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No seasonal changes in cognitive functioning among high school football athletes: Implementation of a novel electrophysiological measure and standard clinical measures

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Abstract

Objective—To evaluate neuroelectric and cognitive function relative to a season of football participation. Cognitive and neuroelectric function declines are hypothesized to be present in football athletes.

Design—Observational

Setting—Athletic fields and research laboratory.

Patients (or Participants)—77 high school athletes (15.9+0.9years, 178.6+7.2cm, 74.4+14.7Kg, and 0.8+0.8 self-reported concussions) participating in football (n=46) and non-contact sports (n=31).

Interventions (or Assessment of Risk Factors)—All athletes completed pre-, mid, and post-season assessments of cognitive and neuroelectric function, self-reported symptoms and quality of life. All athletes participated in their respective sports without intervention, while head impact exposure in football athletes was tracked using the Head Impact Telemetry (HIT) System.

Main Outcome Measurements—Cognitive performance was based on Cogstate Computerized Cognitive Assessment Tool Processing Speed, Attention, Learning, Working

Memory Speed and Working Memory Accuracy scores. ElMindA Brain Network Activation Amplitude, Synchronization, Timing and Connectivity BNA scores demarcated neuroelectric performance. Quality of life was assessed on the Health Behavior Inventory and Satisfaction with Life Scale and symptoms on the SCAT3 inventory.

Results—Football and control sport athletes did not show declines in cognitive or neuroelectric function, quality of life measures, or symptom reports across a season of sport participation.

Conclusions—These findings refute the notion that routine football participation places athletes at risk for acute cognitive declines. The lack of impairment may be associated with no association with head impacts and cognitive function, increased physical activity offsetting any declines, and/or test sensitivity. How these findings are associated with long term cognitive function is unknown.

Keywords

repeated head impacts; concussion; cognitive decline

INTRODUCTION

Declines in cognitive functioning and the onset of Chronic Traumatic Encephalopathy (CTE) are thought to have their beginnings at a young age with head impacts resulting in concussion and/or repeated head impacts (RHI) in absence of concussive signs and symptoms¹⁻⁸. Presently, there is no methodology to diagnose CTE in the living, but graded sets of clinical signs and symptoms have been developed that are thought to be associated with the various stages of disease^{5,9,10}. The clinical presentation has been tied to pathological findings described in post-mortem case series evaluating the cerebral tissue in mostly contact and collision sport athletes ranging from the high school through professional athletes that have been retired for a number of years⁵.

Despite the significant attention that has been placed on these case studies, there is considerable debate on the prevalence of CTE in an athletic population^{11,12}, its potential overlap with other neurodegenerative diseases common to aging both in clinical presentation and pathologically¹³⁻¹⁵, and how many RHIs are required to trigger a clinically meaningful decline in cognitive functioning¹⁶. One hypothesis surrounding the time delay between exposure to RHI and clinically meaningful cognitive declines suggests that each head impact results in cellular level damage that by itself has no clinical meaning. Over time and with ongoing exposure however, the sum exposure to RHIs reduces the finite number of cerebral neurons to a point that will eventually exceed a threshold for clinical impairment¹⁷.

Talavage¹⁸ was the first to support this hypothesis in an investigation of high school level football athletes. In this study, the authors found that those athletes sustaining a higher number of head impacts demonstrated alterations in the dorsolateral prefrontal cortex when evaluated using functional magnetic resonance imaging (fMRI) that corresponded with cognitive changes on ImPACT cognitive test performance. A follow-up investigation by Breedlove¹⁹ using a larger, similarly aged sample reported similar findings, whereby non-concussed football athletes demonstrated changes on fMRI in the upper parietal and

occipital regions that were significantly associated with head impact exposure. Evaluations of collegiate level football athletes have reported similar findings when evaluated prior to and both immediately and at 6-months following the competitive season. In the absence of clinically evident concussions, diffusion tensor imaging analysis has demonstrated changes in fractional anisotropy and mean diffusivity, with the most notable changes occurring at the corpus callosum at both post-season time points^{20,21}. A larger investigation of collegiate football athletes yielded similar results with changes in corpus callosum white matter diffusivity correlating with head impact exposure²². Notably, no consistent pattern of cognitive impairment or other clinical declines (e.g., self-report symptoms) has been observed among these studies.

