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Research Article

Retired Professional Contact Sport Athletes are more sedentary and consume fewer brain healthy nutrients than Non-contact Sport Controls

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Abstract

Early onset neurodegeneration and cognitive impairment is suspected to be prevalent in retired contact sport athletes and the most common etiology is suspected to be repetitive head injuries during sport. Other causes of early neuro degeneration, like nutrition and lifestyle, have been studied to a lesser extent. We compared the nutritional and physical activity habits of retired contact sport athletes (CS) and age-matched, retired non-contact sport athlete controls (NS). Standardized questionnaire was used to obtain demographics, vascular risk factors, and sport history. Nutritional intake was calculated using Food Frequency Questionnaire (FFQ) and physical activity using Yale Physical Activity Questionnaire (YPAS). Mild Cognitive Impairment (MCI) was diagnosed using Comprehensive Criteria. Estimated IQ was measured using Wide Range Achievement Test-4. CS (n = 21, 56.3 ± 10.9 years, 29.7 ± 3.6 kg/m2) were significantly more overweight than NS (n = 21, 56.7 ± 9.5 years, 24.5 ± 2.6 kg/m^2, $p < 0.001$). Estimated Energy Expenditure (EEE) was not significantly different, however, total time spent doing physical activities was significantly lower in CS than NS (51.1 ± 15.0 hours/week vs. 22.5 ± 18.7 hours/week, $p < 0.001$). Macronutrient intake was not significantly different. Many brain healthy micronutrients including copper, selenium, folate, manganese, and riboflavin were lower in CS than NS but did not reach statistical significant after correcting for multiple pair wise comparisons. Our study suggests that retired contact sport athletes are more overweight and more sedentary than age-matched, retired non-contact sport athletes making them at risk for early cognitive decline.

Keywords: Aging; Athlete; Energy expenditure; Nutrition

Abbreviations

YPAS: Yale Physical Activity Survey; FFQ: Food Frequency Questionnaire; CTE; Chronic Traumatic Encephalopathy; NFL: National Football League; NHL: National Hockey League; DHA:Docosahexaenoic Acid; EPA: Eicosapentaenoic Acid.

Introduction

The prevalence of cognitive impairment is suspected to be high in the retired contact sport athlete population [1]. Mild traumatic brain injury (mTBI), commonly referred to as concussion in the sports literature, is a common injury that National Football League (NFL) and National Hockey League (NHL) athletes suffer from repeatedly throughout their professional careers. It is thought that repeated mTBI throughout life may lead to the development of Chronic Traumatic Encephalopathy (CTE), which is a progressive neurodegenerative disorder [2]. There has been much research done on CTE and post-mortem analysis shows that 80% to 99% of contact sport athletes, mainly former NFL and NHL players, had observable tau deposition which was characteristic of CTE [3,4]. However, this tau deposition has not been linked to the suspected clinical manifestations of CTE and there is much controversy regarding the subject [5,6].

There has been some research on other possible causes of neurodegeneration, including cardiovascular risk and cognitive reserve, [7-9] in the retired contact sport population, but little attention has been given to their nutritional and general lifestyle habits. Lack of micro- and macro-nutrients in the diet and the decline of regular physical activity has been linked to neuro-degradation and early aging of the brain [10,11]. Nutrition is a fundamental component of an athlete's training regimen; however, news articles suggest many current professional football players are not aware of their specific dietary recommendations. Athletes just starting out in their professional careers lack proper dietary and life style education for post-professional sport life and may develop improper habits during retirement. The purpose of this study is to determine nutritional and lifestyle habits of retired professional contact sport athletes and compare with age-matched, retired non-contact sport athletic controls. This study furthermore aims to provide a descriptive report of any differences in nutritional intake between contact and non-contact sport athletes after retirement and suggest a diet rich

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in micro-nutrients which may have a role in brain health after mTBI. We hypothesize that retired contact-sports athletes would have lower energy expenditure, higher calorie intake, and lower micronutrients than retired non-contact sport athletes.

