

A New Collegiate Model: Intra-Collegiate Athletics at BYU Idaho

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Aaron Kelly, a highly respected college sport consultant, is charged with the task of presenting a new model of intercollegiate athletic administration to a panel of leaders in the field. Coincidence and research led him to a successful National Junior College Athletic Association athletic program that was discontinued in pursuit of a new model of competitive intra-collegiate athletics when the institution transitioned to a four-year university. Given the purpose of athletics within the academe to facilitate an educational experience difficult to replicate through any other opportunity, (Brand, 2006; NCAA 2010; Rader, 1999) this program sheds light on a new way to view this tradition we have come to know as college sport. The purpose of this case is to highlight the tremendous potential for innovation that exists within the intercollegiate athletic model. While financial challenges make it difficult for many institutions to sponsor broad-based intercollegiate athletics programs, this model presents a design that can reduce expenditures and provide additional participation opportunities for education through athletics. As Kelly prepares for his presentation, he questions whether this model is ideal and how the landscape of intercollegiate athletics might be affected if implemented on a national scale.

In the wake of rampant deficit spending, disgraceful scandals, and vocal calls for reform, the building inertia for a college sport renaissance seemed palpable. It was this sense of urgency that spawned the Summit on Intercollegiate Athletic Reform. Several weeks ago, Aaron Kelly, a highly respected sport administration consultant and former Division I East Coast Conference commissioner, received a phone call from the president of the Summit with an invitation to present a proposed solution to some of the financial issues that university athletics programs were facing. The Summit organizer emphasized it was time for a new model of intercollegiate athletics to be presented and charged Kelly with the task of developing a model that could decrease the financial burden of struggling universities without decreasing the number of participation opportunities. Kelly was honored and hesitantly accepted the task not knowing whether this was a feat that could be accomplished. After grappling with the charge for several months and finding little direction through a variety of research methods, a conversation he overheard at his favorite local pizza joint led him to his first breakthrough – and possibly a direction for his presentation.

While anxiously waiting for his fully loaded stuffed-crust pizza, he overheard a conversation at an adjacent table between two missionaries and a young couple. The couple asked the young men what they planned to do when they finished their service as missionaries. Both missionaries emphatically stated they hoped to go to BYU Idaho in order to be involved in competitive sports. One mentioned he hoped to coach basketball at the university because his playing days were over due to a high school injury, and the other said he wanted to play competitive football and baseball. The couple asked some follow-up questions about how they intended to do this, and the missionaries explained that several years ago church leaders had directed the successful National Junior College Athletic Association athletic

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program at Ricks College to be discontinued in pursuit of a new model of competitive intra-collegiate athletics when the institution transitioned to a four-year university. The missionaries went on to recount experiences of leadership and athletic skill development that they had heard about from friends who were alums of the university. These athletic opportunities, they mentioned, were the driving force behind their hopes to attend the university. As Kelly finished his third slice, he thought about the enthusiasm he had sensed from the missionaries and made the decision to box up the remaining slices of his pizza so he could get to a computer to learn more about this intriguing university and its athletic program.

Several weeks later, after researching and visiting the campus of BYU Idaho, Kelly believed the philosophy and organization of this athletics program met or exceeded all of the specifications he was hoping to fulfill in the proposed new vision the Summit leaders desired. Kelly hoped the BYU Idaho competitive athletics model would inspire Summit participants to recognize the tremendous potential there is for innovation within college sport, and that this recognition would spur a rich discussion leading to additional organizational models. As enthusiastic as he was, he also held a fair amount of worry about how the model would be received by those in attendance (including a selection of athletics directors in addition to the presidents of the National Collegiate Athletic Association (NCAA), National Junior College Athletic Association (NJCAA), National Association of Intercollegiate Athletics (NAIA), and numerous conference commissioners and university presidents). As the presentation drew near, Kelly debated whether this model was truly the best answer to the issues currently faced in the industry of intercollegiate athletics.

BYU Idaho

For most of its history BYU Idaho was known as Ricks College. Initially established in 1888 as the Bannock Stake Academy by members of The Church of Jesus Christ of Latter-day Saints, the school was renamed in honor of founder Thomas E. Ricks in 1923 (BYU Idaho, 2006; Crowder, 1997). Except for a brief period between 1948-1956 when it operated as a four-year institution, Ricks College primarily functioned as a two-year junior college. By 2000, Ricks College had over 7,500 students, making it the largest private junior college in the country (Encyclopedia of Mormonism, 1992).

In June of 2000, Gordon B. Hinckley, President of The Church of Jesus Christ of Latter-day Saints, announced that Ricks College would become a four-year institution. Among the changes mentioned was the discontinuation of the varsity intercollegiate athletics program and institution of a competitive intra-collegiate athletics program. The athletics announcement took the campus by surprise given the historical success of the athletic program (BYU Idaho 2011b). As a member of the Scenic West Athletic Conference in the National Junior College Athletic Association, Ricks boasted one of the top five junior-college athletic programs in the country. The football program was especially strong, winning several Western States Football League championships and routinely sending players to Division I powerhouses. Between 1980 and 2000, nearly 25 Ricks College alumni had played in the National Football League or the Canadian Football League (Jacobson, 2005).

Located in Rexburg, Idaho, Ricks College officially became known as Brigham Young University Idaho on August 10th, 2001 (BYU Idaho, 2006) and continues to offer several Associate degree programs in addition to Bachelor degree programs. Since the transition from Ricks College to BYU Idaho, the school has experienced tremendous growth doubling enrollment from the Ricks College era. During the fall semester of 2011 there were 13,368 full-time students enrolled at the institution (BYU Idaho Newsroom, 2011). Despite this growth, the campus is considered geographically, ethnically, and religiously homogeneous, with 90% of the students being Caucasian and 99% members of The Church of Latter Day Saints (BYU Idaho Institutional Research, 2011).

Although numerous changes have taken place subsequent to the transition from Ricks College to Brigham Young University Idaho, university and church leaders have made an effort to continue the “Spirit of Ricks” - a campus emphasis of service, hard work, friendliness, and compassion. In this spirit, much of BYU Idaho student life revolves around organized student activities which consist of dances, outdoor excursions, concerts, fitness programs and service projects (BYU Idaho, 2010). These university initiatives along with the sports events, recreational sports and competitive sports tracts of the sport division are housed within the Student Activities Program.

The Student Activities Program

Introduced in the transition from two-year Ricks College to four-year BYU Idaho, the student activities program provides engagement opportunities for everyone on campus regardless of their skill level, or degree of commitment. A variety of activities are offered throughout the year in an effort to provide students with opportunities to be involved, gain leadership skills, and serve others while enriching the university experience for themselves and those around them (Student Activities Program, 2011). The activities program is guided by five foundational principles that contribute to a complete education enabling students to learn and grow through ‘taking action’ in the pursuit of developing “the best, most interesting and capable versions of themselves” (Activities Guiding Principles, 2011):

Principle #1: A wide range of activities will meet the diverse interests and abilities of students.

Principle #2: Students are the participants rather than the spectators.

Principle #3: Participants have the opportunity to act rather than be acted upon.

Principle #4: Students choose their level of participation.

Principle #5: Participants will develop personal and spiritual qualities that prepare them for life.

Elder Henry B. Eyring, the Church’s commissioner of education, said the activities program is intended to build leaders and is “leadership training of the broadest and most exciting kind” (qtd. in Gardner, 2001). The goal is for participants in the program to be “leaders who know how to teach and how to learn... [who] will become legendary for their capacity to build the people around them and add value wherever they serve” (Eyring, 2001). Students can choose to participate or become involved in leading the program through being a coach, manager, coordinator, director, or area director. Area directors and directors are scholarship positions, with coordinators, managers, and coaches as volunteer opportunities. The Student Area directors oversee the seven program divisions including outdoor, service, talent, wellness, social, life skills and sports.

The sports division uses a three track system to provide options for students to lead and participate in sports events, recreational sports, and/or competitive sports. Annual sports events include golf scrambles, 3-on-3 basketball tournaments, and themed races. Recreational sports, similar to most university’s intramural programs, include a variety of options with the average participation requirements of one game per week and a tournament if eligible. The competitive sports track emulates collegiate varsity sport participation with the inclusion of try-outs, regular practices, trained coaches, and a full season and post-season of competition all of which takes place on campus between other university competitive sport teams.

The Competitive Sports Program

The Competitive Sports Program “provides opportunities for individuals to develop personal honor in a disciplined team environment” (BYU Idaho Competitive Sports Program, 2011, ¶ 6). When a student chooses to participate in the BYU Idaho Competitive Sport Program, he or she is held to a high standard of commitment that includes many of the features of traditional intercollegiate varsity athletic programs including regular team practices led by trained coaches, team uniforms, and post-season play.

Participants in the competitive program must go through a try-out process. In order to play competitive football, for instance, students must attend a registration meeting and three days of tryout sessions. Following this, a public draft is held that eligible student-athletes must attend in business attire. If drafted onto one of the teams, athletes then attend a team combine to begin the season. Once committed to a competitive team, students are required to attend all practices and competitive events. The competitive baseball league, for instance, states “if you miss practice unexcused you will have to sit out for the games that weekend. If you quit playing during the semester you will be ineligible to play competitive sports the following semester” (“League Description”, 2011, ¶ 1). The tryouts and drafts held each semester as opposed to traditional intercollegiate athletics team recruiting are implemented in order to maintain a level

of competitive parity within the leagues, and this parity has been instrumental in providing an optimal experience for spectators and participants as there are many close finishes and exciting playoffs (J. Garner, personal communication, November 4, 2011).

Along with competing in the campus-wide leagues, students also maintain the athletic fields and serve as coaches and/or athletic administrative staff. The organizational structure of the competitive sports area within the activities program is very similar to a traditional intercollegiate athletic department; however the complex structure is overseen by only four full time university employees. These advisors oversee several different sports per semester and serve in a primarily administrative function. Advisors have a light touch at different levels throughout the organization, however day-to-day activities are largely student run (J. Garner, personal communication, November 16, 2011). In order to split up the workload, simulate the real world, and provide tremendous opportunities for leadership, each semester a student is selected to serve as the area director and two students are selected to serve as directors of programs and operations, respectively. Coordinators are then chosen to oversee each sport and finally coaches are selected for each team (J. Garner, personal communication, November 16, 2011). In 2010, there were 386 students who served in leadership capacities within the Competitive Sports Program. An additional 277 were employed in games management or officiating positions, and many more volunteered in other capacities to help the program run (J. Garner, personal communication, November 10, 2011). Because of the significant time commitment required of participants in the Competitive Sports Program, students are limited to participation in one sport per semester, and most sports have the option to simultaneously be an administrator or coach and participant.

Outcomes of the Transition from Intercollegiate Athletics to Competitive Intra-Collegiate Athletics

The new model of competitive intra-collegiate athletics that has been implemented at BYU Idaho has brought a number of effects to the university and community. The most notable impacts on the university from an administrative standpoint have been the tremendous cost savings the new program has facilitated, and the ability to accommodate all students who hold the interest and ability to participate in competitive intercollegiate athletics. From a student standpoint, perhaps the most notable impact of the transition has been the addition of impactful leadership opportunities that participants consistently describe as highly educational, worthwhile and valuable experiences that are instrumental in enhancing personal growth (see Table 1). Other direct impacts of the Competitive Athletics Program have been noted in student recruiting and donor philanthropy. Supporters of the intra-collegiate program cite the administrative, sportsmanship, and leadership skills that students are able to develop as the primary benefits of the model with the cost-savings as an added bonus (Jacobson, 2005).

The decision to replace intercollegiate athletics with a competitive intra-collegiate athletic program was largely driven by economics. Though David A. Bednar, who served as the university's president during the transition, declined to disclose financial figures for Ricks College varsity athletics, he did state publically that the intra-collegiate activities program would operate at a fraction of a traditional intercollegiate budget as travel often demands a large percentage of the funds (J.Garner, personal communication, November 16, 2011; Jacobson 2005). Evidence of this forecast has come to fruition.

Staffing cost savings related to the administrative and coaching salaries have decreased significantly as the Competitive Sports Program operates with just a fraction of the staff members that were employed by the Ricks College athletic department. Comparing operational budgets of sports offered under the Ricks College athletic department with the same sports offered through the Competitive Sports Program, sport-by-sport operational budgets operate at between 3-13% of what was spent under the traditional intercollegiate athletics model. This figure accounts for the average decreases in sport-by-sport expenditures related to travel; (representing approximately 34% of the operational budgets); scholarships (representing approximately 58% of the operational budgets), and "other" expenditures including equipment costs, office supplies, equipment repairs, cell phone costs, etc. (representing approximately 8% of the operational budgets). In aggregate, these sports operate in the Competitive Sports Program for less than 8% of the Ricks College system operational budgets while serving 920% more students (J.Garner, personal communication, June 9, 2012).

Table 1 Student Leader Experiences (Fall 2010, Winter 2011, Spring 2011)

	Mean	SD
Transferable Skills Developed*	6.30	1.15
<i>I learned leadership principles that I can apply to other settings.</i>		
Experience was Educational*	6.29	1.16
<i>My experience in Activities helped me learn practical leadership skills.</i>		
Experience Facilitated Personal Growth & Development*	6.27	1.15
<i>I have personally grown and developed from this leadership experience.</i>		
Experience was Worthwhile*	6.22	1.25
<i>My leadership experience in the Activities Program was worthwhile...</i>		
Felt trusted in role*	6.09	1.25
<i>I felt trusted in my role running a program or event.</i>		
Felt Empowered*	5.96	1.31
<i>I was able to act, have my ideas heard, and make a difference.</i>		
Felt Influential*	5.82	1.37
<i>I felt like I was in a position where I could “grow” people under my supervision.</i>		
Experience was Critical to BYU Experience	5.10	1.51
<i>How critical was the leadership role to your overall BYU experience?</i>		

Note. The scale ranged from Strongly Disagree or Not Critical (1) to Strongly Agree or Extremely Critical (7)

* $p < .001 (\mu \geq 5)$

n = 267

Participation in the competitive intra-collegiate athletic program has increased participation opportunities dramatically. Under the old intercollegiate model in 2000, 264 athletes played nine varsity sports. Ten years later, under the new model, 2,433 athletes participated in competitive intra-collegiate athletics with 212 unique teams in 19 sports (see Table 2). Because of the minimal financial investment required to field and operate the teams, a broad-based program is possible. As of 2010, nineteen different sports were fielded, many with male and female leagues and between three and thirty-four teams per league, with football capped at eight teams (see Table 3) (J. Garner, personal communication, November 16, 2011; Competitive Sport Program, 2011). The addition of sports has come based on the interests and abilities of students, and that level of interest and ability continues to rise as the program gets more visibility. This program provides many individuals the opportunity to compete after high school that they wouldn't otherwise be able to do. These participation and leadership opportunities fit with one of the guiding principles of the Activities Program that students should be participants rather than spectators (J. Garner, personal communication, November 4, 2011).

Chris Moore, the Director of Philanthropies at BYU Idaho, recalled the initial hit in giving that was taken when it was announced the athletic program would be discontinued. “People were upset,” he recollects (personal communication, June 12, 2012). One donor in particular who had been a long-time supporter of the renowned athletics program was so upset that he asked for a significant ~\$30,000-40,000 gift back. While there was an initial dip in giving after the athletics program was discontinued, there has been an overall increase in giving for the institution since the transition. It is difficult to pinpoint the source of this increase, however, because the entire institution has changed. Before, if someone wanted to give to athletics, they could make a direct donation, but now those funds would be directed toward the general scholarship fund.

Discussing some of the direct benefits of the Competitive Athletics Program as seen in the philanthropies office, Moore mentioned the “amazing graduates” from the first 3-5 years of BYU Idaho who already major donors and some

Table 2 Competitive Athletic Participation Opportunities in Ricks College Athletics (2000) and BYU Idaho Competitive Sports Program (2010)

	Ricks College Athletics		BYU Idaho Competitive Sports	
	Men's	Women's	Men's	Women's
Baseball	x			x
Basketball	x	x	x	x
Cross Country	x	x	x	x
Football	x		x	
Golf			x	x
Ice Hockey			x	
Lacrosse			x	x
Soccer			x	x
Fast Pitch Softball		x		x
Swimming			x	x
Tennis			x	x
Track & Field	x	x	x	x
Ultimate Frisbee			x	x
Volleyball		x	x	x
Wrestling	x		x	
Spirit/Dance	x	x	x	x
Total # of Athletes	264		2883*	

*Represents total participation opportunities from teams in fall (n = 918), winter (n = 710), and spring (n = 1255). During this time frame there were a total of 2433 unique participants

Table 3 Teams in the 2010 BYU Idaho Competitive Sports Program

	Fall			Winter			Spring			Total
	M	W	C	M	W	C	M	W	C	
Baseball	6						6			12
Basketball (Varsity)				12	6		10	6		34
Basketball (JV)				12	6		10			28
Cheer Squad		1				1			1	3
Color Guard		1								1
Cross Country	5	5								10
Dance Team		1			1			1		3
Football	6									6
Golf								4	4	
Ice Hockey				6						6
Lacrosse							4	3		7
Soccer	6	6					6	6		24
Softball		4					4			8
Swimming			3					3		6
Tennis						6				6
Track & Field	5	4					5	4		18
Ultimate Frisbee							4	10		30
Volleyball	4	12								3
Wrestling				3						
Total Teams	32	33	4	33	13	1	51	34	11	212

Note. M = men's teams, W = women's teams, C = combined teams

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of the school's best alumni. Moore believes their experiences with leadership in the Activities Program were integral in tying them to the university. Because of the number of opportunities present in this program, Moore expects the benefits in terms of giving to continue to increase as the program matures (personal communication, June 12, 2012). Related to how much the Competitive Sports Program is present in donor visit conversations, "the Activities Program is a great story, and we love to talk about the wonderful things happening there...but because there isn't a whole lot of money needed to run the program, it's not the best pitch to donors. (T. Moore, personal communication, June 12, 2012). The Activities Program generally comes up in discussions as a wonderful leadership program, but the majority of funding conversations center on the educational initiatives that are in need of funding (personal communication, June 12, 2012).

Tyler Williams, the Director of Admissions at BYU Idaho, commented on his observations related to direct impacts of the Competitive Athletics Program and its "resoundingly positive impact on quality student recruitment" (personal communication, June 8, 2012). As he and other members of the admissions office have travelled to meet with prospective students, they have noted a tremendous amount of enthusiasm when they describe the Competitive Athletics Program. "When students learn there are still opportunities to participate in sport at a high level beyond intramurals – that they get coaching time, practice time, and structure – they are excited about that" (T. Williams, personal communication, June 8, 2012). Williams also mentioned the influx of transfer students who are athletes that have noted the Competitive Athletics Program as an element of their transfer decision. These students have expressed a desire to get to do what they love to do – play their sport – while being able to focus on academics. These transfer athletes have noted the stress, intensity, and travel schedule they were required to maintain as collegiate athletes and chose the BYU Idaho model as one that would fit their individual educational needs more fully.

There are other students and parents who are disappointed to find out that the school doesn't have a football or basketball program that they can root for...but these voices have been the minority. "Most believe it is more exciting to participate than to watch others participate. That is the kind of student that is attracted to our school" (T. Williams, personal communication, June 8, 2012). The recruiting office has also received its fair share of occasional parents that offer negative feedback related to the program who reflect on their nostalgia from the old glory days of the athletic department. Generally, Williams noted, once these students or parents get to campus and gets a feel for the Activities Program, their pre-conceived notions of what campus might be missing without the varsity athletic program melt away. "Upon observation, it is evident the Activities Program provides a laboratory for learning. Students are able to put into practice what they are taught and can develop new leadership skills - it's a very effective model" (personal communication, June 8, 2012).

While the opportunities for experience are great within the model, there are challenges. The primary challenge in this model is the customary turnover of student-leadership which creates a continual staffing concern. This turnover can at times lead to inefficiency. Additionally, because the students are fulfilling all the roles from umpire and referee to athlete, coach, and administrator, there have been isolated instances where a student has a conflict of interest that hurts the integrity of the system (J. Garner, personal communication, November 4, 2011).

The role of the community has also taken a different form - there are generally not a lot of fans in the stands. A large crowd for a championship typically consists of 800-1000 people while average games only draw a few hundred spectators. Where at one point there was a fair amount of community involvement as fans followed their nationally ranked teams, the new model which involves a shuffle of teams each semester is not as fan friendly (J. Garner, personal communication, November 4, 2011). This leaves somewhat of a void in opportunities to unite the students and create school spirit and strong community relationships through athletics. The reality of the intra-collegiate athletic model is that the institution will never be better than .500, and while the level of play is good across the leagues due to the competitive parity, it is not equal in quality to that of intercollegiate sports.

Conclusion

In 2010, only 22 athletics programs in the NCAA Football Bowl Subdivision—the wealthiest intercollegiate athletic division—reported positive net generated revenue without allocated institutional support - up from 14 schools in 2009 (NCAA, 2011). Most athletic programs through all levels of intercollegiate athletics rely heavily on institutional support to supplement the salary, scholarship, travel, and other administrative and infrastructural financial demands inherent in

athletic operation (NCAA, 2006), and despite this support there is systemic deficit spending (Dadigan, 2010; Knight Commission 2010; NCAA 2011). The competitive nature of athletics and the lure of commercial enticements at some levels have also led to patterns of abuse in athletic administration that in many ways threaten the sanctity of college sport (Dadigan, 2010; Splitt, 2009; Upton, 2011). These compounding issues have led many intercollegiate athletic stakeholders to question the current model (Branch, 2011; Byers, 1995; Sack, 2009; Splitt, 2009).

The BYU Idaho intra-collegiate athletic model offers an illustration of an administrative structure that dramatically increases participation opportunities while decreasing expenditures. While the model is quite a divergence from traditional varsity athletics, it fulfills an educational mission “providing opportunities for individuals to develop personal honor in a disciplined team environment” (BYU Idaho Competitive Sport Program, 2011). Given the purpose of athletics within the academe - to facilitate a holistic education difficult to replicate through any other educational opportunity (Brand, 2006; NCAA 2010; Rader, 1999) - this program sheds light on a new way to think about this tradition we have come to know as college sport.

As Aaron Kelly reviewed his notes for the Summit presentation, he reflected upon his first visit to BYU Idaho and his meeting with the Director of the Activities Program, Justin Garner. When the announcement came to the students, staff, and community that Ricks College would discontinue its intercollegiate athletics program and begin a new model of competitive intra-collegiate athletics, Garner was coaching and serving as an assistant athletic director of a very successful National Junior College Athletic Association program. “We didn’t know what the new model would look like, or what it meant to the university,” he recalled, “but we believed in our leader and so we rolled up our sleeves and went to work. Looking back,” he reflected, “I don’t think any of us would have believed how great the new model would become. The quality is remarkable – it’s a different quality, but it meets our mission and it is a great program” (J. Garner, personal communication, November 4, 2011). It is this great program that Aaron Kelly hoped the Summit attendees would embrace.

In just a few days, Kelly would be sharing the BYU Idaho intra-collegiate athletics model to a panel of leaders in the industry. While confident in his research and excited about the recommendations he would propose, he was troubled by the reality that this model might be too different, and that it might not be an ideal fit for many institutions – particularly those with “big time” athletics programs. As he contemplated the potential reaction of those that would soon enter the Summit venue, he questioned whether this was the best model to present, and how the landscape of intercollegiate athletics would be affected if this model were instituted on a national scale.

Case Questions

1. If an institution is hoping to decrease costs and increase participation opportunities in their intercollegiate athletics program, what are some important considerations in evaluating competitive intra-collegiate athletics as a potential model?
2. What are the benefits and/or drawbacks of the competitive intra-collegiate athletics model?
3. What factors should an administrator consider in maximizing the educational potential of athletics within their institution?
4. What factors should an administrator consider in maximizing the commercial potential of athletics within their institution?
5. What impact would the competitive intra-collegiate athletics model have on the institution’s brand and/or admissions?
6. What would be the implications of implementing this model at your institution?
7. How might the dropping of intercollegiate athletics affect an institution’s local economy? What factors in this case minimized the negative effects dropping intercollegiate athletics might have on the local economy?
8. What factor would the *public* or *private* status of an institution play in the administration’s ability to drop intercollegiate athletics in favor of an intra-collegiate athletic model?
9. What impact might there be on achieving racial diversity or gender equity in an intra-collegiate model compared to the intercollegiate model?

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Is Bigger Really Better? Obesity Among High School Football Players, Player Position, and Team Success

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Abstract

Objective. American football is one of the most common high school sports in the United States. We examine obesity among high school football players, and variations based on positions, team division, and team success. **Patients and methods.** We used 2 data sets from the North Carolina High School Athletic Association ($n = 2026$) and MaxPreps ($n = 6417$). We examined body mass index, calculated using coach-reported height and weight, by player position, division, and success based on win-loss percentage. **Results.** Most players (62%) were skill players, with 35% linemen and 3% punters/kickers. Most skill players (62%) were healthy weight and 4% obese or morbidly obese. In contrast, only 8% of linemen were healthy weight, with 21% morbidly obese. Team success was correlated with size only for skill players. **Conclusions.** Obesity is a significant problem for high school football players. Pediatricians should consider the context of football playing in assessing long-term health risks for these young men.

Keywords

obesity, physical activity, adolescents

American football is one of the most common high school sports in the United States, one deeply ingrained in many communities throughout the country. In the context of the current childhood obesity “epidemic,” clinicians and others commonly recommend physical activity, including team-based sports, as a prevention and treatment strategy. Football, however, has unique characteristics that may not promote healthy weight for the 1.1 million high school youth participating in 11-player football.

Given the widespread perception that football players should be “big,” an increased prevalence of obesity among its players may seem obvious. Football, though, unlike many other sports, has significant within-sport variation by position in the “ideal” size of players. Much previous research has examined obesity among college, National Football League (NFL), and retired football players.^{1–4} Obesity has been shown to be a significant problem and to be particularly concentrated in playing positions where larger size is seen as an advantage.⁵ One small study of high school football players showed that 45% of offensive linemen were obese.⁶ However, collegiate and NFL players have gone through significant selection based on their playing skill and success. High school football, with its much wider reach in terms of

the number of players, may not have the same patterns of obesity throughout its players. Even so, taller children are more likely to be overweight, and selection of players based on both greater height and weight may result in disproportionate numbers of overweight football players.⁷

As these players experience the natural growth and development of adolescence, high school may be a time period during which some players believe they should purposefully gain weight, perhaps beyond healthy levels. This is a time they are most likely to emulate their college and NFL heroes, and they want to increase size, speed, and strength to earn college scholarships.⁸ Larger size is a social norm among football players, and they

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may receive encouragement from parents, coaches, and friends who also hope for long-term success for the player. Coaches in particular may encourage greater size as a means of improving the success of the team.

For many young athletes, football is seen as a path to success—whether in the form of a college scholarship or a professional career. The vast majority of high school football players will never reach these higher levels, but they will carry with them their experiences as part of their teams. Because of the sheer number of young players, identifying whether those playing certain positions are more likely to be obese can help to target obesity interventions to particularly high-risk individuals. The goal of this study is to examine the presence and distribution of obesity among high school football players, and examine variations based on positions, division in which their school football team participates, and success of the team.

Methods

Data

We obtained 2 different but similar data sets through the North Carolina High School Athletic Association (NCHSAA), each of which conferred different advantages in terms of size and included information. The first data set (MaxPreps) was obtained by NCHSAA from MaxPreps, a national company that collects data on high school sports throughout the country. For this study, we used data they collected for North Carolina athletes. To ensure confidentiality of the players, these data were provided as a completely de-identified data set, with team names removed. Although MaxPreps collects data for players across the country, we used only North Carolina data because of our access to the second data set, which included additional useful information. The second (NCHSAA) includes data collected directly by the NCHSAA, primarily for media purposes during the playoff period for football teams within the state and represents a subset of the MaxPreps data. Importantly, this data identified the school, allowing us to assign a win/loss record in order to determine team success. All results were nearly identical with the 2 data sets. Because the MaxPreps data represents a larger group, we present results using this group of data, except for analyses requiring the measurement of team success.

Measures

Coaches reported height and weight for each player. From these reports, we calculated body mass index (BMI). We classified obesity based on adult criteria

using BMI: morbidly obese (>35), obese (30–35), overweight (25–30), healthy weight (18.5–25.0), and underweight (<18.5). We chose to use adult criteria because age was not available to us, only grade. Although we could estimate age from grade, we could not predict the effect of the error of this process. For this adolescent group, BMI values based on the child percentiles approach the adult values, and misclassification would result in a healthier weight using adult criteria than child criteria—a bias to the null, which is preferable to an unpredictable bias.

We anticipate the possibility of some overreporting by coaches of weight to make players appear “larger.” Along the same lines, we also expected overreporting of height, so impact on the calculated BMI should be minimal. Additionally, any bias in reporting should be consistent across teams—all coaches would likely overreport height and weight. To examine this effect, we repeated all of our analyses with an assumed overreported height of 2 inches and weight of 20 pounds. Mean BMI based on actual report and this assumption of overreporting were within 1.5 points of each other for all players and by player position. Because of the modest differences, we present only data based on coach reporting.

Positions

We initially classified players into mutually exclusive positions based on the first listed position as shown in Table 1. Players rarely play only a single position, particularly in high school football. Because we wanted mutually exclusive categories to better test for differences in weight, we also classified players into 3 broader groups: skill players, linemen, and punters/kickers (Table 1).

Class was based on coach-reported grade level for the traditional high school grades 9 to 12: freshmen, sophomores, juniors, and seniors. “Division” represents the state-classified division in which the team plays. Divisions are based on school size, and range from 1A (smallest) to 4A (largest). For our purposes here, we excluded the small number of teams that played in 8/9/10-man football divisions or independent school divisions. In addition to the very small number of players in these divisions, we believed they may represent a different population than traditional high school football teams.

Ranking

We developed our ranking of success based on win percentage. We used the win/loss records of all available

Table I. Position Classification.

Broad Classification	Classification	Specific Positions
Skill players	Tight ends	Tight ends
	Offensive backs	Running backs, quarterbacks, tailbacks, fullbacks, halfbacks, wingbacks
	Receivers	Wide receivers, split ends
	Linebackers	Linebackers, outside linebackers, middle linebackers, inside linebackers
	Defensive backs	Cornerbacks, safeties, strong safeties, free safeties
Linemen	Offensive linemen	Offensive linemen, guards, offensive tackles, centers
	Defensive linemen	Defensive linemen, nose guards, defensive tackles, nose tackles
Punters/kickers	Punters/kickers	Punters, kickers

teams in the NCHSAA data and categorized them into quartiles, representing 4 groups of “success” from worst to best.

Missing Data

Weight and height, needed to calculate BMI, were missing in 25% of the MaxPreps data. The majority of missing data occurred as part of an entire team, indicating that the coach did not report weight and height on any players. However, we examined whether there were differences in missing data based on other important variables. There were no significant differences in missing data based on the type of position played, the primary variable of interest. There were significantly more freshmen missing data on weight, compared with older students, though 70% of freshmen had complete BMI data. Because players are classified as skill players versus linemen early in their “careers” and the relatively small number of freshmen, we do not believe the increased missing data on freshmen will affect the results examining position.

Statistical Analysis

Using the MaxPreps data, we compared mean BMI across position, class, and division, using Wald tests to examine differences. We also examined obesity classification across position, using χ^2 tests to examine differences. Using the NCHSAA data, we examined the mean BMI of all players and by position based on win percentage quartile.

The study was determined by the University of North Carolina Institutional Review Board to be exempted from human subjects review.

Results

After excluding players with missing data, the NCHSAA data included 2026 players and the MaxPreps data

included 6417 players. Table 2 shows the distributions for each data set, and demonstrates strong similarities between the 2. Most players (61% to 62%) were skill players, with 35% to 36% linemen and 3% punters/kickers. Most players were upperclassmen. Finally, only 43% to 44% of all players were healthy weight, with 35% overweight, 14% to 15% obese, and 7% morbidly obese.

Table 3 presents mean BMI by player position, class, and division. Mean BMI for linemen was 31, whereas mean BMI for skill players was 24, and mean BMI for punters/kickers was 23. Differences by class were significant but small, with a mean BMI for freshmen of 26 and 27 for seniors. There were also statistically significant, but modest, differences by division, with 1A (smallest schools) having overall player BMI of 26, whereas 2A to 4A schools had mean player BMIs of 27.

Table 4 shows the distribution of weight status by player position. Skill players and punters/kickers were overwhelmingly healthy weight or overweight. Most skill players (62%) were healthy weight, with 34% overweight, and only 4% obese or morbidly obese. An even greater proportion of punters and kickers were healthy weight (74%) with virtually all of the remaining overweight. Linemen, however, showed a much different distribution. Only 8% of linemen were healthy weight, with 37% overweight, 34% obese, and 21% morbidly obese.

Table 5 shows the relationship between player size and team success, based on the win percentage. The only statistically significant difference by team success was for skill players, with larger players in the most successful teams. The difference from best to worst was less than 1 BMI point.

Discussion

Obesity is a significant problem for high school football players, but certain positions clearly attract players of

Table 2. Description of Samples for Each Data Set (Percentages).

	North Carolina High School Athletic Association (n = 2026)	MaxPreps (n = 6417)
Position		
Skill players	61.1	62.4
Linemen	35.6	34.5
Kickers/punters	3.3	3.1
Class		
Freshmen	6.6	4.1
Sophomore	19.7	14.4
Junior	34.6	39.3
Senior	39.1	42.2
Division		
1A	18.8	17.6
2A	20.6	19.9
3A	35.0	29.1
4A	25.6	33.4
Weight category		
Morbidly obese	7.0	7.4
Obese	15.4	14.0
Overweight	34.9	34.8
Healthy weight	42.5	43.6
Underweight	0.3	0.3

Table 3. Mean Body Mass Index by Position, Class, and Division (n = 6417).

	Mean	95% Confidence Interval	P ^a
Position			
Skill players	24.44	[24.35-24.52]	<.001
Linemen	31.27	[31.05-31.48]	
Kickers/punters	23.44	[23.04-23.84]	
Class			
Freshmen	25.62	[25.02-26.22]	<.001
Sophomore	26.18	[25.88-26.48]	
Junior	26.62	[26.43-26.82]	
Senior	27.20	[27.01-27.39]	
Division			
1A	26.23	[25.94-26.52]	<.001
2A	26.90	[26.63-27.18]	
3A	26.73	[26.50-26.95]	
4A	26.98	[26.78-27.19]	

^aP values are from Wald tests.

Table 4. Weight Status by Player Position, Percentage (n = 6417).

	Skill players	Linemen	Kickers/Punters	P ^a
Morbidly obese	0.3	20.8	1.0	<.001
Obese	3.5	34.1	1.0	
Overweight	34.0	37.3	23.0	
Healthy weight	61.9	7.8	73.5	
Underweight	0.4	0.0	1.5	

^aP value is from χ^2 test.

Table 5. Mean Body Mass Index Based on Team Success From (1) Best to (4) Worst (n = 2026).

	Mean	95% Confidence Interval	P
All players			
1	26.99	[26.61-27.37]	.737
2	26.86	[26.49-27.24]	
3	26.68	[26.28-27.08]	
4	26.79	[26.03-27.55]	
Skill players			
1	24.81	[24.54-25.08]	.005
2	24.55	[24.30-24.81]	
3	24.37	[24.07-24.68]	
4	23.94	[23.53-24.35]	
Linemen			
1	31.27	[30.63-31.90]	.796
2	31.09	[30.43-31.74]	
3	30.97	[30.28-31.66]	
4	31.68	[30.34-33.03]	
Kickers/punters			
1	23.44	[22.34-24.54]	.644
2	23.56	[22.60-24.51]	
3	22.73	[21.75-23.70]	
4	23.13	[19.85-26.40]	

greater weight. Although we are unable using these data to determine if players in these positions are attempting to gain weight, larger size is considered advantageous for these positions. Although there is little information about the dietary behaviors of high school football players, a study of college players showed significantly increased total energy, carbohydrate, and protein intakes during the playing season.⁹

Importantly, we did not just identify differences based on player position; we were able to demonstrate the magnitude of obesity among high school football players. That only 8% of linemen were healthy weight, and >20% of them had a BMI greater than 35, demonstrates a significant concern about obesity in this group. These findings reflect those that have been seen in other populations, particularly college and NFL players who play a line position.

One particularly unique finding is that weight of players did not appear to have a significant impact on success of the teams. Although the differences were statistically significant, the magnitude of difference was small, with the largest players in the second-ranked group, not the first. Of course, overall team success is not the only reason for players to feel pressure to increase their size, as shown in previous research. Coaches may encourage linemen to gain weight because they think it will help their team win and also because they might believe it will help the individual have a better chance to play in college.

Although we do not know the breakdown for North Carolina athletes specifically, NCAA statistics show that only 6.4% of high school football players go on to play in college, and only 0.08% of high school football players ever make it to the professional level.^{10,11} Despite these statistics, many young athletes aspire to football as their career. However, an overwhelming majority of these high school players will never participate at a higher level. Their efforts to gain weight in adolescence may lead to the development of ongoing obesity that becomes even more severe when they no longer have the physical activity to compensate for caloric intake.

One question we are unable to answer is whether the physical activity associated with playing football provides any protection against the development of cardiovascular risks among obese players. Previous research in collegiate and NFL athletes has shown mixed effects on cardiovascular risks, despite the regular vigorous physical activity. One study demonstrated that NFL players had similar or better values for several cardiovascular risk factors.⁵ Another study of college football players showed increased in total cholesterol and low-density lipoprotein during the playing season.⁹ This increased risk is particularly concerning for young high school athletes, who develop the dietary habits and weight they believe will improve their football skills. At the end of their high school careers, most will go on to occupations that do not provide similar levels of activity, carrying with them continued obesity into adulthood.

In the context of the clinical encounter, these findings demonstrate the importance of considering not only overall activity of adolescents, but the kinds of sports and specific roles they have on any sports teams. With well more than half a million high school-aged boys playing a lineman position, virtually all of whom are overweight or obese—and 126 000 of them who are morbidly obese, based on adult standards—this a problem that will regularly be encountered by the primary care physicians caring for these boys. Although sports medicine physicians and coaches are certainly concerned about the health of their players, they may have greater focus on immediate athletic performance. Pediatricians and primary care physicians may be in a better position to focus on long-term health. When asking adolescents about their diet and activity, pediatricians should carefully consider the importance of the kind of activities or sports, and the potential influence this has on their behaviors. Future research should carefully examine differences across the country and also compare adolescents playing different sports.

One particularly important issue is how to improve obesity in this population while not reducing participation in football or other physical activities. Ensuring that football players, as well as the coaches, administrators, and parents who guide them, have access to good nutrition information is critical. For example, previous work has shown that nearly a third of high school football coaches believe protein shakes are an important part of weight gain efforts for their players.¹² Developing healthy behaviors during high school is critical to prevention of further obesity development in adulthood. Primary care physicians can ensure that football players feeling pressure to gain weight have information about healthy nutrition that focuses on building lean mass.

An important consideration for anyone assessing these young athletes is the use of BMI as an assessment tool for evaluating variation and distribution of obesity in high school football athletes. Among collegiate athletes, higher frequencies of overweight/obesity by BMI in football groups appear to be driven by elevated fat-free mass rather than percentage fat, although linemen had the highest percentage fat.¹³ Other researchers have shown that age-adjusted BMI percentile rank in high school football players was not effective for determining overweight/obesity levels (based on adiposity) and lead to misclassification for weight status especially for “big” athletes.¹⁴ However, the utility of BMI for measuring fat mass versus fat-free mass is less important from the perspective of long-term obesity risk. Even if young football players have higher BMI due to fat-free mass, this is unlikely to continue to hold true once they discontinue the sport.

Limitations

The most obvious limitation is that we were only able to examine players in a single state. A second important limitation is that we relied on self-reported heights and weights. We anticipated the possibility of some over-reporting by coaches of height and weight to make players appear “larger”; however, we expect that the impact on BMI to be minimal assuming size adjustments to be roughly proportional and similarly consistent across teams.

Conclusions

A significant percentage of North Carolina high school football players are overweight or obese, particularly among linemen. Although increased size of linemen is seen as an “occupational hazard” of football, the severe obesity among linemen is cause for concern. More than one third of the 1.1 million high school players are linemen, creating a large population of players whose obesity is likely to persist into adulthood, placing them at risk for significant cardiovascular and metabolic health problems. Future research should examine whether football playing in high school actively encourages additional weight gain, and what this means for the long-term health of these young men.

Declaration of Conflicting Interests

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49.**FIREARM ACCESS DOCUMENTATION IN HIGH RISK CLINICAL SITUATIONS: MISSED OPPORTUNITIES***Eric Sigel, MD.**University of Colorado Denver School of Medicine.*

Purpose: The American Academy of Pediatrics, among other groups, strongly encourages screening for firearm access in pediatric patients, as there is significant morbidity and mortality related to having access to firearms, particularly among youth at risk for suicide. The current study was designed to determine whether health care providers (HCPs) miss opportunities to detect youth access to firearms in particularly high risk situations such as the presence of depression or violence risk.

Methods: Adolescents ages 12-17 scheduled for a routine physical exam in an urban, adolescent medicine clinic between Oct 2010-December 2011 were eligible. Assent from adolescents and consent from parents (both by phone) was obtained to participate in a study assessing violence risk. As part of usual care, youth had the opportunity to answer a standardized health assessment, including whether there were guns in their home. Chart review was completed one month after the appointment. Data collected included youth self-report of violence risk, depression (PHQ-9) and access to firearms in the home and provider documentation of violence risk, mental health diagnosis and the presence of firearms in the home. Rates of each were calculated. Bivariate analysis determined whether there were associations between provider documentation of mental health issues or violence risk and documentation of firearms in the home.

Results: 549/784 (70%) of eligible youth consented), and 85% of those (n= 466) actually completed their appointment. Mean age was 14.5 (sd 1.6); 65% female, 45 % Hispanic, 38% Black, 17% White. 300/466 (64%) completed the health screening questionnaire. Providers documented a mental health diagnosis in 22% of all patients, including 14% with Depression, 4% with ADHD, and 4% all others. Providers only documented either the presence or absence of a firearm in just 15.9% of these patients, with 2% documentation of the presence of a firearm. Youth who were diagnosed with a mental health issue reported having firearm access 13.8% of the time- only 20% of the time did providers recognize this. For the 14.6% of youth who were positive for moderate-severe depression on the PHQ-9 -in only 8.1% was there documentation of access to firearms. Similarly, 14.6% were positive for future violence risk, and in only 17% was there documentation by providers of firearm access. No demographic characteristics, including age, race/ethnicity, gender or SES were associated with whether a provider documented the presence of a firearm in the home for these high risk youth.

Conclusions: Despite the prevalence of mental health diagnosis and violence risk in this adolescent population, providers are largely not documenting youth access to firearms. Missed opportunities to counsel parents and youth about the risk of firearms in high risk situations can lead to potentially devastating consequences. Ways to document access to firearms needs to be improved.

Sources of Support: Colorado Injury Control Research Center, Children's Hospital Colorado Research Institute, Deans Academic Enrichment Fund.

50.**2011 NORTH CAROLINA YRBS: ATHLETIC PARTICIPATION, VIOLENCE, AND BULLYING**

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Purpose: Athletic participation may prevent youth from engaging in risky behavior including violence. We sought to determine the association of athletic participation with participation in violence-related activities.

