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# Effect of Repetitive Head Impacts on Saccade Performance in Canadian University Football Players

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**Abstract**

**Objective:** Investigate the effect of cumulative head impacts on saccade latency and errors, measured across two successive football seasons. **Design:** Participants were acquired from a sample of convenience—one Canadian university football team. Head impacts were collected during training camp, practices, eight regular season games, and four playoff games in each season. Saccade measurements were collected at five time points—before and after training camp, at midseason, after regular season, and after playoffs. **Setting:** Two seasons following players from a single USports football team during practices and games. **Participants:** Players who completed a baseline saccade measurement and a minimum of one follow-up measurement were included in the study. A total of 127 players were monitored across two competitive seasons, including 61 players who participated in both seasons. **Independent Variables:** Head impact measurements were collected using helmet-mounted sensors. **Main Outcome Measures:** Saccade latency and number of errors were measured using high-speed video or electro-oculography. **Results:** On average, each head impact increased prosaccade latency by $5.16 \times 10^{-3}$ ms (95% confidence interval [CI], $2.26 \times 10^{-4}$-$1.00 \times 10^{-2}$, $P = 0.03$) and antisaccade latency by $5.74 \times 10^{-3}$ ms (95% CI, $7.18 \times 10^{-4}$-$1.06 \times 10^{-2}$, $P = 0.02$). These latency increases did not decrease between the two seasons; in fact, prosaccade latencies were 23.20 ms longer (95% CI, 19.40-27.14, $P < 0.001$) at the second season's baseline measurement than the first. The number of saccade errors was not affected by cumulative head impacts. **Conclusions:** Repetitive head impacts in Canadian university football result in cumulative declines in brain function as measured by saccade performance. **Clinical Relevance:** Football organizations should consider implementing policies focused on reducing head impacts to improve player safety.

**Key Words:** head impacts, football, prosaccade, antisaccade, latency

(*Clin J Sport Med* 2024;34:280–287)

## INTRODUCTION

Concussion research has gained prominence following the discovery of short-term and long-term consequences of head impacts, including chronic traumatic encephalopathy (CTE).[1–4] Initially, multiple concussions were thought to be the cause of CTE, but recent research has shown that CTE is associated with repetitive head impacts, not concussions.[3,5,6] While a single concussion can cause acute damage to the brain, symptoms usually resolve within 30 days.[7] In contact sports, repetitive head impacts are more frequent than concussions and have been linked with CTE cases in football, hockey, and boxing athletes.[3–5]

Some studies have examined the short-term effects of repetitive head impacts. For example, imaging studies have identified brain structure changes following as little as one season of contact sport.[8–13] On the other hand, studies have reported no differences in neurocognitive tests between preseason and postseason,[9,14–16] but these tests may not be adequately sensitive.[17,18]

Neurocognitive concussion tests, such as the Vestibular Ocular Motor Screening and the Sport Concussion Assessment Tool-5th Edition, commonly assess executive function—the cognitive processes that enable a person to make decisions, adapt to situations, and pay attention to relevant information. Executive function is important for team sport athletes who must adapt to game situations and inhibit responses.[19] For example, college football players possess more proficient executive control over their motor systems than nonathletes.[20] A noninvasive assessment of executive function is the measure of saccadic eye movements. Prosaccades are the reflexive response to look toward a stimulus. Prosaccade latencies are associated with information processing efficiency and attentional control.[21] Antisaccades require the suppression of this reflexive response and generation of a saccade in the stimulus' opposite location.[22] Accordingly, the antisaccade task tests inhibitory control and the ability to generate voluntary actions; both processes are indicators of executive function.[23] Decreases in executive function in concussed individuals compared with healthy controls have been measured using saccadic eye movements.[24–29]

*Submitted for publication April 18, 2023; accepted November 7, 2023.*

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*This study was funded in part by grants from the Natural Sciences and Engineering Research Council and Ontario Centres of Excellence. Instrumentation was provided as an in-kind contribution from GForce Tracker. James P. Dickey acknowledges NSERC (RGPIN-2016-05187) for support.*

*The authors report no conflicts of interest.*

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*http://dx.doi.org/10.1097/JSM.0000000000001202*

However, there is sparse information evaluating the cumulative effects of repetitive head impacts using executive function measures. The purpose of this study was to determine whether cumulative head impacts in Canadian university football players affect executive function, as reflected in saccadic eye movements. We hypothesized that saccade latencies and the number of errors will increase with cumulative head impacts.

