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Head Impact Exposures In Youth Canadian Football Players Aged 12-14 Do Not Significantly Impair Antisaccade Performance

William J. Smith, *The University of Western Ontario*

Supervisor: Dickey, Jim, *The University of Western Ontario*

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Abstract

There is a growing body of research evaluating head impacts in sport, but studies have not evaluated youth football in Canada. It is important to evaluate football in Canada as the rules and field size are different than American football, and this may influence the impact exposures. The purpose of this study was to quantify the head impact exposures in youth Canadian football and determine if a season's worth of impacts could cause impairments in executive function. Players (n=50) had their head impacts recorded using an impact measuring device (GForceTracker) throughout all contact practices and games. A subset of players (n=28) completed a pre- and post-season antisaccade protocol to determine whether there were any changes in executive function. No statistically significant executive functioning deficits were detected in this study. It appears that the head impacts accumulated during one season of youth football do not lead to decrements in executive function.

Keywords

Subconcussive head impact, Canadian football, youth, antisaccades, electrooculography, concussion, executive function

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1 Introduction

1.1 Concussion

The first reference to mild traumatic brain injury (mTBI) as "concussion" was in the 10th century [1]. While our understanding of concussion has evolved, this was the first time that concussion was referred to as being distinctly different than other head injuries. The current definition of concussion is a mTBI induced by biomechanical forces that can be transmitted to the brain by impacts to either the head or body [2]. Concussion can only be diagnosed by a health care professional and should be suspected if an individual presents with any of the associated physical signs or clinical symptoms [2]. There is a wide variety of possible concussive symptoms, with the most common being headache, dizziness and difficulty concentrating [3]. It is important to note that symptoms vary between individuals and no symptom presents in every case of concussion [3].

The occurrence and management of head injuries are currently some of the most discussed topics in sports. Sports related concussions (SRC) are one of the largest problems for people in North America; particularly for adolescents and youths. Nearly 40% of youth patients admitted to emergency rooms in Canada with sport related head trauma were diagnosed with concussions [4]. In the youth population, American football is the sport responsible for the most SRC associated hospital visits [5]. Concussions account for roughly 8% of all injuries sustained by youth football players [6].

Head injuries can have long term effects for anyone who suffers them, but may be most detrimental to youths. In fact, 16% of youth football players aged 5-14 with SRC took longer than 30 days to return to play compared to 7% of college athletes [3]. At these ages, the brain undergoes a period of development in which white matter increases and continues to develop into adulthood [7]. While the exact reasons for age related differences in recovery are not fully understood, it is possible that these neurological changes make youth athletes vulnerable [8]. A continuously growing body of evidence is

showing that long term detrimental effects on the brain may be present even when a concussive injury has not been diagnosed. A recent study evaluated executive function in retired National Football League (NFL) players that began their playing careers before the age of 12. They found that players who began playing before the age of 12 demonstrated significantly greater impairments of their executive function compared to those that began playing after 12 [9]. This is despite there being no statistically significant difference in the number of concussions sustained throughout their career [9]. This indicates that long term neurological issues could be a result of exposure to head impacts, not necessarily SRC.

1.2 Subconcussion

Subconcussion refers to impacts to the head or body that cause an acceleration of the brain, but do not cause concussive symptoms [10, 11]. Accumulation of these subconcussive impacts has been linked to Chronic Traumatic Encephalopathy (CTE) [12, 13] and various short term cognitive deficits [14-16]. One of the difficulties of researching subconcussive impacts in humans is the ethical implications of knowingly inducing head trauma in participants. For this reason, most of the research has come from observing head impacts in sports such as boxing, soccer and football.

1.2.1 Subconcussive Impacts

Several studies have determined the characteristics of the impacts that athletes are exposed to by participating in sporting events. A common methodology involves conducting kinematic analyses of video to extract kinetic data from head impacts which are then recreated in a laboratory setting [17-19]. Collecting game video for analyses can work when analyzing a small number of impacts, but it is difficult in sports such as football when there can be hundreds of impacts per game. Proper analysis of video requires multiple camera angles and video that is clear enough to see precise details, yet zoomed out enough to have every player in the frame at all times. Additionally, the unknown relative angles between the camera and the impact site can lead to difficulty locating a precise impact point and can result in errors calculating the players velocity of

up to 11.3% [20]. An alternative to collecting impact data using video replication is the use of in-helmet instrumentation.

Instrumentation has been used in football helmets to provide real time feedback on the characteristics of impacts since the 1970's [21]. More recently, researchers have evaluated both the number and magnitudes of head impacts sustained by collegiate American football players during a season [22-26]. Peak linear and rotational accelerations were 200 g [22] and 9,922 rad/s^2 [24] respectively. Mean linear accelerations were 20.9 g [23] and 32 g [22], and mean rotational accelerations were 1,355 rad/s^2 [24]. The maximum number of head impacts that collegiate level players were exposed to in a season ranged from 1,022 – 1,444 [26]. The impacts measured in American college football are different than those observed in the high school and youth levels of the game.

In contrast to collegiate players, high school players experience a lower number of head impacts and reduced impact magnitudes. The peak linear and rotational accelerations experienced by high school athletes were 152.3 g and 7,701 rad/s^2 respectively [27]. Mean linear accelerations were recorded at 25.9 g with mean rotational accelerations of 1,694.9 rad/s^2 [28]. Athletes were exposed to a maximum of 1,258 impacts [27] with a mean of 774 impacts per season [28]. These findings show that the number of impacts and the peak linear and rotational acceleration magnitudes in collegiate football are higher than high school football, despite having similar mean impact values. The peak values are likely larger due to the increased size, speed, and strength of the players in the collegiate level. Relatively few impacts occur in the highest acceleration range compared to the lower range, which could explain why the higher peak values in the collegiate game did not show a large increase in the mean acceleration magnitudes.

There has been a recent shift of focus to explore the characteristics of head impacts that youth athletes (aged 5-14) are exposed to while playing American football [29-33]. The peak linear and rotational accelerations have been reported at 175.9 g [32] and 7,694 rad/s^2, respectively [31]. Peak linear values are comparable to those seen in both

collegiate and high school levels. Mean linear accelerations have been measured between 19.8 and 22 g, and mean rotational accelerations have been measured between 1,099 and 1,114.6 rad/s^2 [33]. These values are less than those experienced by collegiate and high school athletes. Quantifying the impacts that youth athletes are exposed to throughout a season of football is important, but it offers little meaning if we do not understand the potential effects these impacts could have on their developing brains.

1.2.2 Effects of Subconcussive Impacts in Football Players

Studies into the effects of subconcussive impacts have yielded some concerning results. A study of high school football players with no concussive symptoms showed athletes had impaired visual and working memory abilities after one season of subconcussive head impacts [34]. Researchers have revealed similar findings in youth football. Youth football players had slower processing speeds and reaction times than non-contact athletes [35]. Other researchers have observed changes in brain function through medical imaging. For example, Bahrami and colleagues (2016) studied fractional anisotropy (FA) of white matter tracts in youth (aged 8-13) American football players [36]. Fractional anisotropy is a measure of permeability that is frequently used to describe the integrity of white matter tracts in the brain. Low FA values indicate that neuronal permeability is being disrupted, which is caused by a loss of structural integrity of the axon [37]. Bahrami et al. (2016) observed a negative linear relationship between head impact exposure and FA. These findings from American football indicate that exposure to subconcussive impacts may impair certain aspects of brain functioning in youth football players. However, there are a number of differences between American and Canadian football that may alter the head impact exposures. To date, no researchers have examined the head impact exposures or their consequences in youth level Canadian football.

1.3 Canadian vs American Football

One of the most notable differences between Canadian and American football is that Canadian rules allow for three attempts (downs) to get a first down [38]. This is different than the four downs allowed in the American game [39]. Due to having fewer attempts to

get a first down, the likelihood of Canadian teams achieving a first down is lower. When a first down is not achieved, the offensive team often elects to kick the ball to the opposing team. Kicking plays are commonly referred to as special teams plays. These plays generally result in greater closing distances prior to colliding with the opposing team, which leads to higher linear and rotational head accelerations on impact [40].

