Ph.D

EXHIBIT “1”

Qualifications and Expert Disclosures:

NAME: HOSHIZAKI, Thomas, Blaine, Full Professor,

Member of the Faculty of Graduate and Postdoctoral Studies

DEGREES:

Ph.D., Exercise Physiology, University of Illinois, Urbana Champaign, Illinois, 1978
M.A., Exercise Physiology, University of South Alabama, Mobile, Alabama, 1975
BPE, Bachelor of Physical Education, University of Calgary, Calgary, Alberta, 1974

EMPLOYMENT HISTORY:

2013 -	Full Professor, Human Kinetics, University of Ottawa
2004 - 2012	Associate Professor, Human Kinetics, University of Ottawa
2002 - 2004	Consultant, Sporting Goods Industry
1997 - 2004	Adjunct Professor, Faculty of Human Kinetics, University of Windsor
1997 - 2002	Vice President, Product Development, The Hockey Company. (CCM)
1995 - 1998	Adjunct Professor, Department of Physical Education, McGill University
1995 - 1997	Research and product design consultant to Canstar / Nike
1989 - 1995	Vice President Research and Development, Canstar Sports Group Inc (Bauer)
1984 - 1994	Associate Professor, Department of Physical Education, McGill University
1980 - 1984	Assistant Professor, Department of Physical Education, McGill University
1979 - 1980	Assistant Professor, Department of Physical Education and Outdoor Recreation, Lakehead University
1978 - 1979	Sessional Lecturer, Department of Physical Education, University of Victoria

ACADEMIC HONOURS:

2015	Invited speaker Hoshizaki T. B. (2015) Mechanisms of Head Injury in Sport and Energy Management Characteristics of Helmet Technologies, Risk and Insurance Management Society, January 20, 2015, Ottawa, ON.
2015	Invited speaker Hoshizaki T. B. (2015) Concussion and the Student Athlete, April 24, 2015 NSBN, Niagara on the Lake, ON
2015	Invited speaker Hoshizaki T.B. (2015) Pathway to a Hit count technical standard. Symposium on Head Acceleration Measurement Sensors, March 9, 2015. Blacksburg, Virginia USA
2015	Invited speaker Hoshizaki T.B. (2015) Mechanics of Head Injuries in Sport and Energy Management Characteristics of Helmet Technologies. Sport Science Conference, January 15 – 16, 2015, London England
2014	Invited speaker Hoshizaki T. B. (2014) Protective Equipment: The relationship between how humans play sport and how helmets protect against head injury. Mini Med School, Oct. 23 2014/May. 14, 2015 Ottawa, ON.
2013	Invited Speaker/Faculty Member, On October 9th and 10th, 2013, Mayo Clinic will host the 2nd “Ice Hockey Summit: Action on Concussion”. Topic: Determining Forces to Head and Brain – PROs and CONS of Reconstruction
2012	Scientific/medical Expert, Sport Legacy Institute: Boston 2012 conference Chronic Traumatic Encephalopathy

2012 Invited Speaker, U.S.A. National conference on Chronic Traumatic Encephalopathy 2012: topic, The Biomechanics of Head Trauma: Measuring Blows to the Head

2011 Expert Scientist, Quoted in the U.S. Senate Committee Hearings, of October 19, 2011 on the Concussion and the Marketing of Sports Equipment, One hundred and twelfth Congress. Identified as a well-respected scientist and biomechanical engineer published excellent work regarding helmet performance and the mechanics of head injury

2012 Globe and Mails Power 50, named as one of the 50 most influential people in Canadian Sport 2012 by The Globe and Mail

2012 Invited Lecture: Canadian National Museum of Nature, Protective Equipment: Why Turtles Have It and Humans Need It?

2010 - 2011 Invited participant Think First: "Think Tank on Brain Injury Prevention" Toronto, Canada, May 11, 2010

1993 Visiting Research Scholar, Visiting Research Scholar, Université Joseph Fournier, Grenoble, France

1986 - 1987 Visiting Research Scholar, Visiting Research Scholar, University of Tokyo, Tokyo, Japan