## MATERIALS AND METHODS

### Participants

Varsity football team members from the Fall 2017 and 2018 USports (Canadian intercollegiate) football seasons were eligible to participate in this prospective cohort study. Western University's Health Sciences Research Ethics Board approved the protocol (protocol 103727), and all participants provided informed consent. Of the 144 unique players on the football team over these two seasons, 127 players were included in this study. These players completed a baseline and at least one subsequent saccade test (Table 1). A subset of 61 players participated in both seasons.

### Helmet Instrumentation

The GForce Tracker (GFT) was used to collect head impact data (Artaflex Inc., Markham, ON, Canada). The device was adhered to the inside of each participant's helmet, to the right of the crown cushion, using a recloseable fastener (3M Dual Lock Recloseable Fastener SJ3551 400 Black, St. Paul, MN). This location and mounting are similar to previous studies[30,31] and have been validated.[32] Data collection was triggered when a linear acceleration on one axis exceeded the user-defined threshold of 15*g*, which is consistent with best practices.[33] Each impact was time stamped and recorded on the device's onboard storage.

### Impact Data Collection Protocol

Participant attendance was documented for each practice and game. A custom LabVIEW program (version 2011; National Instruments, Austin, TX) recorded which participants were on the field during games and timing of practice periods. These data were later compared with impact time stamps to ensure that only impacts occurring to participants competing on the field were included for analysis, which is an important mechanism for excluding false impacts.[34] The GFT data were transferred to a laptop after each athlete exposure for analysis.

**TABLE 1. Participant Demographics and Data Characteristics**

| Characteristics | Participants (n = 127) |
|---|---|
| Age, mean (SD), yr | 20.8 (2.0) |
| Mass, mean (SD), kg | 98.3 (18.4) |
| Height, mean (SD), cm | 184.6 (5.9) |
| Head impacts, total no. | 77 707 |
| Testing sessions, total no. (%) | 826 (95.7)* |
| Saccade trials, total no. (%) | 32 486 (98.3)† |
| Incorrect saccade trials, total no. (%) | 1167 (3.5) |

** Thirty-seven sessions were missed due to player injury, removal from team, or scheduling conflict.*
*† Trials displaying an anticipatory response or missing data accounted for 1.7% of trials and were excluded from analysis.*

### Saccade Apparatus and Procedure

Saccades were collected in four testing sessions during the season: baseline and preseason tests were performed before and after training camp, respectively, a midseason test occurred after the fourth of eight regular season games, and a final test was completed after playoffs concluded. In the second season, a fifth testing session was included after the regular season ended, before playoffs (Table 2).

Room lights remained on during the testing procedure. Participants sat at a table (height 77.5 cm) with their head placed in a fixed head-chin rest throughout testing. Visual stimuli were presented on a custom-made light board centered on the participant's midline and located at a 55 cm viewing distance. Light emitting diodes (LEDs) were embedded in the light board and covered with black stereo cloth. A LED (48 cd/$m^2$) located at participants' midline at eye level served as the fixation point, and LEDs located 15.5 degrees left and right of the central fixation LED served as target stimuli, consistent with other studies.[35,36] Each trial began with the illumination of the fixation LED, signaling the participant to direct their gaze to the central fixation. The target LED was presented (50 ms), serving as the signal to initiate the prosaccade or antisaccade, following a randomized fore period (1000-2000 ms). The fixation LED was visible throughout the trial (no-gap paradigm).[37] Photodiodes captured light from the LEDs to capture the light timing and enable subsequent characterization of the indicated saccade's direction. Prosaccades and antisaccades were completed in separate blocks. The target location was pseudorandomly ordered (pick without replacement) to ensure that each block contained 10 left and 10 right targets.

