Concussion Incidence and Risk Factors in the National Hockey League between the 2005-2006 and 2011-2012 Seasons

By

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Abstract

Ice hockey has been identified as a sport with a high risk for concussions, and given the injury's serious health consequences, it has become a major concern within the sporting community. While research is available at the youth and junior levels, there are few reports at the professional level describing concussion incidence and risk factors. The purpose of the present study was to provide a descriptive analysis of concussion as well as identify concussive risk factors for a sample of National Hockey League players. Data on player position, age, height, weight, average time on ice (ATOI) and reported concussions were collected from *The Hockey News* for the 2005-2006 to 2011-2012 seasons. Chi square analysis revealed significant effects for season $\chi^2(6, N = 6145) = 28.67, p = .00$, age $\chi^2(2, N = 6145) = 5.70, p = .05$ and ATOI $\chi^2(6, N = 6145) = 241.53, p = .00$. One-way ANOVA revealed reported concussion differences amongst the seven seasons for age: $F(6, 6138) = 2.40, p = .03$, weight: $F(6, 6138) = 2.85\ p = .01$ and concussion incidence: $F(6, 6138) = 4.80, p = .00$. Logistic regression revealed increased risk of concussion for the 2010-2011 (1.7 times) and 2011-2012 (2.12 times) seasons as well as the 15-20 minute (2.64 times) ATOI category. The results indicate that concussion incidence is rising in the NHL and that ATOI is a risk factor for concussive injury. Implications of these findings are discussed.

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"A teacher affects eternity; he can never tell where his influence stops." –Henry B. Adams-

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Introduction

Introduction to Concussion

In the United States, approximately 1.6 to 3.8 million cases of sport- and recreation-related traumatic brain injuries (TBI) take place each year[1]. Concussion, a specific form of mild TBI, is particularly prominent in sport, and has been estimated by the National Collegiate Athletic Association (NCAA) to have steadily increased by 7.0% annually between the 1988-1989 and 2003-2004 seasons[2]. The rising frequency has been attributed to better detection and reporting methods, as well as reflecting a true increase in concussion incidence[3]. While increasing prevalence *and* better detection have led to the increased incidence rates, the reported rates of concussion are believed to be low due to the issue of underreporting. For example, many athletes withhold information regarding their symptomatology in order to avoid missing playing or practice time. Additionally, the signs and symptoms of concussion often resolve rapidly and spontaneously without the knowledge of many athletes, coaches and medical personnel realizing the injury had taken place[4]. Due to the pervasive influence of concussion in sport and the short- and long-term complications associated with it, medical personnel, coaches, players and parents need to be well informed regarding the injury in order to make informed decisions about their athletes.

Concussion incidence has become a topic of considerable discussion and debate amongst researchers, media and the general public. In particular, American football and ice hockey, at all levels of the game, have received a great deal of attention from the media due to the sensational nature of the hits and collisions that perpetuate the injury. Just as more attention has been afforded to this type of injury from the general public and media, so too have researchers studied concussion with greater interest. The number of publications on sport concussion and mild TBI

in sports published from 2000-2006 surpassed the entirety of the literature published on the subject from 1980-2000[5]. Since 2000, there have been four international conferences on concussion in sport, the most recent held in November of 2012 in Zurich, Switzerland[6,7,8]. The aim of these conferences has been to streamline information and provide guidelines for diagnosis and management by medical personnel. Furthermore, these conferences have served to outline directions for future investigation, encouraging more basic and applied science research in targeted areas of need.

Biomechanics

The third international conference on concussion in sport defined concussion as "a complex pathophysiological process induced by traumatic biomechanical forces[8]". It is a form of mild TBI attributable to the transmission of force to the brain from either a direct or indirect impact to the head, face, neck or elsewhere along the body. A recent review article by Meaney and Smith (2011) helped summarize and explain the specific mechanism of action that induces a concussion[9]. There are two categories of force that can impact the skull and brain; contact and inertial. Contact forces occur during impact loading where the head strikes, or is struck by, an object. The primary injury caused by contact forces are skull fractures, which can occur in isolation or lead to subsequent complications such as epidural bleeding. While these types of injuries are common in moderate and severe brain injury, they are largely absent in cases of concussion[10]. The second category of force to impact the skull are inertial forces; those that occur as a result of impulsive head motions (the absence of the head striking an object) and are believed to be more directly responsible for concussion.

While the type of forces applied (contact or inertial) dictate the nature of injury, the manner in which the brain responds to the force is just as significant. There is considerable

evidence that the primary cause of concussion is the acceleration loading experienced by the brain at the moment of contact. Acceleration can be broken down into two components, linear and rotational forces, both of which are found to occur in nearly every instance of concussion. Linear acceleration correlates well as a predictor of the peak pressure that occurs within the brain[11]. The transient increase in pressure caused by the force of impact causes injury to brain tissue and subsequently invokes neurologic dysfunction[12,13]. However, while the brain is highly resistant to changing its shape in response to slow or transient forces, it deforms quite easily when shearing forces are applied, due to the physical properties of the brain itself[14,15,16]. Rapid head rotations generate shear forces in the brain, and therefore, rotational acceleration forces applied to the skull have a high potential to cause shear-induced damage to brain tissue. A number of studies have led to the belief that shear deformation caused by rotational acceleration is the predominant mechanism of action in concussive injury[17,18,19]. Further to the point that rotational acceleration is the critical component in concussive injury, research shows that traumatic unconsciousness is difficult to produce in the absence of rotational forces and their resultant shear deformations[20].

The cervical spine also appears to play an important role in preventing the rotational and linear forces that lead to concussive injuries. Increased neck stiffness has been shown to decrease acceleration of the head in football[21] leading to the hypothesis that increased neck stiffness can decrease concussive impacts; however, a similar relationship was not established in a study of youth ice hockey players[22]. It is believed that in order for cervical muscle strength to have an effect on impact, the musculature must be tense and contracted in order to impart protective stiffness[23]. This may explain concussive incidence in the NFL where the player receiving the hit is concussed more often than the player delivering the hit[24]. The player delivering the hit has

sufficient time to contract the muscle of the neck, sufficiently stabilizing the cervical spine. This is in contrast to the player receiving the hit, who may not be aware of the incoming player, and may not have the requisite muscular contraction required to dampen impact forces.

Pathophysiology

Concussions and their acute symptoms typically represent a functional disturbance within the brain rather than a structural injury, reflected by the generally unremarkable neuroimaging test results[25]. Concussive injury is believed to result from a series of metabolic events within the brain and have been summarized in detail by Giza and Hovda (2001) as well as Giza and DiFiori (2011)[26,27]. Acceleration of the head leads to neuronal deformation and rapid influx/efflux of ions to and from the cell, causing a depolarization of the neuron and release of glutamate, an excitatory amino acid. Glutamate acts to further amplify the excitation phase by promoting further ion release and depolarization of neurons by the activation of N-methyl-d-aspartate (NMDA) receptors. This cascade of events is thought to result in many of the acute signs and symptoms associated with concussions. These include, but are not limited to, confusion, vision/balance deficits and seizures. NMDA also allows a massive influx of calcium into the cell that has a number of consequences including the disruptions of cell mechanisms, inflicting structural damage to the cell. The calcium can also overload the mitochondria, reducing its ability to produce adenosine triphosphate (ATP) and lead to the creation of reactive oxygen species that can cause cell damage and death. The decrease in mitochondrial functioning and ATP production requires the brain to rely on less efficient means of ATP manufacturing, and a mismatch between ATP supply (low) and demand (high) emerges. The end result of the neuronal ion imbalance, compromised mitochondrial respiration and decreased ATP production is a

reduction in cerebral cell viability, eventually leading to apoptosis and consequent neurologic dysfunction.

Signs and Symptoms

The clinical signs and symptoms of a concussion can vary widely and are believed to result from the aforementioned metabolic disturbances occurring within the brain. The signs and symptoms may include: somatic symptoms such as headaches, cognitive symptoms such as the athlete reporting "feeling in a fog," emotional symptoms such as lability, physical signs such as loss of consciousness and amnesia, behavioral changes such as irritability and the exhibition of irregular personality traits, cognitive impairment such as slowed reaction times and/or, sleep disturbances such as unexplained drowsiness[6]. These signs and symptoms may appear immediately, have a delayed onset, overlap or wax and wane throughout the recovery period. Typically, recovery from a concussion manifests in three stages: An acute stage lasting up to 1 week post-injury, a post-concussive stage which may last up to 6 weeks and a prolonged post-concussion syndrome lasting up to 6 months. In rare cases, a fourth stage may appear in the form of chronic traumatic encephalopathy (CTE), a long-term disability similar to neurodegenerative diseases such as Parkinson's and Huntington's disease, and which may manifest after a long and variable latent period[28].