Methods

This IRB-approved case control study was completed as a part of the Healthy Aging Mind Project [12] at the University at Buffalo, SUNY.

Participants

Participants were invited to complete a series of neurocognitive tests, a blood draw and questionnaires about their lifestyle. Retired professional contact sport athletes (CS) were contacted and recruited through NFL and NHL alumni associations. Criteria for inclusion in this group included (1) played a professional contact sport for two or more seasons; (2) currently retired from competitive sports; and (3) between the ages of 36-72 years. Criteria for exclusion included (1) unwilling or medically unable to receive an MRI, (2) sustained a concussion within the past two years, (3) history of moderate to severe brain injury (4) history of cerebrovascular event that could lead to hypoxia, and (5) history of a learning disability. Non-contact sport athletic controls (NS) were recruited by contacting athletic clubs for runners, swimmers or tri-athletes with rosters that included older athletes. Criteria necessary for inclusion were (1) athletes who participated in local or national competitive individual non-contact sport such as running, cycling, or swimming when they were younger; (2) currently retired from competitive sports; (3) between the ages of 36-72 years; and (4) no history of self-reported or documented concussions. Exclusion criteria were the same as the retired professional contact sport athlete group.

Outcome measures

Demographics: A standard questionnaire was used to obtain demographics and medical, vascular risks factors, and sport history. History of alcohol abuse, drug abuse, hypertension, diabetes and hypocholesteremia were self-reported. Venous blood sample was collected by phlebotomist at first visit, and sent to in-site laboratory for analysis. High density lipoproteins (HDL) and low density lipoproteins (LDL) were measured using standard laboratory protocols and cut-off values were 40-60 mg/dL and 0-100 mg/dL respectively [13]. Resting blood pressure after 2-minute sitting (using Welch Allyn Connex ProBP 3400 Digital Blood Pressure Device) was measured to calculate Atherosclerotic Cardiovascular Disease (ASCVD) 10-year risk using the Framingham Heart Study equation. The ASCVD risk calculator uses a high blood pressure cut-off value of ≥ 140/90 mmHg and cardiovascular event risk of 7.5% or higher is considered as elevated risk [14].

Mild cognitive impairment (MCI): MCI was diagnosed by a neuropsychologist using the Comprehensive Criteria, which is that at least two performances within a cognitive domain fall below the established cutoff in order for that domain to contribute to the MCI classification [6,15].

IQ estimation: IQ was estimated using Wide Range Achievement Test-4 (WRAT-4) which was performed by a neuropsychologist.

Estimated energy expenditure (EEE): Yale Physical Activity Survey (YPAS), [16] a self-reported questionnaire, was used to assess the subjects' EEE. It includes seasonal modifications that take in to account the time of year that the activity is being completed and has documented repeatability and validity [17]. The YPAS asks subjects to recall various activities that they regularly perform and creates a score based on the intensity, season, and duration of the activity, and is measured by estimated calories burned while doing various tasks. The calories of each physical activity are added to the Basal Metabolic Rate (BMR, calculated by Harris-Benedict equation) to obtain the EEE. This questionnaire was completed at home with instructions.

Food frequency questionnaire (FFQ): FFQ was used to estimate nutritional intake [18]. The FFQ comprises a detailed self-report daily, weekly, and monthly nutrition intake which is used to obtain a year-long recall of diets and has been validated for reproducibility in a variety of populations [19,20]. Nutritionist Pro software (Nutritionist Pro™, Axxya Systems, USA) is then used to calculate the macro- and micro-nutrient intake. Micronutrients are divided by the daily recommended intake values to calculate Percent Daily Recommended Intake (%DRI). This questionnaire was completed at home with instructions.