The rules regarding the number of players and the dimensions of the playing field may also contribute to differences in closing distances. The Canadian game has 24 players participating at a time, and the field is 150 yards long, and is 65 yards wide [38]. In comparison, the American game has 22 players on at a time, and the field is 120 yards long, and is roughly 53 yards wide [39]. The larger field provides Canadian players with 406.25 yd^2 of free space per player compared to the 289.09 yd^2 on an American field. More free space gives players a greater opportunity to increase their velocities prior to contacting the opposing team, resulting in increased linear and rotational head accelerations on impact. The rules that govern American and Canadian football vary considerably, meaning impact exposures and their associated cognitive effects collected from American football should not be extrapolated to a Canadian population.

1.4 Antisaccades

1.4.1 Executive Function

The executive functions are a specific set of cognitive skills that most everyday tasks that humans perform heavily rely on. These functions include things like planning goal direction behaviours, initiating actions, inhibiting reflexive movements, and the ability to self-correct behaviours in response to changes in a planned task [41]. While impairments in executive function are often seen in cases of brain lesions following TBI [42, 43], some individuals with mTBI display impairments even without any additional cognitive symptoms [42, 43]. One of the commonly used methods of detecting executive functioning impairments is the antisaccade task.

1.4.2 Antisaccade Task

The antisaccade task is an oculomotor test of executive function [44]. Participants are instructed to focus on a fixation point (FP) centrally located in their field of view. When a target stimulus (TS) is flashed peripherally to either their left or right, individuals must suppress the natural reflex to look at the stimulus (a prosaccade) and instead generate a new saccade to the mirror symmetrical location of the stimulus [45]. While the antisaccade task is simple for participants to complete, it requires complex coordination of several regions of the brain. This includes the cerebral cortex, basal ganglia, thalamus, superior colliculus, brainstem, cerebellum and reticular formation [45]. For a complete neurophysiological review of the antisaccade task, refer to Munoz and Everling, 2004.

A standardized antisaccade data collection protocol recommends that researchers evaluate performance using both the reaction time (RT) latencies and error rates of all trials [46]. Most antisaccade errors result in the generation of a reflexive prosaccade toward the stimulus, followed by a brief pause, then the correct antisaccade movement [47]. This indicates that the errors seen in individuals with impaired executive function are likely caused by an inability to suppress the prosaccade reflex [45]. In cases where the prosaccade reflex is successfully inhibited, impaired individuals will likely display greater latencies due to their compromised ability to plan and generate the mirror symmetrical saccade. Researchers can determine both RT and direction of a participant's saccade using electrooculography (EOG,[[48-51]).

1.4.3 Electrooculography

Electrooculography (EOG) can be used to detect the onset and direction of eye movements. The eye has an electrical di-pole between the positively charged cornea at the front of the eye and the negatively charged retina at the back. This is known as the standing retinal potential (SRP) [52]. The SRP can be used to gain valuable information regarding the onset and direction of eye movement by placing an electrode on the outer (lateral) canthi of each eye. When the eyes are oriented straight ahead, the relative

voltage between the two electrodes is zero. As participants look left or right, the positive cornea rotates towards one electrode while the negative retina rotates toward the other. The electrode closest to the cornea will measure a positive charge while the electrode closest to the retina will measure a negative charge. This means that the saccade will always be in the direction of the positively charged electrode and the onset of movement occurs at the time when the voltage between electrodes changes from zero.

2 Purpose Statement and Hypothesis

2.1 Purpose Statement

This study has two purposes. First, to quantify the cumulative number of head impacts and the resulting linear accelerations and rotational velocities that Canadian youth football players are exposed to throughout a competition season. Second, to determine the effects that these subconcussive head impacts have on athletes executive functioning.

2.2 Hypotheses

1) A seasons worth of head impact exposures will cause impairments in players' executive function.

2) Players who sustain a higher number of impacts throughout the season will experience greater executive functioning deficits than those who sustain a lower number of impacts.

3 Methods

3.1 Participants

This study was approved by the Western University Health Sciences Research Ethics Board (protocol 11054). Players from a bantam level youth football program volunteered to participate in this study. Each player and their parent/guardian gave informed consent prior to participating in any aspect of this study. Participants completed a health history questionnaire to determine if any concussive symptoms or neurological disorders were present (Appendix A). Athletic trainers were present at all practices and games to monitor athletes for possible signs and symptoms of concussion.

3.2 gForce Tracker

The gForce Tracker (GFT) is an inertial measurement unit that measures head impact kinematics that result from impacts to the head or body. These devices contain a tri-axial linear accelerometer and a tri-axial gyroscope and they measure linear accelerations, rotational velocities, and impact locations. The devices are triggered to record data when the accelerometers detect a linear acceleration above the user defined threshold in any axis. The threshold was set to 15 g to remain consistent with previous research [53-55]. The devices record 40 ms of data when the threshold is reached (8 ms before and 32 ms following the threshold trigger). Signals from the tri-axial accelerometer are sampled at 3,000 Hz and low pass filtered with a cut-off frequency of 300 Hz. Signals from the tri-axial gyroscope were recorded at 800 Hz and low pass filtered with a cut-off frequency of 100 Hz. Impacts are time stamped and stored in an onboard memory system. Impact data are downloaded from the devices following each game or practice.

Participant's helmets were equipped with a GFT device for the duration of the season. The devices were fastened to the inside crown of the helmets shell using an industrial strength re-closeable fastener (3MTM Dual LockTM Re-closeable Fastener SJ3551 400

Black, 3M Global Headquarters, St. Paul, MN). The device was placed within the natural spaces between each helmet's padding (Figure 3.1). Once fastened, according to manufacturer guidelines, an alignment procedure was performed to orient each device to its location relative to the rest of the helmet. This involved calibrating the devices with the helmet in three different positions, one in each cardinal plane. The devices remained in the same position for each collection period to ensure that all data remained accurate.

Figure 3.1: Figure depicts a GFT, circled in red, fastened to the inside crown of the helmet.

3.3 Impact Collection

3.3.1 Practices

All of the devices were turned on prior to each practice. During practice, attendance was recorded and absent player's devices were turned off. Players were instructed by the coaches to keep their helmets on their heads at all times while on the playing field. Athletes were monitored any time they were off the field (e.g. for injuries or water breaks) to ensure that any incidences when their helmets came off their head were recorded. Any impacts recorded during these times were excluded from the analysis. Devices were turned off at the conclusion of each practice session. Following each practice, the devices were charged and the impact data was downloaded from the helmets

onto a secure cloud server. Helmets remained in the possession of the research team while not in use to ensure that the devices remained uncompromised.

3.3.2 Games

All of the devices were turned on prior to each game. The team participated in only one game per week with no bye-weeks between games. During games, a custom game day LabVIEW program (LabVIEW 2010, National Instruments, Austin TX) enabled the researchers to record the exact time periods that players were on the field. This program was used to exclude all impacts recorded when a player was not on the field of play. Devices were turned off at the conclusion of each game. Following each session, the devices were charged and the impact data was downloaded from the helmets onto a secure cloud server.

3.4 Antisaccade Testing Procedure

Participants completed an antisaccade testing protocol at two time points throughout the study. The pre-season protocol took place immediately prior to the first contact practice and the post-season follow-up protocol was conducted between two and seven days following the final game. Testing protocol's ran approximately five minutes in duration. Participants were seated at a table with their heads placed on a chin rest for the duration of the collection protocol. A light board was constructed based on digitally controlled colour LEDs (WS2801, www.adafruit.com) connected using a USB to Multi-Protocol Synchronous Serial Engine (MPSSE) Cable (C232HM-EDHSL-0, Future Technology Devices International, Ltd., Glasgow, UK) and controlled using a custom LabVIEW program (LabVIEW 2010, National Instruments, Austin TX). The light board was oriented perpendicular to the participant's gaze and located 55 cm in front of the chin rest. The saccadic FP and TS were displayed on the light-board. The FP was centered horizontally on the light board with TS located 15.5 ° to the left and right along the same horizontal axis. The FP was illuminated to indicate to each participant that the trials had begun. This light changed from green in prosaccade trials to red in antisaccade trials to remind the participant which was required. Each testing session was comprised of 20 prosaccades followed by 20 antisaccades; each block consisted of 10 trials to the right

and 10 to the left in a randomized order. There was a randomly assigned 1,000 to 2,000 ms latency period between trials. After which, the TS was illuminated for 50 ms to trigger the response and indicate the desired direction of the saccade. The FP remained illuminated for the entire duration of the testing protocol (no-gap paradigm).