Diagnosis

No one single approach or test is used in the diagnostic process and a multi-disciplinary approach is used instead. It is important to note that previous grading systems have been abandoned in favor of a more individualized, patient-centred approach. As outlined in the 3rd International Conference on Concussion in Sport (2009)[8], diagnosis of concussion should involve a detailed history as well as assessment of clinical symptoms, physical signs, behavior,

balance, sleep and cognition. Sideline evaluation of a concussion in an athletic setting should first and foremost rule out injury to cervical spine and skull. Once critical injuries have been excluded, the athlete should be removed from the field of play and appropriate testing protocols instituted. The Sideline Concussion Assessment Tool 2nd Edition (SCAT2)[29] was developed during the 2nd International Conference on Concussion in Sport (2005)[6] and is a compilation of tools used to aid in concussion diagnosis including a symptom checklist, concentration and memory tasks as well as neurologic screening. While a full neurological exam including cranial nerves, peripheral nerves and cerebellar functioning is often unremarkable, it is important to conduct in order to rule out possible intra-cranial hemorrhaging. Any athlete who is suspected to have suffered a concussion should be immediately removed from their sport and disallowed to return to play on the same day as the injury.

Neuropsychological Testing

Traditional medical tests such as x-ray, magnetic resonance imaging, computed tomography and blood laboratory testing are typically unremarkable in cases of concussion. Coupled with the knowledge that simple intellectual and mental functioning tests do not reveal the true scope of cognitive decline in concussion patients[28], it became clear that a better model of evaluation was required. The use of neuropsychological testing in sports-related concussion was pioneered by Jeff Barth at the University of Virginia in 1989 when his group implemented the evaluation paradigm that is still used to this day[30]. Neuropsychological testing is performed in controlled clinical environments and involves the administrations of a variety of tests that examine cognitive abilities, psychological functioning and sensory and motor functioning in traditional paper/pen, computerized or hybridized models[31]. The premise of neuropsychological testing is that their implementation can be used to detect and quantify cognitive and behavioral

deficits resulting from concussive injury by examining brain-behavior relationships[32]. Testing is performed after a concussion, but only when the athlete has reported a resolution of symptoms. Due to high individual variability in testing and lack of standardized scoring norms, each athlete must have their own pre-season baseline test score for post-concussion analysis and comparison[31]. The popularity and implementation of neuropsychological testing has grown over the years with many elementary, high-school and collegiate football and ice hockey programs throughout Canada and the United States requiring their use in returning athletes to sport after concussive injury[33].

While neuropsychological testing has gained widespread acceptance and is mandated by the National Hockey League (NHL) and National Football League (NFL) for use by all teams, it has been done largely in the absence of evidence that it modifies risk for athletes[33]. Neuropsychological testing requires a player be symptom-free before return to sport; however, same-season repeat concussions are infrequent and the risk of suffering a concussion does not appear to be diminished by ensuring a player is symptom-free before return-to-play[34]. Furthermore, neuropsychological testing does not appear capable of predicting second impact syndrome (a potentially fatal rapid brain swelling after repeat concussion) or late-life consequences of repeated concussions, and as such, their usefulness and widespread acceptance is questionable[33]. Finally, the psychometric properties of neuropsychological testing are not well established, and many athletes have reported purposely failing, or, "sandbagging" their baseline test in order to easily pass their post-concussion test[31,33,35,36,37]. In the absence of valid and reliable tools, neuropsychological testing has filled a void in the diagnosis and management of concussions. While it does serve a useful purpose, it is clear that more research is required before establishing it as a gold standard.

Concussion Management

There are no specific treatments available for concussion and the prevailing protocol involves letting the brain heal by employing patient education, cognitive rest and time[38]. Pharmacologic interventions have been proposed to manage specific symptoms such as anxiety and to modify the pathophysiology of concussion in an attempt to shorten the duration of symptoms. Amitriptyline, corticosteroids, non-steroidal anti-inflammatories, calcium-channel antagonists and nicotinamide have all been used with varying success. The efficacy of such treatments has been questioned as their use is based on severe brain injury research, and therefore, may not be transferrable to the treatment of concussion[38].

In athletic settings, return-to-play protocols are often used to gradually return the athlete to their sport and are based on symptom resolution. Think First is a Canadian non-profit organization dedicated to the prevention of brain and spinal cord injuries that has developed a return-to-play protocol based on the 3rd International Conference on Concussion in Sport (2009)[8,39]. It involves 6 steps (minimum of 1 day per step) that begins with complete cognitive rest and culminates in game play. Any return in symptoms at any point in the process necessitates the athlete return to the preceding step.

The most significant issue regarding return-to-play protocols is athlete compliance. Athletes may often ignore, minimize or hide their symptoms in order to return to their sport earlier[40]. This occurs most often due to parents, teammates and/or coaches placing pressure on the athlete to return to play for a myriad of reasons and significantly decreases the efficacy of return-to-play protocols. One retrospective longitudinal study on 23 hockey players from a variety of skill levels reported that while 65% of the athletes were advised to retire by a neurosurgeon, 33% were non-compliant and returned to play[41]. Unfortunately, a gold standard

for concussion detection and management has yet to be established and current management techniques such as symptom resolution, neuropsychological testing and sideline evaluation tools such as the Sport Concussion Assessment Tool 2 (SCAT2) are the best implements available. It has been suggested that clinical information and physician judgement be given significant consideration in addition to symptom resolution, cognitive assessment and balance testing when assessing the readiness of an athlete to return to play[38].

Long-Term Consequences of Concussion

The long-term sequelae of concussion are still unclear and new research is constantly emerging[42]. Approximately 22 500 new patients each year demonstrate long-term deficits as a result of concussion, approximately equal to the number of patients diagnosed annually with breast cancer, multiple sclerosis and traumatic spinal cord injury combined[9]. The first report of long-term, permanent neurologic sequelae from repeated mild TBI was reported by medical examiner and pathologist Harrison Martland (1928)[43]. The condition was observed in boxers and coined Dementia pugilistica, literally meaning "dementia in a fighter," and known as "punch drunk" by fans due to the symptoms displayed by the fighters. The condition was found to affect boxers of the slugging variety; those who would receive a great deal of punishment to the head while waiting for the perfect opportunity to deliver a knockout punch. It was also commonly found in second-rate fighters who were used for training purposes and likely to be knocked down several times a day. Early symptoms appeared in the extremities and included an occasional flopping of one foot or leg while walking, a slight unsteadiness in gait, periods of slight mental confusion and slowing of muscular action. These early symptoms were well known to boxing fans, and they would often shout "cuckoo" at boxers exhibiting symptoms during a match. Martland hypothesized that 50% of boxers would develop the condition if they continued their

boxing career long enough and further described cognitive, behavioral and motor abnormalities that would emerge as the condition progressed. Dementia pugilistica was compared to other neurodegenerative diseases such as Parkinson's disease and the authors postulated that the neurodegenerative symptoms exhibited were a consequence of repeated mild TBIs over the course of the boxers' careers.

A significant amount of research has been conducted in the decades since the original paper by Martland[43]. Dementia pugilistica was discovered in sports other than boxing and autopsy reports found that the pathology was similar to other neurodegenerative diseases[44]. It was ultimately deemed to be a separate and unique entity, and the term chronic traumatic encephalopathy (CTE), first coined by Miller in 1966, became widespread, eventually replacing dementia puglistica altogether[45]. CTE is thought to be a long-term consequence to repeated concussions, manifesting after a variable latency period, usually after retirement from sport[46]. The condition progresses like most other neurodegenerative diseases that cause dementia; it has a gradual onset and a slow progressive decline in function is observed, ultimately culminating in complete dementia[46]. While exact epidemiological data are unclear, a conservative estimated lifetime prevalence of at least 3.7% has been suggested, which represents a significant public-health risk for those who suffer repetitive concussions[46].

In 2008, the Centre for the Study of Traumatic Encephalopathy (CSTE) was formed as a collaborative effort between the Boston University School of Medicine and Sports Legacy Institute[47]. Their purpose was to conduct research on the neuropathology, pathogenesis, clinical presentation, genetics, risk factors and prevention of CTE. A brain donation registry that allows current and former athletes and military personnel to donate their brain and spinal cord for research purposes after their death has lead to valuable information regarding brains that have

suffered repeated trauma. The CSTE has published several research articles and case reports advancing the knowledge of CTE, mostly on boxers and American football players. In 2009, the CSTE announced that former NHL player Reggie Fleming, an aggressive forward and defenseman with a career spanning twenty years, had suffered from CTE[48]. He was the first NHL player to be diagnosed with the disease, and since then, additional case reports have emerged, including noted enforcer and fighter Bob Probert[49].