Given the early evidence linking repeated head impacts with long term neurodegenerative decline, there is a need to apply novel research methodologies to fill the current literature gap between early stage changes in brain function and end stage brain decline. Event related potentials (ERP), a subset of electroencephalography, have previously been used to evaluate the acute²³ and long term effects of concussion (for review see Broglia²⁴). More recently, Brain Network Activation (BNA) scores, with foundations in ERP analysis, have been introduced as a global measure of brain function. Unlike ERPs which evaluate the electrical signal at one electrode at a time following a stimulus, BNA is a measure of interconnectivity of multiple channels, or networking, following a stimulus and has recently been applied in the context of assessing athletes for concussion^{25–28}. For example, BNA scores have been assessed following concussion both in a single subject²⁸ and at the group level²⁶. The ability of BNA to differentiate between those with and without post-traumatic migraine following concussion has also been reported²⁹.

How BNA may change following exposure to RHIs in the absence of concussion has not yet been evaluated, but such an investigation may provide additional insight into the suggested link between head impact exposure and subclinical neurological changes that is proposed to be associated with later life cognitive declines. Therefore, the purpose of this investigation is to evaluate the relationship between a season of high school football exposure and changes in cognitive function with particular emphasis on neuroelectric functioning. We hypothesize there will be an overall decline in cognitive and neuroelectric functioning among football athletes that is proportional to head impact exposure.

METHODS

From 2013 through 2015, athletes (N=77, 15.9±0.9years, 178.6±7.2cm, 74.4±14.7Kg, and 0.8±0.8 self-reported concussions) participating in high school football (n=46) and non-contact sports (n=31) were enrolled and completed this investigation. In instances where athletes participated in more than one year of the investigation, only data from the first year were analyzed. An additional 9 football athletes who were originally enrolled in this study sustained a diagnosed concussion and 9 matched controls from non-contact sports were removed prior to analysis and are reported on separately. Each participant was given a description of the study design and procedures and asked to provide written informed assent and parental consent according to the University Institutional Review Board guidelines.

Upon agreeing to participate, each football athlete's helmet was equipped with a Head Impact Telemetry (HIT) System encoder that recorded all head impacts during football practice and game participation. Athletes were prioritized for fitting with the system starting with first string varsity athletes at the greatest risk for injury³⁰. Football athletes with the HIT System helmet participated in games and practices as they normally would, without intervention by the investigators. Control athletes were recruited from non-contact sports such as: track/cross country (n=8), baseball (n=9), swimming (n=1), basketball (n=8), golf (n=1), and tennis (n=4) and aside from not undergoing head impact monitoring, otherwise completed the same testing timeline and protocol as the football athletes.

Testing Timeline

All enrolled athletes completed a battery of tests at three time points: pre-season, mid-season, and post-season. The testing battery included a demographics questionnaire and other variables of interest (e.g. concussion history), Brain Network Activation (BNA) analysis generated from responses to an auditory oddball task, the Cogstate computerized cognitive assessment tool (CCAT), the SCAT 3 symptom inventory, and two quality of life questionnaires (all described below). Pre-season testing was completed from June to mid-August and ended prior to any pre-season practices. On a weekly basis throughout the 9 week regular football season (September to early-November), athletes were randomly selected (without replacement) to repeat the testing procedure so that each athlete completed one mid-season reassessment. At the conclusion of the competitive season, all athletes completed one additional post-season assessment (November to December). Control athletes were evaluated prior to, during and following their respective season on a timeline matched to their football counterpart.

Equipment

HIT System—The HIT System (Simbex, Lebanon, NH) is the most widely used device capable of recording head impact location and magnitude during sporting events³⁰. The device consists of two units: the encoder located within the helmet and a sideline computer. The encoder contains 6 thimble sized single-axis accelerometers recording at 1000Hz, a telemetry unit, a data storage device, and a battery pack. The memory device can record and store 100 impacts when out of range of the sideline computer. The components are sealed in waterproof plastic and retrofitted between the pads of any standard Riddell football helmet. At the time of impact, data are transmitted to a sideline computer and stored for later analysis. The sideline computer can receive impact data from up to 100 players up to 150 yards away. Importantly, athletes like linemen who experience head impacts during the majority of plays, but whose impacts may be obscured from video capture, can be monitored. The data available from the software contains all pertinent impact data including: peak linear acceleration, peak rotational acceleration, and time stamp. HIT System equipped helmets look and function identically to other helmets and meet National Operating Committee on Standards for Athletic Equipment (NOCSAE) safety standards.