Statistical analysis

Based on computed power analysis, a total of 20 participants in each group was required in order to achieve a power of 0.80 with a one sided test at level 0.05. A series of t-tests (equal varices) and Chi-squared tests were used to assess group-wise differences in demographics, estimated IQ, vascular risk factors, and sport history. P-values less than 0.05 were considered significant. Non-parametric t-test was used to examine group wise differences in each EEE, macronutrient (grams) and micronutrient (%DRI). Bonferroni correction was used to account for multiple pair wise comparisons in micronutrients and a p-value of 0.001 was considered significant (0.05/27). Logistic regression was performed to see if there was any association between diagnosis of MCI (binary response) and age, BMI, calorie intake, EEE and hours spent performing physical activity. Statistical analysis was performed using SPSS Version 24 (Armonk, NY: IBM Corp).

Results

A total of 27 retired professional football and hockey players took part in the study. Five participants withdrew from the study before completing all the procedures and one was excluded due to a serious brain injury from a motor vehicle accident which was only revealed during imaging. Hence, 21 retired NFL and NHL athletes made up the CS group. All participants correctly filled out the YPAS, but only 12 out of 21 participants correctly filled out the FFQ so demographics and EEE are calculated from 21 participants and nutritional intake is calculated on 12 participants. Completion rates of the FFQ were significantly different, but there were no statistically significant differences in demographics, vascular risk factors, or physical activity between those participants who completed the FFQ correctly and those

who did not. A total of 24 non-contact sport retired athletes signed up to participate but three withdrew from the study before completing all the procedures. All participants correctly filled out the YPAS and FFQ, hence 21 participants made up the NS group. CS were significantly more overweight than NS (30.1 ± 3.5 vs. 24.5 ± 2.5 kg/m^2, p < 0.001), had lesser education (p = 0.024), lesser estimated IQ (49.29 ± 6.76 vs. 57.57 ± 8.82, p = 0.01), and had lower HDL cholesterol (42.6 ± 8 vs. 49.8 ± 11, p = 0.017). More CS met the criteria for MCI than NS (8 vs 3, p = 0.083), but this did not reach significance. None of the other variables were significantly different. Demographics, estimated IQ, incidence of MCI, and vascular risk factors are presented in Table 1.

CS had a significantly higher BMR (1960.8 ± 217 vs. 1628.2 ± 177 kCal/day, p < 0.001) than NS. There was no significant difference in EEE between CS and NS (2229.7 ± 371 vs. 2123.7 ± 272 kCal/day, p = 0.297), however, CS spent significantly less time performing physical activities than NS (22.5 ± 18.7 vs. 51.1 ± 15.0 hours/week, p < 0.001). Individual domain scores from the YPAS are presented in Figure 1. Vigor (p < 0.001), walking (p = 0.009), and sitting (p = 0.021) scores were significantly different between groups.

There were no significant differences between the CS and NS's estimated macronutrient intake. Additionally, there were no statistically significant differences in the different fats, i.e. oleic, linoleic, linolinic, eicosapentaenoic (EPA) and docosahexaenoic (DHA) acid. Riboflavin (p = 0.047), biotin (p = 0.012), folate (p = 0.02), vitamin D (p = 0.035), copper (p = 0.019), selenium (p = 0.037), potassium (p = 0.02), phosphorous (p = 0.043), manganese (p = 0.002), and fiber (p = 0.02) were lower in CS than NS, but were not significant after Bonferroni correction. Figure 2 shows the macronutrient intake and Figure 3 shows the micronutrient %DRI of the 12 CS and 21 NS participants who correctly completed the FFQ.

On regression analysis, diagnosis of MCI was not significantly associated with age (p = 0.199), calorie intake (p = 0.604), BMI (p = 0.067), EEE (p = 0.669), or hours spent doing physical activity (p = 0.069).

Discussion

The purpose of the Healthy Aging Mind study was to extensively evaluate the cognitive, behavioral, and lifestyle characteristics of athletes who had professional careers playing

Table 1: Demographics, estimated IQ and Vascular Risk Factors of Study Population.

