THE CUMULATIVE EFFECTS OF PROLONGED REPETITIVE HEAD IMPACT EXPOSURE INTO ADULTHOOD

by

Katherine J. Hunzinger

A dissertation submitted to the Faculty of the University of Delaware in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Biomechanics and Movement Science

Winter 2022

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ACKNOWLEDGMENTS

If you were to ask me 15 years ago if I saw myself doing a PhD I would have laughed. If you were to ask me 7 years ago if I saw myself doing a PhD, I still would have laughed and had you admitted to an asylum. However, life has a funny way of working out and going full circle. So as much as I am grateful for the opportunities afforded to me which permitted my success, I am equally grateful for the opportunities that did not happen that led me here.

First and foremost, I would like to thank my advisors Dr. Tom Buckley and Dr. Buz Swanik. Graduate school and PhD life are filled with horror stories, but I can truly say I didn't experience that. Both Tom and Buz have always been my number one fans, nudging and pushing me when I needed guidance and motivation, but also mentoring me and serving as supporters and teammates in my moments of need. I have always felt respected as an equal. They have never doubted my abilities and always allowed me to do anything I wanted (without reason, of course) to improve my skillset and gain experience. I am eternally grateful to you both.

In addition, I would like to thank my committee members: Dr. Jaclyn Caccese, Dr. Rebekah Mannix, Dr. Bill Meehan III, and Dr. Jocelyn Hafer. Thank you for your countless hours, advice, and commitment to my academic improvement, betterment, and success.

Next, I would like to thank my other academic mentors who helped guide me to along a winding pathway that led me to doing a PhD: Dr. Anthony DeLegge, Dr. Stephen Howell, Dr. Andreana Holowatyj, Dr. Peter Brubaker, Dr. Stephen Messier, and Dr. Michelle Ness. In their own unique ways, they have all mentored and guided me to be as successful as I am, in addition to providing me with invaluable research

and clinical experience. It is crazy to think a failed military career, brief collegiate basketball career (thanks Coach Gillespie), and collegiate rugby career paired with multiple undergraduate major changes led me right back to where I started and where I always wanted to be: biomedical/biomechanical/epidemiological research involving the military and athletes. So, although there's too many to name, thank you to those who "failed" me, leading me to a path of eventual success filled with many lessons learned.

I would not be half the person without the rugby community which has afforded me numerous opportunities for development as a referee, player, researcher, mentor, coach, partner, and human. Thank you to the rugby community for teaching me to accept myself, love myself, and be the best version of myself; I could not have done this dissertation without the support of such an amazing community. You are my family.

Thank you to all my CrossFit family here in Newark, DE and Elkton, MD. CrossFit has provided me with physical, mental, and social wellness in addition to lifelong friends during my four years here. All of you have personally celebrated my academic and research PRs as much as my barbell PRs.

Next, thank you to my peers: Dr. Dave Grenet, Dr. Jessie Oldham, Dr. Melissa DiFabio, Shawn Hanlon, Dr. Tori Wahlquist, Dr. Nao Ito, Dr. Rachel Knobel, Dr. Elanna Arhos, Dr. Fernando Santos, Rebecca Daniels, Alissa Strouse (Dragon), Dr. Corey Koller, James Tracy, and Jacky Chou. From movie nights and lunch dates, frantic phone calls for help and advice, random dance parties in the kitchenette, biomechanics lab dad jokes, to beers and complaining. Thank you all for being a friend.

I value all my other friends from my various places of residence who have continued to support me. A special shout out goes to “One Croc to Rule Them All”. Jaclyn and Kelsey have been my biggest supporters, friends, and mentors since Day 1 at the University of Delaware, and I am thankful to have you both in my professional and personal life. Furthermore, what would I do without my work wife, Dr. Kelsey Bryk (read: BRICK HOUSE). It is not often you love your lab mates enough to have them as your maid of hono(u)r in your wedding, but there’s no one else I’d rather do a dissertation with during a pandemic in a foreign country with than you! Thanks for being my international travel buddy, teacher, mentor, confidant, and best friend.

Lastly, I want to thank my family. Although they may not truly understand what it takes to do a PhD or what my research is about, they have always shown interest and supported me through all the ups and downs. Thank you for the countless phone calls where you provided a soundboard for me allowing me rant without feedback. Thank you for the random care packages and notes of love and support. Thank you for always supporting my journeys, no matter how crazy. More importantly, thank you to my fiancée, Ash. You have been on this journey with me since practically Day 1. You have always supported me and my workaholic tendencies all the while providing me with your chaotic nature as good balance. You make me a better human every day. I love you always and could not have done this without you.

This PhD is the product of everyone listed here in addition to many more. This PhD is for all of you.

We did it. Roll Hens! Go Deacs! Let’s Go Huskies!

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ABSTRACT

BACKGROUND: Roughly 70-90% of the 2.8 million traumatic brain injuries (TBIs) that occur annually are mild (mTBI). Although a fairly benign injury, acutely, with clinical sign and symptom resolution occurring devoid of intervention in a few weeks, subtle neurophysiological deficits may persist beyond standard clinical recovery timelines of about one month. These deficits may be linked to dual-task and/or neuromuscular control dysfunction which may be the cause for the increased risk of subsequent lower extremity musculoskeletal injury (LE-MSI) post-concussion reported in youth, collegiate, and professional athletes; however, data are limited on females and community athletes. Further, beyond concussion, the long-term effects of repetitive head impacts (RHI) or impacts that do not result in clinical signs and symptoms of concussion remain a concern. Data are mixed on the long-term effects of RHI measured by age of first exposure (AFE) to contact/collision sports with some studies suggesting cognitive and emotional dysfunction in middle age and other studies suggesting no effect. Yet, these studies are limited to football and soccer playing middle aged males in addition to lacking objective measures of neurophysiological health and comparison groups. Further, the sex-specific response to RHI and the moderating effect of physical activity remain to be elucidated across the age spectrum. **PURPOSE:** The overall purpose of this dissertation was to identify the long-term effects of collision sport participation across individuals with varying levels of lifetime RHI exposure and physical activity levels. **METHODS:** For Aim 1, 1,037 (31.6 $\pm$ 11.3 years, 40.9% female) community level rugby players completed an

online survey via Qualtrics detailing demographics, sport history and AFE to sport, concussion and LE-MSI history, and patient reported outcomes (Brief-Symptom Inventory-18 (BSI-18), Short-Form 12 (SF-12 Physical and Mental Component Summary (PCS/MCS), and Satisfaction with Life Scale (SWLS)). Odds ratios (OR) by sex were performed to determine the odds of LE-MSI given history of concussion and a binary logistic regression to determine if there were sex differences in risk. Mann-Whitney U tests were performed to compare AFE groups (<12 and $\geq$ 12 yr) for BSI-18, SF-12, and SWLS. Generalized linear models were performed to examine the association between AFE (continuous) and patient reported outcomes; predictors included age, career duration (i.e., cumulative years contact/collision sports), and concussion history (yes/no). For Aims 2 and 3, 113 adults were recruited representing four groups: 1) Former non-contact athletes/non-athletes who are not physically active (NON; N=28, 35.4 $\pm$ 14.1 years); 2) Former non-contact athletes who are physically active (NCA; N=29, 33.9 $\pm$ 10.8 years); 3) Former contact/collision sport athletes who participated in high risk for RHI sports (i.e., boxing, football, ice hockey, lacrosse, soccer, wrestling) who are physically active (HRS; N=29, 33.3 $\pm$ 8.4 years); and 4) current and former rugby players, with a history of playing rugby after the age of 22 (e.g., prolonged RHI exposure into adulthood after the age at which most collision sport participation ceases) (RUG; N=27, 38.1 $\pm$ 13.0 years). All participants completed an online questionnaire of demographics, injury history, sport history, and patient reported outcomes, in addition to common concussion assessments measuring multiple domains and systems, and single (ST) and dual-task (DT) inertial

measurement unit instrumented gait and tandem gait. For Aim 2, a multiple regression was used to predict ST and DT gait outcomes in collision sport athletes from career duration. Groups were compared on all outcomes using Kruskal-Wallis test with a pairwise comparison procedure and Bonferroni correction for multiple corrections. Follow up analyses utilized covariates (concussion, learning disorder history, and career duration) in a one-way ANCOVA to compare groups on all outcomes. The relationship between ST gait speed and performance on clinical tests and dual task cost (DTC) gait speed and performance on clinical tests were analyzed using multiple Spearman's rank-order correlations. For Aim 3, a linear regression was utilized to understand the effect of career duration and sex on gait outcomes (i.e., ST & DT gait speed, double support, stride length, and tandem gait, and their corresponding DTC outcomes). Further, a dummy variable was created to model the sex*career duration interaction and was also included in the model alongside sex and career duration in Block 1 using the "Enter" method. A second block with potential confounding factors (i.e., concussion history, LD/ADHD history, age, height) was also performed.

RESULTS: For Aim 1, there was a significant association between diagnosed concussion and any LE-MSI($\chi(1) = 13.055$, $p < 0.001$, OR = 2.30 [95%CI: 1.45, 3.65]). There were no differences between sex for risk of LE-MSI ($R^2 = 0.024$, $p = 0.999$). Whether analyzed continuously or dichotomously, younger AFE was not associated with worse patient-reported outcomes for either men or women. For Aim 2, career duration did not predict any of the gait outcomes ($p>0.05$). In adjusted models, there were no group differences on multiple outcomes. However, on 6/25 outcomes

(SF-12 PCS, Apathy Evaluation Scale, SWLS, Montreal Cognitive Assessment (MOCA), Balance Error Scoring System (BESS), ST tandem gait) the NON group did significantly worse than the NCA group. The NON group also performed worse than RUG on BESS and ST gait speed. Spearman correlations revealed weak correlations between ST gait speed and SCAT5 Symptom Severity (r_s = 0.282, p=0.002), Trails B (r_s=-0.219, p=0.021), and SWLS (r_s =0.282, p=0.002). As well as weak correlations between DTC gait speed and Trails A (r_s = -0.216, p=0.022), Trails B (r_s = -0.306, p<0.001), and MOCA (r_s = 0.248, p=0.008). For Aim 3, there was no effect of sex on the relationship between career duration and gait outcomes (p>0.05). **DISCUSSION:** Collectively, these results suggest that contact/collision sport participation does not affect early- to mid-adulthood neurophysiological health in males and females. Further, career duration was not a sensitive enough measure to detect any group differences on assessment of neurophysiological health, warranting future research utilizing more precise measures of lifetime RHI exposure to determine if there truly is not an effect present. However, concussion history remains a significant predictor of quality of life and wellness in addition to its relationship with increased odds of LE-MSI. Lastly, there does not appear to be a significant effect of RHI on sex among measurements of ST and DT gait implying that both sexes are equally unaffected. These findings add to the growing body of evidence that contact/collision sport participation and RHI do not negatively affect mid-life neurophysiological health and function.

Chapter 1

LITERATURE REVIEW

Defining Concussion

The 5th International Consensus Statement on Concussion in Sports (5th CIS) defines a concussion as a "traumatic brain injury (TBI) induced by biomechanical forces" resulting in various clinical signs and symptoms.[1] These neurological signs and symptoms (e.g., headache, cognitive dysfunction, loss of consciousness, sleep and balance disturbances, and behavioral changes) have been considered the hallmark of a concussion and can arise in the absence of macroscopic neural damage.[2] These symptoms may include a variety of psychological and somatic symptoms, impairments in neuropsychological and cognitive function, and alterations in postural stability.[2–4] The biological mechanisms and underpinnings of these signs and symptoms may be the result of a neurometabolic cascade.[5] Moreover, individuals affected by a concussion, or mild traumatic brain injury (mTBI), typically reach clinical recovery in 10-14 days[1] with return to play at 12 days.[6] More recently, data from the NCAA-DoD Concussion Assessment, Research and Education (CARE) Consortium revealed that 50% of student-athletes were symptom free by day 6 post injury, with 92% of student athletes having begun or completed return to play (RTP) protocol at 28 days post-injury, and 85% having returned to sport.[7] Importantly, data were highly variable, yet most athletes were deemed recovered by about 1 month post injury.[7]

Pathophysiology of Concussion

A concussion will occur when rotational and/or linear forces are transmitted to the brain via indirect or direct physical contact; however, there is no established biomechanical threshold for the diagnosis of a clinical concussion.[8] A biomechanical injury may cause neuronal shearing and stretching, which in turn, produces an ionic flux and hyperacute indiscriminate glutamate release.[5,9] Specifically, there is an efflux of potassium ions paired with an influx of sodium and calcium due to mechanoreception of the lipid membranes in the cells.[5] This ionic influx of positively charged ions creates depolarization, which can subsequently trigger ligand- or voltage-gated ion channels, creating a diffuse "spreading depression-like" state which may be the biological mechanism for acute post-concussion signs and symptoms (e.g., migraine, phonophobia, and photophobia).[5]

To restore homeostasis within the cells, membrane pumps become activated, requiring the use of large amounts of adenosine-triphosphate (ATP).[5,9] As a result of the increased energy demand, hyperglycolysis occurs as ATP is broken down into ADP and an inorganic photsphate.[5,10] This aerobic metabolism, which relies on a demand for glucose to produce ATP, and hyperglycolysis creates a discrepancy in glucose supply and demand, thus, reducing cerebral blood flow.[5] Due to this increased production in ATP and subsequent breakdown of ATP into ADP+P_i, waste products such as lactate are formed, causing excess accumulation. Additionally, the calcium ion influx can leads to buildup of calcium into the mitochondria to prevent excess intracellular levels of calcium; however, this results in potential mitochondrial

dysfunction, which could worsen the cellular energy crisis by affecting ATP production.[5]

Of note, after the initial changes in ion concentration, glucose metabolism rates remain impaired for up to 7 to 10 days in adult animals; this impairment is correlated with behavioral impairments in spatial learning.[5] Moreover, a second head injury during this alteration in glucose metabolism may result in worsened neurocognitive function and reduced glucose metabolism, giving rise to the theory of metabolic vulnerability post-concussion.[5]

Epidemiology of Concussion

Early literature describing the epidemiology of concussions in the United States estimated that 1.6-3.8 million sport related concussions occurred annually.[11] However, this number is likely an inaccurate representation of the number of concussions in the U.S. due to factors like reporting and various definitions of concussion.[12] In American high school and collegiate athletes, concussions comprise ~13-19% of all sport related injuries.[13,14] Specifically, rugby athletes exhibit the highest concussion risk (when assessed as concussions per athletic exposure (AE)) at 28.25 concussions per 10,000 AEs, a rate 3 times that of American football, with women having elevated risk for concussion compared to men.[15]

In the United States, roughly 2.8 million TBI-related emergency department visits, hospitalizations, and deaths occurred in a single year; with ~70% of TBI-related visits for concussion, costing $21.4 billion in hospital admission costs alone.[16] This

has resulted in approximately $75.6 billion (2019 USD) in annual indirect costs and $93.0 billion (2019 USD) in lifetime costs.[16,17] Individually, adults spend ~$13,564 within the first year following a concussion, with majority of payments occurring in the first three months towards inpatient services and physician appointments.[18]

Diagnosing Concussion

Clinical signs and symptoms are the hallmark of a concussion and diagnosing a concussion.[1] In particular, diagnosis of a concussion can include one or more of the following clinical areas: 1) physical signs (e.g., neurological deficits, post-traumatic amnesia), 2) cognitive (e.g. "feeling in a fog"), emotional (e.g. anxious), and/or somatic symptoms (e.g. headache), 3) behavioral changes, 4) sleep/wake disturbances (e.g. too much or too little), 5) balance alterations (e.g. altered postural control), and/or 6) cognitive impairments (e.g. alterations in reaction time).[1] However, these signs and symptoms are non-specific to concussion, so knowledge of baseline signs and symptom reporting is helpful in diagnosis of concussion as baseline levels of signs and symptoms (e.g., trait anxiety), indicating the criteria for persistent concussion symptoms, have been reported in non-concussed athletes.[1,19]

Of these clinical signs and symptoms, headache is the most commonly reported symptom, being present in ~71-95% of sport related concussions.[20,21] Some of the least reported symptoms include loss of consciousness and various types of amnesia. In regards to sex differences, it has been reported that not only do the sexes differ in symptom reporting, with females having higher symptoms post-concussion,

but females are more likely than males to report a symptom at baseline.[22,23] Unfortunately, due to underreporting of concussive symptoms, a diagnosis of concussion solely based on signs and symptoms may be difficult.[24,25] Therefore, the 5th CIS recommends a multifaceted assessment of concussion at baseline (i.e. pre sport participation) and post-injury.[1] Some of these assessments include measures of neurocognitive function (e.g. immediate post-concussion assessment and cognitive test, ImPACT, or standard assessment of concussion, SAC), balance and postural control (e.g., balance error scoring test, BESS), along with common sideline tools to assess symptoms (e.g., sport concussion assessment tool, 5th edition, SCAT5).[26,27] Recently, there is emerging evidence to include measures of postural control and neurocognitive function by assessing tandem gait and dual task gait.[28,29] Data from the CARE Consortium (SCAT symptoms, BESS, SAC, time of injury, and student athlete demographics) were utilized to estimate risk scores to aide in the diagnosis of either a definite, probable, possible, or unlikely concussion; data were categorized using a classification and regression tree (CART).[30] Collectively, the algorithm was highly accurate at determining probable and definite concussions (Sensitivity ranging from 91.07-97.40%). Unsurprisingly, those determined to have a definite concussion by the model had lower SAC, but higher SCAT symptom and BESS scores.[30]

Acute Effects of Concussion

Clinical Recovery

Historically, it has been reported that most athletes return to baseline values on clinical tests paired with symptom resolution around 7-10 days.[4,31] This was based off of a 2003 report from the NCAA Concussion Study (1999-2001) which included NCAA football players that showed football players returning to baseline graded symptom checklist (GSC) levels by day 7 post-injury. Additionally, within this cohort, the concussed individuals recovered cognitively—based on SAC total score—by day 7 and postural stability—assessed by BESS total score—resolved by day 5. The highlight of this study was that symptoms, cognitive functioning, and postural stability all resolved over time within this cohort of collegiate football players. Moreover, each of these three domains appeared to recover independent of one another, and varied from player to player, highlighting the heterogeneity of a concussive injury.[4] Another key report from this study was a theorized period of "cerebral vulnerability", within 7-10 days post-injury, in which an individual who had suffered a concussion was at the greatest risk for a repeat concussion within the same season.[31] Herein researchers hypothesized that the brain was especially vulnerable post-concussion for another concussive injury because it was not physiologically recovered, exposing it to heightened risk for repeat injury; this period also corresponds to the period of altered glucose metabolism.[4,5,31]

However, in recent years, data from a large longitudinal study have posited that the recovery timeline may be longer than the previously thought 5-7 days. Researchers from the CARE Consortium (2014-2017) analyzed return to play data from the NCAA Concussion study (1991-2001) and the CARE Consortium.[6] In general, athletes from the NCAA study had a median symptom duration of 2 days, compared to ~6 days in the CARE study. The symptom free waiting period was around 1 day for NCAA study athletes and 6 days for CARE athletes. Lastly, athletes in the NCAA study returned to play (RTP) by a median of 3 days (mean = 6.67 $\pm$ 11.40 days) compared to a median of 12 days (mean = 16.08 $\pm$ 14.39 days) in the CARE consortium.[6] Lastly, the NCAA cohort had 92% of repeat concussions occur within the first 10 days of initial injury, compared to only 3.7% in the CARE cohort.[6]

Expanding on the CARE Consortium study, data from the entire CARE study (n=34,709 student athletes) were utilized to determine and define the natural history of concussion in collegiate sporting populations.[7] Median symptom duration was ~6 days with median RTP duration lasting ~13 days (IQR: 8.7-20.1 days). Researchers defined normal recovery as a process that can, and should take, up to one month. Lastly, demographic factors such as male sex, increased assessment frequency, and ADHD medication usage were reported to be related to shorter RTP duration. On the contrary, greater levels of post-injury symptom severity, training related concussion, and a history of 3 or more concussions were all associated with greater recovery timelines.[7]

Collectively these data highlight the advances in clinical management of concussion in collegiate athletes. Moreover, the greater acknowledgement of

symptoms and symptom duration period in the current CARE cohort may be the result of increased concussion symptom recognition, awareness, and improved reporting behaviours.[6,32] This has yielded a longer recovery time following sport related concussion, evidenced by the more conservative based strategies compared to the NCAA study. However, underlying subclinical deficits may persist beyond clinical recovery (i.e., return to baseline on concussion assessment battery).[33] Additionally, it is important to note that these data and recovery timelines are limited to collegiate student athletes and may not be generalizable to non-sporting and older populations.[6,7]

Neurophysiological Time to Recovery

A 2017 review aimed to identify the physiological time to recovery after concussion categorized into the following modalities: functional MRI (fMRI), diffusion tensor imaging (DTI), magnetic resonance spectroscopy (MRS), cerebral blood flow, electrophysiology, heart rate, exercise, fluid biomarkers, and transcranial magnetic stimulation (TMS).[33] Summary of findings are summarized below.

fMRI: Changes in cerebral activation persisted from 3 days to 23 months after clinical recovery of concussion or RTP.[33]

DTI: Changes in white matter have been detected and resolved before RTP,[34] whereas other methods and regions of interest have showed changes beyond RTP.[35,36]

MRS: Cerebral metabolic disturbances have been reported up to 30 days post injury despite RTP at day 15 post-injury.[35,37]

Cerebral Blood Flow: Abnormalities are present acutely post-injury; however, they tend to be resolved by day 30 in most cases. Yet, evidence is lacking so a true time course cannot be determined.[38,39]

Electrophysiology (electroencephalogram/EEG and event-related potential/ERP): There are possible alterations in electrophysiological measures beyond symptom resolution and clinical recovery, however, more rigorous studies are needed to confirm these results.[40,41]

Heart Rate: There is insufficient data at the present time to conclude that heart rate measure dysfunction persists beyond clinical recovery.[33] However, more recent studies in recently concussed individuals have reported blunted cardiovascular responses to the cold pressor test, indicating sympathetic dysfunction,[42] as well as long-term dysautonomia, evidenced by longer time to baseline heart rate variability measures post exercise in those with a history of 2 or more concussions.[43]

Exercise: At the time of the review, postinjury time course of exercise tolerance impairments remained to be defined.[33] More recently, numerous studies have reported the benefits of post-concussion aerobic exercise (>24-48 hours post injury) on reducing symptoms.[44–50]

Fluid Biomarkers: At the time of the aforementioned systematic review (2016 data synthesis), authors concluded that there still remained no reliable markers to monitor recovery.[33] However there have been promising data in recent years regarding the use of biomarkers collected at baseline and acutely following sport related concussion. Specifically, the Quanterix Neurology 4Plx "B" (N4PB) multiplex assay, consisting of

glial fibrillary acidic protein (GFAP), ubiquitin c-terminal hydrolase L1 (UCH-L1), total tau (T-tau), and neurofilament light (NF-L) have been used to discriminate between healthy controls and recently concussed collegiate student athletes.[51] Acutely post-concussion, GFAP and NF-L showed excellent discrimination between SRC and controls. However, these biomarkers were not related to clinical outcomes of symptoms, balance, and neurocognitive assessments or recovery trajectory.[51] Thus, future research is needed involving neuroimaging modalities to validate fluid biomarkers for diagnosing and tracking concussion recovery.

TMS: Abnormalities in cortical excitation have been reported beyond clinical recovery,[52] but further research is warranted to determine the true window of alterations in cortical excitability following concussion.[33]

Aside from the 2017 Kamins review paper,[33] a 2018 review which included 43 articles sought to review the criteria used to define recovery from sport related concussion in youth athletes (i.e., elementary to college age groups).[53] Despite varying definitions of concussion in the included articles, the most common method used to determine recovery from concussion was self-reported symptoms.[53] This is in line with data reporting that symptom (score and/or severity) have the greatest sensitivity and specificity when diagnosing a concussion.[54–57]

Taken together, these data highlight that neurophysiological recovery from concussion is well beyond the clinical recovery timeline of concussion. Moreover,

single task (ST) gait and dual-task (DT) gait data have also highlighted lingering deficits beyond clinical recovery.

Post-Concussion Gait

Single task gait consists of walking along a straight pathway at a self-selected pace. This movement is primarily controlled via subcortical locomotor processing, requiring minimal executive control in healthy individuals.[58] A variety of studies have investigated the effects of concussion on ST gait ranging from cross-sectional to longitudinal follow-up study designs using a variety of methods including 3-dimensional motion capture systems with integrated force platforms, gait mats, as well as a simple stopwatch and a walkway.[59]

A 2018 review assessed 26 articles that investigated differences in ST gait performance following concussion. Of this, 19 studies utilized adult populations with the other 7 involving adolescents;[59] of note, there is a paucity of research on ST gait in older adults (i.e., beyond college aged) following concussion. These studies investigated a variety of kinematic and kinetic variables during single task gait over a range of concussive injury timelines (e.g. $\leq$ 10 days, 11-90 days, 91-365 days, and > 1 year).[59]

Most of the included studies investigated the effects of concussion on gait speed (m/s) and found that it was primarily unaffected beyond the acute phase in adults with a concussion compared to controls; this null finding was also present for adolescent populations during all time groups of analysis.[60–67] However, some studies

did report that adults with concussion had significantly slower gait speed compared to healthy controls during the acute phase of $\leq$ 10 days.[59,60,62,68–71] As such, ST gait speed as a standalone assessment for measurement of clinical recovery from concussion is not considered a sufficiently sensitive assessment tool since it may recover before other assessment of concussion recovery return to baseline.[7,33,72]

Stride length during ST gait has been assessed via various measures including GAITRite[68] mats and motion analysis systems.[61] Similar to gait speed, stride length also had mixed results across the variety of recovery timelines which may be the result of individual characteristics (e.g., RTP progression timeline, concussion history) or measurement methods. The majority of studies for acute through 1-year recovery periods reported no significant differences between healthy and concussed or changes in stride length from baseline to post-concussion.[61,62,65,70,71] Only a few studies investigated stride time, with one finding a small increase in the acute stage,[69] yet a separate study found no change at 11-90 days.[62] Step width showed similar results with predominantly null findings across the recovery timeline.[59] Lastly, time spent in double support did not differ in the acute phase or long-term assessment timepoint, with no clear trend across the recovery timeline.[67,73–75] Thus, similar to gait speed, these measures during ST gait fail to be sensitive enough to detect subtle neurophysiological deficits of concussion.

Utilizing more sensitive methods (i.e., integrated force plates and 3-dimensional motion capture cameras), some studies did show increases in mediolateral (ML) movement of the center of mass (COM) during both the acute phase[65,69] as well

as up to 90 days post-concussive injury; this change may be indicative of altered postural control.[65] Contrarily, anteroposterior (AP) COM movement showed no differences, however, some studies did find a decrease in AP COM movement depending on the method used.[62,69,70]

Lastly, single-task gait assessment yielded a variety of null findings in which researchers did not find significant differences between groups or baseline and post-concussion assessments at any post-injury timepoint. These assessments included trunk fluidity,[64,76] trunk stability,[60] and joint coordination.[71]

Thus, although single task gait may highlight some impairments or abnormalities immediately (i.e., acutely) post-concussion, these differences between baseline performance or compared to healthy controls typically resolve over time with symptom resolution.[59] Furthermore, the subtle differences (i.e. altered ML COM movement) that do exist are not easily identifiable with simple biomechanical methods, requiring advanced and sometimes cumbersome equipment, which may lead to difficulty for clinicians in ascertainment and detection of dysfunction post-concussion. Therefore, more sensitive measures are needed to highlight balance and postural control deficits post-concussion, especially given that these deficits may be linked to subsequent musculoskeletal injury.[77–79] Moreover, of the studies included in the review, 21% found subacute deficits during simple ST gait, whereas 95% of the studies found deficits during DT or complex (e.g., obstacle clearance) gait, highlighting the need for more cognitively challenging tasks during gait to highlight subtle post-concussion impairments such as DT gait.[59]

Gait Initiation and Termination

Aside from steady-state ST gait, researchers have also investigated how concussion may affect gait initiation (GI) and gait termination (GT). Both GI and GT offer unique insights into postural control as they are inherently destabilizing tasks, challenging the postural control and balance systems to transition from either stable, static balance to gait, a continuously destabilizing task, through the generation of propulsive forces or vice versa with GT.[80,81] Both of these tasks are typically analyzed and described in reference to the subject's center of mass (COM) and pressure (COP) displacement, velocity, and acceleration, as well as foot position and muscle activity of the lower extremity.[80,82]

GI consists of three phases: anticipatory postural adjustment (APA) phase, S1; Transitional phase, S2; and Locomotor, S3.[81,83] Acutely post-concussion, individuals exhibit a conservative GI strategy, evidenced by reductions in posterior and lateral COP displacement during APA phase, compared to healthy controls and their own baseline (pre-injury) data.[81] Further, injured athletes initiated gait with a shorter step length and slower velocity. These deficits may be indicative of post-concussion neurophysiological dysfunction, specifically executive dysfunction and motor planning, which is controlled by the supplementary motor area.[81,84] However, these deficits may persist, on a more subtle level in those with a history of $\geq$3 concussions, further providing evidence of subtle deficits across the lifespan indicative of a conservative gait, which may be a fall prevention mechanism.[85]

Conversely to GI, GT recruits the CNS and feedforward mechanisms to anticipate, control, and brake/slow an individual's forward momentum by restricting the COM within one's base of support.[82,86] GT consists of three phases: Braking, S1; Transition, S2; and Stabilization, S3.[86] Similar to GI, acutely post-concussion

individuals display a conservative postural control strategy during GT evidenced by reductions in braking mediolateral COP displacement and velocity, and heightened and transitional anteroposterior COP displacement and velocity.[86] Thus, these data may highlight further subtle neurophysiological dysfunction and motor control alterations post-concussion.[86]

Dual-Task Gait

Purposeful locomotion commonly requires concurrent cognitive tasks while walking such as thinking about future actions, conversing, or holding an object.[87] Thus, gait with a concurrent task places a large demand on cognitive and sensory systems, requiring more executive functioning skills than ST gait.[88] The demands placed on the cognitive system during locomotion can be investigated using DT methods; in doing so, researchers can identify where performance is impacted the most during DT conditions (i.e. cognitive task or motor task) and even the prioritization of tasks.[87]

During DT gait, healthy adults tend to walk slower than ST conditions.[87] Additionally, there is a reduction in gait cadence in various DT conditions such as mental tracking, reaction time, verbal fluency, and working memory compared to ST.[87] DT gait also leads to a significant reduction in stride length.[87]This reduction in stride length is coupled with an increase in stride time and stride time variability.

Overall, DT conditions affect spatiotemporal parameters resulting in reduced gait speed, stride length, and cadence, and increased stride time and stride time

variability; in essence, healthy individuals slow down during dual task conditions.[87] This slowing down during DT suggests that cognitive systems are involved in the control of gait speed, specifically the ability to divide attention, proposed by the capacity-sharing theory.[89] Interestingly, gait speed has been shown to be associated with performance on executive functions and memory tests, and as such, gait speed control may involve executive functioning skills.[87] Performance on these combined tasks and the potential decrement in performance on either task allow researchers to classify the individual's prioritization of the task (i.e. do they put posture first or posture second) and see the extent to which the two tasks share attentional resources.[88,90] The attentional demands required for postural control during gait are dependent on the task at hand, an individual's balance skills, and the age of the individual among other things.[90] This difficulty in performing concurrent tasks has led to the development of multiple theories: the capacity-sharing theory, the bottleneck theory, and the multiple resources model.[89]

Dual-Task Prioritization

The capacity-sharing theory suggests that attentional resources are limited, and as such performing two attention-demanding tasks at the same time (e.g., walking and subtracting by 7) will produce a reduction in performance in at least one of those tasks.[89] Due to this shared capacity, if the time between the presentation of the two tasks is decreased, the time required to process will increase.[91] An assumption of the capacity-sharing theory is that one can voluntarily distribute attentional resources to a specific tasks, hence why performance of a secondary task (e.g., DT gait using serial 7's) during gait produces kinematic changes like a reduction in speed.[87,89]

The bottleneck theory proposes that if the two tasks are processed using the same neural networks or processor, a "bottleneck" will be created when processing the information, implying that only a set amount can be "sent through" at a time.[89] Thus, the processing of the added task is subsequently delayed until the neural processor has processed the initial task.[89] This theory is used to explain the delays seen in reaction times of a second task (e.g., when an individual has slowed responses to cognitive task on DT gait).[89] Similar to the capacity-sharing/limited resources theory, the bottleneck theory posits that DT gait produces slowed gait or impaired performance on the cognitive tasks, but only if the neural networks involved in the two tasks overlap; if they do not overlap then there should not be a reduction in performance.[89] Thus, selection of cognitive task for DT gait is important when investigating task-prioritization and performance deficits.

Lastly, the multiple resource model hypothesizes that neural processing requires an assortment of resources.[92–94] For instance, if the two tasks during DT testing do not require the same resources, then interference will not occur and there will not be a reduction in performance. This theory is used to explain why some DT paradigms do not show reductions in performance like performing a cognitive task (e.g., talking) while walking, whereas execution of other tasks, like doing a second motor task (e.g., holding an object), while walking results in performance changes due to utilization of the same resources.[89,95]

These theories have been used to describe the responses to DT paradigms in a variety of populations including the elderly, neurological disease (e.g., Parkinson's Disease), post-concussion, etc.[89,96] Other than requiring attentional resources, these DT paradigms cause the individual to prioritize tasks. Thus, researchers have

categorized DT responses as “posture first”, meaning the individual prioritizes postural stability and dynamic balance over the cognitive task, or “posture second” meaning the other task (e.g., object holding or mental math) is prioritized over maintaining postural stability.[89] Healthy adults and elderly individuals give priority to the stability of gait when walking and performing a concurrent cognitive task, meaning they put posture first (e.g., prioritize postural stability); this is evidenced by a decrease in step width variability in healthy adults and reductions in stride length and gait speed in older adults in an attempt to maintain upright posture and avoid falls.[59,97] This posture first strategy might be employed by the elderly and neurologically impaired populations as a means of increasing stability and preventing falls.[59,97]

Contrarily, we have seen posture second strategies employed in certain clinical populations. For instance, individuals with Parkinson’s Disease inaptly display a posture second strategy, evidenced by more motor errors (e.g., slowed/altered performance or cessation of walking) than healthy controls, which can result in an increased risk of falls. As such, fall risk may be increased by an individual’s motor impairments, reduced executive function skills, and an inability to appropriately divide attention and allocate attentional resources.[89]

Post-Concussion Dual-Task Gait

Unlike ST gait which utilizes minimal executive function for control, DT gait employs frontal lobe (i.e., dorsolateral prefrontal cortex[87]) executive functioning to enable the processing of concurrent cognitive and motor demands.[89] DT gait deficits appear to be present beyond symptom resolution.[66,98] DT function is commonly measured as dual-task cost (DTC), which represents the change between undivided

(i.e., single-task) and divided (i.e. dual-task) attention conditions.[99] These changes in DTC have been shown to differentiate control subjects from those with a concussion,[65] be predictive of prolonged concussive symptoms,[100] and outline post-concussion gender differences.[101]

Similar to ST, DT gait speeds are slower acutely post-concussion,[60,62,68–71,102] with some studies showing no change,[65,75,103] and unchanged at later periods of recovery in adult populations, indicating gradual resolution over time.[60,62,76] However, adolescents have not seemed to demonstrate changes in DT gait speed across all timepoints.[64,65,75] Furthermore, a systematic review and meta-analysis which utilized individual participant data revealed that individuals exhibited reductions in gait speed under DT conditions up to 2 months post-concussion, highlighting the clinical utility of DT assessesments.[104] However, the difference compared to controls was only 0.06 m/s which is not considered a clinically meaningful difference for gait velocity.[105]

Investigations into DT stride length have produced inconclusive results with the majority of studies reporting no change across all time points,[62,65,69–71,75] however, two studies have shown decreases in the acute phase when comparing DT gait to ST.[62,103] This significant decrease in stride length (e.g.1.34 m during ST vs. 1.23 m during DT in concussed individuals)[103], can be viewed as a compensatory mechanism to increase stability during gait, and that a concurrent cognitive task may impair gait performance.[103] Another strategy individuals may use to increase stability during gait and/or DT gait is to alter their stride width, however, studies have not shown any changes in DT stride width during the acute, 91-365 days, or > 1 year phases in

adolescents and adult subjects.[62,65,69–71,103] Moreover, early investigations did find a significant increase in stride time during DT gait.[69,70] Yet, three other studies reported no change during the acute phase as well as 11-90 days.[62,75,103] Lastly, DT double support time yielded no significant differences in double support time during the acute or long term periods.[61,74,75]

Dynamic stability measures are crucial to assess at post-concussion timepoints as impairments may go undetected during common clinical tests, yet results have proven to be quite mixed.[72] For instance, multiple studies have shown increases in ML COM movement between acute recovery and up to 90 days,[62,65,69,70,75,103] whereas others showed no change,[62,69,70,103,106] and one study showed a decrease during DT gait.[106] One thing to consider about these mixed findings is they are a result of researchers reporting different outcome measures of ML COM movement. More specifically, when comparing acutely concussed (<48 hours) to controls, ML COM displacement was smaller,[106] not significantly different,[62,66,70,107] or larger.[62,70] Maximum ML COM velocity was either not different[62,103] or faster[69,70] between acutely concussed adults and controls in the acute phase.[59] Lastly, ML COM acceleration was significantly slower during the transition between single support to double support portion of the gait cycle only in the acute and subacute phases.[106] Interestingly, one study found that DT ML COM displacement (r= -0.52, p = .004) and peak ML COM velocity (r= -0.37, p = .048) were significantly correlated with return to activity day in high school and collegiate athletes.[108] This result implies that those who return to activity sooner demonstrated more gait instability, which may

predispose individuals to subsequent injury risk.[108] In summary, the majority of COM changes have been reported in the frontal plane and minimal changes have been reported in the sagittal plane.[72]

Analysis of AP COM displacement yielded mixed results with some studies indicating reduced,[107] increased,[109] or no significant difference in concussed adults compared to controls during the acute phase, yet no differences at subacute.[62] Analysis of AP COM velocity produced null findings between groups,[62,107] whereas AP COM maximum velocity was typically slower in concussed athletes during the acute phase.[70] These results imply that post-concussion, individuals do not rely on sagittal plane motion as a compensatory mechanism, or it is unaffected, and as previously mentioned, changes are more likely to be observed in the frontal plane.[72]

During DT gait concussed individuals exhibit worse trunk stability and greater lower extremity joint variability between the knee and hip joints, and knee and ankle joints compared to controls in the acute and/or subacute phases.[60,71] This lack of limb coordination may impair balance and be indicative of sensory input and motor output deficits.[103] As a result of the added cognitive task during DT, which requires divided attention, these deficits may manifest. This is an important finding, as impairments in sensory input and motor output may produce insufficient reactive movement patterns during sport, which can increase one's risk for injury.

The main finding from the review on dual task gait in concussion is the fact that 18/19 studies found significant impairments during DT or complex gait.[59] Further, a review of individual participant data also confirmed this finding having reported that

DT deficits appear to exist upwards of 2 months post-concussion.[104] Particularly, DT gait impairments tend to manifest in the acute and subacute phase. This finding is critical for concussion management, as DT testing may be an advantageous addition to a concussion testing battery due to its greater clinical utility than ST gait by providing evidence of lingering motor and/or executive dysfunction deficits. Furthermore, compared to healthy individuals, those with a concussion exhibit a more conservative gait strategy characterized by a slower gait velocity, increased COM frontal plane displacement (ML), and a restricted COP-COM separation.[59,104] These biomechanical adjustments to gait may be a strategy utilized post-concussion to maximize dynamic stability, and a method of "posture-first" when performing DT functions.[59,98,103,108]

Concussion and Long-Term Health Outcomes

Studies on the relationship between concussion and long-term health outcomes have yielded mixed and inconsistent results due to the changing nature of the definition of concussion,[1] self-reported concussion history, and the variety of methods used.[110–112] Primarily, investigations have focused on cognitive, neuropsychiatric, and motor performance[113,114] in addition to mental health outcomes.[115,116]

One limitation of research on the long-term effects of concussion is that the research will always be limited to cohort longitudinal and epidemiological data due to the ethical concerns of a randomized controlled trial investigations into concussion and long-term health outcomes. In regards to mental health outcomes, Kerr et al. reported a relationship between the number of self-reported concussions and

diagnosed depression in former NFL players.[116] Moreover, epidemiological studies have found a relationship between concussion history and depression in a cohort from the National Survey of Children's Health 2007-2008 (3.3-fold increased risk)[117] as well as increased risk of dementia (HR=1.44) in those with a history of head injury in those in the Atherosclerosis Risk in Communities (ARIC) Study.[118] These data from the ARIC study revealed a dose-response relationship of head injuries and dementia risk, with stronger associations among females (HR=1.69 vs. 1.15 in males).[118]

More recently, data from the NFL-LONG study revealed a small, but significant relationship between depression symptom severity and concussion history; interestingly, years of participation of football and concussion history were not associated with the rate of change in depressive symptoms from 2001-2019.[115] Of note, a heightened decline in self-rated physical function was predictive of a steeper increase in depression symptoms over time, indicating that physical function may have a greater effect on quality of life (QoL) than history of concussion.[115]

In regards to neuropsychological performance and history of concussion, a meta-analysis revealed no overall effect of self-reported mTBI and worse performance on measures of executive functioning and delayed memory.[119] An early study also revealed no cumulative effect for 1-2 previous concussions among male high school and university athletes on ImPACT.[120] Moreover, a 2018 review highlighted no reported effect of concussion on neurocognitive assessment, despite the number of previous concussions being differentiated. This may be due to the fact that the majority of these neurocognitive assessments were designed and validated for short-

term assessment of concussion and may not be sensitive enough to detect subtle long-term impairments.[113] Aside of American football, data from the BRAIN Study, a cross-sectional cohort of elite male rugby union players older than 50 years, revealed no association between concussion history and cognitive function as measured by the Preclinical Alzheimer Cognitive Composite.[121] Yet, those older than 80 years with a history of 3 or more concussion did have worse cognitive functioning that those without a history of concussion, further highlighting the need to control for the interaction effects of age when investigating concussion and long-term health outcomes.[121] Lastly, a 2020 review of concussion and long-term cognitive functioning among elite/professional athletes reported the lack of high-quality or powered epidemiological studies on this topic.[122] Moreover, of the 14 included studies—which included ice hockey, rugby, boxing/mixed martial arts, and American football athletes—the data poorly suggested a small association between concussion history and worse cognitive function in later life, yet the clinical relevance is unknown.[122] Collectively, the magnitude of the effect of concussion on cognition is unclear, additionally the differences found may not be exclusively due to concussion and likely are affected by confounders (e.g., psychosocial risk factors, drug and alcohol use, lifestyle).[122]

Concussion and Subsequent Lower-Extremity Musculoskeletal Injury

The last decade has not only shown an increase in concussion research and education, but a shift towards post-concussion outcomes, particularly injury risk.

Given the increased risk for another concussion after RTP[31] and persistent deficits beyond clinical recovery and RTP,[78,123] post-concussion injury is highly plausible. As such, many researchers have sought to identify and quantify the odds and incidence rate ratios of musculoskeletal injury (MSI) after a concussion.

A 2018 systematic review and meta-analysis of articles published between January 2000 and November 2017 identified 8 articles investigating concussion and subsequent MSI.[77] The results of the meta-analysis revealed that athletes with a concussion were over 2 times as likely to suffer an MSI than athletes without a concussion (OR: 2.11; 95%CI 1.46-3.06).[77] Furthermore, those with a concussion had a statistically significant higher incidence of MSI after RTP compared to athletes without a concussion (IRR: 1.67; 95CI: 1.42-1.96).[77]

A limitation of this review was that it only included athletes (primarily collegiate and professional, with one high school cohort[124]) who were mostly male, limiting its generalizability to non-athletes and females.[77] However, the same results of increased MSI risk in those with a history of concussion have been found in Active Duty Soldiers[125], Reserve Officers' Training Corps cadets[126], amateur athletes,[127] and the general population (specifically ACL injury post-concussion).[128] Despite the growing abundance of research investigating concussion and subsequent MSI there is no confirmed mechanism behind this relationship for increased injury risk, yet, there are multiple theories. The most prominent theorized mechanisms for this increased risk of injury post-concussion appear to be disruptions in perception-action coupling loops[79] and altered neuromuscular control.[78]

Altered Neuromuscular Control

Specifically, Howell et al. hypothesized that neuromuscular control deficits, which may persist beyond apparent clinical recovery, may be a factor in increased injury risk.[78] In this case, neuromuscular control refers to the contributions to how the nervous system controls muscle activation (and inhibition), and in the end postural control.[78] Moreover, the authors suggested that divided attention during exercise and sport may place increased demands on cognitive loading (e.g. dual task deficits), causing an injury to happen due to an inability to accurately divide attentional resources.[78] This hypothesis is supported by research highlighting larger dual-task deficits in recently concussed individuals as cognitive tasks increased in complexity.[66,78,129] More specifically, the dual-task dysfunction is a result of the brain's inability to properly divide attentional resources when exposed to two different tasks (e.g. tandem gait and cognitive task like Serial 7's).[78,103,130] Lastly, it has been reported that gait impairments persist beyond clinical recovery with dual-task assessments identifying deficits beyond apparent recovery, suggesting dual-task neuromuscular control dysfunction.[59,61,78,106,131] Moreover, a prospective study found that NCAA athletes with concussion who sustained a LE-MSI in the year following concussion had significantly lower cognitive accuracy, walked slower, and spent more time in double support during DT gait compared to athletes with concussion who did not suffer an LE-MSI post-concussion.[132] These data highlight the evidence of a conservative gait strategy post-concussion which may aide in highlighting contributing factors to subsequent MSI.

Perception-Action Coupling Loop Disruption

Eagle et al.[79] argue against the neuromuscular control theory because the "top down" approach to the sensorimotor system creates a computational problem whereby the computational requirements of an indirect perception model propose that perception and subsequent action must be tightly "coupled" in order to accomplish goal-driven movement. As such, the authors propose an alternative explanation for subsequent injury, the direct perception theory.[79] This theory suggested that information from one's environment is directly detected by the individual and acted upon, without the requirement of expanding upon the information internally.[79,133]

Expanding upon previous research, Eagle and colleagues included concussion "clinical profiles" into their model whereby the characteristics of these profiles, and their associated symptoms may lead to dysregulation of the perception-action coupling process.[79,134] Specifically, the concussion may cause symptoms and/or impairments that force the injured individual to recalibrate their altered perceptual system in order to accurately control movement. The authors contend that without sufficient recovery time, this dysregulated perception-action coupling loop may lead to an increased risk of musculoskeletal injury post-concussion. [79] However, unlike the other theorized mechanisms, it is important to highlight that no study, to date, has reported data that may link perception-action coupling dysfunction with subsequent musculoskeletal injury.