Auditory Oddball Task—A 3-stimulus auditory oddball task was selected as the cognitive task during the BNA assessment to elicit all of the necessary ERP components needed to calculate the BNA scores. Participants were required to respond as quickly and as

accurately as possible with a right hand button press to a randomly occurring, infrequent target stimulus tone while ignoring all other auditory stimuli. Target stimuli were 1000 Hz tones that occurred with a probability of 10%, and non-target stimuli were 2000 Hz tones that occurred with a probability of 80%. The remaining 10% of all tones consisted of novel stimuli (e.g., white noise, phone ring, knock on door, etc.). Stimuli were presented binaurally using a headset over a total duration of 16 minutes.

EIMindA Brain Network Activation (BNA)—The BNA algorithm is a novel analysis applied to standard EEG technology as a measure of brain connectivity. As a sub-component of EEG, Event Related Potentials, represent time-locked patterns of neuroelectric activation that occur in preparation for, or in response to, an auditory event (i.e., the Oddball Task). Neuroelectric data were gathered using a standard 256 wet lead EEG cap using previously described standard procedures³¹. The BNA algorithm is described in detail elsewhere^{32–35}. Briefly, the BNA analysis is completed in two separate stages (see Figure 1). The group pattern analysis stage (blue arrows) was completed independently by EIMindA during product development prior to the initiation of this study, and the individual subject evaluation stage (red arrows) was then applied to each individual testing session during this study. In both stages, raw EEG data undergo data preprocessing (A), band-passing (B), discretization (C), and normalization (D). During the group pattern analysis stage, a set of ERP patterns found to best characterize the reference group during Network Analysis (E) was generated to define the reference BNA networks. In the individual subject evaluation stage, Network Analysis (E) was performed on the ERP data from each individual testing session to compare each individual's ERP patterns to those of the reference group as previously derived during the group pattern analysis stage.

BNA Scores are based on how well the individual's ERP patterns (frequency band, scalp location, amplitude, and timing) match the reference BNA network patterns derived during the group analysis stage. An index score, ranging between 0 and 100, is calculated to establish each BNA score for an individual subject and can be interpreted as the percent similarity between the individual's ERP pattern and the reference BNA network pattern. A total of twelve BNA scores are generated for each subject and include an Amplitude, Synchronization, Timing and Connectivity score for each of the three tones associated with the Oddball Task. (i.e., Frequent, Target, and Novel Stimuli tones).

Cogstate—The Cogstate Computerized Cognitive Assessment Tool (CCAT) is a commonly used clinical concussion assessment tool that measures speed and accuracy during four simple cognitive tasks to generate Processing Speed, Attention, Learning, Working Memory Speed and Working Memory Accuracy scores. Each of the four tasks is based on a response to playing cards that are presented on the computer screen and the entire test takes <15min to complete. The Cogstate CCAT has demonstrated high sensitivity (71%) to cognitive declines associated with concussion³⁷.

Health Related Quality of Life—The Satisfaction with Life Scale (SWLS) and the Health Behavior Inventory (HBI) were used to evaluate participants' quality of life. The SWLS is a 5-item survey assessing the participant's overall current satisfaction with life. Each item is responded to using a 7 point Likert scale, with higher scores representing better

Health Related Quality of Life. The SWLS has consistently been found to have a high internal reliability³⁶ and has been deemed valid^{36,37} and highly correlated to health and self-esteem³⁸. The Health Behavioral Inventory (HBI) is a 20-item survey assessing personal health beliefs. Summative cognitive and somatic scores are generated from the 20 responses, with higher score representing lower quality of life^{39,40}.

Symptom Inventory—The SCAT3 symptom inventory⁴¹ assesses the presence and severity of 22 different symptoms that are commonly associated with concussion (e.g. nausea, dizziness, headache, etc.). Each symptom is graded on a scale of 0–6 (none to severe) and the values are summed to generate a total symptom severity score.

Statistical Analyses

Following completion of the investigation, measures of central tendency were calculated for each outcome measure by group and time point. A two-way (group $\times$ time) repeated measure Analysis of Variance (ANOVA) was used to compare pre-season to mid-season to post-season changes in each outcome measure between the football and non-contact control athletes. Significant main effects were further evaluated using paired contrasts against the pre-season measure. A Greenhouse-Geisser correction was applied when sphericity was violated and Bonferroni corrections were applied when multiple tests were completed.