Methods: We used data from the 2011 North Carolina Youth Risk Behavior Survey. This survey is administered to high school students and provides state-representative data on a variety of behaviors. We used two questions related to sports, which asked if the student played a school-sponsored sport that was team-based (e.g., football) or individual-based (e.g., track). We then examined reports of fighting, carrying weapons, and bullying. We used adjusted Wald tests to examine differences in violence by the type of sport played, which adjust for the complex survey design of the NC YRBS.

Results: Half of the 1820 surveyed students ages 14-18 years reported participation in a school-sponsored sport: 25% team sports, 9% individual sports, and 17% both types. Girls who played sports were less likely to have been in a physical fight in the last year (14% vs. 22%, p < 0.05); there were no differences for boys or by type of sport. Girls playing sports were also less likely to have carried a weapon to school in the past 30 days (6% vs. 11%, p < 0.05); however, there were no differences for boys or type of sport. There was a non-significant trend towards boys playing sports being less likely to report having been bullied (20% vs. 25%, p = 0.17). Yet, boys playing only individual sports were more likely to report having been bullied than those playing team sports (29% vs. 18%, p < 0.01) with a similar trend for girls (41% vs. 32%, p = 0.14).

Conclusions: Our results suggest that childhood sport participation may have important relationships with violence-related activity. While girls were less likely to fight or carry a weapon when involved in sports, the same protective effect was not seen for boys. Boys who played team sports were less likely to report being bullied than boys who played individual sports. It is not known if boys who play team sports are less likely to be the perpetrators of bullying. Future research should examine why sports may be protective for girls' but not boys and if interventions involving pediatricians or schools can influence these relationships.

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51.**A RANDOMIZED CONTROLLED EVALUATION OF THE EFFECTS OF THE FAMILIAS EN ACCIÓN SCHOLARSHIP PROGRAM ON ADOLESCENT VIOLENCE, SUBSTANCE USE, AND UNSAFE DRIVING**

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prevalence. Activity was most protective for those who played football in high school, while obesity increased among those who played basketball or no sports regardless of activity levels.

Conclusions: High school sports participation had different effects on physical activity levels and the prevalence of obesity among young adult males. Unlike adolescents that played no sports or football exclusively, athletes that participated in basketball and football, or basketball exclusively experience a lower prevalence of obesity in early adulthood. Orientation to physical activity played an important role in the increased levels of obesity as adolescents' transitioned to young adulthood. Our findings suggest that as basketball players become less active in late adolescence, the prevalence of obesity in this group begins to mirror football players. Further research is needed to determine the reason for the decreased level of physical activity as basketball players reach early adulthood.

Sources of Support: NC TraCS Translational Research Pilot grant #2KR451216.

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REPORTED SPORTS PARTICIPATION, SEX, AND OBESITY IN A NATIONALLY REPRESENTATIVE SAMPLE

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Purpose: Participation in organized sports is thought to provide beneficial opportunities for physical activity among adolescents. Identifying demographic and weight status patterns of sports participation among youth is essential to developing effective obesity interventions. We sought to examine how sex and obesity relate to sport participation in a nationally representative sample of adolescents.

Methods: Adolescents 12–19 years from the National Health and Nutrition Examination Survey (NHANES, 1999–2006) reported leisure activities over the last month and had measured height and weight. We used these reports to identify participation in the five most common male and female sports. We created mutually exclusive categories of the “most frequent” sport to maximize the likelihood that participation was part of a team, based on time spent playing the sport, level of activity, and number of occasions playing the sport. We used adjusted Wald tests to examine the differences in sports participation by demographics and prevalence of obesity (BMI = 95%) by sport, for boys and girls separately.

Results: For girls, 52% participated in at least one of the five most common sports, compared to 71% of boys; participation declined from middle to high school for both genders. Running and basketball were the most common sports played by girls, while running, basketball, and football were most popular among boys. Overall, girls playing sports were no less likely to be obese than non-players; only runners had a significantly lower prevalence of obesity (11% vs. 17%, $p < 0.01$). In contrast, the prevalence of obesity among boys who did not play sports was higher than those who did (23.6% vs. 16.6%, $p < 0.01$). This was true for all sports, except football, whose players’ obesity rates did not differ from those not playing sports.

Conclusions: Our findings demonstrate that male and female adolescents who participate in organized sports have a lower prevalence of obesity than non-athletes or participants in other activities.

However, the effect of organized sports participation on obesity for female adolescents is not significant except for running/track. Physicians who counsel parents and girls toward sports participation should recognize that while participation in sports may have significant benefits, the relationship between participation and healthy weight is not as clear, especially for girls. Physicians and policy-makers should consider the gender and type of sport played when recommending participation as an obesity prevention strategy, and focus on encouraging continued participation as adolescents age.

Sources of Support: Provided by UNC CTSA RR025747. The Carolina Postdoctoral Fellowship Program offered support for Dr. Turner. Dr. Skinner is currently supported by BIRCH (K12-HD01441).

155.

IT'S NOT YOU IT'S ME: ASSOCIATION BETWEEN MATERNAL PERCEPTIONS OF RESPONSIBILITY FOR ADOLESCENTS' EXCESS WEIGHT AND PEDIATRIC WEIGHT MANAGEMENT PROGRAM ATTRITION

Paul R. Joseph, BS, Acham Gebremariam, MS, Bethany J. Gaffka, PhD, Susan J. Woolford, MD, MPH.

University of Michigan Health System.

Purpose: Obese adolescents 15–17 years old are nearly 18 times as likely to become obese adults as their normal weight peers. Pediatric weight management programs have the unique opportunity to help adolescents achieve a healthy weight at this critical time in their life. However, such programs typically achieve only modest success in part due to problems with patient engagement and attrition. This research aims to explore possible associations between maternal perceptions of their responsibility for their adolescent's excess weight and whether they join and remain in a multidisciplinary weight management program.

Methods: This retrospective study utilized survey data from obese adolescents (12–18 years old, BMI $\geq 95^{\text{th}}$ percentile) who attended an initial visit in the Michigan Pediatric Outpatient Weight Evaluation and Reduction (MPOWER) program between March 2007 and July 2010. The following question on a pre-enrollment survey assessed maternal perceptions of responsibility: How much do you blame yourself for your child's weight problem? Response options were 0–no blame, 1–little blame, 2–some blame, 3–lots of blame. For this analysis, we dichotomized responses into little responsibility (0 and 1) and lots of responsibility (2 and 3). To assess joining status, patients were assigned a 1 if they joined the program or a 0 if they did not join. Among those who joined, attrition was assessed as a binary variable where 1 = patients who remained in the program for 3 months or longer and 0 = patients who dropped out before 3 months. Responsibility, joining status and attrition were compared using bivariate analysis and multivariate regression modeling.

Results: Program participants ($n = 351$) had a mean age of 14.4 years and a mean BMI of 41.8 kg/m^2 at the start of the program. Only cases in which surveys were completed by the mother were included in this analysis (84%). No statistically significant associations were found between joining status and degree of responsibility ($p = 0.15$). We found that a greater percentage of families with mothers who reported high levels of responsibility for their child's excess weight stayed in the program for 3 months or longer when compared to families in which mothers endorsed little responsibility (72% vs. 35% respectively, $p = 0.03$).

presence of a psychiatric co-morbidity was not significant. However a trend was observed with patients in the lowest weight gain quartile and no psychiatric co-morbidity having a predicted probability of readmission of 21.8% (SE = 19.2), while those patients with a psychiatric comorbidity and in the highest quartile of rate of weight gain had a probability of readmission of 48.4% (SE = 17.3).

Conclusions: This study was not sufficiently powered to detect the effect of rate of weight gain on readmission. However the observed trend of increased readmissions with rapid weight gain strongly indicates that further study is indicated. Future studies should aim to be powered to detect interactions with psychiatric co-morbidity and rate of weight gain.

Sources of Support: ACGME Fellowship through Texas Children's Hospital 2531202692.

OBESITY

152.

DIAGNOSTIC EVALUATION AND COMORBIDITY SCREENING FOR POLYCYSTIC OVARY SYNDROME IN ADOLESCENTS: DOES IT DIFFER ACROSS SUB-SPECIALTIES?

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Texas Children's Hospital.

Purpose: Polycystic Ovary Syndrome (PCOS) is a diagnosis that can have significant long term health implications. Forty to 85% of adolescents with PCOS are overweight or obese and > 60% have at least one component of metabolic syndrome. The diagnostic criteria for PCOS in adolescents have been subject to much debate. There are no consensus guidelines regarding comorbidity screening. This leads to considerable practice variability between providers and among specialties. Lack of sufficient evaluation risks underdiagnoses and unnecessary tests add to the cost of care. The purpose of this project was to evaluate and characterize this practice heterogeneity.

Methods: Retrospective chart review of a random sample of 103 electronic medical records of adolescent girls (11 – 21 years) who presented to the adolescent medicine (AM), pediatric endocrinology (ENDO) and gynecology (GYN) clinics at a large children's hospital over a 1 year period (2011-2012), with visit diagnosis codes of PCOS, menstrual cycle disorders or hirsutism was performed. Patients diagnosed with a non-PCOS cause for symptoms were excluded. Data abstracted included details on age, age at menarche, symptoms, BMI, BMI percentile, examination findings, laboratory tests ordered and management plan. The evaluations performed were compared across specialties using Chi -square, Fischer exact and Kruskal-Wallis test, as appropriate.

Results: Fifty-seven patients (18 AM, 20 GYN and 19 ENDO) were eligible for the study. Baseline characteristics including age, reproductive age, age at menarche, menstrual symptom and presence of overweight or obesity were similar across the clinics. The proportion of patients who received LH, FSH, testosterone, prolactin and TSH testing were also similar. As compared to patients in GYN and ENDO, AM patients were less likely to be tested for levels of DHEA-sulfate (95 and 75%, respectively vs. 33%, p < 0.001) and 17 hydroxy progesterone (90 and 78%, respectively vs. 11%, p < 0.001). They were also less likely to receive a pelvic ultrasound (95% in GYN and 47% in ENDO vs. 11% in AM, p < 0.05).

Thirty percent of patients with BMI in the overweight and obese range were not identified. Girls in AM and ENDO were more likely to be identified as having elevated weight (92.8% and 100%, respectively vs. 16.7% in GYN, p < 0.001), acanthosis nigricans (85.7% and 72.7%, respectively vs. 33.3% in GYN, p < 0.001) and to receive advice or referrals for weight management (71.4% and 72.7%, respectively vs. 16.7% in GYN p < 0.001). Proportion of patients who received tests for dyslipidemia (51.4%), diabetes (56.7%) and liver enzyme derangement (54.1%) were not significantly different among clinics.

Conclusions: There is variability across specialties in evaluating adolescent PCOS with significant under evaluation of co-morbidities. The balance between cost and benefit needs to be considered. In order to better identify and treat complications of PCOS, consensus amongst specialists is needed, with subsequent emphasis on dissemination and provider education.

Sources of Support: ACGME Fellowship-Texas Children's Hospital 2531202692.

153.

HIGH SCHOOL SPORTS PARTICIPATION: RISK AND PROTECTIVE FACTORS FOR EARLY ADULTHOOD PHYSICAL ACTIVITY AND OBESITY IN A REPRESENTATIVE LONGITUDINAL STUDY

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Purpose: To determine the association between adolescent boys' participation in high school sports (basketball, football, or both) and early adulthood by: (a) levels of physical activity and (b) obesity prevalence.

Methods: The data come from the in-school student questionnaire, the in-home student interview and the parent interview during multiple waves of the National Longitudinal Study of Adolescent Health (Add Health), a school-based study of the early life course. We examined the association of adolescent male obesity with participants in basketball and football (as well as students that reported participating in both sports and no sports). We confined the final analysis to 9,267 male adolescents with valid survey weights that had weight and activity data for at least one of the four survey waves during a 15-year period, Wave I in 1994-1995 to Wave IV in 2008-2009. Measures of BMI and physical activity were collected in every wave while the majority of demographic information was collected in the first two waves. Parent and household information (parental education, household structure, and family income) were also only collected during Wave I and II. We used questionnaire items to construct measurements of recreational sports, traditional sports and cardio are identical in Wave I and Wave II. These items are also identical between Wave III and Wave IV. The differences between the first two waves and the latter two waves are that activities are spread across a greater number of items.

Results: Boys playing no sports were less likely to participate in traditional sports or cardiovascular activity at waves 1, 2, and 3. By wave 4, there were no significant differences in activity based on sports participation in high school. Obesity prevalence was significantly greater at all waves for boys who played football in high school and lowest for boys who played basketball. Higher levels of activity were consistently associated with lower obesity

prevalence. Activity was most protective for those who played football in high school, while obesity increased among those who played basketball or no sports regardless of activity levels.

Conclusions: High school sports participation had different effects on physical activity levels and the prevalence of obesity among young adult males. Unlike adolescents that played no sports or football exclusively, athletes that participated in basketball and football, or basketball exclusively experience a lower prevalence of obesity in early adulthood. Orientation to physical activity played an important role in the increased levels of obesity as adolescents' transitioned to young adulthood. Our findings suggest that as basketball players become less active in late adolescence, the prevalence of obesity in this group begins to mirror football players. Further research is needed to determine the reason for the decreased level of physical activity as basketball players reach early adulthood.

Sources of Support: NC TraCS Translational Research Pilot grant #2KR451216.

154.

REPORTED SPORTS PARTICIPATION, SEX, AND OBESITY IN A NATIONALLY REPRESENTATIVE SAMPLE

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Purpose: Participation in organized sports is thought to provide beneficial opportunities for physical activity among adolescents. Identifying demographic and weight status patterns of sports participation among youth is essential to developing effective obesity interventions. We sought to examine how sex and obesity relate to sport participation in a nationally representative sample of adolescents.

Methods: Adolescents 12–19 years from the National Health and Nutrition Examination Survey (NHANES, 1999–2006) reported leisure activities over the last month and had measured height and weight. We used these reports to identify participation in the five most common male and female sports. We created mutually exclusive categories of the “most frequent” sport to maximize the likelihood that participation was part of a team, based on time spent playing the sport, level of activity, and number of occasions playing the sport. We used adjusted Wald tests to examine the differences in sports participation by demographics and prevalence of obesity (BMI = 95%) by sport, for boys and girls separately.

Results: For girls, 52% participated in at least one of the five most common sports, compared to 71% of boys; participation declined from middle to high school for both genders. Running and basketball were the most common sports played by girls, while running, basketball, and football were most popular among boys. Overall, girls playing sports were no less likely to be obese than non-players; only runners had a significantly lower prevalence of obesity (11% vs. 17%, $p < 0.01$). In contrast, the prevalence of obesity among boys who did not play sports was higher than those who did (23.6% vs. 16.6%, $p < 0.01$). This was true for all sports, except football, whose players’ obesity rates did not differ from those not playing sports.

Conclusions: Our findings demonstrate that male and female adolescents who participate in organized sports have a lower prevalence of obesity than non-athletes or participants in other activities.

However, the effect of organized sports participation on obesity for female adolescents is not significant except for running/track. Physicians who counsel parents and girls toward sports participation should recognize that while participation in sports may have significant benefits, the relationship between participation and healthy weight is not as clear, especially for girls. Physicians and policy-makers should consider the gender and type of sport played when recommending participation as an obesity prevention strategy, and focus on encouraging continued participation as adolescents age.

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Athlete Graduation Rate Gaps at Division-I State Flagship Universities: An Exploratory Analysis Emphasizing Black Males

**Robert W. Turner II
Richard M. Southall
Woody Eckard**

ABSTRACT: Discrepancies in Black male graduation rates at NCAA Division-I state flagship institutions have raised questions about claims of unilateral academic progress among certain revenue sport athletes. Researchers have identified gaps in NCAA and federal graduation rates between athletes and non-athletes based on race and type of sport participation. This exploratory study examines the degree to which graduation rates vary between football and male basketball athletes and male undergraduates at state flagship institutions. We pay particular attention to gaps in graduation rates for Black male athletes. We then seek theoretical explanations for these gaps by drawing on the athletic role-engulfment and key-player hypothesis, the mismatch education hypothesis, and the institutional isomorphism theory.

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While affirmative action has and continues to be a major concern on college campuses, there's one place where there does not seem to be a need for it: intercollegiate athletics, especially in National Collegiate Athletic Association (NCAA) Football Bowl Subdivision (FBS) football and NCAA men's basketball. Within athletic departments one finds huge racial discrepancies with African Americans dominating some Division-I teams, but being totally absent from others. Indeed, in the most recent report Black males outnumber Whites in Division-I football for the first time since the NCAA began tracking data in 1999. In NCAA D-I men's basketball, Black athletes now account for nearly 61% of participants (Associated Press, 2010). An awareness of the seeming over-representation of African Americans in certain sports on many NCAA D-I campuses led to analysis and then concern about these students' educational outcomes. The issue first gained national attention in the late 1980s when two former NCAA and Olympic basketball players worked with members of Congress to force U.S. colleges and universities to publish athlete graduation rates (Selingo, 2012). Attention crested in 1999 as many observers were troubled by low graduation rates among Division-I football and men's basketball athletes.

Historically, low NCAA Division-I Black male football and basketball graduation rates have been a lightning rod issue. This criticism recently resurfaced in response to data released by the University of Pennsylvania that suggests these athletes graduated at a rate 22 percentage points lower than the general undergraduate student population and 5 percentage points lower than Black male undergraduates (Harper, Williams, & Blackman, 2013). Additional research has identified significant gaps between the graduation rates of full-time male NCAA Football Bowl Series (FBS) athletes, Division-I men's basketball players, and other full-time male students (Eckard, 2010; Southall et al., 2012a, 2012b).

Statistics on athletic graduation rates have evolved as the main measure of athlete success or failure at NCAA Division-I member institutions (Watt & Moore, 2001). Critics argue that while beneficial, a consequence of this development may be that institutions are tempted to cluster athletes in more academically friendly majors, create fake classes, or pressure academic support services into maintaining eligibility rather than encouraging athletes to pursue their own educational goals (Ridpath, 2002, 2008, 2010; Barrett, 2014). Although NCAA reports suggest Black scholarship athletes across all sports tend to graduate at higher rates than other Black students, only 20 of the 50 flagship public universities post a Federal Graduation Rate (FGR) for Black male athletes higher than the overall Black male student population (JBHE, 2005). These discrepancies prompted the *Journal of Blacks in Higher Education* (JBHE) to question if academically selective flagship institutions were fulfilling their

public mandate of educating all students equally (JBHE, 2005). Hawkins (2010) claims that because they are a small percentage of students at Predominantly White Colleges and Universities (PWCUs), Black athletes are simultaneously visibly noticeable and invisible due to a preconceived notion that they are not academically prepared. The hyper-visibility v. invisibility dichotomy results in some Black athletes at PWCUs feeling alienated or racially isolated (Hawkins, 2010). When a Black male athlete begins his career the main focus is often on athletic prowess rather than academic achievement. When Black male college athletes' academic progress is a focal point, it is often treated as an exceptional occurrence. This results in many Black male college athletes being viewed as "minority" affirmative action admits (Hawkins, 2010).

We have two primary aims in this paper: first, we empirically explore the degree to which Black and White male NCAA Division-I football and basketball players (and students more generally) at flagship state universities graduate at different rates. Second, we interpret our findings drawing on three well-known theoretical frameworks: athletic role-engulfment and key-player hypothesis, mismatch education hypothesis, and institutional isomorphism hypothesis. But first, we provide a brief description of the Black male athlete, and then explain the different methods used to measure graduation rates: the Federal Graduation Rate (FGR), the Graduation Success Rate (GSR), and the Adjusted Graduation Rate (AGR).

BLACK MALE ATHLETES AT PREDOMINANTLY WHITE COLLEGES AND UNIVERSITIES

Black male college athletes occupy an important and unique place in the NCAA Division-I collegiate model of athletics (Brand, 2004). Over the past four decades Black men have increasingly come to dominate football and basketball programs at PWCUs (Brand, 2006). The *2009–10 Student-Athlete Race and Ethnicity Report* revealed that for the first time African Americans comprised the highest percentage of NCAA Division-I football players (Zgonc, 2010). The study also reported that 60.9% of NCAA Division-I men's basketball players were Black (Brown, 2011). By contrast, Black males accounted for just 2.8% of full-time, degree-seeking undergraduate college students (Harper, Williams, & Blackman, 2013).

As Harrison and Lawrence (2004) noted, as Black male participation in NCAA football and men's basketball has increased, so has the widely held belief in Black athletic superiority (Hoberman, 1997). College-sport fans' current infatuation with Black athleticism perpetuates the negative stereotype of

Blacks as physically superior but intellectually inferior to Whites. Harrison and Lawrence (2004) contend the manner in which this debate has been framed is biased, political, and limited in analysis. They contend the scientific preoccupation with racially linked genetic differences is racist, since it is founded on and naturalizes racial categories as fixed and unambiguous biological realities, thus obscuring the political processes of racial formation (Harrison & Lawrence, 2004).

The stereotypical belief of Black males' athletic superiority and intellectual inferiority (Harrison & Lawrence, 2004; Hawkins, 1999, 2010) is fueled by the overrepresentation of Black male college athletes in the high-profile revenue sports of football and men's basketball as well as the underrepresentation of Black male students in the general student body at PWCUs. As Harper et al. (2013) noted, "Between 2007 and 2010, Black men were 2.8% of full-time, degree-seeking undergraduate students, but 57.1% of football teams and 64.3% of basketball teams" (p. 1).

Reacting to the existence of these attitudes, the NCAA contends its Academic Progress Program (APP) reform efforts have successfully changed college sport's "dumb jock" culture (Porter, 2011). This cultural change has been highlighted in a public service announcement entitled *Dumb Jocks* and remarks by NCAA president, Mark Emmert. In addition, the NCAA points to its reports that athletes, particularly African American males, are graduating at higher rates than their counterparts in the general student body in almost every category (NCAA Research, 2011).

Since Black college students are more likely to drop out for financial reasons, an athletic grant-in-aid (GIA) may be vitally important to economically disadvantaged Black players (JBHE, 2005). In short, FBS football and NCAA D-I men's basketball players are working their way through school by "playing" football or basketball. These athletes' graduation rates have economic relevance, since over their lifetime Black male college graduates have twice the mean earning capacity of Black high school graduates—an absolute difference of \$1.03 million on average (Sum et al., 2007). Additionally, the average Black male college graduate will pay nearly \$500,000 more in taxes compared to the average Black male high school dropout, who receives nearly \$190,000 more in cash and in-kind government benefits than he will pay in payroll and income taxes over his working life (Sum et al., 2007).

While such data are heartening, Comeaux and Harrison (2011) contend college athletes, especially Division-I revenue-sport athletes, not only face "... all of the challenges [e.g., social and academic adjustments to college] experienced by other students in the general population... [but also face] demands

imposed by their sports, which create considerable challenges to student life” (p. 236). These demands include over 40 hours a week devoted to practices, travel, team meetings, and midweek game schedules. The demands of these athletic “jobs” (Southall & Weiler, 2014) result in mental fatigue, physical exhaustion, and nagging injuries. In addition to having less time to devote to academic pursuits, by choice or heavily influenced by the athletic structure, college athletes also live, eat, study, and socialize together and are even tracked into the same majors (Comeaux & Harrison, 2011; Southall & Weiler, 2014). The resulting isolation poses a challenge to their academic success and—in many ways—affects the quality of their college experience. Harper et al. (2013) highlight the fact that “97.4% of institutions graduated Black male student-athletes at rates lower than undergraduate students overall. On no campus were rates exactly comparable for these two comparison groups” (p. 1).

In light of these data, Howard (2014) contends Black males, including college athletes, often succeed not because of, but in spite of their schools. In 2013, Jean Boyd, president-elect of National Association of Athletic Academic Advisors (N4A), commented on the competing interests at play in big-time college sport: “Big time college athletics is a business and anytime you have games on Tuesday nights, it is not in the best interest of the student-athlete. It’s in the best interest of the institution” (Robinson, 2013, para. 21). Reflecting this struggle, Southall, Eckard, Nagel, and Randall (in press) examined Football Bowl Subdivision (FBS) football and NCAA D-I men’s basketball programs and found a significant relationship between a team’s athletic success and lower graduation rates among Black players.

With conflicting and competing measures of academic success, as well as media and NCAA reports of record graduation rates for college athletes, an understanding of the various graduation-rate metrics is important. Since many readers may have only a cursory knowledge of graduation rates, in the following sections we delineate three graduation rates, and then discuss the study’s methodology and results.

COMPETING MEASURES OF GRADUATION RATES

Federal Graduation Rate

In 1995, the U.S. Department of Education (DOE) operationally defined the Federal Graduation Rate (FGR) as the percentage of full-time, first-time bachelor-degree-seeking students enrolled in any fall semester who had completed their degree requirements within 150% of the normal time span (Sack, Park, & Thiel, 2011; Selingo, 2012). The FGR involved a straightforward calculation based on the question, “How many students who initially enroll as

first-time, full-time first-year students at a given university graduate from that university within six years?" Since students are not removed from the cohort if they later switch to part-time enrollment or transfer, the FGR provides a mechanism to determine the extent to which colleges and universities retain and graduate enrollees who begin as full-time students. The strength of the FGR is its focus on student retention; however, transfer students are treated as non-graduates from their original institutions even if they graduated from another institution at a later date (Southall, 2012). Critics note that one-third of all college students in the US transfer at least once within five years (Selingo, 2012). Despite its limitations, the FGR remains the only easily available college performance-measure applicable to the general student body.

In addition to providing information about the general student body, the FGR is calculated for full-time athletes who receive athletic aid (i.e., a grant, scholarship, tuition waiver, or other assistance awarded on the basis of athletic ability) for any period of their enrollment. Just as other students, athletes who do not graduate from the school in which they initially enroll within six years count as non-graduates against the school's FGR (Hosick, 2010; Selingo, 2012). The FGR is often criticized for not including athletes who transfer and then graduate from other schools (Hosick, 2010; Zhong, 2008). However, it offers the additional benefit of allowing for the calculation of a Federal Graduation Gap (FGG), reflecting the difference (e.g., gap) in graduation rates between cohorts. For example, if the FGR for a university's overall male student body is 65% and the FGR for its Black male students is 49%, the Black male student FGG would be -16, while the overall male student body FGG would be +16.

In 1999 when the first series of disclosure reports were published for the 1995 cohort, many observers, including the Knight Foundation Commission on Inter-collegiate Athletics (KCIA), were troubled by college athletes' low graduation rates, particularly in NCAA D-I football (37.5%) and men's basketball (33%) (KCIA, 1999). With low college football and men's basketball FGRs persisting, as well as ongoing academic scandals occurring at high-profile flagship schools (e.g., Auburn University, University of Michigan, University of North Carolina at Chapel Hill, and University of Tennessee-Knoxville), some critics question the alignment of "big-time" college sport with universities' academic missions (e.g., Maloney & McCormick, 1993; Heydorn, 2009; Nocera, 2012; Harper et al., 2013).

Graduation Success Rate

As part of its 2003 academic reform program, the NCAA developed and promoted an alternative athlete graduation rate metric: the Graduation Success Rate (GSR) (Christianson, 2005). Justifying the GSR, the NCAA national

office noted, "... [university] presidents had long been disappointed with a federal methodology in which so many student-athletes are simply lost in the calculation" (Hosick, 2010, p. 6). Since the inception of the GSR, NCAA officials have consistently contended it "... is a more accurate rate since it credits institutions for incoming transfers who graduate, and it removes from the calculation transfers who leave the institution in good academic standing" (NCAA News Archives, 2010).

While the GSR is an important internal graduation rate calculation for athletic departments, its sample and methodology are different from the FGR, which does not account for transfers into or out of an institution. When the first GSR report was released in 2005, it was not intended to replace the FGR, but was designed to complement it, since general student body transfer or retention is not aggregated and widely reported on a national basis (NCAA, 2005; NCAA Research Staff, 2013). Yet, over time the NCAA national office has increasingly highlighted GSR rates in its yearly graduation report and referred to it as "a more accurate measure of graduation," which indicates "... the federal rate might actually be underestimating the long-term student-level graduation performance" (Hosick, 2010, p. 5, 14). As a result of data being drawn from a different sample and employing a different methodology, the GSR rate is almost always higher than the FGR (Southall, 2012). In addition, while the GSR removes athletes who transfer (or leave a school in good academic standing, but do not enroll in another school) from a university's GSR cohort, it cannot shed light on why athletes left a school: "Did a player leave seeking a professional-sport opportunity, transfer to another school, or simply drop out and go home?" In effect, it treats dropouts as transfers, and so overestimates graduation rates.

Despite the FGR's limitations, LaForge and Hodge (2011) note any comparison (inadvertent or purposeful) of athletic GSRs to overall student-body FGRs is methodologically inappropriate. While such comparisons generally cast an athletic team in a more favorable light, they are invalid since the two samples are drawn from different populations (LaForge & Hodge, 2011). In addition, Gurney and Southall (2012) contend:

By consistently asserting the GSR "more accurately assesses the academic success" of college athletes and steadfastly referring to GSR rates, NCAA members have convinced the media to almost exclusively use the new, more-favorable metric. Intentionally or not, the NCAA's Academic Progress Rate (APR) and GSR metrics confuse the media, fans and the general public. (p. 17)

These scholars note that while using the GSR to highlight graduation “success” may be a savvy marketing and public relations tool, it has “... increasingly fostered acts of academic dishonesty and devalued higher education in a frantic search for eligibility and retention points” (Gurney & Southall, 2012, p. 17). Although the NCAA emphasizes the GSR metric, there is no corresponding GSR for non-athletes. Therefore GSRs cannot be used for comparisons with general student body graduation rates, and so the GSR was excluded from our study.

Adjusted Graduation Rate

Heydorn (2009) argues a graduation gap is an appropriate indicator when comparing college athletes to other undergraduates. Rische (2004) further contends a graduation gap comparison makes sense because it is a more standardized measure of relative success. The Adjusted Graduation Rate (AGR) model compensates for any potential downward bias in the general student body FGR through regression-based adjustments for the percentage of part-timers in a school’s student body. Part-time students take longer to graduate and therefore pull down general student body FGRs, which include part-timers who switch from full-time after their initial enrollment. In this study we utilize the Adjusted Graduation Gap (AGG) to explore differences in athlete graduation rates. The AGG calculates the difference (e.g., gap) between the AGR for full-time male students (both Black and White) and the FGR of both full-time Black and White football and men’s basketball players.

RESEARCH SETTING

Before outlining the methodology and reporting the results, several items are noteworthy and delineate this study’s research setting:

1. Neither the Federal Graduation Rate (FGR), mandated by Congress, nor the NCAA’s GSR is perfect or inherently a more accurate metric; they utilize different sampling and statistical analyses to examine different cohorts. In short, they are different graduation rates.
2. The GSR consistently returns a “success” rate 12–25% higher than the FGR. As far back as 1991 (NCAA, 1991), the NCAA knew that by removing 1/4 to 1/3 of what it referred to as “eligible dropouts” from the sample would result in a markedly higher “success” rate.
3. A comparison of published FGRs of NCAA athletes and the general student population includes a significant number of part-time students at many schools. This is problematic because NCAA athletes must be “full-time.” Consequently, it makes sense to compare full-time college athletes with other full-time students. Without adjusting for the possible downward “part-timer bias” in the student-body rate, any comparison may be

distorted—or somewhat skewed. Because part-time students take longer to graduate, reported general student-body FGRs may be significantly reduced, making the relative rate of college athletes at many schools and conferences appear more favorable.

4. Finally, since there is no comparable national-level GSR for the general student body, GSR and FGR data should NOT be reported simultaneously. To do so in press releases or dataset tables invites inappropriate comparisons and fosters confusion. (Southall, 2014a, pp. 5–6)

METHODS

Description of Samples

Our study focuses on Division-I state flagship universities to explore the degree to which graduation rates vary between football and male basketball athletes, and male undergraduates. In determining universities to include in the study's sample, the first criterion was based on institutions possessing at least one of Berdahl's (1998) three attributes of flagship universities:

[1] These institutions formed the core of the public systems of higher education in their respective states.... [2] In most cases, these institutions were the first public universities to be established in their states.... [3] They became the centers for research and graduate education and they developed an array of professional schools that added to their size, scope, and preeminence. (pp. 5–6)

The next inclusion requirement was the university must be listed in the annual *USA Today College and Tuition and Fees Survey of 75 Public Flagship Universities*. Finally, this group was delimited to universities ($N = 60$) that were members of the NCAA D-I Football Bowl Subdivision (FBS). Table 1 lists all flagship universities in our sample.

The sample's graduation rate data were obtained from the 2012–13 NCAA FGR database (NCAA, 2015) and 2012 College Sport Research Institute (CSRI) AGG Reports for FBS football and men's basketball (Southall et al., 2012a, 2012b, 2013). Eckard's (2010) regression modeling was utilized to estimate full-time male adjusted graduation rates (AGRs). Since all NCAA athletes must be full-time students, it was not necessary to adjust reported FGR data for football or men's basketball players (Eckard, 2010). Each flagship university's Black and White student enrollment data (e.g., percent part-time and ethnic breakdowns) were obtained from the National Center for Educational Statistics and *Integrated Postsecondary Education Data System*, National Center for Educational Statistics.

Table 1. NCAA Division-I FBS Flagship Universities (N = 60)

University	University
Arizona State University	University of Hawaii, Manoa
Auburn University	University of Idaho
Clemson University	University of Illinois, Champaign
Colorado State University	University of Iowa
Florida State University	University of Kansas
Indiana University	University of Kentucky
Iowa State University	University of Maryland, College Park
Kansas State University	University of Michigan
Louisiana State University	University of Minnesota, Twin Cities
Michigan State University	University of Mississippi
Mississippi State University	University of Missouri, Columbia
New Mexico State University	University of Nebraska, Lincoln
North Carolina State University	University of Nevada, Las Vegas
Ohio University	University of Nevada, Reno
Oklahoma State University	University of New Mexico
Pennsylvania State University	University of North Carolina at Chapel Hill
Purdue University	University of Oklahoma
Rutgers, St. University of New Jersey	University of Oregon
Texas A&M University, College Station	University of South Carolina, Columbia
The Ohio State University	University of Tennessee, Knoxville
U. at Buffalo, The St. U. of New York	University of Texas, Austin
University of Alabama	University of Utah
University of Arizona	University of Virginia
University of Arkansas, Fayetteville	University of Washington
University of California, Berkeley	University of Wisconsin, Madison
University of California, Los Angeles	University of Wyoming
University of Colorado, Boulder	Utah State University
University of Connecticut	Virginia Polytechnic Institute & State U.
University of Florida	Washington State University
University of Georgia	West Virginia University

Statistical Analyses

Reported graduation gaps (i.e., FGG and AGG) were obtained by comparing various FGR and AGR cohorts. The resulting gaps are expressed as either a negative or positive value, negative (-) if the second listed integer is less than and positive (+) if it is greater than the first. In order to determine if there were significant differences in various cohort mean graduation rates, paired-sample T-tests were performed.

RESULTS

Frequencies and Descriptive Statistics

While all flagship universities in the sample have Football (FB) FGRs, eight men's basketball programs had reported FGRs from only one ethnic group.¹ Seven schools have no White FGR and one has no Black FGR. As a result, the sample for Black basketball player (BB) FGRs and AGGs was $n = 59$, and the White Basketball (BB) FGR and AGG sample was $n = 53$. Initial frequencies and descriptive statistics (summarized in Table 2) reveal a range of cohort graduation rates. Within all cohorts White males had higher graduation-rate means than Black males. In addition, consistent with previous research (Southall et al., 2012a, 2012b, 2013), within each ethnic group full-time male students have higher graduation rates than football and men's basketball players. Athlete FGR means ranged from 36.4% for Black men's basketball players to 65.3% for White football players.

Table 2. Flagship Universities' Graduation Rates Descriptive Statistics

	Black	White	Black	White	Black	White	Black	White	Black	White
	AGR	AGR	BB FGR	BB FGR	FB FGR	FB FGR	BB AGG	BB AGG	FB AGG	FB AGG
<i>N</i> =	60	60	59	53	60	60	59	53	60	60
<i>Mean</i> =	56.6	76.4	36.4	59.5	46.0	65.3	-20.2	-16.9	-10.6	-11.1
<i>Median</i> =	56.4	77.2	38.0	67.0	46.0	67.5	-22.1	-16.0	-13.0	-12.9
<i>StDev</i> =	9.9	10.0	22.3	32.8	10.7	12.8	24.7	30.7	12.9	14.1
<i>Min</i> =	37.0	54.3	0.0	0.0	25.0	33.0	-63.6	-79.0	-42.7	-48.0
<i>Max</i> =	80.2	94.7	100.0	100.0	82.0	93.0	51.4	35.3	17.5	25.0

Graduation Gap Summaries

Utilizing the mean AGRs and FGRs described in Table 2, we examine graduation gaps within and between various cohorts. As a result of different sample sizes, presenting the basketball and football AGG comparisons in one table is problematic. Therefore Table 3 summarizes comparisons for basketball, while Table 4 presents football AGGs.

Consistent with overall-sample results, within-ethnic-group comparisons of flagship university men's basketball programs ($n = 52$) reveal double-digit positive graduation gaps for White males (see Table 3, "Within Cohort Gap" column). Among full-time males, White students have a significant AGG of +19.8 ($t = 15.0$) relative to Black students. All 52-flagship schools have White male student body AGRs that exceed those of Black males. Comparing men's basketball FGRs, White players had a +23.8 FGG ($t = 4.74$) relative to Black players. For 41 sample schools (79%), White basketball players' FGRs exceed those of Black players. The within-group comparisons show significant negative athlete graduation gaps. The Black athlete-student AGG is -22.3 and the White athlete-student AGG is -18.3. Interestingly, when the FGRs of Black and White men's basketball players are compared to the AGRs of full-time male students of the same ethnicity, the resulting difference in AGGs is insignificant. This difference is -4.0 (-22.3 vs. -18.3), with a t-stat of 0.85.

The last column of Table 3 shows cross-group comparisons. The Black basketball FGR compared to the White student body AGR yields an AGG of -42.1. In effect, the negative within-Black AGR/BB FGR gap (-22.3) is

Table 3. Flagship Graduation Gaps Summary Table: Basketball (N=52)

Cohort	Mean	Within Cohort Gap	Athlete - Student AGG [^]	Student v. Athlete B v. WAGG
1. Black Male AGR	57.2	+19.8 (2-1)	—	—
2. White Male AGR	77.0	($t = 15.0$)*	—	—
3. Black Men's BB FGR	34.9		-22.3 (3-1) +23.8 (4-3)	-42.1 (3-2) ($t = 12.2$)*
4. White Men's BB FGR	58.7	($t = 4.74$)*	-18.3 (4-2) ($t = 4.33$)*	+1.5 (4-1) ($t = 0.35$)

* p-value < 0.0001; paired difference-between-means test; null hypothesis: difference = 0.

Note. ^Athlete - Student (Same ethnicity).

Table 4. Flagship Graduation Gaps Summary Table: Football (N=60)

Cohort	Mean	Within Cohort Gap	Athlete - Student AGG [^]	Student v. Athlete B v. WAGG
Black Male AGR	56.6	+19.8 (2-1)	—	—
White Male AGR	76.4	(t = 15.2)*	—	—
Black FB FGR	46.0		-10.6 (3-1) (t = 6.37)*	-30.4 (3-2) (t = 17.5)*
White FB FGR	65.3	+19.3 (4-3) (t = 8.5)*	-11.1 (4-2) (t = 6.09)*	+8.7 (4-1) (t = 4.99)*

* p-value < 0.0001; null hypothesis: difference = 0.

Note. ^Athlete - Student (Same ethnicity).

compounded by the negative Black-White AGR gap (-19.8). In contrast, the White BB FGR compared to the Black AGR yields an AGG of +1.5. In other words, White basketball players on average graduate at a rate slightly higher than the Black male student body, although the difference is not statistically significant ($t = 0.35$). Here the negative within-White AGR-BB FGR gap (-18.3) is offset by the positive White-Black AGR gap (+19.8).

Table 4 summarizes the graduation-gap analysis for 60 FBS football flagship universities. A within-cohort examination disclosed similar significant positive graduation gaps (i.e., AGGs and FGGs) for White males: the White male AGG equals +19.8 ($t = 15.2$) and the White FB FGG is +19.3 ($t = 8.5$). As with men's basketball, all 60 FBS flagship schools have White AGRs that exceed Black AGRs. For 53 of the 60 schools (88%), the White football players' FGRs exceed Black football FGRs. In addition, Black football players had a -10.6 AGG ($t = 6.37$) when compared to Black full-time male students, while the White FB AGG was -11.1 ($t = 6.09$). Consistent with the men's basketball sample, the difference between Black and White FB AGGs was almost zero (0.5), with an insignificant t-stat (0.20).

The last column of Table 4 shows cross-group comparisons. The Black FB FGR compared to the White AGR yields an AGG of -30.4. In effect, the negative within-Black AGR-FB FGR gap (-10.6) is compounded by the negative Black-White AGR gap (-19.8). In contrast, the White FB FGR compared to the Black AGR yields an AGG of +8.7 ($t = 4.99$). In other words, White football players on average graduate at a rate higher than the Black male student body by a statistically significant amount. Here the negative within-White AGR-FB

FGR gap (-11.1) is more than offset by the positive White-Black AGR gap (+19.8).

DISCUSSION

Graduation Gap Explanations

Our results show substantial gaps between the graduation rates of revenue-sport athletes, both football and basketball, and their ethnic peers in the general student body at state flagship universities. In light of these results and given the findings of related studies (Harper, Williams, & Blackman, 2013; JBHE, 2005; NCAA Research Staff, 2011; Eckard, 2010; Southall et al., 2012a, 2012b, 2013; TIDES, 2012), we explore the utility of three theoretical constructs: one emphasizing the individual level (role-engulfment), the structural level (mismatch education hypothesis), and the organizational level (institutional isomorphism).

Role Engulfment and the Key-Player Hypothesis

Our findings support Shulman and Bowen's (2001) research, which revealed that not only do collegiate athletes under-perform academically, these deficiencies were more pronounced in men's basketball, football, and hockey. Shulman and Bowen (2001) along with other scholars (Aries et. al., 2004; Cantor & Prentice, 1996; Simons et al., 1999) argue that the time demands faced by these athletes force them to make academic sacrifices. Susceptibility to *athletic role engulfment* and a decreased ability to balance athletic and academic burdens may occur when professional career aspirations and motivations are closely linked (Adler & Adler, 1989, 1991; Simons, Van Rheenen, & Covington, 1999; NCAA Research, 2011).

A related factor that may impact the academic performance of college athletes is the "key-player" hypothesis, which posits that key players (e.g., starters and stars) face disproportionate time demands, logging more playing time over the course of their college career (Bowen & Levin, 2003). Today's profit-sport environment, in which a large majority of state flagship Division-I football and men's basketball starters (key players) are Black athletes, many of whom were granted "special-admit" status, poses unique challenges at state flagship institutions.