Trials were deemed successful if the saccade was generated towards the TS in a prosaccade, and away from the TS in an antisaccade (Figure 3.2). Trials were considered to be errors if the initial eye movement was in the wrong direction, even if participants subsequently looked in the correct direction. The change in number of directional errors and RT latencies from pre- to post-season were used to measure executive functioning impairment.

The timing of the stimulus was captured simultaneously with EOG signals. The voltage across a 100 kΩ resistor in parallel with the photodiode was used to quantify the level of light emitted by the LED. Illumination of the LED resulted in a 200mV signal that had a rise time of less than 10 μS. Photodiodes were placed beside the TS LEDs, oriented towards them, and covered to eliminate ambient light. The signals from the photodiodes were used to determine the timing and direction of the desired saccade. EOG was used to measure the latency and direction of participant's saccades.

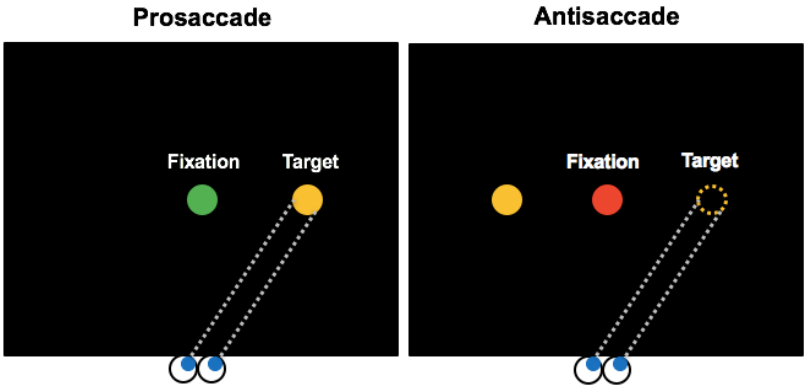

Figure 3.2: Correct performance of prosaccade and antisaccade trials. Colour of fixation light indicates upcoming trial (green = prosaccade, red = antisaccade). Blue dots represent participant's eyes. Yellow dots represent target stimulus.

3.4.1 EOG Recordings

Participants were fitted with three disposable surface Ag-AgCl electrodes (AM-N00S/E, AMBU Blue Sensor Adhesive Snap Electrode, Ambu Inc, Glen Burnie, MD, USA); one located at the outer canthi of each eye and one ground electrode on the center of their forehead (Figure 3.3). The voltages from the electrodes were amplified 1000x and filtered from 0.5-100 Hz using an isolated electrophysiological amplifier (Model 2024F, Intronix Technologies Corporation, Bolton, Ontario, Canada) and sampled at 1000 Hz with a 16 bit analog-to-digital converter (USB 6211, National Instruments, Austin TX) using a custom LabVIEW program (LabVIEW 2011, National Instruments, Austin TX). Signals were displayed in real time on the computer monitor so that investigators could evaluate the quality of the signals.

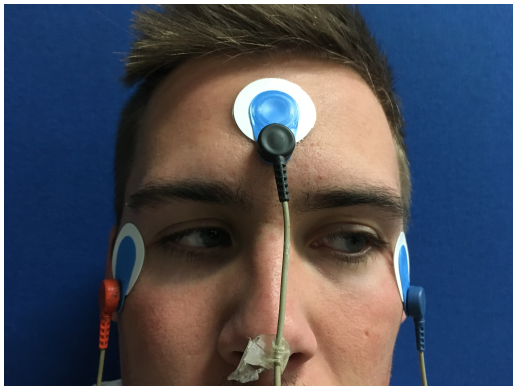

Figure 3.3: Placement of the EOG electrodes; one on the outer canthi of each eye, and one ground electrode on the forehead.

3.5 Data Analysis

3.5.1 Head Impact Analysis

Head impacts were identified using the student investigators practice notes and the game day LabVIEW program to remove all impacts that did not occur while the player was participating in football activity. The GFT's record data from the outer shell of helmets,

so a correction algorithm was used to determine the linear accelerations and rotational velocities of the center of mass of participants heads [56].

3.5.2 Antisaccade Analysis

The raw saccadic voltages collected via EOG were post-processed using a custom LabVIEW program. As a start, the EOG voltages were band-pass filtered from 0.05 to 20 Hz using a 2nd order Butterworth filter. The start of each trial was determined as the onset of the change in voltage from photodiodes measuring the TS illumination using an automated onset detection algorithm [57]. The onset of the saccade was determined as the onset of the change in voltage of the EOG signal using the same onset detection algorithm [57]. The latency was calculated as the difference in timing between the onsets of the TS and the EOG signal (Figure 3.4). The direction of the EOG signal voltage was used to determine saccade direction. For antisaccades, trials were recorded as correct if the TS signal from the photodiode and the saccadic signal from the EOG were in opposing directions (Figure 3.4). In some cases, the EOG signal was some value other than zero when the TS was illuminated, indicating the participant was either blinking or did not have their eyes focused on the FP. When this happened, trials were labelled as a "blink" and excluded from further analysis.

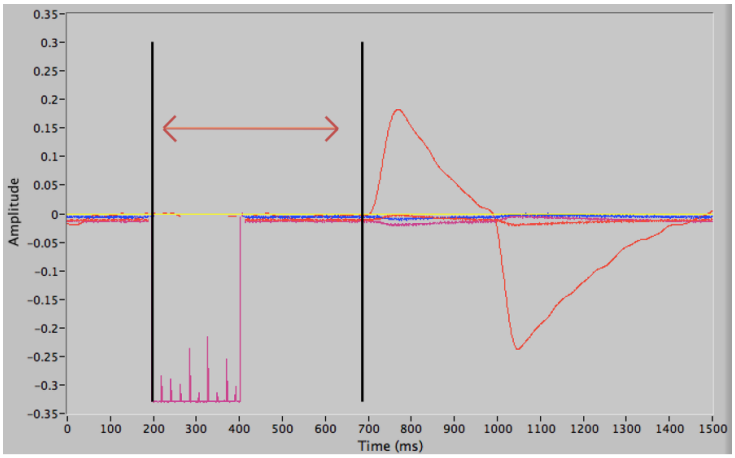

Figure 3.4: Depiction of a correct antisaccade trial. The downwards change in voltage at approximately 200 ms (pink line) is the voltage from the photodiode reflecting the TS. The onset of the TS, as determined using an onset detection algorithm, is depicted by the vertical black line at approximately 200 ms. The movement of the eyes is illustrated by the red waveform line on the right side of the graph. The onset of eye movement, as determined using an onset detection algorithm, is depicted by the vertical black line at approximately 700 ms. The RT is calculated as the difference between these lines, as depicted by the red arrow.

3.6 Statistical Analysis

An alpha level of 0.05 was assigned for all statistical analyses. Mean ± SD were used to evaluate the distributions of the cumulative number of impacts, linear accelerations, and rotational velocities. Head impacts were divided into practices and games to determine the exposure characteristics for each type of event.