In addition, to evaluate for individual changes in cognitive functioning and head impact exposure, stepwise linear regression models were built to evaluate the relationship between cumulative head impact exposure as measured by the HIT System (i.e., total head impacts, cumulative linear acceleration, cumulative rotational acceleration, cumulative HITsp) and pre-season to post-season BNA score changes. Variables significantly improving the regression model ($p < 0.1$) were retained and variables with high collinearity ($r > 0.7$) were excluded. All analyses were completed using SPSS (version 22) and α was set *a priori* at 0.05.

RESULTS

Demographics for the 46 football athletes (15.9 ± 0.8 years, 179.5 ± 6.7 cm, 79.8 ± 14.1 kg, 1.0 ± 0.8 previous concussions) and 31 non-contact control athletes (15.9 ± 1.0 years, 177.4 ± 7.8 cm, 66.5 ± 11.9 kg, 0.4 ± 0.6 previous concussions) were captured the time of enrollment. The football athletes weighed significantly more ($p < 0.001$) and had a greater number of previous concussions ($p = 0.002$) than the control athletes.

Over the course of the investigation, football athletes participated in an average of 59.7 sessions per season, including 49.6 ± 8.2 practices and 10.1 ± 1.1 games. These sessions resulted in 513.9 ± 395.8 head impacts with 309.3 ± 248.4 occurring during practices and 204.6 ± 168.6 during games. Impact magnitudes averaged 26.3 ± 2.8 g (median 25.8 g), 1186.0 ± 124.4 rad/s/s (median 1197.3 rad/s/s), and 16.0 ± 1.2 for HITsp (median 15.9). The mean cumulative impact magnitude exposure per athlete in a single season was $14,007.1 \pm 11,668.3$ g, $620,358.7 \pm 481,702.1$ rad/s/s, and 8420.0 ± 6625.7 for HITsp. Sport participation data were not collected on control athletes.

BNA Findings

Frequent Stimuli—BNA scores for the football and control athlete groups on the Oddball Frequent stimuli are presented in Table 1. ANOVA analysis indicated a significant time main effect for Amplitude ($p=0.040$), but no group main effect ($p=0.433$) or group by time interaction ($p=0.30$). Post-hoc analyses showed a combined group decrease from pre-season to mid-season ($p=0.019$), but no difference between pre-season to post-season ($p=0.716$). Synchronization analysis indicated a significant time main effect ($p<0.001$), but no group main effect ($p=0.89$) or group by time interaction ($p=0.388$). Post-hoc analyses showed all athletes improved from pre-season to post-season ($p<0.001$), but no difference between pre-season to mid-season ($p=0.254$). Timing analysis indicated a significant time main effect ($p=0.017$), but no group main effect ($p=0.080$) or group by time interaction ($p=0.706$). Post-hoc analyses showed a combined group improvement from pre-season to post-season ($p=0.009$), but no difference between pre-season to mid-season ($p=0.135$). Connectivity analysis indicated a significant time main effect ($p=0.010$), but no group main effect ($p=0.163$) or group by time interaction ($p=0.186$). Post-hoc analyses showed a combined group improvement from pre-season to post-season ($p=0.016$), but no difference between pre-season to mid-season ($p=0.791$).

Target Stimuli—BNA scores for the football and control athlete groups on the Oddball Target stimuli are presented in Table 1. ANOVA analysis indicated a no significant time main effect for Amplitude (Greenhouse-Geisser adjusted $p=0.120$), group main effect ($p=0.093$), or group by time interaction ($p=0.941$). Synchronization analysis indicated a no significant time main effect ($p=0.337$), group main effect ($p=0.103$), or group by time interaction ($p=0.822$). Timing analysis indicated a no significant time main effect ($p=0.881$), group main effect ($p=0.225$), or group by time interaction ($p=0.580$). Connectivity analysis indicated a no significant time main effect (Greenhouse-Geisser adjusted $p=0.679$), group main effect ($p=0.075$), or group by time interaction ($p=0.963$).