Other Proposed Mechanisms

Although the primary theories behind the increased risk for injury post-concussion lie in neuromuscular control and alterations in perception-action coupling mechanisms, other potential contributing factors may exist. One factor may be preexisting conditions; for instance, those with ADHD tend to have lower neurocognitive baseline performance than those without ADHD[135] which is associated with increased injury risk[136,137], and those with ADHD may be at increased risk for injuries than those without.[138] Additionally, neuromuscular control deficits have been associated with ADHD and injury risk.[139,140]

Moreover, personality traits, which may affect how one participates in sport, may influence one's risk for MSI post-concussion. Traits such as lack of caution or sensation seeking, have been mildly associated with MSI risk.[141–143] However, the literature is scarce and warrants further investigation on personality traits and post-concussion MSI risk.[78] Additionally, data from collegiate student athletes noted that common concussion assessments (i.e., symptoms, SAC, BESS, ImPACT, clinical reaction time, and King-Devick) in addition to demographic factors did not predict subsequent MSI post-concussion.[144] Thus, other tests and research are needed to identify clinical predictors of post-concussion MSI.

Repetitive Head Impacts

Not every impact will result in a diagnosed concussion; these "subconcussive" impacts have been termed repetitive head impacts (RHI)[145] and occur through routine

participation in contact/collision sports (i.e., football, soccer, ice hockey, boxing, rugby, lacrosse, and wrestling).[146] It has been hypothesized that these RHI may have adverse effects on long-term health outcomes.[147] Although biomechanical data have been collected, in primarily American football players, to quantify the magnitude, frequency, and effects of RHI, these data are highly variable and do not necessarily correlate with likelihood of sustaining an mTBI.[148,149] Moreover, single-season data have not shown consistent relationship between RHI suffered and changes in cognitive functioning,[147] postural control (assessed by ST and DT gait),[150] or clinical concussion assessment outcomes.[151] Due to lack of readily available biomechanical data, researchers have recently relied on age of first exposure to quantify RHI.

Age of First Exposure to Repetitive Head Impacts

As a means to quantity lifetime RHI, researchers have utilized age of first exposure (AFE), or the age at which one starts participation in contact/collision sports.[152,153] The past decade has seen a growing number of research studies investigating the relationship between AFE and long-term health outcomes. One of the first papers was by Stamm and colleagues which found that former, clinically symptomatic NFL players who started playing football before age 12 (an age researchers considered to be critical for neurodevelopment—indicated by peak myelination rates[154–156]) performed more poorly on neurocognitive testing than those who started playing at or after the age of 12.[153]

Since then, numerous studies have been conducted highlighting methodological issues in the Stamm 2015 paper such as the biased cohort (i.e., participants in Diagnosing and Evaluating Traumatic Encephalopathy using Clinical Tests (DETECT) – were more likely to have issues and seek help), small sample size

(n=42), arbitrary dichotomizing of groups at age 12 instead of investigating age as a continuous variable, self-reported measures, etc. Furthermore, multiple letters to the editor of *Neurology* were written in response to the Stamm 2015 paper criticizing the article and its methods further.[157–159] These authors highlighted that the study subjects included individuals who participated in the 1960s-1980s which is not representative of the safer playing practices of modern day football and youth football in the United States.[159] Furthermore, socioeconomic differences and parenting styles may confound and influence an individual's response to head impacts. In addition to the self-report nature (e.g., one individual reported almost 400 concussions in their lifetime), researchers critiqued the measures such as the Wide Range Achievement Test, 4th edition (WRAT-4) as an insufficient neuropsychological test.[158,159] Further, one group noted that AFE group differences may have simply been the result of premorbid differences in WRAT-4 ability and not the result of RHI exposure, in addition to the authors failing to account for Learning Disability (LD) and attention deficit hyperactivity disorder (ADHD), a known counfounder.[157] As such, this has led to a growing amount of AFE research in various cohorts such as the CARE Consortium,[160–164] and former high school[165]/collegiate/professional football players[166–174] with outcome measures ranging from physical performance, executive functioning and neurocognitive function, and psychological well-being with many of these findings showing no or no clinically meaningful differences between AFE groups on most if not all outcomes, and a sparse few finding significant group differences.

As a result of the confluence of conflicting research from various research groups, a 2021 narrative review sought to summarize the controversies in AFE

literature as well as identify any potential biases and clarify interpretation of the data. This review included 21 studies: 11 among former athletes, 8 with current athletes, and 2 studies with current and former athletes.[152]

High School Football and Later Life Neurological Health

To date, 8 studies have examined the relationship between high school American football participation and later in life cognitive dysfunction and mental health issues.[173,175–181] Within these studies, among early adults (i.e., 20-30 years old) it has been reported that high school football participation is not associated with elevated lifetime rates of anxiety,[176] depression or current symptoms of depression,[175,176,178] substance abuse,[176] or suicidal ideation within the previous year.[178]

When investigating the relationship between high school football participation and mid-life neurological health, researchers reported no significant association. Specifically, there was no association between middle aged cognitive functioning, self-rated physical health, or mental health.[177,179] Lastly, two other studies reported no increased risk for later-life neurodegenerative disease (i.e., dementia, Parkinson's Disease, amyotrophic lateral sclerosis (ALS)) in those who played high school football via medical-record data of community dwelling individuals.[180,181] Collectively, these data suggest that high school football participation may not be related to early adulthood and later in life cognitive functioning, self-rated function, and mental health.

Professional Football and Later Life Neurological Disease

Neuroimaging data remains highly contentious regarding the clinical meaningfulness of findings; nonetheless, it still provides a valuable research tool for investigations into neurological health. Microstructural differences in white matter pathology and cerebral blood flow (CBF) have been reported in former NFL players when compared to age, education, and IQ-matched controls.[182] Further, macrostructural differences in white matter integrity, assessed by diffusion tensor imaging (DTI) fractional anisotropy (FA), have been identified in former NFL players; these changes were significantly associated with history of RHI, concussion, and depressive symptoms.[183] As a means to account for limitations in AFE as a measure of RHI, researchers quantified a cumulative head impact index (CHII) to estimate RHI in former NFL players. In a sample of symptomatic NFL athletes, there was direct relationship between reductions in cellular energy metabolism and neurochemistry, assessed by magnetic resonance spectroscopy (MRS), and RHI compared to asymptomatic controls with head trauma history.[184] However, the clinical meaningfulness and relevance of these findings remain to be determined.

Aside from neuroimaging, a relationship between neuropsychological testing, lifelong cognitive dysfunction, and RHI has been reported in former NFL players whereby former NFL players perform worse than healthy controls.[182,185,186] In particular, studies have suggested greater prevalence of neurodegenerative disease[187] and cognitive impairment[188] among retired NFL players; one limitation is the unknown quantifiable risk of neurodegenerative disease in these players.[186] In regards to neuropsychological testing, few studies have reported reductions in verbal memory, yet are limited by sample size and lack of long-term data.[153,182,185,186]

In summary, the relationship between RHI exposure and cognitive dysfunction in later life among NFL players remains mixed. However, it has been reported that former NFL players have reduced mortality rates than the general population,[189] which is similar to data from former Scottish soccer players which found reduced all-cause mortality up the age of 70.[190] Interestingly, these soccer players had reduced mortality from common diseases (e.g., heart disease, cancer), but higher mortality from neurodegenerative disease, which may be a result of survival bias and longer lifespan.[190] All in all, AFE may not be an accurate measure of RHI and better methods are needed to accurately quantify exposure such as career duration.[191]

Current Athletes

To date, no research study of current contact/collision sport athletes have revealed a statistically significant relationship between earlier AFE (i.e., AFE<12 years of age) and worse clinical outcomes of concussion or neurological function.[160,161,163–165,192–194] Within these data, a large cross-sectional study among high school football players (N=1,802) revealed no differences in AFE groups on objective clinical outcomes of cognition, psychological health, oculomotor function, postural stability, and behavior, even after controlling for age, race, ADHD, and concussion history.[165] Among current collegiate athletes with data from the CARE Consortium, whether AFE was treated as a binary (AFE<12 or AFE≥12) or continuous variable, there was no relationship between AFE and neuropsychological outcomes of ImPACT in football players,[160,161,163,164,192] non-contact athletes,[160,163] service academy cadets,[163] male/female contact and non-contact athletes, as well as mood (assessed via Brief-Symptom Inventory 18, BSI 18), balance (BESS),[161] and reading ability in football players,[193] and sensorimotor processing in soccer players.[194]

Authors of the review highlight that these studies of 4 independent cohorts included almost 10,000 athletes with 20 outcome measures, yet, there was not a single measure whereby earlier AFE to football or contact/collision sports was associated with meaningful worse neurological functioning.[152] Moreover, the majority of these studies highlighted the methodological flaw of the Stamm paper's[153] failure to account for ADHD/LD history, as other research have found ADHD/LD and academic issues to be associated with worse cognitive functioning and heightened symptoms.[195–198] However, it should be noted that although these data provide a large cohort and wealth of evidence against the relationship between AFE and neurological health, the included measures may not be sensitive enough to detect the subtle effects of RHI. Moreover, these long-term effects of RHI may not have manifested yet as these athletes are young adults. However, it can be suggested that the effects of RHI and AFE on neurological health does not appear to affect current high school and collegiate athletes.[152] Lastly, it appears that AFE may not be the largest contributing factor to long-term neurological health in contact/collision sport athletes. Instead, substantial exposure to RHI ($\geq$5 years of RHI sports or $\geq$5 years of American football with $\geq$2 years at the high school level) and measures of RHI exposure, for example, career duration (or years participated in sport), may provide more insight into the effects on long-term health.[191]

Literature Gap: Career Duration and Sex-Specific Responses to RHI

Specifically to American football, earlier AFE, whether analyzed dichotomously or continuously, to contact football does not appear to be related to worse subjective anxiety, depression, or cognitive functioning in retired NFL players.[199] Large scale studies of current high school and collegiate collision sport

athletes have failed to show a significant or clinically meaningful relationship between AFE and various outcomes (e.g., postural stability, neurocognitive functioning, symptoms).[160,161,163–165] Yet, small scale convenience sample data of former NFL players (i.e., a cohort with extreme RHI exposure) utilizing more sophisticated measures of neuroimaging and other clinical outcomes have reported a negative relationship between AFE and these outcomes.[153,166,169,200] Thus, there may not be a relationship between RHI suffered through routine contact sport participation, but there may be an effect of RHI when experienced at extreme exposure levels. However, future research including age groups not explored (i.e., 30-50 years old) across sexes and the sport spectrum (i.e., not American football) using more objective assessments are needed.

A 2021 National Institute of Neurological Disorders and Stroke Consensus for diagnostic criteria for traumatic encephalopathy syndrome (TES), the clinical condition associated with post-mortem diagnosed Chronic Traumatic Encephalopathy (CTE), noted that the majority of RHI research was limited to American football; as such, it is impossible to define specific exposure thresholds for other contact/collision sports and for women.[191] Further, experts suggest that career duration may be a more impactful measure of RHI exposure than AFE.[191] Thus, research on AFE and RHI needs to be expanded to cohorts that include women collision sport athletes, across the age spectrum, and in particular sex- and rule-matched sports might provide further insight into the relationship between RHI and neurophysiological functioning.

Chapter 2

RELATIONSHIP BETWEEN ADULT RUGBY PARTICIAPTION AND PATIENT-REPORT OUTCOMES AND INJURY HISTORY IN MEN AND WOMEN COMMUNITY RUGBY PLAYERS

(Hunzinger, K. J., Costantini, K. M., Swanik, C. B., & Buckley, T. A. (2020). Diagnosed Concussion is Associated with Increased Risk for Lower Extremity Injury in Community Rugby Players. *Journal of Science and Medicine in Sport*, *24*(4), 368–372. https://doi.org/10.1016/j.jsams.2020.10.013 *Updated with recent literature)[127]

(Hunzinger, K. J., Caccese, J. B., Costantini, K. M., Swanik, C. B., & Buckley, T. A. (2021). Age of First Exposure to Collision Sports Does Not Affect Patient Reported Outcomes in Women and Men Community Rugby Players. *Medicine & Science in Sports & Exercise*, *53*(9), 1895–1902. https://doi.org/10.1249/MSS.0000000000002657 *Updated with recent literature)[201]

Introduction

Rugby union is the most participated collision sport with over 8 million players worldwide; furthermore, it is the only collision sport in which men and women play by the same rules/laws.[202,203] As worldwide popularity increases, leading to large monetary sums at stake for winning teams, injury risk and prevention strategies have become of particular interest for World Rugby and participating teams.[204] Specifically, male professional rugby union players suffer 92 injuries per 1,000 players hours during matches with concussions representing ~20% of these injuries, yielding a mean

severity of 19 days of time loss to injury (1-2 fixtures).[205] With an ever growing concern about concussion in rugby, many programs (e.g., Activate) have been implemented, yielding limited reductions in overall injuries, but showed potential for concussion and lower limb injury incidence reduction.[206] However, the long-term sequalae of concussion beyond lost playing time may still be burdensome on rugby players such as reduced quality of life, impairments in cognitive function,[122] and subsequent injury risk.[207]

In addition to concussions, the collision aspect of rugby has led to a high incidence of musculoskeletal injuries (MSI), with 80% of match injuries being associated with contact events.[208] Particularly, muscle/tendon (40/1,000 player hours) and joint/ligament injuries (34/1,000 player hours) are the most common time loss injuries among professional rugby union players.[209] Furthermore, among U.S. collegiate rugby players MSI are also the most prevalent time loss injury (15 days)[209] at an overall injury rate of 15.2 per 1,000 AE, a rate 3x higher than American football, with the lower extremity being the most commonly injured body region.[210] Moreover, joint sprains and muscles strains have the highest incidences of injury in amateur male rugby players.[211] In addition to time loss to injury, rugby players report continued impact (e.g., surgery, pain, numbness) from previous MSI, leading to recurrent injuries[212] as well as poorer long term health outcomes, with retired players reporting a greater prevalence of osteoarthritis, joint replacements, and osteoporosis than the general population.[213] As such, documenting and prevention of MSIs are imperative as retired amateur rugby players are prone to suffer from osteoarthritis, one of the leading causes of disability worldwide, creating a substantial socioeconomic burden.[212,214]

Both concussions and MSI are important considerations independently; however, emerging evidence suggests a potential linkage between the two conditions.[77] Indeed, male professional rugby players with a concussion were 60% more likely to sustain a time loss (e.g., MSI or concussion) injury than those who did not have a concussion; however the relationship in community rugby players is unknown.[207] This ~2x elevated risk of subsequent lower extremity musculoskeletal injury (LE-MSI) post-concussion has been reported in high school athletes[124], collegiate athletes[215], military cadets[126], and professional athletes[207]; however the relationship in community rugby, with over 30,000 American participants, is unknown.[216] Furthermore, these studies are primarily focused on elite (e.g., Division I or professional) athletes and have been comprised of largely male cohorts. Indeed, the effect of sex on concussion and subsequent injury remains inconclusive with only one large cohort highlighting a greater risk in females, warranting further investigation.[215] Additionally, with women's rugby growing at a faster rate than men's rugby, it is imperative to identify any sex-differences in injury risk.[203,216]

With an established risk of post-concussion LE-MSI found in professional rugby players[207] and the high burden of MSI in amateur players[211] a need exists to study this relationship among community rugby players. Furthermore, with women's rugby growing internationally at a faster rate than men's rugby and the similar nature of men's and women's rugby injury risks,[203,216] a need exists to determine the effect of sex on injury risk in community rugby players.

In addition to concussion, the effect of routine exposure to repetitive head impacts (RHI) on neurophysiological functioning has become another area of research. Routine exposure to repetitive head impacts (RHI) through contact/collision sports has

been associated with both short- and long-term molecular, structural, and functional changes in the brain.[182,200,217,218] Younger age of first exposure (AFE) to RHI, particularly before age 12, may negatively affect neurodevelopment thereby increasing vulnerability to adverse long-term neurological outcomes.[154–156] Thus, numerous studies have examined the effects of AFE to RHI on later-in-life brain health.[153,160,161,163–165,167,169–171,194,199] Initial studies on small cohorts of former amateur and professional American football players suggested worse cognition and greater behavior/mood impairments with younger AFE [153,169]; however, a growing body of literature in current high school and collegiate contact/collision sport athletes [160,161,163–165,194], and in other cohorts of former professional American football players[167,169–171,174,199] suggests no association between AFE and later-in-life brain health problems. Studies on the long-term effects of AFE to RHI in older cohorts have been limited in sport and sex to former male American football players[153,166,167,169,170,199,200]; therefore, a critical gap in knowledge remains regarding the long-term effects of younger exposure to other contact/collision sports, such as rugby, as well as the effects on females.

The sport of rugby offers a unique insight into the relationship between AFE and RHI on brain health as many rugby players in the United States continue playing for adult club teams beyond college (evidenced by similar registered player numbers between college and adult club teams), exposing them to RHI further into adulthood.[216] Although many rugby players start playing rugby in college, they commonly report adolescent participation in other contact/collision sports.[127,219] Furthermore, rugby is the only collision sport whereby men and women play by the same laws, and the variable aspects of the game permit a diverse player population

with varying levels of skills and body types.[220] Therefore analysis of rugby players will extend previous findings by examining early adulthood outcomes in current and former women's and men's rugby players – providing critical data in women, who have different neurodevelopmental trajectories than men.[154–156] Finally, it will provide insight on athletes who continue to experience RHI outside of football, but beyond high school and college.[154–156]

Although rugby participation may offer regular exercise and its associated positive physical and mental health benefits, the collision aspect of the sport is linked to high musculoskeletal injury and concussion rates.[221,222] For example, routine rugby participation has been linked with microstructural changes in the brain in female rugby players, which may be linked to later in life dysfunction.[223] As a result of prolonged RHI exposure via collision sport participation, researchers posit that the higher prevalence of anxiety and depression in rugby players compared to the general population[224] and reduced QoL[213,221] may be the result of prolonged effects of concussion and cognitive abnormalities.[212,225]

The relationship between prolonged RHI and patient-reported outcomes among community level male and female rugby players remains to be elucidated. As such, with the continued growth of women's sports it is imperative to enhance our understanding of the effects of RHI in male and female collision sport athletes. Further, analysis of AFE alone may not be an appropriate measure of lifetime RHI as suggested by the 2021 National Institute of Neurological Disorders and Stroke (NINDS) consensus on diagnostic criteria for traumatic encephalopathy syndrome (TES).[191] Thus, a need exists to determine the effects of prolonged RHI exposure as measured by career duration in adult collision sport athletes across sex.

Specific Aim 1 Description

To determine the associations between adult rugby participation and injury history and patient-reported outcomes in males and females.

H.1.1: Rugby players with a concussion history would be more likely to have an LE-MSI than those without a history of concussion; females would have a greater risk of LE-MSI given history of concussion than males.

H1.2: Career duration and AFE will not predict worse self-reported psychological well-being or QoL.

Methods

Participants

Current and former rugby players aged 18 years or older with at least one year of contact rugby playing experience were recruited for this study via rugby specific forums on social media (e.g., Reddit, Facebook). This recruitment method in addition to the data collection modality (i.e., online), which have been previously utilized in health-related research, permitted mass recruitment.[164,165,226] Overall, 1,376 individuals responded to recruitment efforts, but 322 (23.4%) were excluded for failure to complete the entire online questionnaire. Of the remaining 1,054 participants, 17 (1.6%) were removed for failing to meet inclusion criteria: 18 years or older (n=9) and minimum one year of full-contact (i.e., tackle) rugby playing history (n=8). Thus, 1,037 (age: 31.6 $\pm$ 11.3 years [range: 18-74; 95%CI: 31.0, 32.2], 59.0% men, 10.1 $\pm$ 8.1 years played) respondents were included in analyses. Relevant injury demographics for Aim 1.1 by sex are presented below. (Table 1) Further, AFE and

relevant demographics are also presented below by sex and AFE group (AFE<12 and AFE≥12). (Table 2) As approved by the University of Delaware's Institutional Review Board (1540689-3), prior to starting the survey, all participants completed an informed consent via the online survey platform (Qualtrics, Provo, UT, USA). (Appendix M)

Table 1 Participant Injury Demographics for Aim 1.1.

	Participants		
	Male (N = 612)	**Female (N = 425)**	**Total (N = 1,037)**
Age (years)	33.6 ± 12.8 [95%CI: 32.6, 34.7]	28.7 ± 7.8 [95%CI: 28.0, 29.5]	31.6 ± 11.3 [95% CI: 31.0, 32.3]
Rugby Playing History (years)	12.2 ± 9.0 [95%CI: 11.5, 12.9]	7.1 ± 5.2 [95%CI: 6.6, 7.6]	10.0 ± 8.1 [95%CI: 9.6, 10.6]
Diagnosed Concussion (n,%)	412 (67.3%)	278 (65.4%)	690 (66.5%)
Any LE-MSI	562 (91.8%)	396 (93.2%)	958 (92.4%)
Ankle Sprain (n,%)	475 (77.6%)	321 (75.5%)	796 (76.8%)
Multiple Ankle Sprains (n,%)	261 (42.6%)	211 (49.6%)	472 (45.5%)
Knee Injury (n,%)	269 (44.0%)	188 (44.2%)	457 (44.1%)
Broken LE Bone (n,%)	155 (25.3%)	112 (26.4%)	267 (25.7%)
LE Muscle Strain (n,%)	314 (51.3%)	208 (48.9%)	522 (50.3%)
ACL Injury (n,%)	30 (4.9%)	15 (3.5%)	45 (4.3%)

Table 2 Participant AFE Demographics for Aim 1.2

Sex	**Females (N=424)**				**Males (N=610)**			
	Overall	**AFE<12**	**AFE≥12**	**P value**	**Overall**	**AFE<12**	**AFE≥12**	**P value**
Total	424	281	143	N/A	610	472	138	N/A
Years Rugby Played	7.1 ± 5.2	7.1 ± 5.0	7.2 ± 5.6	0.882	12.2 ± 9.0	12.4 ± 8.7	11.5 ± 10.0	0.027*
Age	28.7 ± 7.9 (range: 18-61)	28.4 ± 7.89	29.4 ± 8.0	0.163	33.7 ± 12.8 (range: 18-74)	32.8 ± 11.9	36.6 ± 15.0	0.018*
AFE to Contact/Collision sports	10.0 ± 5.6	6.7 ± 2.4	16.5 ± 4.3	<0.001*	8.7 ± 4.2	6.9 ± 2.2	15.0 ± 3.4	<0.001*
Cumulative Years of Contact/Collision Sports	12.9 ± 8.8 (range: 1-56)	13.4 ± 9.1	11.8 ± 8.2	0.082	19.4 ± 12.2 (range: 1-79)	20.8± 11.7	14.7 ± 12.9	<0.001*
Diagnosed History of Concussion (%)	34.4%	32.0%	39.2%	0.11	32.5%	29.7%	42.0%	0.006 *

Data presented as mean + standard deviation. * denotes significant difference between AFE<12 and AFE>12.

Instrumentation and Procedures

For Aim 1.1, participants completed an 85-item online questionnaire to ascertain injury history. Specifically, the questionnaire consisted of 30 demographic questions and 55 questions from the reliable Gilbert injury history questionnaire[227,228] to identify self-reported history of diagnosed concussions (i.e., by a health professional) and LE-MSI (e.g., muscle strains, ligament sprains) history. The Gilbert injury history questionnaire (Appendix A) was initially designed to determine concussion reporting rates, but also included injury history questions for common musculoskeletal injuries and has been used to investigate concussions and LE-MSI previously.[126,227,228] The test-retest reliability of this questionnaire was excellent (ICC = 0.92) among collegiate student athletes tested 4 months apart.[227,228] Participants were asked to report lifetime incidences of concussions (no definition was provided) and various LE-MSI; while dates of injury were collected, an order effect between concussion and LE-MSI was not determined due to incomplete (e.g., missing) or inconsistent reporting (e.g., "in high school", "summer 2017 or 2018").

For Aim 1.2, in addition to the previously completed concussion history (yes/no) and demographics, participants self-reported AFE to various contact and collision sports (Appendix B). For concussion history, participants were not provided a definition of concussion, and simply asked "Have you ever suffered a concussion?" For AFE, participants were asked: "At what age did you START playing the following sport(s)?" and "How many YEARS (not seasons) did you play the following

sport(s)?" to determine sport history. Contact/collisions sports with routine RHI exposure included: boxing, American football, ice hockey, lacrosse, rugby, soccer, and wrestling.[146] Total years of contact/collision sport history was calculated as the sum of years an individual played each/any of the aforementioned contact/collision sports, regardless of overlapping seasons. Additionally, participants completed 3 commonly utilized patient-reported outcome measures: Brief-Symptoms Inventory 18 (BSI-18), Short Form Health Survey 12 (SF-12), and Satisfaction with Life Scale (SWLS). The first recruitment effort occurred on March 9, 2020, and yielded 764 valid responses, the second effort was on March 27, 2020, and yielded an addition 86 valid responses, and the final effort was on April 7, 2020, providing an additional 187 valid responses.

Brief-Symptom Inventory 18 (BSI-18)

The BSI-18 is a self-report tool comprised of 18 questions to assess current (last 7 days) psychological distress in adults older than 18. Respondents rate their distress using a 5-point Likert scale, whereby 0 is none at all and 4 is extremely often.[229] It is both reliable (ICC: 0.91) and valid among individuals with brain injury[229] and an American norm sample.[230] The BSI-18 has 3 clinical sub-scores comprised of 6 questions each: somatization, depression, and anxiety. These subscales can range from 0 (none/best) to 24 (worst). Additionally, a composite score is created, the global severity index (GSI), by summing the three sub-scores; thus, a respondent's overall score can range from 0-72 with a lower score again reflecting better outcomes.[229,230]

Short Form Health Survey 12

The SF-12 was developed as a shortened version of the Medical Outcomes Study 36-item Short-Form Health Survey SF-36 and is used to assess self-reported health-related QoL.[231] It is both reliable (test re-test reliability = 0.864) and valid in multiple populations.[231] The SF-12 contains 12 questions that measure eight health domains used to asses physical (general health, physical functioning, role physical, and body pain) and mental health (vitality, social functioning, role emotional, and mental health). The SF-12 utilizes a scoring algorithm from the general population to score the 12 questions into two components: Physical Component Summary (PCS) and Mental Component Summary (MCS). Scores can range from 0-100, whereby a lower score represents worse physical and mental QoL, and a score of 50 is representative of the average American population.[231]

Satisfaction with Life Scale (SWLS)

The SWLS assessed self-reported satisfaction with one's life as whole.[232] The SWLS has shown good convergent validity with other scales assessing well-being, shows temporal stability (0.54 for 4 years), strong internal reliability (α=0.87), and is sensitive to detect change in one's satisfaction with life during clinical intervention.[232] The SWLS consists of five statements related to one's self-reported satisfaction with their life; respondents indicate their level of agreement with each statement using a 7-point Likert scale ranging from 1 (strongly disagree) to 7 (strongly agree). The questions are summed, so a total score can range from 5 (lower levels of satisfaction) to 35 (highest level of satisfaction with one's life).[232]

Statistical Analysis

For Aim 1.1, consistent with previous studies[126,228], one dichotomous independent variable was created, self-reported diagnosed concussion history (yes/no), along with seven dichotomous (yes/no) dependent variables: 1) ankle sprain, 2) multiple ankle sprains, 3) knee injury, 4) fractured LE bone, 5) LE muscle strain, 6) ACL injury, and 7) Any LE-MSI. Data were exported via Excel (Microsoft Excel, Redmond, WA) and coded using a custom script (MATLAB 2017; MathWorks, Inc, Natick, MA).

Variance inflation factor (VIF) was used to check the data for multicollinearity, or high correlation between variables prior to analysis (VIF<5 was acceptable);[233] multicollinearity did not exist (VIF: 1.04-1.70) among the various dependent variables. Chi-square analyses were performed to identify the association between diagnosed concussion and each of the 7 LE-MSI variables. An odds ratio with 95% confidence interval was calculated to determine the risk of each LE-MSI among rugby players given a history of diagnosed concussion. Odds ratios were also calculated for risk of LE-MSI given history of concussion among males and females individually. A binary logistic regression with any LE-MSI (yes/no) as the outcome and concussion (yes/no) and sex (male/female) as predictors was performed to determine if there was a sex by concussion interaction (i.e., differences in risk of LE-MSI given history of concussion between males and females).

For Aim 1.2 a generalized linear model (GLM) was used to examine the association between AFE as a continuous variable and patient-reported outcome

measures by sex. Predictors in the models included AFE (continuous), age (years), cumulative years of contact/collision sports history (years) (i.e., sum of years played of each reported contact/collision sport), and concussion history (yes/no) due to significant differences on these measures between AFE groups. Models were fit based on the error distribution of the response variables. The best fitting model was determined as the one with the lowest Akaike information criterion (AIC) value.[110] A negative binomial distribution with a log link best fit BSI-18 Somatization, Depression, Anxiety sub-scores, and GSI. A Poisson loglinear model best fit SWLS. A Gaussian distribution best fit SF-12 PCS and MCS.

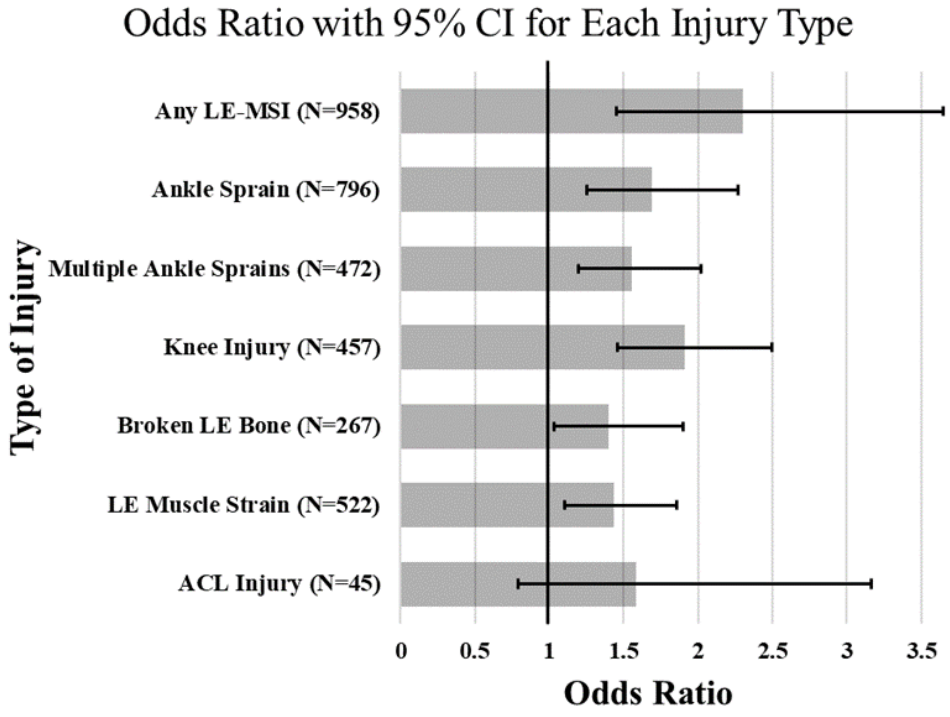
Because AFE differed between men (N=610) and women (N=424) ($p<0.05$), and because neurodevelopmental timelines differ by sex, sex-specific analyses were used. Participants were also dichotomized into two groups based upon their self-reported AFE to contact/collision sports (<12 or ≥ 12); AFE to rugby was not used since most participated in other collision sports prior to rugby. (Table 2) Male and female data were independently tested for normality using a Kolmogorov-Smirnov test and were not normally distributed ($p < 0.05$), so a Mann-Whitney U test was performed to compare outcomes between AFE groups. AFE, BSI-18/SWLS, and SF-12 data were missing or incomplete for 4, 21, and 34 participants respectively. Significance was set at $p<0.05$. All analyses were performed with SPSS v. 26 (SPSS Inc, Chicago, IL).

Results

Concussion & LE-MSI

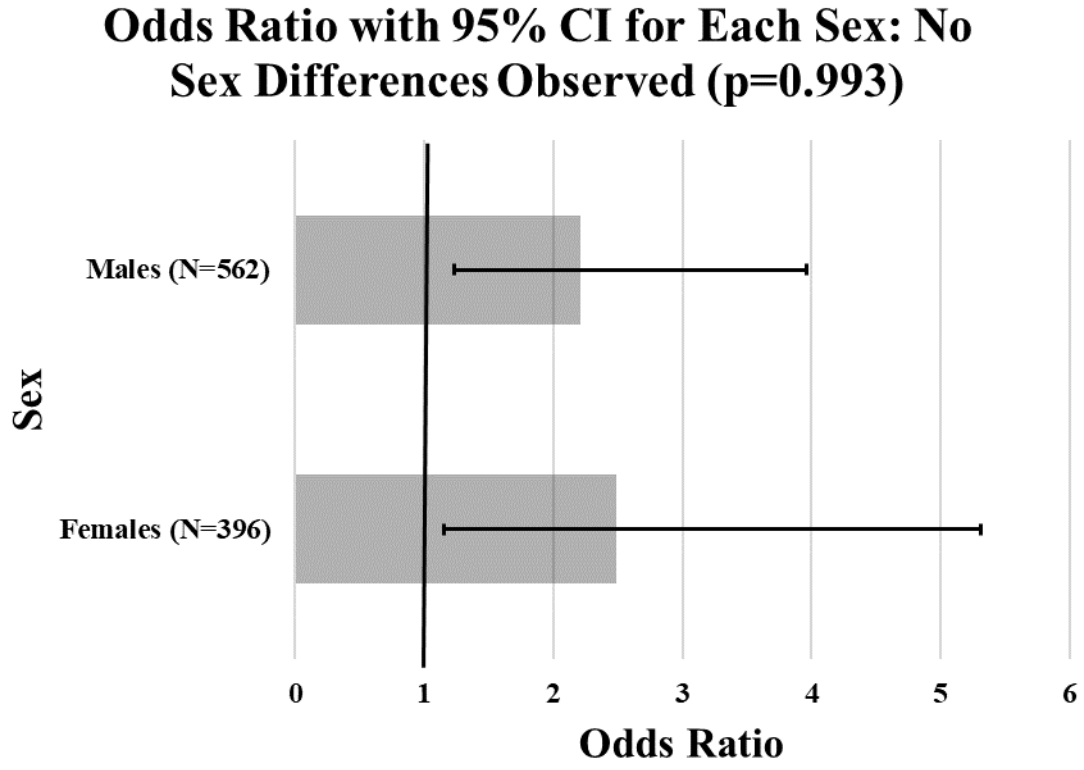
There was a significant association between diagnosed concussion and any LE-MSI ($p<0.001$). Rugby players with a history of concussion were 2.3x more likely (OR: 2.29 [95%CI: 1.45,3.65]) to experience any LE-MSI than those without a history of concussion. For specific LE-MSI, there was a significant association between diagnosed concussion and all LE-MSI outcomes except ACL injury ($p= 0.190$). (Figure 1)

Figure 1 Odds Ratios for Concussions and LE-MSI Risk. All LE-MSI were significant (*) except for ACL injury.



There was a significant association between diagnosed concussion and any LE-MSI for both males (p= 0.006) and females (p=0.016). Males with a history of concussion were 2.2x more likely (OR: 2.21 [95%CI: 1.24,3.96]) and females with a history of concussion were 2.5x more likely (OR: 2.48 [95%CI: 1.16,5.31]) to experience any LE-MSI compared to those without a history of concussion. There was no difference in the odds of LE-MSI given history of concussion between males and females (p = 0.993, R^2 = 0.024). (Figure 2)

Figure 2 Sex-Specific Odds Ratios for Concussion and LE-MSI Risk. There was no significant difference between sexes for post-concussion LEMSI risk (p=0.993)



AFE & Patient Reported Outcomes

Women – AFE

Findings from the generalized linear models suggested that AFE to contact/collision sports was not a significant predictor for any of the patient-reported outcomes among women. (Table 3) Furthermore, there were no significant differences between AFE <12 and AFE ≥12 groups for any of the patient-reported outcomes among women. (Table 4) (Appendix C)

Women – Co-variates

Cumulative years of contact/collision sport history was not a significant predictor for any patient-reported outcomes among women. (Table 3) Concussion history was a significant predictor for all patient-reported outcomes; those with a history of concussion reported worse outcomes than those without a history of concussion. (Table 3) Women with a history of concussion had higher (worse) BSI-18 Somatization (79.9%), Depression (34.6%), Anxiety (52.2%) sub-scores, and higher (worse) GSI (50.2%) than those without a history of concussion. Women with a history of concussion also had lower (worse) SWLS (-4.9%), SF-12 PCS (-1.96), and SF-12 MCS (-2.89) scores than those without a history of concussion. Age was a significant predictor for BSI-18 Depression and Anxiety sub-scores, BSI-18 GSI, and SF-12 PCS and MCS scores, whereby psychological health outcomes improved with age and physical health outcomes worsened with age. For every year increase in age, there was an associated reduction (improvement) in BSI-18 Depression (-1.6%) and Anxiety (-2.4%) sub-scores, a reduction (improvement) in BSI-18 GSI (-1.7%), and

increase (improvement) in SF-12 MCS (0.28), but a reduction (worsening) in SF-12 PCS (-0.16). (Table 3)

Men - AFE

Findings from the generalized linear models suggested that AFE to contact/collision sports was not a significant predictor for any of the patient-reported outcomes among men. (Table 3) Furthermore, there were no significant differences between AFE <12 and AFE ≥12 groups for any of the patient-reported outcomes among men. (Table 4)

Men – Co-variates

Cumulative years of contact/collision sport history was only a significant predictor for BSI-18 Depression and SF-12 MCS, whereby each year increase in cumulative years of contact/collision sport history was associated with a reduction (improvement) in BSI-18 Depression (-1.1%) and an increase (improvement) in SF-12 MCS (0.11). (Table 3) Concussion history was the only significant predictor for all BSI-18 outcomes, whereby those with a history of concussion reported higher (worse) BSI-18 Somatization (+53.1%), Depression (+27.8%), Anxiety (+44.9%) sub-scores, and GSI (+39.0%) than those without a history of concussion. (Table 3) Age was a significant predictor for all patient-reported outcomes, except for BSI-18 Somatization, whereby psychological health outcomes improved with age and physical health outcomes worsened with age. For every year increase in age, there was an associated reduction (improvement) in BSI-18 Depression (-2.3%) and Anxiety (-2.4%) sub-scores, a reduction (improvement) in BSI-18 GSI (-1.9%), increase

(improvement) in SWLS (+0.002 score), and increase (improvement) in SF-12 MCS (+0.15 score), but a reduction (worsening) in SF-12 PCS (-0.15 score). (Table 3)

Table 3 Generalized Linear Model Results by Sex.

Sex	Dependent Variable	Concussion History (Yes)	AFE (years)	Age (years)	Cumulative Years Contact/Collision Sport History (years)
Women	**BSI-18 Somatization (Exp(B))**	1.80 (1.40, 2.31)**	1.01 (0.99, 1.03)	0.99 (0.98, 1.00)	1.00 (0.98, 1.01)
	BSI-18 Depression (Exp(B))	1.35 (1.07, 1.69)*	1.01 (0.99, 1.03)	0.98 (0.97, 0.997)*	1.00 (0.99, 10.01)
	BSI-18 Anxiety (Exp(B))	1.52 (1.20, 1.93)**	1.00 (0.982, 1.02)	0.98 (0.96, 0.99)**	0.99 (0.99, 1.01)
	BSI-18 GSI (Exp(B))	1.50 (1.21, 1.87)**	1.01 (0.99, 1.02)	0.98 (0.97, 0.99)**	1.000 (0.99, 1.01)
	SWLS (Exp(B))	0.95 (0.91, 0.99)*	1.00 (0.99, 1.00)	1.00 (1.00, 1.01)	0.99 (0.99, 1.00)
	SF-12 PCS (B)	-1.96 (-3.47, -0.44)*	-0.01 (-0.14, 0.12)	-0.16 (-0.25, -0.07)**	0.06 (-0.02, 0.14)
	SF-12 MCS (B)	-2.89 (-5.26, -0.53)*	0.02 (-0.19, 0.22)	0.28 (0.14, 0.42)**	0.03 (-0.10, 0.15)
Men	**BSI-18 Somatization (Exp(B))**	1.53 (1.23, 1.91)**	1.00 (0.98, 1.03)	0.99 (0.98, 1.00)	1.00 (0.99, 1.01)

	BSI-18 Depression (Exp(B))	1.28 (1.04, 1.57)*	0.99 (0.97, 1.01)	0.98 (0.97, 0.99)**	0.99 (0.98, 1.00)*
	BSI-18 Anxiety (Exp(B))	1.45 (1.30, 1.61)**	0.99 (0.98, 1.01)	0.98 (0.97, 0.98)**	1.00 (0.99, 1.01)
	BSI-18 GSI (Exp(B))	1.39 (1.15, 1.68)**	0.99 (0.97, 1.02)	0.98 (0.97, 0.99)**	0.99 (0.99, 1.00)
	SWLS (Exp(B))	0.98 (0.95, 1.02)	1.00 (0.99, 1.00)	1.00 (1.00, 1.00)*	1.00 (1.00, 1.00)
	SF-12 PCS (B)	-1.08 (-2.27, 0.11)	-0.08 (-0.22, 0.07)	-0.15 (-0.20, -0.10)**	-0.03 (-0.08, 0.03)
	SF-12 MCS (B)	-1.42 (-3.28, 0.44)	0.11 (-0.12, 0.34)	0.15 (0.07, 0.23)**	0.11 (0.02, 0.20)*

GLM results are presented as Exp(B) and B values and their 95% Wald Confidence Intervals where appropriate. *: $p<0.05$ and **: $p<0.007$.

Table 4 AFE and Quality of Life Outcomes by Sex and AFE Group.

Sex		N	Mann-Whitney U value	P-value	Overall Mean ± SD	AFE <12	AFE≥12	η^2
Women	**BSI-18 Somatization**	408	16,985	0.190	2.67 ± 3.19	2.57 ± 3.17	2.87 ± 3.25	0.004
	BSI-18 Depression	408	18,327	0.927	4.55 ± 4.92	4.48 ± 4.80	4.67 ± 5.18	<0.001
	BSI-18 Anxiety	408	18,141	0.796	3.81 ± 3.82	3.81 ± 3.74	3.82 ± 3.98	<0.001
	BSI-18 GSI	408	18,013	0.711	11.02 ± 10.10	10.86 ± 9.89	11.36 ± 10.58	<0.001
	SWLS	408	18,251	0.874	24.41 ± 6.33	24.46 ± 6.12	24.29 ± 6.78	<0.001
	SF-12 PCS	403	17,037	0.370	52.38 ± 7.45	52.49 ± 7.56	52.13 ± 7.27	0.002
	SF-12 MCS	403	17,373	0.555	43.36 ± 11.64	43.21 ± 11.36	43.64 ± 12.23	<0.001
Men	**BSI-18 Somatization**	611	31,635	0.691	2.11 ± 2.83	2.15 ± 2.89	1.99 ± 2.62	<0.001

	BSI-18 Depression	611	32,119	0.904	3.96 ± 4.90	3.97 ± 4.93	3.96 ± 4.85	<0.001
	BSI-18 Anxiety	611	29,694	0.138	2.99 ± 3.67	3.09 ± 3.77	2.66 ± 3.33	0.002
	BSI-18 GSI	611	31,323	0.576	9.06 ± 9.71	9.21 ± 9.96	8.60 ± 8.84	<0.001
	SWLS	611	31,646	0.704	25.15 ± 6.28	25.23 ± 6.14	24.81 ± 6.75	<0.001
	SF-12 PCS	603	30,406	0.495	52.42 ± 7.11	52.62 ± 6.95	51.71 ± 7.66	<0.001
	SF-12 MCS	603	28,940	0.132	48.09 ± 10.93	47.87 ± 10.76	48.89 ± 11.44	0.004

There were no significant differences between AFE groups for all outcomes among Females and Males.

Discussion

Concussion & LE-MSI

The purpose of this study was to assess the association between diagnosed concussion and LE-MSI among a novel cohort of community playing rugby players, which comprise the majority of rugby union players worldwide.[203] The primary finding from this study suggests these individuals have a ~2.2x increased odds of experiencing any LE-MSI given a history of diagnosed concussion, albeit these results must be viewed in consideration of the lack of temporal association. Furthermore, this relationship was identified across numerous LE-MSI, except ACL injuries, suggesting that no one injury is driving this relationship among community rugby players. However, the binary logistic regression revealed there were no sex differences in Odds Ratios related to concussion and LE-MSI risk between males and females (p=0.993). (Figure 2) The increased odds of LE-MSI given a history of concussion and these injuries' respective incidence rates among this cohort, highlight the need for the utilization of established injury prevention programs through World Rugby.

The results of this study failed to identify any sex differences in LE-MSI risk, however there was a ~2.2x elevated risk of LE-MSI in the entire cohort of community rugby players with a history concussion. Houston et al.[215] had reported a 1.9 – 2.5x elevated risk of MSI post-concussion in female collegiate athletes, but found that male athletes did not experience greater odds of post-concussion MSI. Herein, the lack of

sex differences is interesting as the study design obtained injury history in a similar manner as Houston et al. via self-reported lifetime injury history with injuries dichotomized (yes/no).[215] These differences in findings may be explained by the study populations whereby Houston et al.[215] included a variety of collision, contact, and non-contact sport athletes, with less than 10% of participants playing collision sports and presumably females were less involved in collision sports, whereas this study contained athletes with rugby as their current primary sport. As such, the overall risk and collision aspect of rugby, in which men and women play by the same laws[203], may be related to the increased risk of LE-MSI in both sexes, however, causality between concussion and subsequent LE-MSI between sexes cannot be inferred from this study. Additionally, despite playing rugby an average of 5 years less than males, females and males in this study exhibited similar concussion rates, unlike the Houston cohort whereby females had a higher rate of concussion.[215] However, the self-reported nature of the concussions and LE-MSI herein and the dichotomizing (yes/no) could influence these outcomes. Therefore, since the majority of studies investigating concussion and LE-MSI have involved primarily male athletes from various sports, have not stratified by sex or were limited in sample size[77,124,207,228,234], future studies should prospectively investigate sex differences as part of study design and analyses to further explain the relationship between concussion and LE-MSI risk by sex.

These findings generally support and expand previous work[77] which reported a 1.4-2.3x elevated overall risk of LE-MSI in those with a concussion by assessing a slightly older cohort (mean age: 31.6 years) of community collision sport athletes as

compared to high school or collegiate student-athletes. Furthermore, there were similar elevated rates for specific LE-MSI (e.g., ankle sprains, knee injury)[228] with the notable exception of ACL injuries. (Figure 1) This study was not designed to investigate the specific mechanisms for this increased risk of LE-MSI, but several theories have been proposed. Specifically, it has been suggested that alterations in perception-action coupling[79], postural control[77], neuromuscular control and dual-task dysfunction[78,99], and gait[132,235] may underlie this increased risk of post-concussion subsequent LE-MSI. These theories are based upon evidence of persistent neurophysiological dysfunction post-concussion beyond clinical recovery and highlight the need for delayed return to sport.[77,78] Therefore, future longitudinal research is warranted to assess these potential mechanisms underlying elevated injury risk to implement targeted and established injury prevention programs.