RELEVANT PUBLICATIONS:

1. Post A, Hoshizaki TB, Gilchrist MD, Brien S, Cusimano MD, Marshall S. "The dynamic response characteristics of traumatic brain injury." Analysis and Prevention. 2015. In Press.
2. Post A, Karton C, Hoshizaki TB, Gilchrist MD, Bailes J. "Evaluation of the protective capacity of baseball helmets for concussive impacts" Computer Methods in Biomechanics and Biomedical Engineering. 2015. doi.org/10.1080/10255842.2015.1029921.
3. Rousseau, P., & Hoshizaki, T. B. "Defining the effective impact mass of elbow and shoulder strikes in ice hockey." Sports Biomechanics. 2015. In Press.
4. Post A, Hoshizaki TB. "Rotational acceleration, brain tissue strain, and the relationship to concussion." Journal of Biomechanical Engineering. 2015. 137, 030801-1030801-9. doi: 10.1115/1.4028983.
5. Oeur R.A., Karton C., Post A, Rousseau P, Hoshizaki TB, Marshall S, Brien S, Smith A, Cusimano MD, Gilchrist MD. "A comparison of the head dynamic response and brain tissue deformation from impacts resulting in concussion, concussion with persistent post-concussive symptoms, and subdural hematoma." Journal of Neurosurgery. 2014. In Press.
6. Post A, Rousseau P, Kendall MK*, Walsh ES*, Hoshizaki TB. "Determination of high-risk impact sites on a Hybrid III headform by finite element analysis." Journal of Sports Engineering and Technology. 2014. doi: 10.1177/1754337114551444.
7. Post, A., Kendall, M.*, Koncan, D., Cournoyer, J.*, Hoshizaki, T.B., Gilchrist, M.D., Brien, S., Cusimano, M. & Marshall, S. "Characterization of Persistent Concussion Syndrome through injury reconstruction and Finite Element Modelling." Journal of the Mechanical Behaviour of Biomedical Materials: Special issue on TBI. 2014. doi: 10.1016/j.jmbbm.2014.07.034.
8. Post, A., Hoshizaki, T.B., Gilchrist, M.D., Brien, S., Cusimano, M. & Marshall, S. "Traumatic Brain Injuries: The influence of the direction of impact." Journal of Neurosurgery. 2015. 76(1), 81-91.