Mismatch Theory and Academic Underpreparedness

In 2009, Dr. Gerald Gurney, president-elect of the National Association of Academic Advisors for Athletics (N4A), took issue with the NCAA's deemphasis of minimum initial eligibility requirements, "[T]oo many athletes are

overmatched in the classroom and cheating and scandal are inevitable" (Wieberg, 2009, p. 1). Despite NCAA graduation success proclamations, Gurney's assertion offers a plausible explanation for the graduation gaps identified in our results, based in part on a mismatched-student hypothesis or *mismatch theory*. This theory claims students whose college admission is dependent on preferential (i.e., affirmative action) policies have less academic success than students who are prepared to flourish in an educationally rigorous environment. Critics of such "special-admission" programs point to the poor academic performance of minorities, athletes, and legacy students at highly selective universities as evidence of an "educational mismatch" (Sanders & Taylor, 2012; Perry, 2012).

In 2013, more than half of NCAA Division-I Faculty Athletic Representatives (FARs) reported their institutions admit players who do not meet standard admissions requirements (Wolverton, 2013). Admissions data submitted by NCAA Division-I athletic programs revealed relaxed special-admission standards; with athletes from 27 identified universities being ten times more likely to benefit from such programs (Scherzagier, 2009). And, according to Comeaux and Harrison (2007) and Sellers (1992), Black college athletes tend to enter college with lower academic credentials.

Although athletes may be admitted with lower academic credentials (Hood et al., 1992; Shulman & Bowen, 2001; Bowen & Levin, 2003), Massey and Mooney's (2007) model remains one of the few studies to test the explanatory powers of mismatch theory for college athletes at competitive universities. Constructed from a sample of nearly 4,000 students, including 294 first-year varsity or junior varsity athletes at 28 elite American colleges and universities, Massey and Mooney found minorities and athletes who received an SAT admissions bonus did not earn significantly lower grades through the end of their sophomore year. Likewise, research by Alon and Tienda (2005) suggest affirmative action programs do not set up minorities (in general) or athletes (in particular) for academic failure in competitive academic environments. However, Massey and Mooney did acknowledge that legacies and athletes who attend a school that practices institutional affirmative action are more likely to leave at higher rates.

Institutional Isomorphism

Rather than conceiving of the NCAA as an independent regulatory agency and Division-I flagship institutions as competing firms, viewing these entities as a single organizational field allows us to explore Black male athlete graduation gaps as a collective action between relevant actors. According to DiMaggio and Powell (1983), once disparate organizations in the same line of

business are structured into an actual field, either by the state, competition, or profession, powerful forces emerge that lead them to become more similar to one another (p. 148). Institutional isomorphism theory posits that organizations constantly try to change, but after a certain degree of maturity occurs in an organizational field, the aggregate effect of such changes is to lessen the extent of diversity within the field (p. 149). It is therefore possible that after decades of interaction regarding the governance of college sports, the NCAA and Division-I flagship institutions have matured to the point that institutional isomorphism now guides the organizational field.

Division-I schools in revenue sports confront common pressures to field winning teams that in turn create tensions between the role of “student” and the role of “athlete,” as described above. Over time, formal and informal rules and procedures have evolved in the organizational field regarding recruiting and academic support for student-athletes that, in fact, have emphasized athletics over academics. Institutional isomorphism suggests that such changes in rules and procedures would tend to reduce the diversity of academic outcomes across schools. Our results are consistent with this, indicating, for example, that in 80% of flagship institutions (4 of 5) both Black and White football players graduate at rates lower than their ethnic peers, and in 88% of institutions (about 8 of 9) Black players have lower FGRs than White ones.

CONCLUSIONS AND IMPLICATIONS

Making sense of athlete and student graduation rates is challenging. This task is initially based on the difficulty of establishing a standard, comparable graduation rate for both cohorts. The American Council on Education acknowledges that while a graduation rate can be a simple matter of developing a standard measure that is easy to calculate, interpreting graduation rates is far more complex and analytically challenging (Cook & Hartle, 2011). In fact, it took the U.S. Department of Education (DOE) five years after Congress enacted the *Student Right-to-Know and Campus Security Act of 1990* (SRTK) to officially operationalize the Federal Graduation Rate (FGR) in 1995.

In response to public outcry over persistently low graduation rates and ongoing academic scandals at high-profile state flagship schools, the NCAA began instituting a series of academic reforms in 2003. Though scholars have examined the merits and impact of these reforms on graduation rates, less attention has focused on gaps in graduation rates that compare college athletes to other undergraduates. Even fewer studies have focused on examining the graduation gaps based on racial differences and types of sport participation.

While the NCAA, in an effort to maintain the perception of a clear line of demarcation between its collegiate model and professional sport, has consistently reported record GSR's and sought to position these reports as the "best" or most accurate graduation rate and utilize GSR and APR scores as evidence that big-time college sport has one clear focus—education—our study offers support for an alternative conclusion. While GSR data may be aggregated to present a more palatable image of the collegiate model, disparities in graduation rates between profit-athletes and the general student body, as well as large-scale clustering of such athletes, are examples of systemic impediments to profit-athletes' equal-educational access.

While the data in this paper are admittedly limited to a select group of universities, these higher-education institutions are—without question—among the most important in the country in terms of size, the role they play in their respective states, and the hugely out-sized role they play in "major" college athletics. Our results both support and challenge the findings of other studies. We especially challenge the NCAA's claims of comparable athlete-student body graduation rates, something that can appear to be the case when considering all student-athletes, but arguable when focusing on racial differences, especially (as in our study) for Black males in major revenue sports.

In sharp contrast to the NCAA's claims of comparable graduation rates based on all sports, our study's results clearly demonstrate negative graduation gaps exist between Black male basketball and football players and full-time male students. This finding supports the effect of restrictions faced by this population of athletes, which are the result of their unique educational and athletic work experiences. Because these athletes are not "regular" students, our major empirical and theoretical conclusion is that a more nuanced theoretical perspective that accounts for individual, structural, and organizational level influences is necessary for any future research.

It is also important to situate our results within a college-sport industry embroiled in ongoing legal and societal challenges. In response to these challenges to its Collegiate Model of Athletics, the NCAA national office has sought to disseminate a rebranded definition of academic success. As Myles Brand (late NCAA President) proclaimed in 2006, "the business of college sports is not a necessary evil, [but] a proper part of the overall enterprise" (p. 8). Consequently, the NCAA and its members have sought to rebrand academic success and blunt criticisms of big-time college sport by pointing to record GSRs as evidence college athletes are provided an opportunity for a world-class education (Southall, 2014b). While such systematic and sustained rebranding is not inherently unethical, its use has successfully obscured the

college sport industry's institutional hegemony and allowed the industry to portray itself as an educational enterprise (Gramsci, 1971; Southall & Staurowsky, 2013). For the past quarter-century the NCAA has been remarkably disciplined and consistently stayed on message. Similar to its use of the term "student-athlete," the association has used its GSR as a rebranded definition of academic success to camouflage its profit-seeking tendencies.

While it is beyond the scope of this project to determine if the time demands or career aspirations linked to role engulfment are responsible for academic graduation gaps, future research would benefit from Division-I state flagship universities becoming more transparent regarding their admission policies. Access to special-admission standards, ACT and SAT scores, and high school grades (in aggregate and deidentified) of FBS football and NCAA D-I men's basketball players would allow scholars to gain greater insights on pressing social issues such as academic preparedness, exploitation, and the stigmatization of the Black male revenue-sport athletes as academically inferior. Although these data are not currently accessible to the general public, the NCAA Eligibility Center and/or Division-I member institutions likely collect such information (NCAA, 2015).

To gain a more nuanced understanding of the NCAA and Division-I flagship institutions' response to graduation rate gaps, we recommend conducting ethnographic investigations that utilize institutional isomorphism as the theoretical starting point. On a practical level, athletes, administrators, and the public will benefit from institutional investigations that lead to effective policy change focused on raising athlete graduation rates (regardless of the metric utilized) and reducing graduation gaps. In terms of the scientific community, an empirical inquiry that employs institutional ethnography can assist in combining theory and method by emphasizing the connections among sites, situations of everyday life, professional practice, and policy making.

NOTES

1. The NCAA does not report an ethnic group's FGR if there are two or fewer students in that group for a particular sport.

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A preliminary study of youth-sport concussions: parents' health literacy and knowledge of return-to-play protocol criteria

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Abstract

Primary objective: Preliminarily explore parents' health literacy and knowledge of youth sport league rules involving concussion education and training, and return-to-play protocols.

Research design and methods: This study was guided by the Knowledge, Attitude, and Practice (KAP) model of health knowledge to examine parents' concussion literacy, and understanding of concussion education and training, and return-to-play protocols in youth sports. The mixed-method design involved 119 participants; that included in-person (n=8) and telephone (n=4) interviews, and web-based surveys administered through Mechanical-Turk via Qualtrics (n=98).

Main outcomes and results: Most respondents were not familiar with concussion protocols, but trust coaches' knowledge in return-to-play rules. More than half of the respondents report that the return-to-play concussion criteria have not been clearly explained to them. The majority of respondents were not familiar with the CDC's "Heads Up" online concussion training program, nor were they familiar with any other educational/training tool. About one fifth of the parents had conversations with a coach or medical staff about youth-sport concussions.

Conclusion: Parents have a general understanding of how to identify concussion symptoms, but lack knowledge of immediate steps to take following an incident other than seeking medical help.

Keywords

Youth Sports; Parents; Education; Concussion literacy; Return-to-play; Health-Literacy; Concussion Education

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1. Introduction

In recent years the topic of sport concussion among children and young adolescent athletes has increasingly captured the interest of the public and researchers. Despite an upswing in the number of reported concussions over the past decade, a lack of data exists concerning the overall incidence of sports-related concussions among youth [1]. Beyond the concerns for health consequences, understandings of social behavior around youth concussions are lagging. We know little about the social determinants of concussions and surrounding behaviors, including the effects of increasing parental literacy and adherence to return-to-play guidelines [1–3]. Upon convening an expert committee to review the science of sports-related concussions in youth, the Institute of Medicine (IOM) and the National Research Council recommends efforts to increase knowledge about the culture (e.g. social norms, attitudes, and behavior) surrounding youth sport concussions [1]. A sociological and behavioral investigation will be particularly useful for identifying practical applications and policy relevant solutions that enhance the health and well-being of youth with concussion.

Between 2009 and 2015, fifty states and the District of Columbia enacted legislation to address youth sports-related concussions. The vast majority of these laws contained a provision for concussion education or training, removing youth a youth athlete from play or practice in an even of a suspected concussion, require clearance by a designated health care provider prior to return-to-play [4]. Because it is difficult to determine the impact of this legislation without adequate measures of parental concussion knowledge and return-to-play protocols, policy makers and various youth sport stakeholders will benefit from an examination of parental concussion health literacy and knowledge of youth sport return-to-play criteria [1,5].

Collecting accurate data on mild traumatic brain injury (mTBI) – often referred to as a sport concussion – is hampered by the culture of sports, which may negatively influence athletes from self-reporting concussions and the adherence to return-to-play guidelines. Although research is providing medical professionals with a better understanding of concussions, young athletes are often guided by the notion of toughness as a key attribute of sports participation, so physicians are frequently not consulted after such injuries [6]. Young athletes, and in some cases parents and coaches, may not fully appreciate the short and long-term sequelae of concussions [7]. Previous research found 91% of students understood that they would be at risk of serious injury if they returned to play too soon, but only 50% of high school football players would “always” or “occasionally” tell their coaches about their concussion symptoms [8–10]. Because of their status as young athletes, sport participation may have important health and behavioral implications, particularly with regard to concussions [6].

Although research on the etiology of concussions is advancing rapidly, work on the social determinants and consequences of concussions have moved more slowly. Despite public health concerns associated with youth sport concussion, it remains difficult to draw strong conclusions about best practices. This challenge suggests the need for field research that examines issues around literacy, culture, attitudes, and behavior involving concussions in youth sports. Such research with parents, coaches, team administrators, and other

stakeholders charged with making youth sport return-to-play concussion decisions would be particularly valuable [11–13].

Limited studies exist that evaluate parental knowledge of youth sport concussions [1–3,14–16]. Yet, research focused on this topic points to differences in concussion literacy among parents of youth athletes. For example, 83% of parents of young rugby athletes in New Zealand were able to provide a list of well-accepted concussion signs and symptoms. In contrast, U.S. data suggests that important misconceptions surrounding concussions continue to persist among parents of youth athletes. Building on the work of Mannings et al., [2014][2] the present study was designed to collect preliminary data on parent's awareness of concussion education websites (e.g. CDC "Heads Up"), knowledge of youth sport league rules involving concussion education and training, and return-to-play protocols.

2. Methods

This study was conducted using a triangulation mixed methods design based on Creswell's typology (chap 17) to test the Knowledge, Attitude, Practice model (KAP) of health knowledge, which suggests that increasing a person's health literacy will prompt a behavior change [17,18]. Qualitative and quantitative data were collected in parallel, analyzed separately then merged. The development and validation (e.g. face, validity, content, criterion) of our data collection instruments were guided by scientific methods used in athletic training survey research [19]. Our domain was identified by conducting a search of Google Scholar, PubMed, Medline, and Web of Science databases for relevant publications on concussion health education and literacy between 2013–2014. Previous concussion and sports injury-related questionnaires were collected and included as items in our data collection efforts to assess participants' concussion literacy, knowledge of league rules involving concussion education, training, and return-to-play procedures for youth sports. Our "proof of concept" study was conducted using multiple sources of data including: 1) in-person (n=8) and telephone (n=4) interviews, and 2) online surveys (n=98). For the purposes of this study youth athletes were defined as children from the age of 5–14 years. Informed verbal or written consent was obtained from all participants prior to participation.

Participants

Inclusion criteria for all participants included: parents and guardians with at least one child between the age 5–14 playing youth tackle football or participating in other organized youth sports (e.g. recreational, in-school, intramural, club, team, and individual), and living in the United States. Each interview lasted approximately 20 minutes. Data from the in-person interviews were collected from 6 parents and 2 parent-volunteer coaches during a youth football game in Washington D.C in fall 2014. An additional cohort of 4 parents living in Anne Arundel County, Maryland (three of whom also served as volunteer youth coaches) completed the survey interview by phone, for a total of 12 in-person and phone survey participants.

Additionally, we created and administered a 20-minute anonymous survey instrument through Mechanical Turk – a marketplace for work that requires human intelligence – via Qualtrics. Survey and interview instruments contained questions about participants'

concussion literacy and knowledge of league rules involving concussion education, training, and return-to-play procedures. A recruitment letter was placed in the MTurk online marketplace to solicit and prescreen adult participants. At the completion of each questionnaire a unique confirmation number was assigned and data was stored securely on a password-protected server.

Data Collection

All participants were asked questions that fell into three categories: demographic (age, gender, race, education, marital status, household composition, income), child-specific, and injury-specific. Child-specific questions included type of sport their child participates in, team vs. individual, child's age, school or league sport, pay for play status, volunteer coach participation, duration of participation, and frequency of practices. Under this category, we asked about sport-related injuries and frequency. We also asked about parents' knowledge of concussion symptoms, sport-league rules, and return-to-play protocols. A snowball sampling technique was used to recruit face-to-face and telephone participants, and a non-probability sampling method was employed to collect data for the web-based survey [20,21].

In the tradition of Gorden [1992][22], the data collected from interviews and web-based surveys were coded and placed into related groupings. We assigned category symbols then classified relevant information, which consisted of underlying pertinent words and phrases to indicate perceptual similarities and differences in concussion literacy and return-to-play guidelines. The coding interview response process permitted for analysis of data that identified patterns and ideas that helped explain those patterns [23].

Statistical analysis

Data were presented using descriptive statistics including means, standard deviations, medians, ranges, and proportions. Dependent variables were captured through a series of health literacy, concussion, and return-to-play protocol related questions (e.g., how familiar do you feel you are with the rules/procedures that determine when a child can return-to-play following an injury, how well do you think coaches know the rules/procedures that determine when a child can return-to-play following an injury, do you know what concussions signs and symptoms to look for). We determined the mean value for questions that used Likert scales; and nominal level data were coded 1=yes, 2=no, 3=not sure. Multi-variable regression analysis was performed to assess significant predictors associated with participant familiarity with the CDC "Heads Up" website [24]. Data was analyzed using STATA, version 12.1.

3. Results

Survey demographic data

Data were collected from a total of 110 adults. The average age of the participants is 34 (19–59), with women accounting for 53% of the respondents. The racial distribution of the group is 82% Non-Hispanic White, 4% Non-Hispanic Black, 2% Hispanic, and 5% Asian. All respondents reported graduating from high school with 40% attaining a bachelor's degree. Sixty-four percent of the participants report being married, 10% indicated being single, 13%

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currently live with partners, and 12% (n= 13) reside in a single-parent household. Household income distribution ranged from 11.3% with \$25K to 7.2% with \$100K (**Table 1**). According to the Current Population Survey, the median household income in 2014 was \$53,657^[25]. The 50th percentile in our study falls in the category of \$45-\$55k. Therefore, the median household income in our study is comparable to the general population, with a slightly higher representation in lower and middle class households.

Youth sport involvement

More than 90% of respondents' reported that their children are engaged in team sports. Parents indicated the top six organized or pick-up sports that their children participate in included soccer (57%), baseball (46%), basketball (27%), gymnastics (23%), swimming (19%), and football (16%). Among children registered for organized youth programs at present, 39% play soccer, 31% play baseball, 17% play basketball, 14% participate in gymnastics, 11% swim, and 10% play football (**Table 2**). The mean age of children – represented in our study – that participated in organized youth programs was 8.6 (ages 5–14), and 53% were males. The average duration of participation was two and a half years (1–8 years, sd: 1.56). On average, youth had 2.8 practices (0–8 practices, sd: 1.45) and played 1.5 games (0–10 games, sd: 1.39) per week. These data show that majority of youth athletes played school-based (60%) and/or recreational league sports (67%). About 90% of the respondents reported paying fees for their child's participation in sports.

Coaching experience

A total of 16 (14.5%) respondents reported serving as a volunteer coach for at least one season of organized youth sports (8 in soccer, 7 in baseball, and 3 in basketball, hockey, and cheerleading). The majority of volunteer parent coaches worked with athletes between the ages of 5 and 10 years old.

Youth sport injury

Overall, 21% (n=23) of the respondents reported children being injured while playing organized sports. Concussions (n=2), bruises and scratches (n=4), fractures (n=6), and joint injuries (n=12), were among the most common injuries sustained by children playing organized youth sports. The most common youth sports in which injuries occur included soccer (n=15), baseball (n=5), football (n=4), and basketball (n=2). Most parents were present when injuries occurred (45%) and went to the school's medical staff, personal doctors, or hospital with their children.

Most respondents were not familiar with concussion protocols (3.09 on a 7-point Likert scale), but trusted coaches' knowledge of return-to-play rules (5.28). (Figure 1.) More than half of the respondents (55%) reported that the return-to-play rules and procedures involving concussions had not been clearly explained to them.

The majority of respondents (73%) have never heard of CDC's website *Heads Up to Youth Sports: Online Training for parents* [24]. Parents knew little about the Heads Up Football Coaches Training Program (2.48 on a 7-point Likert scale), and only 6% were aware of the American Youth Football & Cheerleading Association rule that required coaches to

successfully complete the CDC Heads Up online concussion training program annually. In the multi-variable regression analysis, only respondents' level of education is positively associated with familiarity with the CDC "Heads Up" website (**Table 3.**). When probed to offer specific details about their knowledge of concussions, 13 respondents (14%) reported they knew nothing about head concussions. Of the remaining 84 participants that answered this question, 51% were aware of life-threatening or life-long effects; 38% specified concussions are diagnosable; 33% stated medical treatment is required; 25% discussed physical signs and symptoms; 17% indicated cognitive signs and symptoms; and 10% stated history or rising public attention on NFL and youth sports concussion rules and prevention. Nearly 1 out of 5 parents have had a conversation with a coach or medical staff about concussions. Parents report they have knowledge of concussion symptoms (4.45 on a 7-point scale). In the response to the open-ended question, "Can you name some of the concussion signs and symptoms to look for?" the top six answers were as follows: dizziness (60%); headache (47%); confusion (43%); nausea (22%); blurred vision (21%), and vomiting (20%). When asked, "Can you describe what steps should be followed upon learning that a child has experienced a sports concussion?", the top responses included: go to the ER/hospital right away (74%); sit them down/take rest (23%); keep them awake (16%); and, assess severity before going to hospital (12%). Women reported knowing more about concussion signs and symptoms than men (**Table 4.**). Respondents were generally concerned with youth sport concussions (4.66 on a 7-point scale). Controlling for gender, age, race, education, and marital status, participants who reported with higher household income intervals showed greater concerns for youth sport concussions than those with lower income levels.

Qualitative data

Three overall themes emerged from the qualitative data: (a) parents had limited specific knowledge about head concussions, (b) parents primarily rely on coaches to identify concussion symptoms and to communicate this information to them, (c) parents that serve as volunteer coaches report receiving concussion education and are knowledgeable about return-to-play protocols.

Parental knowledge of sport concussions

Qualitative interviews revealed that parents have limited knowledge about head concussions in youth sports. This quote by a 35-year-old married mom of an 8-year-old male football player from Anne Arundel County Maryland is indicative of the typical response.

Well, as soon as you start asking about that, I'm like gosh, I really don't know that much, but I know it's a huge concern with football in particular. I've heard, only anecdotally, about other parents and their sons who have had concussions and what that entails, sort of having them not go to school, not watch TV, how to be aware of bright light conditions, and sustain that for a matter of days until they're given the go-ahead to resume. But I've never experienced any of that first hand, it's all been word of mouth and anecdotal, and because it hasn't touched me yet. I haven't really ever done much research or Googling or anything about it.

While acknowledging concerns about youth sports concussions, none of the parents in our study had consulted with medical staff or health care professionals about identifying concussion symptoms or treating an athlete with concussion. Likewise, parents disclosed that they did not consult the CDC Heads Up website, or any other webpage for youth sport concussion education materials.

Reliance on coaches to identify sports concussions

A second theme present in the qualitative data is parent's reliance on coaches to identify concussion symptoms and their dependence on emergency room treatment. Although most parents are unaware if coaches receive formal concussion training, they are confident in their coaches' ability to instruct them once an injury occurs. Seven of the twelve parents we interviewed said they were not aware if the coaches or athletic trainers on their child's team received concussion training. A mother in Anne Arundel County offered the following response to a question about proof of team staff receiving youth sport concussion training. "I'm kind of embarrassed to admit it honestly, but I've been relying on our football coaches too much. I just figure they know about concussions." Likewise, eight parents stated that, to their knowledge, no member of their child's coaching or medical staff explained rules involving the return-to-play concussion protocol.

Coaches' concussion & return-to-play education

Face-to-face interview and telephone survey data also indicates that volunteer parent coaches are knowledgeable of youth concussions and return-to-play procedures. Volunteer parent coaches in both Washington DC and Anne Arundel County confirmed that concussion education and return-to-play training are mandatory league requirements. The commissioner of the Washington DC youth football team stated that it is compulsory for coaches in their league to complete the CDC Heads Up online concussion training program. His response is consistent with the rules posted on the American Youth Football & Cheer website [²⁶], 'for the 2014 season, all coaches are required to complete the online CDC Heads up Online Concussion Training.' To ensure that coaches are properly trained and educated about youth sport concussions, the DC commissioner said teams are required to retain a copy of each coaches' certificate of competition each season.

Similarly, a father in Anne Arundel County explained that parents are required to know the return-to-play concussion protocol before they can volunteer as youth sport coaches. "In my league it's been very clearly explained to the coaches that hey, if a kid want to come back after a head injury, they have to have a doctor's note, or even if it's a broken bone. I require a doctor's note because, you know, some parents won't actually do it." According to this father, no such requirement exists for non-volunteer parents to know the return-to-play rules and procedures. "I would say on the teams my kids play on, no, parents aren't required to know anything about concussions; which, is kind of interesting now that you've pointed it out." This father's comments suggest that volunteer parent coaches may have a higher level of concussion education than less-active parents of youth sport athletes. Likewise, our qualitative data suggests volunteer parent coaches have greater knowledge of return-to-play protocols and procedures than their peers.

4. Discussion

Results from our online survey and qualitative data collection methods consistently show that parents of youth athletes are not familiar with return-to-play concussion protocols. Both interview and survey data reveal the rules and procedures involving youth sport concussions have not been clearly explained to parents and guardians. Our mixed methods approach also discovered that parents indicate an overwhelming reliance on youth sport coaches to inform them of a child's injury. The majority of youth injuries reported in this study were joint-related therefore, the opportunity to analyze parents' behavior following a concussive event was limited.

Recent data indicate that 300,000 sport-related concussions occur annually; however, data are significantly lacking about concussions among grade school and middle school athletes [6]. Knowledge and misconceptions about mTBI have been assessed in other populations, yet concussion symptoms may be under-identified by parents and coaches of young athletes [27,28]. The misunderstandings parents and coaches often have about concussions may be improved through education [13,29], but there is a paucity of research on the effectiveness of sport concussion education [30]. Such uncertainties highlight the benefits of conducting an examination of practices and behaviors around youth sport concussions and repetitive head-impacts. It is crucial that such studies explore and identify predictors and modifiers of outcomes, including the possible influences of SES, race and ethnicity, sex, co-occurring conditions, climate (e.g. policies, practices, and procedures for the treatment of concussions), and culture (e.g. norms and values around dealing with concussions), as well as the roles of parental literacy, education, and the factors that make reporting most likely [1].

The mixed method design used for this study accomplished multiple objectives. It allowed us to gain insight on parental health literacy involving youth sport concussions, and knowledge of league rules related to education, training and return-to-play protocols for concussions. Additionally, the data provides insight into the extent that parents depend on coaches to inform them about youth sport concussion. Rather than rely on their own (perceived) ability to identify concussion symptoms, parents indicated a preference to empower youth sport coaches to inform them when or if children are injured. According to our data, emergency room staff, pediatricians, athletic trainers, and school nurses would generally be consulted only once a coach informed parents their child might have experienced a concussion. These data also suggests that parents are often unfamiliar with the CDC Heads Up website and do not typically use educational resources to learn about youth sport concussions or return-to-play rules.

Statistically significant differences emerged from the data when parents were asked if they believed they know how to identify concussion signs and symptoms. Parents who reported having college degrees were positively associated with awareness of the CDC "Heads Up" website. Parents from higher income households are more likely to show concern for youth sport concussions than those with lower income levels. Our results suggest that lower parental socio-economic-status (SES) may adversely influence the recognition of concussion symptoms, and knowledge of return-to-play rules and procedures.

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Limitations of this study include a small sample size and the lack of a racially diverse population. As a result, we are unable to determine how race and SES impacts parents' concussion literacy, knowledge of youth sport league rules involving concussions education, training, and return-to-play protocols. While participants in this study reported most athletes were engaged in youth team sports, only one-fifth of the children experienced some type of injury. Of all the reported cases of injury (20), only 2 were the result of concussion (10%). These results might not be representative of a national sample due to the low number of reported concussions and smaller presence of football participation, which nationally has the most participants in youth sports [31]. Future research that contains large-scale data collected from a nationally representative sample might impact these overall findings.

5. Conclusion

Although youth sport-concussions are a central public health priority, understandings of the etiology and epidemiology of concussions have run ahead of empirical understandings of the social influences and consequences of behaviors that surround concussions. Despite clear theoretical and clinical relevance of maintaining a healthy physically active lifestyle, the field lacks knowledge of the negative and positive reinforcement mechanisms needed to study safety, reduction and control, prevention of, and response to sports injuries. Recent advances in sports medicine discuss the need for research on real-life sport injury [32]. Such views call for a more behavioral focused approach when it comes to research and clinical practice around youth concussions and other sport injuries. Similar to findings in previous studies, the data from this study indicate a greater need for research that considers the attitudes and behavior of parents and coaches around youth concussions, while attending to the contexts in which concussion symptoms are diagnosed, and return-to-play decisions occur.

Our study represents an important next step in understanding parental health literacy involving youth sport concussions. Beyond the importance of expanding upon previous sports injury research, data in this study suggests a clear need for a more extensive study in response to the Institute of Medicine (IOM) and the National Research Council recommendation to increase knowledge about the culture (e.g., social norms, attitudes, and behavior) surrounding youth sport concussions [1].

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<p>Are you familiar with the rules/procedures that determine when a child can return to play following an injury?</p> <p>Not at all familiar <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> Somewhat familiar <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> Very Familiar <input type="checkbox"/></p>					
<p>How well do you think your coaches know the rules/procedures that determine when a child can return to play following an injury?</p> <p>Not at all familiar <input type="checkbox"/> <input type="checkbox"/> Somewhat familiar <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> Very Familiar <input type="checkbox"/></p>					
<p>For the start of the 2013 season, American Youth Football required coaches to complete the online CDC Heads Up Online Concussion Training. The National Pop Warner Youth Football organization implemented a similar requirement.</p>					
<p>How familiar are you with this rule?</p> <p>Not at all familiar <input type="checkbox"/> <input type="checkbox"/> Somewhat familiar <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> Very Familiar <input type="checkbox"/></p>					

Figure 1.
Example of Likert Scale Concussion Survey

Table 1.

Distribution of household income

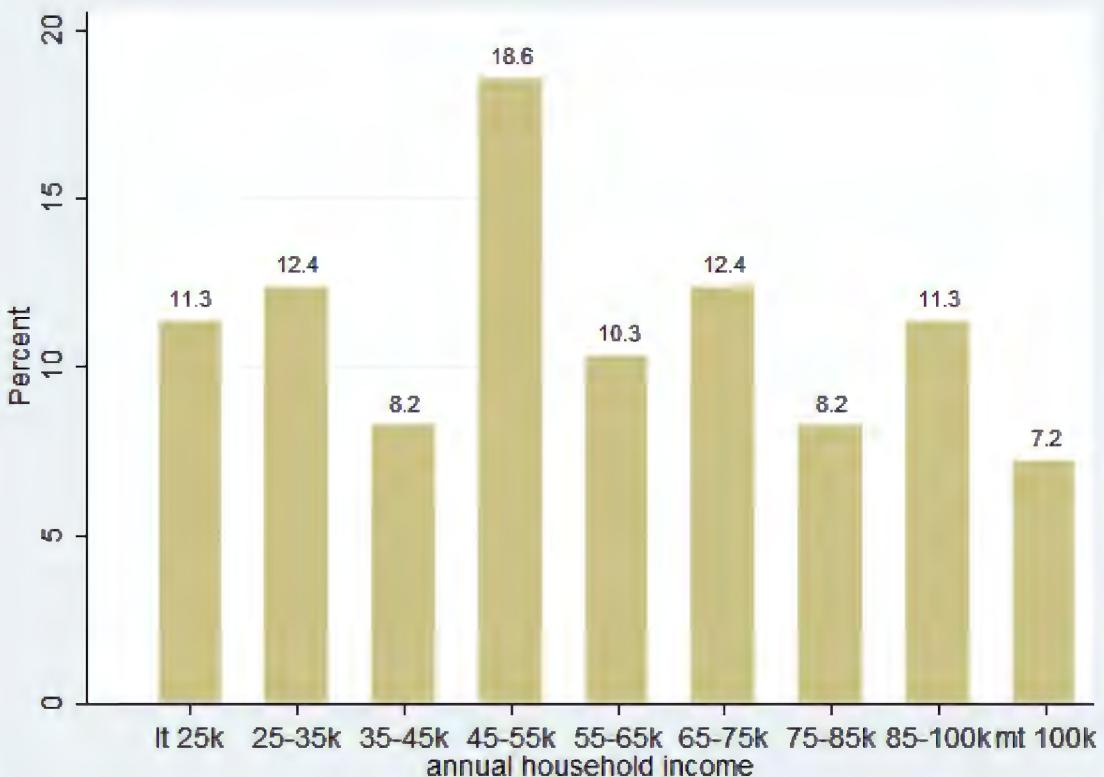


Table 2.

Type of sports participated & registered

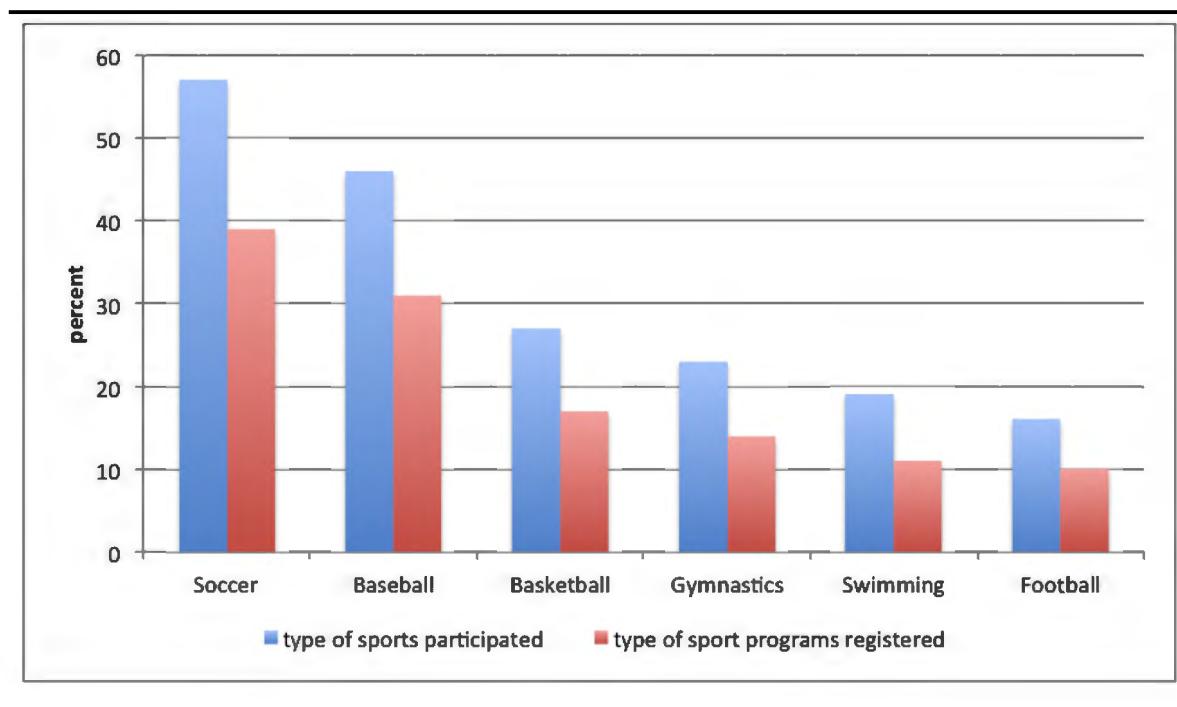


Table 3.

Familiarity with CDC “Heads Up” website.

Familiar with CDC “Heads Up”	Coef.	T-score	P> t
Gender	.4319403	1.52	0.132
Age	-.0106321	-0.50	0.621
Education	.2591754	2.39	0.019
Marital Status	-.1402572	-0.66	0.513
Race	.015626	0.10	0.922
Household Income	-.036614	-0.60	0.301

Table 4.

Concussion signs and symptoms *

Knowledge of concussion signs	Coef.	T-score	P> t
Gender	.7538341	2.27	0.025
Age	.0068848	0.28	0.784
Education	-.1364397	-1.08	0.283
Marital Status	-.1542426	-0.62	0.537
Race	-.1649793	-0.89	0.377
Household Income	.1007088	1.40	0.164

* Measured on a 7point Likert scale (continuous dependent variable analyzed in linear regression model)

Functional Limitations Mediate the Relationship Between Pain and Depressive Symptoms in Former NFL Athletes

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Abstract

The objective of this study was to analyze data from the National Football League Player Care Foundation Study of Retired NFL Players to understand potential risks for depressive symptoms in former athletes by investigating the relationship between pain and depressive symptoms in a multivariate context, while simultaneously exploring the potential connection with functional limitations. Descriptive statistics were used to describe the study sample and to conduct bivariate comparisons by race and age cohort. Linear regression models were conducted in the subsample of respondents reporting on depressive symptoms using the PHQ-9. Models examine the relationship of bodily pain, injury as a reason for retirement or not re-signing with a team, length of NFL career, sociodemographic characteristics, chronic conditions, and functional limitations to depression. Interaction terms tested whether race and age moderated the effect of bodily pain and functional limitations on depressive symptoms. Bivariate associations revealed no significant differences between younger and older former players in indicators of pain and only slightly higher functional limitations among younger former players. In the multivariate models, pain was significantly associated with depressive symptoms ($\beta = 0.36$; $p < .01$), net of a range of relevant controls. Adding an index of functional limitations reduced this association by nearly half ($\beta = 0.20$; $p < .01$) and functional limitations was significantly associated with depressive symptoms ($\beta = 0.40$; $p < .01$). No statistically significant interactions were found. Overall, bodily pain was strongly associated with depressive symptoms. After accounting for the effects of functional limitations, this association was notably reduced. These results may be useful in identifying aging-related physical declines in relatively younger adult men who may be at the greatest risk for depression. They highlight how physical functionality and activity may mitigate the risk of depression, even in the presence of significant bodily pain.

Keywords

aging, pain, NFL athletes, depressive symptoms, functional limitations

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The mental health and well-being of former National Football League (NFL) athletes is of increasing public interest (Dean & Rowan, 2014; Kerr, Marshall, Harding Jr., & Guskiewicz, 2012; Weir, Jackson, & Sonnega, 2009). Previous research has focused on the link between mild traumatic brain injury (mTBI) or concussion and depressive symptoms (Guskiewicz et al., 2007; Kerr et al., 2012; Solomon, Kuhn, & Zuckerman, 2016; Stern et al., 2011). While prevalent, results from a multiyear study suggest most NFL athletes have not been diagnosed with concussion, nor do they display recognized symptoms of head injury during their careers (Casson, Viano,

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Powell, & Pellman, 2010). Some former athletes without a reported history of sports-related concussion experience depressive symptoms (Schwenk, Gorenflo, Dopp, & Hippie, 2007). Little research has examined whether other, non-concussion-related injuries increase the risk of depressive symptoms (Guskiewicz et al., 2007; Holsinger et al., 2002).

Given the well-documented association between pain and depressive symptoms (Feeley et al., 2008; Shankar, Fields, Collins, Dick, & Comstock, 2007), surprisingly few studies have examined this link in former NFL athletes. Schwenk et al. (2007) reported a 73% comorbidity of high levels of self-reported pain and high depressive symptoms in retired NFL athletes. That study examined only bivariate associations, leaving open the question of what other physical factors may be associated with depressive symptoms. A leading contender is functional limitations, which are defined by Verbrugge and Jette (1994) as “restrictions in performing fundamental physical actions used in daily life” (p. 3). Functional limitations are associated both with pain (Liechtenstein, Dhanda, Cornell, Escalante, & Hazuda, 1998) and with depressive symptoms, especially in older populations (Bair, Robinson, Katon, & Kroenke, 2003; Ormel, Rijssdijk, Sullivan, Van Sonderen, & Kempen, 2002; Schieman & Plickert, 2007; Yeom, Fleury, & Keller, 2008). Because of the extreme demands of NFL play, functional limitations may be a cause for concern even in younger former players who may exhibit diminished physical capability associated with a pattern of accelerated aging (Belsky et al., 2015), with, for example, rates of arthritis comparable to much older men (Weir et al., 2009).

Despite recent public interest in the health of NFL athletes, a dearth of literature exists on the relationship between injury, pain, functional limitation, and depressive symptoms in this population. Using cross-sectional data from the National Football League Player Care Foundation Study of Retired NFL Players, this study builds on Schwenk et al. to examine the relationship between pain and depressive symptoms in a multivariate context permitting the exploration of the potential connection with functional limitations. Patterns of physical decline might be expected to appear at an earlier stage in the life course in this population. Indeed, prior work has reported high levels of pain in younger retired NFL athletes (Turner, 2018). The present study investigates potential age differences in the associations among pain, functional limitations, and depressive symptoms. Similarly, as the demographic profile continues to shift from a once-White majority sport to one where nearly 70% of the NFL athletes are Black (Turner, 2018), it has become important to elucidate potential racial differences in the risk pathways. Therefore, this study examines the potential effects of racial group differences.

In the case of NFL football players, injury emerges as an occupational hazard that exposes healthy young men to increased risk of functional limitations as they age. This is similar to the cases of many other collision sport athletes (e.g., boxing, roller derby, ice hockey, soccer, lacrosse, rodeo, full-contact martial arts; *Segen's Medical Dictionary*, 2011) who may equally be at risk for premature functional decline and potentially elevated depressive symptoms (Gouttebarge, Aoki, Lambert, Stewart, & Kerkhoffs, 2017; Hume et al., 2017; Manley et al., 2017; Marshall et al., 2015). Improved understanding of risks for depressive symptoms in former NFL athletes may serve as a potential model for pain, functional status, and aging to support efforts in depression treatment and intervention.

Method

Sample

Data are from the National Football League Player Care Foundation Study of Retired NFL Players (Weir et al., 2009), a survey of retired players conducted at the University of Michigan’s Institute for Social Research with a cross-sectional field period that spanned 2006–2007. The Foundation provided a complete listing of 6,983 former NFL players with vested rights (3–4 years active career) in the NFL’s pension system. A stratified random sample of 1,625 players was selected for telephone interviews. Strata were based on age and disability pension status, with older age groups and those with disabilities oversampled. The overall response rate was 65.4%, resulting in an analytic sample of 1,063. Sampling weights were used to adjust for the sample design and nonresponse (Weir et al., 2009).

Measures

Depressive symptoms. Depressive symptoms were measured in a subsample of the total sample using the Patient Health Questionnaire-9 (PHQ-9; Kroenke, Spitzer, & Williams, 2001). All respondents completed the following four screening questions (Jackson et al., 2004): “Have you ever in your life had a period of time lasting several days or longer when most of the day you felt sad, empty or depressed?”; “Have you ever in your life had a period of time lasting several days or longer when most of the day you were very discouraged about how things were going in your life?”; “Have you ever in your life had a period of time lasting several days or longer when you lost interest in most things you usually enjoy like work, hobbies, and personal relationships?”; and “Have you ever in your life had a period of time lasting several days or longer when most of the time you were very irritable, grumpy or in a bad

mood?" The fourth question is thought by some researchers to capture a more masculine form of depression, that is, irritability rather than sadness (Jackson et al., 2004). Respondents who answered yes to at least one of these questions were also asked if they had those feelings within the past month. If they responded affirmatively to having these feelings in the past month, they were then asked to answer the nine items of the PHQ-9.

Questions on the PHQ-9 asked how much in the past 2 weeks respondents were bothered by the following: "Having little interest or pleasure in doing things"; "Feeling down, depressed, or hopeless"; "Trouble falling asleep, staying asleep, or sleeping too much"; "Feeling tired or having little energy"; "Poor appetite or overeating"; "Feeling bad about yourself—or that you are a failure or have let yourself or your family down"; "Trouble concentrating on things, such as reading the newspaper or watching television"; "Moving or speaking so slowly that other people could have noticed—or the opposite—being so fidgety or restless that you have been moving around a lot more than usual"; and "Thoughts that you would be better off dead, or of hurting yourself in some way." Responses ranged from 0 to 3, and the resulting scale ranged from 0 to 27.

NFL career variables. We created a dichotomous indicator measuring the importance of injuries in the respondents' decision to retire or in their not re-signing with a team, where 1 indicated *very important* and 0 indicated *somewhat* or *not very important*. Tenure in the NFL indicated number of years playing in the NFL (range 3–29).