However, this study did not find a significant relationship between concussion and ACL injury, contrary to the findings of McPherson et al. which found an increased odds (OR: 1.6 [95%CI: 1.1-2.4] for ACL injury post-concussion among a general population cohort.[128] McPherson et al. suggest that neurocognitive and executive functioning skill reduction may be associated with neuromuscular control dysfunction, increasing one's risk of an ACL injury.[128,136] However, the lack of significance ($p = 0.190$) herein for concussion and ACL injury may be the result of an underpowered analysis ($\eta^2 = 0.002$, observed power = 0.258) and skewed sample whereby only 45 (4.5%) individuals had an ACL injury. Additionally, our cohort is comprised of athletic individuals, specifically who participated in the high injury risk sport of rugby,

as compared to McPherson's general population cohort which may also impact the relationship between concussion and ACL injury risk. The lack of ACL injury incidence herein is less than the findings of Hind et al., whereby 13% of retired amateur rugby players had surgery for an ACL tear; interestingly, Hind's cohort cited ACL tears and concussions as the most common injury to affect them in retirement.[212] Therefore, future research should continue to assess concussion and specific LE-MSI risk such as ACL injury to further elucidate this relationship in other cohorts as neuromuscular deficits are a commonly recognized risk factor for ACL injuries particularly in females[136]

This study was limited by the utilization of a retrospective design which did not allow the research team to determine a causative or directional relationship between concussion and LE-MSI; however, this approach is consistent with several prior studies.[215,228] A strength of this approach was that it did allow for the recruitment of a large, novel cohort of adult community rugby players which exceeds many prior studies. Regarding participant responses, we have no true way to measure response rate, and could only measure the number of completed and valid responses out of how many people entered the survey and consented (75.4%); as such, there may be potential respondent bias. However, the 322 participants that did not complete the survey were similar in age to the analyzed sample herein, albeit contained slightly more males than females (67.7% male, age: 30.3 $\pm$ 11.3 years). Furthermore, the self-reported injury history of these athletes is limited by memory recall, accuracy of the information reported, and honesty as females are less likely to underreport injuries

than males.[227,236] Additionally, participants were not provided a definition for concussion, so reporting of diagnosed concussion may be limited by knowledge of concussion and/or self-diagnosis. Also, we did not have participants clarify whether their LE-MSI was a contact or non-contact injury which may have provided further insight related to underlying motor control deficits. Finally, while the participants herein were older (mean age: 31.6 years old) than the prior studies of high school and college athletes, it is unknown if or how this relationship will change as the participants age and continue to compete in rugby; it should be noted that these findings are only generalizable to community rugby players, so future research should be performed to expand these findings to other sporting groups.

In summary, there was a ~2.2x elevated risk of LE-MSI amongst community rugby players who had also had a history of diagnosed concussion. However, there were no differences for injury risk between male and female rugby players, athletes participating in the only collision sport in which the rules are same for both sexes.[203] Future research should investigate potential neurophysiological mechanism(s) responsible for the increased incidence of LE-MSI following concussion. Additionally, teams and clubs should aim to develop strategies to support long-term health of amateur rugby players, such as required sports medicine providers.

AFE and Patient Reported Outcomes

This study sought to determine the relationship between AFE to contact/collision sports and various patient-reported outcomes among current and

former women and men community level rugby players. Among this cohort of over 1,000 rugby players, we did not observe an association between younger AFE and worse self-reported psychological distress or QoL. However, there was a significant relationship between self-reported history of concussion and worse psychological distress and QoL in both women and men. Furthermore, self-reported mental health outcomes improved with age, but self-reported physical health outcomes worsened with age in both sexes. Collectively, these findings suggest that younger AFE to contact/collision sports is unrelated to cognitive, mental health, and physical problems in early adulthood in community rugby players.[160,161,163–165]

The main finding of this study was that younger AFE to contact/collision sports, whether analyzed continuously or dichotomously at age 12, was not associated with worse patient reported outcomes among adult (31.6 $\pm$ 11.3 years (range: 18-74)) male and female community rugby players. These findings further prior cohort studies that found no effect of AFE on various neurophysiological and neuropsychological outcomes in current high school and collegiate student-athletes.[160,163–165] For example, similar to our findings, Caccese and colleagues reported that younger AFE to contact/collision sports was not associated with worse self-reported somatization, depression, or anxiety as measured by the BSI-18 in current collegiate student-athletes.[164] Contrary to previous work in current high school and college athletes, our study had an older average age (31.6 $\pm$11.3, Range: 18–74), and a larger proportion of women (41%) who had participated in contact/collision sports. Furthermore, a recent study of middle-aged men (aged 35-55) who played high school football, also reported

no association between younger AFE and depression when analyzed continuously and dichotomously at age 12, providing further evidence that AFE to contact/collision sports may not affect psychological functioning in early adults.[171] This expands on previous research in older adults, who participated in high school football, which reported no increased risk of neurodegenerative diseases compared to aged-matched individuals, who participated in non-contact varsity sports or who reported no organized sports participation history.[180,181] The findings herein furthers understanding of the early adulthood and mid-life effects of younger AFE on patient-reported outcomes in early adults across sexes. To our knowledge, this is the first study to include an analysis of AFE on women older than college-aged individuals. However, over 75% of the participants herein were under the age of 50, so the later life effects of prolonged RHI exposure in females remain to be elucidated.

While AFE was not associated with poorer outcomes in this study, participants with a self-reported concussion history had poorer psychological well-being and lower QoL which is consistent with the findings of Caccese et al.[164] and Meehan et al.[237]. Specifically, women with a history of concussion had worse scores on all seven outcomes (BSI-18 Anxiety, Depression, Somatization, and GSI, SF-12 PCS, SF-12 MCS, and SWLS), and men with a history of concussion performed worse on all four BSI-18 outcomes than those without a history of concussion. However, similar to previous studies, the clinical meaningfulness of these significant findings is likely low as evidenced by small effect sizes.[163,164] It should be noted that the BSI-18 is designed to measure current psychological distress (last 7 days)[230], so other patient-reported

outcomes may be necessary to determine the chronicity of psychological distress. It was noted that women with a history of concussion performed worse on measures of physical function (i.e., SF-12 PCS) which may be related to previous research highlighting persistent physical impairments (i.e. conservative gait) in those with a history of concussion.[73] Collectively, this finding of worse psychological distress and QoL among men and women with a history of concussion is concerning and warrants further investigation into the mechanisms underlying persistent impairments in men and women rugby players.

Cumulative years of contact/collision sport history was only a significant predictor for BSI-18 Depression and SF-12 MCS in men; whereby more years of participation was associated with lower depression and better self-reported mental health. Although this finding may seem counterintuitive, it may be result of the positive psychological effect of sport participation as lower depression symptoms have been reported in adults playing team sports [238] and high school athletes participating in contact/collision sports compared to non-contact sport athletes.[239] It should be noted, that most non-contact sports included in the previous study by Howell et al.[239] were individual sports, and therefore the effects of team sports versus individual sports on well-being and depression may have been driving this relationship and not necessarily the collision aspect (non-contact versus contact) of the sport played. However, several large scale studies have shown that contact/collision sports participation during high school is not associated with worse mental health problems in adulthood.[173,175–179] Moreover, team sport participation is associated with higher self-esteem, life

satisfaction, and decreased psychological distress, which may explain our findings among rugby players (i.e. team sport athletes).[240,241] Furthermore, rugby participation is associated with lower prevalence of cardiovascular disorders and metabolic dysfunction,[221] and middle-aged male rugby players reported self-actualism, making friends, and fulfillment in regards to rugby participation.[242] These positive psychosocial benefits of rugby participation have been associated with increased physical and mental activity,[242] resulting in improved overall player QoL, and may be potential mediators of our findings. As such, despite the potential risk for injury, consistent with previous findings, prolonged participation in contact/collision, or rather team sports like rugby may improve overall patient-reported outcomes of QoL.[238,242]

In both sexes, older age was associated with lower (i.e., better) BSI-18 Depression, Anxiety, and GSI, and higher (better) SF-12 MCS; older age was also associated with higher (better) SWLS in males. Similar to the aforementioned benefits of team sports participation, these improvements in psychological well-being with increased age may in part be the result of increased years of sport participation as a result of regular physical and social activity.[240,241] However, this analysis controlled for cumulative years of contact/collision sport participation, as such, this improvement in mental wellness may be representative of the reported linear increases in mental health through adulthood.[243] Lastly, across both sexes, increasing age was associated with lower (worse) SF-12 PCS which is most likely the result of age-related deteriorations in physical functioning.[243] Additionally, this decrement of physical

functioning with age may be compounded by continued or prolonged rugby participation as middle aged former rugby players report higher rates of musculoskeletal disorders (e.g., Standardized morbidity ratio (SMR): 4.00 for osteoarthritis) and morbidity (e.g., SMR: 2.69 for osteoporosis) compared to an age-standardized general population cohort (60% vs. 15% prevalence of osteoarthritis).[213]

The most notable limitation of this study was a self-reported anonymous online questionnaire, as opposed to in-person assessments, to acquire the data which may produce respondent bias; however, this is commonly utilized in public health and concussion literature and allows for the collection of larger sample sizes.[164,165,226] Additionally, AFE to contact/collision sports was self-reported, and may be subject to recall bias, however, this method has been consistently used in pervious AFE research.[160,161,163,165,171] It should be noted that men and women have differing neurodevelopmental trajectories[154], and therefore a dichotomous AFE for both sexes at age 12 may not have been appropriate, however, we aimed to mitigate this by analyzing AFE as a continuous variable. Additionally, we did not collect data on the number of RHI suffered for everyone, as such, the varying nature of average number of head impacts incurred, the forces incurred, and frequency may result in different dosages of RHI suffered by each individual. While some researchers have attempted to quantify RHI suffered over the course of one's athletic career,[167] we were unable to do so herein due to multi-sport athletes, thus, the utilization of AFE to contact/collision sports to assess RHI may be biased, despite this method being used extensively in previous research.[153,160,161,163–167,169–171,194,199] (Appendix D) Furthermore, concussion

history was examined as a binary (yes/no) and not a continuous variable, so conclusions on a "dosage effect" cannot be made. Consistent with previous studies, participants were asked to self-report their history of concussion, but were not provided a definition[126,127,228], which may result in underreporting.[25,244] The outcomes herein focused on psychological distress and QoL, so findings cannot be extended to other domains of brain health (e.g., neurocognitive and executive function). The results of this study extend previous findings to an early adulthood and mid-life population; however, only a small percentage (~10%) of participants were over 50 years old and, thus, later life conclusions remain to be elucidated. (Appendix E) Lastly, and importantly, ~25% of these data were collected following the novel COVID-19 related shutdowns in early March of 2020; this is important as this subset of participants may have experienced increased symptoms of psychological distress, which was reported in healthy 18-29 y.o. as a result of the pandemic.[245] In fact, post-hoc comparisons revealed significant differences between the three recruitment waves for BSI-18 Anxiety (i.e., the second and third waves had lower anxiety compared to the first wave ($p=0.048$)), SWLS (i.e., the second and third waves reporting higher (better) SWLS ($p=0.024$)), and SF-12 MCS, (i.e., the second and third waves reporting slightly better SF-12 MCS ($p=0.039$)). Interestingly, across all domains, psychological distress and QoL improved over time (i.e., the early group had the worst, the second wave was in the middle, and the latest wave had the best). However, AFE did not differ between timepoints (Wave 1 (N=764) AFE = 9.4 ± 5.0; Wave 2 (N=86) AFE =

8.7 $\pm$ 4.6; Wave 3 (N=187) AFE = 8.8 $\pm$ 4.6 (p=0.185)), so it is unlikely to have biased the AFE findings. (Appendix F)

In conclusion, consistent with recent cohort studies in college aged adults, younger AFE to contact/collision sports was not associated with worse patient-reported outcomes of psychological distress and QoL in early adult men and women rugby players. This was the first study to examine AFE in women beyond college. Whether analyzed continuously or dichotomously at age 12, younger AFE was not associated with worse psychological distress or QoL. However, prior concussion history was associated with worse self-reported outcomes on some assessments. Contact/collision sport participation alone was not associated with worse brain health, but playing contact/collision sports does increase risk of concussion, which may in turn lead to worse long-term psychological distress and QoL.[237] These findings add to the growing body of literature that younger AFE to contact/collision sports (i.e., boxing, American football, ice hockey, lacrosse, rugby, soccer, and wrestling) is unrelated to patient-reported outcomes of psychological distress and QoL in young and middle aged adults; however, further exploration is required in older adults.

Chapter 3

ASSOCIATION BETWEEN RHI FROM COLLISION SPORTS INTO ADULTHOOD ON DYNAMIC POSTURAL CONTROL AND PATIENT REPORTED OUTCOMES

Introduction

Rugby provides a unique opportunity to investigate the effects of prolonged RHI exposure since most American rugby players start playing rugby in college after a career of other collision sports (e.g., football, wrestling, ice hockey) and continue participation in organized leagues (i.e., practices and games) beyond college for adult club teams.[216] For example, our previous research provided insight that the average American adult rugby player had a cumulative contact/collision sport history of ~13 years for females (range: 1-56) with an AFE of ~10 years old, and ~19 years for male players (range: 1-79) with an AFE of ~9 years old, highlighting the prolonged participation beyond the typical retirement age of most collision sport athletes.[127,201] In this cohort, earlier AFE to contact/collision sports was not associated with worse QoL/patient reported outcomes, however, cumulative years of contact/collision sports was a significant predictor of better self-rated depression and mental health in men.[201] Although this expands AFE/RHI research into female and older cohorts, we did not collect data on physical or behavioral measures (e.g., postural control) which may be affected by prolonged exposure to RHI.[167,246] Therefore, a need exists to investigate the relationship between prolonged RHI exposure (i.e., career duration) on dynamic

postural control. This is in line with the NINDS consensus for TES suggestion that investigations on RHI would benefit from measure of career duration over AFE, in addition to expanding research utilizing more objective behavioral measures such as gait.[191]

One of the main reasons for the NINDS push for expanded research is the homogeneity of study populations and mixed findings among the research on the effects of RHI and short- and long-term health outcomes.[191] The short term effects of RHI have shown minimal to no effect on neurological and behavioral health among collegiate American football players and contact/collision sport athletes.[151,152,164,246] Further, high school football participation has been shown to be unrelated to later-life risk of neurodegenerative diseases[180,181] and cognitive and mental health dysfunction among community dwelling individuals.[176,177] However, cohort data of symptomatic former NFL players have shown negative consequences to contact sport participation such as increased odds of CTE (although the base rate of CTE remains unknown), neuropsychological dysfunction, and white matter abnormalities; it is worth mentioning that these data remain topic of debate due to potentially biased sampling and recruitment methods and reliance on self-report data.[153,166,169,200] Additionally, individuals who play in the NFL make up an extremely small proportion of U.S. men with a history of playing American football in addition to their prolonged playing careers (i.e., prolonged RHI exposure), thus limiting generalizability to other cohorts. These data are challenged by other cohorts of American football players in mid-life whereby earlier age of first exposure to American football and participation in football

was not related to later-in-life cognitive dysfunction, midlife brain health, or suicide risk.[152,171,173,174,178] Beyond the original Stamm study, AFE research has been limited to studies among collegiate student athletes or middle aged males with a history of football participation, primarily utilizing subjective outcomes measures and/or retrospective self-report data.[152] As such, a need exists to expand research to other contact/collision sport athletes, with the inclusion of sex-matched sports, such as rugby, to investigate the effects of RHI on long-term brain health across the lifespan utilizing objective measures of neurological health.

The long-term effects of concussion, and more specifically, RHI in rugby players and other contact/collision sport athletes remains to be elucidated.[114,147,191,246] It has been reported that former rugby players report a high cumulative injury burden, osteoarthritis, and multiple concussions.[212] Similar to studies of retired football (i.e., soccer) players,[190] former rugby players reported lower frequencies[247] and incidences[248] of chronic conditions (e.g., hypertension) compared to age matched controls, which may be the result of greater lifetime physical activity levels compared to population controls.[247,248] It can be posited that the improved physical health of retired rugby players[221] and soccer players[190] compared to age matched controls are due to greater physical activity levels as physical activity is associated with positive long-term reductions in incidence of Alzheimer's disease and dementia, risk of coronary heart disease, type 2 diabetes mellitus, and other non-communicable diseases.[249] Thus, it is important to consider physical activity and lifetime exercise

habits as potential confounders and moderators of the relationship between RHI exposure on neurophysiological health in ageing populations.

Although former contact/collision sport participation appears to yield improved physical health outcomes in later life,[190,221] this may not translate to improved long-term mental health despite the link between physical activity and mental health.[238,250] For instance, retired rugby players[251] report greater prevalence of anxiety and depression (28%) than the current professional rugby players[224] in addition to symptoms of common mental disorders (e.g., distress (25%), adverse alcohol use (24%)); of note, these data are limited to male populations and did not control for years of sport participation.[224,251] Conversely, years of football participation among former NFL players was not associated with increased depressive symptom severity.[115] Thus, a knowledge gap remains to determine the effects of career duration on long-term mental health outcomes beyond early-adulthood.

The side effects of rugby participation (e.g., RHI exposure and concussion) on neurological health is also mixed and poorly developed.[252] Short-term data from current players are also inconclusive with some studies showing reductions in cognitive function[221,248,253,254] following rugby matches, drills, practices, etc., but other studies show no effect.[221,247] Further, neuroimaging and brain function (e.g., transcranial magnetic brain stimulation) studies have reported mixed short-term and long-term effects of rugby participation.[255,256] Yet, data from the BRAIN study of 146 former elite English rugby players found no relationship between concussion and cognitive function.[121] Lastly, a 2020 systematic review noted a lack of high quality

and properly designed studies on the long-term effects of concussion and cognitive health, with some studies showing a negative effect, but the clinical relevance remaining unknown.[122] However, none of these investigations particularly studied the long-term effects of RHI, and primarily focused on concussion—a less prevalent occurrence than RHI—thus, a gap in the literature exists to determine the effects of rugby participation (prolonged RHI exposure) on long-term neurological and behavioral outcomes.

A critical limitation of previous research was the lack of comparison groups to populations of interest. In particular, comparison groups are needed to further separate the potential neuroprotective effects of exercise and the negative effects of RHI on neurodegeneration.[257] It is imperative to account for the effects of exercise, as exercise significantly reduces the majority of characteristics of brain aging such as cognitive decline in addition to causing enhanced glucose utilization, regulating glycolysis, increasing neurogenesis and network connectivity, reducing oxidative damage, improving calcium homeostasis and energy metabolism, and increased mitochondrial health and function.[258] Thus, a need exists to determine the effects of RHI on neurophysiological functioning across the sporting and physical activity levels spectrum (i.e., non-contact athletes, contact/collision sport athletes, and non-physically active individuals). Furthermore, it is imperative to utilize more objective measures of neurophysiological function, such as gait and other clinically viable tests, as previous research has been primarily limited to subjective outcome measures.[147,152,246]

Gait, mainly walking speed, is considered to be the 6th vital sign since it correlates with balance, functional ability,[259] cognitive status,[89] postural control,[90] and mental health status,[260,261] hospitalization,[262] and death.[263] Additionally, impairments in gait and postural control are reported in athletes beyond clinical recovery from concussion and even in those with a history of concussion,[73,74,78,96,264,265] with both tandem gait and instrumented gait using inertial measurement units (IMUs).[265,266] Also, the addition of a cognitive challenge during a motor task, termed "dual task" (DT), has been utilized to identify post-concussion and persistent (lasting months to years) subclinical deficits in executive function and neurophysiological impairments in collegiate athletes and adolescents.[72,132,267,268] Further, one can calculate the dual task cost (DTC), which is commonly used to assess the negative changes in performance in DT gait relative to ST conditions.[59] DTCs are an important variable in neurologic populations as they provide insight in the interaction between the motor and cognitive system. By analyzing DTCs, we can highlight any subtle gait and/or cognitive deficits in individuals that have experienced prolonged RHI exposure.[59,102,246,269]

The inclusion of cognitive tasks, during gait, requires dorsolateral prefrontal cortex executive functioning in order to enable simultaneous processing of the motor and cognitive demands.[89] This is noteworthy because white matter and metabolic abnormalities have been shown in the frontal lobes of retired collision sport athltes.[256,270] However, a majority of investigations into gait abnormalities as a result of RHI exposure or neurotrauma have been limited to younger (e.g., college or high school) male collision sport athletes, or male and female contact sport

athletes.[150,246,271–274] Therefore, a need exists to extend previous findings on the effects of RHI exposure on postural control by including an older sample of middle-aged collision sport athletes. In doing so, we can provide insight onto the mid-life effects of various collision sports and RHI exposure. Furthermore, IMUs based tandem or ST/DT gait provide a clinically feasibly, ecologically valid, and portable method to assess the neurophysiological effects of RHI exposure on dynamic postural control.

An accumulation of RHI through routine contact/collision sport participation has been theorized to lead to long-term neurological dysfunction. A 2021 systematic review highlighted the heterogeneity of the effects of RHI on postural control (i.e., negative, no change, or improvements) in addition to homogeneous study populations (i.e., young and healthy adolescent and college aged); as such, authors suggested that future research is needed on different athlete populations.[246] As such, these data are lacking in generalizability, and similar to AFE research, are limited to short-term studies on young, healthy adolescent and college aged athletes.[147,246,270] Further, a majority of these studies lacked a measure of RHI exposure and simply group individuals into RHI or no RHI groups, failing to account for the broad range of RHI exposure differences between sport and years played.[246] Therefore, a gap exists to expand these inquiries into the mid-life effects (i.e., ages 30-50 years old) of prolonged RHI across the sporting spectrum and sex. Rugby players will provide exclusive insight into the effects of prolonged RHI exposure in men and women athletes into adulthood, given their prolonged playing careers as amateur athletes. Thus, expansion of research to these rugby athletes, age groups, and the inclusion of

comparison groups (i.e., no RHI, former RHI exposure, and prolonged RHI exposure) of varying physical activity levels (i.e., yes/no meeting physical activity guidelines) can help provide insight onto the subtle postural control impairments and neurophysiological dysfunction that may be caused by RHI exposure.

Specific Aim 2 Description

To determine the associations between RHI from contact/collision sports into adulthood on neurological health in adults with no history of, a history of (up to age 22), and a history of prolonged exposure (beyond age 22) to RHI.

H2.1: Longer career duration will be associated with conservative gait strategy and worse dual task costs (DTC) in collision sport participants.

H2.2: Those with a history of prolonged RHI who are physically active and those with no history of RHI who are not physically active will perform worse on gait and clinical assessments than those without a history of RHI who are physically active.

H2.3: Slower ST gait speed and worse gait speed DTC will be associated with worse performance on tests of executive function, psychological well-being, and QoL.

Methods

Participants

We recruited 113 adults representing four distinct groups via word of mouth, e-mail, local rugby teams and adult recreational groups, community programs, and local gyms. (Table 5) All participants provided oral and written informed consent in accordance with the University of Delaware's IRB (#1605665-6). (Appendix M)

Table 5 Aim 2 Group Description and Inclusion/Exclusion Criteria

Group ID	Description	Inclusion Criteria	Exclusion Criteria
NON	Former non-contact athletes/non-athletes (NON) who are not physically active (no RHI exposure)	- Never played organized contact/collision sports - Do not self-report currently meeting the ACSM physical activity guidelines (150 minutes of moderate or 60 minutes of vigorous physical activity per week)[275]	Self-report: - Current pregnancy - Any acute or chronic impairment that would interfere with normal gait and balance (e.g., vestibular disorders) - Lower-extremity musculoskeletal injury at the time of testing - Concussion within 6 months of the test date - Any pre-existing neurological, balance, hearing, vestibular or ocular disorders - History of stroke or neurodegenerative disease - Unstable cardiac or pulmonary disease
NCA	Former non-contact athletes (NCA) who are physically active. (no RHI exposure)	- Never played organized contact/collision sports - Self-reported meeting ACSM physical activity guidelines	
HRS	Former contact/collision sport athletes who participated in high-risk sports (HRS) for RHI sports (i.e., boxing, football, ice hockey, lacrosse, soccer, wrestling) who are physically active (previous RHI exposure)	- History of organized contact/collision sport participation but ceased by age 22. - Self-reported meeting ACSM physical activity guidelines	
RUG	Current and former rugby players, with a history of playing rugby (RUG) after the age of 22 (e.g., RHI exposure into adulthood after the age at which most collision sport participation ceases) (prolonged RHI exposure)	- Older than 22 years old with a history of playing a least one year of full contact (i.e., tackle) rugby after the age of 22 - Self-reported meeting ACSM physical activity guidelines	

Participants enrolled prior to June 30, 2021, 20 were randomly selected to receive a $20 Amazon.com gift card using a random number generator (random.org). Following June 30, 2021, and IRB amendment approval, all newly enrolled participants who completed the study were compensated $20 in the form of an Amazon.com gift card. Lastly, those enrolled after September 29, 2021, received a $40 Amazon.com gift card.

Instrumentation and Procedures

Most of the Aim #2 testing occurred in the Concussion Research Lab at the University of Delaware's STAR Health Science Complex. However, testing was portable and could be completed in any quiet and safe location with a 7m flat surface and internet connection. Post-hoc analyses were performed and determined testing location did not influence any participant normalized gait task outcomes (e.g., DTC) (p=0.332-0.766).

Participants were screened via phone or e-mail for eligibility and if eligible subsequently scheduled for testing. Testing lasted ~90 minutes and consisted of the following items in order: informed consent, online questionnaires, clinical assessments, and ST and DT gait assessments (tandem and instrumented gait). DT tandem and DT gait utilized the same order of tasks per participant (i.e., Trial 1-5: Words spelled backwards, Subtraction 7s, Months in reverse, Words, Subtraction 6s). If a participant reported English as a second language (n=8), the words task was omitted and replaced with subtraction and months tasks; post-hoc analyses revealed no

differences in cognitive accuracy between native and non-native English speakers (p=0.374).

Questionnaires

To assess various measures of psychological well being, AFE to contact/collision sport, sport history, and injury history, participants completed a set of questionnaires via Qualtrics. (Qualtrics, Provo, UT, USA). The questionnaires also asked demographic questions such as age, height, weight. Lastly, various measures of psychological well-being and QoL were used to expand on previous AFE literature. (Table 6) (Appendix G).

Table 6 Online Questionnaires and Outcome Measures

Questionnaire	**Outcome Measure**	**Range/Normative Data**
AFE/Organized Sport History	A custom MATLAB script will be utilized to determine the youngest AFE to contact/collision sports. (i.e., minimum AFE reported for all contact/collision sports will be reported AFE age)	N/A
Career Duration	Years played in each contact/collision sport will be summed to create a cumulative years contact/collision sport variable.[201] (ex.: 1 year football and 1 year of ice hockey during the same season would be 2 years career duration)	N/A
Brief Sensation Seeking Scale (BSSS)	8 questions on individual's interests and preferences for each statement Rating: 1 (Strongly Disagree) to 5 (Strongly Agree)[276,277]	Range: 1-5; score reported as a mean of the 8 questions[278] Higher scores indicate greater sensation seeking

Injury History	14 questions on diagnosed, unrecognized, and unreported concussion history and LE-MSI history[227,228]	N/A
BU Head Impact Exposure Assessment	Cumulative Head Impact Index (CHII) Metric to quantify lifetime RHI exposure using self-reported athletic exposure and extrapolated objective measures based on sport/position played from accelerometer data.[167]	N/A
SCAT 5 Symptoms	22 items whereby individuals rate their symptoms based on how they typically feel[279] Rating: 0 (None) to 6 (Severe)	Range: 0-132 Higher scores indicate higher symptom burden
Short Form Health Survey (SF-12)	12 questions on one's functional health & well-being Rating: differs by question (e.g., 'none of the time' to 'all of the time' or 'poor' to 'excellent')	Range: 0 – 100[231] Higher scores indicate better QoL; 50 is representative of the average American.[231]
Satisfaction with Life Scale (SWLS)	5 statements assessing judgement of life satisfaction Rating: 1 (Strongly Disagree) to 7 (Strongly Agree)	Range: 5 (lowest) – 35 (highest satisfaction)[232]
Apathy Evaluation Scale (Self-Rated)	18 questions on one's self-reported apathy in the last 4 weeks Rating: Differs by question 1-4 (e.g., 'A lot' to 'Not at all characteristic')	Range: 18 – 72[280] Higher scores indicate worse apathy, AES >34 is clinically meaningful apathy.[280]

Clinical Measures

The following clinical measures were used to assess neurophysiological function (Appendix H):

Cognitive Assessments: Participants were asked to perform the following 3 assessments of memory, attention, processing speed, cognition, and executive function.

a) Trail Making Test A (TMT-A):[281,282] Participants used their finger as a stylus on the iPad to connect the circles numerically from 1-25 as quickly and accurately as they could. 1 trial was performed; time to completion and errors were recorded.

b) Trail-Making Test B (TMT-B):[281,282] Using the same methods as above, participants connected the circles in a sequential number-letter-number pattern (i.e., 1-A-2-B). 1 trial was performed; time to completion and errors were recorded. A composite measure, Trails Diff, was created by subtracting the TMT-A time from the TMT-B time as well as Trails Ratio (TMT-A divided by TMT-B). Both TMT-A and TMT-B have good reliability and construct validity, with re-test reliability ranging from 0.76-0.94.[282]

c) Montreal Cognitive Assessment (MOCA):[283] Using an interview format, participants completed a 30-point test consisting of memory recall, visuospatial, TMT-B, verbal abstraction, attention, concentration, verbal fluency, language, and orientation tasks. The outcome measure was total score (0-30; $\geq$26 is normal cognitive function).[283] The MOCA has high specificity and sensitivity for mild cognitive impairment, good internal consistency (0.83), and good test-retest reliability.[283]

Oculomotor/Vestibular-ocular Tests:

a) King-Devick (KD):[284] Participants utilized horizontal saccadic eye movement reading numbers left to right across 3 cards of varying difficulty as quickly as possible without errors. 2 trials were performed and the fastest time without errors was reported. KD has high specificity (90%) and sensitivity (86%) in identifying concussed versus control athletes.[285]

b) Vestibular/Ocular Motor Screen (VOMS):[286] A 6-item battery assessing smooth pursuits, horizontal and vertical saccades, near point convergence (NPC), horizontal and vertical vestibular ocular reflexes, and visual motion sensitivity. Prior to the VOMS and after each assessment participants rated four symptoms (headache, dizziness, nausea, fogginess) 0(none)-10(worst possible). Symptom count for all 6 tasks were recorded, 3 NPC distances (cm), as well as if the test was terminated prematurely due to symptom provocation of 2 or more. VOMS has established internal consistency and sensitivity for identifying those with concussions.[286]

Postural Control:

Balance Error Scoring System (BESS):[287] Consists of 3 positions: feet together, single leg (non-dominant foot), and tandem (non-dominant foot in back) performed twice, once on a firm surface and once on a foam surface. Stances are held 20s and an error point is awarded if eyes open, hands lift off of hips, stumbling, lifting forefoot or heel, falling out of position, hip abduction greater than 30°, or the participant remains out of position for greater than five seconds. The sum of error counts for all 6 tests were reported. BESS has moderate-good reliability for assessing static balance and correlates with more sophisticated measures of balance.[287]

Tandem Gait (TG):[28,288] A clinically valid test involving participants walking heel-toe 3m, turning 180°, and returning to the start line walking heel-toe. Four single task (ST), or walking only, trials and 4 dual task (DT) were performed whereby participants completed a cognitive task (i.e., spelling five-letter words backwards, serial 6s/7s, or months in reverse order). The dependent variable was dual task cost

(DTC) for completion time. DTC is calculated as a percentage change between ST and DT conditions: (DT-ST)/(ST), which is consistent with previous work.[97]

Instrumented Gait:[289–291] Participants completed 5 ST walking trials and 5 DT walking trials using the same cognitive tasks as TG while simultaneously wearing three triaxial accelerometers (Opal Sensor, APDM Inc., Portland, OR). Opals were placed on the dorsal surface of each foot and L5 vertebrae. Walking trials consisted of gait initiation in response to an acoustic stimulus (i.e., beep), walking 7m, turning around, and walking 7m back to the starting position. Data were collected at 128 Hz and analyzed using Mobility Lab software.[292] This method, when used for level overground gait, has excellent strength-of-agreement (LCC=0.95, r=0.95, ICC=0.94) and is highly reliable and accurate for spatiotemporal gait parameters.[293,294] Dependent variables were double support (%), gait speed (m/s), and stride length (SL) (m) for ST, DT, and the associated DTC for each. All IMU gait, DT, and assessments were performed in a manner consistent with established procedures.[132,267,268,295]

Statistical Analysis

Career Duration and Gait Outcomes in Collision Sport Athletes

A multiple regression using the enter method was utilized to predict ST, DT, and DTC for gait outcomes (i.e., tandem gait, stride length, gait speed, and double support) in collision sport athletes (i.e., HRS and RUG) from contact/collision sport career duration (years), age (years), sex (M/F), and height (m).

Linearity was assessed using partial regression plots and plots of studentized residuals versus the predicted values. Independence of residuals was assessed with a

Durbin-Watson statistic. Data were checked visually for homoscedasticity. Multicollinearity did not exist (VIF<10).

Effect of Sport Participation on Clinical and Gait Outcomes

Data were tested for normality using a Shapiro-Wilk statistic. For non-normally distributed data (i.e., Shapiro-Wilk $p<0.05$) a Kruskal-Wallis test was used to compare groups, for normally distributed data a one-way ANOVA was used to compare groups. All variables were non-normal except for the BSSS (Shapiro Wilk $p>0.05$). Thus, Kruskal-Wallis H-test chi-square values are reported for all variables except the BSSS which is reported as an ANOVA F-test value. Effect sizes are reported as eta-squared for Kruskal-Wallis test and partial eta-squared for the ANOVA test. Effect sizes were interpreted as small = 0.01; medium = 0.06; and large = 0.14.[296] Further, for comparisons between two groups a Mann-Whitney-U test was used. Initial analyses compared groups on all gait and clinical outcomes without covariates with either a Kruskal-Wallis test with a pairwise comparison procedure and Bonferroni correction for multiple comparisons or a one-way ANOVA with a Tukey post-hoc for significant outcomes.

Follow up analyses utilized covariates (i.e., age, sex, concussion and LD/ADHD history, career duration, and height) in a one-way ANOCVA to compare groups on the same clinical and gait outcomes to see if covariates affected the model. Post-hoc analyses were performed for significant outcomes with a Bonferroni test for multiple comparisons.[297]

Clinical Outcomes & DTC:

The relationship between ST gait speed and performance on clinical tests (TMT, SF-12, AES-S, Symptom Severity, SWLS, BSSS, MOCA, King-Devick, and BESS) as well as DTC gait speed and performance on clinical tests were analyzed using multiple Spearman's rank-order correlations since data were not normally distributed. The strength of Spearman's Rho (r_s) was interpreted as the follows: no correlation (0-0.19); low correlation (0.20-0.30); moderate correlation (0.40-0.59); moderately high correlation (0.60-0.79); and high correlation ($\geq$0.80).[298] Significance was set a $P<0.05$ and all analyses were ran using SPSS v. 26 (SPSS Inc, Chicago, IL).

Results

113 participants completed this study across the four groups: Former non-contact athletes/non-sporting individuals who are not physically active (NON)=28; former non-contact athletes who are physically active (NCA)=29; former contact/collision sport athletes who are physically active (HRS)=29; and current and former rugby players with prolonged playing history beyond age 22 who are physically active (RUG)=27. Demographic data by group are reported in Table 7. The groups did not differ on age or sex ($p>0.05$). (Table 7)

Table 7 Aim 2 Participant Demographics by Group

	Group 1 (NON)	**Group 2 (NCA)**	**Group 3 (HRS)**	**Group 4 (RUG)**	**Overall**	**Kruskal-Wallis Test $X^2(3)$ Value/Mann-Whitney U Value**	**P-Value**	**Effect Size (Eta Squared)**
N	28	29	29	27	113	N/A	N/A	N/A
Age (Years)	35.43 ± 14.17 (Range: 18-67) [95%CI: 29.93-40.92]	33.90 ± 10.79 (Range: 23-67) [95%CI: 29.87-37.93]	33.29 ± 8.39 (Range: 22-58) [95%CI: 29.03-35.54]	38.07 ± 12.98 (Range: 22-67) [95%CI: 32.94-43.21]	34.88 ± 11.80 (Range: 18-67) [95%CI: 32.68-37.07]	2.435	0.487	0.005
Sex (M/F)	9/19	11/19	17/11	16/11	53/60	6.582	0.086	0.033
BMI (kg/m^2)	26.90 ± 6.56 (Range: 17.85-44.63) [95%CI: 24.35-29.44]	24.46 ± 3.47¥ (Range: 19.97-37.92) [95%CI: 23.17-25.76]	26.04 ± 4.38 (Range: 19.20-41.09) [95%CI: 24.33-27.73]	29.31 ± 5.01† (Range: 22.46-40.18) [95%CI: 27.33-31.30]	26.61± 5.20(Range: 17.85-44.63) [95%CI: 25.64-27.58]	13.027	0.005	0.092
Height (m)	1.70 ± 0.10 (Range: 1.55-	1.71 ± 0.10 (Range: 1.52-	1.73 ± 0.10 (Range: 1.57-	1.73 ± 0.10 (Range: 1.52-	1.72 ± 0.10 (Range: 1.52-1.98)	2.755	0.431	0.021

	1.96) [95%CI: 1.66-1.73]	1.93) [95%CI: 1.67-1.75]	1.98) [95%CI: 1.69-1.77]	1.91) [95%CI: 1.69-1.77]	[95%CI: 1.70-1.73]			
LD/ADHD (Y/N)	4/24	4/25	5/24	5/22	18/95	0.325	0.955	0.025
Concussion History (Y/N)	7/21	11/18‡¥	18/11*	19/8*	55/58	14.661	0.002	0.107
AFE Contact/Collision Sports	N/A	N/A	7.97 ± 3.53¥	13.30 ± 6.56‡	10.54 ± 5.82	180.500	<0.001	0.485
Contact/Collision Career Duration (Years)	N/A	N/A	14.48 ± 9.30	16.33 ± 11.98	15.37 ± 10.62	363.500	0.646	2.961

Data were not normally distributed (Shapiro Wilk $p<0.05$), thus a Kruskal-Wallis H test was performed to compare group means for demographic data. *: significant difference from NON. †: significant difference from NCA. ‡: significant difference from HRS. ¥: significant difference from RUG.

Career Duration and Gait Outcomes in Collision Sport Athletes

Results of the multiple regression models are shown below. (Table 8). There was no evidence of multicollinearity for any of the models (VIF<10). Further, there was independence of the residuals based upon the Durbin-Watson statistic. Homoscedasticity existed and was confirmed for each model via visual inspection of the studentized residuals and unstandardized predicted values plot.

None of the models significantly predicted any of the gait outcomes ($p>0.05$). For many of the models, none of the variables added statistically to the model ($p>0.05$). However, age added statistically for the model for ST double support ($p=0.012$), DT double support ($p=0.039$), and ST tandem gait ($p=0.017$) whereby increased age resulted in a more conservative gait performance (i.e., increased ST/DT double support, and increased ST tandem gait time).

Table 8 Multiple Linear Regression: Collision Sport Athletes and Gait

Outcome	F-value	p-value	Adj. R^2	Durbin-Watson Statistic
ST Gait Speed	0.976	0.429	-0.002	1.619
DT Gait Speed	0.619	0.651	-0.029	1.801
DTC Gait Speed	0.233	0.918	-0.059	1.892
ST Double Support	1.968	0.113	0.066	1.566
DT Double Support	1.441	0.234	0.031	1.393
DTC Double Support	1.718	0.160	0.050	1.844
ST Stride Length	1.677	0.170	0.047	1.694
DT Stride Length	1.553	0.201	0.039	1.902
DTC Stride Length	0.516	0.724	-0.036	2.032
ST Tandem Gait	1.738	0.156	0.052	2.269
DT Tandem Gait	1.613	0.185	0.043	0.050
DTC Tandem Gait	0.952	0.442	-0.004	1.820

None of the models significantly predicted gait outcomes ($p>0.05$).

Effect of Group/Sport Participation on Psychological Outcomes

Means, standard deviations, range, and 95% confidence intervals (CI) for each psychological variable (i.e., SF-12 PCS and MCS, AES-S, SWLS, BSSS, and the MOCA) are presented in Table 9. The percentage of abnormal responses (i.e., meeting the standard of a clinical cut point) are also highlighted.

There was a significant difference between groups for SF-12 (PCS) with a significant difference between NON and RUG (adjusted p=0.017). There was a significant difference between groups for AES-S with post-hoc analyses revealing a significant difference between NON and NCA (adjusted p=0.04). There was a significant difference between groups for SWLS with post-hoc analyses reporting a significant difference between NON and HRS (adjusted p=0.041) and NON and NCA (p=0.033). Lastly, there was a significant difference between groups for the MOCA with a post-hoc indicated difference between NON and NCA (adjusted p=0.024). There were no significant group differences for SF-12 (MCS) or the BSSS. (Table 9) These results are graphically represented in Appendix I.

Table 9 Psychological Outcomes by Group

	Group 1 (NON)	Group 2 (NCA)	Group 3 (HRS)	Group 4 (RUG)	Overall	Kruskal-Wallis Test $X^2(3)$ Value/ANOVA F Value	P-Value	Effect Size (Eta Squared)
N	28	29	29	27	113	N/A	N/A	N/A
SF-12 (PCS) Abnormal (<50)	53.23 ± 6.16† (Range: 33.8-61.4) [95%CI: 50.84-55.62] **5/28 (17.9%) abnormal**	56.69 ± 2.22¥* (Range: 52.28-65.03) [95%CI: 55.84-57.53] **0/29 (0%) abnormal**	55.05 ± 2.91 (Range: 46.8-60.4) [95%CI: 53.95-56.16] **1/29 (3.4%) abnormal**	52.80 ± 6.12* (Range: 30.7-57.5) [95%CI: 53.95-56.16] **4/27 (14.8%) abnormal**	54.48 ± 4.86 (Range: 30.7-65.0)	10.464	0.015	0.068
SF-12 (MCS) Abnormal (<50)	51.00 ± 8.27 (Range: 32.7-61.3) [95%CI: 47.79-54.21] **10/28 (35.7.%) abnormal**	51.82 ± 7.25 (Range: 29.70-58.08) [95%CI: 49.06-54.58] **8/29 (27.6%) abnormal**	52.32 ± 6.21 (Range: 36.3-60.8) [95%CI: 49.95-54.67] **8/29 (27.6%) abnormal**	49.36 ± 10.42 (Range: 22.6-60.3) [95%CI: 45.24-53.48] **10/27 (37.0%) abnormal**	51.16 ± 8.11 (Range: 22.6-61.3)	0.443	0.931	0.023
AES-S	28.54 ± 5.75†	24.34 ± 4.22*	24.93 ± 5.54	28.00 ± 7.85	26.41 ± 6.15	10.630	0.014	0.070

Abnormal (≥34)	(Range: 19-41) [95%CI: 26.31-30.77] **5/28 (17.9%) abnormal**	(Range: 18-34) [95%CI: 22.74-25.95] **1/29 (3.4%) abnormal**	(Range: 18-37) [95%CI: 22.83-27.04] **4/29 (13.8%) abnormal**	(Range: 19-56) [95%CI: 24.89-31.11] **4/27 (14.8%) abnormal**	(Range: 18-56)			
SWLS Abnormal (<20)	25.18 ± 5.88†‡ (Range: 9-35) [95%CI: 22.90-27.46] **4/28 (14.3%) abnormal**	29.24 ± 4.93* (Range: 16-35) [95%CI: 27.37-31.12] **1/29 (3.4%) abnormal**	29.21 ± 3.77* (Range: 20-35) [95%CI: 27.77-30.64] **0/29 (0%) abnormal**	26.44 ± 6.41 (Range: 12-35) [95%CI: 23.91-28.98] **5/27 (18.5%) abnormal**	27.56 ± 5.54 (Range: 9-35)	10.191	0.017	0.066
BSSS	2.84 ± 0.67 (Range: 1.25-4) [95%CI: 2.58-3.10]	2.74 ± 0.71 (Range: 1.0-4.25) [95%CI: 2.47-3.01]	2.88 ± 0.69 (Range: 1.75-4.88) [95%CI: 2.62-3.14]	3.00 ± 0.66 (Range: 1.88-4.25) [95%CI: 2.75-3.27]	2.87 ± 0.68 (Range: 1.0-4.88) [95%CI: 2.74-2.99]	0.726	0.539	0.020
MOCA Abnormal (<26)	26.79 ± 1.73† (Range: 24-30) [95%CI: 26.11-27.46]	28.03 ± 1.43* (Range: 24-30) [95%CI: 27.49-28.58]	27.03 ± 1.72 (Range: 23-30) [95%CI: 26.38-27.69]	27.07 ± 1.82 (Range: 23-30) [95%CI: 26.36-27.79]	27.24 ± 1.72 (Range: 23-30)	9.550	0.023	0.06

	6/28 (21.4%) abnormal	**2/29 (6.9%) abnormal**	**5/29 (17.2%) abnormal**	**4/27 (14.8%) abnormal**				

Data were not normally distributed (Shapiro Wilk $p<0.05$); thus, a Kruskal-Wallis H test was performed to compare group medians for all data except for BSSS (Shapiro Wilk $p>0.05$), so ANOVA was ran). Data reported as means $\pm$ SD. *: significant difference from NON. †: significant difference from NCA. ‡: significant difference from HRS. ¥: significant difference from RUG.

Effect of Group/Sport Participation on Concussion Battery Outcomes

Means, standard deviations, range, and 95% confidence intervals (CI) for each concussion battery variable (i.e., SCAT 5 Symptom Severity, TMT-A, TMT-B, TrailsDiff, TrailsRatio, King-Devick, and BESS) are presented in Table 10. The percentage of abnormal responses (i.e., meeting the standard of a clinical cut point) are also highlighted. There were no significant differences between group on any of the clinical outcomes.