All antisaccade latencies were summarized using the median for each participant at both pre- and post-season testing, as recommended by Antoniades (2013). The number of antisaccade errors that each participant committed were summed at both pre- and post-season testing sessions. The total hit count that players accumulated throughout the

season was compared to the change (post-season - pre-season values) in median latencies, and also to the number of errors. A Shapiro-Wilks test was used to determine the normality of the distribution of cumulative head impact exposure. This showed that the distribution was non-normal ($p=0.01$). Due to the non-normal distribution of head impact data, Spearman's rank order correlation analyses were performed. Spearman's correlation coefficients (r_s) were calculated for each relationship. Based on previously published guidelines, we defined the strength of each relationship based on r_s of 0-0.3 as "negligible", 0.3 – 0.5 as "low", 0.5 – 0.7 as "moderate", 0.7 – 0.9 as "high", and 0.9 – 1 as "very high" [58]. The strength of the linear relationship between the peak resultant linear acceleration and the peak resultant angular velocity was assessed using a Pearson Product-Moment correlation. P-values were calculated to determine if the observed relationships were statistically significant, and $p<0.05$ was the threshold defining statistical significance.

4 Results

In the follow section, data are displayed as mean ± standard deviation.

4.1 Head Impacts

4.1.1 Participants

Fifty-seven athletes (56 boys, 1 girl) between the ages of 12 and 14 years old (13, ± 0.61) volunteered to participate in this study. All participants were members of the same bantam Ontario Provincial Football League (OPFL) team. None of the participants who had previously been diagnosed with a concussion reported having any concussive symptoms at the beginning of the study. Seven players quit the team prior to the first game, thus their data was excluded from the analysis. The head impacts recorded in all contact practices (n=27) and games (n=9) were recorded from the remaining 50 players during the 13 week season.

4.1.2 Head Impact Characteristics

Linear accelerations and rotational velocities were collected for all head impacts that participants received throughout the course of the season. In total, 10,063 head impacts were recorded for 50 players with instrumented helmets. The total number of accumulated head impacts per player ranged from 10 to 572. The average number of head impacts per player was 6.5 ± 4.2 per contact event, and 201.3 ± 131.6 throughout the season. The number of head impacts was compared across practice and games. Of the 10,063 total impacts, 5,701 (56.7%) occurred in practices and 4,362 (43.3%) occurred in games. Players were exposed to an average of 5 ± 3.4 impacts per practice and 9.7 ± 8.4 impacts per game.

Linear accelerations ranged from 13.1 to 106.6 g. The distribution of linear acceleration was right skewed with a mean of 23.1 ± 6.3 g, a median of 22.1 g, and a 95^{th} percentile

value of 33.9 g. Rotational velocities ranged from 98.9 to 2,804°/s. The distribution of rotational velocities was right skewed with a mean of 585.5 ± 217.3 °/s, a median of 545.7°/s and a 95th percentile value of 995.2 °/s. The distributions of head impact data are presented in Figure 4.1.

Impact magnitudes were analyzed for practices and games. Linear accelerations in practices ranged from 13.2 to 94.2 g with a mean of 22.4 ± 5.5 g. The median and 95th percentile linear accelerations in practices were 21.7 and 31.2 g respectively. Rotational velocities in practices ranged from 98.9 to 2,804.2°/s with a mean of 561 ± 196.8°/s. The median and 95th percentile rotational velocities were 533 and 921.3°/s respectively. Linear accelerations in games ranged from 13.1 to 106.6 g with a mean of 24.1 ± 7.0 g. The median and 95th percentile linear accelerations in games were 22.7 and 36.6 g respectively. Rotational velocities in games ranged from 111.0 to 2,573.8°/s with a mean of 617.5 ± 237.7°/s. The median and 95th percentile rotational velocities were 567.2 and 1,071.1°/s respectively. A summary of practice and game head impacts data is presented in Table 4.1.

There was a strong linear relationship between the peak resultant linear acceleration and the peak resultant angular velocity (r=0.72; Figure 4.2).

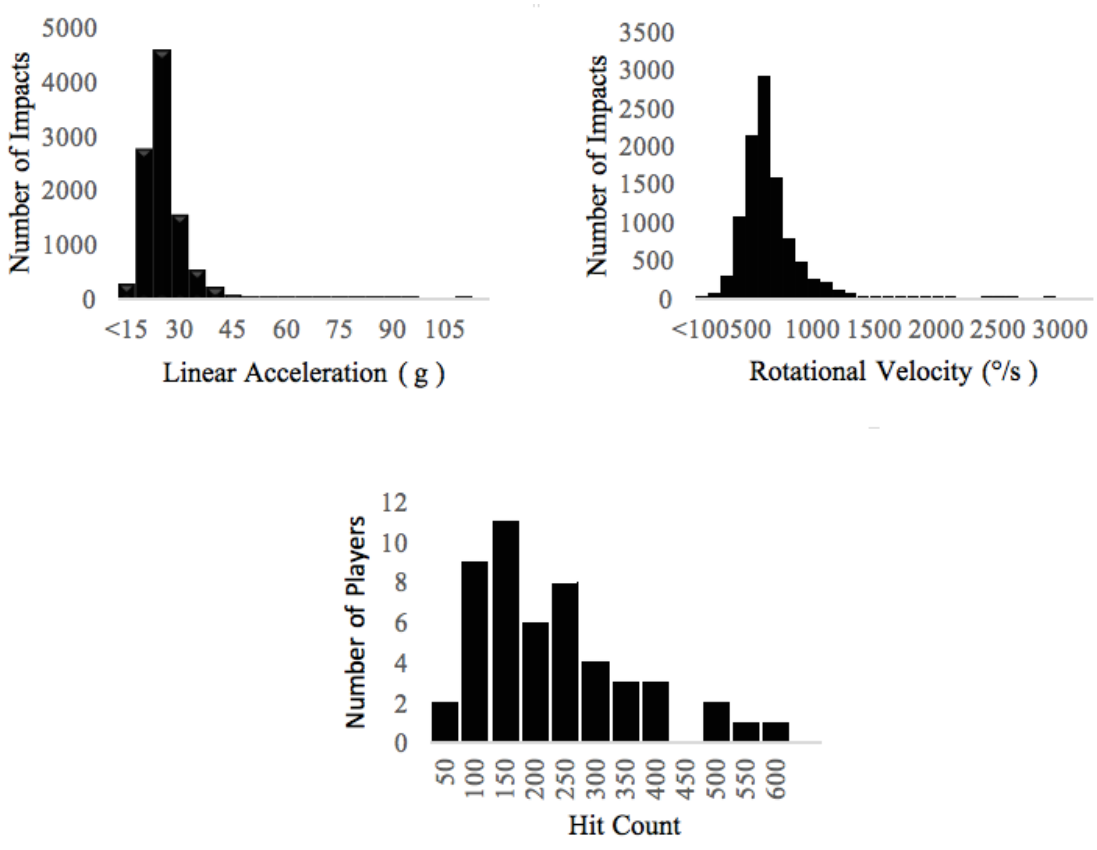

Figure 4.1: Distributions for head impact parameters for linear acceleration, rotational velocity, and hit count.

Table 4.1: Head impact magnitudes for practices and games.

	Practices		*Games*	
	Linear Acceleration (g)	**Rotational Velocity (°/s)**	**Linear Acceleration (g)**	**Rotational Velocity (°/s)**
Mean ± SD	22.4 ± 5.5	561.1 ± 196.8	24.1 ± 7.0	617.5 ± 237.7
Median	21.7	533	22.7	567.2
95th Percentile	31.2	921.3	36.6	1071.1
Max	94.2	2804.2	106.6	2573.8
Min	13.2	98.9	13.1	111.0

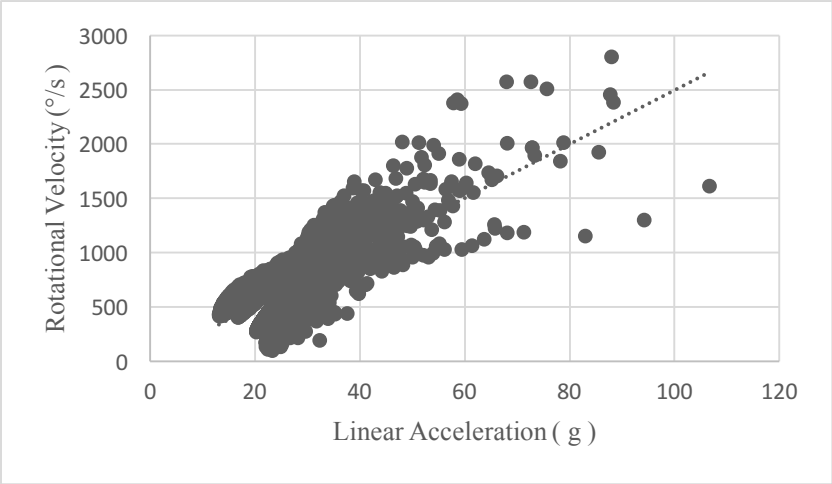

Figure 4.2: Relationship between the peak resultant linear acceleration and peak resultant rotational velocity for all recorded head impacts.