9. Hoshizaki, T.B., Post, A., Oeur, R.A. & Brien, S.E. “Current and future concepts in helmet and sports injury prevention.” Neurosurgery: Sports Concussion Supplement, 2014. 75(4), S136-S148.
10. Post A., Hoshizaki T.B. and Gilchrist M.D., “Comparison of MADYMO and physical models for brain injury reconstruction”, International Journal of Crashworthiness, 2014. doi: 10.1080/13588265.2014.897413.
11. Post A., Oeur A., Hoshizaki T.B. and Gilchrist M.D., “Differences in region specific brain tissue stress and strain due to impact velocity for simulated American football impacts”, Journal of Sports Engineering and Technology – HPD Special Issue., 2014. doi: 10.1177/1754337114527742
12. Post A., Hoshizaki T.B., Gilchrist M.D., Brien S. Cusimano M. and Marshall S., “The influence of acceleration loading curve characteristics on traumatic brain injury”, Journal of Biomechanics, 47(5), 1074-1081, 2014. doi.org/10.1016/j.jbiomech.2013.12.026.
13. Hoshizaki T.B., Post A., Kendall M., Karton C. and Brien S., “The relationship between head impact and brain trauma”, Special issue of the Journal of Neurology & Neurophysiology: Traumatic Brain Injury Diagnosis & Treatment, 5, 181, 2013.
14. Dawson L., Oeur A., Rousseau P. and Hoshizaki T.B., “The influence of impactor striker size on the dynamic response of a Hybrid III headform”, Journal of ASTM international, 2013. doi: 10.1520/STP155220120176
15. Rousseau P., Hoshizaki T.B. and Gilchrist M.D., “Protective capacity of ice hockey helmets against puck impacts”, Journal of ASTM international, 2013. [In Press]
16. Bishop P., Kendall M., Post A. and Hoshizaki T.B., “Performance criteria or a child specific helmet”, Journal of ASTM international, 2013. [In Press]
17. Kendall M., Rousseau P. and Hoshizaki T.B., “The effect of shoulder pad design on reducing peak resultant linear and rotational acceleration in shoulder to head impacts”, Journal of ASTM international, 2013. doi:10.1520/STP155220120150
18. Oeur A., Hoshizaki T.B. and Gilchrist M.D., “The influence of impact location and angle on brain tissue deformation”, Journal of ASTM international, 2013. doi: 10.1520/STP155220120160
19. Post A., Hoshizaki T.B., Gilchrist M.D., Brien S., Cusimano M. and Marshall S., “The influence of dynamic response and brain deformation metrics on the occurrence of subdural hematoma in different regions of the brain”, Journal of Neurosurgery, 120(2), 453-461, 2013.
20. Nishizaki K., Marino W., Hoshizaki T.B., Post A., Oeur A., Walsh E.S. and Gilchrist M.D., “Evaluation of dynamic response and brain deformation metrics using a centric/non-centric impact protocol using a monorail impacting system”, Journal of ASTM international, 2013. [In Press]
21. Post A., Hoshizaki T.B. and Brien S., “Head injuries, measurement criteria and helmet design”, Routledge handbook of ergonomics in sports and exercise. New York: Routledge, 2013.
22. Karton C., Hoshizaki T.B. and Gilchrist M.D., “The influence of inbound mass on the dynamic impact response of the Hybrid III headform and tissue deformation response characteristics”, Journal of ASTM international, 2013 doi: 10.1520/STP155220120175
23. Karton C., Rousseau P., Vassilyadi M. and Hoshizaki T.B., “The evaluation of speed skating helmet performance through peak linear and rotational accelerations”, British Journal of Sports Medicine, 48, 46-50, 2013. doi:10.1136/bjsports-2012-091583
24. Post A., Oeur A., Walsh E.S., Hoshizaki T.B. and Gilchrist M.D., “A centric/non-centric impact protocol and finite element model methodology for the evaluation of American football helmets to evaluate risk of concussion”, Computer Methods in Biomechanics and Biomedical Engineering. 2013. Doi.org/10.1080/10255842.2013.766724