Table 10 Concussion Battery Outcomes by Group

	Group 1 (NON)	**Group 2 (NCA)**	**Group 3 (HRS)**	**Group 4 (RUG)**	**Overall**	**Kruskal-Wallis Test $X^2(3)$ Value/ANOVA F Value**	**P-Value**	**Effect Size (Eta Squared)**
N	28	29	29	27	113	N/A	N/A	N/A
SCAT5 Symptom Severity	8.91 ± 9.66 (Range: 0-41) [95%CI: 4.74-13.09]	6.07 ± 6.70 (Range: 0-28) [95%CI: 3.52-8.62]	7.14 ± 7.68 (Range: 0-35) [95%CI: 4.16-10.12]	11.56 ± 12.65 (Range: 0-49) [95%CI: 6.55-16.56]	8.30 ± 9.4 (Range: 0-49) [95%CI: 6.55-10.06]	3.077	0.380	0.001
Trails A (s)	20.36 ± 5.15 (Range: 12.30-30.90) [95%CI: 18.37-22.36]	18.63 ± 5.17 (Range: 11.80-30.20) [95%CI: 16.66-20.59]	19.02 ± 5.36 (Range: 9.70-34.90) [95%CI: 16.98-21.06]	21.76 ± 5.31 (Range: 14.10-37.30) [95%CI: 19.66-23.86]	19.91 ± 5.32 (Range: 9.70-37.30) [95%CI: 18.91-20.90]	6.734	0.081	0.043
Trails B (s)	45.75 ± 17.50 (Range: 23.40-84.20)	38.68 ± 10.16 (Range: 23.70-58.20)	36.06 ± 7.41 (Range: 24.80-55.60)	39.31 ± 10.55 (Range: 22.20-63.20)	39.91 ± 12.33 (Range: 22.20-84.20)	3.949	0.267	0.018

	[95%CI: 38.97-52.53]	[95%CI: 34.82-42.54]	[95%CI: 33.24-38.88]	[95%CI: 35.13-43.48]	[95%CI: 37.61-42.21]			
TrailsDiff (s)	23.72 ± 12.68 (Range: 7.2-55.0) [95%CI: 18.24-29.20]	20.05 ± 9.54 (Range: 6.0-43.7) [95%CI: 16.42-23.68]	16.74 ± 7.24 (Range: 4.1-38.7) [95%CI: 13.93-19.55]	17.55 ± 10.82 (Range: -5.10-39.10) [95%CI: 13.27-21.83]	20.00 ± 11.19 (Range: -5.1-58.3) [95%CI: 17.92-22.09]	4.855	0.183	0.017
TrailsRatio	0.48 ± 0.12 (Range: 0.27-0.75) [95%CI: 0.43-0.53]	0.50 ± 0.13 (Range: 0.25-0.78) [95%CI: 0.45-0.55]	0.54 ± 0.14 (Range: 0.30-0.83) [95%CI: 0.49-0.59]	0.58 ± 0.19 (Range: 0.35-1.16) [95%CI: 0.51-0.66]	0.52 ± 0.15 (Range: 0.25-1.16) [95%CI: 0.50-0.55]	4.704	0.195	0.016
King-Devick (s)	39.82 ± 6.63 (Range: 25.94-51.55) [95%CI: 37.25-42.39]	39.51 ± 5.30 (Range: 30.03-50.40) [95%CI: 37.49-41.52]	38.58 ± 6.33 (Range: 29.36-52.68) [95%CI: 36.18-40.99]	42.64 ± 8.60 (Range: 28.23-65.71) [95%CI: 39.23-46.04]	40.09 ± 6.86 (Range: 25.94-65.71) [95%CI: 38.82-41.37	1.830	0.146	0.048
BESS (errors)	22.52 ± 4.46 (Range: 13-33) [95%CI:	19.45 ± 6.47 (Range: 8-31) [95%CI:	20.00 ± 5.66 (Range: 10-33) [95%CI:	19.93 ± 6.41 (Range: 6-29) [95%CI:	20.37 ± 5.89 (Range: 6-33) [95%CI:	1.361	0.259	0.038

	20.59-24.45]	16.99-21.91]	17.85-22.15]	17.39-22.46]	19.25-21.49]			

Data were not normally distributed (Shapiro Wilk $p<0.05$); thus, a Kruskal-Wallis H test was performed to compare group medians for all data except for K-D and BESS (Shapiro Wilk $p>0.05$), so ANOVA was ran). Data reported as means $\pm$ SD.

Effect of Group/Sport Participation on Gait Outcomes

Means, standard deviations, range, and 95% confidence intervals (CI) for each gait variable of interest (i.e., gait speed, stride length (SL), double support (DBS), tandem) and their associated DT outcomes are presented in Table 11. Kruskal-Wallis values are reported for non-normal data and ANOVA values are reported for normally distributed variables. The calculated DTC for each of these outcomes are reported in Table 12.

There were no significant differences between groups for DT gait speed, ST stride length, or DT stride length. There was a significant difference between groups for ST tandem gait time ($p=0.002$). Post hoc analyses revealed a statistically significant difference between NON and HRS (Adjusted $p=0.001$). There was a significant difference between groups for DT tandem gait ($p=0.007$) with a post hoc revealing significance differences between NON and HRS ($p=0.007$). There was a significant difference between groups for ST gait speed, but post-hoc analyses indicated no significant differences between groups (Adjusted $p>0.05$). There was a significant difference between groups for ST double support with post hoc analyses revealing a significant difference between RUG and NON ($p=0.031$) and HRS ($p=0.029$). Lastly, there was a significant difference between groups for DT double support with a post-hoc revealed significant difference between HRS and RUG ($p=0.011$). (Table 11)

There were no significant differences between groups for the DTC values for all gait outcomes ($p>0.05$). (Table 12)

Table 11 Single and Dual-Task Gait Outcomes by Group

	Group 1 (NON)	**Group 2 (NCA)**	**Group 3 (HRS)**	**Group 4 (RUG)**	**Overall**	**Kruskal-Wallis Test $X^2(3)$ Value/ANOVA F Value**	**P-Value**	**Effect Size (Eta Squared)**
N	28	29	29	27	113	N/A	N/A	N/A
ST Tandem Gait (s)	18.84 ± 3.57‡ (Range: 14.24-26.63) [95%CI: 17.45-20.22]	16.30 ± 3.26 (Range: 10.40-23.00) [95%CI: 15.06-17.54]	15.11 ± 2.75* (Range: 9.12-20.78) [95%CI: 14.06-16.15]	16.54 ± 2.93 (Range: 11.52-22.03) [95%CI: 15.36-17.72]	16.68 ± 3.39 (Range: 9.12-26.63) [95%CI: 16.05-17.32]	15.524	0.002	0.123
DT Tandem Gait (s)	26.94 ± 6.61‡ (Range: 16.62-49.07) [95%CI: 24.38-29.50]	23.09 ± 6.11 (Range: 12.76-37.09) [95%CI: 20.78-25.42]	21.48 ± 4.42* (Range: 12.06-29.13) [95%CI: 19.80-23.16]	22.23 ± 5.05 (Range: 13.08-30.93) [95%CI: 20.13-24.27]	23.44 ± 5.93 (Range: 12.06-49.07) [95%CI: 22.33-24.55]	11.993	0.007	0.091
ST Gait Speed (m/s)	1.07 ± 0.16 (Range: 0.83-1.59) [95%CI: 1.01-1.14]	1.13 ± 0.15 (Range: 0.86-1.45) [95%CI: 1.07-1.19]	1.12 ± 0.15 (Range: 0.83-1.45) [95%CI: 1.07-1.18]	1.03 ± 0.12 (Range: 0.83-1.59) [95%CI: 0.98-1.08]	1.09 ± 0.15 (Range: 0.83-1.59) [95%CI: 1.06-1.12]	10.103	0.018	0.074

DT Gait Speed (m/s)	0.88 ± 0.18 (Range: 0.57-1.49) [95%CI: 0.81-0.95]	0.94 ± 0.20 (Range: 0.56-1.38) [95%CI: 0.86-1.02]	0.98 ± 0.17 (Range: 0.64-1.44) [95%CI: 0.91-1.04]	0.89 ± 0.15 (Range: 0.51-1.26) [95%CI: 0.83-0.95]	0.92 ± 0.18 (Range: 0.51-1.49) [95%CI: 0.89-0.95]	7.451	0.059	0.050
ST Stride Length (m)	1.14 ± 0.12 (Range: 0.95-1.45) [95%CI: 1.09-1.18]	1.16 ± 0.12 (Range: 0.95-1.42) [95%CI: 1.12-1.21]	1.17 ± 0.09 (Range: 0.96-1.37) [95%CI: 1.13-1.20]	1.11 ± 0.09 (Range: 0.94-1.27) [95%CI: 1.07-1.14]	1.14 ± 0.11 (Range: 0.94-1.45) [95%CI: 1.12-1.16]	1.935	0.128	0.051
DT Stride Length (m)	1.04 ± 0.13 (Range: 0.85-1.45) [95%CI: 0.99-1.09]	1.06 ± 0.14 (Range: 0.85-1.39) [95%CI: 1.00-1.11]	1.08 ± 0.10 (Range: 0.87-1.26) [95%CI: 1.04-1.12]	1.03 ± 0.11 (Range: 0.77-1.28) [95%CI: 0.98-1.08]	1.05 ± 0.12 (Range: 0.77-1.45) [95%CI: 1.03-1.08]	3.952	0.267	0.018
ST Double Support (%)	20.98 ± 3.12 (Range: 14.46-26.04) [95%CI: 19.77-22.19]	20.35 ± 2.89¥ (Range: 14.44-26.43) [95%CI: 19.25-21.44]	20.33 ± 2.65¥ (Range: 13.69-26.18) [95%CI: 19.32-21.34]	22.42 ± 2.73†‡ (Range: 17.30-27.11) [95%CI: 21.31-23.52]	21.00 ± 2.94 (Range: 13.69-27.11) [95%CI: 20.46-21.55]	3.452	0.019	0.087

DT Double Support (%)	23.98 ± 3.31 (Range: 14.91-31.01) [95%CI: 22.70-25.26]	23.41 ± 4.05 (Range: 14.73-31.00) [95%CI: 21.87-24.95]	22.912 ± 2.96¥ (Range: 13.23-30.07) [95%CI: 20.99-23.25]	24.81 ± 2.42‡ (Range: 20.85-28.80) [95%CI: 23.83-25.79]	23.57 ± 3.36 (Range: 13.23-31.01) [95%CI: 22.94-24.19]	3.539	0.17	0.089

Data were not normally distributed (Shapiro Wilk $p<0.05$), thus a Kruskal-Wallis H test was performed to compare group medians for demographic data. An ANOVA was ran for ST stride length, ST double support, and DT double support since data were normal (Shapiro-Wilk $p>0.05$). Data reported as means ± SD. *: significant difference from NON. †: significant difference from NC. ‡: significant difference from HRS. ¥: significant difference from RUG.

Table 12 Gait Outcomes DTC by Group

	Group 1 (NON)	**Group 2 (NCA)**	**Group 3 (HRS)**	**Group 4 (RUG)**	**Overall**	**Kruskal-Wallis Test $X^2(3)$ Value/Mann-Whitney U Value**	**P-Value**	**Effect Size (Eta Squared)**
N	28	29	29	27	113	N/A	N/A	N/A
Tandem Gait DTC (%)	43.59 ± 24.85 (Range: 7.47-105.57) [95%CI: 33.95-53.23]	42.29 ± 27.89 (Range:-3.89-103.84) [95%CI: 31.68-52.90]	44.47 ± 32.16 (Range: 2.02-149.31) [95%CI: 32.23-56.70]	35.36 ± 27.87 (Range: 5.67-126.45) [95%CI: 24.10-46.62]	41.57 ± 28.19 (Range: -3.89,149.31) [95%CI: 36.29-46.85]	3.246	0.355	0.002
DTC Double Support (%)	15.10 ± 10.61 (Range: -2.4-38.09) [95%CI: 10.99-19.21]	15.67 ± 12.41 (Range: -2.13-50.48) [95%CI: 10.5-20.39]	11.56 ± 10.07 (Range: -3.35-30.12) [95%CI: 7.73-15.39]	11.88 ± 10.11 (Range: -1.0-45.75) [95%CI: 7.8-15.96]	13.56 ± 10.83 (Range: -3.35,50.48) [95%CI: 11.54-15.58]	2.908	0.406	0.001
DTC Gait Speed (%)	-17.04 ± 11.51 (Range: -47.36,1.71)	-15.96 ± 12.58 (Range: -46.94,2.62)	-11.69 ± 8.67 (Range: -26.54,5.58)	-12.64 ± 10.13 (Range: -46.67,5.89)	-14.49 ± 10.97 (Range: -47.36,5.89)	2.957	0.398	<0.001

	[95%CI: -21.5,-12.57]	[95%CI: -20.74,-11.17]	[95%CI: -14.98,-8.39]	[95%CI: -16.74,-8.55]	[95%CI: -16.54,-12.45]			
DTC Stride Length (%)	-8.18 ± 6.34 (Range: -22.88,3.61) [95%CI: -10.64,-5.72]	-9.05 ± 6.43 (Range: -20.92,1.96) [95%CI: -11.5,-6.6]	-7.13 ± 5.16 (Range: -15.67,2.75) [95%CI: -9.09,-5.16]	-7.05 ± 6.63 (Range: -27.9,5.28) [95%CI: -9.73,-4.37]	-7.89 ± 6.10 (Range: -27.9,5.28) [95%CI: -9.02,-6.75]	0.651	0.584	0.018

Data were not normally distributed (Shapiro Wilk $p<0.05$), thus a Kruskal-Wallis H test was performed to compare group medians for demographic data. An ANOVA was ran for DTC SL since data were normal (Shapiro-Wilk $p>0.05$).
Data reported as means ± SD.

One-Way ANCOVAs: Psychological Variables

A one-way ANCOVA was performed to compare the relationship between each dependent variable (i.e., psychological variables, gait variables, clinical outcomes, and DTC for gait variables) when controlling for concussion and LD/ADHD history, sex, age, and career duration. Results are reported below in Table 13. Adjusted means and standard errors are provided below for significant outcomes unadjusted values can be found in Table 9.

After adjustment for age, LD/ADHD history, concussion history, and career duration, there was a statistically significant difference in SF-12 (PCS), AES-S, SWLS, and MOCA between groups.

Post-hoc analysis revealed SF-12 (PCS) was significantly higher in the NCA group (adjusted mean: 56.369 $\pm$ 0.949) compared to the NON group (adjusted mean: 52.911 $\pm$ 0.972) ($p=0.030$) and significantly greater AES-S score in the NON group (adjusted mean: 28.786 $\pm$ 1.264) compared to the NCA group (adjusted mean: 24.360 $\pm$ 1.234) ($p=0.034$). Post-hoc observed differences revealed a greater SWLS in the NCA group (adjusted mean: 30.136 $\pm$ 1.104) compared to the NON group (adjusted mean: 25.987 $\pm$ 1.131) ($p=0.023$) and greater MOCA scores in the NCA group (adjusted mean: 27.727 $\pm$ 0.339) compared to the NON group (adjusted mean: 26.442 $\pm$ 0.347) ($p=0.021$). There were no significant differences between groups for SF-12 (MCS) or BSSS ($p>0.05$).

Table 13 One-Way ANCOVA: Psychological Variables

Measure	F Value	p-value	η_p^2	Observed Power	Post-hoc differences
SF-12 (PCS)	3.365	0.021*	0.088	0.748	NCA>NON
SF-12 (MCS)	1.585	0.197	0.044	0.407	N/A
AES-S	4.107	0.008*	0.106	0.836	NON>NCA
SWLS	4.740	0.004*	0.120	0.889	NCA>NON
BSSS	0.514	0.673	0.015	0.152	N/A
MOCA	3.111	0.030*	0.082	0.711	NCA>NON

*: indicates significance at the 0.05 level with adjustment for multiple comparisons.

One-Way ANCOVAs: Concussion Battery Variables

Concussion battery variable ANCOVA results are shown below. (Table 14) Adjusted means and standard errors are provided below for significant outcomes unadjusted values can be found in Table 10. After adjustment for covariates, there was a statistically significant difference between groups for TrailsDiff, TrailsRatio, and BESS.

Post hoc test revealed no significant individual group differences ($p>0.05$) for TrailsDiff. However, there was a significantly greater TrailsRatio in RUG (0.607 ± 0.034) compared to NON (0.457 ± 0.032) ($p=0.029$). For BESS, RUG (17.952 ± 1.120) ($p=0.005$) and NCA (20.235 ± 1.077) ($p=0.040$) groups had statistically less errors than the NON group (24.186 ± 1.179). There were no significant differences between groups for SCAT5 Symptom Severity, TMT-A, TMT-B, or King-Devick.

Table 14 One-Way ANCOVA: Concussion Battery Variables

Measure	F Value	p-value	η_p^2	Observed Power	Post-hoc differences
SCAT5 Symptom Severity	2.467	0.066	0.067	0.598	N/A
TMT-A	2.660	0.052	0.071	0.635	N/A
TMT-B	2.314	0.080	0.063	0.568	N/A
TrailsDiff	2.954	0.036*	0.079	0.686	None.
TrailsRatio	2.912	0.038*	0.077	0.679	NON<RUG
King-Devick	1.107	0.350	0.031	0.291	N/A
BESS	4.633	0.004*	0.123	0.880	NON>NCA; NON>RUG

*: indicates significance at the 0.05 level with adjustment for multiple comparisons.

One-Way ANCOVAs: ST & DT Gait Variables

Gait variable ANCOVA results are shown below. Covariates included age, sex, concussion and LD/ADHD history, career duration, and height. (Table 15) Adjusted means and standard errors are provided below for significant outcomes unadjusted values can be found in Table 11. A significant effect for group existed for ST and DT tandem gait, ST gait speed, and ST and DT double support.

Post-hoc tests showed that the NON group (18.624 $\pm$ 0.649) had greater ST tandem gait times than both the NCA (16.279 $\pm$ 0.635) (p=0.026) and the HRS groups (15.473 $\pm$ 0.629) (p=0.012). For DT tandem gait the NON group (27.433 $\pm$ 1.141) had greater (i.e., slower) times than the HRS (21.406 $\pm$ 1.107) (p=0.005) and the RUG group (21.000 $\pm$ 1.218) (p=0.005). There was a significant effect of group for ST gait speed (p=0.030), but there were no post-hoc group differences (p>0.05). There was a significant effect of group for ST double support (p=0.038), but there were no post-hoc group differences (p>0.05). DT double support had post-hoc difference between

HRS (22.765 ± 0.674) and RUG (25.292 ± 0.726) (p=0.031). There were no significant group differences for DT gait speed, ST stride length, or DT stride length (p>0.05).

Table 15 One-Way ANCOVA: Gait Variables

Measure	F Value	p-value	η_p^2	Observed Power	Post-hoc differences
ST Tandem Gait (s)	4.391	0.006*	0.114	0.861	NON>NCA; NON>HRS
DT Tandem Gait (s)	5.081	0.003*	0.130	0.911	NON>HRS; NON>RUG
ST Gait Speed (m/s)	3.434	0.030*	0.083	0.708	None.
DT Gait Speed (m/s)	2.091	0.161	0.049	0.445	N/A
ST Stride Length (m)	2.327	0.114	0.056	0.508	N/A
DT Stride Length (m)	1.513	0.293	0.036	0.328	N/A
ST Double Support (%)	3.012	0.038*	0.079	0.677	None.
DT Double Support (%)	3.020	0.039*	0.078	0.674	HRS<RUG

*: indicates significance at the 0.05 level with adjustment for multiple comparisons.

One-Way ANCOVAs: DTC Gait Variables

DTC for gait variable ANCOVA results are shown below. Covariates included age, sex, concussion and LD/ADHD history, career duration, and height. (Table 16) There were no significant differences between groups when covariates were included in the model (p>0.05).

Table 16 One-Way ANCOVA: DTC for Gait Variables

Measure	F Value	p-value	η_p^2	Observed Power	Post-hoc differences
Tandem Gait DTC (%)	1.929	0.130	0.054	0.485	N/A
DTC Double Support (%)	0.099	0.961	0.003	0.067	N/A
DTC Gait Speed (%)	0.179	0.911	0.005	0.082	N/A
DTC Stride Length (%)	0.230	0.875	0.007	0.092	N/A

*: indicates significance at the 0.05 level with adjustment for multiple comparisons.

Clinical Outcomes & DTC:

Results of the Spearman correlations for both ST gait speed and DTC gait speed and clinical outcomes are presented below in Table 17.

There was a statistically significant, low negative correlation between ST gait speed and SCAT5 Symptom Severity (r_s (111) = -0.211, p=0.025) and TMT-B (r_s (111) = -0.219, p=0.021). There was a statistically significant, low positive correlation between ST gait speed and SWLS (r_s (111) = 0.282, p=0.002). All other clinical outcomes were not statistically significantly correlated with ST gait speed (p>0.05).

There was a statistically significant, low negative correlation between DTC gait speed and TMT-A (r_s (111) = -0.216, p=0.022) and TMT-B (r_s (111) = -0.306, p<0.001). There was a statistically significant, low positive correlation between DTC gait speed and MOCA (r_s (111) = 0.248, p=0.008). All other clinical outcomes were not statistically significantly correlated with DTC gait speed (p>0.05).

Table 17 Spearman Correlation: Clinical Outcomes and ST and DTC Gait Speed

	ST Gait Speed		**DTC Gait Speed**	
Clinical Measure	**Spearman's Rho (r_s)**	**p-value**	**Spearman's Rho (r_s)**	**p-value**
SF-12 PCS	0.015	0.875	0.012	0.901
SF-12 MCS	0.116	0.220	0.100	0.293
AES-S	-0.133	0.161	-0.055	0.562
Symptom Severity	-0.211	**0.025***	0.020	0.831
SWLS	0.282	**0.002***	0.156	0.100
BSSS	-0.066	0.490	0.025	0.793
TMT-A	-0.161	0.088	-0.216	**0.022***
TMT-B	-0.219	**0.021***	-0.306	**<0.001***
MOCA	0.167	0.077	0.248	**0.008***
K-D	-0.109	0.249	-0.097	0.307
BESS	0.055	0.571	0.105	0.279

*: indicates significance ($p<0.05$).

A summary of all significant findings and interpretations from Aim 2 can be found in Appendix J.

Discussion

RHI exposure may put individuals at risk for subtle acute and chronic neurophysiological changes and/or deficits;[114,147,151,246,299–302] however, research on the long-term effects into adulthood are mixed and limited in age and sport history, having primarily focused on either collegiate athletes in the short-term or middle aged to older adult men in the long term.[151,152,178,191,153,160,163,164,167,169,171,173] The purpose of this study was to expand on previous research in terms of age and sex, having multiple comparison groups, and the utilization objective clinical gait outcomes when investigating the effects of RHI from contact/collision sports on neurophysiological health in individuals with varying levels of lifetime RHI exposure and physical

activity levels. The primary finding of this study was that participation in contact/collision sports, including prolonged rugby participation into adulthood (i.e., after age 22), was not related to adverse outcomes in early to mid-adulthood. Moreover, those who were not physically active without a history of RHI exposure performed worse on some assessments of neurophysiological health (8/25 measures, 32%), representing multiple systems (e.g., cognition, quality of life, postural control), than physically active individuals, regardless of RHI exposure history. Consistent with emerging data among middle aged males who played football,[173,174,178] these results suggest that potential negative consequences of RHI exposure through contact/collision sports (if any), regardless of career duration or beyond the normal timeframe (i.e., rugby players participating past college age), do not manifest in early-mid adulthood in physically active individuals. Therefore, these findings add to the growing body of literature that contact/collision sport participation does not negatively affect neurophysiological health in early- to mid-adulthood, as remaining physically active, regardless of RHI exposure, may in fact be beneficial to long-term health compared to those who are physically inactive.

The main finding of this study was that career duration, as assessed by cumulative years of contact/collision sport participation, did not predict worse neurobehavioral function, as assessed by ST and DT instrumented gait, in current and former contact/collision sport athletes (mean age: 35.1 $\pm$ 11.1, 41.1% female). Although our findings did not confirm our hypothesis of worse gait function, the results herein expand upon Iverson et al.'s AFE study among populations of middle aged men with a history of playing football by including measures of career duration and gait.[171,173,178] However, this null finding is similar to that of middle aged men

whereby football career duration was not significantly related to current depression, cognitive dysfunction, or post-concussion like symptoms[171,174] or mild cognitive impairment diagnosis.[188] Further, amongst a larger sample of middle aged men who played high school football (N=435), career duration was not related to later life health conditions (e.g., memory loss, diabetes, headaches, psychological treatment), or current concussion-like symptoms (e.g., concentration problems, headaches), or depression symptoms.[173] Our findings herein agree with our previous finding among amateur rugby players,[201] whereby cumulative years of contact/collision sports was not related to worse patient reported measures of QoL, self-rated physical and mental health, or depression and anxiety among early to middle-aged adults. Taken together, data among early-mid adult collision sport athletes (i.e., ages 22-50) suggest that career duration does not seem to affect measures of self-reported cognitive functioning, mental health, and objective measures of neurobehavioral health (i.e., gait, a known factor associated with physical and mental health[89,90,259–261]); however, the later life effects remain unknown.

Although we did not confirm our hypothesis and failed to show an effect of career duration on neurobehavioral health in early to mid-life adults, career duration may still be an important factor for later life (i.e., $\geq$70 years) neurological health, as data from male football and soccer players suggest that career duration may be related to neurodegenerative disease risk as well as mortality.[166,303,304] Interestingly, despite former NFL players having lower mortality rates compared to the general population, those who participated in a greater number of games had higher mortality rates than those who played in fewer games, suggesting a lifetime effect of RHI exposure.[189] As such, career duration remains a metric of interest for long term health, given the

NINDS TES statement recommending its use as a metric for RHI exposure.[191] Similarly, post-mortem data of football players noted a dose-response relationship between career duration and CTE odds existed (30% increased odds for each additional year played, but the baseline risk of CTE is unknown),[303] yet, career duration was not associated with CTE severity.[166] This is similar to data from former male Scottish soccer players whereby a career duration ≥15 years was associated with the highest risk for neurodegenerative disease compared to age, sex, and socioeconomic status (SES) matched general population controls.[304] Of note, 41.1% of our cohort of former and prolonged collision athletes (HRS and RUG) had career durations ≥15 years, with an overall average career duration of 15.4 years. Collectively, our null findings paired with previous research suggest that career duration, regardless of AFE to contact/collision sport, appears to not affect neurophysiological health in early to mid-adulthood,[152,173,174,188,201] but it may be an important consideration for long-term neurological health in older adults (i.e., 70+ years old).[166,303,304]

One of the largest limitations of research investigating the long-term effects of RHI on neurophysiological health has been the lack of comparison groups and age, being limited to primarily cohort data of college aged or middle ages males.[114,147,151,171,178,191,246,303] By incorporating comparison groups of similar age and varying minimal levels of physical activity, we created a unique insight into the potential effects of RHI into early-mid adulthood. Contrary to our hypothesis, a secondary finding of this study was that physically active individuals with a history of RHI (HRS and RUG) did not perform worse on 25 different assessments of neurological, psychological health, cognition, and gait than those who are physically

active without a history of RHI exposure (i.e., NCA); interestingly, those who were not physically active without a history of RHI exposure (i.e., NON) performed worse on 6/25 assessments compared to those who are physically active without a history of RHI (i.e., NCA). After adjusting for known covariates, including contact sport career duration, the rugby group did not perform worse than non-contact athletes or former contact/collision sport athletes on any assessment ($p>0.05$). (Tables 13-16). This finding implies that any neurophysiological consequences of RHI exposure may not manifest in early to mid-adulthood (i.e., ages 22-50) which is interesting as cognitive decline has been noted to start in middle age (ages 45-59).[305] Of note, our mean age was ~35 years old, with only 18.6% of our sample over the age of 45, thus cognitive decline may not be a large contributor to our findings herein.[305] However, this finding may be the result of the neuroprotective effects of exercise outweighing the potential negative effects of RHI exposure.[250,258,306] Indeed, head injury has been associated with increased risk of incident dementia in a dose responsive manner even after controlling for physical activity levels; however this did not take into account RHI and utilized self-reported head injury that warranted physician/hospital care which is more likely a mild-moderate TBI.[118] Further there exists a significant increased odds of stroke among former NFL players with a history of 10 or more concussions, highlighting the potential link between vasculature damage as a consequence of head injury and RHI and cerebrovascular health.[307] However, former NFL players had overall lower prevalence of stroke compared to the general population which may be a result of the cumulative effects of increased levels of lifetime physical activity.[307] This may partially explain our lack of group differences as the HRS and RUG groups were

physically active, whereby the chronic benefits of exercise may have hid the negative consequences, if any, of RHI.

It is well established that healthy lifestyle choices (e.g., diet and exercise) and reductions in cardiovascular risk factors (e.g., cholesterol, lipids, hypertension, diabetes) and their levels at mid-life are important for lifelong dementia risk.[305] Indeed a dose-response relationship was found whereby individuals with greater measured physical activity levels had a lower risk of dementia compared to those with the lowest level of physical activity, even after accounting for comorbidity, lifestyle, and SES factors.[308] This is in line with the consensus that "what is good for our hearts is also good for our heads",[305,309,310] given the known benefits of exercise and physical activity on mood, anxiety and depression, dementia, cardiovascular disease, metabolic syndrome, and other noncommunicable diseases.[250,311] Therefore, the fact that neither RHI exposed group (HRS and RUG) performed worse on any outcome than the NCA group may be the result of the neuroprotective and beneficial effects of exercise and regular physical activity and the fact that these participants were in their mid-30's. Additionally, the fact that the physically inactive group performed worse than the NCA group on 6 different outcomes across multiple domains of health (i.e., SF-12 (PCS), AES-S, SWLS, MOCA, BESS, ST tandem gait) despite never experiencing RHI through contact/collision sports further highlights the detrimental effects of physical inactivity and presumably low levels of cardiorespiratory fitness which have been linked as a major risk factor for many diseases (e.g., cardiometabolic disease, all-cause deaths, systemic inflammation, stress, anxiety, Alzheimer's Disease).[250,312,313]

It is noteworthy to mention that although there were not many group differences (Tables 13-16), there were a considerable proportion of participants within

each group with abnormal psychological scores, or scores below clinical cut points (Table 9-11), as summarized below. (Table 18)

Table 18 Summary of Abnormal Psychological Scores by Group

	Group 1 (NON)	**Group 2 (NCA)**	**Group 3 (HRS)**	**Group 4 (RUG)**
N	**28**	**29**	**29**	**27**
SF-12 (PCS) Abnormal (<50)	5/28 (17.9%) abnormal	0/29 (0%) abnormal	1/29 (3.4%) abnormal	4/27 (14.8%) abnormal
AES-S Abnormal (≥34)	5/28 (17.9%) abnormal	1/29 (3.4%) abnormal	4/29 (13.8%) abnormal	4/27 (14.8%) abnormal
SWLS Abnormal (<20)	4/28 (14.3%) abnormal	1/29 (3.4%) abnormal	0/29 (0%) abnormal	5/27 (18.5%) abnormal
MOCA Abnormal (<26)	6/28 (21.4%) abnormal	2/29 (6.9%) abnormal	5/29 (17.2%) abnormal	4/27 (14.8%) abnormal

For instance, we observed the NON group self-reported significantly lower SF-12 (PCS) scores in the adjusted models than the NCA group. As evidenced in the above table, the NON and RUG groups had a nontrivial proportion of participants below the population average of 50, whereas the NCA group had no one below the population average score.[314] Additionally, clinically meaningful apathy is considered a score of 34 or greater for the AES-S; herein, the NON, HRS, and RUG groups had ~15% of their participants with clinically meaningful apathy compared to only 3% in the NCA group.[169,280] In regards to lower SWLS, ~1/6 of the NON and RUG groups reported scores below 20/35, indicating dissatisfaction with life, compared to only a small, if any, proportion of the NCA and HRS groups.[232,315] For cognition, assessed by the MOCA, we reported adjusted group differences between NCA and the NON group (Table 13), however, nearly 1/5 of the NON, HRS, and RUG groups had scores below

26, a clinical cutoff used to differentiate mild cognitive impairment from normal cognitive functioning individuals.[283] Herein lies a unique trend. Although most of our group differences showed worse performance in the NON group compared to the NCA group in the adjusted models, signifying that former contact/collision sport athletes (i.e., HRS and RUG) are not performing worse than physically active individuals who never experienced RHI, there is still a significant proportion of individuals in the NON, HRS, and RUG groups with clinically meaningful dysfunction. Although the progression of this dysfunction cannot be determined from these data, it highlights a clear trend that there do exist individuals in these cohorts with worse performance compared to their peers that warrants future investigation to determine what their late life function entails.

Despite failing to support our hypothesis, our data highlight the lack of effects of RHI and prolonged RHI exposure in early to mid-adulthood on measures of neurophysiological health among physically active individuals. Further, the differences between the NON group and NCA group further highlight the detrimental effects of physical inactivity and the beneficial effects of physical activity.

Gait speed is a strong predictor of physical and mental health due to its ability to correlate with various domains of health, wellness, and morbidity.[89,90,259–263] A tertiary purpose of this study was to expand insights into the relationship between gait and neurophysiological health by including measures of RHI exposure (i.e., career duration). Our hypothesis that slower ST gait speed and worse gait speed DTC would be related to worse patient-reported outcomes was partially supported in some findings (SCAT5 Symptom Severity, SWLS, MOCA, TMT-A, TMT-B), but overall most outcomes did not support our hypothesis. In terms of psychological findings, ST gait

speed with weakly negatively correlated with SCAT5 symptom severity whereby higher symptom severity was associated with slower gait speeds (r_s= -0.211, p=0.025) but not DTC gait speed (r_s= 0.020, p=0.831). In line with our hypothesis, the significant relationship between ST gait speed and symptom severity is consistent with findings among individuals with chronic mTBI whereby they walked significantly slower under ST and gait performance worsened with symptom severity.[269] However, the lack of relationship between symptom severity and DTC gait speed is interesting as DT gait requires additional attentional resources, which in theory would be hindered by existing symptom severity. In turn, this may be the result of low levels of baseline symptom severity among our participants (Table 9). Yet, this finding is supported by data from collegiate student athletes (noncontact/contact/collision) whereby the presence of symptoms were unrelated to DTC gait speed (ES: 0.25, p>0.05).[316] SWLS was also weakly positively correlated with ST gait speed (r_s= 0.282, p=0.002) whereby a faster gait speed (i.e., higher number) was associated with a greater SWLS (i.e., better SWLS). This finding is somewhat consistent with the literature showing that gait speed, as a measure of overall health, is strongly correlated with quality of life; in particular slow gait speeds are associated with lower quality of life.[259,261,317] However, the lack of strength in the relationship herein may be the result of differing subject populations (e.g., young college students, older adults) and potential negative effects of the COVID-19 pandemic on one's mental health.[318–320] Overall, these findings demonstrated that measures of psychological health are weakly correlated with neurobehavioral health (i.e., gait) in these populations, highlighting the continued need for inclusion of measures of psychological well-being in gait studies.

In terms of cognitive outcomes, our hypothesis was supported whereby TMT-B was significantly negatively correlated with both ST gait speed (r_s= -0.219, p=0.021) and DTC gait speed (r_s= -0.306, p<0.001), implying worse gait speeds (i.e., a lower number/slow gait speed) and worse DTC (i.e., more negative number/greater change between ST and DT) with higher TMT-B times (i.e., worse). Additionally, our hypothesis was supported whereby TMT-A was weakly negatively correlated with DTC gait speed (r_s= -0.216, p=0.022); whereby an increase in TMT-A time (worse) was associated with lower DTC for gait speed (i.e., more negative, worse). The significant relationships herein between ST and DTC gait speed and Trail Making Test A & B are consistent with the literature as gait speed is highly correlated to executive function.[88,89] For instance, poor and intermediate performance (i.e., slower times) on TMT tests have been associated with reduced gait speed under complex gait conditions such as obstacle crossing.[89,321] The final significant finding was that there was a low positive correlation between MOCA and DTC gait speed; whereby an increase in MOCA score (i.e., improvement) was associated with an increase in DTC for gait speed (i.e., a more positive number, improvement, and less discrepancy between ST and DT). Similar to the relationship between TMT-A and TMT-B, the relationship between improved MOCA and improved DTC for gait speed is in line with the literature as greater MOCA scores indicate higher cognitive ability and executive functioning; additionally, improved DTC represents an ability to appropriately manage two concurrent tasks and executive function skills.[88,89,283] However, it is important to note that overall MOCA scores in this cohort were generally normal with a small range across the sample (Table 9), which may have caused lack of strength of this finding. Taken together, these findings add to the

growing body of literature that highlights the large cognitive component of gait and dual task gait.

Lastly, counter to our hypothesis, SF-12 (PCS & MCS), AES-S, BSSS, King-Devick, or BESS did not significantly correlate with ST gait speed and DTC gait speed. Although these measures can be used to assess determinants of physical and mental health they may not necessarily correlate with physical function (i.e., gait speed). Worse SF-12 PCS and MCS scores have been self-reported in individuals with slowed walking speeds due to neurological conditions.[322] The lack of relationship found herein may be the result of a wide range of SF-12 PCS (30.7-65.0) and MCS scores (22.6-61.3) in which a significant amount of participants reported scores below population norms (50).[231,314] Similarly, there was a significant proportion of individuals with clinically meaningful apathy (AES-S $\geq$34) and a large range among the sample (18-56). The heterogeneity of these scores may be the result of COVID-19 pandemic related changes in mental and physical health which may explain the lack of relationship between SF-12, AES-S, and gait speed observed in our study.[318,319] It is interesting there was not a relationship for King-Devick, a measurement of horizontal saccadic eye function, as saccadic abnormalities have related to gait abnormalities in neurologic populations.[323] However, this finding may be due in part to the normal neurological status of our participants. Lastly, BESS scores did not correlate with gait speed outcomes. BESS is a measure of posture stability which in theory should correlate to postural control.[287] However, the BESS test has poor reliability and is vulnerable to inconsistent scoring by the tester;[287] despite having the same researcher score the BESS test in this study, scoring is subjective and may have affected these outcomes and explain the lack of hypothesis support herein. These data showed that

gait speed did not correlate with various measures of physical and mental health. Future research should continue utilizing these various measures of physical and mental health in addition to a multifaceted neurological screening to fully encompass the relationship between RHI and health.

A primary limitation of this study included the participants' wide age range (18-67 years) in addition to range for career duration (0-47 years) providing a very heterogeneous sample which may contribute additional confounders that were not accounted for (e.g., socioeconomic status, early childhood events, etc.). Further, the use of career duration as a metric for RHI exposure is limited since we summed the total years, not seasons, of each contact/collision sport played. Therefore, an athlete who played ice hockey and football in the same year would have reported 2 years of contact/collision sports, potentially biasing our data. However, this method has been previously utilized and provides a simple metric to quantify RHI exposure beyond the capabilities of AFE as an RHI exposure metric.[201] Further, the TES statement suggests the use of career duration over AFE as a measurement of cumulative RHI exposure since there are no known thresholds for contact/collision sports outside of American football.[191] Additionally, the prolonged RHI exposure group (RUG) did not report any clinically significant group differences from the primary comparison group (NCA—no RHI exposure), implying a lack of effects of prolonged RHI on neurophysiological function in early adulthood. However, this may be the result of survivor or respondent bias as the players who had neurophysiological dysfunction may have removed themselves from participation in contact/collision sports or failed to volunteer for the study, whereas the healthy and resilient individuals continued to participate in sport and subsequently volunteered for study participation. Lastly, this study relied on self-

reported measures of physical activity as a dichotomous outcome (yes/no for meeting ACSM physical activity guidelines). Although self-report measures of physical activity have been used in previous research,[324] it may fail to account for the accuracy and precision needed to truly detect the effect of exercise (i.e., frequency, intensity, type, time) on the included outcomes. Further, this study wasn't intended to determine the effect of exercise on neurophysiological health and future investigations should utilize validated measures of physical activity to determine the effects of physical activity levels as a moderator for the relationship between RHI exposure and neurophysiological health in mid to late adulthood. Ultimately, these findings herein provide merit to the NINDS TES call to action for more research across the lifespan investigating the prolonged effects of RHI across sporting groups.

This study identified the lack of effect of prolonged RHI exposure and lifetime RHI exposure, as assessed by career duration, on multiple domains of neurobehavioral functioning in early- to middle-aged adults. Some limited significant differences were observed between groups on measures of neurological health, mental health, and gait and balance; however, these differences were primarily limited to differences between individuals with no history of RHI who were either physically active (NCA) or not physically active (NON). Those who were not physically active (NON) performed significantly worse on 6/25 measures of neurophysiological health compared to those who were physically active without a history of RHI exposure through sport (NCA), in addition to performing worse than contact sport athletes on 2 other measures. The lack of differences observed among physically active RHI exposed groups (RUG and HRS) highlights the potential neuroprotective effects of lifetime physical activity and/or the inability for any neurological detriments to manifest in early to mid-adulthood.

Therefore, our findings suggest that the risk of neurological dysfunction and injury from RHI exposure and contact/collision sports may be outweighed by the neuroprotective benefits and effects of lifelong physical activity.

Chapter 4

EFFECT OF SEX ON THE RELATIONSHIP BETWEEN PROLONGED RHI AND DYNAMIC POSTURAL CONTROL AND PATIENT REPORTED OUTCOMES

Introduction

It is well established that males and females have different responses to neurotrauma.[22,325,326] In particular, females tend to have worse outcomes (e.g., executive and cognitive dysfunction, greater symptom count),[22,327,328] longer symptom burden compared to age matched males,[23] and more persistent cognitive and somatic symptoms than males.[329] Yet, more recent cohort data from the NCAA-DoD CARE Consortium reported no sex differences in concussion recovery; yet, a multitude of intrinsic and extrinsic factors may contribute to previous sex differences reported in the literature.[23] Thus, sex should still be considered a factor in TBI research, and the effects of sex on outcomes from repetitive neurotrauma remain to be elucidated. Further, animal models reported that females experienced worse outcomes (evidenced by behavioral changes and mRNA expression) to repetitive mTBI when performing both motor and balance tasks.[330] In addition to differing responses to TBI and repetitive head impacts (RHI), females also have different neurodevelopmental timelines (from adolescence to adulthood) compared to males;[154–156] these differences are important to consider for sport given Title IX efforts to increase female athlete participation[331] as well as differing AFE to contact/collision sports whereby females tend to start at a later age.[201] The long term effects of sex on RHI are further confounded given the fact that females have higher rates for certain neurodegenerative disease (i.e., Alzheimer's and dementia)[332] as well as different physiological effects of aging (e.g., rate, functional capabilities, effect of sex hormones).[333,334] It is theorized

that sex differences in successful ageing arise from intrinsic factors such as reproductive physiology, sex hormones, and genes in addition to extrinsic factors such as exercise/physical activity, lifestyle habits, and nutrition.[334] Furthermore, there exist differences by sex for aging in regards to neuroanatomy (e.g., progressive decreases in brain volume affecting frontotemporal regions of the brain females less than males) and glucose metabolism (higher in males, but is known to decrease with age across sexes),[335] as well as the rate of aging—assessed by five biomarkers (with males having faster rates, but females experiencing a sharp increase in rate around age 65).[334] Despite the biological differences across sex and differing responses to neurotrauma, the long-term effects of RHI are relatively unknown in females. Thus, a gap remains on the effects of RHI among females, and especially females who participated in collision sports such as rugby.

Although research in retired American football players has highlighted an association between RHI and later-in-life cognitive dysfunction, the studies are limited to male athletes.[153,167,169] Despite the fact that females make up a significant proportion of the population susceptible to concussion and other forms of neurotrauma (e.g., RHI) through sport, the research on the effects of sex (male vs. female) has received limited attention beyond college-aged individuals which may be confounded by the fact that most collision sports are male-dominated sports.[325,331] Females in contact sports suffer worse outcomes following concussions,[23,329] suffer more concussion than males in matched sports,[15,336] and may have a lower threshold for concussive head impact.[337] Yet, females have been vastly underrepresented in both concussion and RHI research.[325] Additionally, studies specifically investigating female collision sport athletes and/or sex-specific effects on the interaction between RHI exposure (i.e.,

career duration) and long-term health outcomes are limited.[164,191,338] As such, in line with the NINDS TES criteria recommendation, a need exists to identify the effects of sex on the relationship between RHI and mid-life neurobehavioral health.

Rugby provides an unparalleled insight into the effects of sex on the relationship between RHI and long-term health given that it is the only collision sport where females and males play by the same laws.[220] With roughly 25% of registered players and 20% of adult senior club players being females in the U.S., there exists a large population of women experiencing prolonged RHI beyond the typical timeline of adolescence through college (i.e., age 22).[216] Further, emerging data from a cohort of American college rugby players (of unknown rugby/collision sport career duration) has shown that males and females experience similar magnitude of impacts in rugby, females had greater DTCs of DT gait at baseline, and females reported more symptoms than males post-concussion.[339,340] Additionally, among this cohort, female rugby players exhibited a more conservative gait and greater DTCs post-concussion than concussed male rugby players.[339] Interestingly, despite suffering more impacts than females, males had no decrements in DT gait performance over the course of one season, whereas females showed improvements in gait performance which could be the result of physical activity and learning effects.[339] However, these data are limited to the course of one season of RHI in collegiate rugby players, thus, a need exists to determine the effects of prolonged RHI exposure on gait across sexes in older populations utilizing a metric of RHI exposure (i.e., career duration).

Healthy adult males have faster ST and DT tandem walking speeds from females,[267] with females demonstrating greater changes from ST to DT gait (slower gait speed, lower cadence, and shorter stride length) and lower cadence DTC than

males,[101] and males demonstrating greater gait speed and stride-to-stride variability than females during DT gait.[341] Combined with the limited data highlighting sex differences in postural control and post-concussion outcomes as well as animal models displaying worse outcomes in females as a result of repetitive neurotrauma,[330] it can be inferred that the effects of career duration/RHI on neurophysiological outcomes may differ by sex. Therefore, in line with the NIH policy on sex as a biological variable, this study aims to determine the relationship between sex, RHI, and postural control.

Although insights on retired American football players have highlighted an association between RHI and later-in-life cognitive dysfunction, the studies are limited to male athletes.[167] Despite exponential growth of females in rugby, comprising 2.7 million players globally,[203] rugby being the only law and sex-matched collision sport, the fact that females make up a significant proportion of sporting population susceptible to neurotrauma, and females may be at increased risk for long-term impairments from neurotrauma, investigations in the relationship between RHI and long-term outcomes among females is lacking. Therefore, this study is innovative in that it is one of the first studies to investigate the effects of sex on the relationship between RHI and neurophysiological functioning in male and female collision athletes providing critical insight into how RHI exposure affects females across their lifetime.

Specific Aim 3 Description

To determine the associations between sex, RHI exposure, and gait outcomes using a clinically feasible measure of instrumented gait.

H3.1: There will be a significant effect of sex for the relationship between career duration and gait outcomes.

H3.2: There will be a significant effect of sex for the relationship between career duration and DTCs.

Methods

Participants

Participants for Aim 3 will consist of the previous participants mentioned in Aim 2. (Table 6) Inclusion/exclusion criteria and IRB approval were the same for Aim 3 as it was a part of the same study in Aim 2.

Instrumentation and Procedures

Instrumentation and testing procedures for Aim 3 remained the same as those previously mentioned in Aim 2. Only the gait measures were used for analysis in this study.