The decision to recommend retirement to an athlete following concussion is highly individualized to the athlete and must be a multi-disciplinary decision based on current knowledge of neurology, sports medicine and concussion management[28]. Retiring an athlete due to a concussion is a complex process, but one that must be taken seriously due to the consequences of concussive injury and potential of developing CTE. Recent guidelines have proposed that an extensive history, physical and neurological examination be performed on an athlete with the following criteria taken into consideration: Chiari malformation (downward herniation of the cerebellar tonsils through the foramen magnum), intracranial hemorrhage, diminished academic performance or cognitive abilities, persistent prolonged post-concussion syndrome, lowering of threshold for concussion, 3 or more major concussions, CT or MRI scan documenting structural brain injury, non-resolving functional MRI scan deficit, signs or symptoms consistent with CTE[28]. While no strict number of criteria must be met, the presence of these items should lead the responsible health care practitioner to encourage retirement from sport[4].

Injury and Concussion in Ice Hockey

Ice hockey is a popular sport in North America. The latest report from Hockey Canada cites youth registration rates of close to 600 000 per year and growing[50]. USA Hockey reports

similar membership numbers, and has seen tremendous growth in the game, reporting a 15.6% increase in participation rates since 2005-2006[51]. The game is a fast moving sport played with 5 players and 1 goalie per side[52]. It is an aggressive contact game in which the players move on skates at speeds up to 30 miles per hours and shoot a piece of black rubber with graphite, carbon and wooden sticks at speeds up to 100 miles per hour[53]. Given the often violent nature of the game, it should come as no surprise that ice hockey has the highest rate of concussion incidence amongst contact sports[54]. Of all helmeted sports, ice hockey has the highest concussion rate with 0.27 concussions per 1000 athletic exposures followed by American football (0.25), men's lacrosse (0.19) and women's softball (0.11)[55]. It has been demonstrated to be a problem, particularly in female ice hockey, where higher incidence rates and time lost compared to men have been exhibited[56,57]. While physical contact is allowed in female ice hockey, body checking, a significant injury risk factor, is illegal at all age and skill levels, making this statistic particularly perplexing[2].

Concussion is a known problem at all levels of the game and has been reported to account for 2-14%[58,59,60,61] of all hockey injuries and 15-30% of all hockey head injuries[53,58]. At the minor level (adolescents), concussion has been shown to represent 15% to 18% of all reported injuries, and the risk of concussion is significantly greater in age and skill groups that permit body checking[62,63,64]. At the Canadian amateur tier II level (16-20 years of age), one study examined the various aspects of concussion over two seasons in the British Columbia Junior Hockey League (BCJHL), noting incidence rates of 7.42% for the first season and 10.66% for the second season[54]. The authors hypothesized that these percentages may represent the lower boundaries of concussion incidence, as the retrospective histories of players participating in both study years may have led to underreporting. One study examining tier IV junior ice hockey

players reported 21.52 concussions per 1000 athlete exposures. Furthermore, the authors reported that 24% of those concussed were directly involved in a fight immediately prior to their diagnosis[65]. No studies have examined tier I amateur hockey in Canada (Canadian Hockey League); however at the American collegiate level (NCAA), concussion incidence rates have varied between studies, ranging from as low as 9.0% to as high as 18.0% overall, and from as low as 0.41 concussions per 1000 athletic exposures to as high as 4.9 per 1000 athletic exposures[2,57,66,67]. Part of the large variance can be explained by different methods and measures employed, as it becomes difficult to draw comparisons and conclusions across studies when injury rates are expressed using dissimilar methods (athletic exposures vs. game-hours vs. overall percentages)[64].

Hockey at the highest level in North America takes place in the NHL. At least eight players between 1993 and 2003 were forced to retire prematurely because of repeated concussions, and many more missed weeks, months and even entire seasons because of lingering symptoms[68]. However, these data are considered conservative and other estimates have placed the number of concussion-related retirements at double or more[69]. Epidemiological data on concussion incidence rates in the NHL are sparse with only four studies found during a literature search[70,71,72,73]. Part of the lack of research is attributable to the reluctance of professional sport leagues in North America to make epidemiological data available to the public due to stated issues of medical confidentiality[74].

Of the four studies conducted, the first by Wennberg and Tator (2003)[70], studied concussion in the NHL using data from published media reports, which have been cited as reliable source of injury information[70,72,74]. In their justification for using media reports, Wennberg and Tator explained that injuries suffered by professional athletes are one component

of the overall entertainment package of professional sport, and that journalists and public demand eventually uncover the nature of most injuries suffered by professional athletes. The authors found the concussion rate in the NHL from the 1997-1998 to 2001-2002 seasons was more than triple the rate of the previous decade. From the 1986-1987 to 1995-1996 seasons there was a mean of 12 concussions per season. From the 1996-1997 to 2001-2002 seasons there was a mean of 56 concussions per season. The authors interpreted this drastic increase as increased medical recognition and reporting of concussion attributable to the commencement of the NHL Concussion Program in 1997.

In the second study, Wennberg and Tator (2008) conducted a follow-up investigation documenting the incidence of concussion and time lost from play due to concussive injury during the 1997-1998 to 2007-2008 seasons[72]. The authors found the mean incidence over the ten seasons to be 1.45 concussions per 1000 athletic exposures, and reported a significant downward trend in concussions over that time period. In contrast to this decrease, a gradual increase in the average number of games missed per concussion was recorded and the mean number of missed games per concussion during the last five seasons was significantly greater than during the first five seasons. The authors hypothesized that this may represent an increase in concussion severity, or perhaps, more stringent concussion management protocols that are more rigid in their return-to-play timelines. Without access to NHL data, an issue fraught with concerns regarding medical confidentiality, it is impossible to discern which is more likely to account for the increase in games missed per concussion in the latter five seasons examined.

Recognizing a growing injury problem within the league, the NHL, in conjunction with the National Hockey League Player Association (NHLPA), established the NHL-NHLPA Concussion Program in 1997, with the purpose of examining the science of concussion as well as

the education of its members on all aspects of the injury[71]. The program helps the NHL accumulate data on concussion incidence, mechanism of injury and return-to-play (RTP) timelines. The data collected have not been released to the public and only two studies have been published to date. The first was an abstract authored by Meeuwisse, Burke and Benson (2003) reporting data collected from team physicians who reported on all players sustaining concussions during the 1997-1998 to 2002-2003 NHL regular seasons[71]. They reported an average of 97 concussions per year and found that the median games missed per concussion doubled from one to two over the 5 years of the study. As it was only an abstract, no detailed statistics were provided.

To date, only one detailed study has emerged from the NHL Concussion Program. Benson et al. (2011) had NHL team physicians document all concussions sustained during the 1997-1998 to 2003-2004 seasons in a prospective case series format[73]. The authors reported a total of 559 physician-diagnosed, regular season in-game concussions with a mean of 80 concussions per season. The median time lost from play was six days. In line with the two studies by Wennberg and Tator[70,72], the authors established that concussion rates were declining, from 7.7 concussions per 100 players during the 2001-2002 season down to 4.9 per 100 players during the 2003-2004 season. The authors concluded that this may be due to a variety of reasons including under-reporting of symptoms by players to avoid missing time, more conservative management by team medical personnel, higher thresholds for diagnosis of concussion and increased use of neuropsychological testing results before making RTP decisions. The results of the studies, while indicating a decrease in concussion incidence, should be taken with caution. The authors stated these results may have been due to underreporting, and that more conservative

management of concussions theoretically helped prevent repeat incidents by ensuring complete recovery before return to play.

Risk Factors for Injury and Concussion in Ice Hockey

Like any injury or disease, it is important to know not merely how often it occurs, but to understand risk factors to aid in preventative methods. Much of the research on ice hockey injury risk factors has been generalized to all injuries (i.e., not concussion-specific). The most commonly investigated risk factors include age, session type, level of play, player position and the presence/absence of body checking[75]. Additional factors such as participation in fair play programmes, aggression and empathy, weight and height, level of hockey experience, relative age and gender have received limited attention and have produced conflicting results[75]. Furthermore, the majority of this research has been conducted on youth hockey, leaving a noticeable gap in our understanding of risk factors for injury and concussion at the adult and NHL levels.

Importantly, the studies that have examined these relationships in younger populations have produced mixed results. For example, of three studies examining the role of player position at the youth ice hockey level, two found forwards to be at higher risk of injury than defensemen,[76,77] while the third reported the relative risk of injury for defensemen to be 2.18 times that of forwards[78]. All three studies found goalies to be at a much lower risk of injury. While the methodological differences (different skill levels, age groups, tournament vs. season play) between the three studies were significant, they do not fully explain the drastically different findings.

There have also been mixed results for the influence of player age on injury and concussion risk. A recent systematic review examining age as a predictor of overall injury in youth ice hockey found that of the eight studies examined, five reported that risk of injury went up with increasing age, while the remaining studies found no difference, or found that older players were at lower risk than their younger counterparts[75]. Interestingly, one study examined the concept of 'relative age' (age relative to peers) and its relationship to injury[79] noting that relatively older children within ice hockey age groups were at increased risk of injury compared to their younger counterparts, and that this risk was exacerbated at higher competition levels. The relationship between age and concussion has been somewhat clearer, with older players at higher concussion risk compared to younger players[80] and Bantam and Pee Wee players at higher concussion risk compared to Atom-aged players[63].