4.2 Antisaccades

4.2.1 Participants

All 50 players participated in the baseline antisaccade testing. Unfortunately, we were unable to schedule six players for the post-season testing session. One participant was diagnosed with a concussion during the season. To avoid exacerbating concussive symptoms, this individual was excluded from the post-season antisaccade testing protocol. Only the saccade data collected from the 43 players who attended both pre- and post-season testing sessions were analyzed further. Preliminary analysis of the pre- and post-season data revealed that some players had longer prosaccade latencies than antisaccade latencies in one of the test sessions. This indicated that these participants did not perform the task correctly. Antisaccade latencies should be longer than prosaccade latencies as correct performance of the antisaccade task encompasses all of the cognitive elements of the prosaccade, plus the cognitive processes associated with supressing the

reflexive prosaccade response [59]. We excluded these participants from further analysis. The data from the remaining 28 players was analyzed to determine the relationship between cumulative head impacts and antisaccade performance.

4.2.2 Latencies and Error Rates

Median pre-season antisaccade latencies ranged from 223.0 – 381.2 ms, with a mean of 294.5 ± 33.2 ms. Pre-season errors ranged from 0 – 10 out of 20, with an average of 3 ± 2.9 per participant. Median post-season antisaccade latencies ranged from 215.4 – 376.9 ms, with a mean of 273.1 ± 41 ms per participant. Post-season errors ranged from 0 – 8 out of 20, with an average of 3.2 ± 2.3 per participant.

4.2.3 Head Impact vs Antisaccade Relationship

There was a statistically significant low negative correlation between the cumulative hit count and the change in median latency from pre- to post-season (r_s = -0.39, p=0.042). Similarly, there was a statistically significant low positive correlation between hit count and the change in error from pre- to post-season (r_s=0.43, p=0.021).

5 Discussion

To our knowledge, this study reports the only collection of head impact data in youth Canadian football players to date. We sought to quantify the cumulative number of head impacts and the resulting linear accelerations and rotational velocities that Canadian youth football players are exposed to throughout a competition season. Additionally, we wanted to determine the effects that these subconcussive head impacts have on athletes executive functioning.

5.1 Head Impact Exposures in Youth Canadian Football

Previous research evaluating head impact exposures in Canadian football has been conducted with University aged players [54, 55]. One such study recorded 20,925 impacts over 55 practices and 11 games. Of these, 10,528 (50.3%) impacts were in practice and 10,396 (49.7%) were in games [54]. This is a lower percentage of impacts recorded in practices than in our study. In contrast, Muise et al. (2106) report that 5,473 hits occurred in 10 training camp sessions, 6,293 hits occurred in 32 practices and 9,184 hits in 10 games. Considering the season as a whole, practice impacts reported by Muise et al. (2016) accounted for roughly 41% of impacts, while game impacts accounted for 59%. This proportion of impacts in games and practices is opposite of our results. The fact that the distribution of impacts varied between Canadian varsity football teams likely indicates that coaching style influences the number of head impacts that players receive. This may also be relevant to youth players, and accordingly our findings may not reflect exposures on other youth teams.

In terms of impact magnitude, Campbell (2014) reported that the median and 95th percentile linear accelerations for all exposures were 20.43 and 36.13 g respectively.

Median linear accelerations were 21.53 g in games and 19.94 g in practice. Interestingly, these median impact magnitudes are similar, but slightly lower than those reported across practices and games in our study. The trend of increased impact magnitudes in games than practices in consistent with our results.

Given that our data stands alone as the only measures of head impact exposures in youth Canadian football players, it is not possible for our data to be compared to Canadian players of similar ages. However, certain metrics of head impact exposure are comparable to those observed in youth American football players. There are a few studies examining head impact exposures in youth American football. However, most of them are conducted in younger age groups, which would be considered a different division in the OPFL. There are two studies examining the head impact characteristics in similarly aged youth American football players.

Munce et al. (2015) recorded head impacts in 22 youth football athletes (12.9 ± 0.6 yearas of age) throughout 27 practices and 9 games [32]. The recording thresholds for their measurement devices were set to 10 g. They recorded 6,183 total head impacts, which equals roughly 281 total impacts per player. This is substantially more than the 201.3 impacts per season that players in our study were exposed to, despite both teams participating in the same amount of practices and games. Setting the recording threshold to 10 g's allowed Munce et al. to record impacts that would not have triggered the devices in our study. It is likely that the number of impacts they recorded between 10 and 15 g's was substantial. This is because using a threshold of 10 g can result in a 45% increase in number of impacts collected compared to a 15 g threshold [60]. Accordingly, the number of impacts between our study and theirs may not have been as different as it seems. Munce et al. determined that 61% of impacts occurred in practice and 39% occurred in games or pre-game warmup. Linear accelerations ranged from 10 to 175.9 g, with a mean of 25.5 g and a median of 20.2 g for all exposures. Linear accelerations recorded in practice averaged 25.5 g with a median of 19.9 g per head

impact. These values are lower than those recorded in games (26.8 g and a median of 20.9 g per head impact). The overall 95th percentile linear acceleration was 57.3 g, with values of 55.0 g in practices and 63.0 g in games. These results are directly comparable to our study. Munce et al. reported substantially higher peak and 95th percentile linear accelerations than our study, indicating that head impacts on the higher end of the spectrum were larger for the American players. The mean linear accelerations in our study were slightly smaller in both practices and games compared to the American players. However, our median linear accelerations were greater in both conditions. These findings are not consistent with the difference in sensor trigger threshold (10 g threshold used by Munce et al.) as that would be expected to reduce the mean and median values of impacts relative to a 15 g threshold [60].

Kelley et al. (2017) recorded head impact data in a 32 youth American football players aged 13 ± 0.5 years old. Athletes participated in approximately 43 ± 2 sessions (games and practices) per season. Recording thresholds for their measurement devices were set to 10 g. Linear accelerations from all exposures had a mean, median and 95th percentile value of 25.6, 22 and 57.9 g respectively. The mean linear accelerations were larger in games than in practices at 27.4 and 24.9 g respectively. The trend of reporting greater linear accelerations in games than practices was consistent for all studies examining youth football (Kelley et al. (2017), Munce et al. (2015) and our study). The peak and 95th percentile linear accelerations were larger in both practices and games compared to our results. This trend reinforces the idea that head impacts on the higher end of the spectrum are larger for American players.

Regulation sized Canadian football fields are larger than those used in American football. This means there is more free space per player allowing them to obtain greater velocities prior to contacting the opposing team (406.25 in Canadian versus 289.09 yd^2 in American). This idea of free space is thought to be responsible for the greater impact magnitudes in passing and special teams plays compared to running plays [40, 61]. Accordingly, we

expected that the Canadian players in our study would have greater linear accelerations compared to those in both Munce et al. and Kelley et al.'s studies. Our results did not support this assumption. It is also important to note that bantam OPFL games are played with four downs, like in the American game, rather than three downs like most Canadian football leagues. This would likely result in a similar number of passing and special teams plays between players in our study and those in Munce et al. and Kelley et al.'s studies.