Statistical Analysis

A linear regression was utilized to understand the effect of career duration and sex on gait outcomes (i.e., ST & DT gait speed, double support, stride length, and tandem gait, and their corresponding DTC outcomes). Further, a dummy variable was created to model the sex*career duration interaction and was also included in the

model alongside sex and career duration in Block 1 using the "Enter" method. A second block with potential confounding factors (i.e., concussion history, LD/ADHD history, age, height) was also performed. Linearity between career duration and gait outcomes were determined via visual inspection of the scatterplot (Appendix K). Independence of observations was confirmed statistically via the Durbin-Watson test. Significant outliers (i.e., $\pm$3 standard deviations from the residual) were removed (N= 0-6). Homoscedasticity was assessed using a visual inspection of the standardized residuals vs. standardized predicted values plot. Significance was set at 0.05 and all analyses were performed on SPSS v. 26 (SPSS Inc, Chicago, IL).

Results

The results for the linear regressions for ST and DT gait outcomes (Aim 3.1) are presented below in Table 19. The dummy variable, sex*career duration, was not associated with any ST or DT gait outcomes ($p>0.05$).

ST and DT Gait Speed

For ST gait speed and DT gait speed neither of the two models were statistically significant, indicating there was no sex by career duration interaction regardless of the inclusion of covariates.

ST and DT Stride Length

Both models were significant for ST stride length (Model 1: R^2=0.135, p=0.001; Model 2: R^2=0.169, p=0.006), but none of the added confounders in Model 2 added statistically to the model; only sex added significantly to each model. Both models were significant for DT stride length (Model 1: R^2=0.082, p=0.024; Model 2: R^2=0.164, p=0.008), with only sex in Model 1 (p=0.011) and concussion history in

Model 2 adding statistically to the model (p=0.007), whereby female sex had a -0.095m reduction in stride length, and individuals with a history of concussion had a 0.069m larger stride length.

ST and DT Double Support

Neither model was significant for ST or DT Double support, however, age added statistically to Model 2 for ST Double support (p=0.026) and DT Double support (p=0.009), whereby increases in age (years) was predictive of increases in double support (%).

ST and DT Tandem Gait

Both Model 1 (R^2=0.084, p=0.023) and Model 2 (R^2=0.190, p=0.002) were significant for ST Tandem gait. Sex was significant for Model 1 (p=0.019) and age was significant for Model 2 (p<0.001). Lastly, only Model 2 was significant for DT tandem gait (R^2=0.164) with height (p=0.037), age (p<0.001), and LD/ADHD history (p=0.048) adding statistically to the model.

Table 19 Linear Regression Outcomes for ST & DT Gait Outcomes

ST Gait Speed	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	0.334 (3)	0.009		0.801	0.415 (7)	0.025		0.798
Career Duration			-0.001 (0.002)	0.503			-0.002 (0.002)	0.365
Sex			-0.032 (0.036)	0.374			-0.047 (0.048)	0.334
Sex*CD			0.003 (0.003)	0.393			0.003 (0.003)	0.423
Height							-0.178 (0.228)	0.437
Age							0.000 (0.001)	0.822
LD/ADHD							0.026 (0.040)	0.511
Concussion History							0.032 (0.032)	0.326
DT Gait Speed	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	0.431 (3)	0.012		0.731	1.344 (7)	0.060		0.468
Career Duration			0.001 (0.002)	0.509			0.000 (0.002)	0.825
Sex			-0.016 (0.044)	0.708			-0.026 (0.058)	0.661
Sex*CD			0.001 (0.004)	0.840			0.000 (0.004)	0.946
Height							-0.189 (0.275)	0.492
Age							-0.001 (0.002)	0.423
LD/ADHD							0.037 (0.048)	0.439
Concussion History							0.074 (0.039)	0.060
ST Stride Length	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	5.650 (3)	0.135		**0.001***	3.043 (7)	0.169		**0.006***

Career Duration			-0.002 (0.001)	0.091			-0.002 (0.001)	0.052
Sex			-0.095 (0.024)	**<0.001***			-0.072 (0.032)	**0.027***
Sex*CD			0.003 (0.002)	0.210			0.003 (0.002)	0.222
Height							0.111 (0.151)	0.464
Age							-0.001 (0.001)	0.406
LD/ADHD							0.011 (0.026)	0.667
Concussion History							0.033 (0.021)	0.123
DT Stride Length	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	3.262 (3)	0.082		**0.024***	2.934 (7)	0.164		**0.008***
Career Duration			0.000 (0.001)	0.898			-0.001 (0.001)	0.433
Sex			-0.075 (0.029)	**0.011***			-0.045 (0.038)	0.236
Sex*CD			0.001 (0.003)	0.779			0.001 (0.003)	0.822
Height							0.106 (0.179)	0.555
Age							-0.001 (0.001)	0.294
LD/ADHD							0.019 (0.031)	0.533
Concussion History							0.069 (0.025)	**0.007***
ST Double Support	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	0.285 (3)	0.008		0.836	0.979 (7)	0.061		0.451
Career Duration			0.018 (0.033)	0.582			-0.003 (0.034)	0.938
Sex			-0.115 (0.709)	0.871			-0.022 (0.941)	0.981
Sex*CD			-0.033 (0.063)	0.602			-0.020 (0.063)	0.746
Height							-1.381 (4.453)	0.757
Age							0.056 (0.025)	**0.026***

LD/ADHD							0.359 (0.776)	0.644
Concussion History							0.675 (0.630)	0.286
DT Double Support	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	0.428 (3)	0.012		0.733	1.225 (7)	0.075		0.296
Career Duration			-0.027 (0.037)	0.467			-0.042 (0.039)	0.278
Sex			-0.186 (0.810)	0.819			-0.590 (1.068)	0.582
Sex*CD			-0.025 (0.072)	0.724			-0.011 (0.071)	0.874
Height							-3.812 (5.054)	0.452
Age							0.074 (0.028)	**0.009***
LD/ADHD							-0.136 (0.881)	0.877
Concussion History							0.125 (0.716)	0.862
ST Tandem Gait	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	3.301 (3)	0.084		**0.023***	3.495 (7)	0.190		**0.002***
Career Duration			-0.014 (0.036)	0.696			-0.038 (0.037)	0.310
Sex			1.891 (0.791)	**0.019***			1.327 (1.010)	0.192
Sex*CD			-0.086 (0.070)	0.220			-0.067 (0.067)	0.318
Height							-6.251 (4.775)	0.193
Age							0.096 (0.027)	**<0.001***
LD/ADHD							0.924 (0.853)	0.281
Concussion History							0.421 (0.680)	0.537
DT Tandem Gait	**Model 1 (Step 1)**				**Model 2 (Step 2)**			

	F(df)	R^2	B(SE)	p	F(df)	R^2	B(SE)	p
	0.781 (3)	0.021		0.507	2.909 (7)	0.164		**0.008***
Career Duration			-0.032 (0.065)	0.626			-0.079 (0.065)	0.227
Sex			0.729 (1.431)	0.611			-0.988 (1.796)	0.584
Sex*CD			-0.117 (0.126)	0.353			-0.090 (0.120)	0.455
Height							-17.927 (8.495)	**0.037***
Age							0.174 (0.048)	**<0.001***
LD/ADHD							3.042 (1.517)	**0.048***
Concussion History							1.244 (1.244)	0.306

*: denotes significance at P<0.05.

The results for the linear regression models for DTC for gait outcomes (Aim 3.2) are presented below in Table 20. None of the models were significant ($p>0.05$). However, concussion history added statistically to Model 2 ($p=0.019$) for DTC stride length, whereby individuals with a history of concussion had an associated 3.10 increase in DTC for stride length (%). Further, career duration added statistically to Model 1 ($p=0.044$) for DTC double support, whereby each increase in career duration years yielded a 0.24 reduction in DTC double support (%).

Table 20 Linear Regression Outcomes for Gait DTC

DTC Gait Speed	Model 1 (Step 1)				Model 2 (Step 2)			
	F(df)	R^2	B(SE)	p	F(df)	R^2	B(SE)	p
	1.269 (3)	0.034		0.289	1.443 (7)	0.088		0.196
Career Duration			0.213 (0.120)	0.079			0.169 (0.126)	0.182
Sex			0.715 (2.616)	0.785			0.830 (3.466)	0.811
Sex*CD			-0.117 (0.232)	0.615			-0.156 (0.231)	0.502
Height							-4.700 (16.405)	0.775
Age							-0.128 (0.091)	0.163
LD/ADHD							0.769 (2.861)	0.789
Concussion History							4.392 (2.323)	0.061
DTC Stride Length	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	1.297 (3)	0.034		0.279	1.548 (7)	0.094		0.159
Career Duration			0.126 (0.067)	0.061			0.086 (0.070)	0.220
Sex			0.845 (1.454)	0.563			1.408 (1.921)	0.465
Sex*CD			-0.130 (0.129)	0.315			-0.141 (0.128)	0.271
Height							-0.699 (9.095)	0.939
Age							-0.040 (0.050)	0.431
LD/ADHD							0.695 (1.586)	0.662
Concussion History							3.066 (1.288)	**0.019***
DTC Double Support	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	1.652 (3)	0.044		0.182	1.491 (7)	0.090		0.178

Career Duration			-0.241 (0.118)	**0.044***			-0.194 (0.124)	0.121
Sex			-1.812 (2.570)	0.482			-5.940 (3.417)	0.085
Sex*CD			0.070 (0.227)	0.757			0.054 (0.228)	0.812
Height							-22.661 (16.175)	0.164
Age							0.040 (0.090)	0.660
LD/ADHD							-2.026 (2.821)	0.474
Concussion History							-3.195 (2.290)	0.166
DTC Tandem Gait	**Model 1 (Step 1)**				**Model 2 (Step 2)**			
	F(df)	**R^2**	**B(SE)**	**p**	**F(df)**	**R^2**	**B(SE)**	**p**
	1.811 (3)	0.048		0.149	1.359 (7)	0.084		0.231
Career Duration			0.102 (0.307)	0.740			0.033 (0.325)	0.919
Sex			-10.692 (6.708)	0.114			-14.817 (8.936)	0.100
Sex*CD			-0.172 (0.591)	0.771			-0.129 (0.595)	0.828
Height							-43.576 (42.266)	0.305
Age							0.301 (0.237)	0.206
LD/ADHD							11.813 (7.548)	0.121
Concussion History							1.770 (6.017)	0.769

*: denotes significance at P<0.05.

Discussion

Responses to RHI/neurotrauma, neurodegenerative disease risk, as well as performance on gait and postural stability differ by sex, making sex a key variable of interest for investigations on RHI and concussion.[191,267,325,332–335,341,342] Thus, this study sought to investigate the effect of sex on the relationship between career duration and ST and DT gait performance in individuals with varying levels of physical activity and RHI exposure. Our hypothesis that there would be a significant effect of sex on the relationship between career duration and gait outcomes was not supported by our data. Our findings indicated that there was a lack of interaction between sex and career duration with any ST or DT gait outcomes ($p>0.05$). (Table 19) Further, there was not a significant interaction between sex and career duration on DTC for any gait outcomes. (Table 20) These findings imply that sex does not affect the relationship between career duration, a pseudo metric for lifetime RHI exposure, and gait in early to middle aged adults.

Although the literature on concussion and gait is well researched,[59,61,74,99,343] little attention has been paid to the chronic effects of RHI on gait across the lifespan. A 2021 review on RHI and postural control—which consisted of primarily short term studies—highlighted the heterogeneous results of studies on RHI and postural control, suggesting the need for more objective measures of clinical balance and studies to determine the chronicity of postural control changes as a result of RHI.[246] To our knowledge, this was the first study to investigate the relationship between RHI and gait in early-mid adults (i.e., age 30-50) across a variety of RHI exposure history. We did not find a relationship between sex and career duration on ST and DT gait outcomes. This was counter to our hypothesis, which was based on the fact that

females tend to perform worse on gait tasks than males, respond differently to concussions and RHI, and age at different rates than males (with the relationship between biological age and chronological age differing by sex across ages 20-90).[191,267,325,332–335,341,342] However, our finding herein implies that despite these intrinsic and extrinsic differences, males and females may respond similarly to RHI when accounting for RHI exposure (i.e., career duration).

Similar to studies on the long-term effects of RHI in middle-aged individuals, the literature on RHI and gait is primarily limited to male participant pools in addition to only short-term insights on RHI and gait. However, there have been studies on the long-term effects of concussion/concussion history on postural control which may provide some insight herein.[151,339] Our finding of no sex differences in relation to gait outcomes is in agreement with a short-term investigation among collegiate rugby players that did not find a relationship between RHI and deficits in gait performance across ST and DT measures.[339] Yet, there were some sex-specific differences, with females having higher DTC (i.e., worse) than males at baseline and pre-season. Males did not improve in gait metrics over the course of a season while females tended to improve gait performance (i.e., increased gait speed, decreased double support time, and increased stride length) assessed by ADPM IMUs, the same method we used, despite experiencing similar peak linear accelerations, albeit less total impacts, than males over the course of the season.[339] This is similar to post-season soccer data whereby females improved (i.e., got faster) their tandem gait times following a season's worth of soccer heading-related RHI; however, those with higher magnitude impacts (>98 g) improved less than those experiencing lower impact thresholds, suggesting the magnitude, and not frequency of impacts may be of more

importance.[151] As such, our metric of career duration, a metric more sensitive to/focused on frequency than magnitude, may explain why we did not see any sex differences. Collectively, these data suggest that a season's worth of RHI is not sufficient to impair gait, although the magnitude of impacts may be a factor, and in fact the improvements in gait may be a result of routine physical activity, a learning effect since improvements in concussion assessment tools are not uncommon over the course of a season, and/or the Hawthorne effect (i.e., modifying one's behavior due to being observed).[344,345] Thus, in line with recent CARE data,[23] we did not find any effect of sex, however, intrinsic and extrinsic factors related to sex (e.g., biology, medical access, training level, etc.) may remain contributing factors and sex should still be considered an important variable in gait research. Future research should incorporate more sensitive measures of postural control (i.e., integrated motion capture, gait initiation/termination) to determine if there are any long-term impairments in postural control from RHI exposure.

Expanding on the effect of the magnitude of impact, literature on the long-term effects of concussion/history of concussion on gait may provide insight into our findings. Individuals with a history of concussion are known to exhibit impaired postural control;[346] in particular, these individuals exhibit a more conservative gait strategy (e.g., more time in double support, slower gait speed) compared to those without a history of mTBI.[74] Additionally, data from concussion recovery have shown minimal effects of sex on post-concussion gait recovery with both sexes exhibiting similar magnitudes of performance reduction compared to baseline.[101] Thus, the lack of sex differences in gait recovery in the concussion (e.g., greater magnitude impact than RHI) literature may explain the lack of effect of sex on the relationship between

career duration and gait outcomes. These results suggest that sex may not be a contributing factor in gait performance following neurotrauma.

Taken together the findings on single-season RHI on gait and the chronic effects of concussion on gait, the relationship between sex and career duration and gait does not appear to be meaningful. Emerging evidence has highlighted little to no difference between responses to concussion by sex in collegiate athletes,[23] while RHI data suggest little to no effect of sex on dynamic postural control as well.[150,151,339] Our finding of no effect of a sex by career duration interaction on gait adds to the growing body of literature that there may be minimal to no effect of sex on post-RHI outcomes, however, other intrinsic and extrinsic factors such as cardiovascular health, physical activity levels, sleep, etc. should be explored as potential confounders and mediators.

A secondary finding of this aim was that we failed to find a significant interaction between sex and career duration on DTC gait outcomes. Gait speed DTC, acutely post-concussion, is a known predictor for prolonged recovery (>28 days) in concussed athletes.[100] Further, despite data highlighting greater DTC in female rugby players compared to males at baseline, post-concussion, and post-seaseon,[339] we did not find an interaction between sex and career duration on gait outcomes. The null finding may be due in part to differences in RHI exposure between males and females in our cohort. As previously noted, females tend to have worse outcomes in response to neurotrauma compared to males,[191,267,325,332–335,341,342] thus, theoretically if females in our cohort suffered less RHI exposure than the males, then any potential differences may not have manifested in our sample. Post-hoc analyses of collision sport athletes only (HRS and RUG) and all participants (NON, NCA, HRS, RUG) revealed significant differences between males and females for career duration (Males: 11.6 $\pm$

12.6 years; Females: 4.5 ± 7.2 years, $p<0.001$). Although career duration was included in the model, differences in RHI exposure through different sports (e.g., soccer vs. American football), and moreover the magnitude of these exposures, may have accounted for the lack of effect of sex on the model. Furthermore, our participants had varying levels of time since sport cessation (i.e., years since RHI exposure), thus, any persistent deficits of RHI exposure may have recovered by the time of testing, and future analyses should incorporate this as a potential modifying factor.

There were a few significant predictors to the models. Sex added significantly to the unadjusted model for ST and DT stride length and ST tandem gait and the adjusted model for ST stride length. Specifically, females had a 0.095 m shorter ST stride length and 0.075m shorter DT stride length; however, this was in the unadjusted model that did not account for height. In the adjusted model, females had a 0.072m ST shorter stride length than males which may be a result of slower gait speed in females.[347] Also, females had 1.89 s slower ST tandem gait, which is greater than ST tandem gait MDC (0.38s),[28] than males in the unadjusted model which may be due to height differences given that this difference was not present in the adjusted model.[348] Height also added significantly to the adjusted model for DT tandem gait whereby an increase in height saw a decrease in DT tandem gait time which is likely the result of increased gait speed or foot size.[263,349] However, future research should incorporate the measurement of leg length and/or foot size. As such, sex and height should be considered as confounders in future gait research.

Concussion history also added significantly to the adjusted model for DT stride length and DTC stride length, whereby those with a history of concussion had greater DT stride length (0.07m) and DTC stride length (3.1%)—both of which are less than

one standard deviation in change from their respective means. Individuals with a history of concussion walk with a more conservative gait (i.e., shorter stride length),[74] so it is interesting that those with a history of concussion had a greater stride length, despite accounting for height. Herein lies a potential limitation, whereby leg length may have been a better covariate than height in this model, however, these data were not collected. However, the more likely reasoning is that this finding may be purely incidental as there is not established literature to suggest why those with a history of concussion would walk with larger stride lengths. Further, this finding may be in part due to physical activity levels[259,261] given the fact that the physically active individuals tended to have greater frequencies of concussion history compared to those who are not physically active. (Table 7) Whereas the greater DTC for stride length in those with a history of concussion indicates a greater reduction in motor performance during DT, which is in line with previous research highlighting DT dysfunction in individuals post-concussion.[62,103] Collectively, this finding adds to the growing body of literature highlighting the long-term effects of concussion history on motor function during DT conditions.[61,72,74,78,113,269,350]

Age added statistically to the adjusted model for ST and DT double support as well as ST and DT tandem gait time, whereby, unsurprisingly, as age increased each of these measures increased (i.e., worsened). Of note, the clinical relevance of these findings is minimal evidenced by the low beta-values. For example, for every one-year increase in age, there is an associated 0.056 increase in ST double support (%) and 0.074 in DT double support (%). However, this does reflect a more conservative gait with aging, in line with literature amongst aging adults.[263,349] Based upon the predictive models, every one-year increase in age there was an associated 0.096 s

increase in ST tandem gait and 0.174 s increase in DT tandem gait times, suggesting a slowing tandem gait speed with age. This is also in line with ageing and overground gait literature highlighting reduction in gait speed with age as well as differences in tandem gait across age groups.[263,349,351] Together, these data build upon aging literature suggesting decreased gait performance with increased age.

Learning disability(LD)/attention deficit hyperactivity disorder (ADHD) is potentially associated with concussion recovery; moreover, individuals with LD/ADHD history have a greater reported lifetime history of concussion.[1,352,353] LD/ADHD history added statistically to the adjusted model for DT tandem gait, whereby those with a history of LD/ADHD had 3.0 s greater (i.e., slower) DT tandem gait completion time. This is a large difference given that the minimal detectable change (MDC) for ST tandem gait has been reported to be 0.38s (the MDC for DT tandem gait remains to be established).[28] There is evidence that those with LD/ADHD perform worse on measures of computerized neurocognitive tests, a measure of executive function.[195] Slower DT tandem gait times may be reflective of executive dysfunction and an inability to concurrently perform two tasks which may explain the group differences herein.[89,90,354,355] Yet, one would expect this executive dysfunction to transpire across both DT gait tasks (i.e., tandem and level overground DT gait). The lack of effect of LD/ADHD on DT gait may be the result of DT gait being less challenging of a task compared to tandem gait. However, although likely accurate, it should be noted that LD/ADHD history was self-reported. In summary, in agreement with previous research, these data suggest that those with a history of LD/ADHD perform worse on concurrent tasks of gait and cognition compared to those without a history of LD/ADHD.

Although the use of IMUs allowed us to collect data during a global pandemic in a portable manner, they failed to provide a more sensitive measure of postural control which could have been captured by integrated motion capture (e.g., kinematics and kinetics), which may have provided additional insights. Additionally, investigations into gait termination and initiation may have also provided unique insights into potential sex differences. However, IMUs have been commonly utilized in post-concussion gait research and are sensitive enough to highlight group differences.[96,266,293,294,356,357] Furthermore, although gait termination and initiation provides additional insight into neurological functinoing,[90,150,358,359] normal gait is an established predictor of multiple outcomes across numerous domains of health.[259,261,263,349] Further, varying levels of lifetime physical activity and RHI exposure, known confounders to neurophysiological health,[147,191,257,306,360] may have affected our outcomes. However, we did attempt to control for this by including career duration in our models, but future research should incorporate more precise measures of lifetime physical activity levels. Furthermore, the use of career duration as a metric for RHI exposure may not be sensitive enough to highlight effects of RHI on neurophysiological function given the broad nature of the measure, the variability in RHI exposure by position in sport (e.g., football place kicker vs. lineman), and its inability to capture RHI from other sources (e.g., domestic abuse, recreational sports, activities of daily living). Lastly, these data were collected during the ongoing COVID-19 global pandemic which may have resulted in healthy person recruitment bias in addition to alterations in pre-pandemic physical activity levels.[318,319] Since we asked participants to report their current physical activity levels, these data may not

highlight the effects of the chronicity of lifelong physical activity. Thus, future research should aim to include more precise measures of lifetime physical activity.

These findings highlight that exposure to RHI may not negatively affect postural control, assessed by instrumented ST and DT gait, differently across sexes in early- to middle-aged adults. Further, our findings add to growing body of literature which fail to provide conclusive evidence for a modifying factor of sex in response to neurotrauma using objective assessments.[23,266,348,361] However, future research should continue to investigate sex as a biological variable and other potential factors that may contribute to any group differences (e.g., SES, education levels). Regardless, females remain an understudied population in RHI literature, and this study provides some insight on the lack of effects of RHI on gait in females (and males) in their mid-30s. As female participant in contact/collision sports continues to grow,[216,331] there is some solace that they may not be more affected by RHI than males.

Chapter 5

DISSERTATION SUMMARY

The overall objectives of this dissertation were to provide insight into the effects of prolonged contact/collision sport participation and patient reported outcomes and health (*Chapter 2*), as well as the effects of prolonged RHI exposure into adulthood on neurophysiological health (*Chapter 3*) across sex (*Chapter 4*).

Adult rugby participation – Numerous studies have highlighted the relationship between concussion and subsequent LE-MSI, yet the effects of sex remained to be elucidated. However, these studies failed to include amateur (i.e., post-college, nonprofessionals) athletes or female collision sport athletes. Our results found a significant relationship between concussion and LE-MSI in male (OR= 2.21) and female (OR= 2.49) collision sport athletes, but no difference in risk between sexes. Further, the literature on AFE and long-term health remains mixed with studies of collegiate athletes highlighting no clinically significant effects of AFE and results among middle aged and older adult male cohorts reporting significant group differences and other studies in similar cohorts reporting no group differences. Hence, the NINDS TES statement call to action for more research across sexes and varying levels of RHI exposure through a variety of sports. A significant limitation of prior research was that these studies failed to include female collision sport athletes or individuals between college and middle age. Our results found that whether analyzed dichotomously or continuously, AFE and career duration did not significantly predict worse patient reported outcomes among male or female collision sport athletes as assessed by BSI-18, SF-12, and SWLS. Collectively, these findings highlight the need

for sports medicine staff among amateur and community level sports teams to help reduce injury risk following concussion. Additionally, our results add to the growing body of literature that younger AFE to contact/collision sports do not result in worse psychological distress or QoL in early- to middle-aged adults. However, future research should expand insights into the relationship between AFE and career duration to collision sport athletes in mid- to late- adulthood.

Career duration and neurophysiological outcomes – With the current NINDS TES recommendations and growing body of evidence on AFE/RHI research, researchers have noted a distinct need to expand research to women and other collision sports outside of football. Further, it has been noted that AFE may not be an accurate measure of lifetime RHI exposure and other metrics such as career duration should be explored. Thus, in line with NINDS TES recommendations, we investigated the effects of career duration on a variety of psychological, cognitive, and gait outcomes amongst individuals with varying levels of physical activity and RHI exposure through sport. Our results demonstrated that individuals with a history of RHI (i.e., individuals who participated in contact/collision sports up to age 22) or prolonged RHI exposure (i.e., individuals who participated in contact/collision sports beyond age 22) do not perform worse on various measures of executive function, psychological distress and QoL, or dynamic postural control than individuals without a history of RHI exposure when accounting for career duration. In fact, the only group differences that did exist highlighted that the group without a history of RHI, who was not physically active performed worse than those without a history of RHI who were physically active, further highlighting the mental and physical health benefits of team sports. Taken

together, these results highlight the potential neuroprotective effects of exercise and/or the potential lack of long-term deficits of RHI exposure among adults in early to mid-adulthood. However, future research should incorporate more sensitive measures of RHI exposure to further encapsulate lifetime RHI as well as validated measures of current and lifetime physical activity levels to parse out the effects of exercise and RHI on neurophysiological health and function.

Sex and Career Duration Interaction on Gait – Sex differences in gait and in response to acute neurotrauma have been well documented. However, recent literature has noted a lack of sex differences in concussion recovery. However, the literature on RHI and ageing utilizing objective measures of neurobehavioral function is limited. Our results demonstrated no interaction between sex and career duration on assessments of ST and DT gait and their associated DTCs, adding to the growing body of literature noting no effect of sex on neurotrauma outcomes. Despite the lack of relationship, sex should still be considered as a biological variable in future insights on the relationship between RHI and gait given the various intrinsic and extrinsic factors associated with sex and the unknown effect of sex in older adults. Additionally, the large range of career duration between males and females may have accounted for the lack of relationship observed. However, to our knowledge this was the first study to investigate the post-young adulthood (i.e., mid-30's) effects of RHI on gait in males and females. We highlighted the lack of effect of RHI on gait in early to mid-adulthood in physically active individuals, demonstrating the lack of effects of RHI and/or the neuroprotective effects of physical activity. Future research should

incorporate more precise measures of lifetime RHI exposure to further confirm these findings.

Conclusion

In summary, this dissertation fills critical gaps in the concussion and RHI literature. We provided insight in the relationship between concussion and LE-MSI across sexes in a novel cohort of amateur collision sport athletes. The incorporation of rugby provided us an unparalleled insight into the effects of concussion and RHI in a sex-matched collision sport whereby men and women play by the same laws. Further, we expanded AFE research to an unexplored age group (i.e., mid 30s) and sporting group, rugby, highlighting the lack of effect of AFE and career duration on patient reported outcomes, adding to the growing body of literature stating no long-term negative consequences of contact sport participation. Additionally, we highlighted the lack of effect of RHI and prolonged RHI exposure on multiple measures of neurophysiological function and in fact showed that those without a history of RHI who are not physically active tended to perform worse overall. However, we did not identify any sex differences, but sex should still be considered an important confounder for future research given differences in ageing and neurodegenerative disease risk across sexes. Ultimately, we filled a critical knowledge gap showing the lack of effect of RHI exposure in early to mid-adulthood in males and females.

Future Directions

The next steps for this dissertation would be to expand our research to older (>50 years old) samples of former and current contact/collision sport athletes.

Although there appears to be no effect of lifetime RHI exposure in early- to mid-adulthood there may be dysfunction in later-life (i.e., older and/or geriatric adults). Further, more sensitive measures of lifetime RHI exposure should be explored that can be utilized for multiple contact/collision sports outside of American football (e.g., similar to helmet measured impacts in football providing a metric for total exposure, using mouthguard measured head impacts in rugby or lacrosse). Additionally, the neuroprotective effects of lifetime physical activity are of particular interest. Future research should incorporate validated and detailed measures of physical activity to elucidate the effects of physical activity and RHI on long-term neurophysiological health. Lastly, other confounding variables on long-term neurological health should also be considered such as early-childhood events, socioeconomic status, smoking status, alcohol abuse, family history of neurodegenerative disease, air pollution, social isolation, obesity, education, diabetes, and hypertension. In summary, RHI exposure does not appear to negatively affect neurophysiological functioning in physically active adults in mid-life.

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Appendix A

GILBERT INJURY HISTORY QUESTIONNAIRE

Directions: Please answer the following questions regarding your **collegiate athletic career** to the best of your knowledge. Your answers will remain confidential and will **NOT** be shared with your coaches or athletic training staff.

Demographics

Sex: M / F Age: ___ Academic year in school: FR SO JR SR 5th Other _____

Sport(s): _____ Position in sport: _____

How many years did you participate in your sport at the collegiate level? _____

Which Division? NCAA I NCAA II NCAA III NJCAA Other: _____

Injury History

1. Have you ever sprained your ankle? YES NO
    a. Was the ankle sprain reported to a healthcare provider? YES NO
    b. Did you complete a rehabilitation program, either on your own or with a healthcare provider? YES NO
        i. If no, why not? _____
2. Have you ever injured a ligament or cartilage in your knee? YES NO
    a. If yes, which one(s)? Meniscus Cartilage MCL ACL LCl PCL
3. Have you ever sprained any other joints (shoulder, wrist, etc.) while playing sports? YES NO
    a. If yes, what body part(s)? _____
4. Have you ever suffered a concussion? YES NO
    a. If yes, how many? _____
    b. If yes, approximately when were they? (Month and year to the best of your memory) _____
5. Did you ever suffer a concussion and not tell anyone? YES NO
    a. If yes, why? (check all that apply)

____ 1. Did not think it was serious

____ 2. Did not know it was a concussion

____ 3. Did not want to be pulled out of the game/practice

____ 4. Did not want to be pulled from future games/practice

____ 5. Did not want to let your teammates down

____ 6. Would have if it was a less important game/practice

____ 7. Other:_____

6. Have you ever hurt your back? YES NO

a. If yes, please explain:_____ _____

7. Have you ever broken a bone? YES NO

a. If yes, which bone(s)?_____ _____

8. Have you ever dislocated your shoulder? YES NO

9. Have you ever pulled, strained, or torn your rotator cuff or any other structure in your shoulder? YES NO

a. If yes, briefly explain: _____

10. Have you ever been knocked out while playing sports? YES NO

a. If yes, how many times?_____ How many were diagnosed as concussions?_____

11. Have you ever pulled, strained, or torn a muscle? YES NO

a. If yes, which muscle(s)? _____

12. Have you ever been "knocked silly/seen stars" (confused/disoriented) while playing sports? YES NO

a. If yes, how many times? __ How many were diagnosed as a concussion?___

b. If yes, did you tell your coach, athletic trainer, or parent? Which one(s)?

_____

13. Have you had multiple ankle sprains? YES NO

a. If yes, how many?_____

14. Have you had any episodes of your ankle giving way? YES NO

a. If yes, how many times?_____

15. Do you have any current residual (lingering) symptoms regarding your ankle sprains? YES NO

a. If yes, what are they? _____

16. Have you ever experienced any season ending injuries? YES NO

a. If yes, what was/were your injury/injuries? _____

b. If yes, did you have surgery on any of these injuries? Which ones? _____

17. During your collegiate athletic career, did you ever have any orthopedic surgeries? YES NO

a. If yes, on what? _____

18. Following a blow to the head, if you had experienced a headache, dizziness, or confusion, would

you report it to your athletic trainer? YES NO

a. If no, why not? _____

19. Have you ever had injuries that you did not tell your athletic trainer about? YES NO

a. If yes, what injuries? _____

20. Have you ever been hit so hard you lost your memory while playing sports? YES NO

a. If yes, how many times? ___ How many were diagnosed as a concussion?__

b. If yes, did you tell your coach, athletic trainer, or parent? Which one(s)? _____

21. During your collegiate athletic career, do you feel like you had a good relationship with your athletic trainer?

YES NO

Appendix B

AIM 1 QUALTRICS DATA COLLECTION SURVEY

Rugby Injury Epidemiology Consent & Survey

Start of Block: Informed Consent

Q1
Welcome to the Rugby Injury Epidemiology Research Study!
Principal Investigator: Katherine J Hunzinger, MS, CEP

Important aspects of the study you should know about:

• **Purpose**: The purpose of the study is to examine the quality of life, and the association between concussion and lower extremity musculoskeletal injury rates in current and/or former full contact (e.g., tackle) rugby players.
• **Procedures**: If you choose to participate, you will be asked to complete an online survey using Qualtrics broken down into two questionnaires. The first questionnaire will be broken down into two parts:
o *Demographics* (3-5 min): You will be asked to answer questions about your background including: age, sex, sport history, rugby playing history, level of rugby played.
o *Injury History Questionnaire* (5-10 minutes): You will be asked questions regarding your injury history during your lifetime and your rugby career. If you have experienced an injury, you will be asked to answer follow up questions regarding that injury and if it occurred while playing rugby.
The second questionnaire consists of the following;
o *Brief Symptom Inventory-18 (BSI-18), Short Form 12 (SF-12), & Satisfaction With Life Scale (SWLS)* (<5 minutes): You will be asked 35 questions regarding your distress, perceptions of your health, and satisfaction with life.
• **Duration**: This will take about approximately 15-20 minutes over the course of one online session.
• **Risks**: There are no known risks associated with these questionnaires. The

questions and injury recall may be challenging to complete, which may cause frustration or slight emotional discomfort.
• **Benefits**: Participants will not benefit directly from taking part in this research study. The results from this study may help further understanding of concussions and lower extremity injury risk among rugby players and age of first exposure to collision sports.
• **Costs and Compensation**: If you decide to participate there will be no additional cost to you. If you complete both surveys you could be compensated up to $50 in the form of a World Rugby Shop gift card. Four participants will be randomly selected to receive this gift card.
• **Participation**: Taking part in this research study is your decision. You do not have to participate in this research. If you choose to take part, you have the right to stop at any time. If you decide not to participate or if you decide to stop taking part in the research at a later date, there will be no penalty or loss of benefits to which you are otherwise entitled.

Contact Information: If you have any questions about the purpose, procedures, or any other issues related to this research study you may contact the Principal Investigator, Katie Hunzinger, at (630) 621-5786 or khunzing@udel.edu, or Dr. Thomas A. Buckley at (302) 831-4783 or tbuckley@udel.edu.

CONSENT TO PARTICIPATE IN THE RESEARCH STUDY:
By clicking the button below, you acknowledge that your participation in the study is voluntary, you are 18 years of age, you have asked any questions you have about the research and the questions have been answered to your satisfaction, and that you are aware that you may choose to terminate your participation in the study at any time and for any reason.

Please note that this survey will be best displayed on a laptop or desktop computer. Some features may be less compatible for use on a mobile device.

- ○ I consent, begin the study (1)
- ○ I do not consent, I do not wish to participate (2)

Skip To: End of Survey If Q1 = I do not consent, I do not wish to participate

N

Q2 Do you currently play, or have you ever played rugby?

- Yes (1)
- No (2)

Skip To: End of Survey If Q2 = No

End of Block: Informed Consent

Start of Block: Demographics

Q3 What is your biological sex?

- Male (0)
- Female (1)
- Prefer not to share (2)
- If your sex is not listed, please disclose here (3) _____

O

Q4 What is your age?

_____

Skip To: End of Survey If Condition: What is your age? Is Less Than 18. Skip To: End of Survey.

P

Q5 What year were you born?

	Year

Please Select: (1) | ▼ 1900 (1 ... 2002 (2002)

Q

*

Q6 What is your height (inches)? [i.e. 5 ft tall = 60 inches]

_____

R

*

Q7 What is your weight (lbs)?

_____

S

Q8 Please check the box(es) below if you have any FAMILY HISTORY of any of the following:

- [ ] None (1)
- [ ] Alzheimer's Disease (2)
- [ ] Dementia (3)
- [ ] Parkinson's Disease (4)
- [ ] Other neurological disease (Please write below) (5)

_____

T

Page Break

Q9 How many **YEARS** (not seasons) did you play the following sport(s)? If you did not play an organized sport(s) you may leave it blank.

- American Football (1) _____
- Badminton (2) _____
- Baseball (3) _____
- Basketball (4) _____
- Boxing (5) _____
- Crew (Rowing) (6) _____
- Cricket (7) _____
- Cross Country Running (8) _____
- Cycling (9) _____
- Dance (10) _____
- Diving (11) _____
- Equestrian/Horse Racing (12) _____
- Fencing (13) _____
- Field Hockey (14) _____
- Flag Football (15) _____
- Golf (16) _____

- ○ Gymnastics (17) _____
- ○ Handball (18) _____
- ○ Ice Hockey (19) _____
- ○ Lacrosse (20) _____
- ○ Rugby (21) _____
- ○ Skiing (22) _____
- ○ Soccer (Football) (23) _____
- ○ Softball (24) _____
- ○ Snowboarding (25) _____
- ○ Swimming (26) _____
- ○ Tennis (27) _____
- ○ Track & Field (28) _____
- ○ Volleyball (29) _____
- ○ Wrestling (30) _____

U

Page Break

Q10 At what age did you **START** playing the following sport(s)? If you did not play an organized sport(s) you may leave it blank.

- American Football (1) _____
- Badminton (2) _____
- Baseball (3) _____
- Basketball (4) _____
- Boxing (5) _____
- Crew (Rowing) (6) _____
- Cricket (7) _____
- Cross Country Running (8) _____
- Cycling (9) _____
- Dance (10) _____
- Diving (11) _____
- Equestrian/Horse Racing (12) _____
- Fencing (13) _____
- Field Hockey (14) _____
- Flag Football (15) _____
- Golf (16) _____

- ○ Gymnastics (17) _____
- ○ Handball (18) _____
- ○ Ice Hockey (19) _____
- ○ Lacrosse (20) _____
- ○ Rugby (21) _____
- ○ Skiing (22) _____
- ○ Soccer (Football) (23) _____
- ○ Softball (24) _____
- ○ Snowboarding (25) _____
- ○ Swimming (26) _____
- ○ Tennis (27) _____
- ○ Track & Field (28) _____
- ○ Volleyball (29) _____
- ○ Wrestling (30) _____

V

Page Break

Q11 On average, how many seasons **per calendar year** of rugby did you play? (i.e. Fall 15s, Spring 15s, and a Summer 7s = 3 seasons/year)

_____

W

Q12 When you played rugby, approximately how many games per calendar year did you play?

_____

X

Q13 When you played rugby, approximately how many practice hours did you have per week?

_____

Y

X→

Q14 What level of rugby did you participate in as a player the most? (i.e. if you did 2 years college, and 4 years club you mostly participated in club).

- ○ High School (1)
- ○ Collegiate (Club/Varsity) (2)
- ○ Adult Club (3)
- ○ Professional (4)
- ○ International (5)
- ○ Other (6) _____

Z

Q15 Did you play rugby in **High School**?

- Yes (1)
- No (0)

AA

Figure 1 *Display This Question:*

Figure 2 *If Q15 = Yes*

Q16 How many years did you play **High school Rugby**?

_____

BB

Figure 3 *Display This Question:*

Figure 4 *If Q15 = Yes*

Q17 What position did you primarily play in H**igh School Rugby**?

- Prop (1)
- Hooker (2)
- Lock (3)
- Flanker (4)
- 8-Person (5)
- Scrumhalf (6)
- Fly-half (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)
- Not Applicable (12)

CC

Page Break

Q18 Did you play rugby in **College/University**?

- Yes (1)
- No (0)

DD

Figure 5 *Display This Question:*

Figure 6 *If Q18 = Yes*

Q19 How many years did you play **Rugby in College/University**?

_____

EE

Figure 7 *Display This Question:*

Figure 8 *If Q18 = Yes*

Q20 What position did you primarily play in **Collegiate Rugby**?

- Prop (1)
- Hooker (2)
- Lock (3)
- Flanker (4)
- 8-Person (5)
- Scrumhalf (6)
- Fly-half (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)
- Not Applicable (12)

FF

Page Break

Q21 Did you play rugby in **an Adult Club League**?

- Yes (1)
- No (0)

GG

Figure 9 *Display This Question:*

Figure 10 *If Q21 = Yes*

Q22 How many years did you play **Adult Club Rugby**?

_____

HH

Figure 11 *Display This Question:*

Figure 12 *If Q21 = Yes*

Q23 What position did you primarily play in **Adult Club Rugby**?

- Prop (1)
- Hooker (2)
- Lock (3)
- Flanker (4)
- 8-Person (5)
- Scrumhalf (6)
- Fly-half (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)
- Not Applicable (12)

II

Page Break

Q24 Did you play **Professional Rugby**?

- Yes (1)
- No (0)

JJ

Figure 13 Display This Question:

Figure 14 If Q24 = Yes

Q25 How many years did you play **Professional Rugby**?

_____

KK

Figure 15 Display This Question:

Figure 16 If Q24 = Yes

Q26 What position did you primarily play in **Professional Rugby**?

- Prop (1)
- Hooker (2)
- Lock (3)
- Flanker (4)
- 8-Person (5)
- Scrumhalf (6)
- Fly-half (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)
- Not Applicable (12)

LL

Page Break

Q27 Did you play **International Rugby**? (i.e. Played for the National Team, TEAM USA, USA U18s in international competitions)

- Yes (1)
- No (0)

MM

Figure 17 *Display This Question:*

Figure 18 *If Q27 = Yes*

Q28 How many years did you play **International Rugby**?

_____

NN

Figure 19 *Display This Question:*

Figure 20 *If Q27 = Yes*

Q29 What position did you primarily play in **International Rugby**?

- Prop (1)
- Hooker (2)
- Lock (3)
- Flanker (4)
- 8-Person (5)
- Scrumhalf (6)
- Fly-half (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)
- Not Applicable (12)

OO

Page Break

Q30 What country did you primarily play rugby in?

▼ United States (1) ... Other (14)

PP

Q31 What was your primary rugby position for 15s?

- Prop (1)
- Hooker (2)
- Lock (3)
- Flanker (4)
- 8-Person (5)
- Scrumhalf (6)
- Fly-half (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)
- Not Applicable (12)

QQ

Q32 What was your primary rugby position for 7s?

- Prop (1)
- Hooker (2)
- Scrumhalf (3)
- Fly-half (4)
- Center (5)
- Wing (6)
- Not Applicable (7)

End of Block: Demographics

Start of Block: Injury History

Q33 Have you ever sprained your **ankle**?

- Yes (1)
- No (0)

RR

Figure 21 *Display This Question:*

Figure 22 *If Q33 = Yes*

JS

Q34 When did you sprain your ankle?

Please select answers to the best of your ability (month/year).

If you do not know the month you can leave it blank.

	Month	Year
Ankle Sprain #1 (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (121)
Ankle Sprain #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (121)
Ankle Sprain #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (121)
Ankle Sprain #4 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (121)

SS

Figure 23 *Display This Question:*

Figure 24 *If Q33 = Yes*

X→

Q35 Was the ankle sprain reported to a healthcare provider?

- ○ Yes (1)
- ○ No (0)

TT

Figure 25 *Display This Question:*

Figure 26 *If Q33 = Yes*

X→

Q36 Did you complete a rehabilitation program either on your own or with a healthcare provider?

- Yes, on my own (1)
- Yes, with a healthcare provider (2)
- No (0)

UU

Figure 27 *Display This Question:*

Figure 28 *If Q36 = No*

Q37 If you did not complete a rehabilitation program, why not?

_____

_____

_____

_____

_____

VV

Page Break

X→

Q38 Have you ever injured a **ligament or cartilage in your knee**?

- ○ Yes (1)
- ○ No (0)

WW

Figure 29 *Display This Question:*

Figure 30 *If Q38 = Yes*

Q39 Which one(s)?

- ☐ Meniscus (1)
- ☐ Cartilage (2)
- ☐ MCL (3)
- ☐ ACL (4)
- ☐ LCL (5)
- ☐ PCL (6)

XX

Figure 31 *Display This Question:*

Figure 32 *If Q38 = Yes*

Q40 Did your knee cartilage or ligament injury/injuries require surgery?

- ○ Yes (explain below) (1)

_____

- ○ No (2)

YY

Page Break

Figure 33 *Display This Question:*

Figure 34 *If Q38 = Yes*

JS X→

Q41 When did your knee injury/injuries occur?

Please describe what the knee injury was in the text box provided (i.e.: ACL Tear)

If you do not know the month you can leave it blank.

	Month	Year
Knee Injury #1 (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Knee Injury #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Knee Injury #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Knee Injury #4 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)

ZZ

X→

Q42 Have you ever sprained any other joints (shoulder, wrist, etc.)?

- ○ Yes (1)
- ○ No (0)

AAA

Figure 35 *Display This Question:*

Figure 36 *If Q42 = Yes*

Q43 What body part(s)?

_____

_____

_____

_____

_____

BBB

Page Break

X→

Q44 Have you ever suffered a **concussion**?

- Yes (1)
- No (0)

CCC

Figure 37 *Display This Question:*

Figure 38 *If Q44 = Yes*

*

Q45 How many concussions have you suffered?

_____

DDD

Figure 39 *Display This Question:*

Figure 40 *If Q44 = Yes*

JS X→

Q46 Approximately when where your concussion(s)?

If the month is unknown you may leave the month blank.

	Month	Year

Concussion #1 (most recent) (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #4 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #5 (5)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #6 (6)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #7 (7)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #8 (8)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #9 (9)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Concussion #10 (10)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)

EEE

Page Break

Q47 Did you ever suffer a concussion and **not tell anyone**?

- ○ Yes (1)
- ○ No (0)

FFF

Figure 41 Display This Question:

Figure 42 If Q47 = Yes

Q48 Why? (Select all that apply)

- ☐ Did not think it was serious (1)
- ☐ Did not know it was a concussion (2)
- ☐ Did not want to be pulled out of the game/practice (3)
- ☐ Did not want to be pulled from future games/practice (4)
- ☐ Did not want to let your teammates down (5)
- ☐ Would have if it was a less important game/practice (6)
- ☐ Other (7) _____

GGG

Page Break

Q49 Have you ever hurt your **back**?

- Yes (1)
- No (0)

HHH

Figure 43 Display This Question:

Figure 44 If Q49 = Yes

Q50 Please explain.

_____

_____

_____

_____

_____

III

Page Break

X→

Q51 Have you ever broken a bone in your **leg, hip, or foot**?