25. Wright R, Post A., Hoshizaki T.B. and Ramesh K.T., “A multiscale computational approach to estimating axonal damage under inertial loading of the head”, Journal of Neurotrauma, 30(2), 102-118, 2013. doi:10.1089/neu.2012.2418
26. Post A., Oeur A., Hoshizaki T.B. and Gilchrist M.D., “An examination of American football helmets using brain deformation metrics associated with concussion”, Materials & Design, 45, 653-662, 2013.
27. Post A. and Hoshizaki T.B., “Mechanisms of brain impact injuries and their prediction: A review”, Trauma, 14(4), 327-349, 2012. doi:10.1177/1460408612446573
28. Robert Cantu, M.D., Pat Bishop, Ph.D., Stefan Duma, Ph.D., Tom Gennarelli, M.D, Richard Greenwald, Ph.D., Kevin Guskiewicz, Ph.D., P. Dave Halstead, M.S., Thomas Blaine Hoshizaki, Ph.D., Albert I. King, Ph.D., Frederick O. Mueller, Ph.D., and Margot Putuk, “Letter to the Editor Helmets”, Journal of Neurosurgery, 187-189, May 11, 2012.
29. Kendall M., Walsh E.S. and Hoshizaki T. B., “Comparison between Hybrid III and Hodgson-WSU headforms by linear and angular dynamic response”, Journal of Sports Engineering and Technology, 0(0), 1-6, 2012. doi: 10.1177/1754337112436901
30. Post A., Hoshizaki T.B., Gilchrist M.D. and Brien S., “Analysis of the influence of independent variables used for reconstruction of a traumatic brain injury incident”, Journal of Sports Engineering and Technology, 226(3/4), 290-298, 2012. doi: 10.1177/1754337112436629
31. Post A., Hoshizaki T.B. and Gilchrist M., “Finite element analysis of the effect of loading curve shape on brain injury predictors”, Journal of Biomechanics, 45(4), 679-683, 2012.
32. Post A., Gimbel G. and Hoshizaki T.B., “The influence of headform circumference and mass on alpine ski helmet performance in laboratory tests”, Journal of ASTM International., 9(4), 5 pages, 2012.
33. Hoshizaki T.B. Vassilyadi M., Post A. and Oeur A., “Performance analysis of winter activity protection headgear for young children”, Journal of Neurosurgery Pediatrics, 9 (2), 133-138, 2012.
34. Kendall M. and Hoshizaki T.B., “Comparison between Hybrid III headforms by linear and angular dynamic impact response characteristics”, Journal of ASTM International, 9(4): 1-8, 2012. doi: 10.1520/JAI104270
35. Walsh E.S., Post A., Rousseau P., Kendall M., Karton C., Oeur A. and Hoshizaki T.B., “Dynamic Impact Response Characteristics of a Helmeted Hybrid III Headform Using a Centric and non-Centric Impact Protocol”, Journal of Sports Engineering and Technology, 226(3-4): 220-225, 2012. doi: 10.1177/1754337112442299
36. Hoshizaki T.B., Walsh E.S., Post A., Rousseau P., Kendall M., Karton C., Ouer A., Foreman S. and Gilchrist M., “The Application of Brain Tissue Deformation Values in Assessing the Safety Performance of Ice Hockey Helmets”, Journal of Sports Engineering and Technology, 226(2/4), 226-236, 2012. doi:10.1177/1754337112448765
37. Post A., Walsh E.S., Hoshizaki T.B. and Gilchrist M., “Analysis of .loading curve characteristics on the production of brain deformation metrics”, Journal of Sports Engineering and Technology, 226(3/4), 200-207, 2012. doi: 10.1177/1754337112449420
38. Post A., Oeur A., Hoshizaki T.B. and Gilchrist M.D., “Examination of the relationship of peak linear and peak angular acceleration to brain deformation metrics in hockey”, Computer Methods in Biomechanics and Biomedical Engineering, 16(5), 511-519, 2011. doi: 10.1080 10255842.
39. Walsh E.S., Rousseau P. and Hoshizaki T.B., “The influence of impact location and angle on the dynamic impact response of a Hybrid III headform”, Sports Engineering, 13(3), 135 – 143, 2011. doi: 10.1007/s12283-011-0060-9.
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41. Lamb L. and Blaine Hoshizaki, “Deformation mechanisms and impact attenuation characteristics of thin-walled collapsible air chambers for head protection”, Journal of Engineering in Medicine, 223(8), 1021 – 1031, 2009.
42. Rousseau P. and Hoshizaki, T.B., “The influence of deflection and neck compliance on the impact dynamics of a hybrid III head form”, Journal of Sports Engineering and Technology, 223(3), 89 - 97, 2009.
43. Rousseau P., Post A. and Hoshizaki, T.B., “The effects of impact management materials in ice hockey helmets on head injury criteria”, Journal of Sports Engineering and Technology, 223, 159-165, 2009.
44. Coulson N.R., Foreman S.G. and Hoshizaki T.B., “Peak translational and rotational accelerations generated during reconstructed ice hockey impacts on a Hybrid III headform”, Journal of American Standards of Testing and Materials International, 2009.
45. Hoshizaki T.B. and Post A., “Impact attenuation characteristics of engineered structures used in protective helmets”, Journal of American Standards of Testing and Materials International, 6, 4 pages, 2009.
46. Lamb L., Post A. and Hoshizaki, T.B., “Development of a new methodology capable of characterizing the contribution of a controlled venting system to impact attenuation in chamber structures for head protection “, Journal of ASTM international, 6(1), 8 pages, 2009.
47. Rousseau P., Post A. and Hoshizaki, T.B., “A comparison of peak linear and angular headform accelerations using ice hockey helmets”, Journal of ASTM International, 6(1), 11 pages, 2009.
48. Gimbel G. and Hoshizaki, T.B., “Compressive properties of helmet materials subjected to dynamic impact loading of various energies”, European Journal of Sport Science, 8(6), 241-249, 2008.
49. Gimbel G. and Hoshizaki T.B., “A comparison between vinyl nitrile foam and new air chamber technology on attenuating impact energy for ice hockey helmets”, International Journal of Sports Science and Engineering, 2(3), 154-161, 2008.
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51. Post, A., Karton, C., Hoshizaki, T.B. & Gilchrist, M.D. (2014). Analysis of the protective capacity of ice hockey helmets in a concussion injury reconstruction. Proceedings of the IRCOBI Conference, Sept 10-12, Berlin, Germany.
52. Oeur, R.A., Zanetti, K. & Hoshizaki, T.B. (2014). Linear and angular acceleration responses of American football, lacrosse, and ice hockey helmets subject to low-level impacts. Proceedings of the IRCOBI Conference, Sept 10-12, Berlin, Germany.
53. Walsh, E.S., Kendall, M., Hoshizaki, T.B. & Gilchrist, M.D. (2014). Dynamic Impact Response and Predicted Brain Tissue Deformation Comparisons for an Impacted Hybrid III Headform With and Without a Neckform and Torso Masses. Proceedings of the IRCOBI Conference, Sept 10-12, Berlin, Germany.
54. Zanetti, K., Post, A., Karton, C., Kendall, M., Hoshizaki, T.B. & Gilchrist, M.D. (2013). Identifying risk profiles for player positions in American football using physical and finite element modeling reconstructions. Proceedings of IRCOBI, Gothenburg Sweden, Sept 14th-17th. (In Press)