	Contact Sport (n=21)	Non-Contact Sport (n=21)	p-value
Age in years, mean	56.7 ± 9.5	55.4 ± 9.3	0.65
BMI in kg/m^2, mean	30.1 ± 3.5	24.5 ± 2.5	< 0.001
Height in meters, mean	1.78 ± 0.06	1.83 ± 0.05	0.001
Weight in kg, mean	77.1 ± 11.0	99.2 ± 13.6	< 0.001
Ethnicity, n (%)			
Caucasians	18 (85.7)	21 (100)	0.23
African-American	3 (14.2)	-	
Education, college degree, n (%)	10 (47.6)	19 (90.5)	0.024
Mild Cognitive Impairment, n (%)	8 (38%)	3 (14%)	0.083
Estimated IQ	49.29 ± 6.76	57.57 ± 8.82	0.01
Sport, n (%)			
Football	8 (36.4)	-	
Hockey	14 (63.6)	-	
Running	-	12 (57.1)	
Cycling	-	6 (28.6)	
Triathlete	-	2 (9.5)	
Track & field	-	1 (4.8)	
History of smoking, n (%)	7 (33.3)	5 (23.8)	0.51
History of alcohol abuse, n (%)	6 (28.6)	1 (4.8)	0.09
History of drug abuse, n (%)	4 (19.0)	1 (4.8)	0.34
History of High Blood Pressure, n (%)	8 (38.0)	4 (19.0)	0.18
History of Diabetes, n (%)	-	-	-
History of High Cholesterol, n (%)	2 (9.5)	4 (19.0)	0.39
One or More Reported VRF[1], n (%)	15 (71.4)	9 (42.9)	0.005
ASCVD mean score	14.11 ± 10.60	10.36 ± 8.44	0.22
ASCVD elevated risk[2], n (%)	14/20[3] (70.0)	13 (61.9)	0.75
Total Cholesterol in mg/dL	189.4 ± 30	182.8 ± 25	0.44
LDL[4] Cholesterol in mg/dL	106.9 ± 39	103.3 ± 33	0.75
HDL[5] Cholesterol in mg/dL	42.6 ± 8	49.8 ± 11	0.017

1: Vascular Risk Factor; 2: American Heart Association ASCVD Risk ≥ 7.5%; 3: One participant < 40 years of age; 4: Low Density Lipoprotein; 5: High Density Lipoprotein