- ○ Yes (1)
- ○ No (0)

JJJ

Figure 45 Display This Question:

Figure 46 If Q51 = Yes

JS X→

Q52 What bone(s) did you break in your leg, hip, or foot?

Please select dates to the best of your ability and fill in the bone location.

	Month	Year
Broken Bone #1 (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Broken Bone #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Broken Bone #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Broken Bone #4 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)

KKK

X→

Q53 Have you ever broken a bone in your **arm, wrist, or hand**?

- Yes (1)
- No (0)

LLL

Page Break

Q54 Have you ever dislocated your **shoulder**?

- Yes (1)
- No (0)

MMM

Q55 Have you ever pulled, strained, or torn your **rotator cuff or any other structure in your shoulder**?

- Yes (1)
- No (0)

NNN

Figure 47 Display This Question:

Figure 48 If Q55 = Yes

Q56 You answered yes to having ever pulled, strained, or torn your rotator cuff or any other structure in your shoulder.

Briefly explain.

_____

_____

_____

_____

_____

OOO

Page Break

Q57 Have you ever been **knocked out while playing sports**?

- Yes (1)
- No (0)

PPP

Figure 49 *Display This Question:*

Figure 50 *If Q57 = Yes*

Q58 How many times have you been knocked out while playing sports?

_____

QQQ

Figure 51 *Display This Question:*

Figure 52 *If Q57 = Yes*

Q59 Approximately when were you knocked out while playing sports?

	Month	Year
Knocked out #1 (most recent) (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Knocked out #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Knocked out #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Knocked out #3 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)

RRR

Figure 53 *Display This Question:*

Figure 54 *If Q57 = Yes*

Q60 How many were diagnosed as a concussion?

_____

SSS

Page Break

X→

Q61 Have you ever pulled, strained, or torn a muscle in your **leg, hip, or foot**?

- Yes (1)
- No (0)

TTT

Figure 55 Display This Question:

Figure 56 If Q61 = Yes

JS X→

Q62 When did you pull, strain, or tear a muscle in your leg, hip, or foot?

Please describe the muscle/injury in the text box provided.

	Month	Year
Injury #1 (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #4 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)

UUU

X→

Q63 Have you ever pulled, strained, or torn a muscle in your **arm, wrist, or torso**?

- Yes (1)
- No (0)

VVV

Figure 57 *Display This Question:*

Figure 58 *If Q63 = Yes*

Q64 Which muscle(s) did you pull, strain, or tear?

_____

_____

_____

_____

_____

WWW

Page Break

Q65 Have you ever been **"knocked silly/seen stars" (confused/disorientated)**?

- Yes (1)
- No (0)

XXX

Figure 59 Display This Question:

Figure 60 If Q65 = Yes

Q66 How many times have you ever been "knocked silly/seen stars" (confused/disorientated)?

_____

YYY

Figure 61 Display This Question:

Figure 62 If Q65 = Yes

Q67 Did you tell your coach, athletic trainer/physio, doctor/physician, or parent that you were knocked silly/saw stars? Select all that apply.

- [ ] Coach (1)
- [ ] Athletic Trainer/Physio (2)
- [ ] Doctor/Physician (3)
- [ ] Parent (4)
- [ ] None of the above (5)

ZZZ

Figure 63 *Display This Question:*

Figure 64 *If Q65 = Yes*

JS X→

Q68 Approximately when were you "knocked silly/saw stars" (confused/disorientated)?

	Month	Year
Injury #1 (most recent) (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #4 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)

AAAA

Figure 65 *Display This Question:*

Figure 66 *If Q65 = Yes*

*

Q69 How many were diagnosed as a concussion?

_____

BBBB

Page Break

Q70 Have you had **multiple** ankle sprains?

- Yes (1)
- No (0)

CCCC

Figure 67 Display This Question:

Figure 68 If Q70 = Yes

Q71 How many ankle sprains have you had?

_____

DDDD

Q72 Have you had any episodes of your ankle giving away?

- Yes (1)
- No (0)

EEEE

Page Break

X→

Q73 Have you ever experienced any season ending injuries?

- ○ Yes (1)
- ○ No (0)

FFFF

Figure 69 *Display This Question:*

Figure 70 *If Q73 = Yes*

Q74 What was/were your injury/injuries?

_____

_____

_____

_____

_____

GGGG

Page Break

Q75 During your lifetime, did you ever have any sports related or orthopedic surgeries?

- Yes (1)
- No (0)

HHHH

Figure 71 Display This Question:

Figure 72 If Q75 = Yes

Q76 Please describe the injuries.

_____

_____

_____

_____

_____

IIII

Page Break

Q77 Following a blow to the head, if you had experienced a headache, dizziness, or confusion, would you report it to your athletic trainer/physio?

- Yes (1)
- No (0)

JJJJ

Figure 73 Display This Question:

Figure 74 If Q77 = No

Q78 Why not?

_____

_____

_____

_____

_____

Page Break

Q79 During your rugby career, did your team(s) have an assigned athletic trainer/physio? (Meaning you had an athletic trainer/physio **at practice or games employed by your team**)

- Yes (1)
- No (0)

Figure 75 *Display This Question:*

Figure 76 *If Q79 = Yes*

Q80 During your rugby career, do you feel like you had a good relationship with your athletic trainer(s)/physio(s)?

- Yes (1)
- No (0)

Q81 Have you ever had injuries that you did not tell your coach or athletic trainer/physio about?

- Yes (1)
- No (0)

Figure 77 *Display This Question:*

Figure 78 *If Q81 = Yes*

Q82 What injury/injuries? Why did you not report the injury?

_____

_____

_____

_____

_____

KKKK

Page Break

X→

Q83 Have you ever been hit so hard you lost your memory while playing sports?

- Yes (1)
- No (0)

LLLL

Figure 79 *Display This Question:*

Figure 80 *If Q83 = Yes*

JS X→

Q84 Approximately when were you hit so hard you lost your memory while playing sports?

	Month	Year

Injury #1 (most recent) (1)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #2 (2)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #3 (3)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)
Injury #4 (4)	▼ January (1 ... December (12)	▼ 1940 (1 ... 2020 (81)

MMMM

Figure 81 *Display This Question:*

Figure 82 *If Q83 = Yes*

Q85 How many times have you been hit so hard you lost your memory while playing sports?

_____

NNNN

Figure 83 *Display This Question:*

Figure 84 *If Q83 = Yes*

Q86 How many were diagnosed as a concussion?

_____

OOOO

Figure 85 *Display This Question:*

Figure 86 *If Q83 = Yes*

Q87 Did you tell your coach, athletic trainer/physio, doctor/physician, or parent?

- [ ] Yes, coach (2)
- [ ] Yes, athletic trainer/physio (1)
- [ ] Yes, doctor/physician (4)
- [ ] Yes, parent (0)
- [ ] None of the above (3)

PPPP

Page Break

End of Block: Injury History

Start of Block: BSI 18

Q88 DIRECTIONS: Below is a list of problems people sometimes have. Read each one carefully and circle the number that best scribes HOW MUCH THAT PROBLEM HAS DISTRESSED OR BOTHERED YOU DURING THE PAST 7 DAYS INCLUDING TODAY. Do not skip any items. HOW MUCH WERE YOU DISTRESSED BY:	NOT AT ALL (0)	A LITTLE BIT (1)	MODERATELY (2)	QUITE A BIT (3)	EXTREMELY (4)
1. Faintness or dizziness (1)	○	○	○	○	○
2. Feeling no interest in things (2)	○	○	○	○	○
3. Nervousness or shakiness inside (3)	○	○	○	○	○
4. Pains in the heart or chest (4)	○	○	○	○	○
5. Feeling lonely (5)	○	○	○	○	○
6. Feeling tense or keyed up (6)	○	○	○	○	○
7. Nausea or upset stomach (7)	○	○	○	○	○
8. Feeling blue (8)	○	○	○	○	○

9. Suddenly scared for no reason (9)	○	○	○	○	○
10. Trouble getting your breath (10)	○	○	○	○	○
11. Feeling of worthlessness (11)	○	○	○	○	○
12. Spells of terror or panic (12)	○	○	○	○	○
13. Numbness or tingling in parts of your body (13)	○	○	○	○	○
14. Feeling hopelessness about the future (14)	○	○	○	○	○
15. Feeling so restless you couldn't sit still (15)	○	○	○	○	○
16. Feeling weak in parts of your body (16)	○	○	○	○	○
17. Thoughts of ending your life (17)	○	○	○	○	○
18. Feeling fearful (18)	○	○	○	○	○

End of Block: BSI 18

Start of Block: SF-12

Q89 In general, would you say your health is:

- Excellent (1) (1)
- Very Good (2) (2)
- Good (3) (3)
- Fair (4) (4)
- Poor (5) (5)

QQQQ

Page Break

Q90 The following two questions are about activities you might do during a typical day. Does YOUR HEALTH NOW LIMIT YOU in these activities? If so, how much? MODERATE ACTIVITIES, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf:

- ○ Yes, Limited A Lot (1) (1)
- ○ Yes, Limited A Little (2) (2)
- ○ No, Not Limited At All (3) (3)

RRRR

Q91

Climbing SEVERAL flights of stairs:

- ○ Yes, Limited A Lot (1) (1)
- ○ Yes, Limited A Little (2) (2)
- ○ No, Not Limited At All (3) (3)

SSSS

Page Break

Q92 During the PAST 4 WEEKS have you had any of the following problems with your work or other regular activities AS A RESULT OF YOUR PHYSICAL HEALTH? ACCOMPLISHED LESS than you would like:

- Yes (1) (1)
- No (1) (2)

TTTT

Q93

Were limited in the KIND of work or other activities:

- Yes (1) (1)
- No (1) (2)

UUUU

Page Break

Q94 During the PAST 4 WEEKS, were you limited in the kind of work you do or other regular activities AS A RESULT OF ANY EMOTIONAL PROBLEMS (such as feeling depressed or anxious)?
ACCOMPLISHED LESS than you would like:

- ○ Yes (1) (1)
- ○ No (1) (2)

VVVV

Q95

Didn’t do work or other activities as CAREFULLY as usual:

- ○ Yes (1) (1)
- ○ No (1) (2)

WWWW

Page Break

Q96 During the PAST 4 WEEKS, how much did PAIN interfere with your normal work (including both work outside the home and housework)?

- Not At All (1) (1)
- A Little Bit (2) (2)
- Moderately (3) (3)
- Quite A Bit (4) (4)
- Extremely (5) (5)

XXXX

Page Break

Q97 The next three questions are about how you feel and how things have been DURING THE PAST 4 WEEKS. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the PAST 4 WEEKS –
Have you felt calm and peaceful?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

Q98 Did you have a lot of energy?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

Q99 Have you felt downhearted and blue?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

Page Break

Q100

During the PAST 4 WEEKS, how much of the time has your PHYSICAL HEALTH OR EMOTIONAL PROBLEMS interfered with your social activities (like visiting with friends, relatives, etc.)?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

End of Block: SF-12

Start of Block: SWL

Q101 **Instructions**: Below are five statements that you may agree or disagree with. Using the 1 - 7 scale below, indicate your agreement with each item by placing the

appropriate number on the line preceding that item. Please be open and honest in your responding.

	7- Strongly agree (7)	6- Agree (6)	5- Somewhat agree (5)	4- Neither agree nor disagree (4)	3- Somewhat disagree (3)	2- Disagree (2)	1- Strongly disagree (1)
In most ways my life is close to my ideal. (1)	○	○	○	○	○	○	○
The conditions of my life are excellent. (2)	○	○	○	○	○	○	○
I am satisfied with my life. (3)	○	○	○	○	○	○	○
So far I have gotten the important things I want in life. (4)	○	○	○	○	○	○	○
If I could live my life over, I would change almost nothing. (5)	○	○	○	○	○	○	○

End of Block: SWL

Start of Block: End of Survey

*

Q102 What is your e-mail address? (this will be used to contact you regarding the gift card if you win it)

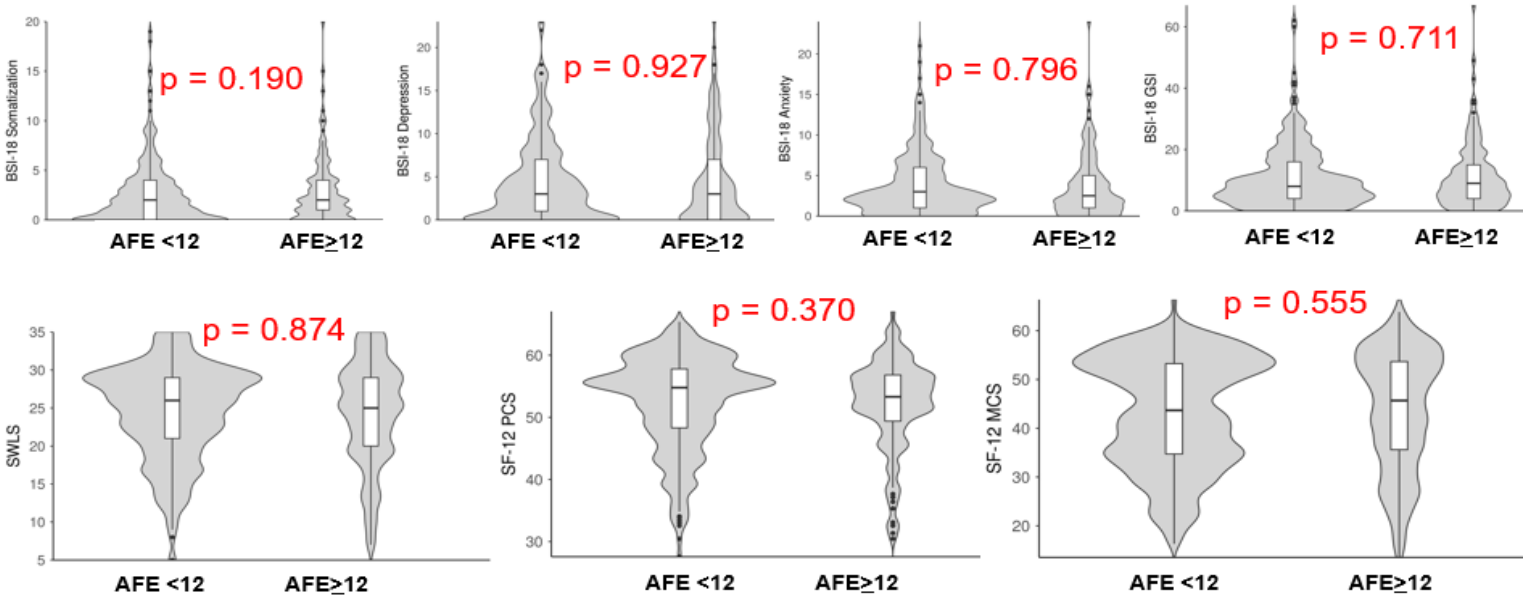
_____

End of Block: End of Survey

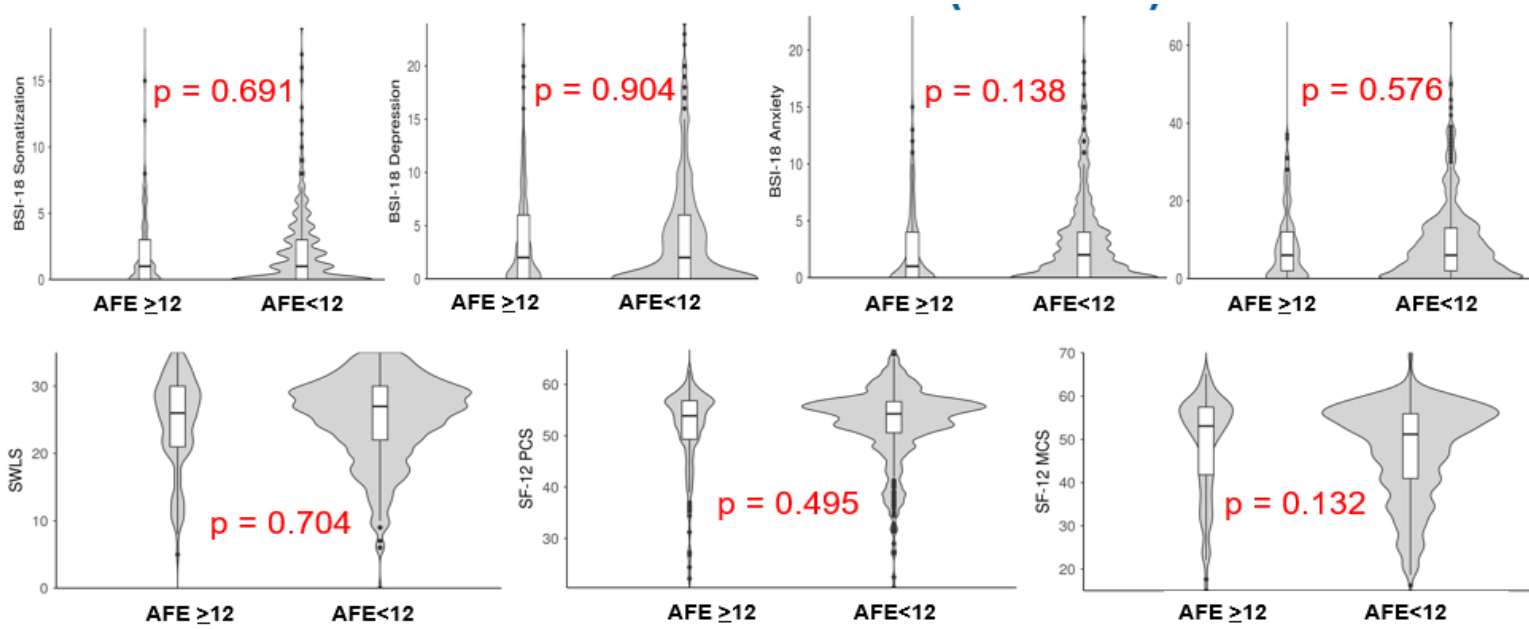
Appendix C

DICHOTOMOUS AFE OUTCOMES BY SEX GRAPHS

Women, N=424



Men, N=611



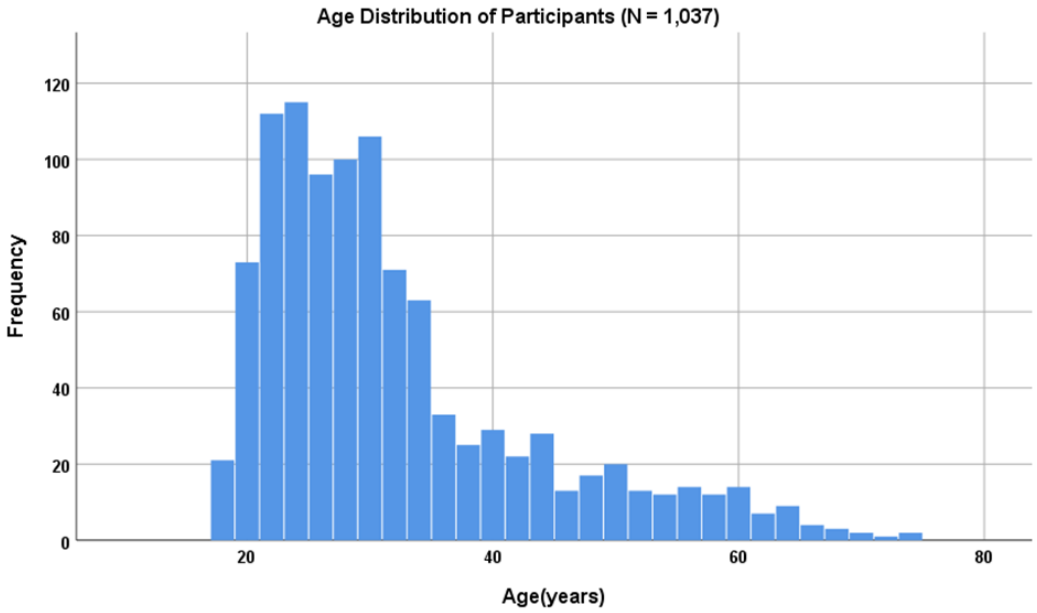
Appendix D

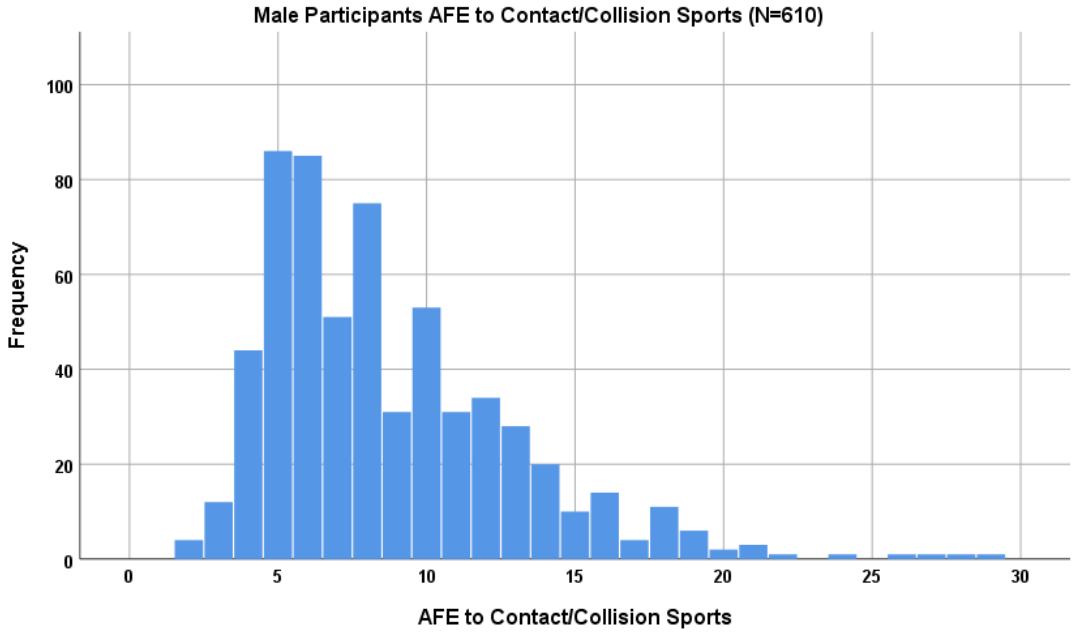
BREAKDOWN OF AIM 1.2 PARTICIPANTS BY SPORT

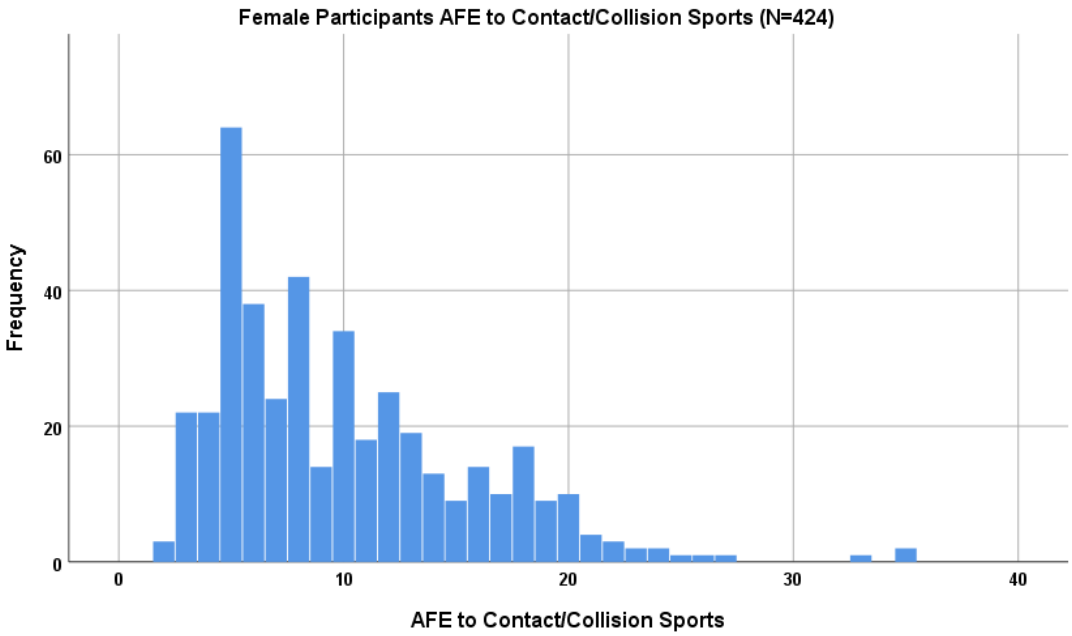
Sport	Number of Participants
American Football	282
Badminton	52
Baseball	254
Basketball	376
Boxing	59
Crew (Rowing)	50
Cricket	103
Cross Country Running	133
Cycling	62
Dance	81
Diving	21
Equestrian/Horse Racing	42
Fencing	27
Field Hockey	59
Flag Football	93
Golf	116
Gymnastics	88
Handball	16
Ice Hockey	86
Lacrosse	90
Rugby	1,037
Skiing	130
Soccer (Football)	429
Softball	186
Snowboarding	113
Swimming	173
Tennis	96
Track & Field	280
Volleyball	144
Wrestling	143

Appendix E

AIM 1.2 AGE AND AFE DISTRIBUTION BY SEX

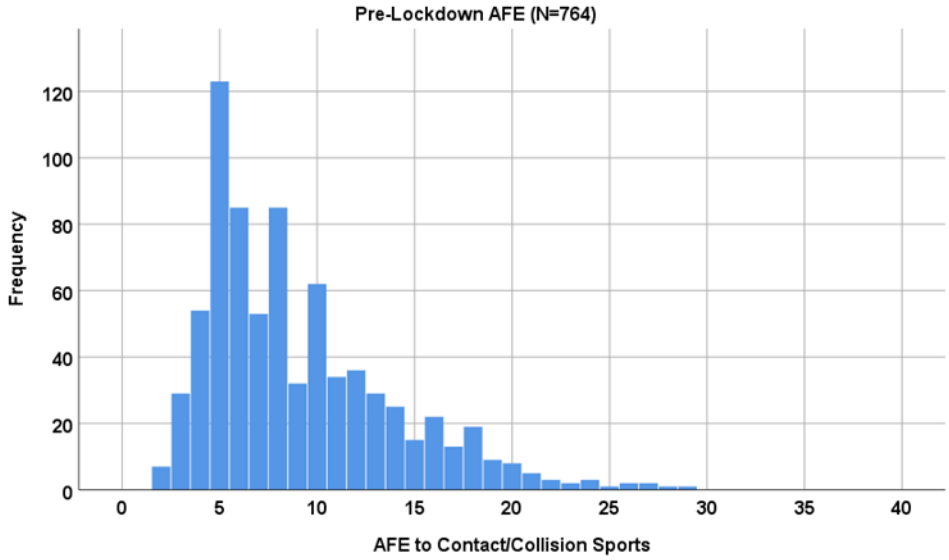


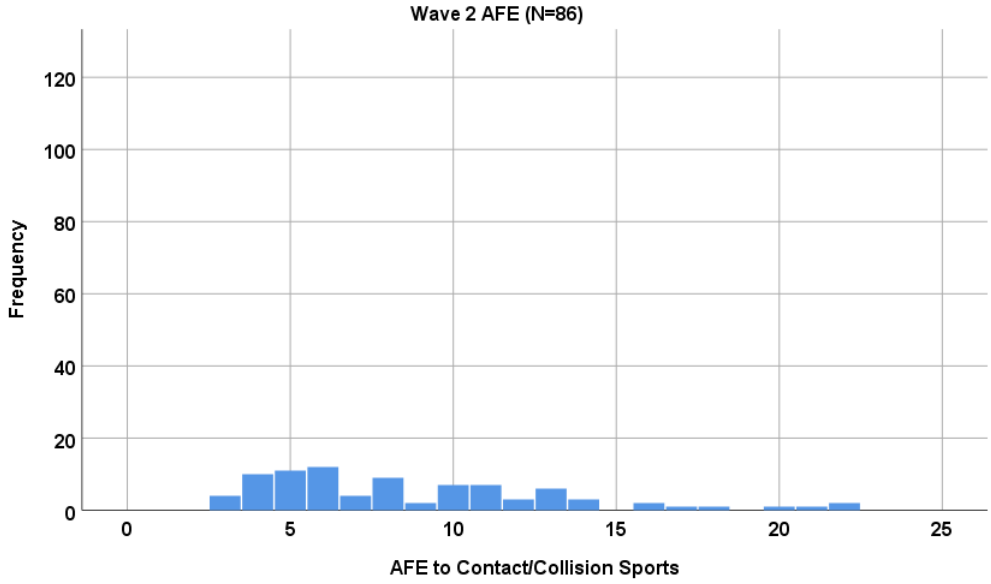


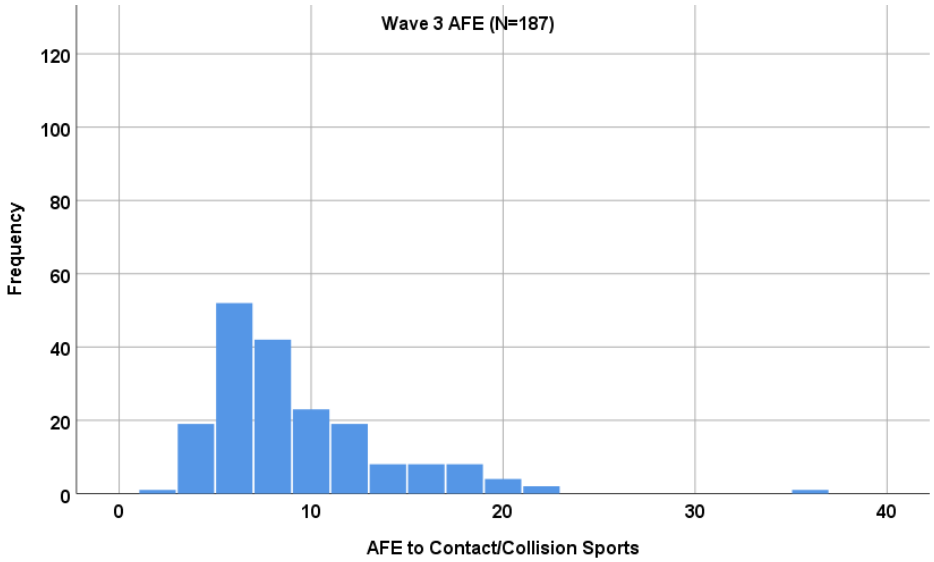

Female Participants AFE to Contact/Collision Sports (N=424)
Frequency
60
40
20
0
0
10
20
30
40
AFE to Contact/Collision Sports

Appendix F

AIM 1.2 AFE DISTRBIUTION BY RECRUITMENT WAVE






Wave 3 AFE (N=187)
Frequency
120
100
80
60
40
20
0
0
10
20
30
40
AFE to Contact/Collision Sports

Appendix G

AIM 2 & 3 QUALTRICS FORM

S.C.R.U.M. Aim 2 Data Collection Forms

Start of Block: Subject ID

*

Q1 **FOR TEST ADMINISTRATOR ONLY:**

Enter Subject ID ACROSTIC:

_____

*

Q2 **FOR TEST ADMINISTRATOR ONLY:**

Enter Subject ID Number:

_____

End of Block: Subject ID

Start of Block: Fatigue Rating (PRE)

Q3 (Pre-test rating):
Compared to how you normally feel and function each day, give a rating between 0 and 100 of how tired you feel right now, with 0 being extremely exhausted and 100 being completely awake and alert _____

0 10 20 30 40 50 60 70 80 90 100

Fatigue Rating (0: extremely exhausted; 100: completely awake and alert) ()	

End of Block: Fatigue Rating (PRE)

Start of Block: Demographics

Q4 Please select the option which best describes you:
Contact/collision sports include: Boxing, American Football, Lacrosse, Ice Hockey, Rugby, Soccer, and Wrestling
Physically Active: 150 minutes/week of moderate physical activity, or 75 minutes/week of vigorous physical activity.

- ○ I have NEVER played contact/collision sports and I am NOT physically active (1)
- ○ I have NEVER played contact/collision sports, but I AM physically active (2)
- ○ I HAVE played contact/collision sports, but have STOPPED; I AM physically active (3)
- ○ I am STILL participating in organized contact/collision sports (4)

Page Break

Q5 What is your city of birth?

_____

Q6 Country of Birth?

- ○ USA (1)
- ○ Other (2) _____

Q7 What is your age? (years)

_____

Q8 Sex assigned at birth?

- ○ Male (0)
- ○ Female (1)
- ○ Other (2) _____

Q9 Current Gender

- ○ Male (0)
- ○ Female (1)
- ○ Transgender (2)

---

*

Q10 What is your height (inches?)

_____

---

*

Q11 What is your weight (lbs)?

_____

---

*

Q12 What is your e-mail for future contact? [optional]

_____

End of Block: Demographics

Start of Block: Age of First Exposure/Organized Sport History

Q13 In your lifetime, how many **YEARS** (not seasons) did you play the following sport(s)? If you did not play an organized sport(s) you may leave it blank.

- American Football (1) _____
- Badminton (2) _____
- Baseball (3) _____
- Basketball (4) _____
- Boxing (5) _____
- Crew (Rowing) (6) _____
- Cricket (7) _____
- Cross Country Running (8) _____
- Cycling (9) _____
- Dance (10) _____
- Diving (11) _____
- Equestrian/Horse Racing (12) _____
- Fencing (13) _____
- Field Hockey (14) _____
- Flag Football (15) _____
- Golf (16) _____

○ Gymnastics (17) _____

○ Handball (18) _____

○ Ice Hockey (19) _____

○ Lacrosse (20) _____

○ Rugby (21) _____

○ Skiing (22) _____

○ Soccer (Football) (23)
_____

○ Softball (24) _____

○ Snowboarding (25)
_____

○ Swimming (26) _____

○ Tennis (27) _____

○ Track & Field (28)
_____

○ Volleyball (29) _____

○ Wrestling (30) _____

Page Break

Q14 At what age did you **START** playing the following sport(s)? If you did not play an organized sport(s) you may leave it blank.

- American Football (1) _____
- Badminton (2) _____
- Baseball (3) _____
- Basketball (4) _____
- Boxing (5) _____
- Crew (Rowing) (6) _____
- Cricket (7) _____
- Cross Country Running (8) _____
- Cycling (9) _____
- Dance (10) _____
- Diving (11) _____
- Equestrian/Horse Racing (12) _____
- Fencing (13) _____
- Field Hockey (14) _____
- Flag Football (15) _____
- Golf (16) _____

○ Gymnastics (17) _____

○ Handball (18) _____

○ Ice Hockey (19) _____

○ Lacrosse (20) _____

○ Rugby (21) _____

○ Skiing (22) _____

○ Soccer (Football) (23)
_____

○ Softball (24) _____

○ Snowboarding (25)
_____

○ Swimming (26) _____

○ Tennis (27) _____

○ Track & Field (28)
_____

○ Volleyball (29) _____

○ Wrestling (30) _____

Page Break

End of Block: Age of First Exposure/Organized Sport History

Start of Block: Medical History

Q15 For <u>every</u> condition below, have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Q16 Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Meningitis

- Yes (1)
- No (0)

Q17
Have you ever been diagnosed by a Physician/MD/DO with: (if you are unsure of the meaning of a condition, please ask the test administrator)

Seizure Disorder

- Yes (1)
- No (0)

Q18
Have you ever been diagnosed by a Physician/MD/DO with:

(if you are unsure of the meaning of a condition, please ask the test administrator)

Diabetes

- Yes (1)
- No (0)

Q19
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Sleep Disorder

- Yes (1)
- No (0)

Page Break

Q20
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Balance Disorder

- ○ Yes (1)
- ○ No (0)

Display This Question:
If Q20 = Yes

Q21 What was your Balance Disorder diagnosis?

- ☐ Vestibular Disorder (1)
- ☐ Vertigo (2)
- ☐ Motion Sickness (4)
- ☐ Meniere's Disease (5)
- ☐ Other (6) _____

Page Break

Q22
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Psychiatric Disorder

- ○ Yes (1)
- ○ No (0)

Display This Question:

If Q22 = Yes

Q23 What was/is the diagnosis for your psychiatric disorder?

- [ ] Unknown (1)
- [ ] Mood Disorder (excluding depression and bipolar disorder) (2)
- [ ] Anxiety Disorder (3)
- [ ] PTSD (4)
- [ ] Somatoform Disorder (5)
- [ ] Alcohol Abuse (6)
- [ ] Drug Abuse (7)
- [ ] Personality Disorder (8)
- [ ] Psychotic Disorder (excluding schizophrenia) (9)
- [ ] Other (10) _____

Page Break

Q24
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Learning Disorder (e.g. dyslexia)

- Yes (1)
- No (0)

Q25
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Attention Deficit-Hyperactivity Disorder (ADD/ADHD)

- Yes (1)
- No (0)

Q26
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Autism Spectrum Disorder

- Yes (1)
- No (0)

Q27
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Depression

- Yes (1)
- No (0)

Q28
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Bipolar Disorder

- Yes (1)
- No (0)

Q29
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Schizophrenia

- Yes (1)
- No (0)

Q30
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Moderate/Severe Traumatic Brain Injury

- Yes (1)
- No (0)

Q31
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Brain Surgery

- Yes (1)
- No (0)

Q32
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Vision Problems (other than glasses/contacts)

- Yes (1)
- No (0)

Q33
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Hearing Problems

- Yes (1)
- No (0)

Q34
Have you ever been diagnosed by a Physician/MD/DO with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Stroke

- Yes (1)
- No (0)

Page Break

Q35 For every condition below have you or a family member ever been diagnosed by a Physician/MD with:
(if you are unsure of the meaning of a condition, please ask the test administrator)

Q36 Please check the box if you or a family member have ever been diagnosed by a Physician/MD with any of the following. If no one has been diagnosed, please leave it blank.

	You (0)	Mother/Father (1)	Sister/Brother (2)	Grandparent (3)
Headaches Disorder (non-migraine) (1)	☐	☐	☐	☐
Migraine Headaches (2)	☐	☐	☐	☐
Parkinson's Disease (3)	☐	☐	☐	☐
Memory Disorder (4)	☐	☐	☐	☐
Alzheimer's Disease (5)	☐	☐	☐	☐
Other non-Alzhemier's Dementia (6)	☐	☐	☐	☐
Mild Cognitive Impairment (7)	☐	☐	☐	☐

Page Break

Q37 Have you ever been under general anesthesia?

- Yes (1)
- No (0)

Display This Question:

If Q37 = Yes

Q38 How many times have you been under general anesthesia?

_____

Q39 Average number of hours of sleep each night (Sunday through Thursday)?

_____

Q40 Average number of hours of sleep each night (Friday and Saturday)?

_____

End of Block: Medical History

Start of Block: Medications

Q41 Are you currently taking prescription medications?

- Yes (1)
- No (0)

Display This Question:

If Q41 = Yes

Q42 What prescription medications are you currently taking? (check all that apply)

- ☐ Antidepressants (1)
- ☐ Anti-anxiety (2)
- ☐ Anti-psychotic (3)
- ☐ Narcotic pain medication (4)
- ☐ Non-narcotic pain medication (5)
- ☐ Sleep aid/sedative (6)
- ☐ Psychostimulant (7)
- ☐ Birth Control (8)
- ☐ Allergy (9)
- ☐ Asthma (10)
- ☐ Acid Reflux/heart burn (11)
- ☐ Other (12) _____

Display This Question:

If Q41 = Yes

Q43 Please provide the name(s) of the prescription medication(s) you are currently taking:

_____

Q44 Are you regularly taking any over-the-counter medications (e.g., Advil/Ibuprofen, Claritin, etc.)

- ○ Yes (1)
- ○ No (0)

Display This Question:

If Q44 = Yes

Q45 What over-the-counter medications are you taking. Please check all that apply.

- ☐ Advil/Ibuprofen (1)
- ☐ Tylenol/Acetaminophen (2)
- ☐ Claritin/Allergy medication (3)
- ☐ Other (4) _____

Q46 Are you taking any over-the-counter supplements (e.g., protein or vitamins)?

- ○ Yes (1)
- ○ No (0)

Display This Question:

If Q46 = Yes

Q47 What over-the-counter supplements are you taking? Please check all that apply.

- ☐ Protein (1)
- ☐ Creatine (2)
- ☐ DHEA (3)
- ☐ Chromium (4)
- ☐ Androstenedione (5)
- ☐ Vitamins (6)
- ☐ Weight loss (7)
- ☐ Other (8) _____

Q48 Have you ever used tobacco (e.g., smoked, dipped) in the past month?

- ○ Yes (1)
- ○ No (0)

Display This Question:

If Q48 = Yes

Q49 How many cigarettes/cigars per week?

_____

Display This Question:

If Q48 = Yes

Q50 How many cans of dip per week?

_____

Q51 Have you used marijuana in the past month?

- Yes (1)
- No (0)

Display This Question:

If Q51 = Yes

Q52 How much per week? (grams)

_____

Q53 Have you used alcohol in the past month?

- Yes (1)
- No (0)

Display This Question:

If Q53 = Yes

Q54 On average, how many **days per week** over the last month did you drink?

_____

Display This Question:

If Q53 = Yes

Q55 On those days, what is the **average number of drinks you consumed**?

_____

End of Block: Medications

Start of Block: Interest and Preference Survey

Q56 Please select the option which best agrees with your interests and preferences for each statement below:

	Strongly disagree (1)	Disagree (2)	Neither disagree or agree (3)	Agree (4)	Strongly agree (5)
I would like to explore strange places (1)	○	○	○	○	○
I get restless when I spend too much time at home (2)	○	○	○	○	○
I like to do frightening things (3)	○	○	○	○	○
I like wild parties (4)	○	○	○	○	○
I would like to take off on a trip with no pre-planned routes or timetables (5)	○	○	○	○	○
I prefer friends who are excitingly unpredictable (6)	○	○	○	○	○
I would like to try bungee jumping (7)	○	○	○	○	○

I would love to have new and exciting experiences, even if they are illegal (8)	○	○	○	○	○

End of Block: Interest and Preference Survey

Start of Block: Other

Q57 Are you financially **independent** of your parent(s)/guardian(s) (i.e., your parent(s)/guardian(s) **do not** claim you as a dependent?

- ○ Yes (1)
- ○ No (0)

Display This Question:

If Q57 = Yes

Q58 Estimated family income?

- $0-30,000 (1)
- $39,991-60,000 (2)
- $60,001-90,000 (3)
- $90,001-120,000 (4)
- $120,001-150,000 (5)
- $150,001-180,000 (6)
- $180,001-210,000 (7)
- $210,001-240,000 (8)
- $240,001-270,000 (9)
- $270,001-300,000 (10)
- +$300,000 (11)
- Unknown (12)

Display This Question:

If Q57 = No

Q59 Estimated family income of parent(s)/guardian(s)?

- ○ $0-30,000 (1)
- ○ $39,991-60,000 (2)
- ○ $60,001-90,000 (3)
- ○ $90,001-120,000 (4)
- ○ $120,001-150,000 (5)
- ○ $150,001-180,000 (6)
- ○ $180,001-210,000 (7)
- ○ $210,001-240,000 (8)
- ○ $240,001-270,000 (9)
- ○ $270,001-300,000 (10)
- ○ +$300,000 (11)
- ○ Unknown (12)

Display This Question:

If Q57 = Yes

Q60 What is your highest level of completed education?

- Unknown (1)
- K through 7th grade (2)
- 8th or 9th grade (3)
- Partial High School (10th or 11th) (4)
- High School Graduate (5)
- Partial College (at least 1 year) (6)
- College Degree (7)
- Graduate Degree (8)

Display This Question:

If Q57 = Yes

Q61 What is your occupation? (If retired choose profession held throughout most working years)

- ○ Unknown/Unemployed (1)
- ○ Student (2)
- ○ Farm/day laborer or Unskilled/service worker (3)
- ○ Machine operator, semi-skilled worker (4)
- ○ Skilled manual worker, craftsperson, police/fire, enlisted/non-commissioned officer (5)
- ○ Clerical/sales, small farm owner (6)
- ○ Technician, semiprofessional supervisor, office manager (7)
- ○ Small business owner, farm owner, teacher, low level manager, salaried worked (8)
- ○ Mid-level manager or professional (ex: architect, engineer, accountant, attorney), mid-sized business owner, military officer (9)
- ○ Senior manager or professional (ex: physician, college professor, minister), owner or CEO of large business (10)
- ○ Other (11) _____

Display This Question:

If Q57 = No

Q62 Mother/Guardian 1 highest level of completed education?

- Unknown (1)
- K through 7th grade (2)
- 8th or 9th grade (3)
- Partial High School (10th or 11th) (4)
- High School Graduate (5)
- Partial College (at least 1 year) (6)
- College Degree (7)
- Graduate Degree (8)

Display This Question:

If Q57 = No

Q63 Mother/Guardian 1 occupation? (If retired/deceased choose profession held throughout most working years)

- ○ Unknown/Unemployed (1)
- ○ Farm/day laborer or Unskilled/service worker (2)
- ○ Machine operator, semi-skilled worker (3)
- ○ Skilled manual worker, craftsperson, police/fire, enlisted/non-commissioned officer (4)
- ○ Clerical/sales, small farm owner (5)
- ○ Technician, semiprofessional supervisor, office manager (6)
- ○ Small business owner, farm owner, teacher, low level manager, salaried worked (7)
- ○ Mid-level manager or professional (ex: architect, engineer, accountant, attorney), mid-sized business owner, military officer (8)
- ○ Senior manager or professional (ex: physician, college professor, minister), owner or CEO of large business (9)
- ○ Other (10) _____

Display This Question:

If Q57 = No

Q64 Father/Guardian 2 highest level of completed education?

- Unknown (1)
- K through 7th grade (2)
- 8th or 9th grade (3)
- Partial High School (10th or 11th) (4)
- High School Graduate (5)
- Partial College (at least 1 year) (6)
- College Degree (7)
- Graduate Degree (8)

Display This Question:

If Q57 = No

Q65 Father/Guardian 2 Occupation (If retired/deceased choose profession held throughout most working years)

- ○ Unknown/Unemployed (1)
- ○ Farm/day laborer or Unskilled/service worker (2)
- ○ Machine operator, semi-skilled worker (3)
- ○ Skilled manual worker, craftsperson, police/fire, enlisted/non-commissioned officer (4)
- ○ Clerical/sales, small farm owner (5)
- ○ Technician, semiprofessional supervisor, office manager (6)
- ○ Small business owner, farm owner, teacher, low level manager, salaried worked (7)
- ○ Mid-level manager or professional (ex: architect, engineer, accountant, attorney), mid-sized business owner, military officer (8)
- ○ Senior manager or professional (ex: physician, college professor, minister), owner or CEO of large business (9)
- ○ Other (10) _____

End of Block: Other

Start of Block: Assessments and Determinants of Concussion Recovery

Q66 Please answer the following questions as honestly as possible. Your answers will remain confidential. Please answer the following questions about your injury history:

Q67
Have you ever suffered a concussion? (diagnosed and undiagnosed)
A concussion is a traumatic brain injury induced by biomechanical forces to the head,

face, neck, or elsewhere on the body with an impulsive force transmitted to the head resulting in clinical signs and symptoms. (e.g., headache, nausea, dizziness, fogginess, loss of balance, light or noise sensitivity, just not feeling "right", etc.)