Different saccade measurement approaches were used in the two seasons. In the first season, a high-speed digital video camera (Exilim EX-FH20; Casio, Tokyo, Japan), placed directly in front of the participant above the light board, recorded the participant's eye movement at 210 Hz with a 480 by 360-pixel image. Fiber optic cables (Simplex 1.0 mm Industrial Fiberoptics, Tempe, AZ), in series with the light board, were secured in the camera field of view to record the timing of the target light on the video system.[36]

In the second season, electro-oculography (EOG) was used to capture participants' saccade latency and direction.[38–41] Participants were fitted with three disposable surface electrodes (AM-N00S/E, AMBU Blue Sensor Adhesive Snap Electrode; Ambu Inc, Glen Burnie, MD) placed at the outer canthi of each eye and the center of the forehead. The voltages from the electrodes were amplified using an isolated electrophysiological amplifier (Model 2024F, Intronix Technologies Corporation, Bolton, ON, Canada; bandwidth DC to 10 kHz) and sampled at 1000 Hz with a 16-bit analog-to-digital converter (USB 6211; National Instruments) using a custom LabVIEW program. Signals were displayed in real time on a computer to monitor signal quality. Although different saccade measurement approaches were used in the two seasons, they yield comparable saccade measurements.[40,42]

### Video Data Analysis

Video data from the high-speed camera was displayed using QuickTime Player (version 7; Apple Inc., Cupertino, CA). Eye

**TABLE 2. Number of Head Impacts, Test Session Completion, and Saccade Errors**

| | Preseason | Post Training Camp | Midseason | Post Regular Season* | Post Playoffs |
|---|---|---|---|---|---|
| Mean number of head impacts (SD) | 0 | 82.1 (94.2) | 218.0 (204.3) | 264.8 (215.9) | 414.9 (351.2) |
| Saccade sessions completed | 190 | 190 | 182 | 93 | 171 |
| Total number of errors from all saccade trials | 234 | 332 | 240 | 181 | 180 |

*In the second season, a fifth testing session was included after the regular season ended, before playoffs.

motion onset was identified by the initial horizontal displacement in the pupil after the stimulus light. Saccade latency was determined based on the number of frames from the onset of stimulus light to the onset of eye motion. This approach for quantifying saccades using high-speed video yields comparable saccade latencies and errors as those measured by an eye-tracker system.[36]

### *Electro-Oculography Data Analysis*

The raw saccadic EOG voltages were postprocessed using a custom LabVIEW program. The EOG voltages were band-pass filtered from 0.05 to 20 Hz using a second-order Butterworth filter.[43] An onset detection algorithm[44] was used to determine the start of each trial from the photodiode signal, and each saccadic eye movement onset from the EOG signal. The latency was calculated as the difference in timing between the onsets of the target LED and the EOG signal. The EOG signal voltage polarity was used to determine saccade direction. Trials with an anticipatory response (ie, latency $<100$ ms[45]) or missing data (eg, target light not visible in video recording, or the participant blinked) were excluded from subsequent analysis.

### *Statistical Analysis*

All statistical analyses were completed using R.[46] Descriptive statistics for saccade latency and the number of errors are reported as mean and SD. Age, mass, and height of participants were measured before training camp.

Linear mixed-effects models were used to identify predictors of a player's saccade latency throughout a season. Cumulative head impacts were entered as a fixed effect, whereas test session, season, and player were modeled as random effects. Separate prediction models were used for prosaccades and antisaccades.

Linear mixed-effects models were also used to examine differences between baseline saccade latencies in successive seasons as well as differences during the offseason. Test sessions were entered as a fixed effect. Player was modeled as a random effect. Separate prediction models were used for prosaccades and antisaccades.

Finally, linear mixed-effects models were used to examine the effect of cumulative head impacts on saccade errors during the tests. Cumulative head impacts were inputted as fixed effects with random effects of season and player. Separate prediction models were used for prosaccades and antisaccades. If a linear model violated the assumption of linearity, homoskedasticity, or normality of residuals, its raw data were transformed. All linear models were visually examined using residual plots, histograms, and quantile–quantile plots.

All mixed-effects models were created using the lme4[47] and lmerTest[48] packages. The lmerTest[48] package was used to run an analysis of variance on the linear mixed-effects models. Experiment-wise alpha was held to 0.05 for all comparisons. Estimated effect sizes for linear models, analogous to Cohen $d$, were calculated.[49] In terms of effect size interpretation, the variance in latencies relative to effect sizes is considerable. Furthermore, the positive correlation between the mean and SD of latency distributions means that the standard deviations will increase if participants' latencies increase. Accordingly, standardized effect sizes in brain injury saccade latency studies can range from 0.19 to 0.61,[50] so it is not appropriate to use the customary thresholds of $d = 0.8$, 0.5, and 0.2 as big, medium, and small effect sizes.