Comparisons between our sample of youth Canadian football players and their age-matched American football counterparts reveal that linear accelerations experienced by the Canadian sample are lower in almost all measures. Interestingly, we observed a strong linear relationship between the peak linear acceleration and angular velocity for the head impacts. Accordingly, while there are thought to be distinct mechanisms of injury for these two kinematic variables [62] we observed that they were strongly related.

Unfortunately, it is not possible to directly compare the rotational impact characteristics as these studies reported rotational acceleration while we reported rotational velocity. However, we were able to analyze the general trends of these values and how they differ between practices and games. Rotational acceleration results presented by both Munce et al. and Kelley et al. followed the same trend as the linear accelerations: both studies observed greater 95^{th} percentile, mean and median magnitudes in games compared to practices. This is consistent with the trend of the rotational velocities observed in our study.

Players in our study received an average of 5.0 ± 3.4 head impacts per practice and 9.7 ± 8.4 head impacts per game. This mean number of impacts that the average player was exposed to is almost double the number of impacts in games than practices. Similarly, the mean and median linear accelerations and rotational velocities were both

larger in games than practices. The reduced impact exposures and magnitudes in practices are likely an intentional design feature implemented by leagues and coaches to protect athletes. In Ontario, a recently passed piece of legislation known as “Rowan’s Law” has drawn national attention to the management of concussions in adolescent [63]. The purpose of this law is to hold coaches and administrators accountable for the prevention and management of head injuries in adolescent athletes. All individuals associated with an athletic organization are required to review concussion awareness resources, and teams are required to create a concussion management plan. There is no direct requirement of coaches to reduce the number of head impacts that their athletes are exposed to. Instead, the law requires that all members of a sport organization become educated on the potential harms of unnecessary head impact exposures. This pressures all individuals involved with adolescent level sporting organizations to do whatever they can to ensure that brain health of players is managed appropriately. It is difficult to control the number and magnitudes of head impacts in games without fundamentally changing the sport. However, it may be possible to reduce the number of head impacts by teaching proper tackling technique. Some evidence suggests that a head-up tackling style decreases the magnitude of head impact accelerations [64]. Another possibility is reducing the head impact exposures in practice sessions. Previous research into the structure of practices has shown that full tackling drills result in the highest magnitude head impacts [65]. Limiting the number of times athletes participate in these drills or placing restrictions on the number of contact practices per week may be an effective way of reducing head impact exposures [28] .

5.2 Changes in Executive Function

On average, participants’ pre-season latencies were 294.5 ± 33.2 ms and their post-season latencies were 273 ± 40 ms. These average latencies are consistent with aged matched participants in previous literature [66].

Participants were evaluated on the change in their performance on an antisaccade task from pre- to post-season to determine if their executive functioning had changed. Performance on the antisaccade task was scored based on participants RT latencies and the number of errors that they committed. Changes in their performance were compared to their cumulative hit count to determine if there was a relationship between subconcussive head impact exposure and executive functioning.

The low negative correlation between the total number of head impacts and change in median saccadic latencies suggests that increasing players head impact exposure will decrease their RT's, reflecting improved executive functioning. It is extremely unlikely that this is true, and it is important to note that the strength of the association is low – only 15.2% of the variance in the change in latency was explained by the total number of head impacts. It is unlikely that this changes in latencies was caused by low test-retest reliability since the correlation coefficient was 0.77 [67], which is considered moderately strong [68]. Alternatively, it is more plausible that confounding variables caused the decrease in RT. One possible confounder is physical fitness. Participating in practice sessions on a weekly basis meant that players were taking part in moderate to vigorous physical activity multiple times a week. Physical fitness has been shown to improve executive functioning [69-72].

As an illustration of the effect of physical activity on executive functioning, Davis et al. (2011) evaluated executive function in sedentary children that were assigned to either a high dose exercise, low dose exercise, or no exercise control condition. Participants assigned to the exercise program completed 13 ± 1.6 weeks of daily moderate to vigorous activity lasting 20 – 40 minutes in duration. At the end of the activity program, these children showed statistically significant improvements in executive functioning when compared to the control condition [70]. Similarly, Chaddock-Heyman et al. (2013) analyzed the effects of a nine-month physical activity program on event related cognitive control tasks. Participants that completed the exercise program showed statistically

significant improvements in RT and percent of correct trials from pre- to post-test. Control participants that did not participate in the exercise program did not show statistically significant improvements in any of the testing metrics [72]. These results show that exercise induced increases in executive function. Participants' RT were improved along with their ability to correctly perform tasks [72]. Participants in our study completed two hours of moderate to vigorous physical activity two to three times per week during the 13-week season. This is roughly equal to the amount of time spent active per week and the number of weeks performing physical activity as participants in the study by Davis et al. [70]. Accordingly, improving physical fitness in a season's worth of games and practices could have been enough to improve our participants executive function, thus improving saccadic RT.

Analysis of the data revealed a statistically significant low positive correlation between hit count and the change in error rates between pre- and post-season testing. The number of errors indicates an impairment in participant's executive functioning. The positive effects of physical activity that were seen in the latency results were not strong enough to completely reduce the change in error rates. However, they may have masked the detrimental effects of increased head impact exposures. While this may have occurred, it is impossible to evaluate using our data.

Munce et al. (2015) examined the effects of subconcussive impacts in youth American football using participants of the same age as our study [32]. They used the change in participants scores on tests of executive function from pre- to post-season to evaluate possible neurological deficits. No statistically significant differences were found between pre- and post-season scores. This result occurred despite the fact that players in their study experiencing greater impact magnitudes, and a greater number of head impacts, than the participants in the present study. This finding is similar to our study and reinforces that the head impact exposure from one season of competitive football does not appear to lead to impairments in executive function.

5.3 Limitations

Our study evaluated a limited representation of youth football as a whole as we only collected data from one team. Other teams may have different head impact exposures due to difference in practice structure or league rules that govern practices and games. Accordingly, it may not be possible to generalize our findings. Additionally, this study evaluated athletes between 12 and 14 years old. This is not representative of the entire population of youth football players as different leagues can include players from 5 to 14 years old.

It is difficult to directly compare our results with other studies due to a number of factors. The devices used in this study recorded rotational velocities, not rotational accelerations. This made it difficult to determine the relationships between rotational head impact characteristics to those previously reported.

Each of the participants in this study played a variety of different positions in practices and games. Accordingly, it was not possible to evaluate differences between the various positional groups, as has been performed in other studies [54, 55].

This study did not utilize a control group of non-contact athletes of similar ages. This would have allowed us to differentiate between the effects of physical fitness and head impacts. Additionally, utilizing more frequent testing sessions could have allowed for a more accurate representation of the changes in executive functions.

This study only examined the relationship between antisaccade performance and hit count. While hit count has been an effective measure in previous research evaluating cognitive function in football [53], it is possible that a more significant relationship could have existed between saccadic performance and another head impact variable, such as those explored by other researchers [16].

We used an automated onset detection algorithm to detect changes in EOG voltages. Other researchers have used specific voltage thresholds as large as 25% of the maximum EOG signal amplitude [73]. Accordingly, the latencies that we report may be different than other studies based on this onset detection approach. However, we believe the advantages of using a consistent and automated analysis were important.

The correction algorithm used to determine the linear acceleration and rotational velocity at the heads COM were calculated using a Riddell Revolution Speed helmet over a Hybrid III head form of a 50^{th} percentile adult male. The head continues to grow until the early 20's [74], so the head sizes of our participants were likely smaller than those used create this algorithm. As well, the players in this study had a variety of helmets. This may have resulted in small calculation errors.

We did not collect the players football participation history. It is possible that the level of previous football experience could have an effect on the athlete's head impact exposures.

6 Conclusion

To the best of our knowledge, this is the first study to report head impact data in youth Canadian football players. We observed that the number of hits ranged from 10 to 572 per player, which is comparable to studies on American youth football players. We observed that the peak linear accelerations ranged from 13.1 to 106.6 g, which is also comparable to studies on American youth football players.

We hypothesized that players who were exposed to a season's worth of head impacts would experience impairments in their executive function. Our results did not support this hypothesis. Therefore, we conclude that a season's worth of head impact exposures does not cause inhibitory executive functioning impairments in youth Canadian football players.