Height and weight have also been purported to be injury risk factors in ice hockey, but the research does not necessarily support this conclusion[75]. Brust et al (1992) found that bodyweight was only a significant risk factor at the Bantam level[80]. The authors postulated that in this age group (13-14 years of age), players' physical development accelerates due to puberty and large size discrepancies may emerge and account for the injury risk. Wiggins (1999) reinforced the notion that bodyweight is a predictor of injury, concluding that lighter players were more likely to be injured than heavier players when bodychecked[76]. Conversely, Smith et al. examined 86 male high school ice hockey players (three varsity and junior varsity teams) during the 1994-1995 season[81] and concluded that neither height nor weight was related to injury, although they noted that weight only narrowly missed significance in their logistic regression analysis. More conflicting and somewhat confusing evidence has demonstrated that bodyweight was a risk factor for shoulder injury among high school players; however, it was the

heavier players at greater risk[82]. It has been hypothesized that concussion incidence and severity will continue to increase if athletes continue to grow bigger, stronger and faster. This has been attributed to simple physics, as the magnitude of any collision is dictated by the size and speed of the two objects colliding[4].

It has been suggested that fatigue contributes to increased injury risk in both rugby[83] and soccer,[84] and it has been reported that concussions are more likely to occur under fatigued conditions in female ice hockey[56]. In male youth and Junior A ice hockey, time-on-ice has been implicated as a risk factor for concussion[85] and injuries in general[81]. One study examining fatigue in the NHL found there was a significant effect for time-on-ice per game[86]. Those who played more minutes over the course of a game had an increased likelihood of suffering a concussion. The authors suggested that in-game fatigue may explain their findings, that is, players may suffer a concussion more often when fatigued due to a reduction in physical abilities and awareness.

Rationale for the Current Study

Most of the evidence for concussion risk factors in the NHL is based on minor, junior and collegiate hockey research, and the results are often conflicting. While some studies have been performed at the NHL level, the majority of the research is epidemiological in nature, and studies examining concussion risk factors are still lacking. While statistics are readily available for position, age, height, weight and average time on ice (ATOI), no relationship has been conclusively established between these variables and concussion risk.

Position has only been considered descriptively, with 341 (64.1%) of concussions suffered by forwards, 179 (31.4%) by defensemen and 24 (4.5%) by goalies, all between the

1997-1998 and 2004-2005 seasons[73]. The only available research on concussion and age in the NHL cited the median age of concussion to be 27 years[73]. As noted earlier, part of the increasing incidence in concussion has been attributed to bigger, stronger and faster athletes, and the correspondingly increased collision magnitudes. Demographic data from the NHL between 1986-1987 to 2001-2002 corroborates the notion that NHL players are getting progressively taller and heavier[70]. Over the 16-year period analyzed, there was a height increase of one inch and weight increase of 10-11 pounds. While it has not been demonstrated via research, it seems reasonable that as players continue to grow in both height and weight, collision magnitudes will continue to increase, and concussion incidence and severity may increase accordingly. The only available data on ATOI in the NHL states that playing more than 15.22 minutes per game (median) had significantly more concussions than those who played less than 15.22 minutes per game[86].

Position, age, height, weight and ATOI are all variables that have some or conflicting evidence and that require further clarification. The objective of this study was to a) provide a descriptive analysis and profile of concussed NHL players and b) determine whether any link exists between concussion incidence and position, age, height, weight and ATOI in NHL players. Based on prior research we hypothesized that forwards, older, taller, heavier and those who play more often will be at greater risk of concussion; however, due to mixed results for many of these relationships in younger samples, we considered this hypothesis to be exploratory.

Methods

Data were collected for every player on all 30 NHL team rosters for the 2005-2006 to 2011-2012 seasons. Any player listed on the NHL roster was considered for the final analysis. Data collection began in the 2005-2006 season because of multiple rulebook changes altering the style and strategies at the professional level that occurred at this time[87]. Specifically, the league office instituted a number of rule changes in an attempt to crackdown on injurious situation, speed up the pace and tempo of the game, as well as allow smaller, more skilled players the opportunity to succeed[87].

The exposure variables were position (forward or defenseman), age (years), height (inches), weight (pounds) and ATOI (minutes:seconds). Information on goaltenders was collected but omitted from the final analysis due to the different nature of the position. Goaltenders are not exposed to the same level of physical contact during the course of play as forwards and defenseman. Additionally, goaltenders are on the ice and involved for the full 60 minute game, while even a cursory review of ice time statistics reveals that forwards and defenseman play a maximum of 20-25 minutes and 25-28 minutes, respectively. The decision to include ATOI was based on previous research identifying it as a risk factor[86]. It is believed that fatigued muscle has a decreased ability to absorb force[88], and as such may, place the player at a greater risk of concussion. The outcome variable was whether the player suffered a concussion, and was simply recorded as "yes," or "no."

Player information (position, age, height, weight, ATOI), with the exception of concussion status, was collected from *Hockey-Reference*, an open-access website dedicated to collecting historical and current NHL statistics[89]. Their data were derived primarily from two

major sources. The first source was Dan Diamond and Associates, the NHL's consulting publisher and producer of hockey statistics. Dan Diamond and Associates have published the annual *NHL Official Guide & Record Book* for the past 25 years. The second major data source was XML *Team Solutions*, a company that provides content solutions for those who depend on sports news and data.

Each season was hand-sorted and data were exported for the individual teams into an excel document for coding and analysis. Position was categorized as forward or defence, with goaltenders omitted for the aforementioned reasons. Age, height and weight were separated into three categories. Within each of the three variables, division into categories was performed in an attempt to achieve equality (33.3% per category) and for easier separation into risk categories during the final analysis. Average age, height and weight data for each season were provided by *The Hockey News* and verified via collected data[90]. These data averages were split into two groups, one for goaltenders and the other for skaters (forwards and defensemen). ATOI was divided into 5-minute categories up until 25-30 minutes. Again, this was done for easier division into risk categories during the final analysis.

Incidence of concussion was extracted from *The Hockey News*[90], a weekly sports magazine that publishes news, editorial content and statistics related to the NHL. Injury data published by *The Hockey News* were derived from team correspondents who file weekly injury reports based on the injury reports released by the NHL, which in turn, are derived from reports submitted by the public relations offices of each team on behalf of the team physician/trainer. The decision to use media-derived injury reports was based on previous work by Wennberg and Tator (2003)[70], who examined concussion data from the 1999-2000 to 2000-2001 and found

nearly identical concussion data reported by the NHL and *The Hockey News*. Other studies, notably Wennberg and Tator (2008)[72] and Stevens et al. (2008)[86] have used similar methods.

Starting from September 2005 until June 2012, weekly injury reports were searched for the following key words: *concussion, head injury* and *headache*. Head injury' and 'headache' were searched given the proximity of these descriptors to the signs and symptoms of what may be reported in a concussion. The injury information was recorded if 'concussion' was listed in the description. If 'head injury' or 'headache' was listed in the injury description, the player's activity was tracked to determine whether the injury descriptor was later changed to 'concussion'. If it was, then the injury was recorded, if it was not changed then it was not recorded. Age was recorded at the time of injury to more accurately describe the age variable (note: age was otherwise recorded as of Feb 1 for each player in the given season). Multiple concussion incidences were recorded for the same player, but only one entry per player per season was used in the final analysis as including multiple entries for the same player would artificially increase the sample size.

The final analysis was performed using IBM SPSS statistics version 20. Descriptive statistics were obtained for the NHL and concussed-only datasets. Chi-square analysis determined whether there were any significant associations between the exposure variables. A one-way ANOVA with Tukey post-hoc analysis was performed to test for differences in concussion reporting across the seven seasons analyzed. Finally, stepwise logistic regression was used to model the concussion risk for each of the exposure variables. This was done by creating two models; the first included all study exposure variables (full model), while the second included only variables that were significant in the chi-square test (parsimonious model). Chi-

square analysis and logistic regression was also performed independently for each individual season. Statistical significance was set at $p < 0.05$ for all of the analyses performed.

Results

A total of 6145 NHL players over seven seasons were included in the final analysis. Table 1 demonstrates the NHL database calculations used to arrive at the final study population. Table 2 demonstrates how the reported concussion totals were tabulated.