55. Kendall, M., Post, A., Zanetti, K., Hoshizaki, T.B. & Gilchrist, M.D. (2013). Comparison of dynamic versus static head impact reconstruction methodology by means of dynamic impact response and brain deformation metrics. Proceedings of IRCOBI, Gothenburg Sweden, Sept 14th-17th. (In Press)
56. Post A., Oeur, A., Hoshizaki, T.B. & Gilchrist, M.D. (2013). The influence of velocity on injury risk in American football. Proceedings of Helmet Performance and Design Conference, London England, Feb 15th.
57. Kendall, M., Post, A., Rousseau, P., Oeur, A., Gilchrist, M. D., & Hoshizaki, T. B, (2012) "A Comparison of Dynamic Impact Response and Brain Deformation of Head Impact Reconstructions Representing Three Mechanisms of Head Injury in Ice Hockey.", International Research Council on Biomechanics of Injury Conference (IRCOBI, Dublin Ireland.
    Author Contribution: co-author
58. Post, A., Oeur, A., Hoshizaki, T. B., & Gilchrist, M. D., (2012). "The Influence of Centric and Non-Centric Impacts to American Football Helmets on the Correlation Between Commonly Used Metrics in Brain Injury Research", International Research Council on Biomechanics of Injury Conference (IRCOBI), Dublin Ireland.
    Author Contribution: Co- Author

SCHOLARLY and PROFESSIONAL ACTIVITIES :