We hope that this research will stimulate more studies exploring the field of subconcussive head impact exposures in Canadian football, specifically in the youth population. Creating a large database for this population will help researchers understand the potential harmful effects on neurological function in both the short and long term.

References

1. McCrory, P.R. and S.F. Berkovic, *Concussion: the history of clinical and pathophysiological concepts and misconceptions.* Neurology, 2001. **57**(12): p. 2283-2289.
2. McCrory, P., et al., *Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016.* British Journal of Sports Medicine, 2017.
3. Kerr, Z.Y., et al., *Concussion symptoms and return to play time in youth, high school, and college american football athletes.* JAMA Pediatrics, 2016. **170**(7): p. 647-653.
4. Government of Canada. *Concussions in sport*. 2018 2018, July 2nd; Available from: https://www.canada.ca/en/canadian-heritage/services/concussions.
5. Apps, J.N. and K.D. Walter, *Pediatric and Adolescent Concussion: Diagnosis, Management, and Outcomes*. 1. Aufl. ed. 2011, New York, NY: Springer-Verlag.
6. Keays, G., D. Friedman, and I. Gagnon, *A 20-Year Comparison of Football-Related Injuries in American and Canadian Youth Aged 6 to 17 Years.* Clinical Pediatrics, 2015. **55**(7): p. 603-613.
7. Blakemore, S.-J., S. Burnett, and R.E. Dahl, *The role of puberty in the developing adolescent brain.* Human brain mapping, 2010. **31**(6): p. 926-933.
8. Anderson, V., M. Spencer-Smith, and A. Wood, *Do children really recover better? Neurobehavioural plasticity after early brain insult.* Brain, 2011. **134**(Pt 8): p. 2197-221.
9. Stamm, J.M., et al., *Age of first exposure to football and later-life cognitive impairment in former NFL players.* Neurology, 2015. **84**(11): p. 1114-1120.
10. Bailes, J.E., et al., *Role of subconcussion in repetitive mild traumatic brain injury.* J Neurosurg, 2013. **119**(5): p. 1235-45.
11. Dashnaw, M.L., A.L. Petraglia, and J.E. Bailes, *An overview of the basic science of concussion and subconcussion: where we are and where we are going.* Neurosurg Focus, 2012. **33**(6): p. E5: 1-9.
12. McKee, A.C., et al., *Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy After Repetitive Head Injury.* Journal of Neuropathology and Experimental Neurology, 2009. **68**(7): p. 709-735.
13. Gavett, B.E., R.A. Stern, and A.C. McKee, *Chronic traumatic encephalopathy: a potential late effect of sport-related concussive and subconcussive head trauma.* Clin Sports Med, 2011. **30**(1): p. 179-88, xi.
14. Hwang, S., et al., *Vestibular Dysfunction after Subconcussive Head Impact.* Journal of Neurotrauma, 2017. **34**(1): p. 8-15.

15. Kawata, K., et al., *Association of Football Subconcussive Head Impacts With Ocular Near Point of Convergence.* JAMA Ophthalmol, 2016. **134**(7): p. 763-9.

16. Slobounov, S.M., et al., *The effect of repetitive subconcussive collisions on brain integrity in collegiate football players over a single football season: A multi-modal neuroimaging study.* NeuroImage: Clinical, 2017. **14**: p. 708-718.

17. Pellman, E.J., et al., *Concussion in professional football: location and direction of helmet impacts-Part 2.* Neurosurgery, 2003. **53**(6): p. 1328-1341.

18. Pellman, E.J., et al., *Concussion in professional football: reconstruction of game impacts and injuries.* Neurosurgery, 2003. **53**(4): p. 799-814.

19. Viano, D.C., et al., *Concussion in professional football: performance of newer helmets in reconstructed game impacts--Part 13.* Neurosurgery - Journal Article, 2006. **59**(3): p. 591.

20. Newman, J.A., et al., *Verification of biomechanical methods employed in a comprehensive study of mild traumatic brain injury and the effectiveness of American football helmets.* Journal of Biomechanics, 2005. **38**(7): p. 1469-1481.

21. Moon, D.W., C.W. Beedle, and C.R. Kovacic, *Peak head acceleration of athletes during competition--football.* Medicine and science in sports, 1971. **3**(1): p. 44.

22. Duma, S.M., et al., *Analysis of Real-time Head Accelerations in Collegiate Football Players.* Clinical Journal of Sport Medicine, 2005. **15**(1): p. 3-8.

23. Brolinson, P.G., et al., *Analysis of Linear Head Accelerations from Collegiate Football Impacts.* Current Sports Medicine Reports, 2006. **5**(1): p. 23-28.

24. Rowson, S., et al., *Linear and angular head acceleration measurements in collegiate football.* Journal of biomechanical engineering, 2009. **131**(6): p. 061016.

25. Funk, J.R., et al., *Validation of Concussion Risk Curves for Collegiate Football Players Derived from HITS Data.* Annals of Biomedical Engineering, 2012. **40**(1): p. 79-89.

26. Crisco, J.J., et al., *Frequency and location of head impact exposures in individual collegiate football players.* Journal of athletic training, 2010. **45**(6): p. 549-559.

27. Urban, J.E., et al., *Head Impact Exposure in Youth Football: High School Ages 14 to 18 Years and Cumulative Impact Analysis.* Annals of Biomedical Engineering, 2013. **41**(12): p. 2474-2487.

28. Broglio, S.P., et al., *Estimation of head impact exposure in high school football: implications for regulating contact practices.* Am J Sports Med, 2013. **41**(12): p. 2877-84.

29. Campolettano, E.T., S. Rowson, and S.M. Duma, *Drill-specific head impact exposure in youth football practice.* Journal of Neurosurgery: Pediatrics, 2016. **18**(5): p. 536-541.

30. Cobb, B.R., et al., *Head impact exposure in youth football: elementary school ages 9-12 years and the effect of practice structure.* Ann Biomed Eng, 2013. **41**(12): p. 2463-73.

31. Daniel, R.W., S. Rowson, and S.M. Duma, *Head impact exposure in youth football.* Ann Biomed Eng, 2012. **40**(4): p. 976-81.

32. Munce, T.A., et al., *Head Impact Exposure and Neurologic Function of Youth Football Players.* Medicine & Science in Sports & Exercise, 2015. **47**(8): p. 1567-1576.

33. Kelley, M.E., et al., *Head Impact Exposure in Youth Football: Comparing Age- and Weight-Based Levels of Play.* Journal of Neurotrauma, 2017. **34**(11): p. 1939-1947.

34. Talavage, T.M., et al., *Functionally-detected cognitive impairment in high school football players without clinically-diagnosed concussion.* Journal of neurotrauma, 2014. **31**(4): p. 327-338.

35. Tsushima, W.T., et al., *Are There Subconcussive Neuropsychological Effects in Youth Sports? An Exploratory Study of High- and Low-Contact Sports.* Appl Neuropsychol Child, 2016. **5**(2): p. 149-55.

36. Bahrami, N., et al., *Subconcussive Head Impact Exposure and White Matter Tract Changes over a Single Season of Youth Football.* Radiology, 2016. **281**(3): p. 919-926.

37. Kochunov, P., et al., *Relationship between white matter fractional anisotropy and other indices of cerebral health in normal aging: Tract-based spatial statistics study of aging.* NeuroImage, 2007. **35**(2): p. 478-487.

38. Johnson, G.H., Jeff; Proulx, Andre, *2017 Rule Book: The Official Playing Rules for the Canadian Football League*. 2017, Canadian Football League. p. 207.

39. Goodell, R., *Official Playing Rules of the National Football League*. 2018, National Football League. p. 89.

40. Ocwieja, K.E., et al., *The effect of play type and collision closing distance on head impact biomechanics.* Ann Biomed Eng, 2012. **40**(1): p. 90-6.

41. Lezak, M.D., *THE PROBLEM OF ASSESSING EXECUTIVE FUNCTIONS.* International Journal of Psychology, 1982. **17**(1-4): p. 281-297.