Table 1. NHL database calculations

Season	NHL Population	Number of Goaltenders Omitted	Players Out for the Season Added	Final Population
2005-2006	961	91	1	871
2006-2007	942	84	2	860
2007-2008	941	89	2	854
2008-2009	974	89	2	887
2009-2010	962	83	1	880
2010-2011	978	87	4	895
2011-2012	983	89	4	898
Totals	6741	612	16	6145

Table 2. Reported concussions calculations

Season	Raw Number of Reported Concussions	Number of Repeat Incidences	Number of Concussed Goaltenders	Final Reported Concussion Totals
2005-2006	55	6	2	47
2006-2007	63	5	3	55
2007-2008	60	5	2	53
2008-2009	63	7	2	54
2009-2010	68	5	1	62
2010-2011	86	6	1	79
2011-2012	117	13	7	97
Totals	512	47	18	447

Descriptive statistics for the NHL database revealed that age decreased slightly over the 7 seasons, from an average of 27.17 years in 2005-2006 to 26.61 years in 2011-2012. Height also remained relatively stable, decreasing slightly from 73.27 inches in 2005-2006 to 73.13 inches in 2011-2012. Weight saw the biggest decrease, from 206.29 pounds in 2005-2006 to 203.28 pounds in 2011-2012. Appendix A demonstrates complete descriptive statistics for the NHL database by season. Descriptive statistics for the concussed sample (N = 447), revealed a mean of 27.75 years of age, 72.99 inches and 203.76 pounds. Appendix B demonstrates descriptive statistics for the concussed sample only.

Of the 6145 players analysed, 447 (7.3%) suffered a concussive injury. There was a gradual increase in the percentage of NHL players concussed per season, from 5.4% in 2005-2006 to 10.8% in 2011-2012. Defensemen had higher reported concussion totals compared to

forwards (7.93% vs. 6.93%), while the greatest proportion of concussions occurred in the oldest age group (29-48) at 8.18%. The highest number of reported concussions for height occurred in the shortest group (65-72 inches) at 7.9%. Weight also saw the highest concussion incidence in the lightest group (144-197 pounds) at 7.8%. There was increasing reported concussions with increasing ATOI up to the 15-20 minute group, where it peaked at 9.48%, and then dropped off slightly in the remaining two categories. Table 3 illustrates the frequency distribution of concussion in the NHL database by exposure variable.

Table 3. Frequency distribution of the sample population by exposure variable

	Concussed	Not Concussed	Total Sample Size	Percentage of Sample Concussed
Season				
2005-2006	47	824	871	5.40
2006-2007	55	805	860	6.40
2007-2008	53	801	854	6.21
2008-2009	54	833	887	6.09
2009-2010	62	818	880	7.05
2010-2011	79	816	895	8.83
2011-2012	97	801	898	10.8
Position				
Forward	280	3760	4040	6.93
Defence	167	1938	2105	7.93
Age (Years)				
18-24	164	2066	2230	7.35

25-28	117	1769	1886	6.20
29-48	166	1863	2029	8.18
Height (Inches)				
65-72	180	2091	2271	7.93
73-74	168	2112	2280	7.37
75-81	99	1495	1594	6.21
Weight (Pounds)				
144-197	157	1857	2014	7.80
198-210	165	2028	2193	7.52
211-270	125	1813	1938	6.45
ATOI				
0-5 min	6	164	170	3.53
5-10 min	41	973	1014	4.04
10-15 min	121	1824	1945	6.22
15-20 min	195	1862	2057	9.48
20-25 min	63	793	856	7.36
25-30 min	5	82	87	5.75
Did Not Play*	16	0	16	100.0
Totals	447	5698	6145	7.3

*Player was listed on the NHL roster but did not play at all that season due to concussion

Chi square analysis revealed a significant effect for season $\chi^2(6, N = 6145) = 28.67, p = .00$, age $\chi^2(2, N = 6145) = 5.70, p = .05$ and ATOI $\chi^2(6, N = 6145) = 241.53, p = .00$ in regards to reported concussions. There were no significant effects for position, height or weight. Figures 1-3 demonstrate significant results.