- ○ Yes (1)
- ○ No (0)

Display This Question:

If Q67 = Yes

Q68 How many concussions have you suffered?

_____

Display This Question:

If Q67 = Yes

Q69 Please list the dates of **ALL** your concussions.

	Month (e.g., Dec = 12) (1)	Year (e.g, 2011) (2)
Concussion #1 (1)		
Concussion #2 (2)		
Concussion #3 (3)		
Concussion #4 (4)		
Concussion #5 (5)		
Concussion #6 (6)		
Concussion #7 (7)		
Concussion #8 (8)		

Concussion #9 (9)		
Concussion #10 (10)		

Q70 Have you ever sprained or otherwise injured your ankle, foot, or knee?

- Yes (1)
- No (0)

Display This Question:
If Q70 = Yes

Q71 When was the last sprain injury? (month/year)

_____

Q72 Have you ever broken a bone in your foot or leg?

- Yes (1)
- No (0)

Display This Question:

If Q72 = Yes

Q73 When was your most recent broken bone in the foot or leg? (month/year)

_____

X→

Q74 Do you have any known balance/metabolic/neurological disorders?

- ○ Yes (Please explain) (1)

_____

- ○ No (0)

X→

Q75 Are you currently taking any medication which affects your balance or cognitive thinking?

- ○ Yes (1)
- ○ No (0)

Page Break

Q76 Have you ever been hit in the head and "knocked silly" or "seen stars"?

- Yes (1)
- No (0)

Display This Question:

If Q76 = Yes

Q77 How many times have you been hit in the head and "knocked silly" or "seen stars"?

_____

Page Break

Q78 Have you ever been knocked unconscious or lost your memory after getting hit in the head?

- Yes (1)
- No (0)
- Unknown (2)

Display This Question:

If Q78 = Yes

Q79 How many times were you knocked unconscious or lost your memory after getting hit in the head?

_____

End of Block: Assessments and Determinants of Concussion Recovery

Start of Block: BU Head Impact Exposure Assessment

Q80 Have you ever participated in organized sports?

Note: Organized is not merely pick-up or neighborhood games; it would include membership on a team, with scheduled practices and games.

- Yes (1)
- No (2)

Skip To: End of Block If Q80 = No

Q81 Have you ever played organized tackle football in your life?

Note: Organized is not merely pick-up or neighborhood games; it would include membership on a team, with scheduled practices and games, including but not limited to, Pop Warner, USA football, Town league, and any school team

- ○ Yes (1)
- ○ No (0)

Skip To: Q106 If Q81 = No

Q82 At what age did you start playing football? (e.g., 5, 18) Please enter the age (years)

_____

Q83 At what age did you stop playing football? (years) [if sill participating enter your current age]

_____

Q84 Did you play football professionally or semi-professionally? Please note that this DOES NOT include college football.

- ○ Yes (1)
- ○ No (2)

Q85 Did you play COLLEGE football?

- ○ Yes (1)
- ○ No (2)

Skip To: Q92 If Q85 = No

Q86 How many FALL seasons of College Football have you played?

- ○ 0 (1)
- ○ 1 (2)
- ○ 2 (3)
- ○ 3 (4)
- ○ 4 (5)

Q87 How many SPRING seasons of College Football have you played?

- ○ 0 (1)
- ○ 1 (2)
- ○ 2 (3)
- ○ 3 (4)

Q88 What was your PRIMARY position while playing COLLEGE Football

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25)

_____

Q89 What was your SECOND most played position in COLLEGE Football

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25)

_____

- ○ None (26)

Q90 What was your THIRD most played position in COLLEGE Football

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25) _____
- ○ None (26)

Q91 Thinking of all your COLLEGE Football experience, what percentage of all the downs (offensive/defensive/special teams) did you play? (only count plays your unit was on the field, if you played offense, only the percentage of offensive plays)

	0 10 20 30 40 50 60 70 80 90 100
Primary Position ()	
Second Position ()	
Third Position ()	

Q92 Did you play HIGH SCHOOL football?

- ○ Yes (1)
- ○ No (2)

Skip To: Q100 If Q92 = No

Q93 How many Fall Seasons did you play tackle football in high school? (Fall = Regular seasons, inclusive of late summer preseason through playoffs)

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)

Q94 How many Fall Seasons did you play tackle football in high school? (Fall = Regular seasons, inclusive of late summer preseason through playoffs)

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)

Q95 How many Spring Seasons did you play tackle football in high school? (Spring = additional season with pads on)

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)

Q96 What was your PRIMARY position while playing HIGH SCHOOL Football

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25)

_____

Q97 What was your SECOND most played position in HIGH SCHOOL Football

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25)

_____

- ○ None (26)

Q98 What was your THIRD most played position in HIGH SCHOOL Football

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25) _____
- ○ None (26)

Q99 Thinking of all your HIGH SCHOOL Football experience, what percentage of all the downs (offensive/defensive/special teams) did you play? (only count plays your unit was on the field, if you played offense, only the percentage of offensive plays)

	0 10 20 30 40 50 60 70 80 90 100
Primary Position ()	
Second Position ()	
Third Position ()	

Q100 Did you play organized tackle/YOUTH football before high school (not merely pick-up games)?

- ○ Yes (1)
- ○ No (2)

Skip To: Q106 If Q100 = No

Q101 How many seasons of YOUTH (before high school) Football did you play?

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)
- 6 (6)
- 7 (7)
- 8 (8)
- 9 (9)
- 10 (10)
- 11 (11)
- 12 (12)
- 13 (13)
- 14 (14)
- 15 (15)

Q102 What was your primary position before high school?

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25)

_____

Q103 What was your second most played position? (pick only one)

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25)

_____

- ○ None (26)

Q104 What was your third most played position? (pick only one)

- Tackle (1)
- Guard (2)
- Center (3)
- Tight End (4)
- Wide Receiver (5)
- Quarterback (6)
- Running Back-Halfback/tailback (7)
- Running Back-Fullback (8)
- Defensive Line- End (9)
- Defensive Line - Tackle (10)
- Linebacker - Weak Side/Outside (11)
- Linebacker - Middle/Inside (12)
- Linebacker - Strong Side/Outside (13)
- Cornerback (14)
- Cornerback-nickleback (15)
- Strong Safety (16)
- Free Safety (17)
- Defensive Back-Dime (18)
- Defense - Elephant (19)

- ○ Place Kicker (20)
- ○ Punter (21)
- ○ Special Teams - Gunner (22)
- ○ Special Teams - Wedge Blocker (23)
- ○ Special Teams - Kick Returner (24)
- ○ Other (please describe) (25) _____
- ○ None (26)

Q105 Thinking of all your YOUTH (before High School) Football experience, what percentage of all the downs (offensive/defensive/special teams) did you play? (only count plays your unit was on the field, if you played offense, only the percentage of offensive plays)

	0 10 20 30 40 50 60 70 80 90 100
Primary Position ()	
Second Position ()	
Third Position ()	

Page Break

Q106 Have you ever played organized SOCCER at any level?

- ○ Yes (1)
- ○ No (2)

Skip To: Q125 If Q106 = No

Q107 At what age did you START playing SOCCER?

_____

Q108 At what age did you STOP playing SOCCER? [if sill participating enter your current age]

_____

Q109 Did you play post-college ADULT club soccer?

- ○ Yes (1)
- ○ No (3)

Skip To: Q111 If Q109 = No

Q110 For how many seasons did you play soccer for your ADULT club team?

_____

Q111 Did you play collegiate/varsity soccer?

- ○ Yes (1)
- ○ No (3)

Q112 For how many seasons did you play soccer for your college team?

- ○ 0 (1)
- ○ 1 (2)
- ○ 2 (3)
- ○ 3 (4)
- ○ 4 (5)
- ○ 5 (6)

Q113 What was your primary position while playing COLLEGE soccer

- ○ Center Back (1)
- ○ Sweeper (2)
- ○ Full-back/Right-back/Left-back (3)
- ○ Wingback (4)
- ○ Other Defender (5) _____
- ○ Midfielder (6)
- ○ Center Midfielder (7)
- ○ Defensive Midfielder (8)
- ○ Winger (9)
- ○ Forward (10)
- ○ Striker (11)
- ○ Secondary Striker (12)
- ○ Other Offensive Player (13) _____
- ○ Goalie (14)
- ○ Other (15) _____

Q114 Did you participate in heading drills when you played COLLEGE soccer

- Yes (1)
- No (2)
- Not Sure (3)

Q115 Did you play soccer for your high school team?

- Yes (1)
- No (2)

Skip To: Q117 If Q115 = No

Q116 For how many seasons did you play soccer for your high school team?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q117 Did you play soccer for a club or town team DURING your high school years?

- Yes (1)
- No (2)

Skip To: Q121 If Q117 = No

Q118 For how many seasons did you play soccer for a club team during your high school years?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q119 What was your primary position while playing HIGH SCHOOL soccer

- ○ Center Back (1)
- ○ Sweeper (2)
- ○ Full-back/Right-back/Left-back (3)
- ○ Wingback (4)
- ○ Other Defender (5) _____
- ○ Midfielder (6)
- ○ Center Midfielder (7)
- ○ Defensive Midfielder (8)
- ○ Winger (9)
- ○ Forward (10)
- ○ Striker (11)
- ○ Secondary Striker (12)
- ○ Other Offensive Player (13) _____
- ○ Goalie (14)
- ○ Other (15) _____

Q120 Did you participate in heading drills at the high school level?

- ○ Yes (1)
- ○ No (2)
- ○ Unsure (3)

Q121 Did you play organized soccer YOUTH (not merely pick-up games) prior to high school?

- ○ Yes (1)
- ○ No (2)

Skip To: Q125 If Q121 = No

Q122 How many seasons/years of YOUTH soccer (before high school) did you play? (summer and fall seasons in the same year would be 1 season.)

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)
- 6 (6)
- 7 (7)
- 8 (8)
- 9 (9)
- 10 (10)
- 11 (11)
- 12 (12)
- 13 (13)
- 14 (14)
- 15 (15)

Q123 What was your primary position while playing youth (before high school) soccer

- ○ Center Back (1)
- ○ Sweeper (2)
- ○ Full-back/Right-back/Left-back (3)
- ○ Wingback (4)
- ○ Other Defender (5)

_____

- ○ Midfielder (6)
- ○ Center Midfielder (7)
- ○ Defensive Midfielder (8)
- ○ Winger (9)
- ○ Forward (10)
- ○ Striker (11)
- ○ Secondary Striker (12)
- ○ Other Offensive Player (13)

_____

- ○ Goalie (14)
- ○ Other (15) _____

Q124 Did you participate in heading drills at the youth level?

- ○ Yes (1)
- ○ No (2)
- ○ Unsure (3)

Page Break

Q125 Did you ever play organized ICE HOCKEY?

- ○ Yes (1)
- ○ No (2)

Skip To: Q138 If Q125 = No

Q126 At what age did you START playing ICE HOCKEY?

_____

Q127 At what age did you STOP playing ICE HOCKEY? [if sill participating enter your current age]

_____

Q128 Did you play adult (post-college) club ICE HOCKEY?

- ○ Yes (1)
- ○ No (2)

Skip To: Q130 If Q128 = No

Q129 For how many seasons did you adult club ICE HOCKEY?

_____

Q130 Did you play ICE HOCKEY in college?

- Yes (1)
- No (2)

Skip To: Q131 If Q130 = No

Q131 For how many seasons did you play ICE HOCKEY in college?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q132 Did you play hockey for your high school team or club level hockey during your high school years?

- Yes (1)
- No (2)

Skip To: Q135 If Q132 = No

Q133 For how many seasons did you play hockey for your high school or club team?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q134 What was your primary position while playing hockey during your high school years?

- Left Wing (1)
- Right Wing (2)
- Winger (3)
- Center (4)
- Forward (5)
- Left Defense (6)
- Right Defense (7)
- Defensemen (8)
- Goalie (9)
- Other (10) _____

Q135 Did you play organized/youth hockey (not merely pick-up games) PRIOR to high school?

- ○ Yes (1)
- ○ No (2)

Skip To: Q138 If Q135 = No

Q136 What was your primary position while playing hockey during your YOUTH ice hockey years?

- ○ Left Wing (1)
- ○ Right Wing (2)
- ○ Winger (3)
- ○ Center (4)
- ○ Forward (5)
- ○ Left Defense (6)
- ○ Right Defense (7)
- ○ Defensemen (8)
- ○ Goalie (9)
- ○ Other (10) _____

Q137 How many seasons of YOUTH ice hockey (before high school) did you play? (Summer and Winter seasons in the same year would be 1 season.)

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)
- 6 (6)
- 7 (7)
- 8 (8)
- 9 (9)
- 10 (10)
- 11 (11)
- 12 (12)
- 13 (13)
- 14 (14)
- 15 (15)

Page Break

Q138 Did you ever play organized LACROSSE

- ○ Yes (1)
- ○ No (2)

Skip To: Q151 If Q138 = No

Q139 At what age did you START playing LACROSSE?

_____

Q140 At what age did you STOP playing LACROSSE?

_____

Q141 Did you play adult (post-college) club LACROSSE?

- ○ Yes (1)
- ○ No (2)

Skip To: Q143 If Q141 = Yes

Q142 For how many seasons did you adult club LACROSSE?

_____

Q143 Did you play LACROSSE in college?

- Yes (1)
- No (2)

Skip To: Q145 If Q143 = No

Q144 For how many seasons did you play LACROSSE in college?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q145 Did you play lacrosse for your high school team?

- Yes (1)
- No (2)

Skip To: Q148 If Q145 = No

Q146 For how many seasons did you play lacrosse for your high school team?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q147 What was your primary position while playing lacrosse during your high school years?

- Defense (1)
- Midfielder (2)
- Attack (3)
- Goalie (4)
- Other (5) _____

Q148 Did you play organized/youth lacrosse (not merely pick-up games) prior to high school?

- Yes (1)
- No (2)

Skip To: Q151 If Q148 = No

Q149 What was your primary position while playing lacrosse during your youth lacrosse years?

- ○ Defense (1)
- ○ Midfield (2)
- ○ Attack (3)
- ○ Goalie (4)
- ○ Other (5) _____

Q150 How many seasons of YOUTH lacrosse (before high school) did you play? (Spring and Summer seasons in the same year would be 1 season.)

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)
- 6 (6)
- 7 (7)
- 8 (8)
- 9 (9)
- 10 (10)
- 11 (11)
- 12 (12)
- 13 (13)
- 14 (14)
- 15 (15)

Page Break

Page Break

Q151 Did you ever play organized RUGBY?

- ○ Yes (1)
- ○ No (2)

Skip To: Q166 If Q151 = No

Q152 At what age did you START playing RUGBY?

_____

Q153 At what age did you STOP playing RUGBY? [if sill participating enter your current age]

_____

Q154 Did you play adult (post-college) club rugby?

- ○ Yes (1)
- ○ No (2)

Skip To: Q157 If Q154 = No

Q155 For how many seasons did you adult club rugby?

_____

Q156 What was your primary position while playing rugby for your adult club team?

- Prop (1)
- Hooker (2)
- Flanker (3)
- Lock (4)
- 8-person (5)
- Scrum Half (6)
- Fly Half/Half back (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)

Q157 Did you play rugby in college?

- Yes (1)
- No (2)

Skip To: Q160 If Q157 = No

Q158 For how many seasons did you play rugby in college?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q159 What was your primary position while playing rugby during your college years?

- Prop (1)
- Hooker (2)
- Flanker (3)
- Lock (4)
- 8-person (5)
- Scrum Half (6)
- Fly Half/Half back (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)

Q160 Did you play rugby for your high school team?

- Yes (1)
- No (2)

Skip To: Q163 If Q160 = No

Q161 For how many seasons did you play rugby for your high school team?

- 0 (1)
- 1 (2)
- 2 (3)
- 3 (4)
- 4 (5)
- 5 (6)

Q162 What was your primary position while playing rugby during your high school years?

- Prop (1)
- Hooker (2)
- Flanker (3)
- Lock (4)
- 8-person (5)
- Scrum Half (6)
- Fly Half/Half back (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)

Q163 Did you play organized/youth rugby (not merely pick-up games) prior to high school?

- Yes (1)
- No (2)

Skip To: Q166 If Q163 = No

Q164 What was your primary position while playing rugby during your youth rugby years?

- Prop (1)
- Hooker (2)
- Flanker (3)
- Lock (4)
- 8-person (5)
- Scrum Half (6)
- Fly Half/Half back (7)
- Inside Center (8)
- Outside Center (9)
- Wing (10)
- Fullback (11)

Q165 How many seasons of YOUTH rugby (before high school) did you play? (Spring and Summer seasons in the same year would be 1 season.)

- 1 (1)
- 2 (2)
- 3 (3)
- 4 (4)
- 5 (5)
- 6 (6)
- 7 (7)
- 8 (8)
- 9 (9)
- 10 (10)
- 11 (11)
- 12 (12)
- 13 (13)
- 14 (14)
- 15 (15)

Page Break

Q166 What other competitive (not recreational) sports did you play in your lifetime? (please list the number of **YEARS** in the text box)

- [ ] Baseball (1) _____
- [ ] Basketball (2) _____
- [ ] Cross Country/Distance Running (3) _____
- [ ] Crew (4) _____
- [ ] Cycling (5) _____
- [ ] Diving (6) _____
- [ ] Equestrian (7) _____
- [ ] Diving (8) _____
- [ ] Field Hockey (9) _____
- [ ] Golf (10) _____
- [ ] Gymnastics (11) _____
- [ ] Horse Jumping (12) _____

- [ ] Powerlifting (13)
- [ ] Skiing (14)
- [ ] Softball (15)
- [ ] Swimming (16)
- [ ] Tennis (17)
- [ ] Track and Field (18)
- [ ] Water Polo (19)
- [ ] Other Sport (name of sport and years played) (20)
- [ ] Other Sport (name of sport and years played) (21)
- [ ] Other Sport (name of sport and years played) (22)

End of Block: BU Head Impact Exposure Assessment

Start of Block: SCAT 5 Symptoms

Q167
Please rate your symptoms based on how you typically feel:

	None (0) (0)	Mild (1) (1)	Mild (2) (2)	Moderate (3) (3)	Moderate (4) (4)	Severe (5) (5)	Severe (6) (6)
Headache (1)	○	○	○	○	○	○	○
"Pressure in head" (2)	○	○	○	○	○	○	○
Neck Pain (3)	○	○	○	○	○	○	○
Nausea or vomiting (4)	○	○	○	○	○	○	○
Dizziness (5)	○	○	○	○	○	○	○
Blurred vision (6)	○	○	○	○	○	○	○
Balance problems (7)	○	○	○	○	○	○	○
Sensitivity to light (8)	○	○	○	○	○	○	○
Sensitivity to noise (9)	○	○	○	○	○	○	○
Feeling slowed down (10)	○	○	○	○	○	○	○
Feeling like "in a fog" (11)	○	○	○	○	○	○	○
"Don't feel right" (12)	○	○	○	○	○	○	○
Difficulty concentrating (13)	○	○	○	○	○	○	○

Difficulty remembering (14)	○	○	○	○	○	○	○
Fatigue or low energy (15)	○	○	○	○	○	○	○
Confusion (16)	○	○	○	○	○	○	○
Drowsiness (17)	○	○	○	○	○	○	○
More emotional (18)	○	○	○	○	○	○	○
Irritability (19)	○	○	○	○	○	○	○
Sadness (20)	○	○	○	○	○	○	○
Nervous or Anxious (21)	○	○	○	○	○	○	○
Trouble falling asleep (if applicable) (22)	○	○	○	○	○	○	○

End of Block: SCAT 5 Symptoms

Start of Block: SF-12

Q168 In general, would you say your health is:

- Excellent (1) (1)
- Very Good (2) (2)
- Good (3) (3)
- Fair (4) (4)
- Poor (5) (5)

Q169 The following two questions are about activities you might do during a typical day. Does YOUR HEALTH NOW LIMIT YOU in these activities? If so, how much?

Q170 MODERATE ACTIVITIES, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf:

- Yes, Limited A Lot (1) (1)
- Yes, Limited A Little (2) (2)
- No, Not Limited At All (3) (3)

Q171 Climbing SEVERAL flights of stairs:

- Yes, Limited A Lot (1) (1)
- Yes, Limited A Little (2) (2)
- No, Not Limited At All (3) (3)

Q172 During the PAST 4 WEEKS have you had any of the following problems with your work or other regular activities AS A RESULT OF YOUR PHYSICAL HEALTH?

Q173 ACCOMPLISHED LESS than you would like:

- Yes (1) (1)
- No (2) (2)

Q174 Were limited in the KIND of work or other activities:

- Yes (1) (1)
- No (2) (2)

Q175 During the PAST 4 WEEKS, were you limited in the kind of work you do or other regular activities AS A RESULT OF ANY EMOTIONAL PROBLEMS (such as feeling depressed or anxious)?

Q176 ACCOMPLISHED LESS than you would like:

- Yes (1) (1)
- No (2) (2)

Q177 Didn’t do work or other activities as CAREFULLY as usual:

- ○ Yes (1) (1)
- ○ No (2) (2)

Q178 During the PAST 4 WEEKS, how much did PAIN interfere with your normal work (including both work outside the home and housework)?

- ○ Not At All (1) (1)
- ○ A Little Bit (2) (2)
- ○ Moderately (3) (3)
- ○ Quite A Bit (4) (4)
- ○ Extremely (5) (5)

Q179 The next three questions are about how you feel and how things have been DURING THE PAST 4 WEEKS. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the PAST 4 WEEKS –

Q180 Have you felt calm and peaceful?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

Q181 Did you have a lot of energy?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

Q182 Have you felt downhearted and blue?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

Q183 During the PAST 4 WEEKS, how much of the time has your PHYSICAL HEALTH OR EMOTIONAL PROBLEMS interfered with your social activities (like visiting with friends, relatives, etc.)?

- All of the Time (1) (1)
- Most of the Time (2) (2)
- A Good Bit of the Time (3) (3)
- Some of the Time (4) (4)
- A Little of the Time (5) (5)
- None of the Time (6) (6)

End of Block: SF-12

Start of Block: SWLS

Q184 **Instructions**: Below are five statements that you may agree or disagree with. Using the 1 - 7 scale below, indicate your agreement with each item by placing the

appropriate number on the line preceding that item. Please be open and honest in your responding.

	7- Strongly agree (7)	6- Agree (6)	5- Somewhat agree (5)	4- Neither agree nor disagree (4)	3- Somewhat disagree (3)	2- Disagree (2)	1- Strongly disagree (1)
In most ways my life is close to my ideal. (1)	○	○	○	○	○	○	○
The conditions of my life are excellent. (2)	○	○	○	○	○	○	○
I am satisfied with my life. (3)	○	○	○	○	○	○	○
So far I have gotten the important things I want in life. (4)	○	○	○	○	○	○	○
If I could live my life over, I would change almost nothing. (5)	○	○	○	○	○	○	○

End of Block: SWLS

Start of Block: Apathy Evaluation Scale (Self-Rated)

Q185 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I am interested in things.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q186 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I get things done during the day.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q187 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

Getting things started on my own is important to me.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q188 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I am interested in having new experiences.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q189 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I am interested in learning new things.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q190 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I put little effort into anything.

- NOT AT ALL (1)
- SLIGHTLY (2)
- SOMEWHAT (3)
- A LOT (4)

Q191 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I approach life with intensity.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q192 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

Seeing a job through to the end is important to me.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q193 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I spend time doing things that interest me.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q194 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

Someone has to tell me what to do each day.

- NOT AT ALL (1)
- SLIGHTLY (2)
- SOMEWHAT (3)
- A LOT (4)

Q195 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I am less concerned about my problems than I should be.

- NOT AT ALL (1)
- SLIGHTLY (2)
- SOMEWHAT (3)
- A LOT (4)

Q196 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I have friends.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q197 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

Getting together with friends is important to me.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q198 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

When something good happens, I get excited.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q199 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I have an accurate understanding of my problems.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q200 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

Getting things done during the day is important to me.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q201 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I have initiative.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

Q202 **Instructions**: For each statement, please select the answer that **best** describes your thoughts, feelings, and activity in the **past 4 weeks.**

I have motivation.

- NOT AT ALL (4)
- SLIGHTLY (3)
- SOMEWHAT (2)
- A LOT (1)

End of Block: Apathy Evaluation Scale (Self-Rated)

Start of Block: Fatigue Rating (Post)

Q203 (Post-test rating)
Compared to how you normally feel and function each day, give a rating between 0 and 100 of how tired you feel right now, with 0 being extremely exhausted and 100 being completely awake and alert _____

0 10 20 30 40 50 60 70 80 90 100

Fatigue Rating (0: extremely exhausted; 100: completely awake and alert) ()	

End of Block: Fatigue Rating (Post)

Appendix H

S.C.R.U.M. DATA COLLECTION FORMS

S.C.R.U.M. Data Collection Form

Examiner's Name:

Individual does not want to be contacted for future studies: □

Online Questionnaires Completed? ☐YES ☐NO

Trail Making Test

Trail Making Test – A (TMT-A)

Practice Time: _____

Practice Errors: _____

TMT-A Time: _____

TMT-A Errors: _____

Trail Making Test – B (TMT-B)

Practice Time: _____

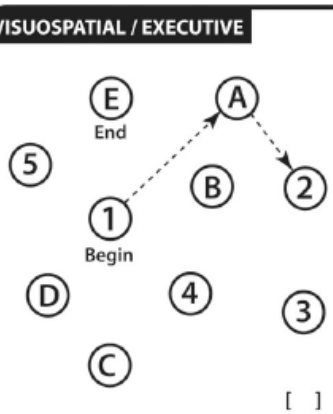
Practice Errors: _____

TMT-B Time: _____

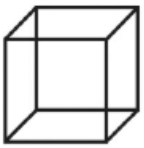
TMT-B Errors: _____

Montreal Cognitive Assessment (MOCA)

VISUOSPATIAL / EXECUTIVE



Copy cube



[ ] [ ]

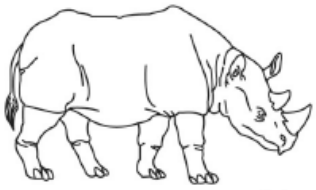
Draw CLOCK (Ten past eleven) (3 points)

[ ] Contour [ ] Numbers [ ] Hands

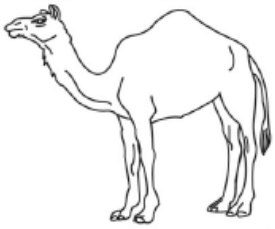
POINTS __/5

NAMING

[ ]



[ ]



[ ]

__/3

MEMORY Read list of words, subject must repeat them. Do 2 trials, even if 1st trial is successful. Do a recall after 5 minutes.

	FACE	VELVET	CHURCH	DAISY	RED
1st trial					
2nd trial					

No points

ATTENTION Read list of digits (1 digit/ sec.).

Subject has to repeat them in the forward order [ ] 2 1 8 5 4

Subject has to repeat them in the backward order [ ] 7 4 2

__/2

Read list of letters. The subject must tap with his hand at each letter A. No points if ≥ 2 errors

[ ] F B A C M N A A J K L B A F A K D E A A A J A M O F A A B

__/1

Serial 7 subtraction starting at 100 [ ] 93 [ ] 86 [ ] 79 [ ] 72 [ ] 65

4 or 5 correct subtractions: **3 pts**, 2 or 3 correct: **2 pts**, 1 correct: **1 pt**, 0 correct: **0 pt**

__/3

LANGUAGE Repeat : I only know that John is the one to help today. [ ]

The cat always hid under the couch when dogs were in the room. [ ]

__/2

Fluency / Name maximum number of words in one minute that begin with the letter F [ ] _____ (N ≥ 11 words)

__/1

ABSTRACTION Similarity between e.g. banana - orange = fruit [ ] train – bicycle [ ] watch - ruler

__/2

DELAYED RECALL

	Has to recall words WITH NO CUE	FACE	VELVET	CHURCH	DAISY	RED	Points for UNCUED recall only
		[ ]	[ ]	[ ]	[ ]	[ ]	
Optional	Category cue						
	Multiple choice cue						

__/5

ORIENTATION [ ] Date [ ] Month [ ] Year [ ] Day [ ] Place [ ] City

__/6

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TOTAL __/30

Add 1 point if ≤ 12 yr edu

Administered by: _____

Oculomotor/Oculovestibular Tests – King-Devick Test

King-Devick Baseline		
	Total Time	Total Errors
Attempt 1		
Attempt 2		
Baseline Time (Faster of Attempts 1 and 2 without error)		

Oculomotor/Oculovestibular Tests – VOMS

Vestibular/Ocular Motor Test	Not Tested	Headache 0-10	Dizziness 0-10	Nausea 0-10	Fogginess 0-10	Comments
BASELINE SYMPTOMS	N/A					
Smooth Pursuits						
Saccades - Horizontal						
Saccades – Vertical						
Convergence (Near Point)						**Near Point(cm)** #1: _____ #2: _____ # 3: _____
VOR – Horizontal						
VOR – Vertical						
Visual Motion Sensitivity Test						

Balance Error Scoring System (BESS)

Error Types
1. Hands lifted off iliac crest
2. Opening eyes
3. Step, stumble, or fall
4. Moving hip into >30 degrees abduction
5. Lifting forefoot or heel
6. Remaining out of test position >5 seconds
BESS is calculated by adding one error point for each error during the six 20-second tests

Which **foot** was tested (i.e., non-dominant foot): ☐ Left ☐ Right		
Participant wore: ☐ Socks ☐ No Socks		
SCORE CARD (# errors)	FIRM Surface	FOAM Surface
Double Leg Stance (feet together)		
Single Leg Stance (non-dominant foot)		
Tandem Stance (non-dominant foot in back)		
Total Scores		
	BESS TOTAL:	

Tandem Gait Score Sheet

	Single-Task Tandem Gait	Dual-Task Tandem Gait	Cognitive Task (e.g., months)	Questions Answers Correctly	Questions Asked
Time Trial 1					
Time Trial 2					
Time Trial 3					
Time Trial 4					
Trials Failed					
Best Time of 4 Trials					

WALKING TRIAL

trial	Words	√ / x	trial	Subtraction √ / x / o = correct/incorrect/omitted	Months	Trial √ / x / o	Trial √ / x / o
___	visit	___	___	85 x6 79 73 67 61 55 49 43 37	December	___	___
___	learn	___	___	64 x7 57 50 43 36 29 22 15 8	November	___	___
___	alert	___	___	95 x6 88 81 74 67 60 53 46 39	October	___	___
___	twist	___	___	71 x6 65 59 53 47 41 35 29 23	September	___	___
___	snack	___	___	98 x7 91 84 77 70 63 56 49 42	August	___	___
___	earth	___	___	66 x7 59 52 45 38 31 24 17 10	July	___	___
___	fence	___	___	61 x6 55 49 43 37 31 25 19 13	June	___	___
___	lemon	___	___	72 x7 65 58 51 44 37 30 23 16	May	___	___
___	crawl	___	___	87 x7 80 73 66 59 52 45 38 31	April	___	___
___	dance	___	___	93 x6 87 81 75 69 63 57 51 45	March	___	___
___	brave	___	___	88 x7 81 74 67 60 53 46 39 32	February	___	___
___	mouse	___	___	70 x7 63 56 49 42 35 28 21 14	January	___	___
___	noble	___	___	69 x6 63 57 51 45 39 33 27 21			
___	guest	___	___	90 x7 83 76 69 62 55 48 41 34			
___	paper	___	___	75 x6 69 63 57 51 45 39 33 27			

Single & Dual Task Gait Score Sheet

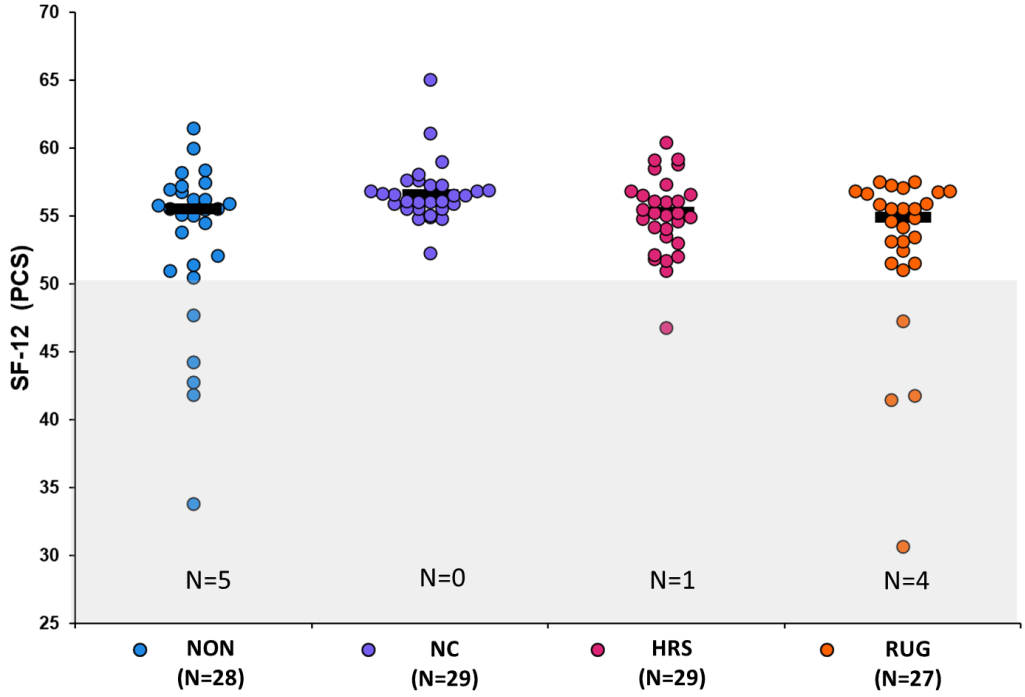
	Single-Task iWalk Gait	Dual-Task iWalk Gait	Cognitive Task (e.g., months)	Questions Answers Correctly	Questions Asked
Trial 1					
Trial 2					
Trial 3					
Trial 4					
Trial 5					

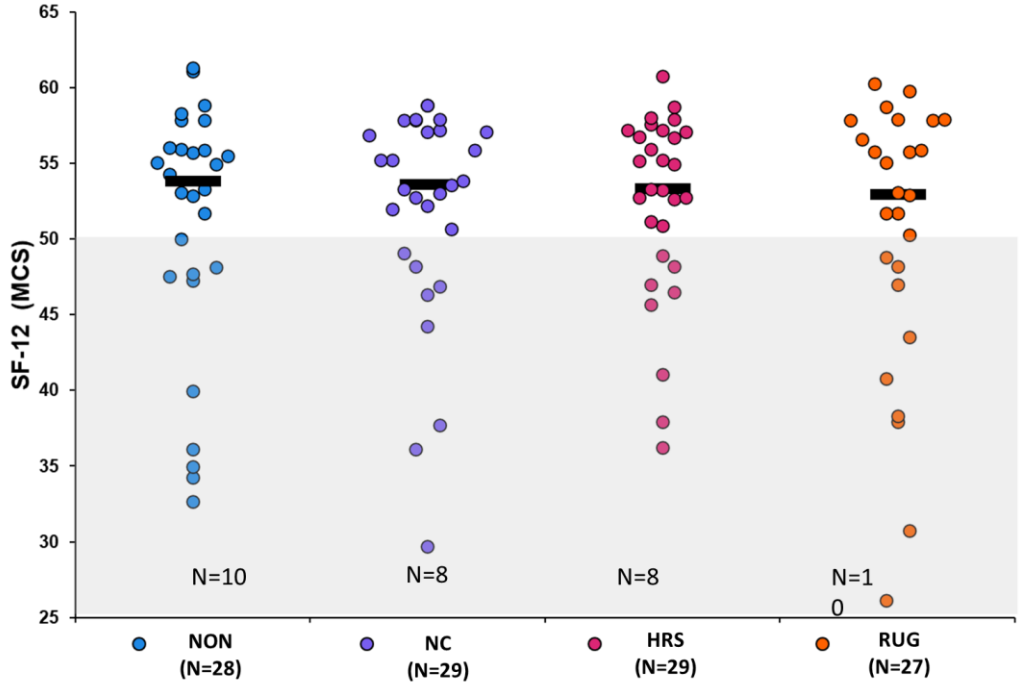
WALKING TRIAL

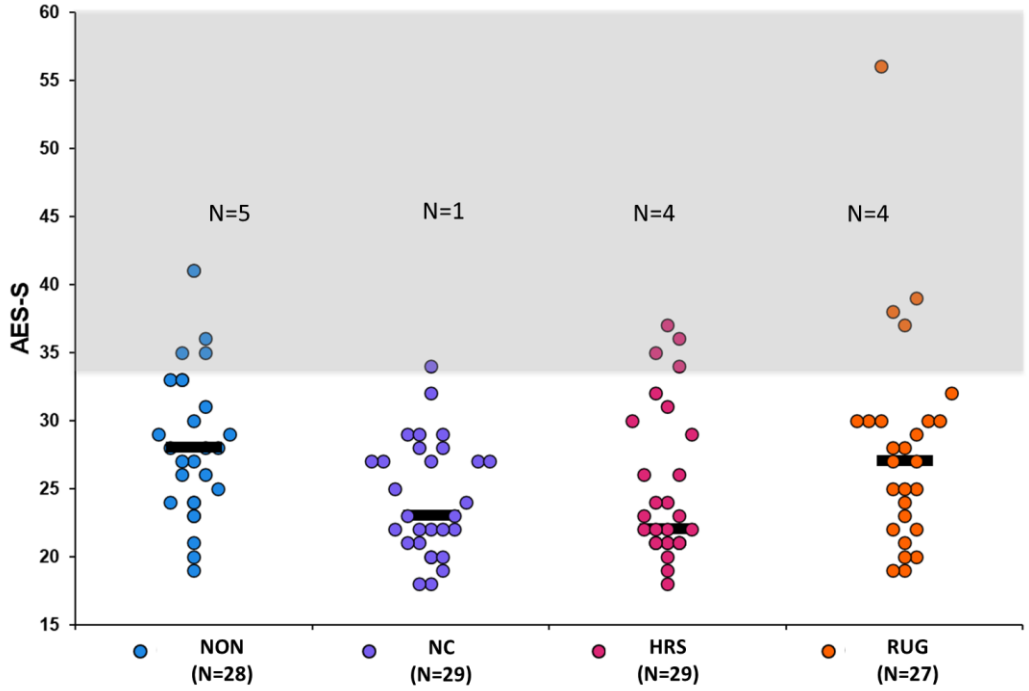
trial	Words	√ / x	trial	Subtraction √ / x / o = correct/incorrect/omitted	Months	Trial √ / x / o	Trial √ / x / o
__	visit	__	__	85 x6 79 73 67 61 55 49 43 37	December	___	___
__	learn	__	__	64 x7 57 50 43 36 29 22 15 8	November	___	___
__	alert	__	__	95 x6 88 81 74 67 60 53 46 39	October	___	___
__	twist	__	__	71 x6 65 59 53 47 41 35 29 23	September	___	___
__	snack	__	__	98 x7 91 84 77 70 63 56 49 42	August	___	___
__	earth	__	__	66 x7 59 52 45 38 31 24 17 10	July	___	___
__	fence	__	__	61 x6 55 49 43 37 31 25 19 13	June	___	___
__	lemon	__	__	72 x7 65 58 51 44 37 30 23 16	May	___	___
__	crawl	__	__	87 x7 80 73 66 59 52 45 38 31	April	___	___
__	dance	__	__	93 x6 87 81 75 69 63 57 51 45	March	___	___
__	brave	__	__	88 x7 81 74 67 60 53 46 39 32	February	___	___
__	mouse	__	__	70 x7 63 56 49 42 35 28 21 14	January	___	___
__	noble	__	__	69 x6 63 57 51 45 39 33 27 21			
__	guest	__	__	90 x7 83 76 69 62 55 48 41 34			
___	paper	____	__	75 x6 69 63 57 51 45 39 33 27			

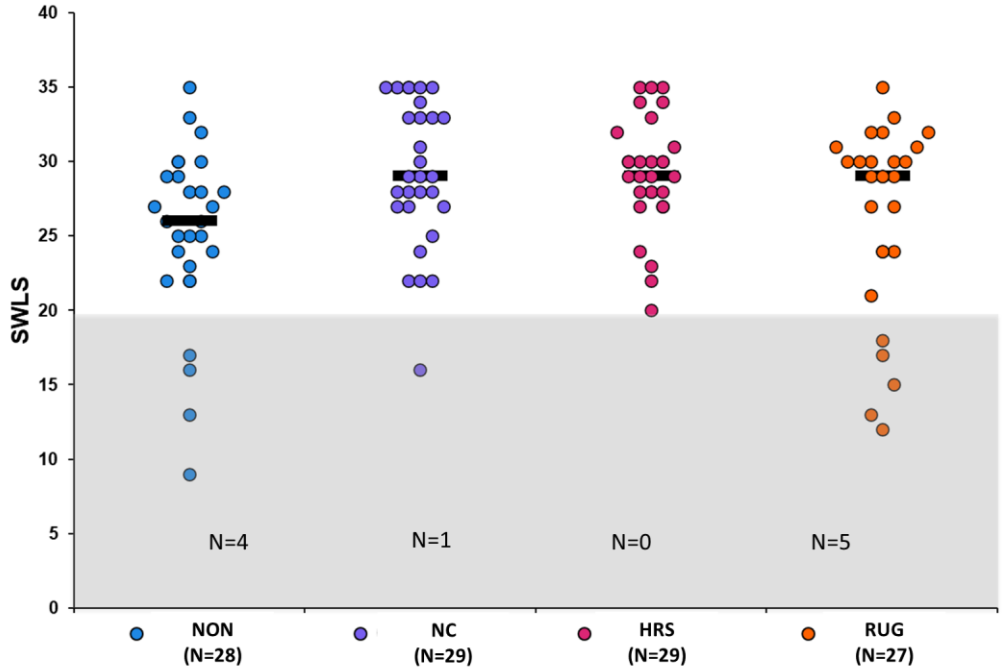
Appendix I

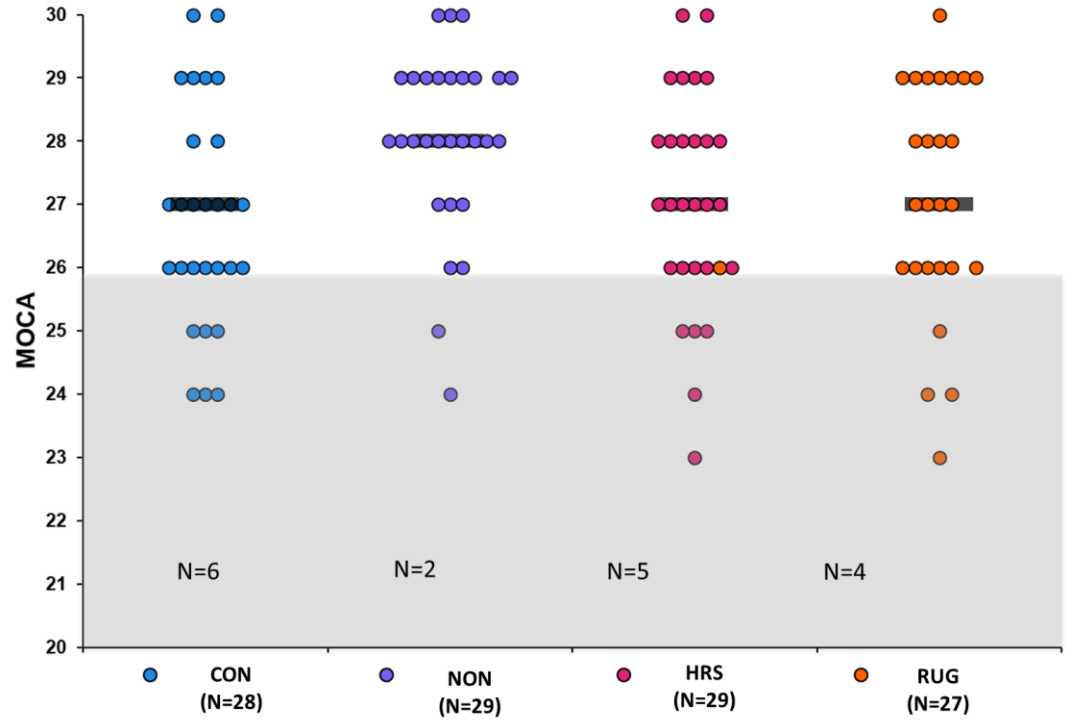
AIM 2 RAW DATA GRAPHS

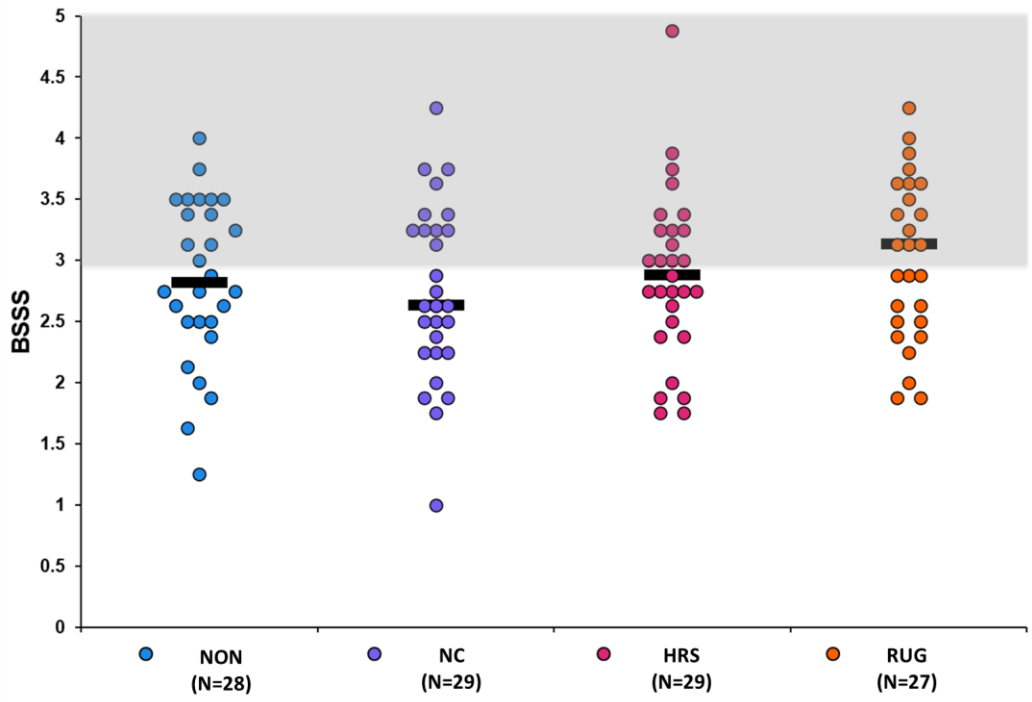


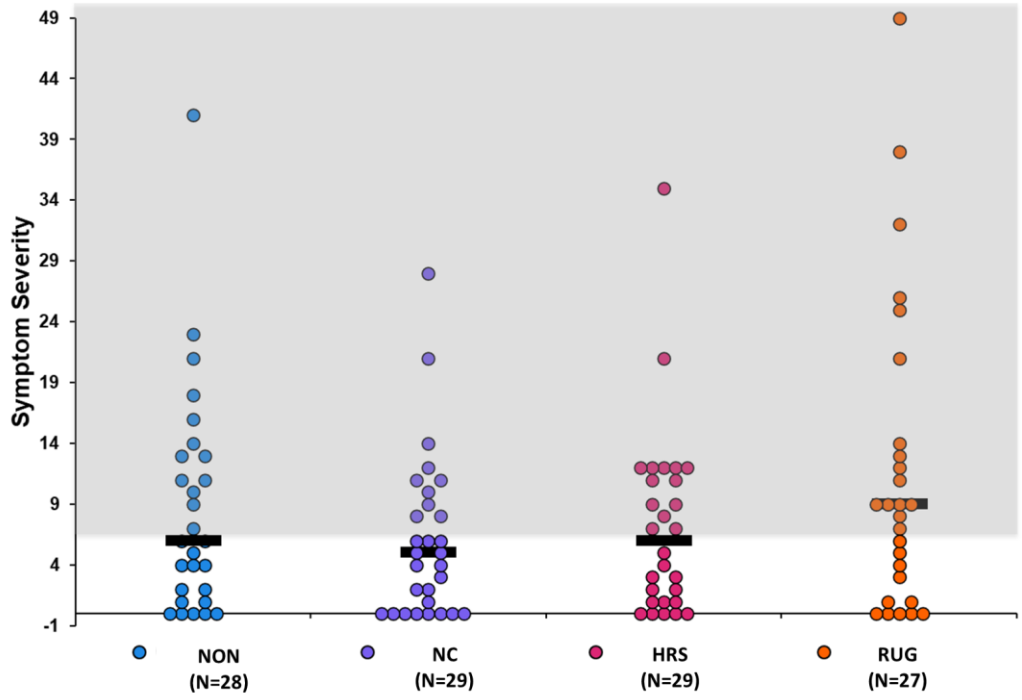


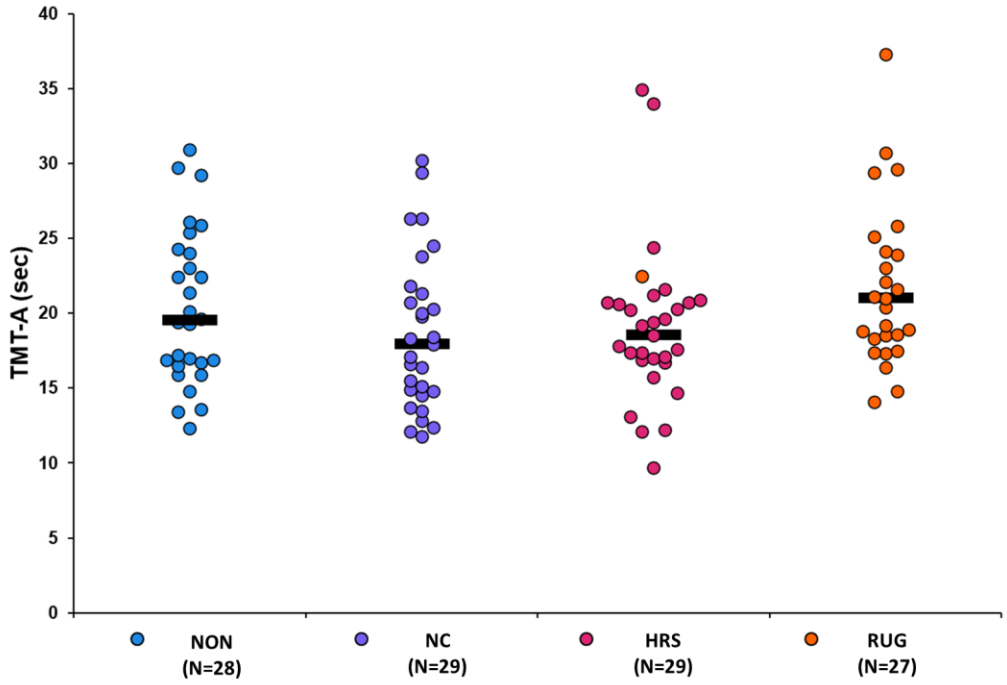

AES-S
60
55
50
45
40
35
30
25
20
15
N=5
N=1
N=4
N=4
NON (N=28)
NC (N=29)
HRS (N=29)
RUG (N=27)