## RESULTS

Antisaccade latencies were longer than prosaccade latencies; a statistically significant and meaningful difference of 26.86 ms (95% confidence interval [CI], 25.62-28.10, $t_{31270} = -42.43$, $P < 0.001$, $d = 0.60$, Figure 1).

Visual inspection of all latency linear models showed violations of homoskedasticity. Accordingly, the inverse latency, calculated as $invLAT = \frac{-1000}{LAT}$, was used. The numerator was set to $-1000$ to maintain similar order of magnitude as latency values and to ensure that the shortest latency was also the shortest *invLAT*. Inverse latency values were transformed back to latency values when reporting results.

Cumulative head impacts were significantly associated with increased prosaccade latencies ($F(1,3022) = 4.48$, $P = 0.03$, $d < 0.01$). Each head impact was associated with a $5.16 \times 10^{-3}$ ms (95% CI, $2.26 \times 10^{-4}$-$1.00 \times 10^{-2}$) increase in prosaccade latency. Similarly, cumulative head impacts were significantly associated with increased antisaccade latencies ($F(1,3066) = 5.47$, $P = 0.02$, $d < 0.01$). Each head impact was associated with a $5.74 \times 10^{-3}$ ms (95% CI, $7.18 \times 10^{-4}$-$1.06 \times 10^{-2}$) increase in antisaccade latency (Figure 2).

Offseason prosaccade latencies of players who participated in both seasons significantly increased in the second season baseline measurement compared with the end of the first season ($F(1,2295) = 176.94$, $P < 0.001$, $d = 0.55$). Prosaccade latencies were 26.32 ms (95% CI, 23.17-29.37, Figure 3) longer at the beginning of the second season than at the end of the first season. Antisaccade latencies did not significantly increase in the offseason ($F(1,2181) = 0.53$, $P = 0.46$, $d = 0.09$).

Baseline prosaccade latencies of players who participated in both seasons significantly increased in the second season baseline measurement ($F(1,2342) = 170.46$, $P < 0.001$, $d = 0.45$). Prosaccade latencies were 23.20 ms (95% CI, 19.40-27.14, Figure 3) longer in the second season than in the first season. However, this change did not occur with the antisaccade latencies; baseline antisaccade latencies of players who participated in both seasons did not significantly change from the first to the second season ($F(1,2231) = 0.50$, $P = 0.48$, $d = 0.02$).