Pain. Respondents were asked whether in the past 3 months, they had experienced severe headaches or migraines and whether in the past 3 months, they had experienced nonminor pain that lasted a day or more in their neck or lower back. Respondents were asked whether they had experienced any joint pain within the past 30 days. Four dichotomous indicators were created for (a) severe headaches or migraines, (b) neck pain, (c) low back pain, and (d) any joint pain. A pain index indicated the number of pain symptoms reported with a range from 0 to 4.

Functional limitations. Functional limitations were assessed using the 12 items of the Nagi scale (Nagi, 1976). Respondents were asked how difficult it was for them to do things like climbing several flights of stairs; stoop, bend, or kneel; or pull or push large objects. A count of limitations was created that ranged from 0 to 12 for the number of activities respondents experienced "somewhat or greater difficulty" performing. This method of assessment provides a direct measure of everyday physical functioning.

Chronic conditions. To assess history of several chronic diseases, respondents were asked, "Have you ever been told by a health care professional that you had (disease)?" A count of the history of the following seven chronic diseases was created: high blood pressure, diabetes, coronary heart disease, stroke, emphysema, cancer, and asthma with a range of 0–7. This simple disease count method is the most commonly used form to determine multiple conditions (Wallace, McDowell, Bennett, Fahey, & Smith, 2016).

Sociodemographic characteristics. Respondents' age in years was coded both continuously and as a dichotomous variable distinguishing younger (30–49 years) and older aged men (50–90 years). Race was dichotomously coded with White equal to 1 and Black equal to 0. Education was a continuous measure of the number of years of schooling. Marital status was coded 1 for currently married and 0 for currently unmarried. Total household income and net household wealth were computed based on a series of questions on various sources of income (including wages, royalties, dividend income, pension income, etc.) and assets (including value of home, vehicles, investments, retirement accounts, etc., minus any debts). These values were reported in 2007 dollars in the table of descriptive results. The natural log of total assets and total income was used in the regression models.

Data Analysis

Descriptive statistics were generated for all study variables in the total sample of 1,063 respondents in Column 1 of Table 1. The rest of Table 1 presents a series of bivariate comparisons. The first comparison was between those who screened into the PHQ-9 section ($n = 207$, Column 2 of Table 1) with those who did not screen into the PHQ-9 ($n = 856$, Column 3 of Table 1). The next two sets of comparisons were within the PHQ-9 subsample. To examine potential race and cohort differences, a comparison between Black men ($n = 104$, Column 4) and White men ($n = 103$, Column 5) and between younger men ($n = 100$, Column 6) and older men ($n = 107$, Column 7) was conducted. Chi-square tests were used to compare dichotomously coded variables, and *t* tests were used to compare means on all continuous variables (Weiss & Weiss, 2012).

Correlations among the study variables were conducted. Linear regression models were employed to examine the independent relationships of study covariates with depressive symptoms in the subsample of men who screened into the PHQ-9 section. Zero-order regression coefficients are first reported. Model 1 evaluated the influence of the pain index and relevant sociodemographic characteristics. Model 2 added an index of

Table I. Descriptive Statistics on Study Variables and Bivariate Comparisons.

Characteristic	Total sample <i>n</i> = 1,063	Sample not in PHQ-9 <i>n</i> = 856	PHQ-9 subsample ^a <i>n</i> = 207	Black men <i>n</i> = 104	White men ^b <i>n</i> = 103	Younger men <i>n</i> = 100	Older men ^c <i>n</i> = 107
Migraine past 3 months (%)	20.98	16.37	39.57**	46.74	31.89*	42.74	34.65
Any back pain past 3 months (%)	52.85	49.45	66.60*	80.38	51.83***	67.76	64.80
Any neck pain past 3 months (%)	35.42	30.85	53.86***	50.32	57.66	53.30	54.74
Any joint pain past 30 days (%)	78.84	76.67	87.54*	90.60	84.27	87.82	87.12
Pain index (0–4)	1.88	1.73	2.48*	2.68	2.26	2.52	2.41
Age (range 27–90 years)	49.89	50.46	47.46	44.28	50.86***	39.07	60.45
White race (%)	53.78	44.85	51.72	—	—	40.98	59.60**
Education (mean years)	17.49	17.56	17.18	16.84	17.54*	17.03	17.41
Married (%)	78.73	80.66	70.80*	61.63	80.40***	65.59	79.29**
Household income (\$)	155,836	163,255	130,858***	123,992	138,212*	139,286	117,799*
Household wealth (\$)	2,805,192	3,008,055	1,986,313***	1,098,209	2,937,676***	2,618,805	1,006,377***
Injury as a reason for retirement (%)	62.32	59.94	73.48***	78.51	68.09***	79.95	63.44***
Tenure in the NFL (mean years)	6.76	6.74	6.81	6.82	6.80	6.37	7.50
Number of chronic conditions	2.53	0.80	1.10	0.96	1.26	0.90	1.41
Number of functional limitations (0–12)	3.20	2.71	5.16***	5.61	4.68	4.71	5.86
Depressive symptoms (0–27)			9.62	10.32	8.87	9.22	10.24

Note. Percentages and means are weighted using sampling weights as described in the text; sample sizes reflect the unweighted *n*. NFL = National Football League; PHQ-9 = Patient Health Questionnaire-9.

^aComparison of PHQ-9 subsample with the sample not in PHQ-9. ^bComparison of Black men and White men. ^cComparison of younger men and older men.

p* < .05. *p* < .01.

chronic conditions and an index of functional limitations. A last set of models added interaction terms to Model 2 for race and age with the pain index and race and age with functional limitations. Standardized regression coefficients are reported and the variance inflation factor (VIF) was used to test for potential collinearity among the independent variables (O'Brien, 2007). All analyses were conducted using SAS 9.4 (Institute, S.A.S., 2014) and included appropriate sampling weights.

Results

The first column of Table 1 displays means and percentages for all study variables in the total sample of 1,063 respondents. The mean age in the total sample was 49.89 years with a range of 27–90 years. Average tenure in the NFL was 6.76 years. Injury as a reason for retirement or not re-signing with the NFL was reported by 62.32%, although some athletes may overstate the role of injury as a more comfortable alternative explanation to age-related decline. Average annual household income was \$155,836 and average household wealth was \$2,805,192. Self-reports of recent pain were high, with 78.84% experiencing any joint pain, 52.85% experiencing any back pain, 35.42% experiencing any neck pain, and 20.98% reporting migraine headache.

Comparing those who screened into the PHQ-9 section to those who did not (Columns 2 and 3), men in the PHQ-9 subsample had significantly less household income and wealth than those not in the PHQ-9 subsample. There were marked differences in health, pain, and functional limitations. For example, 16.37% of those not in the PHQ-9 subsample had experienced recent migraine compared to 39.57% of men in the PHQ-9 subsample ($p < .01$). The report of any joint pain was fairly high even in the subsample who did not screen in but was significantly higher among those in the PHQ-9 subsample (76.67% vs. 87.54%; $p < .05$). Those who reported that injury was a very important reason for retirement or not re-signing with a team were more likely to screen in (73.48% vs. 59.94%; $p < .01$).

The comparison by race (Columns 4 and 5) revealed older average age among Whites (50.86% versus 44.28%; $p < .01$), a higher rate of current marriage in Whites compared to Blacks (80.40% vs. 61.63%; $p < .01$), and higher levels of household income and wealth. In terms of NFL experience, Black men were more likely to report injury as a reason for retiring or not re-signing (78.51% vs. 68.09%; $p < .01$), but there were no differences in the average tenure in the NFL. White men had significantly lower rates of migraine (31.89% vs. 46.74%; $p < .05$) and back pain than Black men (51.83% vs. 80.38%; $p < .01$).

Comparing younger and older men (Columns 6 and 7), 59.96% of the older men were White compared to nearly

40.98% of the younger group, which reflects the fact that more Blacks entered the NFL over time. Younger men were more likely to report that injury played a significant role in their retirement or not re-signing decision (79.95% vs. 63.44%; $p < .01$). Older men reported a slightly higher number of functional limitations than younger men (5.86% vs. 4.71%; $p < .05$), but strikingly, there were no age-related differences in any of the four individual pain measures or the pain index. In the total PHQ subsample, an average of 9.62 depressive symptoms was reported within the past 2 weeks. There were no statistically significant differences across race and age groups in mean depressive symptoms.

Table 2 presents correlation coefficients for all study variables within the PHQ-9 subsample. Depressive symptoms were positively and statistically significantly associated with migraine ($r = 0.33$, $p < .01$), neck pain ($r = 0.33$, $p < .01$), back pain ($r = 0.30$, $p < .01$), joint pain ($r = 0.25$, $p < .01$), injury as a reason for retirement or not re-signing ($r = 0.33$, $p < .01$), and functional limitations ($r = 0.53$, $p < .01$). Depressive symptoms were inversely correlated with net household wealth ($r = -0.28$, $p < .01$). All four pain measures were positively intercorrelated ($r = 0.22$ – 0.31 , $p < .01$) and were all positively correlated with functional limitations ($r = 0.25$ – 0.38 , $p < .01$). The pain index was significantly associated with functional limitations ($r = 0.48$, $p < .01$).

Turning to the regression results (Table 3), in Model 1, the pain index and injury as a reason for retiring or not re-signing were both positively associated ($\beta = 0.36$, $p < .01$ and $\beta = 0.23$, $p < .01$, respectively) and household assets were negatively associated ($\beta = -0.18$, $p < .01$) with depressive symptoms. In Model 2, functional limitations exerted a large and statistically significant risk for depressive symptoms ($\beta = 0.40$, $p < .01$) and reduced the effects of pain by nearly half ($\beta = 0.20$, $p < .01$). Higher household wealth remained significantly associated with lower depressive symptoms ($\beta = -0.17$, $p < .05$). VIF was no higher than 1.57 for any covariate, indicating no significant multicollinearity (Allison, 2012). Interaction terms with age and race and pain as well as age and race and functional limitations were not statistically significant, that is, the relationship between any bodily pain and depressive symptoms and between functional limitations and depressive symptoms did not differ by either race or age. This outcome may be at least partially the result of conducting three-way interaction tests of race, pain, and functional limitation in a relatively small sample.

Discussion

The major findings of this study build upon Schwenk et al. (2007), who pointed to a strong association between

Table 2. Correlations Among Study Variables in PHQ-9 Subsample ($n = 207$).

Characteristic	Migraine	Neck pain	Back pain	Joint pain	Pain index	Injury	Tenure	Age	White race	Education	Married	Income	Assets	Chronic	Functional limitations	
PHQ	0.33***	0.33***	0.30***	0.25***	0.46***	0.33***	-0.04	-0.004	-0.11	-0.15*	-0.10	-0.08	-0.28**	0.07	0.53***	
Migraine		0.29***	0.22*	0.26***	0.68***	0.07	0.03	-0.12	-0.15*	-0.09	-0.03	-0.03	-0.27**	-0.04	0.28***	
Neck pain			0.31***	0.29***	0.73***	0.11	0.03	-0.04	0.07	-0.15*	-0.02	-0.05	-0.00	-0.02	0.38***	
Back pain				0.17*	0.66***	0.13	0.21*	0.01	-0.30***	-0.16*	-0.02	-0.01	-0.15*	-0.05	0.36***	
Joint pain					0.57***	-0.03	-0.03	-0.07	-0.10	-0.07	-0.01	-0.01	-0.15*	-0.10	0.25***	
Pain index						0.12	0.10	-0.08	-0.18***	-0.18*	-0.03	-0.03	-0.04	-0.22*	-0.06	0.48***
Injury							-0.07	-0.20***	-0.12	-0.11	-0.06	-0.01	0.07	0.03	0.31*	
Tenure								0.29***	-0.00	0.02	0.14*	-0.04	0.04	-0.10	0.09	
Age									0.27***	0.11	0.19*	-0.04	-0.14*	0.30***	0.17*	
White race										0.23***	0.21*	0.05	0.19*	0.14*	-0.11	
Education											0.10	0.05	0.12	0.01	-0.29***	
Married											-0.00	0.09	0.06	-0.13		
Income												0.18*	0.07	0.00		
Assets													-0.08	-0.18***		
Chronic conditions														0.15*		

Note. PHQ-9 = Patient Health Questionnaire-9.

* $p < .05$. ** $p < .01$.

Table 3. Linear Regression of PHQ-9 Depressive Symptoms.

Parameter	Model 1	Model 2
	β	β
Pain index	0.36**	0.20**
Injury as reason for retirement	0.23**	0.14*
Tenure in the NFL	-0.07	-0.07
Age	0.06	-0.06
White race	0.01	0.03
Years of education	-0.03	0.06
Married	-0.06	-0.01
Household income	-0.02	-0.05
Household assets	-0.18**	-0.17**
Chronic conditions		0.01
Functional limitations		0.40**
R ²	0.31	0.41
Adjusted R ²	0.27	0.37

Note. Regressions weighted using sampling weights as described in the text. NFL = National Football League; PHQ-9 = Patient Health Questionnaire-9.

* $p < .05$. ** $p < .01$.

pain and depressive symptoms in former NFL athletes. The findings extend those Schwenk et al. by reporting that much of this relationship is accounted for by functional limitations. The Nagi scale (Johnson & Wolinsky, 1993; Nagi, 1976; Unger, McAvay, Bruce, Berkman, & Seeman, 1999), a well-regarded measure of physical functioning, was used to ascertain former athletes' perceived difficulties in performing 12 functional tasks. While bodily pain was strongly associated with depressive symptoms, after accounting for the effect of functional limitations, the association was reduced by nearly half. Prior research has identified that adults who self-reported significant pain prematurely developed functional limitations typically associated with those experienced by older age adults. For example, results from a nationally representative study of community-living persons age 50 years and older found that participants with significant pain had high rates of functional limitations similar to those of participants two to three decades older (Covinsky, Lindquist, Dunlop, & Yelin, 2009), although a causal association could not be determined.

Declines in functional limitations that impair independent living often occur with age. These functional limitations—such as difficulty with basic body functions, for example, lifting and carrying, climbing, or walking upstairs—rather than the more usual activities of daily living (ADLs) and independent activities of daily living (IADLs; Freedman & Martin, 1998) are so strongly associated with aging that they are regarded as a core part of the aging phenotype. Previous empirical analyses provide support that functional limitations are important initial markers of later life disability (Altman, Seelman, & Bury,

2001; Guralnik, Ferrucci, Simonsick, Salive, & Wallace, 1995; Johnson & Wolinsky, 1993; Lawrence & Jette, 1996). A key finding of this study is that younger former NFL athletes (30–49 years) experienced only slightly lower functional limitations than their more senior peers (ages 50+ years). While research on functional limitations among former athletes is sparse, these results align with a cohort study of 103 female soccer players between the ages of 26 and 40 years who sustained an anterior cruciate ligament (ACL) injury 12 years earlier (Lohmander, Östberg, Englund, & Roos, 2004). In both studies, a very high prevalence of arthritis, pain, and functional limitations was reported in these former athletes prior to age 50 years. The findings of the present study suggest that in the context of high levels of pain among former NFL athletes, the early onset of functional limitations may well lead to an increased likelihood of depressive symptoms in adulthood.

These results are consistent, albeit indirectly, with research suggestive of protective effects of physical activity on depressive symptoms. It may be that the strong results in this study related to functional limitations represent the obverse effect, especially for former NFL athletes who were accustomed to high rates of physical activity as young adults (Turner, Barlow, & Heathcote-Elliott, 2000). Additionally, the hypothesis that patterns of physical decline appear at an earlier stage in the life course was confirmed. Among former NFL athletes, functional limitations may lead to reduced physical activity and have a similar negative psychosocial impact on mental health similar to, for example, osteoarthritis (Turner et al., 2000). While restricted physical activity may be related to depressive symptoms among older men, it might be especially so in younger men. For example, outcomes from a study of community-dwelling adults demonstrated the protective effects of physical activity on depression among older adults (Strawbridge, Deleger, Roberts, & Kaplan, 2002). Similarly, a longitudinal study of elite former male athletes revealed that low or decreased physical activity at baseline was related to greater risk of depression over time (Bäckmand, Kaprio, Kujala, & Sarna, 2003).

Considerable effort has been directed at documenting the relationship between depressive symptoms and physical functioning, disability IADLs and ADLs, and mobility. Findings from the Italian Longitudinal Study on Aging indicate that baseline depressive symptoms were associated with higher rates of reported disability in men and women over a 3.5-year period (Dalle Carbonare et al., 2009). In a separate study of high-functioning older adults free of any disability, Bruce, Seeman, Merrill, and Blazer (1994) determined that increased levels of depressive symptoms predicted an increased risk of the onset of disability in basic ADLs over a 2.5-year interval. As the population of older

adults continues to expand, the need to examine the relationship between functional limitations and depressive symptoms will become even more pronounced. The results of our study align with findings of investigators who reported a relationship between functional limitations and depressive symptoms in an older adult population (Gallo, Rabins, Lyketsos, Tien, & Anthony, 1997; Hybels, Pieper, & Blazer, 2009; Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999). Nevertheless, the current analysis uncovered similar findings in the younger cohort of former NFL athletes, which may again suggest an accelerated aging process in functional limitations.

Reducing costs and managing burdens associated with aging depends on understanding the progression from health to chronic disease and disability (Belsky et al., 2015). It is valuable to study this progression in younger samples of adults with extremely physically demanding occupations earlier in life, which may lead to premature age-related functional limitations and can place them at risk for depressive symptoms in older age.

Limitations

Several limitations must be considered when interpreting the results of this study. These data are based on self-reported measures and did not use direct measures of disease and functional limitations. Retired professional athletes are a population for whom physical performance was critical to occupational success and they may well be more medically aware than the general population, thus resulting in an over-reporting bias. Prior findings suggest that these reports benchmark well to the general population (Weir et al., 2009). Nonetheless, the National Football League Player Care Foundation Study of Retired NFL Players did not include questions about the use of pain medications and the treatment of depression or other mental health disorders.

The current study did not include information on position played and what, if any, injuries were sustained during tenure in the NFL. This information would improve the capability to more accurately identify exposure. Former players in this sample were eligible for an NFL pension, including between 3 and 4 years of NFL league experience. These results may not generalize to all retired players. Furthermore, the cross-sectional design limited the exploration of potential causal associations with depressive symptoms. Future research should focus on obtaining more than one observation to provide stronger evidence about the relationships among pain, functional limitations, and depressive symptoms over time.

Implications

Given the growing concerns of illicit drug use disorders and nonmedical use of prescription drugs among adults

age 50+ years (Wu & Blazer, 2011), this study highlights the importance of developing alternative methods to manage pain that may be associated with depressive symptoms. In the case of NFL football players, injury emerges as an occupational hazard that exposes healthy young men to increased risk of functional limitations as they age. The finding that injury is a major reason for retirement or not re-signing suggests that this group of retirees may merit a special focus for intervention. While participation in this study was limited to retired NFL athletes with vested retirement rights, there is reason to believe that aging collegiate football players may be at similar risk of experiencing depressive symptoms. This concern is highlighted by high rates of injury experienced by individuals in this population (Hootman, Dick, & Agel, 2007).

In addition to vested former NFL athletes, the finding from this study may extend to nonvested former NFL athletes, Arena Football League (AFL), Canadian Football League (CFL), semiprofessional, and collegiate football players as well as. Football players often experience a long apprenticeship playing in high school and college prior to working on the professional level (Turner, 2018). Epidemiological research in the United States indicates American football is the leading cause of sport-related injuries among high school and collegiate athletes (Shankar, Fields, Collins, Dick, & Comstock, 2007). The present study finds that functional limitations, even after accounting for bodily pain, pose a significant risk for depressive symptoms. This information can inform the development of programs to help former football players.

Conclusion

Existing research has focused on assessing the prevalence of depressive symptoms and difficulty with pain in retired football players. In the present study, in a sample of former NFL athletes, the association between pain and depressive symptoms and premature aging-related functional limitations was studied. The variability of these relationships across race and age cohort was examined. In line with earlier research among former NFL athletes (Schwenk et al., 2007), pain was studied in relation to depression symptoms without assuming a pathway of influence exists in either direction.

The results of this study highlight the importance of examining the relationship among physical activities covariates, depressive symptoms, and gender, and may be useful for clinicians and others that treat men who were exposed to intensely physically demanding occupations as young adults. In summary, the findings from this study suggest that diminished physical function is particularly important for the mental health of former NFL athletes

and largely explains the relationship between pain and depressive symptoms. Future studies focused on the risk factors for depressive symptoms associated with functional limitations should expand beyond vested former NFL athletes to consider studying male and female collision sport athletes (e.g., hockey, soccer) who face high levels of bodily pain and injury.

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Statement of Ethics Approval Is Not Required

Ethics approval is not required for this article, given that it does not rise to the level of human subject research. No data were collected from human subjects. This is an analysis of secondary data intended to contribute generalizable knowledge about functional limitations, pain, and depressive symptoms among former NFL athletes. The primary beneficiaries of the research are other researchers and scholars in the field of social science and medicine.

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Original Research Article

Sleep Difficulties and Cognition for 10 Years in a National Sample of U.S. Older Adults

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Abstract

Background and Objectives: Sleep difficulties are common among older adults and are associated with cognitive decline. We used data from a large, nationally representative longitudinal survey of adults aged older than 50 in the United States to examine the relationship between specific sleep difficulties and cognitive function over time.

Research Design and Methods: Longitudinal data from the 2004–2014 waves of the Health and Retirement Study were used in the current study. We examined sleep difficulties and cognitive function within participants and across time ($n = 16201$). Sleep difficulty measures included difficulty initiating sleep, nocturnal awakenings, early morning awakenings, and waking up feeling rested from rarely/never (1) to most nights (3). The modified Telephone Interview for Cognitive Status was used to measure cognitive function. Generalized linear mixed models were used with time-varying covariates to examine the relationship between sleep difficulties and cognitive function over time.

Results: In covariate-adjusted models, compared to “never” reporting sleep difficulty, difficulty initiating sleep “most nights” was associated with worse cognitive function over time (Year 2014: $b = -0.40$, 95% CI: -0.63 to -0.16 , $p < .01$) as was difficulty waking up too early “most nights” (Year 2014: $b = -0.31$, 95% CI: -0.56 to -0.07 , $p < .05$). In covariate-adjusted analyses, compared to “never” reporting waking up feeling rested, cognitive function was higher among those who reported waking up feeling rested “some nights” (Year 2010: $b = 0.21$, 95% CI: 0.02 to 0.40 , $p < .05$).

Discussion and Implications: Our findings highlight an association between early morning awakenings and worse cognitive function, but also an association between waking up feeling rested and better cognitive function over time.

Translational Significance: Sleep difficulties are common among older adults yet reduce the quality of life and also contribute to the development of and potentially accelerate cognitive decline. This study examines specific sleep difficulties (e.g., difficulty falling asleep) and their unique relationship to cognition over time among older adults in the United States. The primary aim of this work is to illuminate the specific sleep difficulties that are most concerning from the standpoint of cognitive impairment so as to inform the design of future tailored sleep improvement programs for older adults.

Keywords: Cognitive function, Gerontology, Healthy aging, Sleep, Translational medicine

Sleep difficulties (e.g., difficulty initiating sleep and early morning awakenings) are more common among older adults (65 years or older) than any other age group in the United States (1). Particularly concerning is that sleep difficulties are associated with adverse outcomes. A review of 85 studies assessing sleep difficulty among older adults showed that sleep difficulties were detrimental to physical health and psychosocial well-being (2). Furthermore, a growing body of evidence suggests a connection between sleep difficulties and cognitive impairment and the development and progression of Alzheimer's disease (AD). With an aging population, it is critical to identify factors, such as sleep difficulty, that contribute to the initiation and worsening of AD and related dementias (3).

Research has examined sleep and cognition among older adults and employed a range of sleep parameters in doing so. In research conducted among a nationally representative sample of men using both objective and self-reported measures, researchers found a proxy for sleep difficulty (i.e., self-reported poor sleep quality) and objectively recorded nighttime awakenings were associated with cognitive decline in the follow-up period, which was between 3 and 5 years (4). Another nationally representative study using both objective and self-reported measures did not find any significant associations between self-reported sleep difficulties and cognition during the 5 years, but researchers did find objective recordings of sleep fragmentation and waking after sleep onset were associated with cognitive decline (5). Results from cross-sectional analyses showed a significant association between insomnia (i.e., chronic sleep difficulty) and reduced performance in learning and temporal order judgment among older adults (6). Similarly, another study found a measure similar to sleep difficulty (fragmented sleep) was a significant predictor of cognitive impairment at follow-up (which averaged 3 years) (7). Finally, a meta-analysis of cross-sectional and prospective studies, broadly defined and inclusive of components such as sleep quality, insomnia, and sleep apnea, found sleep difficulty was associated with cognitive impairment among older adults (8).

The literature to date is limited in several ways. One clear critique of the literature is that the conceptualization of sleep difficulty has ranged widely from one study to another. Indeed, as noted above, research has employed a range of sleep difficulty measures, ranging from sleep apnea to daytime sleepiness. Furthermore, existing literature often develops summary scores for sleep difficulty as opposed to measures of specific sleep difficulties (i.e., difficulty initiating sleep, nocturnal awakenings, and early morning awakenings) (8,9). Also, much of the evidence is based upon either cross-sectional designs or prospective observational study designs with short follow-up periods.

Moreover, to date, no longitudinal studies have examined associations of sleep difficulty (i.e., difficulty initiating sleep, nocturnal awakenings, early morning awakenings, or waking not feeling rested) with cognitive function using national data.

The present study addressed these gaps in the literature by examining specific symptoms of sleep difficulty across time and their association to cognitive function using 10 years of longitudinal, national data from the Health and Retirement Study (HRS). We hypothesized that each sleep difficulty (i.e., difficulty initiating sleep, nocturnal awakenings, early morning awakenings, or not waking up feeling rested) will be associated with worse cognitive function over time in covariate-adjusted analyses.

Method

We analyzed data from the HRS, an ongoing nationally representative longitudinal survey of adults aged older than 50 in the United States. The study began in 1992, with a core interview administered every 2 years. In 1998, HRS instituted a steady-state design, enrolling a new birth cohort every 6 years. The Health and Retirement Study uses a multistage area probability sampling design that includes geographic stratification, clustering, and oversampling of African American, Hispanic, and minority households (10). During each biennial wave of data collection, approximately 20,000 participants are surveyed. The HRS also uses proxy respondents for those individuals who are unwilling or unable to complete an interview, which has been shown to reduce attrition bias in longitudinal studies (11). Additional information regarding the study design and content is available elsewhere (12). For most variables, we used a clean and ready-to-use version of the data (13). All respondents provided consent, and the study protocol was approved by the University of Michigan Institutional Review Board.

Analytic Sample

We began with 18,327 respondents who participated in the 2004 wave of the HRS. While the sleep difficulty variables were available beginning in the prior wave in 2002, we chose 2004 as our baseline in order to maximize our sample for longitudinal analysis because 2004 was the enrollment year for the Early Baby Boomer cohort (individuals born between the years 1948 and 1953). We selected 17,592 respondents who demonstrated no significant signs of cognitive impairment at baseline, as evidenced by a score of 7 or higher on the validated Telephone Interview for Cognitive Status (TICS-m) during the baseline year for this

analysis (2004) representing 96.0% of respondents. We then selected 16,494 respondents (90.0% of the population) who were aged older than 50 years. We excluded those respondents who were represented by a proxy ($n = 293$). The final analytic sample was 16,201 respondents (88.4% of the population). The Health and Retirement Study collected information on sleep difficulties biennially until 2006, then in alternate waves thereafter. A new birth cohort was enrolled in 2010, but we excluded these new participants from our sample to maintain a consistent follow-up interval for all respondents. Thus, our analytic sample included all respondents surveyed in 2004 and at follow-up (Years 2006, 2010, and 2014).

Measures

Sleep difficulty

Sleep difficulty was measured with four items. Participants were asked to report the frequency of the following: (i) difficulty initiating sleep, (ii) difficulty maintaining sleep (nocturnal awakenings), (iii) early morning awakenings, and (iv) waking up feeling rested. Response options included 1 (most nights), 2 (sometimes), and 3 (rarely/never). We reverse-scored the items so that higher scores indicated more sleep difficulty (1 = rarely/never, 2 = sometimes, and 3 = most nights) with the exception of feeling rested, for which higher values indicated more feelings of being rested. Finally, we created a new variable to indicate those who experienced concurrent sleep difficulties. Specifically, we identified those individuals who responded either “most nights” to two or more difficulties in a single wave (i.e., difficulty initiating sleep, maintaining sleep, or early morning awakenings) or “rarely/never” to the question regarding waking up and feeling refreshed.

Cognitive function

Cognitive function was measured using the TICS-m, which included immediate and delayed recall items, serial 7s subtraction, and counting backward. Immediate list recall included presenting 10 English nouns audibly to participants at a 2 seconds/word rate (14). Participants were then asked to recall the words in any order they could remember orally, and the examiner recorded their responses. Serial 7s included a serial subtraction by 7s from 100 for five trials. This task taps numerical ability and working memory or the ability to hold and transform information simultaneously. The modified Telephone Interview for Cognitive Status has been used to screen for dementia and mild cognitive impairment. The total cognitive score was measured continuously using the composite measure, which ranged from 0 to 27. As a reference, a score ranging from 0 to 6 is consistent with dementia, 7–11 is consistent with cognitive impairment without dementia, and 12–27 is considered normal (14,15).

Covariates

We controlled for a number of covariates that are known to be associated with cognitive function (13) and sleep difficulties (16). Several covariates were taken from the respondent’s baseline interview including age in years; gender coded as 1 = female, 0 = male; race coded as 1 = white, 0 = nonwhite; ethnicity coded as 1 = Hispanic, 0 = non-Hispanic; marital status coded as 1 = married or living with a partner, 0 = unmarried (never married, widowed, and divorced); education coded as years of schooling (0–17 years or more); and total self-reported financial assets coded by quartile (1 = <\$55,000, 2 = \$55,000 to \$182,999, 3 = \$183,000 to \$464,999, and 4 = \$465,000 or higher). For all other covariates, we included wave-specific measures. Chronic medical conditions were a count variable of up to eight conditions: high blood pressure, diabetes, cancer, lung disease, heart disease, stroke, psychiatric problems, and arthritis (range 0–8), consistent with previous research (17). A modified eight-item version of the Center for Epidemiologic Studies Depression scale (CES-D) was used for the measurement of depressive symptoms. For this modified CES-D, participants reported on the extent to which in the previous week, they felt depressed, everything was an effort, sleep was restless, that he or she could not get going, lonely, that he or she enjoyed life, sad, and happy. To minimize the operational confounding of this covariate with the outcome, we removed the CES-D item that pertained to sleep. We reverse-coded the positive items and summed the remaining seven items for a count of recent depressive symptoms ranging from 0 to 7, consistent with prior research (18). While sleep apnea, which is a well-documented contributor to sleep difficulties, was not measured in this study, as a proxy for sleep apnea, we control for high body mass index (BMI), which is a known risk factor for sleep apnea (19). Body mass index was calculated according to the standard formula (weight in kilograms by the square of height in meters) from self-reported height and weight at each wave. We create a variable to signify high BMI as a score of 27 or higher, consistent with previous research, and control for this in our fully adjusted models (20).

Statistical Analyses

Baseline sample characteristics are reported in Table 1. We computed descriptive statistics for sleep difficulty and cognitive function by year (Table 2). Analysis of variance was used to examine the associations between sleep difficulty and cognitive function during the 10-year duration, followed by post-hoc comparisons using the Tukey’s honestly significant difference test. Post-hoc comparisons were conducted between the sleep disturbance levels and cognitive impairment by year. Finally, a repeated-measures generalized linear mixed model (GLMM) with maximum likelihood estimation (21)

Table 1. Demographic Characteristics of the Sample in the First Wave (Year 1, 2004) ($n = 16,201$)

		Mean \pm SD or N (%)
<i>Demographic and Health Characteristics</i>		
Age		67.51 \pm 10.8
Gender	Male	8173 (42.4%)
	Female	11,107 (57.6%)
Marital status	Married	11,833 (80.4%)
	Living with partner	174 (1.2%)
	Separated	543 (3.7%)
	Divorced	315 (2.1%)
	Widowed	1844 (12.5%)
Race	White/Caucasian	15,569 (80.8%)
	Black/African American	2770 (14.4%)
	Other	938 (4.9%)
Assets	$\leq \$55,000$	4350 (26.4%)
	$\$54,999$ to $\$182,999$	4151 (25.2%)
	$\$18,300$ to $\$464,999$	4037 (24.5%)
	$\geq \$465,000$	3960 (24.0%)
Hispanic ethnicity		1783 (9.2%)
Body mass index		27.4 \pm 5.6
Self-rated health	Excellent	800 (7.0%)
	Very good	3241 (28.4%)
	Good	3895 (34.1%)
	Fair	2539 (22.2%)
	Poor	951 (8.3%)
Depressive symptoms (CES-D)		1.46 \pm 2.0
Chronic conditions	0	3305 (17.2%)
	1	5035 (26.1%)
	2	4908 (25.5%)
	≥ 3	6023 (31.2%)
<i>Sleep Difficulty Characteristics</i>		
Difficulty initiating sleep	Never	5332 (51.4%)
	Sometimes	3552 (34.3%)
	Most nights	1482 (14.3%)
Nocturnal awakenings	Never	3607 (34.9%)
	Sometimes	3947 (38.3%)
	Most nights	2766 (26.8%)
Waking early	Never	5441 (52.5%)
	Sometimes	3599 (34.8%)
	Most nights	1317 (12.7%)
Feeling rested	Never	5089 (57.6%)
	Sometimes	2621 (29.7%)
	Most nights	1124 (12.7%)

Note: CES-D = Center for Epidemiologic Studies Depression scale.

and robust standard errors (22) examined associations between sleep difficulties and cognitive function longitudinally over time (Tables 3–5). To address our primary question of interest, each model specified the main effects for each sleep difficulty, time, and their interaction (time \times sleep difficulty). As sleep difficulties may not change linearly over time between assessments, time was represented as a categorical variable in the repeated

measures models with the baseline assessment in 2004 used as the reference. This analysis was repeated for each sleep difficulty, which was entered into the model with the ordinal structure from 1 (rarely/never) to 3 (most nights). All models controlled for time-varying covariates including chronic conditions, depressive symptoms, and high BMI. Several covariates including age at baseline, marital status, gender, race, Hispanic ethnicity, assets, and education were drawn from the baseline interview data. The cutoff statistical significance for retaining variables in the model was set to p value less than .05, and different models were compared based on Akaike's information criterion. Model fit was assessed using the Wald statistic to test the null hypothesis, which was that the coefficients are simultaneously equal to zero. All analyses were performed using Stata software, version 16 (StataCorp, College Station, TX).

Results

Summary statistics for all demographic and sleep difficulty variables at baseline are given in Table 1. The average age of the sample at baseline (Year 1, 2004) was 67.51 years ($SD = 10.8$ years). Participants reported a mean total asset value of \$409,730 ($SD = 1,080,667.7$). Among participants, self-reported health was rated “excellent” by 7.0%, “very good” by 28.3%, “good” by 34.1%, “fair” by 22.2%, and “poor” by 8.3%. Among participants, 80.4% were married, 80.8% were white/Caucasian, and 9.3% were Hispanic/Latino. The average BMI of the participants was 27.4. Approximately one quarter (26.1%) reported one chronic medical condition and another quarter (25.5%) reported two chronic conditions.

Regarding sleep difficulty, the majority of individuals reported “never” having difficulty initiating sleep (51.4%), followed by those reporting experiencing difficulty “some nights” (34.3%) and “most nights” (14.3%). Regarding difficulty with nocturnal awakenings, the majority responded “some nights” (38.3%), followed by “never” (34.9%) and “most nights” (26.8%). Regarding difficulty waking too early and not being able to return to bed, the majority of participants reported “never” experiencing this difficulty (52.5%), followed by “some nights” (34.8%) and “most nights” (12.7%). Regarding waking and feeling rested, the majority of the participants reported “never” waking and feeling rested (57.6%), followed by “some nights” (29.7%) and “most nights” (12.7%). Overall, the average cognitive function score was 15.6 ($SD = 3.9$).

Figure 1 displays cognitive function, which declines over time. Figure 2A–D displays wave-specific descriptive statistics for cognitive function based on each reported sleep difficulty. Overall, the figures reflect the decreasing level of cognitive function during the 10-year period.

Using analysis of variance, we found significant associations between each sleep difficulty and cognitive function (difficulty initiating sleep: $F(2, 46,497) = 1.53$,

Table 2. General Linear Mixed Models Examining Cognitive Function and Difficulty Initiating Sleep ($n = 16,201$)

	Model 1			Model 2			Model 3		
	95% CI		<i>p</i> Value	95% CI		<i>p</i> Value	95% CI		<i>p</i> Value
	<i>b</i>	Lower		<i>b</i>	Lower		<i>b</i>	Lower	
<i>Difficulty Initiating Sleep</i>									
Direct effect of difficulty									
Never	Reference	-0.43	-0.24	0.000	-0.34	-0.43	-0.24	0.000	-0.25
Sometimes	-0.19	-0.32	-0.06	0.004	-0.19	-0.32	-0.06	0.004	-0.15
Most nights	-0.21	-0.39	-0.03	0.023	-0.21	-0.39	-0.03	0.023	-0.13
Direct effect of time									
2004	Reference	-0.34	-0.43	-0.24	-0.34	-0.43	-0.24	0.000	-0.35
2006	-1.18	-1.28	-1.09	0.000	-1.18	-1.28	-1.09	0.000	-1.15
2010	-1.68	-1.78	-1.57	0.000	-1.68	-1.78	-1.57	0.000	-1.09
2014	-0.03	-0.20	0.14	0.740	-0.03	-0.17	0.13	0.840	-0.03
Time × Difficulty									
2006 × Sometimes	-0.02	-0.19	0.15	0.848	-0.03	-0.19	0.15	0.781	-0.05
2010 × Sometimes	0.03	-0.15	0.20	0.763	0.03	-0.15	0.20	0.763	0.02
2014 × Sometimes	-0.23	-0.45	-0.01	0.042	-0.21	-0.43	-0.02	0.044	-0.19
2006 × Most nights	-0.13	-0.35	0.11	0.266	-0.13	-0.36	0.10	0.266	-0.14
2010 × Most nights	-0.40	-0.64	-0.17	0.001	-0.40	-0.63	-0.15	0.001	-0.40
2014 × Most nights									

Notes: Model 1: models include no covariates. Model 2: models include all demographic and health covariates. Model 3: models include all demographic and health covariates. Bold indicates significance less than .05.

Table 3. General Linear Mixed Models Examining Cognitive Function and Nocturnal Awakenings ($n = 16,201$)

	Model 1			Model 2			Model 3				
	95% CI		<i>p</i> Value	95% CI		<i>b</i>	95% CI		<i>p</i> Value		
	<i>B</i>	Lower	Upper	Lower	Upper		Lower	Upper			
<i>Nocturnal Awakenings</i>											
Direct effect of difficulty											
Never	Reference	-0.11	-0.24	0.03	.121	-0.10	-0.23	0.04	.166		
Sometimes		-0.09	-0.26	0.09	.326	-0.02	-0.17	0.13	.823		
Most nights											
Direct effect of time	Reference	-0.39	-0.51	-0.27	.000	-0.32	-0.44	-0.20	.000		
2004		-1.19	-1.32	-1.07	.000	-1.16	-1.29	-1.04	.000		
2006		-1.65	-1.78	-1.52	.000	-1.65	-1.78	-1.52	.000		
2010											
2014											
Time × Difficulty	Reference	0.06	-0.12	0.24	.497	0.07	-0.11	0.24	.466		
2006 × Sometimes		0.06	-0.13	0.24	.542	0.03	-0.15	0.21	.746		
2010 × Sometimes		-0.05	-0.23	0.14	.632	-0.06	-0.24	0.13	.552		
2014 × Sometimes		0.02	-0.17	0.21	.819	0.06	-0.12	0.25	.496		
2006 × Most nights		-0.10	-0.29	0.09	.315	-0.08	-0.27	0.11	.422		
2010 × Most nights		-0.19	-0.39	0.01	.065	-0.18	-0.38	0.02	.075		
2014 × Most nights											

Notes: Model 1: models include no covariates. Model 2: models include all demographic and health covariates. Model 3: models include all demographic and health covariates. Bold indicates significance less than .05.

Table 4. General Linear Mixed Models Examining Cognitive Function and Early Morning Awakenings ($n = 16,201$)

	Model 1			Model 2			Model 3					
	95% CI		<i>p</i> Value	95% CI		<i>b</i>	95% CI		<i>p</i> Value			
	<i>b</i>	Lower		Upper	Lower		Lower	Upper				
<i>Early Morning Awakenings</i>												
Direct effect of difficulty frequency												
Never	Reference											
Sometimes	-0.14	-0.26	-0.01	.036	-0.11	-0.24	0.02	0.086	-0.09			
Most nights	-0.23	-0.41	-0.05	.014	-0.14	-0.32	0.05	0.143	-0.12			
Direct effect of time	Reference											
2004	-0.34	-0.43	-0.24	.000	-0.27	-0.37	-0.18	.000	-0.26			
2006	-1.20	-1.29	-1.10	.000	-1.17	-1.27	-1.07	.000	-1.09			
2010	-1.63	-1.73	-1.52	.000	-1.64	-1.74	-1.53	.000	-1.50			
2014									-1.61			
Time × Difficulty	Reference											
2006 × Sometimes	-0.02	-0.19	0.14	.795	0.03	-0.13	0.20	.707	0.05			
2010 × Sometimes	0.00	-0.17	0.17	.990	0.01	-0.16	0.18	.924	0.01			
2014 × Sometimes	-0.14	-0.31	0.03	.111	-0.12	-0.29	0.05	.163	-0.10			
2006 × Most nights	-0.20	-0.43	0.03	.084	-0.15	-0.38	0.08	.195	-0.06			
2010 × Most nights	-0.03	-0.27	0.20	.788	-0.04	-0.28	0.19	.722	0.01			
2014 × Most nights	-0.38	-0.62	-0.13	.003	-0.36	-0.61	-0.12	.004	-0.31			

Notes: Model 1: models include no covariates. Model 2: models include all demographic and health covariates. Model 3: models include all demographic and health covariates. Bold indicates significance less than .05.

Table 5. General Linear Mixed Models Examining Cognitive Function and Reports of Waking and Feeling Rested ($n = 16,201$)

	Model 1			Model 2			Model 3			
	95% CI		<i>p</i> Value	95% CI		<i>p</i> Value	95% CI		<i>p</i> Value	
	<i>b</i>	Lower	Upper	Lower	Upper		<i>b</i>	Lower	Upper	
<i>Feeling Rested</i>										
Direct effect of difficulty frequency										
Never	-0.13	-0.27	0.01	.067	-0.10	-0.23	0.04	.163	-0.09	
Sometimes	-0.15	-0.34	0.04	.124	-0.12	-0.31	0.07	.222	-0.11	
Most nights										
Direct effect of time										
2004	-0.09	-0.19	0.01	.088	-0.09	-0.19	0.02	.096	-0.08	
2006	-1.00	-1.11	-0.90	.000	-1.00	-1.10	-0.90	.000	-0.95	
2010	-1.65	-1.75	-1.55	.000	-1.66	-1.76	-1.55	.000	-1.55	
2014										
Time × Difficulty										
2006 × Sometimes	-0.12	-0.31	0.07	.206	-0.13	-0.32	0.06	.190	-0.09	
2010 × Sometimes	0.20	0.00	0.39	.046	0.19	0.00	0.38	.051	0.21	
2014 × Sometimes	0.00	-0.18	0.19	.958	0.01	-0.18	0.19	.939	0.06	
2006 × Most nights	-0.14	-0.39	0.11	.264	-0.15	-0.39	0.10	.246	-0.08	
2010 × Most nights	-0.02	-0.26	0.23	.896	-0.01	-0.25	0.23	.923	0.04	
2014 × Most nights	-0.21	-0.46	0.05	.108	-0.19	-0.44	0.06	.133	-0.12	

Notes: Model 1: models include no covariates. Model 2: models include all demographic and health covariates. Model 3: models include all demographic and health covariates. Bold indicates significance less than .05.