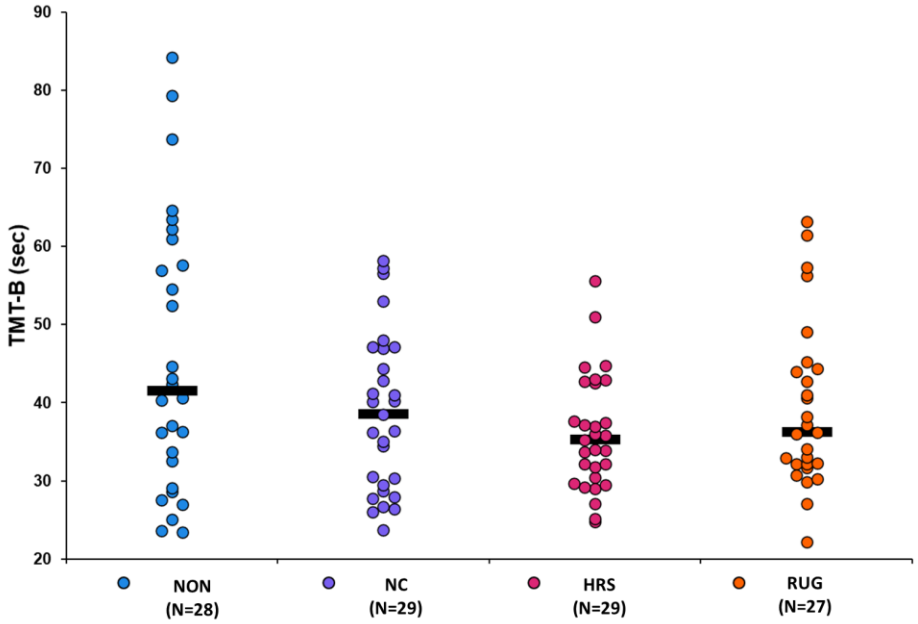

SWLS
40
35
30
25
20
15
10
5
0
N=4
N=1
N=0
N=5
NON (N=28)
NC (N=29)
HRS (N=29)
RUG (N=27)

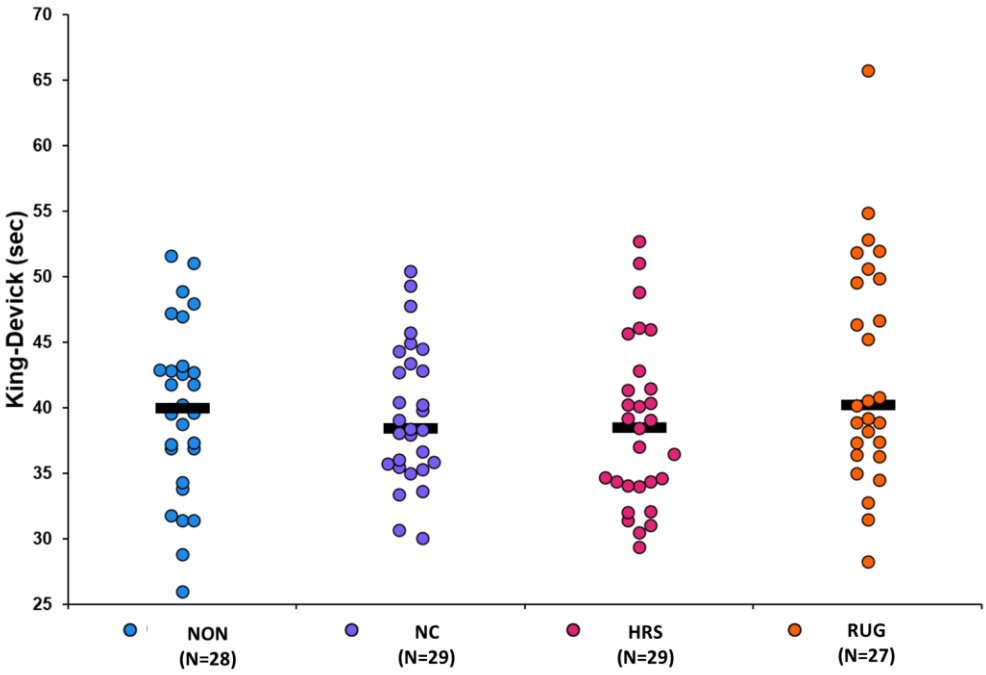

MOCA
30
29
28
27
26
25
24
23
22
21
20
N=6
N=2
N=5
N=4
CON (N=28)
NON (N=29)
HRS (N=29)
RUG (N=27)

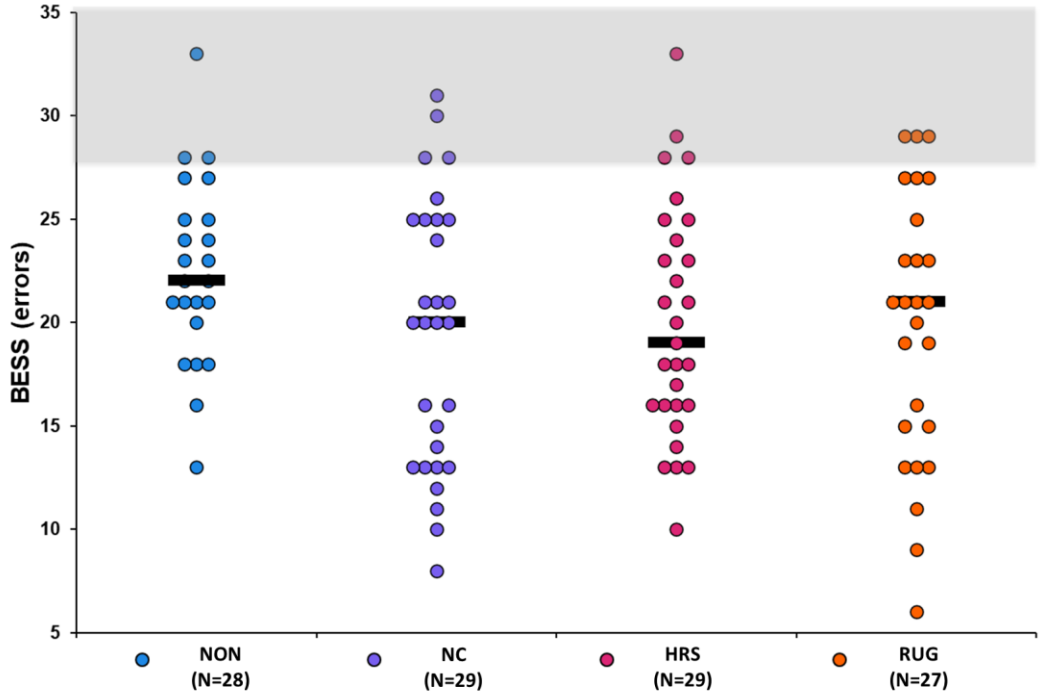

BSSS
5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
NON (N=28)
NC (N=29)
HRS (N=29)
RUG (N=27)

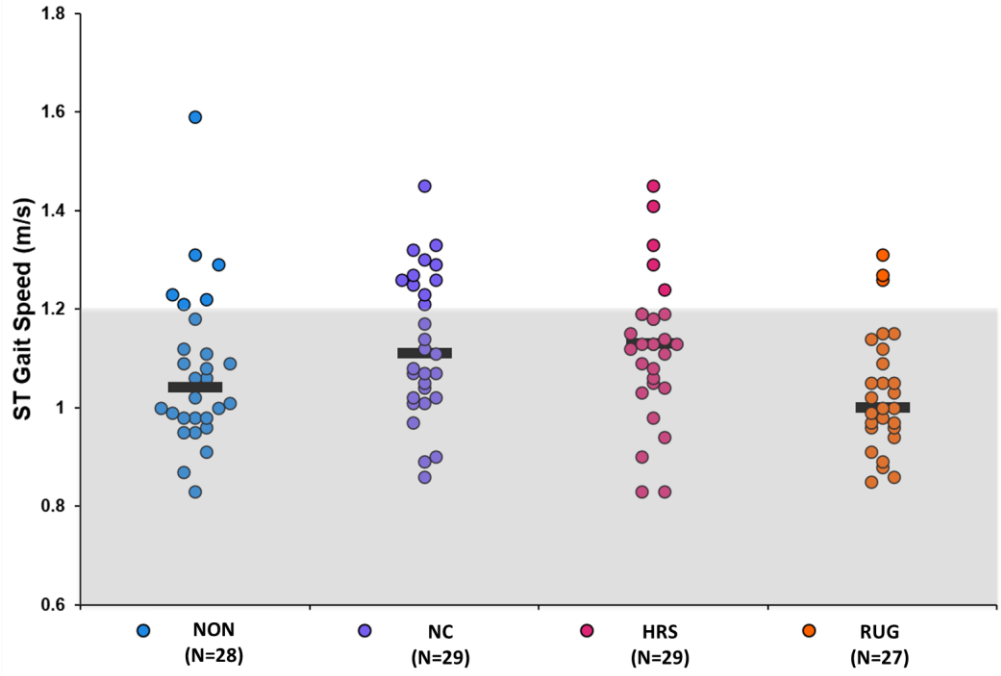

Symptom Severity
49
44
39
34
29
24
19
14
9
4
-1
NON (N=28)
NC (N=29)
HRS (N=29)
RUG (N=27)

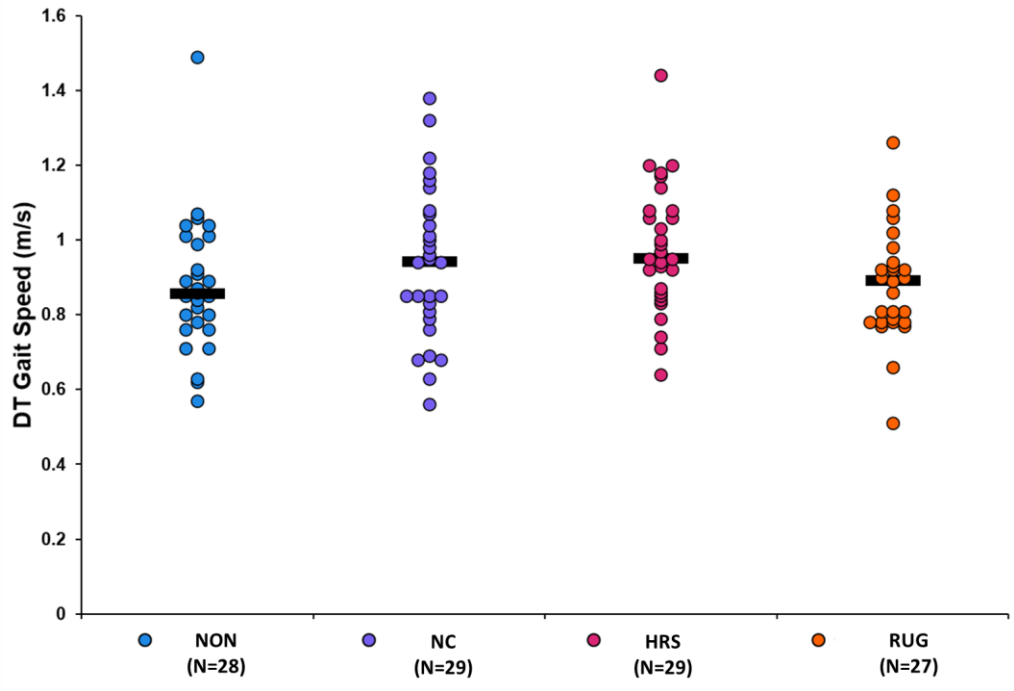

TMT-A (sec)
40
35
30
25
20
15
10
5
0
NON (N=28)
NC (N=29)
HRS (N=29)
RUG (N=27)

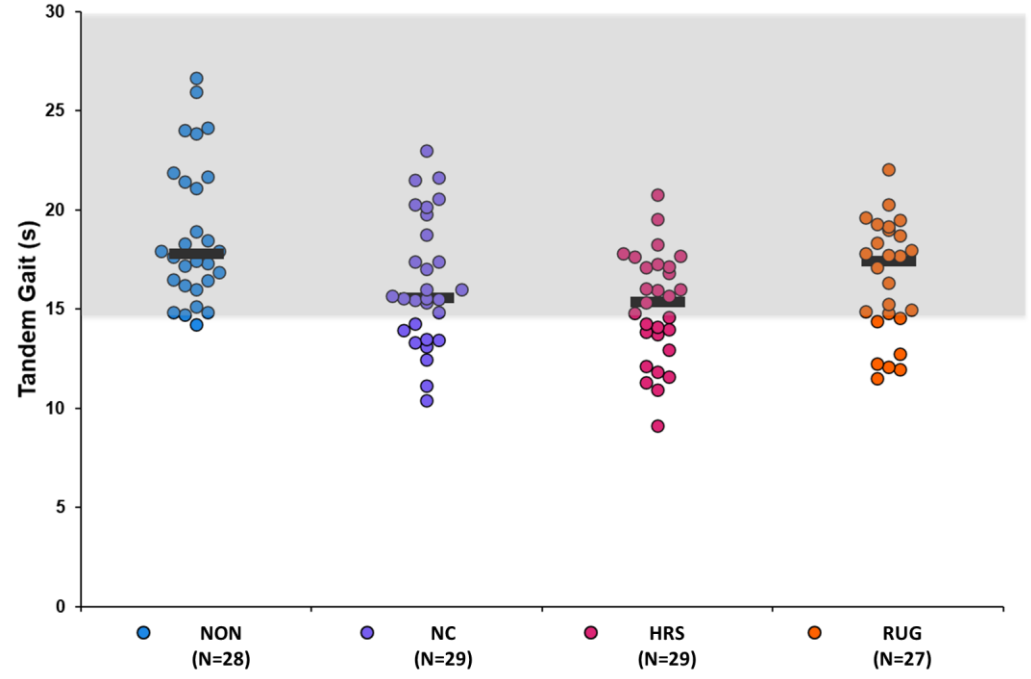

TMT-B (sec)
90
80
70
60
50
40
30
20
NON
(N=28)
NC
(N=29)
HRS
(N=29)
RUG
(N=27)

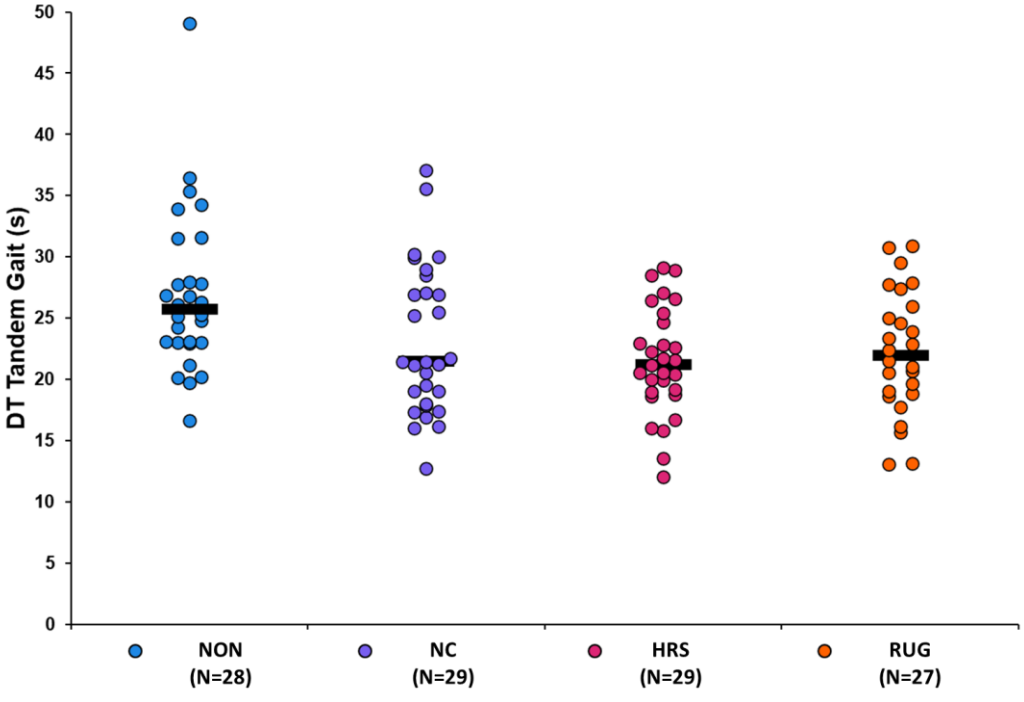

King-Devick (sec)
70
65
60
55
50
45
40
35
30
25
NON
(N=28)
NC
(N=29)
HRS
(N=29)
RUG
(N=27)

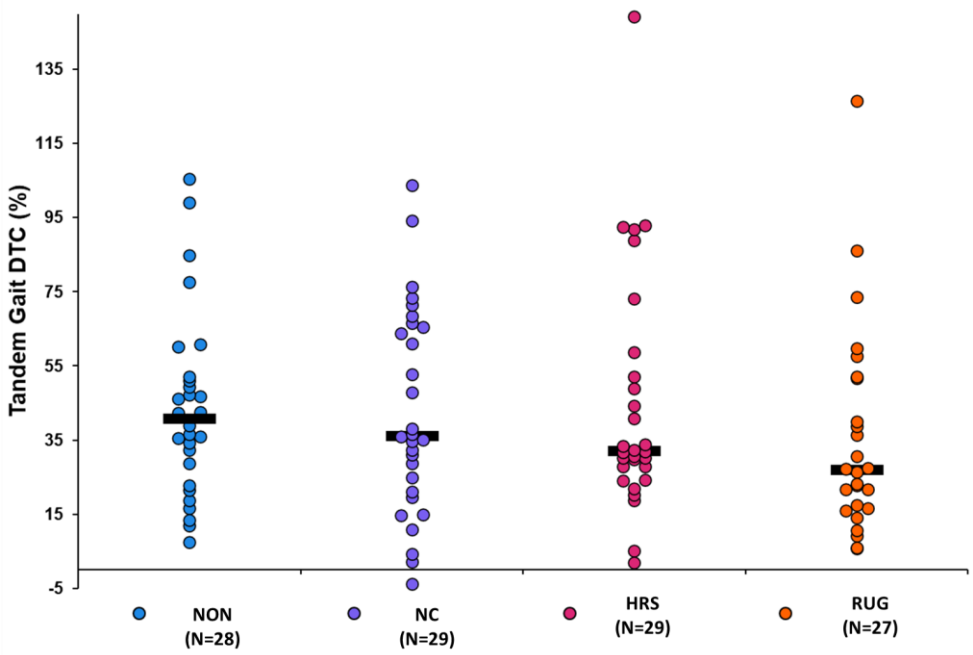

BESS (errors)
35
30
25
20
15
10
5
NON
(N=28)
NC
(N=29)
HRS
(N=29)
RUG
(N=27)


ST Gait Speed (m/s)
1.8
1.6
1.4
1.2
1
0.8
0.6
NON
(N=28)
NC
(N=29)
HRS
(N=29)
RUG
(N=27)


DT Gait Speed (m/s)
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
NON (N=28)
NC (N=29)
HRS (N=29)
RUG (N=27)


Tandem Gait (s)
30
25
20
15
10
5
0
NON (N=28)
NC (N=29)
HRS (N=29)
RUG (N=27)


DT Tandem Gait (s)
50
45
40
35
30
25
20
15
10
5
0
NON
(N=28)
NC
(N=29)
HRS
(N=29)
RUG
(N=27)


Tandem Gait DTC (%)
135
115
95
75
55
35
15
-5
NON
(N=28)
NC
(N=29)
HRS
(N=29)
RUG
(N=27)

Appendix J

AIM 2 SIGNIFICANT FINDINGS SUMMARY

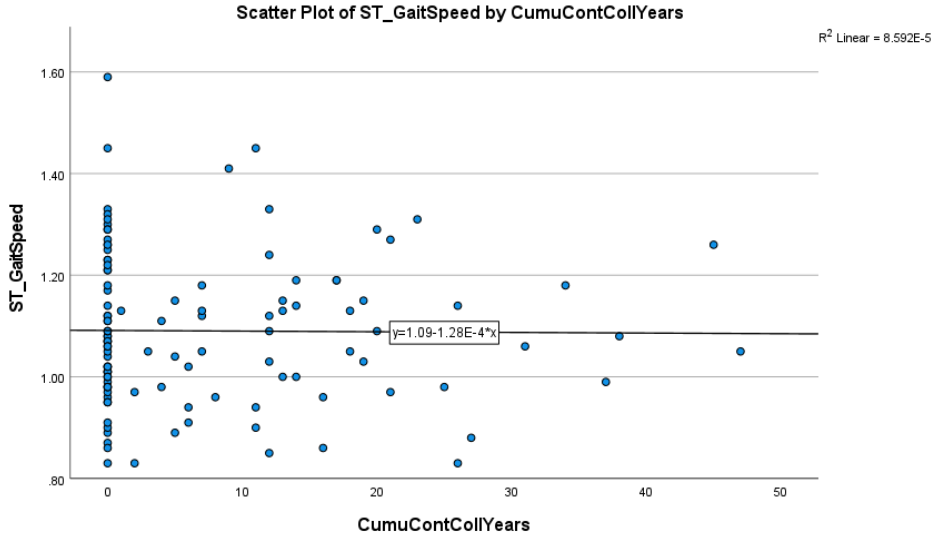
Outcome Measure	Result	Finding	Interpretation
SF-12 (PCS)	ANCOVA: p=0.021, n_p^2=0.088, observed power=0.748	Post hoc differences: NCA>NON	Non physically active individuals with no history of RHI exposure had lower (i.e., worse) self-rated physical health than physically active individuals without a history of RHI.
AES-S	ANCOVA: p=0.008, n_p^2=0.106, observed power=0.836	Post hoc differences: NON>NCA	Non physically active individuals with no history of RHI exposure had higher (i.e., worse) self-rated apathy than physically active individuals without a history of RHI.
SCAT5 Symptom Severity	Spearman Correlation with ST Gait speed: r_s= -0.211, p=0.025	Weak, negative correlation	Individuals with increased symptom severity (i.e., worse) had slower (i.e., worse) ST gait speeds.
SWLS	ANCOVA: p=0.004, n_p^2=0.120, observed power=0.889	Post hoc differences: NCA>NON	Non physically active individuals with no history of RHI exposure had lower (i.e., worse) satisfaction with life than physically active individuals without a history of RHI.
	Spearman Correlation with ST Gait speed: r_s=0.282, p=0.002	Weak, positive correlation	ST Gait speed was weakly, positive correlated with satisfaction with life. Whereby, faster gait speeds were associated with increased satisfaction with one's life.

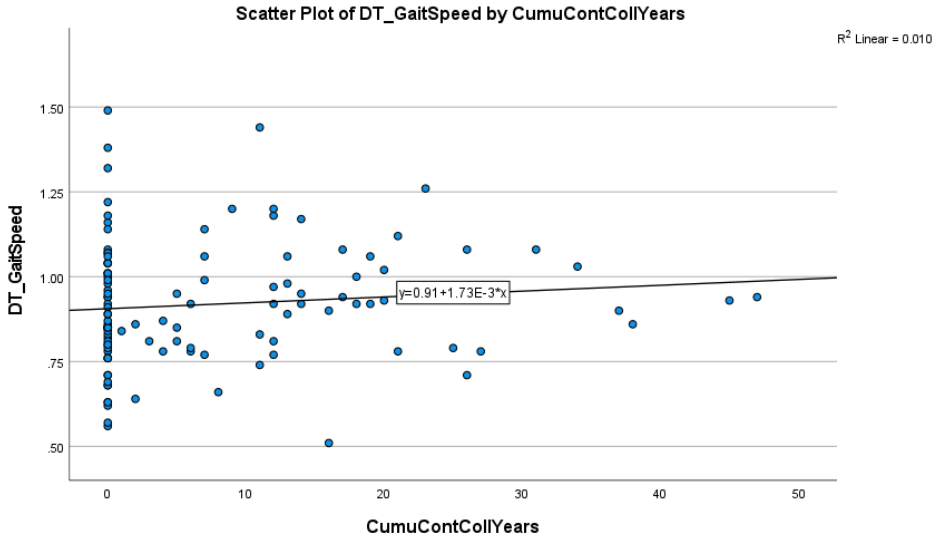
MOCA	ANCOVA: p=0.030, n_p^2=0.082, observed power=0.711	Post hoc differences: NCA>NON	Non physically active individuals with no history of RHI exposure had lower (i.e., worse) cognition than physically active individuals without a history of RHI.
	Spearman Correlation with DTC Gait speed: r_s= 0.248, p=0.008	Weak, positive correlation	As MOCA scores increased (i.e., improved), DTC gait speed increased/got less negative (i.e., improved).
TMT-A	Spearman Correlation with DTC Gait speed: r_s= -0.216, p=0.022	Weak, negative correlation	As TMT-A time increased (i.e., worsened), DTC gait speed decreased/got more negative (i.e., worsened).
TMT-B	Spearman Correlation with ST Gait speed: r_s= -0.219, p=0.021	Weak, negative correlation	As TMT-B time increased (i.e., worsened), ST gait speed decreased (i.e., worsened).
	Spearman Correlation with DTC Gait speed: r_s= -0.306, p<0.001	Weak, negative correlation	As TMT-B time increased (i.e., worsened), DTC gait speed decreased/got more negative (i.e., worsened).
TrailsRatio	ANCOVA: p=0.038, n_p^2=0.077, observed power=0.679	Post hoc differences: NON<RUG	Non physically active individuals with no history of RHI had a smaller TrailsRatio, meaning a greater discrepancy between performance in TMT-A and TMT-B, than those who are physically active with a history of prolonged RHI.
BESS	ANCOVA: p=0.004, n_p^2=0.123, observed power=0.880	Post hoc differences: NON>NCA; NON>RUG	Non physically active individuals with no history of RHI exposure had higher (i.e., worse) balance errors than physically active individuals without a history of RHI, and physically active individuals with a history of prolonged RHI exposure.

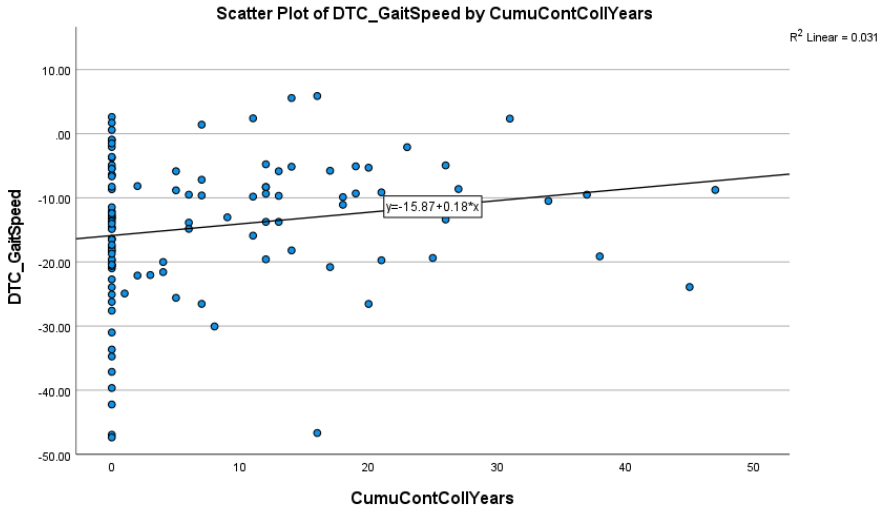
ST Tandem Gait	ANCOVA: p=0.006, n_p^2=0.114, observed power=0.861	Post hoc differences: NON>NCA NON>HRS	Non physically active individuals with no history of RHI exposure had higher (i.e., worse) ST tandem gait times than physically active individuals with a history of no or previous RHI exposure.
DT Tandem Gait	ANCOVA: p=0.003, n_p^2=0.130, observed power=0.911	Post hoc differences: NON>HRS NON>RUG	Non physically active individuals with no history of RHI exposure had higher (i.e., worse) DT tandem gait times than physically active individuals with a history of previous or prolonged RHI exposure.
ST Gait Speed	ANCOVA: p=0.020, n_p^2=0.091, observed power=0.757	Post hoc differences: NCA>RUG	Physically active individuals with a history of no RHI exposure had faster (i.e., better) ST gait speeds than physically active individuals with a history of prolonged RHI exposure.
DT Double Support	ANCOVA: p=0.033, n_p^2=0.081, observed power=0.696	Post hoc differences: HRS<RUG	Physically active individuals with a history of previous RHI exposure spent less time in double support during DT (i.e., less conservative) than physically active individuals with a history of prolonged RHI exposure.

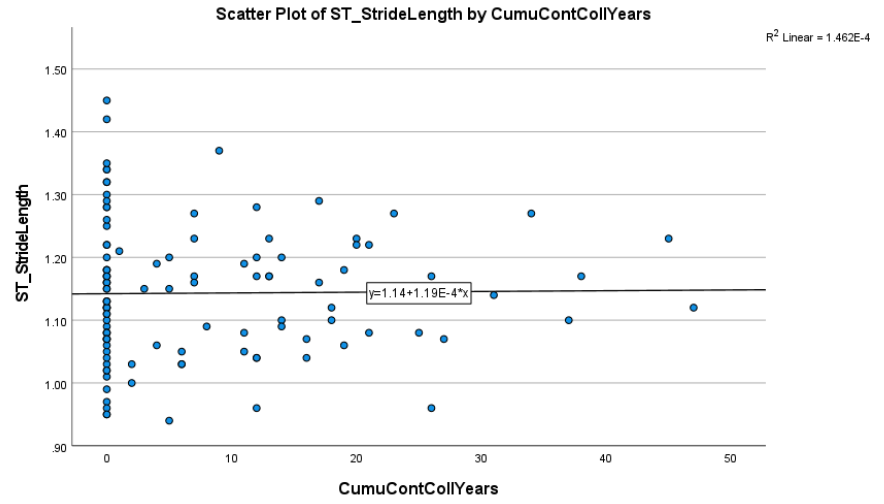
Appendix K

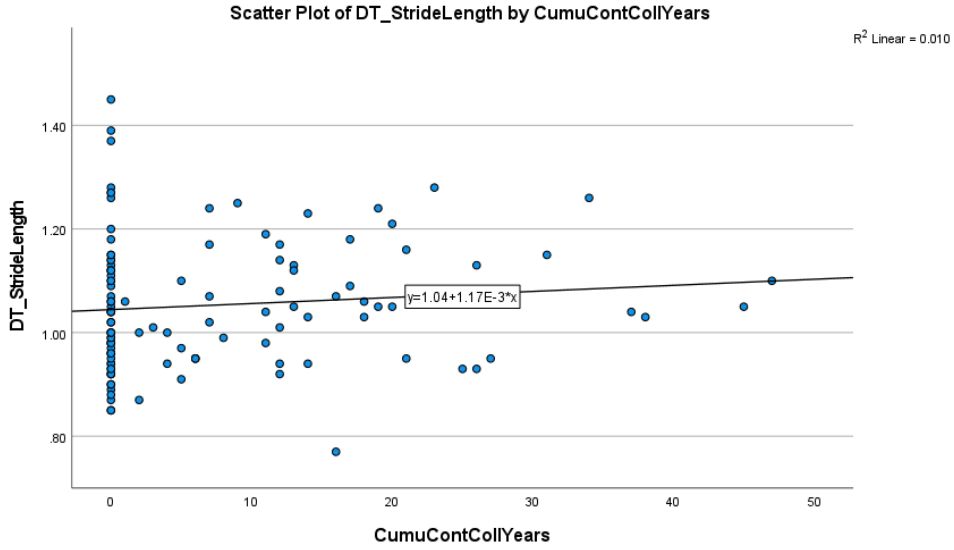
AIM 3 SCATTER PLOTS FOR LINEARITY ASSUMPTIONS

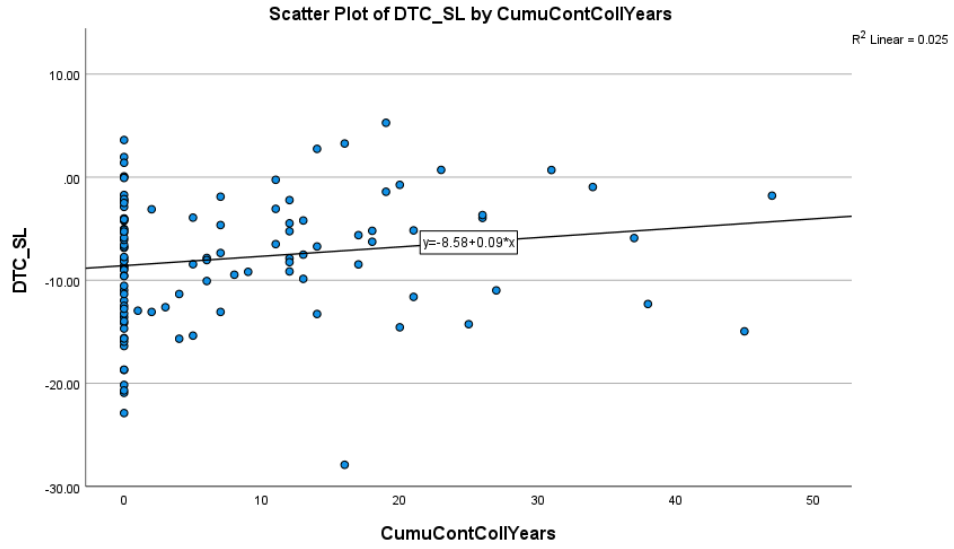


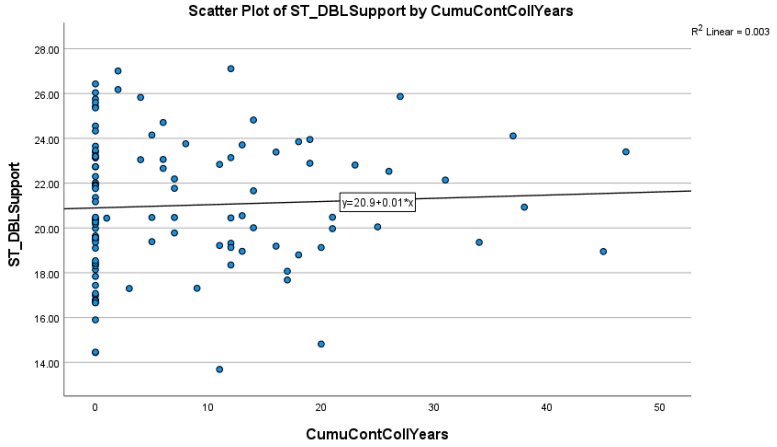


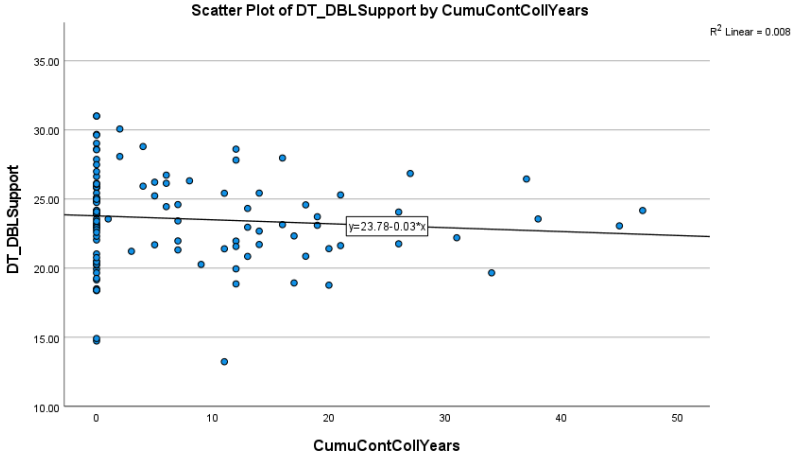

Scatter Plot of DTC_GaitSpeed by CumuContCollYears
R² Linear = 0.031
y=-15.87+0.18*x
DTC_GaitSpeed
CumuContCollYears
10.00
.00
-10.00
-20.00
-30.00
-40.00
-50.00
0
10
20
30
40
50

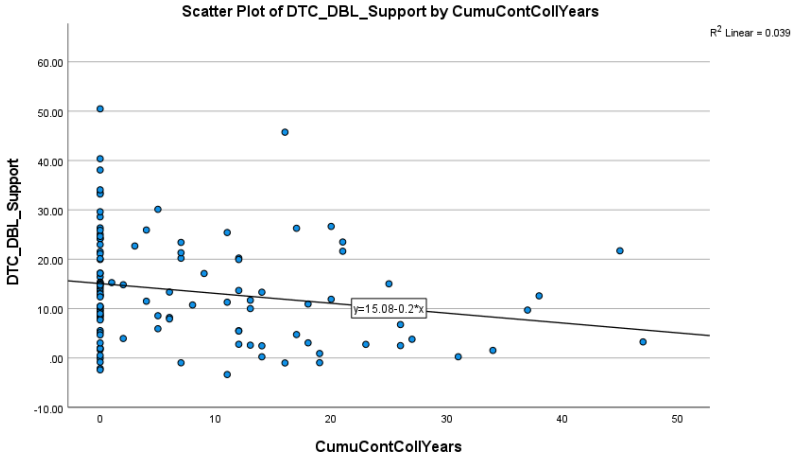

Scatter Plot of ST_StrideLength by CumuContCollYears
R² Linear = 1.462E-4
y=1.14+1.19E-4*x
ST_StrideLength
CumuContCollYears
1.50
1.40
1.30
1.20
1.10
1.00
.90
0
10
20
30
40
50


Scatter Plot of DT_StrideLength by CumuContCollYears
R² Linear = 0.010
DT_StrideLength
1.40
1.20
1.00
.80
y=1.04+1.17E-3*x
0
10
20
30
40
50
CumuContCollYears


Scatter Plot of DTC_SL by CumuContCollYears
R² Linear = 0.025
DTC_SL
10.00
.00
-10.00
-20.00
-30.00
y=-8.58+0.09*x
0
10
20
30
40
50
CumuContCollYears


Scatter Plot of ST_DBLSupport by CumuContCollYears
R² Linear = 0.003
ST_DBLSupport
28.00
26.00
24.00
22.00
20.00
18.00
16.00
14.00
y=20.9+0.01*x
0
10
20
30
40
50
CumuContCollYears


Scatter Plot of DT_DBLSupport by CumuContCollYears
R² Linear = 0.008
DT_DBLSupport
35.00
30.00
25.00
20.00
15.00
10.00
y=23.78-0.03*x
0
10
20
30
40
50
CumuContCollYears


Scatter Plot of DTC_DBL_Support by CumuContCollYears
R² Linear = 0.039
DTC_DBL_Support
60.00
50.00
40.00
30.00
20.00
10.00
.00
-10.00
y=15.08-0.2*x
0
10
20
30
40
50
CumuContCollYears

Appendix L

PERMISSIONS

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Link to Final Publication: https://doi.org/10.1016/j.jsams.2020.10.013

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Appendix M

IRB/HUMAN SUBJECTS APPROVAL

Aim 1 Study Approval Letter:

Institutional Review Board
210H Hullihen Hall
Newark, DE 19716
Phone: 302-831-2137
Fax: 302-831-2828

DATE:	March 6, 2020
TO:	Katherine Hunzinger, MS
FROM:	University of Delaware IRB
STUDY TITLE:	[1540689-3] Rugby Injury Epidemiology
SUBMISSION TYPE:	Amendment/Modification
ACTION:	DETERMINATION OF EXEMPT STATUS
EFFECTIVE DATE:	March 6, 2020
REVIEW CATEGORY:	Exemption category # *(2)*

Thank you for your Amendment/Modification submission to the University of Delaware Institutional Review Board (UD IRB). According to the pertinent regulations, the UD IRB has determined this project is EXEMPT from most federal policy requirements for the protection of human subjects. The privacy of subjects and the confidentiality of participants must be safeguarded as prescribed in the reviewed protocol form.

This exempt determination is valid for the research study as described by the documents in this submission. Proposed revisions to previously approved procedures and documents that may affect this exempt determination must be reviewed and approved by this office prior to initiation. The UD amendment form must be used to request the review of changes that may substantially change the study design or data collected.

Unanticipated problems and serious adverse events involving risk to participants must be reported to this office in a timely fashion according with the UD requirements for reportable events.

A copy of this correspondence will be kept on file by our office. If you have any questions, please contact the UD IRB Office at (302) 831-2137 or via email at hsrb-research@udel.edu. Please include the study title and reference number in all correspondence with this office.

Aim 2/3 Study Approval Letter:

Institutional Review Board
210H Hullihen Hall
Newark, DE 19716
Phone: 302-831-2137
Fax: 302-831-2828

DATE:	September 29, 2021
TO:	Katherine Hunzinger, MS
FROM:	University of Delaware IRB
STUDY TITLE:	[1605665-6] SCRUM (Sub-Concussive impacts in Rugby Union Members)
SUBMISSION TYPE:	Amendment/Modification
ACTION:	APPROVED
EFFECTIVE DATE:	September 29, 2021
NEXT REPORT DUE:	September 8, 2022
REVIEW TYPE:	Expedited Review
REVIEW CATEGORY:	Expedited review category # *(4,7)*

Thank you for your Amendment/Modification submission to the University of Delaware Institutional Review Board (UD IRB). The UD IRB has reviewed and APPROVED the proposed research and submitted documents via Expedited Review in compliance with the pertinent federal regulations.

As the Principal Investigator for this study, you are responsible for, and agree that:

- All research must be conducted in accordance with the protocol and all other study forms as approved in this submission. Any revisions to the approved study procedures or documents must be reviewed and approved by the IRB prior to their implementation. Please use the UD amendment form to request the review of any changes to approved study procedures or documents.
- Informed consent is a process that must allow prospective participants sufficient opportunity to discuss and consider whether to participate. IRB-approved and stamped consent documents must be used when enrolling participants and a written copy shall be given to the person signing the informed consent form.
- Unanticipated problems, serious adverse events involving risk to participants, and all non-compliance issues must be reported to this office in a timely fashion according with the UD requirements for reportable events. All sponsor reporting requirements must also be followed.

The UD IRB REQUIRES the submission of a PROGRESS REPORT DUE ON September 8, 2022. A continuing review/progress report form must be submitted to the UD IRB at least 45 days prior to the due date to allow for the review of that report.

If you have any questions, please contact the UD IRB Office at (302) 831-2137 or via email at hsrb-research@udel.edu. Please include the study title and reference number in all correspondence with this office.