Novel Stimuli—BNA scores for the football and control athlete groups on the Oddball Novel stimuli are presented in Table 1. ANOVA analysis indicated a significant time main effect for Amplitude ($p=0.018$), but no group main effect ($p=0.891$), or group by time interaction ($p=0.687$). Post-hoc analyses showed a combined group increase from pre-season to post-season ($p=0.007$), but no difference between pre-season and mid-season ($p=0.344$). Synchronization analysis indicated a significant time main effect ($p=0.008$), but no group main effect ($p=0.247$), or group by time interaction ($p=0.657$). Post-hoc analyses showed a combined group increase from pre-season to post-season ($p=0.003$), but no difference between pre-season and mid-season ($p=0.471$). The Timing analysis indicated a significant time main effect (Greenhouse-Geisser adjusted $p=0.040$), but no group main effect ($p=0.693$), or group by time interaction ($p=0.575$). Post-hoc analyses showed a combined group increase from pre-season to post-season ($p=0.017$), but no difference between pre-season and mid-season ($p=0.241$). Lastly, Connectivity analysis indicated a significant time main effect ($p=0.003$), but no group main effect ($p=0.525$), or group by time interaction ($p=0.512$). Post-hoc analyses showed a combined group increase from pre-season to post-season ($p=0.002$), but no difference between pre-season and mid-season ($p=0.252$).

Cogstate Findings

Cogstate scores for the football and control athlete groups are presented in Table 2. ANOVA analysis of Processing Speed indicated a significant group main effect ($p=0.001$), but no significant time effect ($p=0.582$), or group by time interaction ($p=0.157$). Post-hoc analyses indicated higher performance by the control athletes at all time points. ANOVA analysis of Attention indicated no significant time (Greenhouse-Geisser adjusted $p=0.217$), group ($p=0.165$), or group by time interaction ($p=0.314$). Analysis of Learning indicated a significant time main effect ($p<0.001$), but no significant group effect ($p=0.096$), or group by time interaction ($p=0.664$). Post-hoc analyses indicated significantly higher combined group scores at mid-season ($p<0.001$) and post-season ($P<0.001$) relative to pre-season. Analysis of Working Memory Speed indicated a significant time main effect ($p<0.001$) and significant group effect ($p=0.038$), but no group by time interaction ($p=0.112$). Follow-up analyses indicated a combined group improvement at the mid-season ($p=0.001$) and post-season ($p<0.001$) time points, relative to the pre-season. Additional analyses indicated significantly higher scores among the football athletes at mid-season ($p=0.021$) and post-season ($p<0.001$) relative to pre-season; and mid-season to pre-season ($p=0.026$) for the control athletes. In addition, significantly higher scores were noted among the control athletes at the pre-season ($p=0.043$) and mid-season ($p=0.008$) compared to the football athletes. ANOVA analysis of Working Memory Accuracy indicated no significant time ($p=0.129$), group ($p=0.911$), or group by time interaction ($p=0.274$).

Self-Reported Symptom and Quality of Life Findings

Symptom and Quality of Life scores for the football and control athlete groups are presented in Table 3. ANOVA analysis the Health Behavior Inventory – Cognitive Score indicated no significant time main effect ($p=0.113$), group main effect ($p=0.804$), or group by time interaction ($p=0.574$). ANOVA analysis the Health Behavior Inventory – Somatic Score indicated no significant time main effect ($p=0.098$), group main effect ($p=0.727$), or group by time interaction ($p=0.126$). ANOVA analysis the Satisfaction with Life – Total Score indicated no significant time main effect ($p=0.188$), group main effect ($p=0.461$), or group by time interaction ($p=0.523$). ANOVA analysis of the SCAT3 Symptom Severity indicated no significant time main effect ($p=0.458$), group main effect ($p=0.081$), or group by time interaction ($p=0.758$).

Regression Findings

Stepwise regression analyses revealed no significant relationships between the pre- to post-season changes in BNA scores and any cumulative head impact exposure variable (p 's >0.05 for total head impacts, cumulative linear acceleration, cumulative rotational acceleration, cumulative HITsp).

DISCUSSION

This investigation sought to examine the relationship between the repeated head impact exposure associated with a season of interscholastic high school football participation and performance on a novel ERP-based measure of brain connectivity and more traditional clinical measures. No declines were identified in any of the outcome measures at a group

level, nor was a relationship between head impact exposure and changes in brain connectivity identified. In fact, some of the Cogstate measures demonstrated improved performance (i.e., Learning and Working Memory Speed) and some of the BNA measures demonstrated higher scores (i.e., Frequent Stimuli – Synchronization; Frequent Stimuli – Timing; Novel Stimuli – Connectivity) among the football athletes as well as the control athletes (i.e., BNA Frequent Stimuli – Synchronization; Novel Stimuli – Connectivity; Cogstate Learning; Cogstate Working Memory Speed) across a season of sport participation. While the biological underpinnings of these findings cannot be determined by this study, we propose three potential explanations: 1) exposure to head impacts may not be associated with alterations in brain network activation, cognitive function, symptoms, or health-related quality of life, 2) the beneficial effects of physical activity may offset any negative effects of repeated head impacts on the study outcomes, and 3) the outcome measures used in this study may be insensitive to the magnitude of changes that are present.