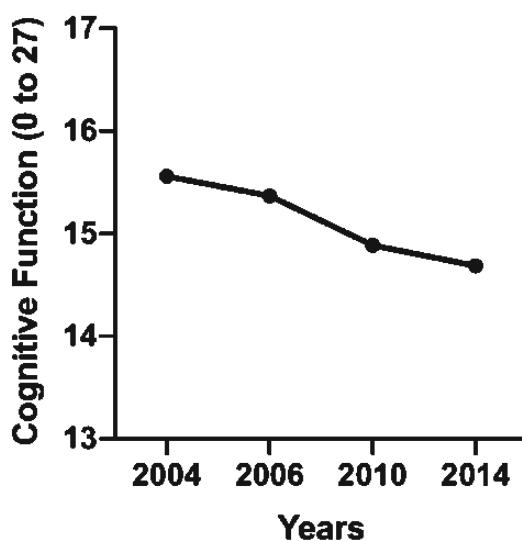


Figure 1. Cognitive function for a period of 10 years.

$p < .001$; nocturnal awakenings: $F(2, 46,425) = 4.19, p = .015$; waking too early: $F(2, 46,480) = 229.02, p < .001$; feeling rested: $F(2, 35,220) = 49.94, p < .001$). Post-hoc comparisons examining pairwise relationships using the Tukey's honestly significant difference revealed that cognitive function was significantly different between those who "never" experienced difficulty initiating sleep and those who reported "sometimes" experiencing difficulties initiating sleep ($p < .001$) and "most nights" experiencing difficulty initiating sleep ($p < .001$). Additional post-hoc pairwise comparisons revealed that cognitive function was significantly different between those who "never" experienced nocturnal awakenings and those who experienced difficulty with nocturnal awakenings "sometimes" ($p < .05$), but not those who experienced nocturnal awakenings "most nights" ($p = .933$). Pairwise post-hoc comparisons revealed that cognitive function was significantly different between those who "never" experienced waking too early and those who experienced difficulty waking too early "sometimes" ($p < .001$) and "most nights" ($p < .001$). Post-hoc comparisons also revealed that cognitive function was significantly different between those who reported "never" waking and feeling rested and those who reported "sometimes" waking and feeling rested ($p < .001$) and those who reported "most nights" waking and feeling rested ($p < .001$).

Tables 2–5 display results of the GLMM examining the relationships between each difficulty (i.e., difficulty initiating sleep, nocturnal awakenings, early morning awakenings, and waking up feeling rested) and cognitive function over time by examining the interaction between difficulty and time. Table 2 displays the GLMM examining difficulty initiating sleep and cognitive function over time. In unadjusted Model 1 analyses, compared to "never" experiencing difficulty initiating sleep, difficulty "most nights" was associated with worse cognitive function in 2006 ($b = -0.23,$

95% confidence interval [CI]: -0.45 to $-0.01, p < .05$) and in 2014 ($b = -0.40, 95\% CI: -0.64$ to $-0.17, p < .01$). In Model 2, partially adjusted analyses, compared to "never" experiencing difficulty initiating sleep, difficulty waking up too early "most nights" was associated with worse cognitive function in 2006 ($b = -0.21, 95\% CI: -0.43$ to $-0.02, p < .05$) and in 2014 ($b = -0.40, 95\% CI: -0.63$ to $-0.15, p < .01$). In Model 3, fully adjusted analyses, compared to "never" experiencing difficulty initiating sleep, difficulty "most nights" was associated with worse cognitive function in 2014 ($b = -0.40, 95\% CI: -0.63$ to $-0.16, p < .01$). The overall test of significance suggested that the coefficients were not simultaneously equal to zero (Wald chi-square = 11,708.9, 2 degrees of freedom, $p < .001$).

Table 3 displays the GLMM examining nocturnal awakenings and cognitive function over time. Nocturnal awakenings were not, in any model, associated with cognitive function over time.

Table 4 displays the GLMM examining early morning awakenings and cognitive function over time. In Model 1, unadjusted analyses, compared to "never" waking up too early and not being able to fall back asleep, difficulty "most nights" was associated with worse cognitive function in 2014 ($b = -0.38, 95\% CI: -0.62$ to $-0.13, p < .01$). In Model 2, partially adjusted analyses, compared to "never" waking up too early and not being able to fall back asleep, difficulty "most nights" was associated with worse cognitive function in 2014 ($b = -0.36, 95\% CI: -0.61$ to $-0.12, p < .01$). In Model 3, fully adjusted analyses, compared to "never" waking up too early and not being able to fall back asleep, difficulty "most nights" was associated with worse cognitive function in 2014 ($b = -0.31, 95\% CI: -0.56$ to $-0.07, p < .05$). The overall test of significance suggested that the coefficients were not simultaneously equal to zero (Wald chi-square = 11,218.3, 2 degrees of freedom, $p < .001$).

Table 5 displays the GLMM examining reports of waking up feeling rested and cognitive function over time. In Model 1, unadjusted analyses, compared to "never" waking up feeling rested, those who reported "sometimes" waking up feeling rested were more likely to have a higher cognitive function in 2010 ($b = 0.20, 95\% CI: 0.00$ to $0.39, p < .05$). In Model 3, fully adjusted analyses, compared to those who reported "never" waking up feeling rested, those who reported "sometimes" waking up feeling rested were more likely to have a higher cognitive function in 2010 ($b = 0.21, 95\% CI: 0.02$ to $0.40, p < .05$). The overall test of significance suggested that the coefficients were not simultaneously equal to zero (Wald chi-square = 11,021.7, 2 degrees of freedom, $p < .001$).

Table 6 displays the GLMM examining concurrent sleep difficulties and cognitive function over time. In Model 1, unadjusted analyses, compared to not having concurrent difficulties, those with these difficulties demonstrated lower cognitive function in 2010 ($b = -0.71, 95\% CI: -1.32$ to

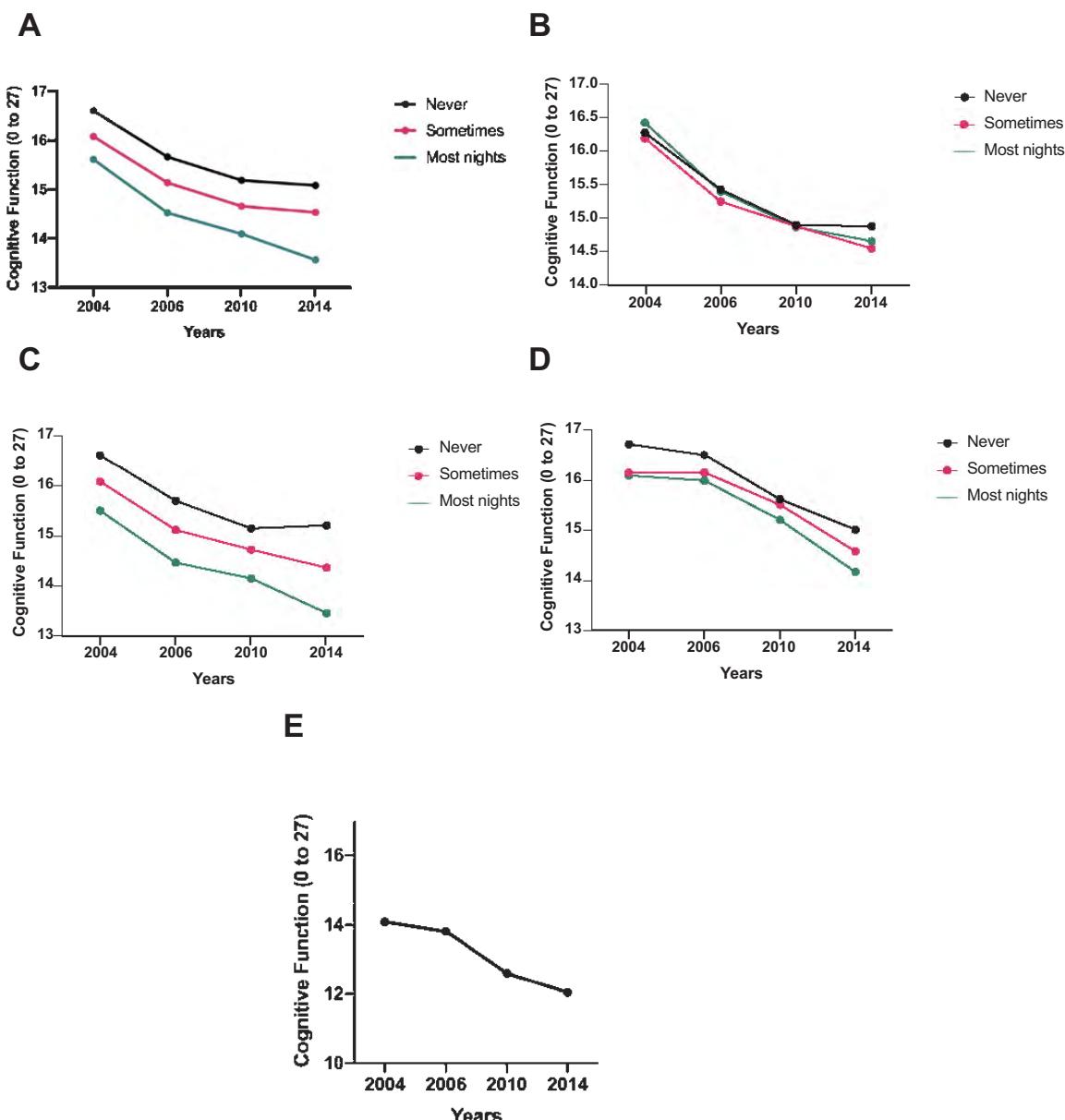


Figure 2. Cognitive function versus (A) trouble initiating sleep, (B) nocturnal awakenings, (C) early morning awakenings, (D) waking feeling rested, and (E) those reporting concurrent sleep difficulties for a period of 10 years.

-0.12, $p < .05$). In Model 2, partially adjusted analyses, compared to those not reporting concurrent difficulties, those who reported these difficulties demonstrated lower cognitive function in 2010 ($b = -0.64$, 95% CI: -1.23 to -0.05, $p < .05$). The overall test of significance suggested that the coefficients were not simultaneously equal to zero (Wald chi-square = 18,443.9, 2 degrees of freedom, $p < .001$).

Discussion

We examined specific sleep difficulties and their relationship to cognitive function during a 10-year period using nationally representative, longitudinal data collected

among U.S. adults aged 51 or older between 2004 and 2014. Findings from our study demonstrate that, after adjusting for demographic and health condition covariates, two of the four sleep difficulties measured in this study were associated with cognitive function over time. First, early morning awakenings were associated with worse cognitive function over time. Specifically, for those individuals who reported experiencing early morning awakenings “most nights,” compared to never reporting such difficulties, there was a significant decline in scores on the validated instruments assessing cognitive function. Second, in adjusted analyses, those who reported feeling rested “some nights” had better cognitive function over time. With an aging population in the

Table 6. General Linear Mixed Models Examining Cognitive Function and Reports of Concurrent Sleep Difficulties ($n = 16,201$)

	Model 1	Model 2	Model 3	95% CI					
				<i>b</i>		<i>p</i> Value		<i>b</i>	
				Lower	Upper	Lower	Upper	Lower	Upper
<i>Concurrent Sleep Difficulties</i>									
Direct effect of difficulty frequency	Reference	Reference	Reference	.286	-0.20	-0.64	0.25	.390	-0.19
Never	-0.24	-0.71	0.21						
Sometimes									
Direct effect of time	Reference	Reference	Reference	.088	-0.28	-0.34	-0.21	.000	-0.25
2004	-0.35	-0.43	-0.28						
2006	-1.18	-1.26	-1.11	.000	-1.16	-1.23	-1.09	.000	-1.08
2010	-1.71	-1.75	-1.63	.000	-1.71	-1.79	-1.64	.000	-1.55
2014									
Time × Difficulty									
2006 × Concurrent difficulties	-0.36	-0.93	0.20	.197	-0.11	-0.66	0.49	.671	0.05
2010 × Concurrent difficulties	-0.71	-1.32	0.12	.019	-0.64	-1.23	-0.05	.034	-0.58
2014 × Concurrent difficulties	-0.58	-1.22	0.05	.079	-0.53	-1.17	0.10	.099	-0.50

Notes: Model 1: models include no covariates. Model 2: models include all demographic and health covariates. Model 3: models include all demographic covariates. Bold indicates significance less than .05.

United States, understanding the contributing factors to cognitive impairment and the initiation and worsening of AD is imperative (5). Results from our study offer clinical and practical utility in identifying the specific sleep difficulties that relate to cognitive function.

Our results extend and refine the published literature on sleep and human aging. Our research is consistent with cross-sectional data which has shown that older adults reporting either insomnia symptoms or late-life insomnia exhibit significantly reduced performance in learning rate and in temporal order judgment tests (6), as well as research that has documented associations between objective, wrist-worn actigraphy recordings of poor sleep (i.e., increased fragmentation) and cognitive decline (4,5,7). Also, our findings on the relationship between sleep difficulties and cognitive function are confirmatory with meta-analysis documenting a strong association between both poor sleep quality and insomnia and greater risk for AD (8). However, findings from research employing both objective and subjective measures of sleep difficulties, has been mixed. Some work has found an association between both self-reported measures (i.e., poor sleep quality) and objective measures (i.e., actigraphy recorded sleep difficulties) of sleep difficulty and cognitive decline (4), but other work has found no relationship between self-reported measures, only between objective measures of sleep difficulty and cognitive function (5). It could be that each sleep difficulty offers a novel contribution or risk factor for cognitive decline. Therefore, retaining the individual difficulties in our analyses may have enabled new insights into distinct sleep difficulties that are most important for cognitive function, which would be challenging to detect with summary scores as have been previously employed in the literature.

There are several mechanisms that may aid in the interpretation of our findings. First, without restorative, sufficient sleep older adults reporting difficulties with early awakenings or not feeling refreshed could have been deprived of the restoration of energy stores for which sleep is known. Second, memory consolidation takes place during sleep (23). Without restorative sleep, this ability is likely deprived, leading to worse performance on the validated cognitive batteries employed in this study. Third, research has uncovered the beneficial role sleep plays in removing dementia and AD biomarkers (24). Finally, while a shift in circadian preference toward earlier rising times is documented in the literature as a normal part of age-related neurological processes (25), it could be that our research highlights a potentially greater decline in cognitive function among those who experience an extreme shift in their rising time.

Also, it is interesting that we find reports of waking up feeling rested “some nights” were associated with better cognitive function over time. Certainly, feeling well-rested is indicative of the absence of sleep difficulty, suggesting a different perspective on the relationship between sleep and

cognitive function. It should be noted, however, that it is somewhat surprising to see the relationship between waking up feeling rested “sometimes” but not “most nights.” One plausible explanation for this finding is that even a healthy sleeper may experience occasional difficulty and associated subsequent low feelings of being rested upon waking from time to time, leading most individuals to report “sometimes” waking and feeling rested as opposed to “most nights.” Another plausible explanation for the relationship between waking up feeling rested “sometimes” and cognitive impairment may simply have to do with unbalanced groups, for the majority of the sample reported “never” waking and feeling rested at baseline (57.6%), followed by approximately one third who reported “sometimes” (29.7%) and a significantly smaller proportion reporting waking and feeling rested “most nights” (12.7%). In either case, our results showed that, even in fully adjusted models, this effect persists.

Although we included a broad array of sleep difficulties in our analysis (i.e., difficulty initiating sleep, nocturnal awakenings, early morning awakenings, and waking up feeling rested), only early morning awakenings and waking up feeling rested were significantly associated with cognitive function over time. The lack of significant findings for the other sleep difficulty items may be understood through the lens of previous literature. First, extensive literature documents that sleep is fundamentally more challenging among older adults compared to their younger counterparts (26). Thus, lifespan-related decay in sleep may be difficult to disentangle from difficulties that may determine the worsening of cognition. Also, research has shown that medical comorbidities among older adults may limit sleep capabilities (27). Specifically, research has shown that, through comprehensive screening to recruit physically and psychologically healthy older adults, insomnia prevalence among older adults is quite low (between 2% and 3%) (28), suggesting that we may have had different findings if we performed our analysis on adults who remained healthy throughout the study. Alternatively, while we control for various evidence-based demographic factors and health conditions, it is possible that these variables may be in the causal pathway between sleep disturbance and cognitive functioning (e.g., sleep disruption may lead to depressive symptoms which then cause reductions in cognitive functioning). Therefore, models with full covariate adjustment should be interpreted cautiously. Another possible explanation may have to do with sleep state misperception. Specifically, research using multiple methods of sleep, including polysomnography and diary reports, found significant night-to-night variability in sleep parameters within older individuals, suggesting some individuals are simply poor judges of sleep depth or quality (29).

Sleep difficulty is more prevalent among older adults than younger adults (30) and in prior studies has been associated with AD and dementia. Furthermore, previous literature has shown that the proportion of older adults who

reported sleep difficulty at baseline nearly doubled after 2 years (27).

Strengths and Limitations

The longitudinal design of the HRS data is a significant strength of this study. They enabled our analysis of cognitive function and sleep difficulty over time. Cross-sectional studies, by comparison, are unable to address the core question of this study that examined the variability in cognitive function due to specific sleep difficulties over time. Unfortunately, while the HRS has been collecting data for nearly one decade prior to the waves selected for this study, questions on sleep were not included until 2004. Furthermore, beginning in the 2006 wave of data collection, the sleep measures were offered only in alternate waves, making for uneven follow-up intervals.

The sleep difficulty measures employed in this study included simplistic measures of frequency (e.g., “never,” “sometimes,” and “most nights”). Also, previous research that has examined self-reported sleep difficulty and objectively recorded sleep (i.e., wrist-worn actigraphy (31) and polysomnography) has shown weak correlations between self-reported sleep difficulty questionnaire responses and objective measures of sleep. Another notable limitation is that the self-reported measures are prone to bias among all groups but particularly among older adults who may be affected by cognitive decline and memory impairment which compromise their ability to offer an accurate rendering of the nature and frequency of sleep difficulties.

An additional limitation is that our regression models used time as a categorical predictor rather than a continuous one. Based on the trends of cognition over time (shown in Figure 2), we did not believe that the relationship between time and cognition was linear. To account for this, time was coded as a categorical variable and all main and interaction effects of time were interpreted with 2004 as the reference. As a result, the interpretation of the sleep difficulty by time interaction is more complex. However, we believe that the additional complexity of these models makes them more accurate. Future analyses should examine whether the trends shown in the current data are maintained at later assessment periods. A related limitation is that the current analysis uses multiple comparisons. The goal of the current analysis is to examine multiple components of sleep disruption, time, and their interaction effects using multiple levels of covariate adjustment. This requires the interpretation of multiple effects. Using methods to adjust for multiple comparisons (e.g., Bonferroni correction) might lead to ignoring moderate effect-size associations. Additional research is required to replicate the findings shown in the current study. Finally, obstructive sleep apnea may contribute to

sleep difficulties, such as early morning awakenings, yet was not provided in this data set. As a proxy for sleep apnea, we control for high BMI, which is a known risk factor for sleep apnea (19). Future research may consider including this condition as a covariate in an exploration of the relationship between sleep difficulty and cognitive function.

Implications and Future Research

Several consensus statements call for increased attention to nature and methods for attenuating sleep difficulties among older adults, including a National Institutes of Health consensus statement, which recently addressed the diagnosis, risks, consequences, and treatment of chronic insomnia among adults (32). The American Academy of Sleep Medicine has published practice guidelines for evaluating and managing sleep difficulties. The current study examined sleep difficulties and cognitive function among older adults, with the aim of informing policies and programs for improving the key aspects of sleep associated with cognitive function and the ultimate aim of improving cognitive function among older adults. Research using data from the Established Populations for the Epidemiologic Studies of the Elderly suggests that treating sleep difficulty among older adults may be a promising approach for decelerating the rate of cognitive decline (33).

Our results also identify a number of opportunities for future research. For instance, research to better identify the causal pathway between sleep difficulty and the onset and development of AD and related dementia is one opportunity for future research. Future research may also consider how to design programs to attenuate sleep difficulty among older adults. For instance, evidence-based programs such as Cognitive Behavioral Therapy for Insomnia (34) may be tailored to the needs of older adults.

Conclusions

Sleep disturbance is more prevalent among older adults relative to their younger counterparts and is associated with cognitive decline and risk for AD. We used a large, national data set to examine sleep difficulties and cognitive function across a 10-year period among older adults in the United States. Our results show that early morning awakenings were associated with worse cognitive function, whereas reports of waking up feeling rested were associated with better cognitive function, over time. Future research may examine methods for attenuating sleep difficulties among older adults.

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Author Contributions

Study concept and design: R.R., A.S., R.W.T., G.J.-L., R.S.O., and M.B. Acquisition of participants and/or data: A.S., K.M.L., and R.R. Analysis and interpretation of data: R.R., A.S., R.W.T., G.J.-L., R.S.O., and M.B. Preparation of the manuscript: R.R., A.S., and R.W.T.

Conflict of Interest

None Declared.

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The Price of Playing Through Pain: The Link Between Physical and Behavioral Health in Former NFL Athletes

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Abstract

Over the past decade, media outlets have drawn attention to some of the health consequences of playing in the National Football League (NFL), including how wear-and-tear and injuries accumulated during athletes' playing years can affect their physical, emotional, and behavioral health after retirement from professional sports. Through a secondary analysis of a cross-sectional telephone survey of former NFL athletes, this study estimated logistic regression models to assess the relationship between several forms of physical pain and anger attacks, controlling for binge drinking, signs of depression, functional limitations, NFL career duration, religious service attendance, and demographic characteristics (age, marital status, race, education, income, and wealth). The analytic sample included 1030 former NFL players. Neck pain, lower back pain, headaches/migraines, and the number of sites of pain were positively and significantly related to anger attacks. There was no significant association between joint pain and anger attacks. NFL career duration was negatively associated with anger attacks, as was religious service attendance. Future research should focus on factors that protect against affective aggression in former professional athletes and how protective factors can be adapted to the broader population.

Keywords

aggression, behavioral issues, mental health, pain, athletes, anger

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Football is one of America's most popular sports, with an estimated 1,006,013 high school males and 73,712 college males having participated during the 2018–2019 school year alone (National Collegiate Athletic Association, 2020). Of the top 50 most-watched television programs in 2019, 47 were National Football League (NFL) games, with approximately 16.5 million fans tuning in each week (Young, 2019). Professional athletes are accorded celebrity status in American society, so their lives off the field attract significant media attention, both positive and negative. Among the most concerning reports have been highly publicized physical altercations involving individual professional athletes, which have raised public concerns about the possibility of a link between participation in football and interpersonal aggression. Interestingly, there is little research evidence that NFL athletes are significantly more aggressive than

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other men, but given the popularity of football, any link between participation in the sport and off-field aggression needs to be understood.

Some scholars have attributed athlete aggression to the features of professional sports culture and institutions. Hattery and Smith (2019), for example, have described college and professional sports as permeated with a hyper masculinist, gender-segregated, internally protected subculture that fosters deviance that carries over into athletes' personal lives (see also Benedict, 2005; Carter, 2009; Smith, 2014). But in recent years, in large part due to an accumulation of evidence of traumatic brain injury (TBI) in former football players (Bailes et al., 2013; Omalu et al., 2005, 2006) and research linking TBI to aggression (Buckley et al., 2017; Farrer et al., 2012; Pouwels et al., 2018), an alternative line of inquiry has developed focusing on player health, and how the physical wear-and-tear, head trauma, and other injuries accrued on the field can affect athletes' emotional and behavioral well-being not only during their playing years but also into retirement (Souter et al., 2018). This study contributes to that literature by examining the relationship between a particular kind of aggression, impulsive, or affective, aggression, and pain (Liu, 2006; McCloskey et al., 2010). The main hypothesis of this study is that high levels of pain may predispose some former NFL athletes to affective aggression, which is characterized by a loss of emotional control and episodic anger attacks.

Pain and Anger Episodes

Affective aggression is defined by impulsivity and a lack of premeditation, which distinguishes it from instrumental, predatory forms of aggression that are characteristically controlled, goal-oriented, and strategically geared toward dominating others (Allen & Anderson, 2017; Liu, 2006). As the term implies, affective aggression is typically accompanied by strong emotions, particularly anger, which manifest in verbal or physical outbursts, directed at individuals, animals, or property (American Psychiatric Association, 2013). Frequent and recurrent anger outbursts are a defining feature of intermittent explosive disorder (IED), which is a clinically diagnosed impulse-control disorder characterized by recurrent episodes of anger that are out of proportion to situational stressors (American Psychiatric Association, 2013).

The centrality of anger to affective aggression is particularly important to understanding the role played by pain. While the pathways linking pain and anger are complex, anger has been reported in the literature as a core component of pain experience (Fernandez & Wasan, 2009). In clinical settings, pain and anger are related in patients experiencing a broad range of health problems,

with anger being the most prominent negative emotion in chronic pain patients (Cosio, 2019). Pain is also associated with anger in individuals who experience frequent headaches (Hatch et al., 1991). The relationship between headaches and anger is especially important for the purposes of this study because of the link between headaches and TBI (Mayer et al., 2013). Given the prevalence of TBI among former NFL players (Bailes et al., 2013; Omalu et al., 2005, 2006; Weir et al., 2009), a finding that headaches are associated with episodic anger in this population could be relevant to research on the pathways from TBI to aggression.

Researchers have also identified significant relationships between pain and IED (Coccaro et al., 2014; Fishbain et al., 1986; McCloskey et al., 2010). Some evidence has pointed to a neurochemical link between pain and outbursts characteristic of IED. Specifically, Coccaro et al. (2014) examined the roles played by C-reactive protein and interleukin-6, which are elevated in patients experiencing inflammation due to painful conditions such as arthritis, spine injury, neuropathy, and headaches (Davenport, 2016; Svensson, 2010; Zhou et al., 2016). Coccaro et al. (2014) found direct correlations between these inflammatory markers and several measures of aggression, including formally diagnosed IED, a composite measure of aggressive behavior, and history of aggression. Consistent with this, an epidemiological survey conducted by McCloskey et al. (2010) found a relationship between IED and neck pain, back pain, headaches, and other forms of chronic pain.

Theoretically, borrowing from the general model of aggression (Allen & Anderson, 2017; Anderson & Bushman, 2002), pain can be understood as situational input that increases the odds of aggression through its influence on affect or emotion (e.g., anger, hostility, irritability). From this perspective, pain does not directly cause aggression, but produces an internal state that potentially, depending on other factors, predisposes an individual to respond aggressively to a given stimulus or situation (Allen & Anderson, 2017). The following section will propose some of those additional factors, particularly as they might apply to former NFL athletes.

Pain and Anger Attacks in Former NFL Athletes

A literature review for this study did not yield any research reporting that former NFL athletes were significantly more likely than men in the general population to experience or display anger episodes. In fact, in a study conducted by researchers at the Institute for Social Research (ISR) at the University of Michigan, a sample of former NFL players were actually less likely than men in the general population to respond affirmatively to

screening questions for IED (Weir et al., 2009). However, NFL players are exposed to injury risk at relatively young ages, and as a result experience pain earlier in life compared to other men. In the ISR study, when a sample of former NFL players ages 30–49 were asked if they had “pain in any joint lasting most of the day” at any point over the past 3 months, 80% of the respondents answered affirmatively, compared to only 20.6% of a national sample of men the same age, and 37.1% of men age 50 and over (Weir et al., 2009). Large differences were also found for neck pain, back pain, and migraine headaches, with younger NFL former players being more likely to report pain than both older and younger men in the general population (Weir et al., 2009). Given the heightened tendency of former NFL players to experience pain, and the relationship between pain and episodic anger, the role that pain might play in predisposing some former players to anger attacks needs to be considered.

Aside from research on TBI, the literature review for this study also did not yield health research on former NFL players that focused specifically on anger episodes. However, a study of former NFL athlete mortality showed that 9.7% of former player deaths were due to either self-harm or interpersonal violence, and an additional 13.9% were due to transportation injuries (Venkataramani et al., 2018, Table 3). These findings were consistent with a media outlet report on arrest rates for players during their playing years. Although player arrests were only 13% of those for men in the general population, among the categories with the highest arrest frequencies were driving under the influence (of alcohol or other drugs), domestic violence, and nondomestic assault (Morris, 2014). Car accidents, self-harm, and interpersonal violence are all behaviors in which impulsivity or anger could play a role.

Other Relevant Correlates of Anger Attacks

One factor that has been linked to IED in the general population is depression (Medeiros et al., 2018). For some athletes, the years following the end of professional play have been reported to be emotionally difficult, as athletes are confronted with losses of identity, social relationships, income, and daily routines. Some athletes respond to these losses with maladaptive coping strategies, depression, hostility, or anger (Wolanic et al., 2015). A research review by Souter et al. (2018) showed this was particularly the case for former athletes who suffered from multiple severe injuries or surgeries during their careers, as they were two to seven times more likely than other elite athletes to suffer from symptoms of common mental disorders (p. 2). Researchers have also identified a link between severe musculoskeletal injuries and

increased symptoms of anxiety and depression in elite athletes (Gouttebarge et al., 2016).

Another factor that has been positively associated with aggression is alcohol consumption (Bushman & Cooper, 1990; O’Leary, 1999; Pappas et al., 2004). Weir et al. (2009) reported that former NFL players were more likely to consume alcohol than were men in the general population. Former NFL players were also slightly more likely to report heavy drinking, defined as more than 730 drinks per year, and binge drinking, defined as five or more drinks on 25 or more days in the past year (Weir et al., 2009). The factors that lead to alcohol abuse are many and varied, and in the absence of research on alcohol consumption among professional athletes, attribution of their drinking to any one factor is difficult. Alcohol consumption habits could be established during playing years as part of the celebrity lifestyle that exists within pro sports (Carter, 2009), or in later years as a mechanism for coping with pain, functional limitations, or new life circumstances (Souter et al., 2018; Weir et al., 2009).

It is also important to consider the influence of length of play in the NFL, which could serve as an environmental modifier that socializes individuals in ways that either predispose them toward or protect them from tendencies toward affective aggression (Allen & Anderson, 2017). Because the effects of socialization do not immediately disappear after individuals leave the environments where socialization occurs, factors associated with NFL acculturation could continue to influence athletes beyond their years of play. If so, the longer the exposure, the stronger its expected influence would be.

As stated previously, some authors have attributed norm violations during pro athletes’ playing years to the cultural and institutional features of professional elite sports, such as hypermasculine socialization, celebrity privilege, and institutional protection from consequences (Benedict, 2005; Carter, 2009; Hattery & Smith, 2019). Some scholars have also proposed that repeated play in contact sports such as hockey, boxing, or football could habituate athletes to aggression in their personal lives (Smith, 2014). However, research on contact athletes’ off-field behavior has not supported this hypothesis (Grange & Kerr, 2011; Lemieux et al., 2002; Zillmann et al., 1974). Furthermore, in two experimental studies, athletes were better able than nonathletes to control their pain responses. Thornton et al. (2020) found that athletes in contact sports were better able than athletes in noncontact sports to maintain their performance on a motor task during painful stimulation, a finding the researchers attributed to contact athletes’ perception of pain as a challenge rather than a threat. Manning and Fillingim (2002) also found that athletes had higher pain tolerances than nonathletes, which the athletes themselves attributed to competitive coping strategies.

Some additional factors associated with professional sports participation may also play a role in reducing propensity toward aggression. For example, income has been identified as negatively associated with domestic violence (Pan et al., 1994), as has been education (Harris & Knight-Bohnhoff, 1996), and retired players have higher median incomes (Weir et al., 2009) and much higher college graduation rates (Weir et al., 2009) than men in the general population. The potentially positive role of some masculine norms should also be considered. For example, Gerdes and Levant (2018) found that norms pertaining to emotional control and primacy of work are negatively related to anger and stress. There could likewise be protective masculine norms inculcated during NFL playing years that carry over after the end of professional play. Consider the research on "mental toughness," which Gucciardi et al. (2017, p. 308) define in terms of positive psychological resources that develop through experience with stress and anxiety: mental toughness "foster(s) goal-directed behavior by enabling individuals to strive (i.e., direction and magnitude of effort expended on a task), survive (i.e., manage everyday challenges or overcome major adversities) and thrive (i.e., experience growth through one's experiences)." While the conventional wisdom has been that mental toughness is negatively related to mental health, Gucciardi et al.'s research review showed that mental toughness can actually protect against symptoms of depression and anxiety.

Another set of protective factors could come from religious participation. For example, Ellison and Anderson (2001) identified a negative relationship between religious attendance and domestic violence, and Bremner et al. (2011) found that prayer reduced both anger and aggressive behavior toward others. Contrary to popular stereotypes, many NFL players are religious, and their religiosity is supported in the NFL through formal chaplaincies and sports ministry organizations. Carter and Carter (2014) found that in a sample of 104 NFL players, 66.3% claimed to have personal relationships with God, and that player religiosity had a deterrent effect on deviant behavior by increasing group cohesion and social support, and reducing anomie that the authors describe as a by-product of professional sports culture. In a survey assessing the overall health and well-being of retired NFL players, Weir et al. (2009) found that former players were almost twice as likely as men in the general population to identify as very religious, and were more likely (35% vs. 24%) than men in the general population to report weekly church attendance. Religious beliefs, habits, coping strategies, support networks, or health-supporting norms could have protective effects that shield former athletes from anger attacks.

Any of the factors reviewed above could be associated with affective aggression among former players just as

they are associated with it or related aspects of emotional or behavioral health for other populations. But former NFL players are different from most populations in one key regard: in their professions, former NFL players push their bodies to their maximum capabilities, and subject them to repeated punishment, strain, and overuse, such that injury is their most recognized risk factor for stress (Souter et al., 2018). The effects of physical stress continue to be felt in later years, with not only older, but even younger former players reporting much higher levels of pain than men in the general population (Weir et al., 2009).

The following analysis extends previous research on affective aggression by examining how pain is associated with affective aggression within a particular group of men, former NFL athletes, who tend to report more pain compared to men in the general population, and whose physical and behavioral health have been a source of public concern. We hypothesize that, even when controlling for factors that have been associated with anger and affective aggression in the literature reviewed for this study, pain will be positively and significantly associated with anger attacks.

Methods

Participants

The present study consisted of a secondary analysis of data that were originally collected for the NFL Player Care Foundation Study of Retired Players, conducted by the ISR at the University of Michigan (Weir et al., 2009). The data did not contain information (e.g., names, current or former places of residence, positions, or teams for which respondents played while in the NFL) that would allow the authors of the present study to identify individual survey participants. Approval for the analysis was obtained from the Institutional Review Board at George Washington University (IRB #180210). The Player Care Foundation provided ISR with a complete listing of 6983 former NFL players with vested rights (minimum 3–4 years active career) in the NFL's pension system in 2006–2007. A stratified random sample of 1625 players was selected. Strata were based on age and disability pension status, with older men and those with disabilities oversampled. The response rate for the ISR study was 65.4%, resulting in a sample of 1063 participants (Weir et al., 2009). For this secondary analysis, 33 cases were missing data due to lack of response for the following variables: 20 for anger attacks, one for married, and 12 for NFL career length. Because of the incomplete data, these cases were eliminated from the analysis. The remaining analytic sample was 1030 (97%).

The ISR survey provided a wealth of information on a population that presents with a unique health profile. On

the one hand, the respondents were elite athletes whose former occupation required that they maintain optimal health. As such, even after the end of their football careers, NFL athletes had better health on average than did men in general population samples on a variety of indicators (Weir et al., 2009). On the other hand, former NFL players tended to suffer from high levels of pain and functional limitations relatively early in life due to overuse and career-related injury (Weir et al., 2009). The ISR-Player Care Foundation survey allows for much needed assessments of the effects of pain on the behavioral health of men within this population.

Measures

Affective aggression was measured using responses to questions adapted from the *The Diagnostic and Statistical Manual of Mental Disorders*, 4th ed. (DSM IV) screen for IED about lifetime occurrence of “anger attacks” (American Psychiatric Association, 1994; Coccato et al., 1997). Anger attacks, a binary variable, was coded 1 for affirmative responses to any of three questions: “Thinking now about your life after football only, have you ever had attacks of anger when all of a sudden you lost control and” (a) “broke or smashed something worth more than a few dollars?” (b) “hit or tried to hurt someone?” or (c) “threatened to hit or hurt someone?” Research testing the three-question scale showed that the internal consistency of the three questions was very good ($\alpha = .87$) and that the scale was appropriate for use in nonclinical samples (Coccato et al., 2017, p. 22). The Cronbach’s α for the anger attacks variable in the present study was .86.

Five pain measures were used, based on four questions adapted from the National Health Institute Survey (National Center for Health Statistics, 2009). The first three questions asked whether respondents had experienced (a) neck pain, (b) lower back pain, and (c) severe headache or migraine that lasted a whole day or more in the past 3 months. Respondents were asked to “not report aches and pains that are fleeting or minor.” A fourth question asked about (d) pain, aching, or stiffness in or around a joint during the past 30 days. Respondents were explicitly asked to not include back or neck pain in their response to this question. In addition to separate measures for each of the four pain sites, a pain index was created to indicate the number of sites of pain, ranging from 0 to 4, based on responses to these four questions.

Several variables were included as controls based on their relationships to either pain or aggression as identified in previous research (Ellison & Anderson, 2001; Holtzworth-Munroe & Stuart, 1994; Hotaling & Sugarman, 1986; Wilt & Olson, 1996). Binge drinking was a continuous variable, based on how many days in the previous year respondents consumed five or more

alcoholic drinks in one day (0–365). Thirty-six cases were missing for the binge drinking variable; their values were set to the mean to prevent losing the cases. The depression screen variable was coded 1 for affirmative response to any of the four screening questions about the lifetime experience of

a period lasting several days or longer when most of the day (a) you felt sad, empty or depressed; (b) you were very discouraged about how things were going in your life; (c) you lost interest in most things you usually enjoy like work, hobbies, and personal relationships; or (d) you were very irritable, grumpy or in a bad mood. (Jackson et al., 2014)

Functional limitations were measured using an index, with values of 0–12, based on the Nagi scale (Nagi, 1976). The Nagi scale measures the number of routine activities, such as climbing stairs, personal care, and preparing meals, that respondents report as being at least “somewhat difficult.” NFL career length was measured using the first and last season played (range 3–20 years). Weekly or more than weekly attendance at religious services was coded 1; responses indicating less than weekly attendance were coded 0.

Sociodemographic controls included age, college education (1 for bachelor’s degree or higher; 0 otherwise), marital status (1 for married at the time of the survey; 0 otherwise), and continuous measures of total income and net wealth computed from self-reported income (including wages, royalties, dividend income, pension income, etc.) and assets (including home value, vehicles, investments, retirement accounts, etc., minus any debts). The natural log of binge drinking, income, and assets was used to correct for skewed distributions.

Race was a binary variable, coded 1 for white, coded 0 for all other races. Because only 19 respondents in the sample identified as being racially other than white or black, identification of their race could have compromised their anonymity. To protect their privacy, their race, along with the race for respondents who identified as black, was coded as non-white.

Data Analysis Procedures

The analyses were performed using STATA 16 SE. Descriptive statistics for all study variables were generated. Bivariate and multivariable logistic regression was performed to evaluate the relationship between anger attacks and the independent variables, including controls.

Results

Descriptive Statistics

The analysis of demographic controls (Table 1) shows that 817 (79%) respondents were married at the time of

Table I. Descriptive Statistics: Characteristics of Retired NFL Athletes Who Responded to the Retired Player Survey ($n = 1030$).

Characteristic	Mean (SD)	Range
Controls		
Age (years)	54.70 (14.17)	26–91
Married (%)	78.83	0,1
White race (%)	58.83	0,1
Income (US\$)	161,732 (335,180)	0–4,259,887
Assets (US\$)	2,856,089 (10,600,000)	0–202,000,000
College graduate (%)	78.45 (0.41)	0,1
Depression screen (%) ^a	19.61 (0.40)	0,1
Functional limitations (0–11) ^b	3.53 (3.25)	0–11
Binge drinking ^c	13.50 (44.93)	0–365
NFL career length (years) ^d	8.10 (3.15)	3–20
Weekly religious attendance (%) ^e	34.47	0,1
Pain^f		
Any joint (%)	78.54	0,1
Headache/migraine (%)	19.13	0,1
Neck (%)	35.63	0,1
Low back (%)	53.40	0,1
Pain index ^g	1.87 (1.19)	0–4
Anger attacks ^h	30.87	0,1

Note. ^aFour-item set of questions on the lifetime experience of a period lasting several days or longer when most of the day you felt sad, empty, or depressed; you were very discouraged about how things were going on in your life; you lost interest in most things you usually enjoy; you were very irritable, grumpy, or in a bad mood. Coded 1 for reporting at least 1 symptom; 0 otherwise.

^b12-item set of questions asking how difficult it was to do things like climb several flights of stairs; stoop, bend, or kneel; or pull or push large objects. A count of limitations was created that ranged from 0 to 12 for how many activities respondents reported as at least "somewhat difficult" to perform.

^cIn the past year, on how many days did you consume five or more alcoholic beverages?

^dCalculated using the first and last season played.

^eHow often do you attend religious services? Coded 1 for weekly or more; 0 otherwise.

^fSurvey asked about pain during the past 30 days that lasted most of the day and was not fleeting or minor.

^gNumber of sites of pain.

^hComposite variable coded 1 for affirmative responses to at least one of three questions: Since you left football, have you ever had attacks of anger when all of a sudden you lost control and (a) broke or smashed something worth more than a few dollars, (b) threatened to hit or hurt someone, or (c) hit or tried to hurt someone?; 0 otherwise.

the survey (90.8% had been married at some point), 809 (78.5%) were college graduates, 606 (58.8%) were white, the average annual income was \$161,732 (range \$0–\$4,259,887), average assets were \$2,856,089 (range \$0–\$202,000,000), and the mean age was 55 years (range 26–91). The average NFL career length was 8.1 years (range 3–20), and 355 respondents (34.5%) reported attending religious services at least weekly. As for substantive controls, 202 (19.6%) respondents reported at least one symptom from the depression screen. Binge drinking was heavily skewed, with a mean of 13.5 days per year, and a median of 0 days per year of consuming five or more alcoholic beverages (range 0–365). Respondents reported an average of 3.5 functional limitations (range 0–11). Respondents reported an average of 1.9 pain sites, with 809 (79%) reporting joint pain, 550 (53%) reporting back pain, 367 (36%) reporting neck pain, and 197 (19%) reporting headaches or migraines. Three hundred and eighteen (31%)

respondents reported a lifetime occurrence of at least one anger attack.