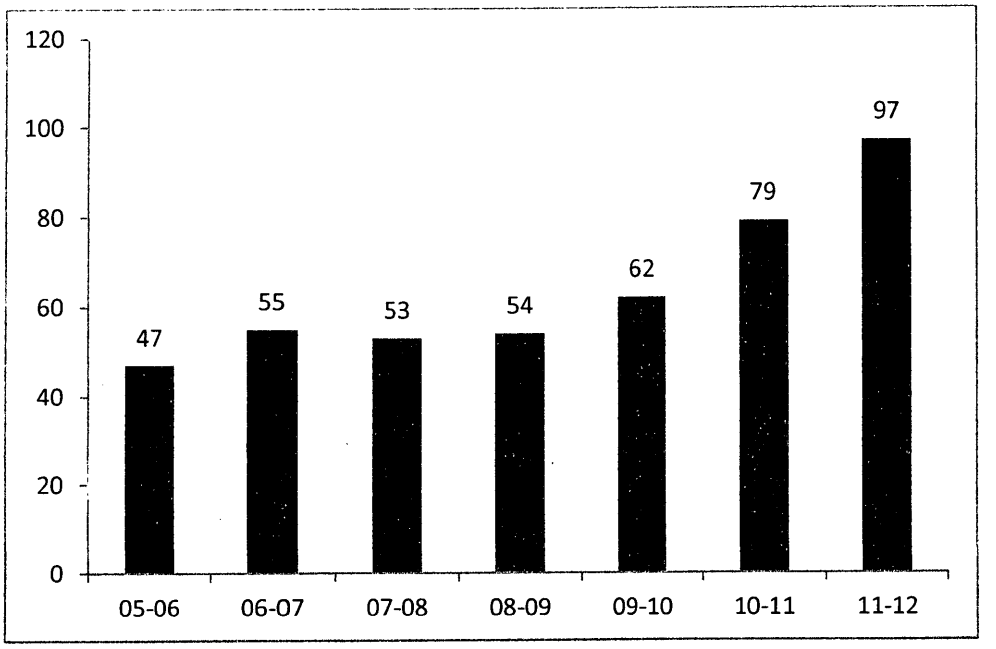

Figure 1. Reported concussion totals in the NHL by season

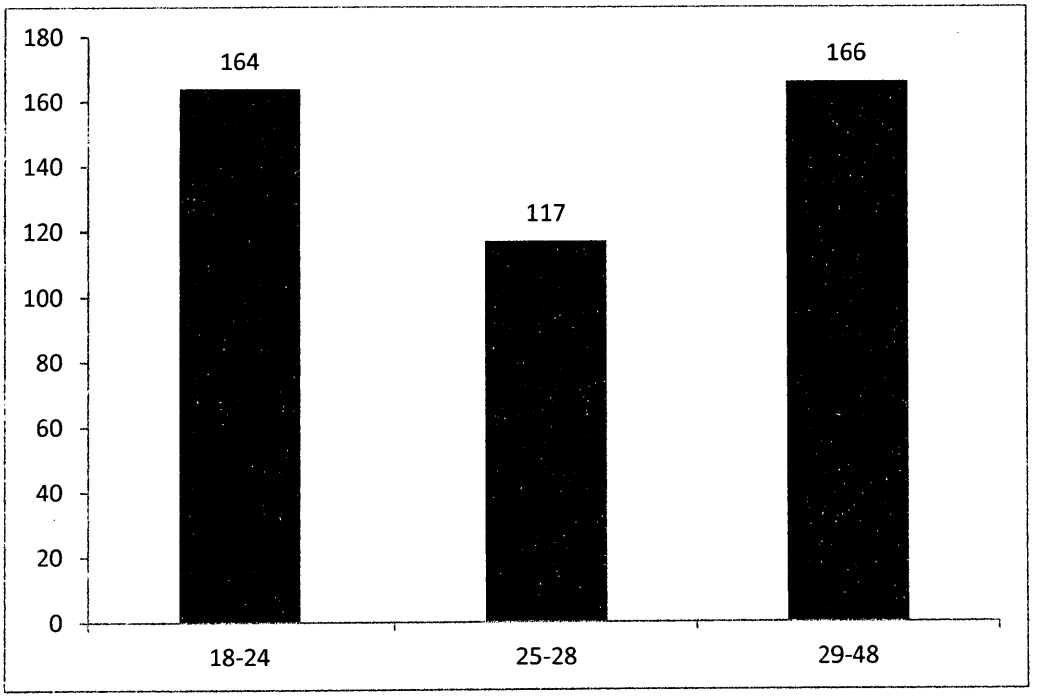

Figure 2. Reported concussion totals in the NHL by age group

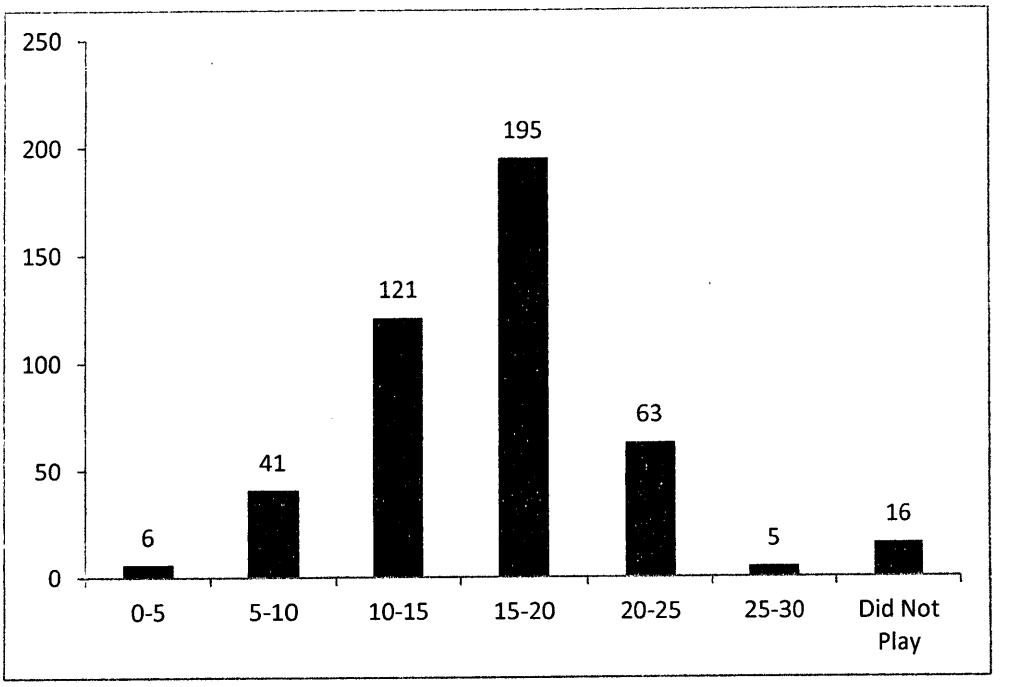

Figure 3. Reported concussion totals in the NHL by average time on ice

When examined independently, chi-square analysis revealed a significant effect for reported concussions in regards to ATOI in the 2005-2006 season: $\chi^2(6, N = 871) = 21.73, p = .001$, 2006-2007 season: $\chi^2(6, N = 860) = 33.16, p = .00$, 2007-2008 season: $\chi^2(6, N = 854) = 34.70, p = .00$, 2008-2009 season: $\chi^2(6, N = 887) = 44.74, p = .00$, 2009-2010 season: $\chi^2(6, N = 880) = 22.24, p = .001$, 2010-2011: season $\chi^2(6, N = 895) = 55.09, p = .00$, and 2011-2012 season: $\chi^2(6, N = 898) = 44.92, p = .00$. Significant effects for age were found in the 2008-2009 season: $\chi^2(2, N = 887) = 6.79, p = .033$ and 2011-2012 season: $\chi^2(2, N = 898) = 8.30, p = .016$.

A one-way ANOVA was performed to test for reported concussion differences amongst the exposure variables during the seven seasons analyzed. The significant variables included age: $F(6, 6138) = 2.40, p = .03$, weight: $F(6, 6138) = 2.85$ $p = .01$ and reported concussions: $F(6, 6138) = 4.80, p = .00$. Tukey post-hoc comparisons indicated a significant difference in age between the 2005-2006 and the 2009-2010 and 2010-2011 seasons. There was also a significant

difference in weight between the 2005-2006 and 2011-2012 seasons. Finally, there was a significant difference in reported concussions between the 2011-2012 season and the 2005-2006, 2006-2007, 2007-2008, 2008-2009 and 2009-2010 seasons. There were no other significant relationships found amongst the variables. As a result of these findings, season was controlled in the regression analysis to prevent its possible confounding influence on the relationships under examination.

Logistic regression was performed to model the odds of concussion risk based on the exposure variables. Significant effects were found for both season and ATOI. The results can be seen in the full and parsimonious models (Tables 4 and 5).

Table 4. Full model logistic regression results

		Odds Ratio	95% Confidence Interval	
			Lower Bound	Upper Bound
Step 1				
	Season			
	2005-2006	1.00	-	-
	2006-2007	1.12	.802	1.79
	2007-2008	1.16	.774	1.74
	2008-2009	1.14	.760	1.70
	2009-2010	1.33	.899	1.97
	2010-2011	1.70	1.17	2.47
	2011-2012	2.12	1.50	3.05
Step 2				
	Position	1.11	.878	1.39

Age				
	18-24	1.00	-	-
	25-28	.818	.636	1.05
	29-48	.992	.782	1.26
Height				
	65-72	1.00	-	-
	73-74	.897	.699	1.15
	75-81	.772	.550	1.08
Weight				
	144-197	1.00	-	-
	198-210	1.02	.791	1.31
	211-270	.980	.705	1.36
ATOI				
	0-5	1.00	-	-
	5-10	1.01	.422	2.44
	10-15	1.53	.659	3.55
	15-20	2.42	1.05	5.59
	20-25	1.82	.759	4.34
	25-30	1.41	.409	4.86
	Did Not Play	-	-	-

Table 5. Parsimonious model logistic regression results

	Odds Ratio	95% Confidence Interval	
		Lower Bound	Upper Bound
Step 1			
Season			
2005-2006	1.00	-	-
2006-2007	1.20	.802	1.79
2007-2008	1.16	.774	1.74
2008-2009	1.14	.760	1.70
2009-2010	1.33	.899	1.97
2010-2011	1.70	1.17	2.47
2011-2012	2.12	1.48	3.05
Step 2			
Age			
18-24	1.00	-	-
25-28	.814	.634	1.05
29-48	.986	.778	1.25
ATOI			
0-5	1.00	-	-
5-10	1.07	.447	2.57
10-15	1.66	.717	3.83
15-20	2.64	1.15	6.07
20-25	2.01	.855	4.75
25-30	1.57	.464	5.31

Did Not Play	-	-	-

When examining the seven seasons independently, logistic regression revealed significant effects for age in the 2008-2009 season. Those in the 25-28 age bracket were 63.1% (CI = .161 - .843) less likely to suffer a concussion than the 18-24 age bracket. Significant effects for age were also found in the 2011-2012 season. Those in the 25-28 age bracket were 46.1% (CI = .306 - .952) less likely to suffer a concussion than the 18-24 age bracket. Finally, logistic regression revealed significant effects for height in the 2010-2011 season. Those in the 75-81 inch category were 59.7% (CI = .173 - .939) less likely to suffer a concussion than the 65-72 inch category.

Discussion

Summary

This thesis provided a descriptive analysis and profile of concussed NHL players over the course of seven seasons (2005-2006 to 2011-2012), and examined relationships between reported concussions and position, age, height, weight and ATOI. There were several significant results: Chi-square analyses displayed a significant effect for reported concussions for the exposure variables of season, age and ATOI. One-way ANOVA demonstrated significant between-season differences for age (2005-2006 compared to the 2009-2010 and 2010-2011 seasons), weight (2005-2006 compared to 2011-2012) and reported concussions (2011-2012 compared to 2005-2006, 2006-2007, 2007-2008, 2008-2009, 2009-2010), however, the significant differences for age and weight disappeared during further testing. Logistic regression analysis revealed that players were 1.7 and 2.1 times more likely to suffer a concussion in the 2010-2011 and 2011-2012 seasons (respectively) than in the 2005-2006 seasons. In regards to ATOI, players in the 15-20 minutes category were 2.6 times more likely to be concussed than those in the 0-5 minute category. No significant findings were present for the exposure variables of position, age, height and weight.

Reported Concussions in the NHL

The descriptive analysis of concussion in the NHL offers valuable insight when placed in the context of relevant studies. The present overall reported concussion totals of 7.3% is lower than both tier II British Columbia Junior Hockey League at 7.42% and 10.62%[54], and NCAA rates, which range between 9.0% to 18.0%[2,57,66,67]. However, the British Columbia Junior Hockey League and the NCAA differ in two significant ways from the NHL, namely age and skill level. As such, it is hard to extrapolate these results to different populations, and thus, they

should be interpreted with caution. Wennberg and Tator (2003)[70] provide some insight into concussion incidence in the NHL, reporting a mean of 12 concussions per season between the 1986-1987 to 1995-1996 seasons, and a mean of 56 concussions per season between the 1996-1997 to 2001-2002 seasons. The drastic increase in concussion incidence from the first to second portions of the Wennberg and Tator study may be attributed to the commencement of the NHL Concussion Program at the beginning of the 1997-1998 season, leading to better recognition and reporting of concussion. The present study reported between 47-97 concussions per season between the 2005-2006 and 2011-2012 seasons, indicating that reported concussions are on the rise. The continuing increase indicates that reported concussions continues to increase despite the NHL-NHLPA Concussion Program and its attempt to curb incidence. Compounding the matter, concussion figures reported here should be considered conservative estimates given the prevalence of underreporting in athletic settings; moreover, the present study did not include goaltender concussions or repeat concussion in the same season.