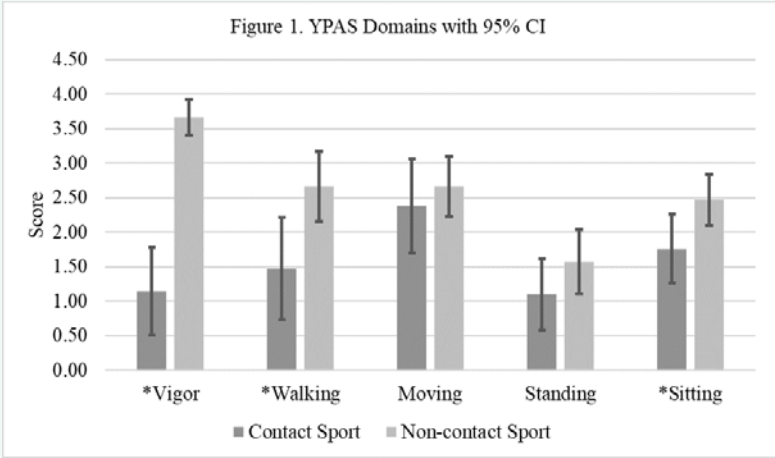

Figure 1 YPAS Domains with 95% CI.
* : significant difference, $p < 0.05$

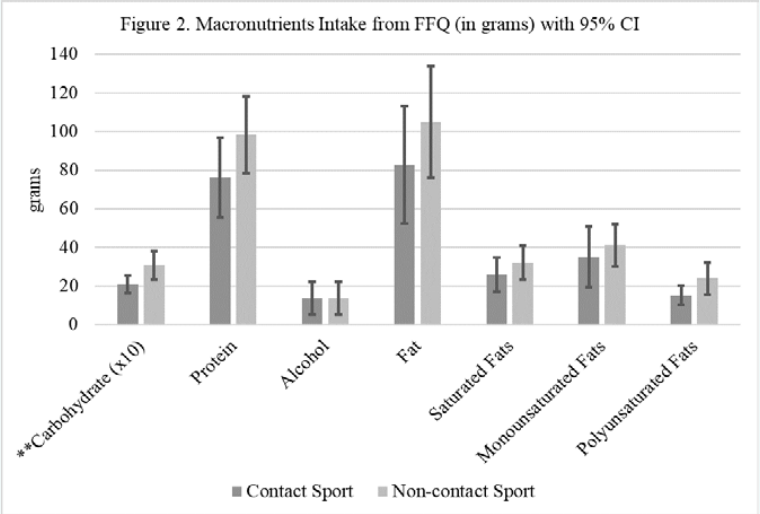

Figure 2 Macronutrients Intake from FFQ (in grams) with 95% CI.
**: Carbohydrates are scaled down to the tenth

contact sports that may have left them vulnerable to CTE and compare them to athletes who competed in individual, non-contact sports. The research and media attention on CTE have given the impression that former contact-sport athletes will experience early onset dementia marked by cognitive impairment from receiving repetitive concussive or sub-concussive blows to the head [21]. Although the CS group scored significantly lower on estimated IQ, this can be explained by the difference in education which has been shown to have a directly proportional relationship with IQ [22]. Based on demographics, the control NS group was considered healthier due to lower BMI and higher HDL, which are known risk factors for early cognitive decline. An investigation in 2012 [9] studied 38 over-weight and 38 healthy-weight retired NFL players (mean age = 57 years, range 25 - 82) and showed that players with higher BMI had significantly more cognitive decline which suggests it is an independent risk factor in retired contact sport athletes. Increased HDL has also been shown to be correlated with increased cognitive function in healthy and pathological conditions, [23] as well as greater grey matter volume [24]. The CS group had a higher incidence of MCI (38% vs 14%) that did not reach significance. This could be due to our small sample size because our power calculation did not include detection of rare events; however, the control group was much superior in physical and cognitive health so we expected this to reach significance. Regression analysis between MCI and main outcome variables (EEE, BMI, calorie intake and hours spent doing physical activity) had interesting results. Although the results were not statistically significant, both BMI and hours spent performing physical activity, were approaching significance ($p = 0.067$ and 0.069 respectively) indicating there may be some correlation. Further research needs to be performed with larger sample sizes. Detailed neurocognitive testing was performed