2013 -	Member of the NCAA National Concussion Committee
2011 -	Member of the Scientific Committee Sport Legacy Institute, Boston.
2010 -	Member of the Scientific Committee NOCSAE
2012 - 2013	Member of the Steering Committee for Ice Hockey Summit II: Action on Concussion, Mayo Clinic, Rochester, USA
2010 - 2011	Co-Host National Hockey Safety Summit Ottawa, ON
2010	Member of the Steering Committee for Ice Hockey summit: Action on Concussion, Mayo Clinic, Rochester, USA
2007 - 2008	Member of the Scientific Organizing Committee for the Fourth International Meeting on mTBI in Sports, St Moritz, Switzerland
2005 - 2008	Ottawa Children's Treatment: Centre Research Advisory Board
2006 - 2007	Member of the Scientific Organizing Committee for the Third International Meeting on mTBI in Sports, St Moritz, Switzerland
2006 - 2007	CCUPEKA Canadian Council of University (President)
2005 - 2006	Member of the Scientific Organizing Committee for the Second International Meeting on mTBI in Sports, St Moritz, Switzerland
2002 -	Advisory Board Committee member for "Think First"
1999 - 2002	Member of the National Hockey League (NHL) Advisory Panel for the Reduction of Injuries in the NHL
1996 - 2000	Chair of the ASTM Sub-Committee for the Standardization of Inline Skate Products F08.11
1994 - 2004	Member of the Working group committee for CEN for head and face protection standard for ice hockey
1994 - 1998	Member of the Working group committee for CEN for head and face protection standard for ice hockey (ISO)
1991 -	Chair; International Standards Organization (ISO), Ice Hockey Equipment Committee
1990 -	Committee member: Canadian Standards Association (CSA) for Ice Hockey Equipment Committee

1989 -	Committee Member: American Standards for Testing Materials (ASTM) for Ice Hockey Equipment
1995 - 1998	Member of the Working group committee for CEN personal protection equipment for ice hockey (ISO)
1992 - 1995	International Society of Biomechanics of Sport Vice-President responsible for Awards
1988 - 1992	International Society of Biomechanics of Sport Vice-President responsible for Publications
1989 - 1991	Certification et d'Accréditation de l'Evaluation de la Condition Physique
1989 - 1991	FCAR Fonds Pour la Formation des Chercheurs et l'Aide à la Recherche. Student Bourse Committee
1989 - 1991	International Symposium on 3-D Analysis of Human Movement Member of the Scientific Committee Organization?
1986 - 1989	External Editor, Journal of Sports Science of Brazil

Expert for cases (past four years):

1) Provided and expert opinion: 2015 Martin Szpiro vs Michel – Anthony Gigante and Mario Gigante, Carole Lecavalier, Rosmere High School, Sir Wilfred Laurier School Board, Greater Montreal English Regional School Sports Association.
2) Provided an expert opinion: 2015 Mr. Mike Caplan
   Suffolk County Office of the Medical Examiner
   P.O. Box 6100
   725 Veterans Memorial Highway
   North County Complex Bldg. #487
   Hauppauge, NY 11788-0099
   Re: Thomas Cutinella (fatal head injury football)
3) Provided an expert opinion: 2014 Steve Moore, Jack Moore and Anna Moore vs Todd Bertuzzi, Oraca Bay Hockey Limited Partnership, Orca Bay Hockey, Inc., dba The Vancouver Canucks Hockey Club.

EXHIBIT “2”

Exhibit 2

- Bain A, Meaney D. 2000. Tissue-level thresholds for axonal damage in an experimental model of central nervous system white matter injury. *J Biomech, 122*, 615-622

- Banks SL, Mayer B, Obuchowski N, et al. 2014. Impulsiveness in professional fighters. *J Neuropsychiatry Cli Neurosci*, 26: 44-50.

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- Cubon VA, Putukian M, Boyer C, Dettwiler A. 2011. A diffusion tensor imaging study on the white matter skeleton in individuals with sports-related concussion. *J Neurotrauma*, 28(2): 189-2011

- Delaney, J. Scott MDCM[*,†]; Lamfookon, Charles MDCM[‡]; Bloom, Gordon A. PhD[§]; Al-Kashmiri, Ammar MD[¶]; Correa, José A. PhD (2015) Why University Athletes Choose Not to Reveal Their Concussion Symptoms During a Practice or Game Clinical Journal of Sport Medicine: March 2015 - Volume 25 - Issue 2 - p 113–125

- Dustin P Joubert, Aaron Caldwell, Steve E Martin and Stephen F Crouse (2015) A longitudinal study examining the effects of a season of American football on lipids and lipoproteins *Lipids in Health and Disease*, 201514:35.