The relationship between repeated head impacts and declines in brain function has not been fully elucidated, particularly among high school athletes. Indeed, the majority of cases in which long term changes in brain functioning in contact and collision sport athletes have been attributed to the adverse effects of head impact exposure have been described in highly selective samples^{1–3,5–8}. These cases have yet to draw a direct link between repeated head impacts, tau deposition and/or progressive clinical decline, leading some to suggest alternative pathologies for these late life outcomes^{13–15,42–44}. For example, some have speculated that alternative neurological disorders may actually be responsible for the clinical effects of neurodegenerative disease that have been attributed to repeated head impacts¹³. Others have demonstrated tau deposition in individuals without a history of repeated head impact exposure⁴⁵, including associations with both opioid⁴⁶ and NSAID use⁴⁷ which are commonly used among contact sport athletes. Lastly, Montenigro¹⁶ recently suggested a dose response effect between repeated head impact exposure and clinically meaningful changes in mood, behavior and cognition. The Montenigro model suggests that a minimum of 2,723 head impacts are necessary to increase the risk of cognitive decline, which is well above the mean number of 514 impacts sustained by our cohort in a season. Indeed, more robust estimates of head impact exposure suggest the average high school football athlete sustains 652 impacts per season⁴⁸, which is still below Montenigro's minimum threshold for meaningful change following a three or four year high school football career.

Converse to the implication that repeated head impacts do not result in long term declines in cognitive functioning, the findings from this investigation could be interpreted to suggest that any declines in cognitive functioning resulting from repeated head impacts are offset by gains derived from physical activity participation. For example, a Cochrane review of randomized controlled trials indicated moderate to large standardized mean difference effects of aerobic exercise on motor function (1.17), auditory attention (0.52) and delayed memory function (0.50) in an older adult population (ages 55+)⁴⁹. Another review noted similar findings in an adult population (ages 18–50), with improvements reported in executive function, memory, and processing speed⁵⁰. Perhaps most germane to this study are investigations among adolescents showing that improved aerobic fitness is associated with improved cognitive control,⁵¹ as well as improved inhibition, cognitive flexibility, attentional resource allocation, and cognitive flexibility⁵² in those adolescents with long term exposure

to exercise. Additionally, acute exercise has been demonstrated to improve response inhibition and response accuracy⁵³. The mechanism for these changes may involve increased basal ganglia⁵⁴ and hippocampal⁵⁵ volume among higher fit children, as well as improved white matter tract integrity^{56,57}.

Third, the BNA technology has thus far only been implemented in a limited number of studies to explore post-concussion changes in brain networking. One investigation differentiated between those with and without post-traumatic migraine using BNA scores²⁹, but another failed to demonstrate any difference in BNA scores between concussed athletes and healthy controls³¹. Furthermore, the stability of several BNA scores has been demonstrated to be lower than is necessary for clinical utility, with reliable change score intervals exceeding 50% or more of the 100 point BNA range⁵⁸. These findings would suggest that BNA analysis may struggle to detect the degree of electrophysiological change associated with concussion, and may thusly be unable to identify more subtle changes that may be associated with repeated head impacts in absence of concussion. Future works should also consider utilizing a visual oddball task as the visual mechanism may be more adversely affected to head impacts^{59,60}. Other outcome measures used in this investigation (i.e., Cogstate) likely face similar sensitivity concerns. Indeed, the Cogstate test is a gross measure of cognitive functioning that was designed to be implemented in the days following acute concussion. Like all tests for cognitive functioning, the largest effect sizes are present immediately following concussion, with diminishing signal over the subsequent days and weeks^{61,62}. Furthermore, a recent review suggests that computerized tests as a whole do not hold the requisite sensitivity to detect subtle changes to neurocognitive functioning⁶³ despite being commonly implemented to do so.