Logistic Regression: The Relationship Between Pain and Anger Attacks

The bivariate models (the first column in Table 2) separately regressed anger attacks on each of the controls and on each of the pain variables. Age ($OR = .98$, 95% CI [.97, .99], $p = .001$), married ($OR = .70$, 95% CI [.51, .95], $p = .024$), and college graduate ($OR = .62$, 95% CI [.46, .85], $p = .003$) were each significantly associated with reduced odds of an anger attack, as were weekly religious attendance ($OR = .56$, 95% CI [.42, .75], $p < .000$) and NFL career length ($OR = .94$, 95% CI [.90, .98], $p = .005$). The depression screen ($OR = 2.82$, 95% CI [2.05, 3.86], $p < .000$), functional limitations ($OR = 1.16$, 95% CI [1.12, 1.21], $p < .001$), and binge drinking ($OR = 1.23$, 95% CI [1.13, 1.34], $p < .001$) were

Table 2. Logistic Regression. Factors Associated With Anger Attacks^a among Former NFL Players (*n* = 1030).

Parameter	Bivariate OR (.95 CI)	Multivariable ^b OR (.95 CI)				Model 5 Pain Index
		Model 1 Any joint Pain	Model 2 Headache/Migraine	Model 3 Neck Pain	Model 4 Lower Back Pain	
Controls						
Depression screen ^c	2.82*** (2.05–3.86)	1.99*** (1.39–2.85)	1.84*** (1.28–2.65)	1.87*** (1.30–2.69)	1.94*** (1.35–2.77)	1.83*** (1.27–2.63)
Functional limitations ^d	1.16*** (1.12–1.21)	1.14*** (1.08–1.19)	1.12*** (1.07–1.18)	1.11*** (1.06–1.17)	1.13*** (1.07–1.18)	1.09*** (1.03–1.14)
Binge drinking ^e	1.23*** (1.13–1.34)	1.19*** (1.08–1.32)	1.20*** (1.09–1.33)	1.20*** (1.08–1.33)	1.19*** (1.08–1.32)	1.19*** (1.08–1.32)
NFL career length ^f	0.94*** (0.90–0.98)	0.93*** (0.89–0.99)	0.93*** (0.89–0.98)	0.93*** (0.89–0.98)	0.93*** (0.89–0.98)	0.93*** (0.88–0.97)
Religious attendance ^g	0.56*** (0.42–0.75)	0.66* (0.48–0.91)	0.64*** (0.46–0.88)	0.65*** (0.47–0.89)	0.66* (0.49–0.93)	0.65*** (0.47–0.90)
Pain^h						
Any joint	2.13*** (1.48–3.07)	1.28 (0.86–1.90)				
Headache/migraine	2.83*** (2.06–3.90)	1.84*** (1.26–2.69)				
Neck	2.62*** (1.99–3.44)		1.90*** (1.40–2.58)		1.46* (1.08–1.99)	
Lower back	2.09*** (1.59–2.74)					1.40*** (1.21–1.62)
Pain index ⁱ	1.66*** (1.47–1.87)	0.40 (0.13–1.25)	0.34 (0.11–1.04)	0.35 (0.12–1.06)	0.40 (0.13–1.19)	0.20*** (0.06–0.64)
Constant		0.10	0.11	0.11	0.10	0.12
Pseudo R ²						

Note. ^aComposite variable coded 1 for affirmative responses to at least one of three questions: Since you left football, have you ever had attacks of anger when all of a sudden you lost control and: (a) broke or smashed something worth more than a few dollars; (b) threatened to hit or hurt someone; or (c) hit or tried to hurt someone? 0 otherwise.

^bControl variables not included in the table: Age, Married, White Race, Income (natural log), Assets (natural log), and College Graduate.

^cFour-item set of questions on the lifetime experience of a period lasting several days or longer when most of the day you felt sad, empty or depressed; you were very discouraged about how things were going on in your life; you lost interest in most things you usually enjoy; you were very irritable, grumpy, or in a bad mood. Coded 1 for reporting at least 1 symptom; 0 otherwise.

^d12-item set of questions asking how difficult it was to do things like climb several flights of stairs; stoop, bend, or kneel; or pull or push large objects. A count of limitations was created that ranged from 0 to 12 for how many activities respondents reported as at least "somewhat difficult" to perform.

^eIn the past year, on how many days did you consume five or more alcoholic beverages? (natural log)

^fCalculated using the first and last season played.

^gHow often do you attend religious services? Coded 1 for weekly or more; 0 otherwise.

^hSurvey asked about pain during the past 30 days that lasted most of the day and was not fleeting or minor.

Number of sites of pain.

* $p < .05$, ** $p < .01$, *** $p < .001$ (two-tailed tests).

Source: NFL Player Care Foundation Study of Retired Players.

significantly associated with increased odds of an anger attack. Each of the individual pain variables and the pain index were associated with increased odds of an attack as follows: any joint ($OR = 2.13$, 95% CI [1.48, 3.07], $p < .001$), headache/migraine ($OR = 2.83$, 95% CI [2.06, 3.90], $p < .001$), neck ($OR = 2.62$, 95% CI [1.99, 3.44], $p < .001$), lower back ($OR = 2.09$, 95% CI [1.59, 2.74], $p < .001$), and pain index ($OR = 1.66$, 95% CI [1.47, 1.87], $p < .001$).

The first four multivariable models (Table 2) regressed anger attacks on the individual sites of pain and controls. Headaches ($OR = 1.84$, 95% CI [1.36, 2.69], $p = .002$), neck pain ($OR = 1.90$, 95% CI [1.40, 2.58], $p < .001$), and back pain ($OR = 1.46$, 95% CI [1.08, 1.99], $p = .015$) were all significantly associated with increased odds of an anger attack. The relationship between joint pain and aggression, though positive, was not statistically significant ($OR = 1.28$, 95% CI [0.86, 1.90], $p = .226$). As for control variables, binge drinking ($p < .01$), the depression screen ($p < .01$), and functional limitations ($p < .001$) were statistically significant and positive across all models 1–4. NFL career length ($p < .01$) and weekly religious attendance ($p < .05$) were significant and negative across the four models. The final multivariable model (5) examines the relationship between the pain index and anger attacks. Each additional site of pain was associated with increased odds of an attack ($OR = 1.41$, 95% CI [1.21, 1.62], $p < .001$), as were binge drinking ($OR = 1.19$, 95% CI [1.21, 1.62], $p < .001$), the depression screen ($OR = 1.83$, 95% CI [1.27, 2.63], $p = .001$), and functional limitations ($OR = 1.09$, 95% CI [1.03, 1.14], $p = .002$). NFL career length ($OR = 0.93$, 95% CI [.88, .97], $p = .002$) and weekly religious attendance ($OR = 0.65$, 95% CI [.47, .90], $p = .009$) were both negatively associated with anger attacks.

Robustness Checks

Several robustness checks were run using alternative constructions of variables. The pain index was the independent variable used to measure pain in each of the robustness check models. First, several alternative measures of alcohol consumption were tested. Thirty-six cases were missing data for binge drinking. In addition to the models reported in Tables 1 and 2, in which the values of the missing cases were set to the mean (13.5), models were run with values for the missing cases set to the median (0), and models were run that excluded the cases with missing data. In all three models, pain increased the odds of an anger attack and the relationships were statistically significant ($p < .001$). Models were also run in which the binge drinking variable was replaced with a variable that measured the number of drinks consumed per week. Here as well, the coefficient for pain was

statistically significant ($p < .001$) and positive. However, the number of drinks per week was not statistically significant ($p = .053$).

Second, for the depression screen, the dichotomous variable was replaced with a scaled variable (0–4) based on the number of depression screening questions to which the respondents answered “yes.” The OR (1.37) for the scaled depression variable was statistically significant ($p < .001$). The coefficient for pain in this model was also positive and statistically significant ($p < .001$).

Third, models were run in which the weekly religious attendance variable was replaced with multiple variables measuring respondents’ self-identification as either “religious” or “spiritual.” None of the alternative measures was statistically significant, and their inclusion in the models did not change the significance of the coefficients for pain.

Fourth, the dichotomous anger attacks variable was replaced with two alternatives. The first alternative was a scaled dependent variable (0–3), based on the number of IED screening questions to which the respondent answered “yes.” The pain index was statistically significant ($OR = 0.10$, $p < .001$). An additional model used a binary variable measuring the occurrence of an anger attack within the past year instead of a lifetime occurrence. The relationship between the pain index and an attack within the past year was also positive and significant ($OR = 1.44$, 95% CI [1.07, 1.95], $p = .016$).

Discussion

In a representative sample of former NFL players, back pain, neck pain, and headaches were all significantly associated with increased odds of an anger attack, as was a cumulative pain index, meaning that with each additional site of pain, the odds of an affective aggression increased. Binge drinking, depression markers, and functional limitations were also significantly and positively related to anger outbursts; however, when controlling for these factors, the pain variables remained statistically significant, indicating that pain plays a distinct role in anger attacks that is not explained by binge drinking, depression markers, functional limitations, or the other controls. These findings are in accord with literature suggesting that pain is a risk factor for anger and affective aggression (Coccaro et al., 2014; Cosio, 2019; Fernandez & Wasan, 2009; Fishbain et al., 1986; McCloskey et al., 2010).

The finding that NFL career length is significantly and negatively associated with affective aggression suggests that longer duration of professional participation in football actually decreases the odds of affective aggression later in life. While care should be taken not to overstate the significance of this finding, it is consistent with research showing that athletes who engage in contact sports are better than others at controlling their emotions

when subjected to pain (Manning and Fillingim, 2002; Thornton et al., 2020). One interpretation of the present study's finding on NFL career duration could be that professional football trains participants to reign in and productively direct aggressive impulses as governed by the rules of the game, even in emotionally charged, pain-inducing situations. If so, longer exposure to the sport could increase the chances that this sort of conditioning would carry over into men's personal lives, in this case after leaving the NFL. Another explanation could be that injury plays a role in shortening career duration, and the higher odds of episodic anger for those with shorter careers reflect the pain suffered from those injuries.

Limitations and Strengths

Several limitations of this study need to be considered. First, the survey did not include a measure of respondents' histories of head injury; therefore, the observed association between headaches/migraines and anger attacks needs to be interpreted with caution and not be taken as evidence for a link between TBI and episodic aggression. The finding does suggest, however, that research on the behavioral effects of TBI might benefit from efforts to disentangle pain from other factors.

The IED measures also present with limitations. While the IED measures allow this study to be compared to other studies on affective aggression that use them, the measures do not operationalize social context; therefore, they do not provide information as to whether reported anger attacks occurred, for example, in family relationships, intimate partnerships, social settings, or in response to provocation. The IED measures also do not specify what kind of "hurt" or "pain" inflicted on others (economic, physical, emotional) is implied when respondents answer affirmatively to the anger attack questions. Furthermore, while the IED survey questions ask about "lost control," the questions do not capture whether self-reported attacks are out of character for the respondents or are instead related to more ingrained patterns of intentional behavior. Research shows that the distinction between affective and instrumental aggression is not clear cut, that motives are often mixed, and that the two forms of aggression are correlated (Allen & Anderson, 2017). Future research on IED should distinguish among these different forms of aggression, how they might at times be interrelated, and how they operate across various contexts and targets. Additionally, the anger attacks variable measures presence or absence of attacks, whereas frequency of attacks over the life course would have allowed for more detailed comparisons. Finally, the survey questions from which the anger attacks variable was created were retrospective, potentially compromising reliability. However, the pain coefficient was also positive and significant in the model

using the dependent variable measuring the presence/absence of an anger attack within the past year (Table 2). This outcome offers some assurance as to the robustness of the main findings.

The fact that the data were cross-sectional also limits interpretability. Consider, for example, the positive and significant coefficients for binge drinking. At first glance, one might assume that alcohol consumption would result in a loss of emotional control that would lead to anger attacks. However, a study by Coccaro et al. (2016) of the temporal relationship between substance use disorders and IED found the opposite, that IED increased the severity of substance use disorder. Fernandez and Wasan (2009) also point out that anger can be a factor that predisposes, precipitates, exacerbates, or perpetuates pain.

Finally, without biomarker information, it is only possible to speculate on underlying mechanisms that might link experiences of pain with affective aggression. Inflammatory markers such as C-reactive protein and Interleukin-6 have been investigated as potential biological mechanisms. This remains a fertile area for future investigation.

Despite these limitations, this study contributes to research on both episodic anger and pain in important ways. The study focuses on a unique population, former NFL athletes, whose physical and behavioral health have been a source of public interest, and addresses two prominent issues: athlete involvement in physical altercations off the field, and injuries, physical impairment, and pain that afflict players during and beyond their playing years. This study speaks to both sets of concerns.

The finding that pain is related to anger attacks among former NFL athletes suggests directions for future research and implications for practice. First, to expand the generalizability of this study's findings, health research examining other occupational sectors that expose workers to high levels of injury and ensuing pain could include measures of IED or other forms of affective aggression. For example, a report from the Massachusetts Department of Public Health (2018) showed significantly higher rates of fatal opioid-related overdose among individuals working in such occupations. The addition of IED variables to surveys could provide a more nuanced understanding of the struggles faced by individuals who endure injury and pain in the context of making a living. Screening for IED could also aid in the development of more comprehensive assessment tools for identifying family and intimate partner violence (Allsworth, 2018). In clinical settings, conversations with patients about chronic pain can open opportunities to inquire about its impact on emotions, relationships, and family life and offer information about supportive services.

The link between pain and opioid addiction has also called forth a need for non-pharmaceutical forms of

support for those who suffer from pain (Gross & Gordon, 2019). The significant and negative relationships between NFL career duration and anger attacks and weekly religious attendance and anger attacks potentially speak to this need. Protective effects of participation in these institutions might accrue from any number of factors, such as social support, self-esteem, a disciplining of emotions, or the inculcation of positive health habits, life skills, or spiritual practices that might help with pain or anger management (e.g., Davis et al., 2020; Gucciardi et al., 2017). This might especially be the case for men who hesitate to seek out mental health support due the associated stigma, which threatens more traditional masculine identities. For example, Nieuwsma et al. (2014) found that military veterans who distrusted mental health professionals were more likely to seek help from pastoral counselors. Gucciardi et al. (2017) similarly suggested that men might be more open to support that is framed in terms of "mental toughness" rather than "mental health," since the former avoids the stigma associated with seeking help for mental health problems. To the extent that sports and some religious cultures, such as the "muscular Christianity" that developed in tandem with men's sports in the United States (Ladd & Mathieson, 1999), resonate in positive ways with traditional masculine identities, some of their beliefs or practices might serve as valuable resources for men who need help with affective aggression. More broadly, research clarifying mechanisms through which sports and religious participation produce health benefits could aid in the development of alternative pain and anger management strategies outside of these institutions, for athletes and nonathletes alike.

Conclusion

Former NFL athletes have significantly greater odds of experiencing physical pain, and earlier in life, when compared to men in the general population. As such, former NFL players are a population that lends itself to the study of the relationship between pain and behavioral health. The present study showed that former NFL players who experienced major headaches, back pain, and neck pain were more likely than former players who were not in significant pain to experience affective aggression, as manifest in episodic anger attacks. More research is needed to establish the generalizability of this finding, and to develop intervention strategies to improve behavioral health among individuals for whom pain is a chronic condition.

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Authors' Note

Dr. Jackson passed away recently. Robert Turner has offered to receive correspondence intended for Dr. Jackson. Dr. Jackson's research administrator, Diana Armistead, also responds to correspondence intended for him (darmis@umich.edu).

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MAJOR ARTICLE



Sleep problems are associated with academic performance in a national sample of collegiate athletes

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ABSTRACT

Objective: Examine associations between a range of sleep problems and academic performance in a national sample of collegiate athletes. **Participants:** Data were obtained from the National College Health Assessment of US college/university students from 2011–2014 ($N=8,312$ collegiate athletes). **Methods:** Univariate comparisons for all sleep variables and demographics were stratified across GPA, using one-way ANOVA for continuous variables and chi-square for categorical variables. Multinomial logistic regression models, with GPA as outcome (reference = A) and sleep variable as predictor, were examined and adjusted for age, sex, and survey year. Ordinal regression examined a 1-level change in GPA associated with each sleep variable, adjusted for covariates. **Results:** Sleep difficulty was associated with increased likelihood of B/C averages. Initial-insomnia was associated with increased likelihood of B/C, and D/F averages. Tiredness was associated with increased likelihood of B/C, and D/F averages. **Conclusions:** Sleep problems are highly prevalent and associated with poorer academic performance in collegiate athletes.

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Introduction

College is a transformative time when many students first gain the autonomy to make decisions that can adversely impact their health and academic performance. The lack of exercise, binge drinking, poor time management, and unbalanced diets are behaviors that have been shown to negatively affect the well-being of college students.^{1–3} In college, students are often challenged to adjust to new peer groups, social settings, greater access to drugs and alcohol, and increased independence.⁴ In addition to these concerns, poor sleep is a persistent problem experienced by many US college students. For example, a study based on the Pittsburgh Sleep Quality Index (PSQI) reported that up to 60% of college students suffered from poor sleep quality.³ Sleep problems, which include sleep difficulties, initial insomnia, insufficient sleep, daytime tiredness, and sleepiness can worsen the academic performance of college students.^{5,6}

Although sleep problems in college students have been well documented,^{7–9} literature that has interrogated the relationship between sleep problems and academic performance in collegiate athletes is limited. Recently, both the NCAA Inter-association Task Force on Sleep and Wellness and the International Olympic Committee have released consensus statements urging universities and colleges to prioritize sleep health among their athlete populations, as sleep has

significant implications for overall health and athletic and academic performance.^{10,11} The research that presently exists with this population has primarily focused on the effect of athletic performance on sleep quality. For example, previous studies indicate that early morning training sessions and late evening games can offset the intrinsic circadian rhythm and impact the amount of sleep collegiate athletes obtain, further exacerbating sleep problems.^{12–14} Several studies indicate that sleep is critical for cognitive function, proper physiological functioning, mental health and is vital for peak performance in collegiate athletes.^{5,15–21} Practice time, travel, competition, balancing athletics and academics, and student life are factors that may adversely affect sleep in collegiate athletes.^{5,15,22–24} Despite athletic performance problems that result from inadequate sleep, the void in research specifically focused on sleep problems and academic performance hinders athletic departments, coaches, and the NCAA, from collaborating with sleep professional to develop targeted intervention solutions for college athletes.

Understanding the impact of sleep problems on academic performance among collegiate athletes is critical for a number of reasons. Poor academic performance during college can jeopardize athletic participation and academic eligibility, as well as limit employment opportunities and career aspirations after graduation, and lead to lower lifetime wages.^{13,25} Future educational opportunities may suffer if academic

struggles lead to suspension or expulsion from college.^{26,27} Additionally, the economic model of human capital suggests academic struggles in college could jeopardize an athlete's long-term benefits (eg, more fulfilling work environment, increased health, extended life, more informed purchases, and lower rates of unemployment).^{28,29} It is clear that the implications for poor academic performance can last a lifetime.

In the present study, we analyzed data from 8,312 US college/university athletes to understand the effects of sleep problems on academic performance. We hypothesized that sleep problems among collegiate athletes are associated with poorer academic performance and that this relationship exists across multiple domains of sleep problems. Findings from this study will contribute to our understanding of the relationship between various types of sleep problems and academic performance among collegiate athletes. Additionally, these results serve as a call to improve and adopt specific interventions that holistically addresses the type of sleep problems that can lead to poor academic performance in collegiate athletes. To our knowledge, this is the first study to examine this relationship in a nationally representative sample of collegiate athletes.

Previous studies

Academic performance and sleep

The effects of sleep on the academic performance in the general student body population have been well documented.⁶ In a study at a large private university involving a random sample of 200 students living in on-campus residence, Trockel et al³⁰ discovered that wake up times and other poor sleep habits were associated with lower grades. Delayed sleep phase syndrome (DSPS), extending the weekend sleep schedule to the weekday, has been shown to induce impaired academic performance in college students. Among 211 first-year psychology college students, academic performance was consistently lower for those suffering from DSPS, compared to non-DSPS students.³¹ A study that used official grades from the university register showed, students with later wake-up and bedtimes on weekend and weekdays performed poorer academically.³⁰ Various performance parameters (eg, GPA, teacher comments, self-rated average grades) have been used to measure academic performance in college students. Yet, these, and any other measures are rarely employed to examine the sleep-academic performance relationship in collegiate athletes.

Athletic performance and sleep

The NCAA annual academic progress report claims that collegiate athletes perform better than the general student body.³² However, a report from the Pacific Athletic Conference indicates that 71% of student athletes mention athletic commitments as the top activity that prevents them from receiving sufficient sleep; and 77% believe they receive less sleep than their nonstudent athlete peers.²⁴ In a qualitative study of a major college basketball programs, athletes

expressed that physical exhaustion and fatigue from the lack of sleep was a catalyst for the loss motivation to perform academically.¹³ Sleep can easily become compromised as student athletes try to balance the multiple demands on their time. Collegiate athletes with sleep deficiency are at increased risk for acute illnesses, traumatic sport injuries, and the development of chronic diseases.¹² The conclusions of these seemly contradictory outcomes raise the possibility that collegiate sport participation may actually hinder athletes' ability to get adequate sleep, thus negatively impacting their ability to perform up to their academic potential.

Athletic sleep interventions

The sleep intervention literature for athletes has focused almost exclusively on athletic performance and recovery. This thread of research concludes that established sleep assessment and intervention strategies are rendered ineffective in athletes due to the uniqueness of their lifestyles and travel schedules.³³ Given the implications for performance and recovery outcomes, there are many explanations regarding why sub-optimal sleep is a cause for concern in athletes. For instance, subjective feelings of hunger and carbohydrate metabolism are negatively affected by poor sleep.³⁴ Another important factor is that sleep restores cerebral glycogen depleted during waking hours.³⁵ Both cerebral glycogen depletion and feelings of hunger can be detrimental to athletic performance in collegiate athletes. The cognitive consequences of sleep deprivation include heightened fluctuation in mood stability, deficits in fine motor movement, memory (eg, consolidation of motor tasks), and decision-making.³⁶ Overall, current data indicate that sleep loss adversely impacts the physical and cognitive performance of athletes.³³ A systematic review of sleep interventions consisting of 10 studies and a total of 218 athletes ranging between 18–24 years old from various sports (eg, swimming, soccer, basketball, tennis), concluded that sleep extension and napping, sleep hygiene, and postexercise recovery strategies were the most effective methods to improve performance and/or recovery outcomes in athletes. While these intervention strategies have produced clear benefits in athletic performance and recovery,^{36,37} the lack of empirical research focused on sleep and academic performance hinders our ability to develop effective intervention methods in this area.

Methods

Data source

Data were obtained from the National College Health Assessment II, an annual survey of US college/university students conducted by the American College Health Association. This health assessment is the largest known data set examining the health of college students in the United States. Complete information about this dataset is available online.^{15,24,25} Data from 2011–2014 were used, since these years included identical questions. Surveys are administered on paper, in person, and online across a number of college campuses each year. Different campuses are

included each year, and thus, years can be combined (as there is no duplication). Institutions are kept anonymous in the survey, as are individuals, in order to promote honest reporting. Previous information about the generalizability, reliability, and validity of the data set is available, reporting a median response rate of 19%.¹⁷ A total of $N=112,849$ students participated during these years. Of these, $N=8,683$ identified themselves as varsity athletes. Approval from a university IRB was received prior to analyzing and reporting the data. For a thorough description of the data source, please refer to the method's section of a manuscript published by Hosik Min.³⁸

Measures

Sleep difficulties. Participants were asked, "Within the past 12 months, have any of the following been traumatic or very difficult for you to handle?" This was followed by a list of conditions, including "sleep difficulties." Those who answered "yes" to this question were coded to have sleep difficulties.

Initial insomnia. Participants were asked, "In the past 7 days, how often have you had an extremely hard time falling asleep?" Responses were recorded as 0–7. Those who reported this problem at least 3 days per week were coded "yes" for initial insomnia.

Insufficient sleep. Individuals were asked, "On how many of the past 7 days did you get enough sleep so that you felt rested when you woke up in the morning?" Responses were coded as 0–7 and recoded to reflect nights per week of insufficient sleep (eg, 0 = 7, 1 = 6, etc).

Daytime tiredness. Participants were asked, "In the past 7 days, how often have you felt tired, dragged out, or sleepy during the day?" Responses were recorded as 0–7. Those who reported at least 3 days per week were coded "yes" for daytime tiredness.

Sleepiness. Daytime sleepiness was assessed with the following item, "People sometimes feel sleepy during the daytime. In the past 7 days, how much of a problem have you had with sleepiness (feeling sleepy, struggling to stay awake) during your daytime activities?" Response choices were "No problem at all," "little problem," "More than a little problem," "A big problem," and "A very big problem." Those who reported sleepiness as being at least "a big problem" were coded "yes" for sleepiness.

Sleep difficulties interfering with academics. Participants were asked, "Within the past 12 months, have any of the following affected your academic performance?" One of the options was "sleep difficulties." Response options were, "This did not happen to me" (reference group), "I have experienced this issue, but my academics have not been affected," or one of four potential ways this could have interfered with academics, including a lower grade on an examination or project, a lower course grade, an incomplete or dropped course, and/or significant disruption in thesis, dissertation, research, or practicum work. Those who endorsed any of these four outcomes were coded as "Yes" for sleep difficulties interfering with academics.

Self-Reported Grade Point Average (GPA). Participants were asked, "What is your approximate cumulative grade average?" Response options included A, B, C, or D/F.

Covariates. All analyses were adjusted for age, sex, and survey year.

Statistical analyses

All variables were examined visually for outliers and implausible values. Descriptive statistics were computed for all variables. Univariate comparisons for all sleep variables and demographics examined were stratified across GPA, using one-way ANOVA for continuous and chi-square for categorical variables. To evaluate whether sleep problems were associated with decreased academic performance, multinomial logistic regression models, with GPA as outcome (reference = A) and sleep variable as predictor were examined, adjusted for age, sex, and survey year. Also, ordinal regression examined a 1-level change in GPA associated with each sleep variable, adjusted for covariates. Since insufficient sleep was assessed as number of days per week, a *post hoc* sensitivity analysis repeated the multinomial logistic regression analyses using each available cut point (eg, at least 1 day/week, at least 2 days/week, etc). All analyses were performed with STATA 14.0,³⁹ with $p < 0.05$ indicating statistical significance.

Results

Characteristics of the sample

Data from $N=8,683$ student athletes between 2011 and 2014 were aggregated. Characteristics of the sample are reported in Table 1. Overall, about 20% of the sample reported sleep difficulty, about 22% reported initial insomnia, about 61% reported daytime tiredness, about 16% reported daytime sleepiness, 28% reported that they experienced sleep problems, but this did not interfere with academics and 18% reported sleep problems that interfered with academics. About 57% of the sample reported insufficient sleep on four nights per week or more.

When results were stratified across GPA groups (reported in Table 1), significant differences were found for age (higher age in D/F students), sex (greater percentage of women with higher grades), and all sleep disturbances.

Table 2 reports correlations between the sleep variables. Although these variables represent overlapping constructs, they correlate with each other only moderately.

Sleep disturbances associated with academic performance

Results of adjusted regression analyses examining relationships between sleep disturbances and academic performance are reported in Table 3. The presence of sleep difficulty was associated with a 47% increased likelihood of a B average, compared to an A. Also, sleep difficulty was associated with a 118% increased likelihood of a C average and a 111%

Table 1. Characteristics of the sample and stratification by academic performance.

Variable	Characteristics	Complete sample	A	B	C	D/F	p-value
N		8,312	3,192	4,381	706	33	***
Age	Years	19.7 ± 1.8	19.6 ± 1.9	19.6 ± 1.7	19.9 ± 1.9	21.5 ± 4.1	***
Sex	Man (%)	38.52	34.20	40.01	48.20	55.56	***
	Woman (%)	61.48	65.80	59.99	51.80	44.44	***
Race/Ethnicity	Non-Hispanic White (%)	75.00	79.51	74.50	58.78	51.52	***
	Black/African-American (%)	4.74	2.26	5.27	12.46	9.09	***
	Hispanic/Latino (%)	6.54	4.61	7.24	11.05	6.06	***
	Asian (%)	4.81	6.20	4.04	3.54	***	***
	American Indian/Alaskan Native (%)	1.42	1.25	1.53	1.56	***	***
	Other	6.00	4.54	6.09	10.91	30.30	***
Year	2011 (%)	25.83	25.47	26.11	25.35	33.33	
	2012 (%)	26.79	24.91	28.21	26.77	21.21	
	2013 (%)	28.89	30.76	27.64	28.33	24.24	
	2014 (%)	18.49	18.86	18.03	19.55	21.21	
Institution type	Public (%)	31.60	27.91	31.68	46.88	51.52	***
	Private (%)	68.40	72.09	68.32	53.12	48.48	***
Sleep-difficulty	Yes (%)	19.80	15.85	21.25	28.04	33.33	***
Initial-insomnia	Yes (%)	21.78	18.24	22.77	30.53	45.45	***
Tiredness	Yes (%)	60.90	57.59	62.25	67.05	69.70	***
Insufficient-sleep	Days /Week	3.83 ± 1.85	3.65 ± 1.85	3.89 ± 1.82	4.24 ± 1.90	4.36 ± 2.51	***
Sleepiness	Yes (%)	32.75	29.53	34.01	39.29	36.36	***
Interfere	Did not experience (%)	54.64	57.17	53.51	51.01	36.36	***
	Did not affect (%)	27.65	31.07	26.80	17.67	21.21	***
	Affected (%)	17.71	11.76	19.68	31.32	42.42	***

*p < 0.05, **p < 0.01, ***p < 0.001.

Table 2. Spearman correlations among sleep variables.

	Sleep-difficulty	Initial-insomnia	Tiredness	Insufficient sleep	Sleepiness
Initial-Insomnia	0.3731				
Tiredness	0.2176	0.1981			
Insufficient Sleep	0.2538	0.1982	0.4636		
Sleepiness	0.2652	0.1626	0.313	0.3618	
Interfere	0.4254	0.3238	0.2515	0.2492	0.2448

Table 3. Associations between sleep disturbances and GPA, relative to likelihood of being an "A" student.

Variable	B Grade		C Grade		D/F Grade		
	RRR	95% CI	RRR	95% CI	RRR	95% CI	
Sleep-difficulty	Yes	1.46***	(1.29, 1.65)	2.11***	(1.73, 2.57)	1.96	(0.81, 4.74)
Initial-insomnia	Yes	1.35***	(1.20, 1.51)	2.04***	(1.68, 2.47)	3.53**	(1.59, 7.80)
Tiredness	Yes	1.27***	(1.15, 1.40)	1.73***	(1.45, 2.07)	3.11*	(1.23, 7.89)
Sleepiness	Yes	1.23**	(1.08, 1.40)	2.03***	(1.64, 2.50)	3.43**	(1.45, 8.12)
Insufficient-sleep	Per Day	1.08***	(1.05, 1.10)	1.21***	(1.15, 1.26)	1.21	(0.98, 1.49)
Interfere	Not experienced	Reference		Reference		Reference	
	Not affected	0.96	(0.86, 1.07)	0.71**	(0.57, 0.90)	1.25	(0.45, 3.51)
	Affected	1.90***	(1.66, 2.19)	3.36***	(2.72, 4.15)	5.37***	(2.18, 13.19)

Note. RRR = relative risk reduction; CI = confidence interval.

*p < 0.05, **p < 0.01, *** p < 0.001.

increased likelihood of a D/F average. Initial insomnia was associated with a 35% increased likelihood of a B average, a 108% increased likelihood of a C average, and 271% increased likelihood of a D/F average. Daytime tiredness was associated with a 25% increased likelihood of a B average, a 62% increased likelihood of a C average, and a 190% increased likelihood of a D/F average. Daytime sleepiness was associated with a 22% increased likelihood of a B average, a 103% increased likelihood of a C average, and a 242% increased likelihood of a D/F average.

Results of analyses examining insufficient sleep are reported in **Table 4**. Insufficient sleep was associated with an 8% increased likelihood of a B average for each day of insufficient sleep. Similarly, insufficient sleep was associated with a 21% increased likelihood of both a C and D/F average for each day reported. Results of a sensitivity analysis,

testing each cutoff, are reported in **Table 3**. Irrespective of cutoff used, increased likelihood of a B average (vs A) was 24–40%, depending on the cutoff used. For increased likelihood of a C average, a cutoff of 1 or more days per week was not associated with increased risk, but all other cutoffs were, with an increased likelihood between 64% and 100%, depending on the cutoff used. For increased likelihood of a D/F average, a significant relationship was only seen for a cutoff of 6 or more days per week (132% increased likelihood) and 7 days per week (328% increased likelihood).

The perception of there being a sleep problem that interfered with academics was associated with actual reported lower GPA, as shown in **Table 3**. Compared to those who reported that they did not have a sleep problem, those who reported that they experienced sleep difficulties that did not interfere with academic performance were at no increased

Table 4. Associations between days of insufficient sleep and academic performance.

Insufficient-sleep	B		C		D/F	
	RRR	95% CI	RRR	95% CI	RRR	95% CI
0 Days	1.000	Reference	1.000	Reference	1.000	Reference
1 or more days	1.34*	(1.06, 1.70)	1.25	(0.83, 1.89)	0.49	(0.14, 1.71)
2 or more days	1.41***	(1.22, 1.64)	1.70***	(1.27, 2.28)	1.30	(0.38, 4.43)
3 or more days	1.27***	(1.15, 1.41)	1.74***	(1.42, 2.14)	1.55	(0.61, 3.90)
4 or more days	1.25***	(1.14, 1.37)	1.82***	(1.53, 2.18)	1.84	(0.81, 4.18)
5 or more days	1.24***	(1.13, 1.37)	1.96***	(1.65, 2.33)	1.79	(0.82, 3.91)
6 or more days	1.24***	(1.10, 1.40)	1.99***	(1.64, 2.42)	2.29	(0.98, 5.35)
7 days	1.25*	(1.05, 1.48)	1.82***	(1.39, 2.39)	3.97**	(1.56, 10.12)

Note. RRR = relative risk reduction; CI = confidence interval.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Table 5. Associations between sleep disturbances and likelihood of a lower grade.

Variable	OR	95% CI
Sleep-difficulty	1.56***	(1.40, 1.74)
Initial-insomnia	1.50***	(1.35, 1.67)
Tiredness	1.36***	(1.25, 1.49)
Sleepiness	1.44***	(1.28, 1.62)
Insufficient-sleep	1.11***	(1.08, 1.13)
Interfere	Not experienced	1.00 Reference
	Not affected	0.90* (0.82, 1.00)
	Affected	2.15*** (1.91, 2.43)

Note. OR = odds ratio; CI = confidence interval.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

likelihood for lower GPA (and showed decreased likelihood for a C average). On the other hand, when students reported that sleep problems interfered with academics, they were 87% more likely to have a B, 216% more likely to have a C, and 428% more likely to have a D/F.

A *post hoc* analysis used ordinal regression to examine whether the presence of sleep disturbance was associated with a change in GPA level, irrespective of actual value. Results of this analysis are reported in Table 5. Overall, sleep difficulty was associated with a 60% increased likelihood of reduced GPA by one grade level, initial insomnia was associated with a 52% increased likelihood of a lower grade level, daytime tiredness was associated with a 32% increased likelihood of a lower grade, and daytime sleepiness was associated with a 44% increased likelihood of a lower grade. Each day of insufficient sleep was associated with an 11% increased likelihood of a lower grade. Having a sleep problem that did not appear to interfere with academics was associated with an 11% likelihood of increasing a grade level compared to those who did not have a sleep problem. However, those with a sleep problem that seemed to interfere with academics, had a 111% increased likelihood of having a reduced grade, compared to those with no sleep problem to report.

Discussion

The present study assessed if sleep problems of collegiate athletes were associated with academic performance. It was hypothesized that sleep problems would indicate an association with lower GPA. The primary findings of this study determined there was a likelihood of collegiate athletes with

sleep problems to earn a lower GPA compared to earning an A average or the highest GPA. Reported sleep problems across multiple domains, including sleep difficulties, insufficient sleep, insomnia, and daytime tiredness, were highly predominant and associated with poorer self-reported GPA in collegiate athletes.

When asked if a sleep problem interfered with academics, of those students who responded positively, 87% were more likely to report a B average, 216% more likely to report a C average, and 428% more likely to report a D/F average. To the best of our knowledge, these associations have not been previous examined in collegiate athletic population data. While previous studies have examined the relationship between quality of sleep and athletic performance, few relate sleep to academics, and even fewer have associated sleep problems with self-reported GPA among collegiate athletes.

A noteworthy finding in the present study is that collegiate athletes who reported having a sleep problem that did not appear to interfere with academics was associated with an 11% likelihood of increasing a grade level compared to those who did not report having a sleep problem. Although it is difficult to identify the specific reason(s) for this outcome, perhaps the perception of a sleep problem causes certain collegiate athletes to adopt effective sleep strategies or study habits that increase academic performance. Conversely, those who reported a sleep problem that appeared to interfere with academics had a 111% increased likelihood of having a reduced grade, compared to those with no sleep problem to report. These findings are consistent with a study of undergraduate college students that concluded the quality, irregularity, and deprivation of sleep were significantly associated with two measures of academic performance.⁴⁰ Given the unique vulnerability of athletes, this outcome suggests that interventions specifically designed for athletes are required. The present results suggest the relationship between sleep problems and academics in collegiate athletes is strong and significant.

Competing athletic and academic demands and rigid scheduling are factors of the collegiate athlete experience that differ most in comparison to nonathlete college students. Traditional college students are generally able to determine and manage their own academic and work schedules, whereas athletes have coaches and advisors who pretermine and monitor their daily routines. In addition to course work and athletic practices, collegiate athletes have

mandatory academic monitoring appointments and tutoring sessions.⁴¹ Additionally, collegiate athletes are restricted from being part-time students. According to NCAA regulations, athletes must be enrolled in at least 12 credit hours per semester to maintain NCAA eligibility.⁴² Finally, collegiate athletes regularly travel for competitions. Extensive travel means athletes often miss classes and exams, which can lead to tensions between students and faculty. Collegiate athletes report being negatively perceived by professors, failing to receive accommodations for athletic competitions, and being openly criticized in the classroom.^{43–45}

A 2003 survey of NCAA institutions revealed that 69.2% employed a maximum of three academic advisors who have been previously trained to understand NCAA rules.⁴⁶ Often, employees designated to support collegiate athletes rely on general campus support systems that have limited knowledge of the challenges faced by this unique population. When collegiate athletic department employees serve as the primary resource responsible for academic progress, athletes may not receive the proper guidance and support needed to address sleep problems.

Solutions

One strategy that may help to enhance collegiate athlete's well-being is to ensure that coaches and athletic department administrators are aware of the empirical evidence that highlights the relationship between sport participation, sleep problems, and academic performance. A study by Watson reveals that collegiate athletes may feel uncomfortable seeking help outside of the athletic department because other members of the university do not understand the needs associated with being an athlete.⁴⁷ Academic support centers for collegiate athletes have been found to primarily prioritize keeping athletes eligible, rather than ensuring academic success and competitive postgraduate opportunities.⁴⁸ Because sleep problems are pervasive among collegiate athletes, university athletic departments should consider employing full-time practitioners to educate coaches and develop research-based interventions that can help guard against declines in academic performance. Having full-time sleep professionals on staff can signal to athletes and coaches alike that sleep health and academic performance are integral components of an athlete's overall well-being.

University mental health counselors may serve as a second layer of support that may advocate for the sleep needs of collegiate athletes. Collegiate athletes underutilize university counseling services,⁴⁹ due to fear of stigmatization by coaches, teammates, peers, and fans,⁵⁰ and because counselors do not understand their unique experiences, pressures, and needs.⁵¹ Broughton and Neyer⁵² reveal that counselors can improve relationships with their collegiate athletes by increasing their knowledge of sports. Advanced knowledge and understanding of athletic life may allow a counselor to more comprehensively address sleep and other issues faced by this population.⁵³

Limitations

There are several limitations in this study that should be considered when interpreting the results. First, this study utilized a cross-sectional design which precludes an inference of causality. The relationships drawn are not causal, rather they are associative. Second, this study used self-reported GPA data, which are at risk of manipulation by the athlete. Athletes may not be accounting for their current semester of work. Measures of sleep were self-reported, rather than collected by a validated survey instrument. Participants could have attributed or misattributed a health issue to a sleep problem. Participants did not receive consultation with a sleep expert before reporting their complaints. Additionally, the sample of study participants was not random. This was a voluntary survey taken by students from various colleges and universities. Another limitation of the data is the inability to determine the Division level (I, II, III) of the athletes. The time constraints placed on athletes may vary by NCAA Division and type of sport (eg, football, basketball, field hockey), which, may cause variation in reported sleep problems. Lastly, there is no objective measure of cognitive performance that could mediate the relationship of variation between collegiate athletes. Despite these limitations, this study is a significant contribution to our understanding of various types of sleep problems experienced by collegiate athletes and the relationship to academic performance.

Conclusion

At this time, there is little known about the association between sleep problems and academic performance among athletes. The results of our study reveal that athletes who report sleep problems are more likely to be academically disadvantaged. This finding has implications for college and sport administrators, coaches, and individual and team performance. Collegiate athletes require quality sleep in order to perform at a high level athletically and academically.^{15,24,54–57} While adjusting practice times, or limiting travel time to sporting events may be impractical, college coaches and administrators should work with sleep professionals that understand collegiate athletics to actively pursue strategies for athletes to attain the highest quality sleep possible.

As reported elsewhere,⁵⁸ the lack of awareness among key stakeholders (eg, collegiate athletes, coaches, parents, administrators) regarding the importance of sleep and the scheduling constraints could lead to insufficient and poor reported sleep in athletes. Implementing techniques to educate collegiate athletes and other key stakeholders regarding the quality, quantity, and environment of sleep could benefit academic performance. Computing technologies (eg, smart-phone applications, web-based video instruction, and online podcast) should also be explored to support healthy sleep behaviors and improve academic performance in current and future generations of collegiate athletes.⁵⁹ Further research is required to understand the intricacies and impact of sleep on the academic performance of collegiate athletes.

Future studies should examine the relation between sleep and other factors such as drug and alcohol use, social jet lag, and rigor of course work, and how they independently and collectively impact the academic achievement of collegiate athletes.

Coaches, teammates, friends, parents, and other university personnel need to be educated in order to maximize the support they can provide to collegiate athletes. Future research also needs to consider how to train collegiate athletes to advocate for the support needed to be able to succeed personally, professionally, and academically.

Conflict of interest disclosure

The authors have no conflicts of interest to report. The authors confirm that the research presented in this article met the ethical guidelines, including adherence to the legal requirements, of the United States and received approval from the Institutional Review Board of the University of Arizona.