42. Brooks, J., et al., *Assessment of executive function in patients with mild traumatic brain injury.* The Journal of trauma, 1999. **46**(1): p. 159.

43. McDonald, B.C., L.A. Flashman, and A.J. Saykin, *Executive dysfunction following traumatic brain injury: neural substrates and treatment strategies.* NeuroRehabilitation, 2002. **17**(4): p. 333-44.

44. Hallett, P.E., *Primary and secondary saccades to goals defined by instructions.* Vision Research, 1978. **18**(10): p. 1279-1296.

45. Munoz, D.P. and S. Everling, *Look away: the anti-saccade task and the voluntary control of eye movement.* Nat Rev Neurosci, 2004. **5**(3): p. 218-28.

46. Antoniades, C., et al., *An internationally standardised antisaccade protocol.* Vision Research, 2013. **84**: p. 1-5.

47. Fischer, B., S. Gezeck, and K. Hartnegg, *On the production and correction of involuntary prosaccades in a gap antisaccade task.* Vision Research, 2000. **40**(16): p. 2211-2217.

48. Chaudhuri, A., A. Dasgupta, and A. Routray. *Video & EOG based investigation of pure saccades in human subjects*. IEEE.

49. Ethridge, L.E., et al., *Consider the context: Blocked versus interleaved presentation of antisaccade trials.* Psychophysiology, 2009. **46**(5): p. 1100-1107.

50. Richards, J.E., *Cortical sources of event-related potentials in the prosaccade and antisaccade task.* Psychophysiology, 2003. **40**(6): p. 878-894.

51. Kirenskaya, A.V., et al., *The Antisaccade Task Performance Deficit and Specific CNV Abnormalities in Patients with Stereotyped Paraphilia and Schizophrenia.* Journal of Forensic Sciences, 2013. **58**(5): p. 1219-1226.

52. Malmivuo, J. and R. Plonsey, *Bioelectromagnetism: Principles and Applications of Bioelectric and Biomagnetic Fields*. 1995, Oxford University Press: New York. p. 437-446.

53. Brooks, J.S., *The use of P3b as an indicator of neurophysiologic change from subconcussive impacts in football players.* Electronic Thesis and Dissertation Repository, 2016. **4253**.

54. Campbell, K., *Quantifying and Comparing the Head Impact Biomechanics of Different Player Positions for Canadian University Football.* Electronic Thesis and Dissertation Repository, 2014. **2259**.

55. Muise, D.P., S.J. MacKenzie, and T.M. Sutherland, *Frequency and Magnitude of Head Accelerations in a Canadian Interuniversity Sport Football Team's Training Camp and Season.* International Journal of Athletic Therapy and Training, 2016. **21**(5): p. 36-41.

56. Campbell, K., et al., *Laboratory Evaluation of the gForce Tracker™, a Head Impact Kinematic Measuring Device for Use in Football Helmets*. Vol. 44. 2015.

57. Santello, M. and M.J. McDonagh, *The control of timing and amplitude of EMG activity in landing movements in humans.* Experimental Physiology, 1998. **83**(6): p. 857-874.

58. Mukaka, M.M., *Statistics corner: A guide to appropriate use of correlation coefficient in medical research.* Malawi medical journal : the journal of Medical Association of Malawi - Journal Article, 2012. **24**(3): p. 69.

59. Coe, B.C. and D.P. Munoz, *Mechanisms of saccade suppression revealed in the anti-saccade task.* Philos Trans R Soc Lond B Biol Sci, 2017. **372**(1718).

60. King, D., et al., *The Influence of Head Impact Threshold for Reporting Data in Contact and Collision Sports: Systematic Review and Original Data Analysis.* Sports Medicine, 2016. **46**(2): p. 151-169.

61. Martini, D., et al., *Subconcussive head impact biomechanics: comparing differing offensive schemes.* Medicine and science in sports and exercise, 2013. **45**(4): p. 755-761.

62. Kleiven, S., *Why Most Traumatic Brain Injuries are Not Caused by Linear Acceleration but Skull Fractures are.* Frontiers in bioengineering and biotechnology, 2013. **1**: p. 15-15.

63. Canada, G.o., *Rowan's Law (Concussion Safety), 2018, S.O. 2018, c. 1 - Bill 193*. 2018.

64. Schussler, E., et al., *The Effect of Tackling Training on Head Accelerations in Youth American Football.* International journal of sports physical therapy, 2018. **13**(2): p. 229-237.

65. Kelley, M., et al., *Head impact exposure measured in a single youth football team during practice drills.* Journal of Neurosurgery: Pediatrics PED, 2017. **20**(5): p. 489-497.

66. Bucci, M.P. and M. Seassau, *Saccadic eye movements in children: a developmental study.* Experimental Brain Research, 2012. **222**(1): p. 21-30.

67. Klein, C. and B. Fischer, *Instrumental and test–retest reliability of saccadic measures.* Biological Psychology, 2005. **68**(3): p. 201-213.

68. Chan, Y.H., *Biostatistics 104: correlational analysis.* Singapore Med J, 2003. **44**(12): p. 614-9.

69. Krafft, C.E., et al., *An 8-month randomized controlled exercise trial alters brain activation during cognitive tasks in overweight children.* Obesity, 2014. **22**(1): p. 232-242.

70. Davis, C.L., et al., *Exercise Improves Executive Function and Achievement and Alters Brain Activation in Overweight Children: A Randomized, Controlled Trial.* Health Psychology, 2011. **30**(1): p. 91-98.

71. Diamond, A. and K. Lee, *Interventions Shown to Aid Executive Function Development in Children 4 to 12 Years Old.* Science, 2011. **333**(6045): p. 959-964.

72. Chaddock-Heyman, L., et al., *The effects of physical activity on functional MRI activation associated with cognitive control in children: a randomized controlled intervention.* Frontiers in human neuroscience, 2013. **7**: p. 72.

73. Aungsakun, S., et al., *Development of Robust EOG-Based Human-Computer Interface Controlled by Eight-Directional Eye Movements*. Vol. 7. 2012. 2196-2208.

74. Huelke, D.F., *An Overview of Anatomical Considerations of Infants and Children in the Adult World of Automobile Safety Design.* Annual Proceedings / Association for the Advancement of Automotive Medicine, 1998. **42**: p. 93-113.

Appendices

Appendix A: Health History Questionnaire

1. How old are you? _____

2. Have you ever been diagnosed with a concussion? Yes ☐ No ☐

If so, how many? _____

3. Are you currently experiencing any concussive symptoms?

_____

Please check all symptoms you have and indicate how long each symptom lasted for and severity (1 = barely symptomatic, 6 = severely symptomatic):

	Symptom	Duration	Severity (1-6)
☐	Headache		
☐	Nausea/Vomiting		
☐	Dizziness		
☐	Light Sensitivity		
☐	Drowsiness/Tiredness		
☐	Confusion/Irritability		
☐	Seizures		
☐	One-sided paralysis or sensory loss		
☐	Unsteadiness		
☐	Memory problems		
☐	Slurred Speech		
☐	Other (specify): _____		
☐	Loss of Consciousness		

4. Do you have any neurological disorders? Yes ☐ No ☐

5. If so, which disorder? _____

Curriculum Vitae

Name: William Justin Smith

Post-secondary Education and Degrees:
Western University
London, Ontario, Canada
2013-2017 B.A.

Western University
London, Ontario, Canada
2017-2018 M.Sc

Honours and Awards:
Deans Honour List
2017

Western Graduate Research Scholarship
2017-2018

Related Work Experience
Graduate Teaching Assistant
Western University
2017-2018

Publications:

Smith, William J.; Richards, Dillon J.; Zhou, Shibo H.; and Kennedy, Tanner (2017) "The Effect of Plantar Flexor Fatigue and Cognitive Recall Task on Standing Balance" *WURJ: Health and Natural Sciences*: Vol. 8 : Iss. 1 , Article 14
DOI: 10.5206/wurjhns.2017-18.11