Notwithstanding, the findings from this investigation diverge slightly from other works that have also evaluated the relationship between football participation in the absence of concussion and various neurological outcomes. For example, work among high school athletes has identified alterations in the dorsolateral prefrontal cortex that were associated with an average of 727 head impacts over the course of a season. However, this same group did not show deficits on clinical measures of cognitive functioning¹⁸. Another investigation of Division I football and ice hockey athletes exposed to an average of 503 head impacts per season demonstrated changes in white matter diffusion relative to control athletes. More specifically, the post-season analysis identified changes in mean diffusivity within the corpus callosum and fractional anisotropy changes in the amygdala. The changes in the corpus callosum were associated with worse performance on the California Verbal Learning Test-II²². A similar evaluation of high school football athletes also demonstrated abnormalities in white matter using diffusion tensor imaging, which were linked to declines on the ImPACT Verbal Memory score⁶⁴. Likewise, Bazarian et al.²⁰ demonstrated changes in mean diffusivity and fractional anisotropy in the corpus callosum, both immediately and at six months following the conclusion of the football season. In that investigation, the imaging changes did not consistently relate to performance on a standard cognitive assessment. As such, while this series of investigations demonstrate some form of neurophysiological change following a season of contact/collision sport participation, the nature and clinical relevance of the changes identified are inconsistent across studies. The lack of consistent clinical outcomes is not clear, but test sensitivity and test stability should be considered⁶⁵ as

should the possibility that while the structural measures may sensitive to sub-clinical changes, the clinical implication of those findings may only manifest under certain circumstances (e.g. taxed cognitive reserve or further structural degeneration).

Limitations

Like all investigations, the findings presented here come with limitations. First, the head impact metrics reported here may not accurately reflect true exposure rates and magnitudes. For example some research suggests the HIT System has an absolute error rate of 15% in magnitude⁶⁶ and will miss 5% of impacts⁶⁷. This limitation however, does not offset the lack of change, or even improvements observed on some of the cognitive evaluations. Indeed, if our cohort did sustain an additional 26 impacts (5% of 514 head impacts), they would still fall below the suggested clinically meaningful threshold value. In addition, as noted above, the BNA and other measures may lack the requisite sensitivity to detect subtle changes in cognitive functioning over the course of a season, should they exist. Future works should consider implementing more challenging clinical measures of cognitive functioning and more refined advanced neuroimaging techniques (e.g., event related potentials and/or electroencephalogram).

CONCLUSION

Regardless of the oft publicized findings that concussions and/or repeated head impacts lead to long term neurological decline, the science in this area is still emerging. The results of this investigation suggest that a season of high school football participation resulting in mean 513 head impacts did not have a negative effect on brain networking, cognitive performance, symptom or quality of life reports. Indeed, these same measures showed improvement among both football and non-contact sport athletes, supporting a multitude of research indicating physical activity has both physiological and cognitive benefits. These findings do not outweigh other works showing long term changes, rather are a reason to pause and fully examine the issue at hand, as there are significant questions to be answered before a direct link between head impact exposure and neurological decline can be made.

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Figure 1.
The process by which standard EEG signals are processed to determine brain networks associated with a given task.

BNA scores by group for each output score based on Auditory Oddball tone. An overall increase in scores were noted for the Frequent Synchronization, Timing, and Connectivity scores, along with the Novel Amplitude, Synchronization and Connectivity scores.

Table 1

	Frequent Stimuli - Amplitude			Frequent Stimuli - Synchronization			Frequent Stimuli - Timing			Frequent Stimuli - Connectivity		
	Pre-Season	Mid-Season**	Post-Season	Pre-Season	Mid-Season	Post-Season*	Pre-Season	Mid-Season	Post-Season*	Pre-Season	Mid-Season	Post-Season*
Football Athletes	72.0 (22.7)	67.1 (25.8)	68.2 (27.5)	72.7 (18.6)	76.5 (17.0)	79.1 (15.0)	70.0 (28.1)	74.7 (22.8)	76.3 (16.5)	71.6 (17.2)	72.8 (14.9)	74.5 (12.5)
Control Athletes	67.9 (17.4)	59.1 (28.5)	68.5 (21.1)	74.7 (20.2)	73.8 (19.6)	81.0 (13.3)	62.3 (24.9)	63.9 (26.7)	67.1 (23.6)	68.3 (15.4)	65.6 (16.2)	72.2 (9.9)
	Target Stimuli - Amplitude			Target Stimuli - Synchronization			Target Stimuli - Timing			Target Stimuli - Connectivity		
	Pre-Season	Mid-Season	Post-Season	Pre-Season	Mid-Season	Post-Season	Pre-Season	Mid-Season	Post-Season	Pre-Season	Mid-Season	Post-Season
Football Athletes	63.7 (27.0)	56.9 (30.1)	57.7 (32.1)	57.7 (29.6)	64.4 (31.8)	59.3 (28.9)	63.8 (31.7)	66.9 (34.4)	65.7 (33.4)	61.7 (25.1)	62.7 (27.3)	60.9 (25.1)
Control Athletes	55.5 (26.1)	50.1 (32.9)	47.6 (31.8)	48.8 (30.6)	57.7 (33.6)	53.3 (35.2)	61.2 (30.3)	58.5 (31.9)	57.5 (39.3)	55.2 (26.1)	55.5 (26.3)	52.8 (30.5)
	Novel Stimuli - Amplitude			Novel Stimuli - Synchronization			Novel Stimuli - Timing			Novel Stimuli - Connectivity		
	Pre-Season	Mid-Season	Post-Season*	Pre-Season	Mid-Season	Post-Season*	Pre-Season	Mid-Season	Post-Season*	Pre-Season	Mid-Season	Post-Season*
Football Athletes	63.0 (29.9)	63.5 (27.1)	71.3 (23.0)	61.2 (28.4)	60.4 (25.0)	69.3 (23.4)	62.1 (33.3)	63.6 (31.1)	68.7 (29.8)	62.1 (26.5)	62.5 (23.1)	69.8 (19.3)
Control Athletes	58.1 (31.9)	66.2 (31.6)	70.6 (28.7)	50.7 (30.7)	58.4 (28.1)	64.7 (26.5)	54.3 (30.6)	64.2 (30.3)	67.1 (23.8)	54.4 (26.8)	62.9 (27.5)	67.5 (22.1)