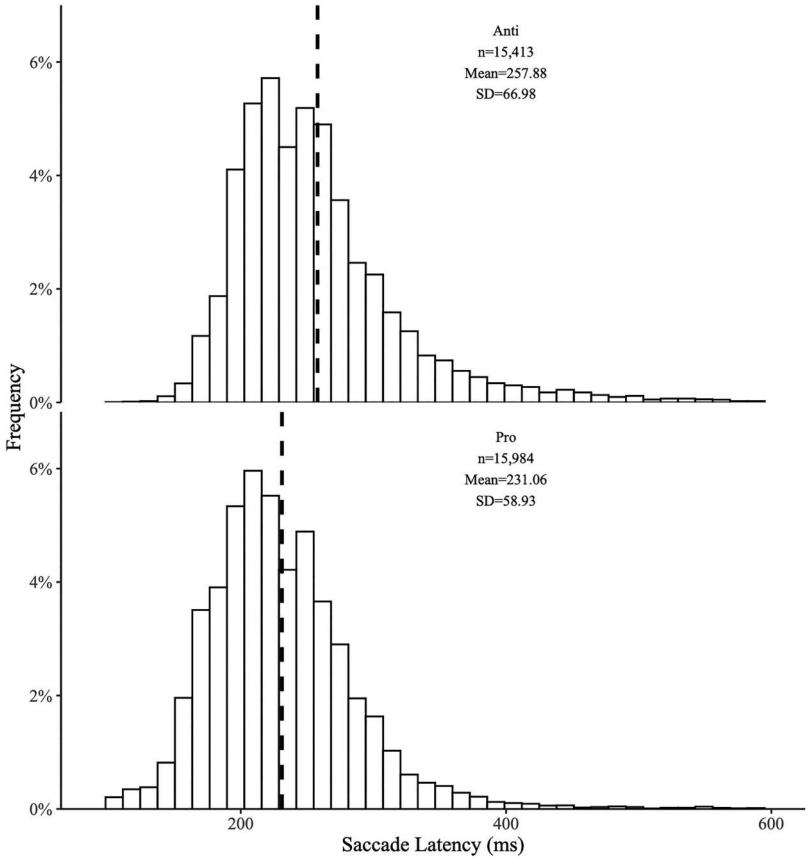


**Figure 1.** Frequency distribution of all correct prosaccade and antisaccade latency trials. Mean latency is indicated by the dotted line. Antisaccade latencies were significantly longer than prosaccade latencies ($P < 0.001$).

Cumulative head impacts were not significantly associated with a change in the total number of errors made during prosaccade trials ($F(1,456) = 2.17$, $P = 0.14$, $d < 0.01$) or antisaccade trials ($F(1,21) = 1.62$, $P = 0.22$, $d < 0.01$).

## DISCUSSION

This study determined that cumulative head impacts experienced by Canadian university football players are associated with a decline in their executive function as measured by saccade latencies. Prosaccade and antisaccade latencies increased for each impact a Canadian university football player received to the head, as hypothesized. In addition, players who participated in both seasons showed a statistically significant and clinically meaningful increase in baseline prosaccade latency from the first season to the second, including prosaccade latency increases in the offseason. Contrary to our hypothesis, cumulative head impacts did not significantly affect the number of errors made during saccade trials.

Players in this study experienced a similar number of head impacts per season as reported in numerous university football head impact exposure studies.[30,31,51–53] A study examining a population of healthy young men[54] measured a mean antisaccade latency (±SD) of 270 ± 39 ms, similar to the 258 ± 67 ms antisaccade latency measured in this study. Another large study[55] reported antisaccade latencies of approximately 300 ms and prosaccade latencies of 236 ms for 20-year-old individuals. The shorter antisaccade latency in our football players may be explained by athletes having shorter latencies than nonathletes.[56] Antisaccade latencies in our study are comparable to athletes in other studies.[26,27,57]

Antisaccade latencies are between 27 to 93 ms greater in concussed individuals compared to healthy participants.[24,26–29,58] Although the participants in this research study were not concussed, they also demonstrated statistically significant increases in saccade latency with head impact exposure. Our determination that each head impact results in an increased saccade latency, regardless of time of season,[59] indicates that there is a cumulative effect of head impacts on brain function. Increased prosaccade latencies are indicative of a player's reduced information processing efficiency and attentional control.[21] Since the antisaccade task involves response inhibition, increased latency demonstrates a select executive-related oculomotor dysfunction. Together, as these latencies increase with accumulated head impacts, football players may have reduced ability to quickly respond to their surrounding environment, which could lead to a greater risk for future head impacts.

The effect size of the difference between prosaccade latencies at baseline from season to season is consistent with prosaccade latency effect sizes measured between mild traumatic brain injury patients and healthy controls.[50] The prosaccade latency difference between baseline measures and during the offseason indicates that oculomotor dysfunction persists from season to season, suggesting a cumulative effect of head impacts from the previous season. Season-to-season