- Elkin BS, Morrison B 3rd. 2007. Region-specific tolerance criteria for the living brain. *Stapp Car Crash J*, 51: 127-138.

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- Jeffrey J. Iliff,[1,2] Minghuan Wang,[1,3] Douglas M. Zeppenfeld,[1,2] Arun Venkataraman,[1] Benjamin A. Plog,[1] Yonghong Liao,[1] Rashid Deane,[1] and Maiken Nedergaard Cerebral Arterial Pulsation Drives Paravascular CSF–Interstitial Fluid Exchange in the Murine Brain J Neurosci. (2013) Nov 13; 33(46): 18190–18199. doi: 10.1523/JNEUROSCI.1592-13.2013 PMCID: PMC3866416

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EXHIBIT “3”

2013-14 (82 regular season games, 30 teams,)
15 games in October
15 games in March

1. Buffalo Sabres Vs. Colorado Avalanche,
2. Los Angeles Kings Vs Calgary Flames
3. Montreal Canadiens Vs. Edmonton Oilers
4. Columbus Blue Jackets Vs. Vancouver Canucks
5. Florida Panthers Vs. Chicago Blackhawks
6. St. Louis Blues Vs. Winnipeg Jets
7. Minnesota Wild Vs. Nashville Predators
8. Tampa Bay Lightning Vs Chicago Blackhawks
9. Boston Bruins Vs. San Jose Sharks
10. Florida Panthers Vs. Vancouver Canucks
11. San Jose Sharks Vs. Anaheim Ducks
12. Pittsburg Penguins Vs Tampa Bay Lightning
13. Toronto Maple Leafs Vs. Anaheim Ducks
14. Winnipeg Jets2 Vs. Washington Capitals
15. New Jersey Devils Vs. Vancouver Canucks
16. Pittsburg Penguins Vs New York Islanders
17. Boston Bruins Vs. New Jersey Devils
18. Dallas Stars Vs. Winnipeg Jets3
19. New York Islanders Vs Philadelphia Flyers
20. Ottawa Senators Vs. New York Rangers
21. Edmonton Oilers2 Vs. Nashville Predators
22. Anaheim Ducks Vs. Washington Capitals
23. Detroit Red Wings Vs. Toronto Maple Leafs
24. Edmonton Oilers3 Vs. Buffalo Sabres
25. New Jersey Devils Vs. Minnesota Wild
26. Arizona Coyotes Vs. Florida Panthers
27. Chicago Blackhawks Vs. Carolina Hurricanes
28. Dallas Stars Vs. Ottawa Senators
29. Philadelphia Flyers Vs. St. Louis Blues
30. Washington Capitals Vs. Los Angeles Kings

2003-04 (82 regular season games, 30 teams,)
15 games in October & November
15 games in March

1. Anaheim ducks vs new york rangers
2. Colorado Avalanche vs New jersey devils
3. Boston bruins vs montreal canadians
4. Calgary Flames vs Dallas Stars

5. Carolina Hurricanes vs Tampa bay lightning
6. Minnesota Wild vs Buffalo Sabres
7. Philadelphia Flyers vs New jersey devils
8. San Jose Sharks vs Tampa Bay Lightning
9. Atlanta Thrashers vs Washington Capitals
10. Boston Bruins vs Pittsburgh Penguins
11. Nashville predators vs St. Louis Blues
12. San Jose Sharks vs Los Angeles Kings
13. Dallas Stars vs Philadelphia Flyers
14. Chicago blackhawks vs Arizona Coyotes
15. Minnesota Wild vs Vancouver Canucks
16. Buffalo Sabres vs Washington Capitals
17. Columbus Blue Jackets vs Edmonton Oilers
18. New York Rangers vs Atlanta Thrashers
19. Detroit red wings vs Nashville predators
20. Vancouver Canucks vs Los Angeles Kings
21. Ottawa Senators vs Ney York Islanders
22. Tampa Bay Lightning vs Carolina Hurricanes
23. Ottawa Senators vs Calgary flames
24. Montreal Canadiens vs Los angeles kings
25. Dallas Stars vs Pittsburgh Penguins
26. Los Angeles Kings vs Arizona Coyotes
27. Florida Panthers vs Toronto Maple leafs
28. Detroit red wings vs Columbus blue jackets
29. Colorado Avalanche vs Edmonton Oilers
30. Chicago Blackhawks vs New jersey devils