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ORIGINAL ARTICLE

Chronotype and social support among student athletes: impact on depressive symptoms

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ABSTRACT

Previous studies have shown individuals with evening chronotype to have a greater likelihood for depression (self-reported and clinical ratings), especially in young adults. However, the mechanisms for this relationship remain unknown. Low levels of social support may be a plausible mechanism: young adults with evening chronotypes are awake when others are sleeping, which may lead to feelings of isolation or low support. This study examined links between chronotype, depression, and social support in relationship subtypes within a group of university student athletes. Data were obtained from 189 NCAA Division-I student athletes across all sports. Chronotype was assessed with the Circadian Energy Scale and ranged from -2 (definitely morning type) to +2 (definitely evening type). Depressive symptoms were assessed with Center for Epidemiological Studies Depression scale. Social support was assessed with the Multidimensional Scale of Perceived Social Support, which included subscales for Family, Friends, and Significant Other. A subscale for Team was created using the items from the Friends subscale (changing the word “friends” to “teammates”). Regression analyses adjusted for age, sex, and minority status. More evening chronotype was associated with higher reported depressive symptoms ($p = .018$), lower overall perceived social support ($p = .001$), and lower perceived social support specifically provided by family ($p < .0001$), friends ($p < .0001$), and teammates ($p = .014$). However, more evening chronotype was associated with higher depressive symptoms for higher, but not lower perceived social support from significant other. Moreover, chronotype-by-support interactions on depressive symptoms were observed; the statistical relationship between chronotype and depression was evident only in those with low (but not high) social support from friends and teammates. These data suggest that having a more evening chronotype may be associated with social isolation, and decreased opportunities for interactions with friends and teammates. This may contribute to the long-standing circadian association seen with depression in college student-athletes. Interventions aimed at increasing university support networks may reduce the impact of depression in students self-identifying with later chronotypes and sleep schedules.

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Introduction

Circadian rhythms broadly dictate human sleep and wakefulness/activity patterns across the day. However, individual preferences for timing of activities can vary significantly within the circadian day, a phenomenon often described as a chronotype. Chronotypes can range for an individual’s activity late at night (late chronotype), early in the morning (early chronotype), or afternoon (intermediate or neutral chronotype). This creates a temporal “phase” relationship between our

internal circadian rhythms and the external world, thereby influencing the timing of our sleep (Levandovski et al. 2013). Chronotype appears to differ across age groups in substantial ways (Fischer et al. 2017; Roenneberg et al. 2004). During adolescence, a shift toward eveningness is seen (Roenneberg et al. 2004). Additionally, late chronotype has been shown to be more common among young adults between ages 18–20, with chronotype scores decreasing (indicating a shift toward earlier chronotype) from there-on

throughout older adulthood. Studies have also shown that chronotype differs by gender, with men exhibiting slightly later chronotype than women (Fischer et al. 2017; Randler and Engelke 2019).

One's chronotype impacts daytime functioning, including academic performance among adolescents (Preckel et al. 2013), daytime sleepiness (Chan et al. 2020), and work-related fatigue (Martin et al. 2012). Furthermore, chronotype may be associated with a variety of physical and mental health domains, such as digestion/metabolism (Kiyotoki et al. 2021) and cardiovascular health (Makarem et al. 2020), as well as depression (Kim et al. 2020; Kivela et al. 2018) and anxiety (Silva et al. 2020). The chronotype-health connection might be mediated through its effects on sleep; for example, both earlier and later chronotypes experience more insomnia symptoms (e.g., difficulty falling asleep for later chronotypes and early morning awakenings for earlier chronotypes) and worse sleep quality (Raman and Coogan 2020).

Depression is an especially important topic when studying mental health in young adults. Approximately 13.1% of young adults (ages 18–25) in the U.S. had at least one prior depressive episode in 2017 (CBHSQ CfBHSaQ 2018). Later chronotype has been associated with depressive mood/symptoms in healthy adults (Hidalgo et al. 2009), and increased depressive symptoms in individuals with a formal diagnosis (Antypa et al. 2017, 2016; Gaspar-Barba et al. 2009; Kim et al. 2020). Gaspar-Barba and colleagues assessed depressive symptoms using the Hamilton Rating Scale for Depression (HRSD) and chronotype using the Morning-Eveningness Questionnaire (MEQ) in 100 participants (Gaspar-Barba et al. 2009). They found that late chronotype was not only associated with increased suicidal thoughts, but also with impairment in activities, paranoia, and higher scores on the anxiety cluster of the HRSD (Gaspar-Barba et al. 2009). Furthermore, Antypa and colleagues showed that rumination was a primary psychological risk factor in the associations between late chronotype and depression (Antypa et al. 2017). Some studies suggest there may be neurobiological and genetic mechanisms that contribute to this relationship, as variants in circadian genes including CRY1 and NPAS2 (Soria et al. 2010) as well as TIMELESS (Park et al. 2019) have been linked to depression. However, another study suggests that chronotype and depressive symptoms, particularly in university students, may have different genetic underpinnings (Jankowski and Dmitrzak-Weglarcz 2017). Therefore, it is important to also consider the environmental and behavioral factors that may influence the relationship.

Suicide fatality is often linked to depression (CDC CfDCaP 2018). Suicide risk is greater for those with insufficient sleep (Baiden et al. 2020; Khader et al. 2020) and more likely to occur at night (Perlis et al. 2016; Persons et al. 2019; Tubbs et al. 2021, 2020b), thus increasing risk for those with late chronotype owing to both increased depression and late-night seclusion. This further signifies the importance of assessing chronotype among university student athletes, who are at higher risk of suicide (CDC CfDCaP 2018).

Student athletes are a particular population where the relationship between chronotype, social support, and depression have not been fully explored. Beyond the common factors that are associated with being a young adult and college student, the additional stressors that come along with being a student athlete are areas of relevance. Notably, student athletes are more likely to experience stress related to increased responsibilities and lack of time (Stevens et al. 2000). Interestingly, while some studies have shown that student athletes are less likely to exhibit depressive symptoms compared to their non-athletic student counterparts (Armstrong et al. 2015; Proctor and Boan-Lenzo 2010), another study has suggested that depression may still be comparably prevalent in student athletes compared to non-athletes (Wolanin et al. 2016). Furthermore, social support has been shown to play a role in depressive symptoms in student athletes, particularly for female student athletes (Hagiwara et al. 2017). Additionally, student athletes have been shown to have poor sleep, including insufficient sleep duration, poor sleep quality, and daytime sleepiness, all of which may have negative implications in health and performance (Mah et al. 2018). To date, however, chronotype has not been a topic thoroughly examined in student athletes. Elucidating the extent to which perceived social support serves as a mechanism for the association between more evening chronotype with depressive symptoms could provide a novel, modifiable target to ameliorate negative mood in at-risk college athletes.

Social isolation – and the subsequent feelings of loneliness that it can offer trigger – are also known to influence the course of depression (Cacioppo et al. 2006; Erzen and Cikrikci 2018). As such, social isolation occurring at night may be a particularly strong factor driving the relationship between chronotype and depression. This is important because those with later chronotypes may be more interested in engaging at night, while others are not (Randler 2019), which may lead to subsequent feelings of loneliness and depressive symptoms. While social isolation is commonly defined as infrequent social contacts, feelings of social isolation, which could be related to perceived social support, may

in itself be considered a risk factor for the development of depression (Erzen and Cikrikci 2018). However, it can also be said that perception of low social support may be a consequence of depression, which may further lead to feelings of loneliness. Late chronotype has been commonly associated with social jetlag (Tassino et al. 2016), defined as a disparity between weekday and weekend sleep midpoints, presumably driven by social/environmental demands that produce advanced and/or shortened sleep during weekdays and delayed/lengthened sleep on weekends (Wittmann et al. 2006). While social “mistiming” may precipitate a lack of engagement opportunities and low social support from others, the association between chronotype and perceived social support has not been thoroughly examined.

The present study sought to determine whether the relationship between chronotype and depression in student athletes is influenced by the presence or absence of perceived social support from family members, friends, significant other, and teammates. The overall hypothesis was that the relationship between chronotype and depression can be partially explained by low perceived social support. The specific hypotheses were that: (1) later chronotype would be associated with increased depressed mood; (2) later chronotype would be associated with less social support overall and lack of perceived support specifically provided by family, friends, significant other, or teammates; (3) there would be a chronotype by social-support interaction on depression, such that the relationship between later chronotype and depression would be stronger in the case of lower social support; and (4) there would be a significant partial mediation of the relationship between chronotype and depression by social support.

Methods

Participants

Data were obtained from N = 189 NCAA Division-I male and female student athletes during the first two weeks of the Fall 2016 semester. Participants included athletes engaged in a variety of sports. All students participated in only one sport, were specifically recruited from the athletics department by flyers, and were at least 18 years of age. The selection of participants favored students who were returning compared to first-year students. All data were obtained via questionnaires which were completed electronically either at home or on a tablet at the athletics facility. Informed consent was obtained from participants, and participants were entered into a raffle for compensation. This study was conducted in accordance with international standards for ethical treatment of human

subjects (Portaluppi et al. 2010), was performed in accordance with the Declaration of Helsinki, and was approved by the Institutional Review Board of the University of Arizona (#1508051282).

Measures

Chronotype was assessed using the Circadian Energy Scale (CIRENS), which has been previously validated to assess chronotype independent of sleep schedule, including in college students (Ottoni et al. 2011, 2012). This measure was chosen because student athletes were likely to have inconsistent or unusual sleep schedules, and the CIRENS is capable of assessing chronotype independent of sleep schedule. This scale consists of three introspective questions, including an optional question, assessing energy level (very low, low, moderate, high, or very high, scored 1–5) in the morning, evening, and afternoon. The total summed score ranged from 3–15. Chronotype was then determined by subtracting morning energy level score from evening energy level score. The spectrum of the CIRENS chronotype score ranged from -4 (definitely morning type) to +4 (definitely evening type). Because chronotype exists along a continuum, CIRENS scores were analyzed as a continuous variable rather than categorically.

Depressive symptoms were assessed using the Center for Epidemiological Studies Depression (CESD) scale (Radloff 1977). The CESD is a validated tool used to screen for depression in the general population (Vilagut et al. 2016), including young adults (Radloff 1991). The CESD is a 20-item measure that asks to rate how often over the past week participants experienced symptoms associated with depression (i.e. restless sleep, poor appetite, feeling lonely). Responses range from 0–3 for each item (0 = rarely or none of the time, 1 = some or little of the time, 2 = moderately or much of the time, 3 most or almost all the time). Scores in the CESD range from 0–60 with a cutoff score of 16 that may identify those at risk for depression. Scores were evaluated as a continuous variable rather than using cutoffs for clinical significance since this was not a clinical sample and would likely reflect variability within a non-clinical range.

Social support was assessed with the Multidimensional Scale of Perceived Social Support (MSPSS) (Zimet et al. 1990), which is a validated scale (Osman et al. 2014) that includes 12 items with subscales for family, friends, and significant-other. A 3-item subscale for teammates was created using the items from the Friends subscale (by simply changing the word “friends” to “teammates” for all the items). Total scores for each subscale ranged from 4–28, and are summed for a total score with a possible range of 16–112. For secondary analyses, MSPSS scores

were categorized as “low” and “high” using median splits (median scores were 87 for total score and 24, 23, 24, and 16 for friends, family, significant other, and teammate subscales, respectively). MSPSS scores were categorized, rather than reported as continuous variables, because perceived social support scores tend to skew toward high support, which would give excess weight to higher scores, and make it more difficult to evaluate relationships with low support. Covariates included self-reported age, gender, and racial/ethnic minority status.

Statistical analyses

Figure 1 depicts the overall structure of the analyses that are reflected in the hypotheses: (1) later chronotype would be associated with higher levels of depressive symptoms; (2) later chronotype would be associated with lower overall social support as well as lower perceived social support from friends, family, and teammates; (3) there would be a significant interaction between chronotype and depression when stratified by social support; and (4) the relationship between chronotype and depression would be partially mediated by social support. Descriptive statistics for all study variables were generated: continuous variables were described using means and standard deviations, while categorical variables were described using count and percentage values.

To determine whether chronotype was associated with depression and total and subscale social support variables, linear regression analyses were conducted and adjusted for covariates, with CESD total score, MSPSS total score, and MSPSS subscale scores as outcomes and CIRENS chronotype score as predictor (Table 2). Then, chronotype-by-social support interaction was computed to evaluate whether the relationship between chronotype and depression depended on high-versus-low social support. Subsequent regression analyses examined relationships between chronotype and depression stratified by low/high social support (Table 3). To determine whether the relationship between chronotype and depression was partially mediated by social support, standard mediation

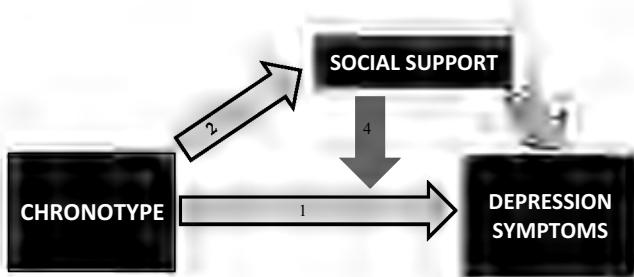


Figure 1. Graphical depiction of study hypotheses.

analyses were performed, examining direct and indirect effects (Table 4). Sobel tests examined whether the proportion of mediation significantly differed from zero. All regression results are displayed as unstandardized coefficients (B) and 95% confidence intervals (CI). *P* values < .05 were considered statistically significant and should be interpreted in the context of the confidence intervals. All analyses were computed using STATA 14.2 (STATAcorp, College Station, TX).

Results

Characteristics of the sample

The characteristics of the sample are shown in Table 1. The mean age of the participants was 19.3 years (*SD* = 5.0). There were 46% females, and most participants were identified as not being from a racial/ethnic minority group. The mean chronotype score indicated neither morning nor evening type, and mean CESD score was 10.8 (*SD* = 7.5), indicating mild depressive symptoms. The average MSPSS total score was 83.4

Table 1. Characteristics of the Sample.

Variable	Units/Category	Value*
Age	Years	19.3 [5.0]
Sex	Male	53.97%
	Female	46.03%
Racial/Ethnic Minority Status	Yes	40.74%
	No	59.26%
Chronotype Score	Range -4 to +4	-0.2 [1.3]
CESD Total Score	Points	10.8 [7.5]
MSPSS Total Score	Points	83.4 [17.2]
MSPSS Family Score	Points	23.4 [5.3]
MSPSS Friends Score	Points	22.2 [5.3]
MSPSS Significant Other Score	Points	22.2 [5.9]
MSPSS Team Score	Points	15.5 [4.8]

*Values represented as percentage or mean ± SD.

CESD: Center for Epidemiological Studies Depression.

MSPSS: Multidimensional Scale of Perceived Social Support.

Table 2. Results of regression analyses examining the role of chronotype score (as continuous variable) and outcomes, including depression and social support.

Variable	B	SE*	95% CI **	p***
CESD Depression Score	1.007	0.422	(0.175, 1.839)	.018
MSPSS Social Support	-3.313	0.959	(-5.204, -1.422)	.001
MSPSS Social Support (Family)	-1.235	0.295	(-1.818, -0.653)	<.0005
MSPSS Social Support (Friends)	-1.062	0.295	(-1.644, -0.479)	<.0005
MSPSS Social Support (Significant Other)	-0.340	0.339	(-1.009, 0.329)	.317
MSPSS Social Support (Teammates)	-0.676	0.272	(-1.213, -0.139)	.014

*SE: Standard Error.

**CI: Confidence Interval.

****p* < .05 is considered statistically significant.

CESD: Center for Epidemiological Studies Depression.

MSPSS: Multidimensional Scale of Perceived Social Support.



Table 3. Relationships between chronotype score as independent variable and depression as outcome, stratified by social support as a moderating variable.

Variable	Low Support					High Support				
	N	B	SE*	95% CI**	p***	N	B	SE*	95% CI**	p***
Total	87	0.776	0.698	(-0.613, 2.165)	.27	102	0.504	0.480	(-0.448, 1.456)	.296
Family	85	1.071	0.714	(-0.350, 2.491)	.138	104	0.341	0.487	(-0.626, 1.308)	.486
Friends	89	1.413	0.704	(0.014, 2.812)	.048	100	0.005	0.504	(-0.996, 1.006)	.992
Significant Other	110	-0.162	0.578	(-1.309, 0.984)	.78	79	2.311	0.603	(1.108, 3.513)	<.0005
Teammates	85	1.649	0.675	(0.306, 2.992)	.017	104	0.128	0.487	(-0.839, 1.096)	.793

*SE: Standard Error.

**CI: Confidence Interval.

***p < .05 is considered statistically significant.

Table 4. Partial mediation analysis.

Mediator Variable	Indirect	Direct	%Mediation	Sobel Test	p_sobel
Total Score	0.626	0.381	0.621	3.044	.002
Family	0.657	0.350	0.652	3.313	.001
Friends	0.593	0.414	0.589	3.050	.002
Significant Other	0.082	0.925	0.081	0.939	.348
Teammates	0.464	0.543	0.461	2.328	.020

(SD = 17.2), with mean subscale scores of 23.4 (SD = 5.3), 22.2 (SD = 5.3), 22.2 (SD = 5.9), and 15.5 (SD = 4.8) for family, friends, significant other, and teammates, respectively. The distribution of chronotype scores is reported in Figure 2. The distribution was generally symmetrical, though with a mean value of -0.19 (SD = 1.27), this represents a slight skew toward morningness.

Chronotype score, depression, and social support

Results of adjusted linear regression models examining the role of comparing chronotype score as a continuous predictor variable for CESD score, MSPSS total score, and MSPSS subscale scores as outcomes, are reported in Table 2. Each row of Table 2 represents a separate regression model. Overall, higher CESD depression score was associated with a later chronotype score ($P = .018$). Regarding social support, those with later chronotype scores were also more likely to have lower total perceived social support scores ($P = .001$), as well as

lower subscale scores for family ($P < .0005$), friends ($P < .0005$), and teammates ($P = .014$). No significant association was noted between chronotype and the MSPSS significant other subscale.

Relationship between chronotype score and depression, stratified by social support

Results of stratified regression analyses are shown in Table 3, stratifying the relationship between chronotype and depression by low or high social support. With social support variables dichotomized to low/high, and with interaction terms computed for social support-by-chronotype interactions on depression, we see significant associations for friends ($P = .048$), significant other ($P < .0005$), and teammates ($P = .017$), but not total score or family score. Reflecting non-significant interactions, relationships between chronotype and depression were not significant for individuals stratified by low vs high total social support score or family score. However, significant interactions were reflected in the other stratified analyses of subscales, such that later chronotype was associated with depression only in those with low social support from friends and teammates (this association may be an intuitive one – these are the affiliative groups students are most likely to reach out to day-to-day). Conversely, later chronotype was only associated with higher depression scores among those with *high perceived social support* from a significant other.

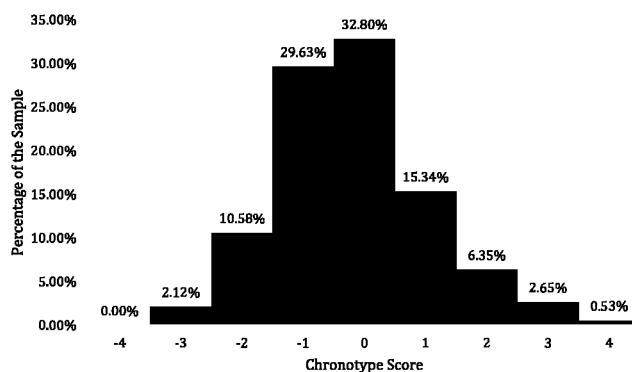


Figure 2. Distribution of chronotype scores.

Mediation of social support on the relationship between chronotype score and depression

Data examining the role of social support as a mediator between chronotype score and depression are presented in Table 4. A traditional mediation test was performed, examining direct and indirect relationships between chronotype and depression, with and without each social-support variable (total score and individual subscale). Sobel tests determined whether partial mediation occurred. Significant Sobel tests were seen for total score (i.e., aggregated from family, friends, and teammates),

Table 5. Zero-order correlations between all variables.

		Chronotype	CESD Depression Score	Total Score	MSPSS Social Support	Family	Friends	Significant Other
CESD Depression Score	r	0.1727						
	p	.0175						
MSPSS Social Support	r	-0.2428	-0.4531					
	p	.0008	<.0005					
MSPSS Social Support (Family)	r	-0.2939	-0.4	0.7988				
	p	<.0005	<.0005					
MSPSS Social Support (Friends)	r	-0.2529	-0.4175	0.8715	0.5797			
	p	.0004	<.0005	<.0005	<.0005			
MSPSS Social Support (Significant Other)	r	-0.0661	-0.2042	0.7515	0.4829	0.4944		
	p	.3662	.0048	<.0005	<.0005	<.0005		
MSPSS Social Support (Teammates)	r	-0.1829	-0.465	0.8026	0.5143	0.762	0.3723	
	p	.0118	<.0005	<.0005	<.0005	<.0005	<.0005	<.0005

CESD: Center for Epidemiological Studies Depression.

MSPSS: Multidimensional Scale of Perceived Social Support.

such that 62% of the relationship between chronotype and depression was explained by variability in total perceived social support. In examining subscales, 65%, 59%, and 46% of the relationship was explained by variability in family, friend, and teammate scores, respectively.

Table 5 displays zero-order correlations between all variables. Chronbach alphas were determined for CESD (0.82) and MSPSS Total (0.93), Family (0.91), Friends (0.91), Significant Other (0.92), and Team (0.94).

Discussion

The purpose of this study was to assess the relationship between later chronotype, depressive symptoms and social support. We hypothesized that social support would mediate the relationship between chronotype and depressive symptoms. Overall, results demonstrated that later chronotype was positively associated with higher depression scores and lower social support (overall, or from family, friends, significant other, or teammates). Further, social support was identified as a partial mediator (partially accounting for the relationship) between later chronotype and depressive symptoms. Specifically, for those with low social support overall, from family, friends, or teammates, we found a significant relationship mediating chronotype and depressive symptoms. Also, those with high social support from significant other demonstrated a similar relationship between chronotype and depressive symptoms. Thus, later chronotype was associated with depressive symptoms among those with low social support from friends or teammates and those with higher social support from a significant other.

Previous studies have shown that later chronotype is associated with depression symptoms, including younger adults (Hsu et al. 2012; Prat and Adan 2013). The present study extends these findings to student athletes as well,

who generally are more likely to have early morning schedules (Driller et al. 2017). In addition, the present study supports previous work indicating associations between social support and depression (Mazza and Reynolds 1998; Teo et al. 2013; Wilson et al. 2014).

Individuals with a later chronotype are more likely to be awake at night, when there is less of an opportunity to engage with other people – especially friends, family, and teammates. This social isolation may contribute to depressive symptoms. Previous studies have found that social isolation is a risk factor for depression (Ge et al. 2017; Matthews et al. 2016). It is plausible that those with a later chronotype are more prone to depressive symptoms because they are feeling socially isolated, as their friends, family, or teammates may not be awake or active that late at night. Although college athletes may have a rigid sleep schedule (i.e. expected to be up early for sports practice), it is possible that those with later chronotypes spend their night hours catching up on schoolwork or working part-time jobs. Although student athletes have exhibited a delayed sleep phase (Monma et al. 2018), and their peers may also be up late at night, it is possible that college athletes may not be communicating with their friends and teammates during these hours, due to expectations that others may be asleep. While variability in sleep schedule, common in athletes (Sargent et al. 2014), has been associated with both circadian misalignment (Giannotti et al. 2002) and depression (Suh et al. 2012), this was not included in the present analysis.

The distribution of chronotype in our sample appeared somewhat normally distributed, with a slight skew toward morningness. While university students generally tend to skew toward eveningness (Zimmermann 2011), a skew toward morningness has been previously seen in athletes (Bender et al. 2019). Thus, this may be one plausible explanation for the relationship between later chronotype,

depression, and low social support from teammates, as student athletes with later chronotype may have less opportunity to socialize with teammates with morning chronotypes.

The results of the “significant other” subscale do not follow the pattern of the impact of low social support on the chronotype-depression connection, which may be influenced by the possibility that individuals spend more time together with their significant other at night (e.g., sleeping in the same bed). However, an interesting finding is that, when stratified by high and low social support, chronotype is only associated with increased depressive symptoms for those with high social support from a significant other. It is possible that there are other factors not addressed in this study that influence this relationship. While few studies have examined the relationship between chronotype, depression, and social support from significant other, previous cross-sectional studies have indicated that concordance of bed partners’ sleep timing may play a role in relationship satisfaction (De Waterman and Kerkhof 1998). This suggests that differing chronotypes between partners may impact their interpretation of social support from their significant other. It has also been shown that significant other’s depression influence patterns of sleep, such as sleep duration (Richter et al. 2016). Overall, it is hypothesized that there is a unique relationship between aspects of sleep, support from significant other, and mental health for those who share sleeping arrangements with their significant other (Troxel 2010). One explanation for our results may be that those with high social support from their significant other may feel dependent on them, and that a misalignment between the student athlete’s later chronotype and their significant other’s sleep timing, may influence these depressive symptoms. This follows the assertion by Troxel (2010) that one’s significant other may act as a social “time-keeper”, influencing their partner’s circadian rhythm. In the present study, it can also be suggested that the lack of a significant other in some participants may be influencing their own responses to “significant other” social support questions in the questionnaire, thus reducing the validity of this subscale’s correspondence with other outcome variables, as participants were not given the option to leave these questions blank.

Other important factors to consider in the relationship between late chronotype and increased depressive symptoms may include poor sleep quality and insufficient sleep, as these are issues faced by many student athletes (Mah et al. 2018). Accordingly, sleep difficulties are related to increased depressive symptoms (Alvaro et al. 2013). Another mechanism which may be important in understanding the relationship between evening

chronotype and symptoms of depression in student athletes is that evening chronotypes are more sensitive to pain (Jankowski 2013), and self-reported pain is associated with significantly increased odds of symptoms of depression in athletes (Yang et al. 2007).

Future applications of this research could investigate the disparities in this mediated relationship in the context of different cultures, as socialization can be influenced by culture and demographics. Furthermore, it would be beneficial to generalize these findings and retest our hypotheses with larger, more population-representative samples (e.g., the hypothesis that late chronotype may be preventing subjects from socializing with others, leading to isolation and perceived lack of social support). It would also be interesting to compare the sleep timing and chronotypes of these individuals with that of their family, friends, significant other, and teammates, as this may help in determining the degree of misalignment in socialization opportunity. This research also suggests interventions for those with later chronotype by way of increased social support. More broadly, these results may facilitate more discussion regarding the various relationships that emerge when studying circadian rhythms and mental health, as chronotype is just one outward manifestation of endogenous circadian biology.

Strengths

One strength of this study is the use of the MSPSS (Zimet et al. 1990) as a validated measure of social support, with the added component of categorizing teammates for questions targeting university student-athlete participants. Further, the sample of elite athletes we recruited was demographically diverse across age, sex, and sport. Another study strength was the ability to quantitatively assess the contribution to depression that social support makes in conjunction with chronotype. Social support is a potentially modifiable aspect of one’s environment, that, with improvement, can lead to a corresponding improvement in depressive symptoms (Teo et al. 2013). Lastly, assessment of social support, depression, and chronotype in university student athletes is itself novel, as this population is understudied in this regard. Although it has been documented that student-athletes are less likely than non-athletes to experience depressive symptoms owing to protective factors such as self-esteem, social support, and connectedness (Armstrong et al. 2015), other data suggest that depressive symptoms can be more prevalent among those engaged in university sports (Wolanin et al. 2016; Yang et al. 2007). This study found that student-athletes with later chronotypes were more likely to exhibit depressive

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Investigating the relationship between mild traumatic brain injury and Alzheimer's disease and related dementias: a systematic review

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Abstract

The objective of this systematic review is to synthesize the relevant literature published after 2016 to ascertain the current landscape of science that relates mild traumatic brain injury (mTBI) to the onset of Alzheimer's disease and related dementias (ADRD) and identify areas of need for future research. We conducted database searches and retrieved articles that were published after 2016 that utilized cognitive assessments to understand the relationship between mTBI and ADRD. We identified eight relevant articles in the review process, four of which presented a significant relationship between mTBI and disease or cognitive impairment outcomes. The studies included in this systematic review underscore the need for future research investigating a possible causal relationship between mTBI and ADRDs given the high prevalence of mTBI among brain injury patients and the lack of literature specifically addressing this issue. Future research should standardize the definitions of mTBI, AD, and ADRDs to create reliable and reproducible results that more comprehensively capture the nuances of this relationship.

Keywords Alzheimer's disease · mTBI · Concussion · Dementia · Traumatic brain injury

Introduction

There are over 5 million people in the United States living with Alzheimer's disease and Related Dementias (ADRDs), and this number continues to rise [1]. Mild, moderate, and severe traumatic brain injury (TBI) may be a risk factor for developing ADRDs. However, the vast majority of literature that seeks to examine the relationship between brain injury and ADRDs specifically focuses on moderate and severe TBI, leaving a clear gap in information concerning mild TBI (mTBI) in particular. While a strong body of evidence points toward moderate to severe TBI as a risk factor for developing ADRDs [2–6], other studies dispute these claims [7, 8]. Inconsistencies stem from methodological differences in the participant inclusion/exclusion criteria and diagnostic definitions of TBI and ADRDs [9]. This discordance in the findings is more pronounced when focusing on only mTBI, which has received less attention as a risk factor for developing ADRDs.

A smaller, more focused body of literature has demonstrated an associative relationship but has not established a causal link between mTBI and ADRDs, finding long-term

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effects such as pathophysiological brain alterations and late-life cognitive impairment [9–11].

Previous studies and reviews that note inconsistent relationships between mTBI and ADRDs explicitly identify a need for larger samples and improved methodological design [9–11]. Julien et al. [10] conducted a systematic review to investigate the relationship between the severity of TBI and onset of Alzheimer's disease (AD). Specifically, the authors examined whether variables related to TBI severity, such as loss of consciousness (LOC), Glasgow Coma Scale (GCS) score, and post-traumatic amnesia (PTA), were predictive of AD risk. The review included 18 original studies published between 1985 and 2015, the majority of which were retrospective and conducted in the United States. In about 55.6% of studies, the incidence of TBI increased the risk of AD. However, the review failed to establish an association between incidence of TBI and risk of AD due to methodological differences between such studies. Diagnostic criteria for TBI and AD, as well as criteria related to TBI severity, differed in the studies. Julien et al., recommended using standardized diagnostic criteria in future studies that examine the association between TBI and AD.

Perry et al. [11] performed a meta-analysis assessing the relationship between clinical history of mTBI and the subsequent development of neurological or psychiatric disease. The analysis included 57 studies published between 1995 and 2012 that met narrow inclusion criteria related to diagnosis of TBI and neurological or psychiatric disease states. The analysis revealed that individuals with prior mTBI were more likely to develop neurological and psychiatric disease than individuals without a history of TBI. This finding points to mTBI as a risk factor for neurological and psychiatric illness. Perry et al., suggested narrower definitions of TBI, including mTBI, and standardized diagnostic criteria in future research.

The present systematic review narrows the focus of previous studies by investigating the association between mTBI and ADRDs while addressing previous methodological challenges by defining stricter inclusion criteria. This review examines the current literature that reports a direct relationship between mTBI and ADRDs, which adds to the understanding of mTBI as a risk factor for subsequent neurodegenerative disease. Specifically, does the presence of mTBI confer a greater risk for developing ADRD?

Methods

A systematic review of peer-reviewed literature published between January 2016 and January 2022 was conducted with Embase, Cochrane Central Register of Controlled Trials (CENTRAL), Medline, PubMed, PsycInfo, and Scopus. Selection of these databases was based on a 2017 systematic

review (Julien et al.) examining the relationship between Alzheimer's disease and TBI, including mild, moderate, and severe injury using articles from Medline, PubMed, Embase, Psycinfo and CENTRAL. Our review included the same databases with the exception of Embase and Scopus due to their limited scope of brain injury literature upon recommendation from a medicine and health sciences librarian (ES). The time period specified in our search began where a recent systematic review [10] ends to examine current literature since the date of that publication. Key search terms included: mild traumatic brain injury, mTBI, concussion, repetitive mild traumatic brain injury, repeated mild traumatic brain injury, repetitive mTBI, repeated mTBI, repeated concussion, repetitive concussions, Alzheimer's Disease, AD, ADRD, dementia, related dementia, NINCDS/ADRDA, and MCI. These terms were chosen to encompass all literature relevant to ADRD and mTBI, including singular and repeated injuries, while intentionally not including other related conditions or severe TBI. Search terms encompassing both AD and general references to dementia were included to ensure all relevant articles pertaining to AD as a subset of dementia were reviewed. After searching, 239 articles were screened by title and abstract. Below is a sample search from Scopus:

(TITLE-ABS ("mild traumatic brain injury" OR "mTBI" OR "concussion" OR "sport concussion" OR "mild TBI") AND TITLE-ABS ("Alzheimer's Disease" OR "AD" OR "dementia" OR "related dementia") AND TITLE-ABS("cognition" OR "cognitive impairment") AND PUBYEAR > 2014 AND PUBYEAR < 2022).

Study selection

After removing duplicate studies, 239 titles and abstracts were screened independently by three reviewers (EC, AK, MM) using Covidence systematic review software (v1780). Studies were excluded if they assessed efficacy of drug treatments, studied patients with other neurological disorders (i.e., Parkinson's, ALS), sampled patients less than 18 years of age, or analyzed individual pathology of ADRD or mTBI without commenting on the presence or absence of a causal relationship or considering measures of patient cognition. Potential conflicts between individual reviewers were discussed and resolved by the senior author (RWT) if a consensus was not reached independently; however, all conflicts were resolved without intervention. There were 30 studies that examined a possible direct relationship between mTBI and ADRD and were published between January 2016 and January 2022 that underwent full-text review. Each reviewer, using the same exclusion criteria, then conducted a full-text screening of all articles independently, and eight full-text studies were eligible for the assessment phase.

Quality assessment

Each of the eight selected articles were assessed by two of the three reviewers (EC, AK, MM) by using the Cochrane Risk of Bias tool in Covidence. The Cochrane Risk of Bias scale assesses risk bias as low, high, or unclear in the following categories: sequence generation, allocation concealment, blinding of participants and personnel, blinding of outcome assessors, incomplete outcome data, selective outcome reporting, and other sources of bias. Selection bias was evaluated through sequence generation, which refers to how sequences for treatment allocation were created, and allocation concealment, which refers to whether the treatment allocation could have been anticipated by participants. Performance bias was considered by assessing the blinding of participants and personnel, and detection bias was noted by considering the blinding of outcome assessors. Attrition bias was assessed by checking for incomplete outcome data, and performance bias was checked by noting any selective outcome reporting. A more thorough explanation of these categories and criteria is available in the Cochrane documentation [15]. After independent review and discussion, all eight studies were deemed to have a low risk of bias and proceeded to data extraction.

Data extraction

Three reviewers (EC, AK, MM) independently extracted data from each selected study. Extracted data included: study setting, study sponsorship, author information, type of study, sample demographics, definitions of mTBI and ADRD, recollection of participants' retrospective information, interventions and measurements, and sample outcomes. This review follows Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

Results

The eight studies we selected examined multiple aspects of the hypothesized relationship between mTBI and Alzheimer's disease and related dementias (Table 1). However, all of the studies that met the inclusion criteria for this review focused specifically on Alzheimer's disease. Four of the studies [12–15] conducted retrospective cohort analyses, three conducted case-control analyses [13, 16, 17], and one conducted a prospective cohort analysis investigating later development of Alzheimer's Disease or AD-related cognitive symptoms [18]. The findings from each of these studies create a comprehensive picture of the current methods for investigating this dynamic and complex relationship. Recent literature shows the possibility of an mTBI event causing not only acute symptoms but initiating a string of longer-term

cognitive deficits and clinical effects similar to AD progression [11, 17, 19]. Mild cognitive impairment (MCI) and AD had a younger average age of onset in mTBI patients than in patients with no mTBI history [12]. One study calculated polygenic risk scores for AD and found that mTBI history among individuals with high polygenic risk scores showed more atrophy in AD susceptible brain regions than non-mTBI controls [15]. Similarly, some association was found between mTBI history, cognitive function, and early-onset AD [20]. However, four studies found no significant relationship between mTBI history and cognitive measures of ADRDs [13, 14, 16, 21]. These articles represent the small, current body of literature investigating long-term cognitive impacts of mTBI as it pertains to AD and suggests possible similarities in cognitive manifestations of AD and mTBI. However, they do not offer a clear picture of the nature or significance of this relationship.

Demographics

In three of the retrospective cohort analyses we reviewed, the mean age of participants ranged from 67 to 75 years old and consisted predominantly of non-Hispanic White adults [12, 14]. One study, which examined polygenic risk scores for AD in relation to mTBI had a mean age of 31 [15].

ADRD definition/criteria

There was variance in the diagnosis criteria for AD across the studies. Two studies utilized the US National Institute of Neurological and Communicative Disorders and Stroke/AD and Related Disorders Association (NINCDS/ADRDA) criteria for diagnosis [12, 14]. One study identified participants who were diagnosed with established criteria for "clinically probably AD dementia" during their most recent Uniform Data Set visit in the National Alzheimer's Coordinating Center (NACC) database [13]. Rostowsky et al. [16] used the ADNI criteria for AD diagnosis, another used the AD-8 Questionnaire, and one identified AD-vulnerable brain regions according to Sanbuncu et al. [15]. Albrecht et al. [18] synthesized a clinical dementia rating through a cognitive testing battery including Mini-Mental State Exam (MMSE), California Verbal Learning Test II (CVLT-II), Wechsler Memory Scale Logical Memory I and II (WMS-LM I & II), Delis-Kaplan Executive Functioning Verbal Fluency Test (D-KEFS VF), Boston Naming Test, Wechsler Test of Adult Reading (WTAR), Wechsler Adult Intelligence Scale III (WAIS-III), the Stroop Test, and the Rey-Osterrieth Complex Figure Test (RCFT). One study utilized ICD-9 dementia diagnoses recommended by the Veterans Affairs Dementia Steering Committee, which were validated in 153 randomly selected cases using medical chart abstractions

Table 1 Author, publication year and country, sample demographics, AD definition, and AD & mTBI inclusion criteria for diagnosis for selected studies

Authors	Year published	Country	Experimental design	Cohort year	Sample	AD/ADRD inclusion criteria	mTBI inclusion criteria	Relationship findings
Hayes et al	2017	United States	Longitudinal Retrospective Cohort Analysis	2014–2016	mTBI: <i>n</i> = 105 rmTBI: <i>n</i> = 49 No mTBI: <i>n</i> = 55 Mean Age = 31.36	Reduced cortical thickness in AD vulnerable regions Low CVLT-II scores	Boston Assessment of TBI Lifetime (BAT-L) Interview; self-reported	mTBI+ High Polygenic risk score for AD associated with reduced cortical thickness and reduced episodic memory recall PRS Threshold P = 0.02 (<i>p</i> = 0.048)
Li et al	2016	United States	Cross Sectional Cohort Analysis	2003–2015	mTBI: <i>n</i> = 73, mean age = No mTBI: <i>n</i> = 1197, mean age = Age Range: 55–90	NINCDS/ADRDA score 0.5 or 1	Self-reported	Age at AD onset was earlier for patients with mTBI history vs without [68.5 (66.3–70.7 CI, <i>n</i> = 56) vs. 70.9 (70.5–71.4 CI, <i>n</i> = 1197)]
Mendez et al	2015	United States	Retrospective Cohort	2005–2014	EOAD: <i>n</i> = 1,449, mean age = 62.7	AD: ADC criteria for clinically probable AD dementia EOAD: Clinically probable AD with cognitive decline onset at < 65 years of age	NACC-UDS survey questions, physician determination of TBI LOAD: Clinically probably AD with cognitive decline onset ≥ 65 years of age	Participants with EOAD and a history of TBI only scored significantly differently than EOAD participants without TBI history in 2 cognitive tests (Logical Memory IA & II A, <i>p</i> < 0.001, <i>p</i> < 0.05) out of 11 tests

Table 1 (continued)

Authors	Year published	Country	Experimental design	Cohort year	Sample	AD/ADRD inclusion criteria	mTBI inclusion criteria	Relationship findings
Rostowsky et al 2021	2021	United States	Case–Control	2004–2010	mTBI: $n=33$, mean age = 62.7	ADNI Inclusion Criteria and clinical dementia diagnosis	GCS > 12, LOC < 30 min, PTA < 24 h, no clinical findings besides CMBs on acute MRI	Similarities in cognitive/clinical effects of AD and mTBI suggest similar manifestations and supports mTBI as a risk factor for AD-like cognitive decline and the possibility of subclinical MCI due to mTBI, as chronic mTBI mirrors AD in many ways

AD: $n=66$, mean age = 75.6

MMSE: Significant differences between control and mTBI participants (Welch's $t = -7.49$, $df \approx 35$, $p = 4.53 \times 10^{-9}$), control and AD participants (Welch's $t = 17.01$, $df \approx 131$, $p < 0.001$) but not mTBI and AD (Welch's $t = 4.59$, $df = 64$, $p = 0.99$)

MoCA: Significant differences between HC and AD participants (Welch's $t = 17.26$, $df \approx 131$, $p = 9.33 \times 10^{-39}$), acute mTBI and AD participants (Welch's $t = 10.65$, $df \approx 59$, $p = 1.04 \times 10^{-15}$), but not between control and acute TBI participants (Welch's $t = -0.29$, $df \approx 42$, $p = 0.39$)

Table 1 (continued)

Authors	Year published	Country	Experimental design	Cohort year	Sample	AD/ADRD inclusion criteria	mTBI inclusion criteria	Relationship findings
June et al	2020	United States	Longitudinal Retrospective Cohort Analysis	Selected after 1958	mTBI: $n=51$, mean age = 65.14	NINCDS/ADRDA criteria	Concussion with LOC < 30 min	The relationship between mTBI and dementia onset is unclear
Albrecht et al	2016	Australia	Longitudinal Prospective Cohort	2006–2012	TBI: $n=53$, Mean Age = 70.2	Clinical Dementia Rating > 0.5	Repetitive self-report	No statistically significant impact of TBI on neuropsychological test scores
Didehbani et al	2020	United States	Case–Control	2010	No TBI: $n=104$, Mean Age = 70.2	Score less than 1.5 SD below adjusted mean	WTAR IQ: $p=0.56$ Clinical dementia rating: $p=0.91$ CDR sum of boxes: $p=0.85$ HADS depression: $p=0.55$ HADS anxiety: $p=0.46$	Self-report evaluated against AN criteria History of multiple TBIs does not significantly affect the neuropsychological profile of patients with MCI

and reviews by a panel of neuropsychologists [21]. Li et al. [15] article calculated polygenic risk scores.

MTBI definition/criteria

As was the case for AD, there was wide variation in the identification of mTBI due to the relative infancy of research investigating mild TBI as a distinct entity from moderate and severe TBI, especially in relation to AD. Mendez et al. [12] used the Mayo Clinic TBI standards, which diagnose mTBI based on a standardized scale of self-reported symptoms. Albrecht et al. [21] used the ICD-9 to define mTBI with additional consideration from multiple Department of Defense and Veterans Affairs data sources to define TBI severity. Self-reported injury of participants with a loss of consciousness for less than 30 min and scores from the Glasgow Coma Scale (GCS), a scale ranging from 13 to 15 with less than 24 h of post-traumatic amnesia, were also used for diagnosing and standardizing mTBI, and determining mTBI incidence for four studies [14–17], while another study classified mTBI as a self-reported concussion with less than 30 min of loss of consciousness [14]. Didehbani et al. [13] also used self-reported TBI history, but the participants' injuries were classified according to the American Academy of Neurology's "Practice Parameter Guidelines for Grading TBI". Lastly, one study recruited TBI participants using the National Alzheimer's Coordinating Center (NACC) Uniform Data Set (UDS), which defined TBI history based upon questions answered during a medical history interview regarding minute loss of consciousness (LOC) [12].