Conflicting results from the NHL Concussion Program further complicates attempts to understand concussion incidence rates. Meeuwisse, Burke and Benson (2003)[71] reported an average of 97 concussions per year between the 1997-1998 and 2002-2003 seasons, a similar time-period to that examined by Wennberg and Tator (1996-1997 to 2001-2002)[70]. However, the former study found much higher concussion incidence rates than the latter. It is possible that the data provided by Meeuwisse, Burke and Benson are more accurate due to direct access to NHL concussion data. Unfortunately these data are not openly accessible to researchers due to issues of medical confidentiality, making it difficult to explain the discrepancy. Benson et al. (2011)[73] reported a total of 559 concussions, and mean of 80 per season between the 1997-1998 and 2003-2004 seasons. The present study reported a mean of 64 concussions per season between the

2005-2006 and 2011-2012 seasons, higher incidence than Wennberg and Tator, but much lower than data provided by the NHL Concussion Program.

The time period examined in the present study may explain this large discrepancy. Data examined here were from the period of time following the NHL lockout that cancelled the entire 2004/2005 season. When the NHL resumed operations the following season, a number of rule changes were implemented to protect players, with one of the stated intentions being to curb concussion incidence[87] (the most current version of the NHL Rulebook can be found at http://www.nhl.com/nhl/en/v3/ext/pdfs/2011-12_rule_book.pdf). The most pertinent changes included granting the linesman discretion to "wave off" icing if the race to the puck was, in the linesman's opinion, a potentially injurious situation. Tougher rules were instituted on players instigating fights, with automatic one-game suspensions and harsher sentences with each additional incident. Finally, officials were instructed to impose a zero-tolerance rule on interference, hooking and holding/obstruction infractions in order to increase the tempo of the game and cut down on potential injuries. It is possible that these rule changes and their stricter enforcement by officials may explain the lower reported concussion totals reported in the current study.

In addition to the rule changes noted above, in March of the 2011-2012 season, the league instituted supplementary processes in an attempt to curb concussion incidence[91]. Measures were taken to reduce the size of equipment to lessen the impact force of hard plastic shells contained within shoulder and elbow pads. Additionally, the NHL Protocol for Concussion Evaluation and Management was revised to reflect more stringent in-game testing, including the mandatory removal of a player from the game if signs and symptoms of concussion are observed by the team medical staff. The player must be evaluated in a 'quiet room' away from the rink and

pass sideline concussion management testing before return to play is permitted. Given how late into the 2011-2012 season these measures were implemented, it is unlikely that their success or failure would have affected the results of the present study.

It is possible that the numerous rule changes implemented after the 2004-2005 lockout to protect players may account for the lower reported concussion totals found presently as compared to Meeuwisse, Burke and Benson (mean of 97 concussions per season between 1997-1998 to 2002-2003)[71] and Benson et al. (mean of 80 concussions per season between 1997-1998 to 2003-2004)[73]. The significant issues of underreporting, more stringent evaluation criteria and conservative management protocols may all have contributed as well. The present study is also the first to examine reported concussions in the NHL following the lockout of 2004-2005. While the present concussion totals are lower than previous work cited by the NHL-NHLPA researchers, there was an upward trend in reported concussions with a sharp increase in the 2011-2012 season. Based on the present findings, it appears that reported concussions are once again rising to pre-lockout levels. It has been over 10 years since the NHL instituted their concussion program, and while our knowledge regarding concussion evaluation and management is constantly evolving and improving, it is reasonable to question whether 'better detection methods' still constitutes a sufficient explanation for rising reported concussions. More research using the NHL's concussion data would likely help solve this question and many of the remaining questions, shedding light on the true nature of concussion incidence in the NHL.

A number of risk factors were examined in an attempt to further understand reported concussions in the NHL. Some of these risk factors help elucidate on the increasing reported concussion totals discussed above and will be discussed in the following sections.

Risk Factors: Position

Research examining differences in injury risk across playing positions has been conflicting[76,77,78]. At the NHL level, Benson et al. (2011)[73] reported that of all concussed players, 64.1% were forwards and 31.4% were defensemen. These statistics are similar to the present study that reported 62.6% of concussions suffered by forwards and 37.3% by defensemen. However, the overall percentage of the sample that suffered a concussion was similar for the two positions, 6.9% of forwards and 7.9% of defenseman. Tests of difference between the positions were non-significant, and as such, there is no previous or current data available to suggest that position is a risk factor for concussion in the NHL.

Risk Factors: Age

The mean age of concussion found presently was 27.25 $\pm$4.9 years, which was similar to data from Benson et al. (2011)[73] who reported a mean age of 27. When examining concussion incidence, age was a significant factor in the chi-square analysis. The 25-28 years of age category (i.e., the middle age category) suffered the lowest incidence of concussions (6.2%) compared to the 18-24 category (7.35%) and the 29-48 category (8.18%). While there was no significant age effect found in the logistic regression for the seven seasons combined, significance was found during the 2008-2009 and 2011-2012 seasons when analyzed independently. During the 2008-2009 season, those in the 25-28 age bracket were 63.1% less likely to suffer a concussion than the 18-24 age bracket. During the 2011-2012 season, those in the 25-28 age bracket were 46.1% less likely to suffer a concussion than the 18-24 age bracket. Additionally, there was a significant difference in age between the 2009-2010, 2010-2011 seasons and the 2005-2006 season.

It has been demonstrated that adolescent brains (high-school students) take longer to recover from sport-induced concussion than early-adult brains (college students), indicating that younger athletes should be managed more conservatively in their return-to-play protocols. However, no research has examined whether older/younger brains or more or less susceptible to the initial concussive insult in an athletic setting[92,93]. Results from the 1996-1997 Canadian National Population Healthy Survey representing approximately 28.6 million Canadian citizens found that amongst males, the 15-34 age group had the highest concussion prevalence rate at 260 concussions per 100 000 citizens. This was followed by the 0-14 age group at 170 per 100 000, and finally, the 35+ age group at 50 per 100 000. However these statistics are not sport specific, the age ranges are quite broad and the 15-34 group crosses key developmental periods during adolescence that could possibly affect the susceptibility of the brain[94]. The somewhat conflicting evidence makes interpreting the results of the current study difficult, highlighting the need for more research to determine whether the brain is more susceptible to concussions at certain ages and developmental periods.

It appears there was a trend to younger and older players suffering concussions at an increased frequency, supported by significant findings in the logistic regression during two seasons (2008-2009 and 2011-2012). One possible explanation for the present findings may be due to the style of play in the younger and older groups. It is possible that the younger age bracket is more reckless in their play. This group is trying to break into the NHL, get noticed and make an impression on their teammates and coaches. Similarly, it is possible that the oldest age bracket is trying to play for one last contract and playing with more reckless abandon in order to remain in the NHL. In contrast, the middle age bracket is likely on their first long term contract and perhaps more comfortable in their surroundings and likely to play a relatively safer and

conservative style. While this is mere speculation, it does offer some thought to future research ideas.

Risk Factors: Height and Weight

It has been purported that part of the concussion problem in the NHL is a result of athletes continually growing bigger, faster and stronger[70]. This assertion was partially validated by Wennberg and Tator (2003)[70] who observed increases in height and weight and corresponding rising concussion incidence. They reported an increase of one inch and almost 10 pounds from the 1986-1987 to 2001-2002 season (72 inches, 191 pounds to 73 inches, 200.1 pounds). However, over the 7 seasons analyzed presently, height remained consistent (73 inches), while weight decreased approximately 3 pounds, from 206.29 pounds to 203.28 pounds. The reduction in weight may be attributable to the rule changes implemented by the NHL after the 2004-2005 lockout. These changed, mentioned previously, were made in an attempt to allow smaller, more skilled players a chance to succeed in a league that had become dominated by larger more physical players. It is possible that the success of the rule changes allowed smaller players a greater chance to succeed, making their presence in the league more likely, and decreasing average weights. For instance, Nathan Gerbe (65 inches, 178 pounds), Brian Gionta (67 inches, 175 pounds), Martin St. Louis (68 inches, 176 pounds) and Claude Giroux (71 inches, 172 pounds) are all established NHL players on the low end of either height or weight categories who have emerged since the 2004-2005 lockout, and otherwise may not have had a chance to succeed. These findings are supported by a significant difference in weight between the 2005-2006 season and the 2011-2012 season.

That height is not a significant factor in concussion incidence may be a result of the relatively homogenous sample used. The average height over the seven seasons analyzed was

just over 73 inches with a standard deviation of approximately 2 inches. Compared to the relatively larger 15 pound standard deviation for weight, it is plausible that at the NHL level, height is not a significant concussion risk factor due to players being largely homogenous for height. While this may explain why height is not a significant risk factor at the NHL level, in youth hockey where height differences may be more pronounced, height has not been found to be a significant predictor of injury or concussion[75].

It should be noted however, that height/weight data provided by the NHL may not be entirely accurate. The height and weight data recorded at the start of every season for a given player does not vary from year to year. This seems unrealistic, especially for some of the younger NHL players who enter the league between 18-20 years of age and are still growing. Repeating the analysis with more accurate height/weight data may help clarify the true relationship between these variables and reported concussions. However, logistic regression did find a significant effect for height in the 2010-2011 season, where those in the 75-81 inch category were 59.7% less likely to suffer a concussion than the 65-72 inch category. This helps reinforce the notion that there may be some merit to the hypothesis put forward presently, and re-analysis should be performed with more accurate data.