1995-96 (82 regular season games, 26 teams,)
15 games in October & November
15 games in March

1. Anaheim mighty ducks vs St. Louis Blues
2. Buffalo Sabres vs Colorado Avalanche
3. Buffalo Sabres vs Chicago Blackhawks
4. Pittsburgh Penguins vs Buffalo Sabres
5. Colorado Avalanche vs Anaheim Mighty Ducks
6. Vancouver Canucks vs Chicago Blackhawks
7. Pittsburgh Penguins vs New York Islanders
8. Anaheim mighty ducks vs Washington Capitals
9. Washington Capitals vs Montreal Canadiens
10. Calgary Flames vs Detroit Red Wings

11. Edmonton oilers vs New york rangers
12. St Louis Blues vs Dallas Stars
13. Boston Bruins vs New Jersey Devils
14. Detroit Red Wings vs Calgary Flames
15. Los Angeles Kings vs Toronto Maple leafs
16. New York Islanders vs Florida Panthers
17. Tampa Bay lightning vs philadelphia flyers
18. Dallas Stars vs Toronto Maple Leafs
19. Tampa Bay Lightning vs Harford Whalers
20. Dallas Stars vs San Jose Sharks
21. Chicago Blackhawks vs Montreal Canadiens
22. Ottawa Senators vs Philadelphia Flyers
23. Montreal Canadiens vs Boston Bruins
24. Calgary flames vs Los Angeles Kings
25. Hartford Whalers vs Ottawa Senators
26. New York Islanders vs Chicago Blackhawks
27. Florida Panthers vs New Jersey Devils
28. Buffalo Sabre vs Montreal Canadiens
29. Detroit Red Wings vs Toronto Maple Leafs
30. Montreal Canadiens vs Buffalo Sabres

1986-87 (80 regular season games, 21 teams)
15 games October, November & December
15 games January, February, March & April

1. Philadelphia Flyers- Boston Bruins
2. Chicago Blackhawks- Toronto Maple Leaf
3. Winnipeg Jets- Philadelphia Flyers
4. New York Rangers- Montreal Canadiens
5. Pittsburgh Penguins- Toronto Maple Leaf
6. Hartford Whalers- Toronto Maple Leafs
7. Boston Bruins vs. Toronto Maple Leafs
8. Washington Capitals- Philadelphia Flyers
9. Los Angeles Kings- Montreal Canadiens
10. Minnesota North Stars- Philadelphia Flyers
11. Philadelphia Flyers- Buffalo Sabres
12. Edmonton Oilers-Toronto Maple leaf
13. Montreal Canadiens- Quebec Nordiques
14. New Jersey Devils- Philadelphia Flyers
15. Washington Capitals- Philadelphia Flyers (86)
16. Buffalo Sabres-Toronto Maple Leaf
17. New Jersey Devils- Toronto Maple Leafs

18. Detroit Red Wings- Toronto Maple Leafs
19. Philadelphia Flyers- Hartford Whalers
20. Philadelphia Flyers- Vancouver Canucks
21. St-Louis Blues- Philadelphia Flyers
22. Philadelphia Flyers- New York Islanders
23. Detroit Red Wings- Philadelphia Flyers
24. Calgary Flames-Toronto Maple Leaf
25. LA Kings- Calgary Flames
26. Vancouver-Toronto Maple Leaf
27. New York Islanders- Calgary Flames
28. Chicago Blackhawks- Toronto Maple Leaf (87)
29. Philadelphia Flyers- Edmonton Oilers
30. Philadelphia Flyers- Los Angeles Kings