* Indicates significant group improvement from Pre-Season

** Indicates significant combined group decline from Pre-season

Table 2

Cogstate scores by group for each performance metric. An overall performance increase was noted for the Learning and Working Memory Speed variables. Group differences were noted for Processing Speed and Working Memory Speed.

	Processing Speed		
	Pre-Season	Mid-Season	Post-Season
Football Athletes	103.1 (6.0)	103.5 (5.4)	102.5 (6.2)
Control Athletes	107.6 (5.9) [†]	105.9 (5.9) [†]	106.6 (4.2) [†]
	Attention		
	Pre-Season	Mid-Season	Post-Season
Football Athletes	105.4 (5.0)	105.2 (4.6)	105.5 (5.2)
Control Athletes	108.4 (4.5)	104.8 (17.7)	108 (3.5)
	Learning		
	Pre-Season	Mid-Season [*]	Post-Season [*]
Football Athletes	101.7 (6.5)	105.5 (8.1)	107.2 (9.6)
Control Athletes	103.7 (8.8)	108.1 (9.6)	111.2 (11.2)
	Working Memory Speed		
	Pre-Season	Mid-Season [*]	Post-Season [*]
Football Athletes	101.8 (6.2)	103.5 (6.2) [*]	105.0 (6.2) [*]
Control Athletes	104.9 (6.6) [†]	107.3 (5.6) ^{*†}	106.4 (6.7)
	Working Memory Accuracy		
	Pre-Season	Mid-Season	Post-Season
Football Athletes	100.9 (7.4)	103.9 (8.5)	103.7 (8.5)
Control Athletes	102.6 (7.0)	101.7 (7.1)	103.7 (8.6)

[†] Significant between group differences for that time point

^{*} Indicates significant combined group improvement from Pre-Season

Table 3

Quality of Life and Symptom Severity scores by group. No changes were seen between groups or over time.

Health Behavior Inventory - Cognitive			
	Pre-Season	Mid-Season	Post-Season
Football Athletes	10.3 (4.6)	9.3 (5.2)	8.8 (5.4)
Control Athletes	10.7 (5.1)	9.7 (5.2)	9.7 (5.5)
Health Behavior Inventory - Somatic			
	Pre-Season	Mid-Season	Post-Season
Football Athletes	3.1 (2.7)	3.3 (3.2)	3.0 (3.5)
Control Athletes	4.3 (3.3)	2.9 (2.5)	3.0 (3.1)
Satisfaction with Life - Total Score			
	Pre-Season	Mid-Season	Post-Season
Football Athletes	30.48 (3.2)	30.2 (3.4)	30.4 (3.1)
Control Athletes	29.9 (2.4)	29.2 (3.3)	30.3 (2.7)
SCAT3 - Symptom Severity			
	Pre-Season	Mid-Season	Post-Season
Football Athletes	1.3 (2.1)	1.3 (2.9)	1.8 (3.9)
Control Athletes	0.6 (1.2)	0.6 (1.4)	0.7 (1.6)