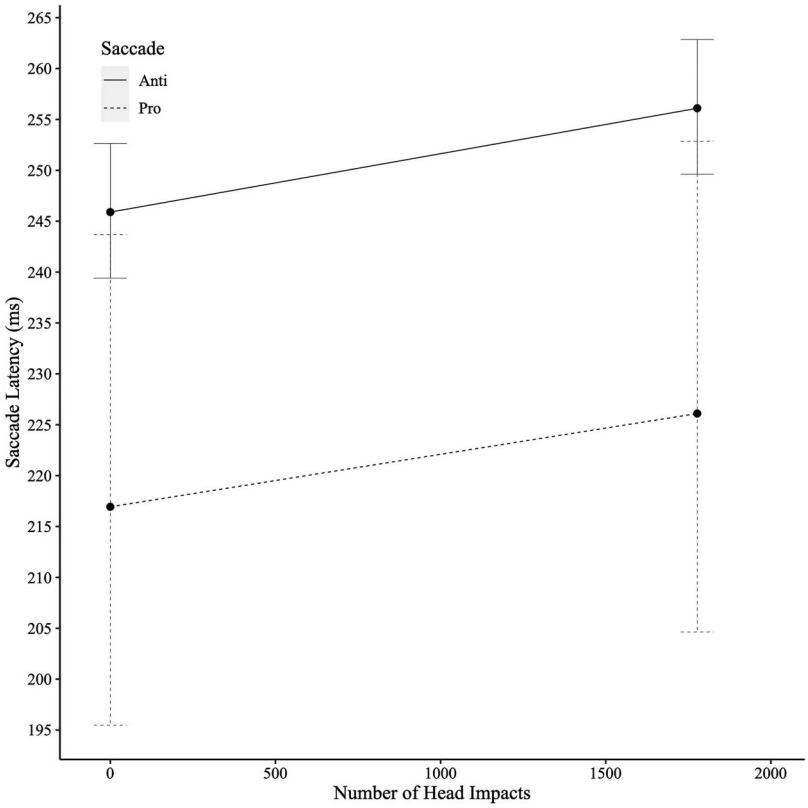


**Figure 2.** Prosaccade and antisaccade latency differences according to the number of head impacts sustained during a season of play. Solid lines indicate antisaccade latencies, and dashed lines indicate prosaccade latencies. Error bars indicate 95% CIs.

brain function changes have been measured in other football,[8,9,60–62] rugby,[11] soccer,[63] and hockey[64] studies. As these deficits accrue over a long period, the effects may not be noticed by the players. However, they represent an alarming change in brain function. For football players—individuals who possess more proficient executive control over their motor systems than nonathletes[20]—a 2.9-ms increase in antisaccade latency is a 12% added "cost" associated with complex decision making. Our model predicts that this 2.9-ms latency increase would occur from experiencing 500 head impacts in a season, of which 30% of this study's participants experienced. This slower executive control could dictate success or failure of a play or lead to an increased injury risk.

Head impact recovery is measured by a return to saccade latency baseline measures before the head impacts were experienced. For example, boxers who receive more head impacts during a match show 20- to 40-ms saccade latency increases immediately after the match but return to baseline after 2 to 3 days of rest.[65] Additional studies have demonstrated immediately increased saccade latencies after concussion, but no significant differences between healthy and concussed individuals at 30 days post injury.[26,58,66] Our study observed persistent changes in prosaccade latencies, with an incremental effect of head impacts on prosaccade and antisaccade latency. This may indicate that players are not receiving enough rest between head impacts to allow the brain to recover. Football players participate in three practices and one game per week and receive an average of 3 to 9 head impacts per practice,[31,51,52] and 12 to 45 impacts per game,[30,31,51,52] depending on their position. This head impact dose appears to have long-term effects as prosaccade latencies did not return to baseline nine months later, at the start of the following season.

The current study has some limitations. Players from a single team were monitored and there was no control group, therefore decreasing external validity. Since different coaching schemes[67] and practice schedules[68,69] influence head impact exposures, this study's results may not represent other youth, university, or professional football teams. In addition, the team underwent coaching staff changes during the study period, which resulted in practice structure changes. This team played in a league that was governed by Canadian football rules. Accordingly, this study's results may not be generalizable to teams that follow American rules. Head impact frequency can vary within positional groups,[70] depending upon seniority and roster depth.[31] This study measured saccade latencies of all roster depths at different positions, including injured players who did not experience any head impacts. Accordingly, position was not differentiated in the analysis. A trigger threshold of 15*g* was used to prevent recording accelerations from normal activities,[71] which is consistent with best practices[33]; however, this study's head impact data are not comparable to studies that used a 10*g* recording threshold.[9,51,68,72] Different instrumentation was used to measure the saccades in each season. The second season's baseline latency EOG measures were longer than the first season's baseline latency measures recorded using high-speed video. Both forms of saccade measurement have been validated against gold standards for eye tracking systems[36,73] and with each