Risk Factors: ATOI

Finally, ATOI was found to be a significant predictor of injury, with those players in the 15-20 minute category 2.6 times more likely to suffer a concussive injury than the 0-5 minute category. These data are similar to prior work in female, youth and junior ice hockey[56,81,85]. At the NHL level, Stevens et al. (2008)[86] reported a significant effect for total time on ice per game, noting those who played more minutes had an increased likelihood of suffering a concussion. In the current study, the 15-20 minute ATOI group suffered the highest incidence in concussion

(9.48%), which is similar to the median data of 15.22 minutes reported by Stevens et al. However, like the Stevens et al. study, the present findings are only correlational and do not suggest any cause and effect links between the two. Stevens et al. suggested that their findings might be due to in-game fatigue, as players who are fatigued will suffer a reduction in physical abilities, and most importantly, awareness, making them more susceptible to concussion. Wilkins et al. (2004) showed differences between fatigued and control participants on a postural stability (balance) task, measured using the Balance Error Scoring System, a sideline test often administered after a mild head injury[95]. As such, it is possible that a fatigued state leading to decreased postural stability and awareness may explain the increased concussion risk associated with higher ATOI.

If fatigue is indeed an underlying causative mechanism, we would have expected to see an increased risk of concussion in the 20-25 minute as well as the 25+ minute groupings. However, this wasn't the case, and more research is required to determine the contributing factors of ATOI to concussion risk. Additionally, we expected to see an increased risk in the 0-5 minute grouping, as this typically represents the players who are the designated 'fighters' for each team. It is possible that there is an interaction between ATOI and skill/performance in that concussions decreased following the 15-20 minute grouping due to an increase in skill in the 20-25 and 25+ minute groupings. Players in those groupings typically represent higher skill levels, and perhaps their increased awareness helps decrease the number of concussions suffered.

Limitations

The current study adds to our understanding of concussion in NHL hockey players; however, there were some limitations to the study. For instance, the design was a retrospective analysis of NHL statistics as they relate to reported concussions and injury risks, and as with all

retrospective studies, the investigators did not have control over data collection methods. In the present study, data were derived from media sources, and as such, the accuracy of the data may be called into question. As noted earlier, the issue of underreporting of concussion is significant. In addition, a criticism of media derived injury information that has been previously discussed[70,72,96] is that team officials may intentionally submit inaccurate injury reports to hide the true nature of the injury, for fear of alerting competitors to vulnerable body parts. It is possible that this occurred in the current dataset; in some cases, the injury description was listed as "upper body injury" or "undisclosed." Many times the nature of an "undisclosed" injury was never revealed, and concussion incidences may have been omitted as a result. However, these unidentified injuries were often labelled at a later date as concussion. Additionally, given the highly scrutinized natured of professional sports by media and the thirst for information by the general public, Wennberg and Tator (2003)[70] suggested that all injury information eventually makes it way to light, a justification for the use of media reports. Furthermore, the authors found that concussion incidence rates are almost identical to reports obtained directly from team physicians, reinforcing the use of media reports in the present study. It is also possible that *The Hockey News* may not be entirely accurate in their retrieval or reporting of information; however, several studies have successfully employed data from these resources[70,72], while other media derived sources of information have successfully been used in separate studies[86,96]. We believe, that like the reporting of professional basketball statistics[74], media injury reports are an accurate source of information that can be used in the absence of official statistics provided by the NHL.

It is important to note that the increased risk of concussion associated with ATOI reported presently can only be extrapolated to professional hockey players in North America. While it is conceivable that comparable results would be found in similar populations such as

adult non-professional leagues, the absence of body-checking in such leagues may mitigate the effects found in this study. Body checking has been found to be a significant risk factor for concussion in ice hockey at all levels, and it is unknown how its removal would affect concussion rates[62,63,64,75]. Similarly, extrapolating the results reported here to hockey players in European leagues may also be problematic. Professional ice hockey is played on a larger ice surface in many parts of Europe, and the effect of this could modify the role risk factors examined in this study play in affecting concussion risk. Furthermore, these results may not apply to youth, junior or women's hockey as too many confounding variables are at play (age, gender, height and weight). Lastly, it is important to note that the significant finding of ATOI is correlational and not causal. While reasonable explanations have been proposed regarding why ATOI may increase concussion risk (fatigue, greater exposure), further experimental research is necessary to determine the true cause of ATOI as a risk factor for concussion.

Future Directions

As noted above, future research should examine the causal issues underlying ATOI as a risk factor for concussive injury. If the underlying explanation for ATOI is indeed heightened fatigue levels as suggested by Stevens et al (2008)[86], then it would be reasonable to expect a higher incidence of concussion near the end of each period, before the teams are granted a 20-minute intermission to recuperate and prepare for the following period. It would also be reasonable to expect more concussions in the third period compared to the second and first. It is plausible that fatigue decreases orientation and attention, causing players to be more susceptible to concussive incidence. It is also possible that fatigued muscle is less capable of absorbing force[88], which may help explain this mechanism. While this is speculative, it is grounds for future research to help clarify causative reasons for ATOI as a risk factor in concussion at the NHL level.

Another avenue for future research is whether specific styles of play can affect concussion incidence. That is, are players who play a more physical game involving hitting, fighting and aggressive behaviors more likely to incur a concussion than players who tend to avoid contact and play a skilled game and tend to spend more time around the perimeter of the ice? There is evidence stating that aggression is a risk factor for injury[75] and this relationship should be further explored. A recent article reported that educational and psychosocial approaches are most effective at reducing aggression-related high-risk behaviors in youth ice hockey[97]. However, the authors stated that rule changes are most effective at changing the culture of sport, clearly defining acceptable behaviors for all involved. A combination of the two is likely most effective as demonstrated by the New Zealand Rugby Union where rule change and nationwide mandatory educational programs for injury prevention were instituted at all levels, leading to a significant decrease in neurologic injuries[98].

Practical Recommendations

Based on the present findings, reported concussions in the NHL are rising. While the exact risk factors and their underlying mechanisms are still unclear, it may be possible to curb the number of concussions by simply increasing the size of the ice surface. An inverse correlation between ice size and collision rates, including head impacts has been previously demonstrated in elite junior hockey tournaments[99]. The NHL often uses their minor league affiliate (American Hockey League) to test the impact of rule changes, and implementing a larger ice surface at that level would serve as a good proving ground for such a measure.

Additionally, mandatory education programs for players, coaches and staff at multiple points throughout the season may help address the significant issue of underreporting. There is immense pressure on everyone involved to return a player to their sport as soon as possible.

Proper and thorough understanding of both the short and long-term sequelae and the importance of treatment may help increase concussion reporting and decrease the pressure on early return-to-play.

Conclusions

The current study adds valuable data to our understanding of concussion in the NHL. It also serves to update descriptive statistics regarding reported concussions. It is the first study to examine a multitude of risk factors for concussion at the NHL level and provides important information regarding position, age, height, weight and ATOI. These data illustrate that concussion incidence is rising in the NHL and that ATOI is a risk factor for concussive injury. Despite stated beliefs to the contrary[70], position, age, height and weight were not found to be significant risk factors for concussion. This information may be relevant for general managers, scouts and other team officials when making personnel decisions on drafting, signing and trading; more specifically, given the resources involved with player acquisition, it is vitally important for teams to know that players being acquired are not at risk for concussion, and therefore less likely to miss extended periods of time due to this injury. While the current study has contributed to our understanding of concussion in the NHL, further research is required to further elucidate risk factors and their underlying causal factors.

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Appendices

Appendix A. Descriptive statistics of the study population by season

	Age	Height	Weight
2005-2006 Season (N = 871)			
Mean	27.17	73.27	206.29
Standard Deviation	4.64	2.04	15.31
2006-2007 Season (N = 860)			
Mean	27.00	73.17	205.55
Standard Deviation	4.62	2.03	15.15
2007-2008 Season (N = 854)			
Mean	27.01	73.16	205.34
Standard Deviation	4.80	2.08	15.45
2008-2009 Season (N = 887)			
Mean	26.82	73.23	205.35
Standard Deviation	4.77	2.09	15.85
2009-2010 Season (N = 880)			
Mean	26.74	73.17	204.74
Standard Deviation	4.69	2.09	15.73
2010-2011 Season (N = 895)			
Mean	26.60	73.16	204.24
Standard Deviation	4.60	2.11	15.57
2011-2012 Season (N = 898)			

Mean	26.61	73.13	203.28
Standard Deviation	4.54	2.09	15.93

Appendix B. Descriptive statistics of the concussed population

	Age	Height	Weight
Concussed Players (N = 447)			
Mean	27.25	72.99	203.76
Standard Deviation	4.90	2.04	15.35