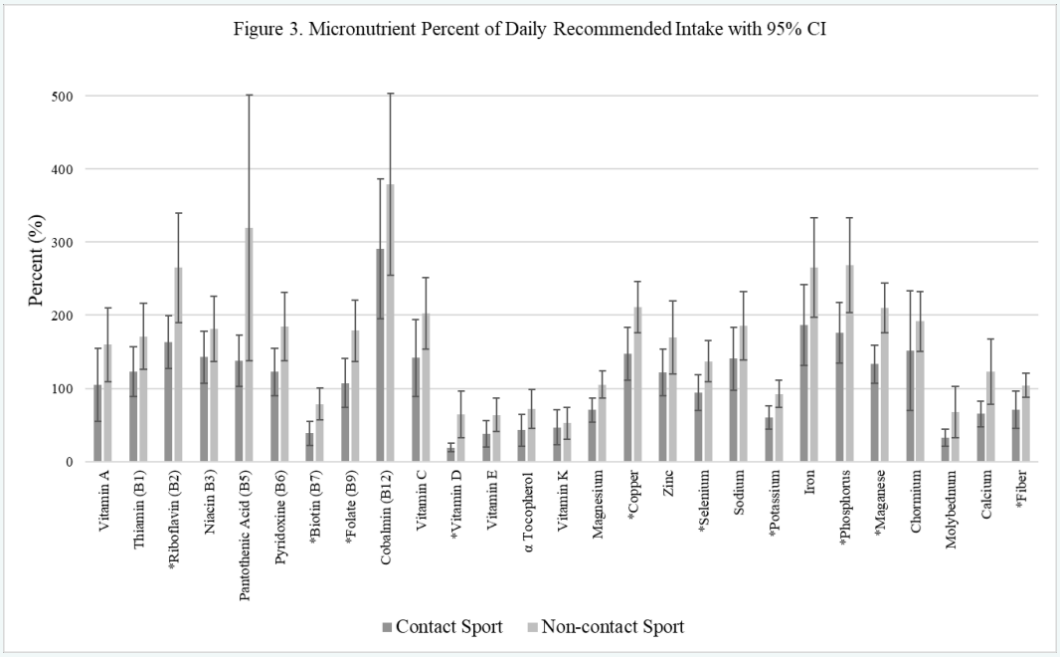

Figure 3 Micronutrient Intake Percent of Daily Recommended Intake with 95% CI.
*: significant difference, $p < 0.05$; 100% represents daily recommended intake

on all participants as part of the extended study protocol; these results are presented in a separate paper [6].

The YPAS provided mixed results for physical activity when we compared the two groups. BMR is the amount of calories the body burns at rest and EEE is the amount of calories it burns when exercise is taken into account. CS spent significantly less time performing physical activities, i.e. were more sedentary, and had significantly less vigor and walking index scores, but had a much higher BMR due to their high BMIs. This led to non-significant differences in EEE between the two groups. Estimated average calorie intake per day can also be obtained from the FFQ and although the results from the two questionnaires were comparable, we presented the values from the YPAS instead of the FFQ because a large proportion of the CS did not correctly complete the FFQ. Still, sedentary life style irrespective of BMR has been associated with a variety of neurocognitive diseases. They have been linked to brain atrophy, [25] neuroinflammation, [26] and vulnerability to trauma and disease, [27] whereas regular exercise has been linked to several benefits [28]. The mechanism of action is suspected to be due to induction of factors that promote neural growth and repair. Brain derived neurotropic factor (BDNF) has been associated with these effects and has been shown to increase hippocampal volume and improve spatial memory [29]. Animal and human studies confirm that exercise increases BDNF levels and brain function as early as 5-6 weeks after initiation of aerobic training [30,31]. The rapid benefit effect of exercise on neuroplasticity suggests that improved neuronal function rather than reduced cerebrovascular disease risk is the cause for functional improvement.

While analyzing the FFQ, it was evident that several members of the CS group did not fill out the FFQ correctly, leaving only 12 sets of FFQ's to analyze against all 21 of the NS group. The main reason for the forms being incorrect was that the CS group checked the different food items instead of writing the amount they consumed. These forms were completed at home and returned to the research center when they returned. Follow-up was attempted to obtain completed FFQ but majority did not want to return. The reason for this drastic difference is uncertain, both groups received the same instructions and there were no significant differences in demographics, except education, between those who completed them correctly and those who did not. There were no significant differences in macro- or micro-nutrient intake, but several brain healthy nutrients (copper, selenium, folate, manganese, vitamin D) were lower in CS than NS. In past studies, low plasma copper levels have been linked to cognitive decline [32]. This indicates a potential correlation between copper intake and cognitive decline, as well as a need for further research on the topic. Recently, researchers have theorized that selenium may play a critical role in preserving the cognition of individuals consuming a high fish diet [33]. Although there is little data on selenium's effect on brain health, if administered intravenously it may help improve neurologic function after suffering a TBI [34]. Foods high in folic acid, which gets converted to folate in the body, are essential components of a healthy diet, and may have an effect on brain health. Numerous studies have shown that lower rates of folate intake correlate to faster cognitive decline [35]. Manganese is transported to the brain through the blood-brain barrier, and when levels are within normative values, may influence synaptic neurotransmission, making it a potential brain-healthy nutrient [36]. In low levels, manganese has proven to lead to neuro-deficiencies, while at high

levels it may act as a neurotoxin, indicating that close maintenance of manganese is important in preventing its negative effects more than improving cognition [37]. There is evidence that low levels of vitamin D is related to lower levels of cognition, yet there is little to no evidence demonstrating that supplementation of vitamin D will improve cognition [38]. In the past, studies have noted the correlation between decreased cognition and less sun abundant months (a major source of vitamin D) [39]. However, determining the effects of dietary vitamin D intake is made difficult due to varying outdoor or sedentary lifestyles in participants.