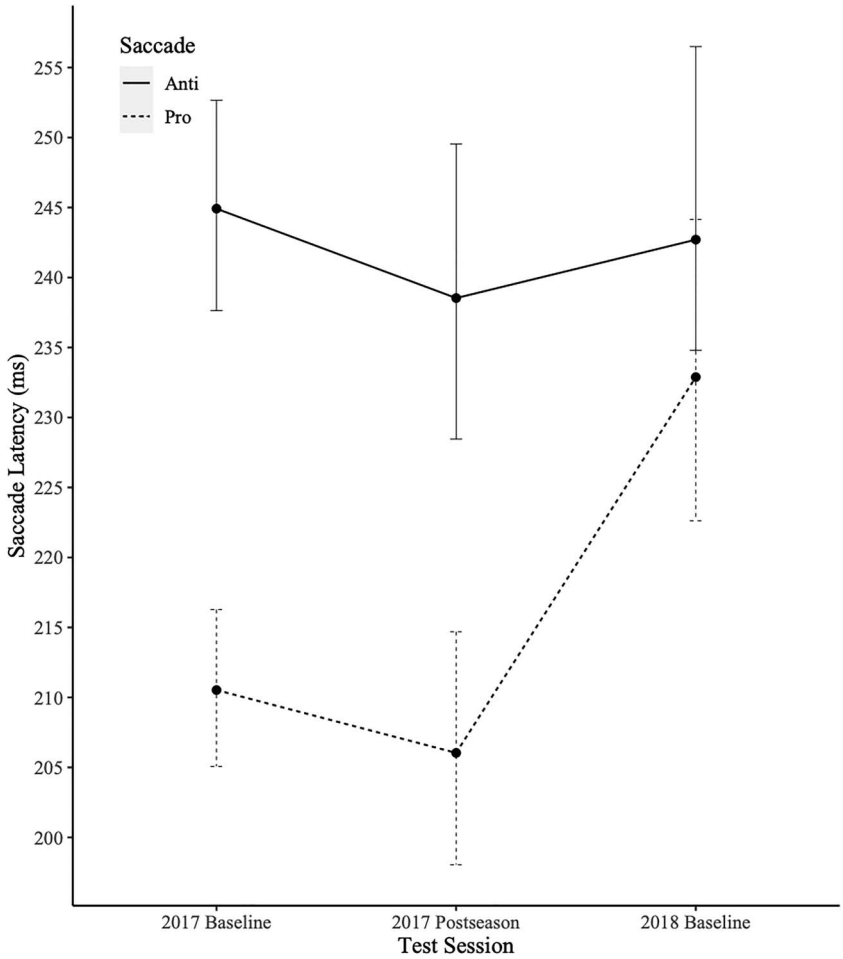


**Figure 3.** Prosaccade and antisaccade latencies between baseline measurements in successive seasons of play. Solid lines indicate antisaccade latencies, and dashed lines indicate prosaccade latencies. Error bars indicate 95% CIs. Prosaccade latencies at 2017 Baseline and 2017 Postseason test sessions were significantly less than those at the 2018 Baseline test session.

other,[40,42] reinforcing that this saccade latency increase represents a measurable difference and not an experimental error. Finally, we acknowledge that specialized equipment, including head impact sensors and a saccade measurement system, are required to perform this study.

## CONCLUSIONS

Football players' prosaccade and antisaccade latencies increase as the number of accumulated head impacts increases. In addition, baseline prosaccade latencies increase over two successive seasons due to cumulative head impacts. These results emphasize the deleterious effects of repetitive head impacts on brain function, which may result in decreased player performance and brain health. Furthermore, the risk of long-term brain sequelae increases steadily every 1000 head impacts a player experiences during their career.[74] In this study, 30% of players accumulated at least 500 head impacts in one season, demonstrating the need to reduce head impacts to safeguard player health. Moreover, we observed that full recovery did not occur within the season or during the offseason. Therefore, head impacts should be reduced through initiatives at both the league[75] and team levels.[76] Future work should evaluate the relationship between career head impact exposure and brain function. Examining whether adequate recovery time between football sessions, such as extending time between games or reducing the number of contact practices, and the amount of exposure, such as the season length or number of games, could enhance players' brain health is also of interest. Future research should evaluate whether the short-term responses observed in this study persist beyond the conclusion of a player's varsity career.

## ACKNOWLEDGMENTS

*The authors acknowledge the help of Wayne Allison, William Smith, Michael Iwamoto, Adam Redgrift, Pawmir Aminullah, and Owen Tirona during the data collection and video analysis of this study. Dr Andrew M. Johnson was consulted for statistical analysis.*

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