There are several vitamins and minerals found in the diet that have been coined as "brain healthy" foods, and regular consumption of foods containing these nutrients has been linked to improved cognition and overall brain health. Some of these brain healthy nutrients which were not different between the two groups are vitamin C and E, omega-3 fatty acids, zinc, niacin, and pyridoxine. Vitamin C is essential for brain function due to its ability to form ascorbic acid, an antioxidant, and prevent cognitive decline. A review from 2014 found a direct effect of vitamin C deficiency on brain development following traumatic injury [40]. Vitamin E has been linked to protecting against oxidative stress in the brain. While past study results vary in their legitimization of vitamin E as a tool in fighting cognitive decline, its anti-oxidative effects seem promising [41]. Little is known about the role of DHA and EPA on the brain in response to mild head trauma, but there has been substantial evidence demonstrating the positive effects of these nutrients in stroke patients, demonstrating that it may have beneficial effects on brain health [42,43]. They have also been shown to prevent the loss of gray matter volume in patients suffering from schizophrenia [44]. Zinc is furthermore useful in the brain because it is needed to metabolize EPA and DHA. A 2005 study found that Zinc supplementation may benefit adolescent cognition; however no such studies have been performed in reference to traumatic brain injury [45]. Niacin therapy has also been proven beneficial; it has improved brain plasticity in post-stroke mice, suggesting that it may be a worthy tool in treating traumatic brain injuries [46]. Pyridoxine is also suspected to be a brain healthy nutrient. A study in 2014 found an inverse correlation between complete vitamin B intake and several cognitive conditions, such as mild cognitive impairment, suggesting a need for further investigation into individual subsets of vitamin B [47]. A suggested brain healthy diet to consume after brain injury, including common sources, is presented in Supplementary File 1.

This study has a number of limitations. The greatest limitation is the unequal sample size for the nutrient intake comparisons. The overall sample size is also small and there is no obvious means for determining the representativeness of the sample to the population of retired professional athletes who played contact sports. We recognize that studies that report no significant differences between groups are often underpowered to make such claims, but our control population was much more superior (retired non-contact master athletes who continue to remain active to some extent, lower BMI, more educated and higher estimated IQ). We hypothesized that these former athletes would be significantly different in lifestyle choices when compared with a superior control group who not only played in their youth, but also continued to stay active since they were members of cycling and running clubs. We were surprised to find so few major differences in food intake or energy expenditure. Still, our sample size is small and the study results need to be replicated with longitudinal assessment. We did not collect information about cause of retirement or if the athletes continued to physically train after retirement, also, the average age of retirement is different for different types of sports, which can confound our results. The body composition between contact sport athletes (e.g. football players) is different from non-contact sports (e.g. runners) which may have contributed to our difference in BMI, however, we found significant differences in hours spent performing physical activities (i.e. chores, work, recreational activities) which should not be affected by body composition. Additionally, all of the forms used were self-reported, therefore subject to possible recall bias.

Conclusion

Retired contact sport athletes were significantly more overweight than age-matched, retired non-contact sport athlete controls and spent significantly less time performing physical activities, which makes them at risk for early neurocognitive decline. However, there was no significant difference between their daily estimated energy expenditure, which can be attributed to their higher BMI. Retired contact sport athletes consumed lesser brain healthy nutrients than healthy controls, but there was no significant evidence that they were lacking in any major macro- or micro-nutrients in their diets. Longitudinal research with larger sample sizes are required to further assess the risk of nutrition and lifestyle choices on early neurocognitive decline in retired contact sport athletes.

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Authors' role

MH, KD, JL, PH and BW designed research. MH, IB, JL and BW collected data. KD, MH and BW analyzed the data. MH, KD IB and JL interpreted the data and prepared the manuscript, all authors approved the final version of the paper.

Conflict of Interest (COI) Statement

The authors report no conflict of interest.

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