Cognitive function evaluations

When examining the studies that measured cognitive function, much overlap can be seen in evaluation methods. The Mini-Mental State Examination (MMSE) was the most frequently used assessment with four out of the eight studies using it to measure cognitive impairment [13, 16–18]. The Boston Naming Test (BNT), and Digit Span and Digit-Symbol Coding subtests of the Wechsler Adult Intelligence Scale (WAIS-III) were used in two studies [13, 18]. The Logical Memory I and II from the Wechsler Memory Scale were also used in the same two studies as well as one other, which used the test to measure age at onset [12]. Albrecht et al. [21] also included cognitive evaluation measures such as D-KEFS verbal fluency subtests, Stroop test, and RCFT. CVLT-II was used to assess episodic memory performance using long-delay free recall in three studies [14, 15, 21]. Additionally, Mendez et al., Didehbani et al., and June et al. included category fluency tests and Trail-Making tests (A and B) [13]. Rotowsky et al., also acquired Montreal Cognitive Assessment (MoCA) scores for their health participants and within 48 h post-injury for their TBI participants.

The only study that analyzed visuospatial perception and motor ability was June et al., which used the Card Rotations Test and the Clock Drawing Test. While there is not a gold-standard diagnostic test for Alzheimer's disease or dementia, different forms of psychometric testing are often used to evaluate a patient's cognition and lead towards a diagnosis. A conclusive diagnosis of AD was only possible at post-mortem autopsy, but now PET imaging capability and developments in blood amyloid testing promises to make a definitive diagnosis of AD more accessible [22, 23]. In particular, the MMSE is the most common neuropsychological test for screening AD and other causes of dementia as it assesses skills such as short-term memory and orientation.

Age of onset

The influence of mTBI on the age of onset of ADRD is a significant finding in this analysis. Two studies included in this analysis identified a relationship between mTBI incidence and age of onset (AOO) of ADRD [12, 20]. One study conducted analyses of variance to compare the AOO of ADRD in participants with and without a history of mTBI [20]. The study observed a significantly earlier AOO among individuals with mTBI history, with a difference of over four years in patients diagnosed with MCI and a smaller difference of two years in patients diagnosed with AD. Mendez et al. [12] utilized data from a large, comprehensive database of AD patients to examine the relationship between mTBI history and early-onset AD (EOAD) versus late-onset AD (LOAD). Mild TBI history was reported to be a specific risk factor for EOAD but not for LOAD, indicating that mTBI may accelerate the onset of ADRD. These findings suggest that a history of mTBI may accelerate neurodegenerative processes and result in an earlier onset of ADRD. This conclusion is clinically significant since it emphasizes the need for clinical diagnosis and surveillance of individuals who may be at risk for developing accelerated ADRD, as well as the use of clinical interventions to mitigate cognitive impairment after mTBI.

Relationship findings

Four of the eight studies included in this analysis identified some relationship between mTBI and ADRD [12, 13, 15, 17]. Three of these focused on the relationship between mTBI and the age of onset of ADRD and reported an association between a history of mTBI and an earlier age of ADRD onset [12, 13, 15]. Didehbani et al. [13] examined whether TBI history was significant among patients with early-onset Alzheimer's disease (EOAD), late-onset Alzheimer's disease (LOAD), or controls. The study reported that incidence of TBI history was significant among those with EOAD compared to controls (11.1%; $\chi^2 = 3.64, p = 0.056$), but not

among those with LOAD when compared to controls (8.7%; $\chi^2=2.73, p=0.100$). Another study that investigated the age of onset (AOO) of AD in relation to mTBI discovered the AOO for patients with a history of mTBI was significantly earlier [68.5 (66.3–70.7 95% CI, $n=56$) vs. 70.9 (70.5–71.4 95% CI, $n=1197$)] [12]. The third study relating mTBI and age of onset of ADRD found that mTBI was significantly associated with early-onset ADRDs (EOD) [aOR(95%CI): mTBI-4.5(2.4–8.9)] [21]. However, this study also observed an association between other neurological and psychological disorders, such as stroke, depression, PTSD, and headache, and a greater incidence of early-onset dementia. One study calculated a polygenic risk score (PRS) for AD onset found that mTBI was associated with a high AD PRS ($p=0.02$) along with reduced cortical thickness and episodic memory recall compared to non-mTBI study participants [15]. Another study observed similarities in the cognitive and clinical effects of AD and mTBI, suggesting similar manifestations of both morbidities, thus supporting mTBI as a risk factor for AD-like cognitive decline and suggesting the possibility of subclinical MCI due to mTBI, as chronic mTBI mirrors AD in many ways [17]. While the above studies offer evidence for mTBI as a risk factor for ADRD, particularly EOAD, four of the studies included in this analysis did not identify any neuropsychological evidence that mTBI is a direct risk factor for AD [14, 16, 18, 21]. This finding may be due to the limited sensitivity of neuropsychological tests. This limitation is not present in neuroimaging studies, which indicate stronger evidence of mTBI as a risk factor for ADRDs. These mixed results found in recent literature are consistent with those in previous reviews, further highlighting the need for a more rigorous and focused investigation of this possible association or lack thereof.

Discussion

Here we examined recently published literature that reported a direct relationship between mTBI and ADRDs. Broadening our search to include studies that were more cognitively centered allowed us to highlight the potential link between ADRDs and mTBI implied by the underlying similarities in pathological factors.

The majority of the studies excluded from this analysis investigated the indirect relationship between mTBI and ADRDs, particularly the similar pathology shared between them. Research has shown that the incidence of repeated mTBI evokes brain pathology that is similar to neurodegenerative disease, which results in acute deposits of dementia-specific biomarkers such as amyloid and tau plaques [24]. Additionally, disrupted sleep (e.g., due to depression) following TBI can affect neuroplasticity and increase the buildup of amyloid-beta plaques, both of which are risk

factors for AD [25, 26]. A disease that shares similar tau pathology and hippocampal shrinkage with Alzheimer's Disease is chronic traumatic encephalopathy (CTE). CTE is a neurodegenerative disease associated with exposure to repetitive head impacts (RHI). Although there are biological similarities between AD dementia and CTE, there is evidence that the two conditions present distinct symptoms [27]. While Alzheimer's Disease shows characteristic short-term memory loss, patients who are diagnosed with CTE from post-mortem autopsy were reported to exhibit depression, mood lability, anger outbursts, substance abuse patterns, and even suicide during life, which are not typical AD-related behaviors [28]. While there is a small overlap, this research intentionally does not investigate CTE as an Alzheimer's related dementia because current literature is insufficient in concluding that there is a definitive relationship between the two diseases, and CTE is not a clinical form of dementia [29].

Despite the similarities in pathology, this review deliberately excluded studies that solely focused on pathology in order to include cognition-centered studies. We believe that establishing such a relationship will help provide the groundwork for future studies regarding the impending AD epidemic. Furthermore, the narrow focus of this analysis is able to contribute a stronger foundation for biomolecular research by synthesizing the current knowledge base to help build consensus within the scientific community.

A limitation of this systematic review is the small number of studies included and analyzed secondary to our strict inclusion criteria and a lack of literature pertaining specifically to a consensus definition of mTBI. Discrepancies in methods and criteria for identifying mTBI greatly reduced the number of articles that were eligible for review, as has been the case for earlier systematic reviews on the same topic [9, 10]. This challenge limits our research findings as each study aims to identify mTBI cases in its own way, which hampers the ability to compare results across studies in a systematic manner. The dearth of studies, discrepant results, lack of methodological consensus, and the small number of studies emphasize why this particular vein of research lacks conclusive findings [29]. Considering these limitations, our review supports existing literature suggesting that mTBI may not be the sole risk factor influencing the onset and progression of ADRDs, but one of many that can impact neurological disease development more broadly [30]. Beyond the lack of a standardized definition and mechanism for diagnosis, mTBI has historically been considered an acute injury without long-term cognitive effects. Unlike moderate and severe TBI, immediate symptoms of mTBI such as headaches, nausea, sensitivity to light, and fatigue often resolve without active neurological treatment. As a result, there has not been a substantial amount of interest in investigating the relationship

between mTBI and ADRD until recently. While increased and consistent incorporation of the Glasgow Coma Scale (GCS) and diagnosis of post-traumatic amnesia (PTA) has shed some light on the long-lasting effects of mTBI, consistent follow-up for long-lasting symptoms continues to be inadequate since many injuries go undiagnosed or misdiagnosed [31]. We recognize and acknowledge the limited value of GCS to accurately assess mTBI due to its lack of specificity; nevertheless, we have highlighted that GCS is the most widely-used and standardized tool cited for diagnosing mTBI within the body of literature we reviewed [32].

The methodology of diagnosing AD was variable across studies, but the framework for establishing a positive diagnosis in all of the studies could be classified as cognition-centric. For example, the NINCDS/ADRDA criteria, AD ICD-9 codes as recommended by the VA Dementia Steering Committee, and NCA criteria are considered cognition-centric as they are rooted in neuropsychological testing and clinical judgment. One study used a set of neuropsychological tests along with the Clinical Dementia Rating scale to classify participants' status as with or without impairment [13]. These approaches provide a comprehensive evaluation of cognitive impairment and AD (Fig. 1).

The differences in outcomes and methods in studies can be explained by evaluating different cognitive endpoints of AD. While NINCDS/ADRDA provides cognitive information about the manifestation of AD in multiple contexts, evaluation of MCI may provide cognitive information about patients in earlier disease stages. Both approaches have benefits and drawbacks, but given the difficulty in diagnosing

AD, it is important to have similar approaches across studies to establish consistent criteria for diagnosis.

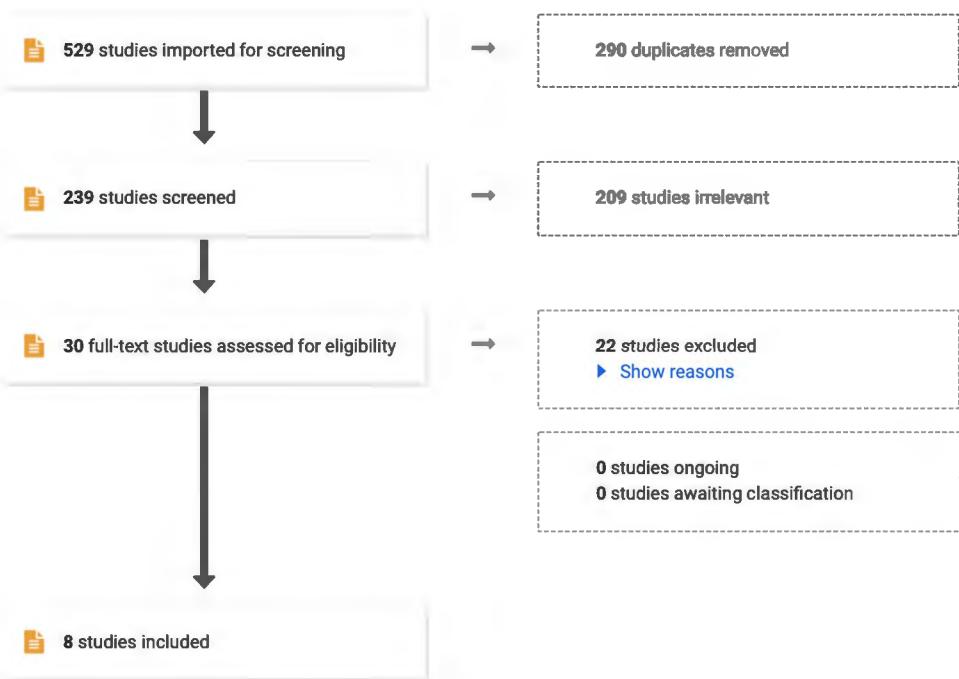
The categorization of mTBI was also variable among studies, and a standardized definition is vital in order to establish a meaningful relationship of any kind with degenerative brain conditions [29]. Due to the wide range of injuries that fall under the umbrella of mTBI and the low reliability of self-reported data, nearly all of the studies analyzed in this review were unable to fully capture nuances of different exposures and symptom presentations of mTBI (e.g., repetitive, including LOC, early vs. late, etc.). Our analysis aimed to examine the impact of mTBI on developing ADRD as a whole, including the impact of multiple mTBIs.

Unfortunately, much of the current literature fails to analyze the difference between a singular mTBI occurrence and multiple events. All the studies we included used inclusion criteria of at least one mTBI resulting in a heterogeneous sample of individuals who had suffered multiple mTBIs and individuals who had only suffered one mTBI. This heterogeneity may have biased the data sets and must be considered when assessing the impact of a singular mTBI on developing ADRD or the age of ADRD onset.

Finally, "grey literature" including conference papers and poster presentations were reviewed and excluded from this study due to our strict exclusion criteria. As a result of the lack of unpublished literature incorporated, our results may be susceptible to publication bias.

Further research should continue to reinforce the presence of an epidemiological association between mTBI and ADRDs in current literature. By standardizing the definition of mTBI and AD across studies, along with standardizing

Fig. 1 PRISMA flowchart of systematic review process



evaluation measures of injury and disease onset, the literature can be readily compared and promote follow-up studies. Once the presence of an association between mTBI and AD is well established, the focus of research can shift towards understanding the mechanism underlying this association. Similarities in pathology between AD and mTBI should also continue to be investigated. Both AD and mTBI are associated with reduced cortical thickness, creating ambiguity in diagnosing the origin of cortical thinning. Further research on the mechanisms and directionality of this relationship is necessary to understand the direct impacts of mTBI on cortical thickness and the onset of Alzheimer's disease unique from cortical thinning caused due to a genetic predisposition to AD, and to increase understanding of how pathological changes may manifest as cognitive changes, and vice versa.

It is reassuring that despite the small breadth of literature in the past few years targeting mTBI and AD, the majority of studies reinforce previous findings. This indicates that the scientific community, despite the lack of a universally accepted definition for mTBI and definitive diagnostic criteria for AD, may be honing in on an important association between the two.

Author contributions RWT conceptualized this systematic review. ES, EC, CM, AK, and SF created the search strategy, exclusion criteria, and methodology. EC and AK screened and selected studies, performed quality assessment, assessed bias, and performed data extraction. All authors discussed the results of the selected studies. EC, CM, AK, and NVN created the initial draft. RWT, RM, EC, NVN, AK, and CM critically reviewed and revised the manuscript. The team was supervised by RWT.

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RESEARCH

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Neuropsychological test performance of former American football players

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Abstract

Background: Patterns of cognitive impairment in former American football players are uncertain because objective neuropsychological data are lacking. This study characterized the neuropsychological test performance of former college and professional football players.

Methods: One hundred seventy male former football players ($n=111$ professional, $n=59$ college; 45–74 years) completed a neuropsychological test battery. Raw scores were converted to T -scores using age, sex, and education-adjusted normative data. A T -score ≤ 35 defined impairment. A domain was impaired if 2+ scores fell in the impaired range except for the language and visuospatial domains due to the limited number of tests.

Results: Most football players had subjective cognitive concerns. On testing, rates of impairments were greatest for memory (21.2% two tests impaired), especially for recall of unstructured (44.7%) versus structured verbal stimuli (18.8%); 51.8% had one test impaired. 7.1% evidenced impaired executive functions; however, 20.6% had impaired Trail Making Test B. 12.1% evidenced impairments in the attention, visual scanning, and psychomotor speed domain with frequent impairments on Trail Making Test A (18.8%). Other common impairments were on measures of language (i.e., Multilingual Naming Test [21.2%], Animal Fluency [17.1%]) and working memory (Number Span Backward [14.7%]). Impairments on our tasks of visuospatial functions were infrequent.

Conclusions: In this sample of former football players (most of whom had subjective cognitive concerns), there were diffuse impairments on neuropsychological testing with verbal memory being the most frequently impaired domain.

Keywords: Alzheimer's disease, American football, Chronic traumatic encephalopathy, Cognitive function, Neuropsychology, Repetitive head impacts

Background

Repetitive head impacts (RHI) from American football have been associated with later-life cognitive symptoms [1–6] and chronic traumatic encephalopathy (CTE) [5, 7–9]. We use the term RHI herein to refer to environmental exposures to repetitive impacts, hits, or blows to the head. These impacts can result in symptomatic traumatic brain injuries (e.g., concussion) and/or less discrete cumulative effects on the brain. The 2021 National

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Institute of Neurological Disorders and Stroke consensus diagnostic criteria for traumatic encephalopathy syndrome (TES) describe the clinical disorder associated with neuropathologically diagnosed CTE [10]. Impairments in memory and/or executive function are core cognitive features of the TES criteria. The specificity of these impairments to CTE and/or RHI is not clear as similar impairments are known to develop in other neurodegenerative diseases (e.g., Alzheimer's disease [AD], frontotemporal dementia) [11, 12]. The TES criteria are informed by retrospective reports from informants of brain donors [10, 13] and prospective objective neuropsychological data on individuals exposed to RHI is lacking. The patterns of cognitive impairments in populations at risk for CTE (e.g., former American football players) are not known. This has led to diagnostic challenges for neuropsychologists and other clinicians.

Neuropsychological evaluation is an integral component of the clinical evaluation of neurodegenerative diseases. This is evidenced by clinical diagnostic criteria for neurodegenerative diseases requiring the presence of cognitive impairment on neuropsychological testing [14–18], often defined by standardized test score(s) of 1 to 1.5 standard deviations below the normative mean [14, 17]. The TES research diagnostic criteria emphasize the need for comprehensive neuropsychological testing to substantiate the presence of cognitive impairment [10]. Neuropsychological test scores serve as outcome measures for large-scale multi-center clinical trials of disease-modifying therapies [19]. It is important that cognitive profiles of populations with exposure to RHI, such as former football players, are delineated for research and clinical purposes.

Research studies on the neuropsychological test performance of older former football players have been limited. Schaffert and colleagues conducted a critical review of 22 studies published between 2013 and 2019 on neuropsychological function in former National Football League (NFL) players [3]. Some, but not all, of the studies found evidence for cognitive impairment, most consistently in verbal episodic memory. There were inconsistencies in the domains impaired across studies. That review highlighted the limitations of the research on this topic that include (1) small sample sizes (e.g., $n = 9$ former NFL players); (2) unknown exposure status of the comparison groups; (3) lack of consistent reporting of effect sizes; (4) substantial variation in the extent and quality of the neuropsychological test battery and associated norming practices; and (5) restricted focus on former *professional* American football players.

The objective of this study was to characterize the neuropsychological test performance of a large sample of former college and professional football players from

the DIAGNOSE CTE Research Project [20]. Neuropsychological function across major cognitive domains was assessed and we report the sample raw and T -scores derived from age, sex, and/or education normative data. Rates of impairment by test and cognitive domain are reported.

Methods

Participants and study design

Participants were from the Diagnostics, Imaging, and Genetics Network for the Objective Study and Evaluation of Chronic Traumatic Encephalopathy (DIAGNOSE CTE) Research Project [20]. The objectives of the DIAGNOSE CTE Research Project are to develop *in vivo* biomarkers for CTE, characterize its clinical presentation, and refine and validate clinical research diagnostic criteria. The study enrolled 240 male participants, ages 45–74, including 120 former NFL players, 60 former college football players, and 60 asymptomatic men without a history of RHI or TBI. All participants volunteered to participate as a part of a research study and they were compensated \$500 for their time. Evaluations and participation were not done as part of clinical care or medico-legal purposes. Baseline evaluations were completed between September 2016 and February 2020. Inclusion criteria included no contraindications for MRI, lumbar puncture, or PET procedures; English as the primary language; and consent to all study procedures. Because RHI can often result in TBI, TBI was not exclusionary in the former football players. The former college football players must have played ≥ 6 years of organized football with ≥ 3 years at the college level. Former professional football players must have played ≥ 12 years of organized football, including ≥ 3 in college and ≥ 4 seasons in the NFL. Although recruitment for the former football players was *not* based on cognitive (or neuropsychiatric) status, most football players had subjective cognitive concerns at the time of study screening based on the AD8 Dementia Screening Interview and Cognitive Change Index (Supplemental Table 1).

The criteria for the asymptomatic unexposed were no self-reported diagnosed history of TBI of any severity at study screening; no participation in organized contact and collision sports (including American football), military combat, or any other activity that can result in RHI; absence of self-reported formal diagnosis or treatment of psychiatric illness or cognitive impairment; and no self-reported cognitive, behavioral, or mood symptoms at study telephone screening (Supplemental Table 1). They had to have a body mass index ≥ 24 in order to facilitate matching on body habitus to the former football players. All participants were required to have an informant and adequate decisional capacity at

the time of their baseline visit to participate. Additional details of enrollment criteria and recruitment methods have been reported [20]. The neuropsychological test performance of the unexposed asymptomatic men is presented and qualitatively described in the supplemental material (Supplemental Tables 2 and 3). These data are not presented in the main text, and statistical tests that compare the former American football players to the asymptomatic unexposed men on neuropsychological outcomes were not conducted because recruitment of participants for the DIAGNOSE CTE Research Project was based on our risk factor of interest (i.e., elite football play with RHI exposure) and symptoms (i.e., unexposed men must have been asymptomatic at screening). This recruitment strategy was designed for biomarker development [20]. However, it is problematic when examining clinical measures as outcomes because estimates of group differences are magnified.

Participants were evaluated at Boston University Chobanian & Avedisian School of Medicine (with MRI conducted at Brigham and Women's Hospital); Cleveland Clinic Lou Ruvo Center for Brain Health in Las Vegas, Mayo Clinic Arizona (with PET scans at Banner Alzheimer's Institute); or NYU Langone Medical Center. Participants underwent a 2-day baseline study visit that included a comprehensive neuropsychological examination and other procedures. All sites received approval by their Institutional Review Board. Participants provided

written informed consent. Research was completed in accordance with the Helsinki Declaration.

Sample size

The final sample size included 59 former college football players and 111 former professional football players. (As shown in the supplement, there were 57 asymptomatic unexposed men.) The sample was reduced after exclusion of participants (across all study groups) for missing data on the primary objective neuropsychological tests ($n=5$) and suboptimal performance validity ($n=8$). Three had missing data on the Golden Stroop Color-Word Test due to colorblindness and were excluded from Golden Stroop Color-Word statistics but were not excluded otherwise.

Objective neuropsychological evaluation

Participants completed an in-person baseline neuropsychological test battery using standard paper-pencil tests administered by fully trained examiners [20]. A complete list of the domains assessed and neuropsychological tests administered are presented in Table 1.

Neuropsychological measures were selected to assure harmonization with data-sharing platforms, such as the National Alzheimer Coordinating Center (NACC). Many instruments and methodologies that overlap with the NACC Uniform Data Set (UDS) v.3.0 were selected [29, 30]. Measures include those that assess cognitive domains relevant to the features described in neuro-pathologically confirmed cases of CTE as reported by

Table 1 DIAGNOSE CTE Research Project Neuropsychological Baseline Measures

Domain	Test/instrument	Normative data source	Demographics used in normative data
Performance validity	Test of Memory Malingering	Tombaugh 1996 [21]	-
Est. premorbid intelligence	Wide Range Achievement Test-Fourth Edition Word Reading subtest	Wilkinson et al. 2006 [22]	Age
Learning and memory	Brief Visuospatial Memory Test-Revised (BVMT-R)	Benedict 1997 [23]	Age
	Neuropsychological Assessment Battery (NAB) List Learning	Stern et al. [24]	Age, sex, Educ.
	Uniform Data Set (UDS) Craft Story 21 Recall	UDS v.3.0, June 2019	Age, sex, Educ.
Executive function	Controlled Oral Word Association Test	Tombaugh et al. 1999 [25]	Age, Educ.
	Golden Stroop Color and Word Interference Test (SCWT)	Golden et al. 2002 [26]	Age, Educ.
	NAB Mazes	Stern et al. [24]	Age, sex, Educ.
	Trail Making Test Part B	UDS v.3.0, June 2019	Age, sex, Educ.
Attention, visual scanning, and psychomotor speed	Symbol Digit Modalities Test	Smith 1973 [27]	Age, Educ
	Trail Making Test Part A	UDS v.3.0, June 2019	Age, sex, Educ.
	UDS Number Span Test	UDS v.3.0, June 2019	Age, sex, Educ.
Language	Animal Fluency	UDS v.3.0, June 2019	Age, Educ.
	UDS Multilingual Naming Test	UDS v.3.0, June 2019	Age, sex, Educ
Visuospatial	Judgment of Line Orientation (odd version)	Woodard et al. [28]	Age
	BVMT-R Copy	Benedict 1997 [23]	-

Abbreviations: UDS Uniform Data Set, Educ education

informants of brain donors [5, 7, 10, 13] and that are part of the TES research diagnostic criteria [10, 13]. Domains assessed included attention, visual scanning, and psychomotor speed; executive functions; learning and episodic memory (verbal and visual); language; and visuospatial abilities. Tests of memory and executive functions were overrepresented given these domains are known to be adversely affected by exposure to RHI [3, 10, 13]. Measures of performance validity and estimated pre-morbid intelligence were administered. Raw scores for all tests were generated according to NACC or test manual protocols. For all tests, the primary raw score outcome was total correct with the exception of Trail Making Test where completion time (in seconds) served as the primary outcome (number of errors is also reported). Neuropsychological test raw scores were converted to *T*-scores using normative data that accounted for age, sex, and education. A small number of tests only accounted for age or age and education. Table 1 provides the normative data source. A *T*-score ≤ 35 (i.e., 1.5 standard deviations [SD] below the normative mean) was considered impaired [14, 17, 31]. The *T*-score range was restricted to 20–80 to limit skewed distributions of the data and outliers.

Suboptimal performance validity was defined by below criterion performance on two out of the following three performance validity measures: Trial 2 of the Test of Memory Malingering (TOMM), reliable number span (modified due to use of the UDS Number Span task), and Neuropsychological Assessment Battery (NAB) List Learning Recognition Hits. Established cutoffs for defining performance validity failure on these measures were used but are not disclosed here to preserve test integrity. While failure of one performance validity test can be indicative of invalidity [32], our decision adheres to the revised Slick criteria (Sherman et al.) for identification of malingered neurocognitive dysfunction that advises failure of 2+ performance validity tests [33]. Of the sample with complete neuropsychological data, 10 (4.2%) had suboptimal performance on the TOMM, 8 (3.4%) had a below cutoff score on the reliable number span, and 50 (21.3%) fell below cutoff on the NAB Recognition Hits trial. Fifty (21.3%) failed at least one performance validity measure, six (2.6%) failed two, and only 2 (0.9%) failed all three. Of note, four participants had one performance validity test missing but were above the cutoff on the other two indices. Taken together, for the current sample, a total of 8 participants were excluded for suboptimal performance validity.

Sample characteristics

Semi-structured interviews were performed, supplemented by online questionnaires, to collect data on

demographics, medical and psychiatric history, athletic history, and other variables not relevant to the present study. An aliquot of whole blood was used for *APOE* genotyping. Race and ethnicity were self-reported. A majority of the sample was Black or White. There was insufficient representation of other racial groups to statistically examine them separately. All racial groups are presented in Table 2.

Statistical analyses

Descriptive statistics were used to characterize the neuropsychological test raw and *T*-scores. Rates of impairment on each test and/or test indices are reported, based on above-described cutoffs (e.g., *T*-score ≤ 35). A neuropsychological domain was considered impaired if at least two tests within that domain had a *T*-score ≤ 35 . This was only done for the attention, visual scanning, psychomotor speed domain, executive function domain, and the episodic memory domain as there was insufficient number of tests for the language and visuospatial domains (as designed). While we report the frequency of those who had one impaired score, our interpretation of an impaired domain is based on 2+ tests falling below the threshold given the high base rates of test impairments on large batteries among normative individuals [31, 35]. For the memory domain, measures that counted towards impairment included the long delay recall trials from the Brief Visuospatial Memory Test-Revised, Neuropsychological Assessment Battery List Learning task, and Craft Story 21 Recall (Paraphrase). Analysis of covariance controlling for age compared the former college and professional football players on the neuropsychological test raw scores. Statistical analyses were conducted using IBM SPSS Statistics, version 27.

Results

Sample characteristics are in Table 2. The sample included 170 former American football players (111 former professional football players, 59 former college football players). The sample of football players was 57.5 ($SD=8.1$) years old and had 16.7 ($SD = 1.5$) years of education, and 55 (32.4%) were Black or African American. Tables 3 and 4 show the neuropsychological test performance of the former American football players. Analysis of covariance controlling for age showed a statistically significant difference between the former college and professional football player groups on only three of the primary neuropsychological tests, all memory (Supplemental Tables 4 and 5), though the significant differences would not have survived multiple comparison adjustments. For this reason, the former college and professional American football players were combined and described as a single group.

Table 2 Sample characteristics

	Former football players (n = 170)	Former professional (n = 111)	Former college (n = 59)	Asymptomatic unexposed men(n = 57)	P-value ^d
Demographics					
Age, mean (SD) years	57.5 (8.1)	59.6 (7.6)	53.6 (7.7)	59.2 (8.3)	0.17
Education, mean (SD) years	16.7 (1.5)	16.6 (1.1)	17.0 (1.9)	17.4 (3.4)	0.14
Race ^a , n (%)					0.58
American Indian or Alaska Native	1.0 (0.6)	1.0 (0.9)	0.0	0.0	
Black or African American	55.0 (32.4)	45.0 (40.5)	10.0 (16.9)	21.0 (36.8)	
Native Hawaiian or other Pacific Islander	0.0	0.0	0.0	1.0 (1.8)	
White	110 (64.7)	63.0 (56.8)	47.0 (79.7)	35 (61.4)	
Multiple races	2.0 (1.2)	1.0 (0.9)	1.0 (1.7)	0.0	
Not reported	2.0 (1.2)	1.0 (0.9)	1.0 (1.7)	0.0	
Ethnicity, n (%)					--
Hispanic or Latino	3.0 (1.8)	3.0 (2.7)	0.0	0.0	
Body mass index , mean (SD) kg/m ²	32.5 (4.5)	31.8 (4.3)	33.7 (4.8)	30.8 (4.6)	0.02
Neurodevelopment					
Attention-deficit/hyperactivity disorder, n (%)	14.0 (8.2)	5.0 (4.5)	9.0 (15.3)	1.0 (1.8)	--
Learning disability, n (%)	5.0 (2.9)	2.0 (1.8)	3.0 (5.1)	0.0	--
Athletic					
Total years of football, mean (SD) years	15.8 (4.4)	18.0 (3.3)	11.5 (2.6)	-	--
Age of first exposure to football, mean (SD) years	11.1 (2.8)	11.6 (2.7)	10.2 (2.6)	-	--
Primary position at highest level of play, n (%)					--
Offensive lineman	42.0 (24.7)	20.0 (18.0)	22.0 (37.3)	-	
Offensive back or receiver	44.0 (25.9)	31.0 (27.9)	13.0 (22.0)	-	
Defensive lineman	19.0 (11.2)	14.0 (12.6)	5.0 (8.5)	-	
Linebacker	27.0 (15.9)	20.0 (18.0)	7.0 (11.9)	-	
Defensive back	34.0 (20.0)	22.0 (19.8)	12.0 (20.3)	-	
Special teams	4.0 (2.4)	4.0 (3.6)	0.0	-	
Reported number of post-definition concussions, median (IQR) ^b	30 (88)	30 (88)	25 (88)	--	--
APOE genotype					
ε4 carrier ^c , n (%)	49.0 (28.8)	30.0 (27.0)	19.0 (32.2)	11.0 (19.3)	0.21

The sample excluded participants who had suboptimal performance on 2+ performance validity tests and was restricted to participants who had complete data on all primary objective neuropsychological tests (n = 13 with missing data [9 former professional, 1 former college, 3 asymptomatic unexposed men]).

^a n = 2 with missing data (2 former professional)

^b All participants were read a definition of concussion prior to being asked to estimate their total number of concussions [34] (n=1 with missing data, former professional football player)

^c n = 9 with missing data (4 former professional, 1 former college, 4 asymptomatic unexposed men)

^d Independent samples t-test compared the former American football players and the asymptomatic unexposed men on continuous outcomes and chi-square was used for binary outcomes

As previously described, participants who had suboptimal performance validity on 2+ measures were excluded from the sample (n=8). However, there remained four participants who had suboptimal scores on TOMM Trial 2 (scores ranged from 32 to 38). These four participants were retained given their adequate performances on the other two validity tests, including reliable number span (scores ranged from 7 to 11) and NAB List Learning Recognition Hits (percentiles ranged from 13 to 50).

Estimated premorbid intelligence

Based on the Wide Range Achievement Test, 4th Edition (WRAT-4) standard score, the estimated premorbid intelligence of the former football players fell in the average psychometric range. Fourteen had below average standard scores (i.e., <85). Five (2.9%) reported a diagnostic history of a learning disability, two of whom had a below average standard score on the WRAT-4.

Table 3 Baseline neuropsychological test performance of former American football players

Domain	Test/instrument	Former American football players (<i>n</i> = 170)			
		Raw			T-score Mean (SD)
		Mean (SD)	Min	Max	
Performance validity	TOMM Trial 2	49.5 (2.4)	32	50	-
	UDS Reliable Number Span	9.9 (2)	6	16	-
	NAB List Learning Recognition	10.4 (1.4)	6	12	37.3%ile (28.2) ^a
Est. premorbid intelligence	WRAT-4 ^{b,c}	62.4 (5.5)	39	70	102.9 (12.7) ^b
	BVMT-R Trials 1–3	20.9 (7.2)	6	35	46.3 (12.6)
Learning and memory	BVMT-R delayed recall	8.6 (2.8)	0	12	49.8 (12.5)
	NAB List Learning trials 1–3	19.6 (4.7)	9	31	40.4 (9.3)
	NAB List Learning SDR	5.7 (2.6)	0	11	39.6 (12.1)
	NAB List Learning LDR	5.2 (2.9)	0	11	38.8 (12.5)
	Craft Story Immediate (Verbatim)	18.8 (6.1)	1	33	44.3 (9.1)
	Craft Story Immediate (Paraphrase)	14.7 (3.9)	1	24	44.8 (9.6)
	Craft Story Delay (Verbatim)	15.7 (6.1)	0	30	43 (9.2)
	Craft Story Delay (Paraphrase)	13.1 (4.2)	0	21	43.2 (9.8)
	COWAT	41.2 (11.4)	3	76	48 (9.6)
	SCWT Interference ^d	36.6 (9.7)	11	67	48.4 (6.6)
Executive function	NAB Mazes	16.0 (6.2)	2	26	53.9 (10.2)
	Trail Making Test Part B	80.4 (43.4)	29	300	44.7 (10.6)
	Trail Making Test Part B errors	0.6 (0.9)	0	4	-
	Symbol Digit Modalities Test	47.4 (9.6)	24	75	48.4 (10.3)
	Trail Making Test Part A	30.2 (10.6)	12	72	44.5 (10.3)
Attention, visual scanning, and psychomotor speed	Trial Making Test Part A errors	0.2 (0.4)	0	2	-
	UDS Number Span Test: Forward	8.5 (2.1)	3	14	49.2 (8.8)
	UDS Number Span Test: FL	6.8 (1.2)	4	9	49.6 (9.6)
	UDS Number Span Test: Backward	6.8 (2.2)	2	13	46.2 (10.1)
	UDS Number Span Test: BL	4.9 (1.3)	2	8	46.2 (10)
	UDS Number Span Test: RL	4.9 (1.3)	2	8	46.2 (10)
Language	Animal Fluency	21 (5.4)	6	35	46 (9.4)
	Multilingual Naming Test	29.2 (2.5)	7	32	43.5 (9.9)
Visuospatial ability	Judgment of Line Orientation (odd version)	12.7 (2.3)	5	15	53 (9.9)
	BVMT-R Copy ^a	11.7 (0.7)	9	12	-

The sample excluded participants who had suboptimal performance on 2+ performance validity tests and was restricted to participants who had complete data on all primary objective neuropsychological tests (across all DIAGNOSE CTE Research Project study groups)

Abbreviations: TOMM Test of Memory Malingering, RH Recognition Hits, WRAT-4 Wide Range Achievement Test-Fourth Edition Word Reading, BVMT-R Brief Visuospatial Memory Test-Revised, NAB Neuropsychological Assessment Battery List Learning [SDR Short Delay Recall, LDR Long Delay Recall], UDS Uniform Data Set, COWAT Controlled Oral Word Association Test, SCWT Golden Stroop Color and Word Test, FL Forward Longest Span, BL Backward Longest Span

^a Percentile reported

^b *n* = 1 former football player with missing data for WRAT-4 and BVMT-R Copy

^c Standard score reported for WRAT-4

^d *n* = 2 with missing data due to colorblindness

Learning and episodic memory

The sample mean *T*-scores for NAB List Learning Trials 1–3 and NAB List Learning Short and Long Delay recall trials were all ~40. Thirty-six (21.2%) had impaired episodic memory, representing the domain with the highest rates of impairment. Eighty-eight (51.8%) had at least one test impaired in episodic memory. Of the sample, impairments were frequent on the

NAB List Learning Trials 1–3 (30.6%, *n*=52) and on the NAB List Learning Short Delay (37.6%, *n*=64) and Long Delay recall trials (44.7%, *n*=74). The participants recalled a mean of 5.2 (of 12) words after a long delay recall. On the recognition trial, 23 participants (13.8%) had impaired false positive errors (mean = 4.8, SD = 3.8) and 33 participants (19.8%) had impaired recognition hits (mean = 10.4, SD = 1.4).

Table 4 T-score distributions of baseline neuropsychological test performance of former American football players

Domain	Test/instrument	Former American football players (n = 170), n (%)			
		≤ 35	36–39	40–49	≥ 50
Learning and memory	BVMT-R Trials 1–3	37 (21.8)	16 (9.4)	44 (25.9)	73 (42.9)
	BVMT-R delayed recall	29 (17.1)	6 (3.5)	41 (24.1)	94 (55.3)
	NAB List Learning trials 1–3	52 (30.6)	19 (11.2)	71 (41.8)	28 (16.5)
	NAB List Learning SDR	64 (37.6)	32 (18.8)	35 (20.6)	39 (22.9)
	NAB List Learning LDR	76 (44.7)	10 (5.9)	47 (27.6)	37 (21.8)
	Craft Story Immediate (Verbatim)	25 (14.7)	25 (14.7)	73 (42.9)	47 (27.6)
	Craft Story Immediate (Paraphrase)	27 (15.9)	19 (11.2)	69 (40.6)	55 (32.4)
	Craft Story Delay (Verbatim)	26 (15.3)	28 (16.5)	78 (45.9)	38 (22.4)
	Craft Story Delay (Paraphrase)	32 (18.8)	16 (9.4)	73 (42.9)	49 (28.8)
Executive function	COWAT	11 (6.5)	23 (13.5)	69 (40.6)	67 (39.4)
	SCWT Interference ^a	6 (3.6)	9 (5.4)	82 (48.8)	71 (42.3)
	NAB Mazes	7 (4.1)	8 (4.7)	41 (24.1)	114 (67.1)
Attention, visual scanning, and psychomotor speed	Trail Making Test Part B	35 (20.6)	12 (7.1)	58 (34.1)	65 (38.2)
	Symbol Digit Modalities Test	20 (11.8)	9 (5.3)	56 (32.9)	85 (50)
	Trail Making Test Part A	32 (18.8)	10 (5.9)	65 (38.2)	63 (37.1)
	UDS Number Span Test: Forward	4 (2.4)	15 (8.8)	74 (43.5)	77 (45.3)
	UDS Number Span Test: FL	14 (8.2)	10 (5.9)	57 (33.5)	89 (52.4)
	UDS Number Span Test: Backward	25 (14.7)	24 (14.1)	62 (36.5)	59 (34.7)
Language	UDS Number Span Test: BL	24 (14.1)	6 (3.5)	89 (52.4)	51 (30)
	Animal Fluency	29 (17.1)	16 (9.4)	71 (41.8)	54 (31.8)
Visuospatial	Multilingual Naming Test	36 (21.2)	18 (10.6)	77 (45.3)	39 (22.9)
	Judgment of Line Orientation (odd version)	12 (7.1)	6 (3.5)	27 (15.9)	125 (73.5)

The sample excluded participants who had suboptimal performance on 2+ performance validity tests and was restricted to participants who had complete data on all primary objective neuropsychological tests

Abbreviations: BVMT-R Brief Visuospatial Memory Test-Revised, NAB Neuropsychological Assessment Battery List Learning [SDR Short Delay Recall, LDR Long Delay Recall], UDS Uniform Data Set, COWAT Controlled Oral Word Association Test, SCWT Golden Stroop Color and Word Test, FL Forward Longest Span, BL Backward Longest Span

^a n = 2 with missing data due to colorblindness

Compared with learning and memory for unstructured verbal stimuli, learning and memory of structured contextualized information (i.e., a story) were better. The sample mean T-scores were in the average psychometric range for Craft Story 21 Immediate and Delay Recall trials (for both paraphrase and verbatim). Rates of impairments were approximately 15% (n=25) for Craft Story 21 Recall Immediate and Delay trials with impairment rates highest for Craft Story 21 Recall Delay Paraphrase (18.8%, n=32).

There was better visual than verbal memory test performance. The sample mean T-scores on indices of learning and episodic memory for figures (BVMT-R) fell in the average psychometric range. Of the sample, 21.8% (n=37) and 17.1% (n=29) had impairments on BVMT-R Trials 1–3 and BVMT-R Delay Recall, respectively. Recognition hits (mean = 5.7, SD = 0.7) and false alarms (mean, SD = 0.1, 0.4) were overall intact with few participants having scores in the impaired range (n = 6 [3.5%] for hits, n = 4 [2.4%] for false alarms).

Executive functions

Mean T-scores were in the average psychometric range and 12 (7.1%) had impaired executive function. Forty-five (26.5%) had at least one test impaired in this domain. Rates of impairments were highest for Trail Making Test Part B (20.6%). On Trail Making Test Part B, 37 had one error, 14 had two errors, and 9 had 2+ errors. Less than 10% of the sample had impaired performance across all other tests.

Attention, visual scanning, psychomotor speed

The sample mean T-scores fell in the average psychometric range for all neuropsychological tests administered in this domain. Twenty-one (12.4%) participants were impaired. Fifty-five (32.4%) had one test impaired and rates of impairments ranged from 2.4% (n=4) on UDS Number Span Forward total correct trials to 18.8% (n=32) on Trail Making Test Part A. On Trail Making Test Part A, 29 participants had one error and two participants had two errors.

Language

The sample mean *T*-scores for measures of semantic fluency (Animal Fluency) and confrontation naming (Multilingual Naming Test, MINT) were in the average psychometric range. Regarding rates of impairments, 21.2% ($n=36$) and 17.1% ($n=29$) of the sample were impaired on the MINT and Animal Fluency, respectively.

Visuospatial

Only 7.1% ($n=12$) were impaired on the Judgment of Line Orientation test. Gross visuospatial abilities on the BVMT-R Copy were intact as raw scores ranged from 9 to 12 (of 12).

Multidomain impairments

We examined rates of multidomain impairments among the memory, attention, visual scanning and psychomotor speed, and executive function domains. Based on our definition of impairment (i.e., 2+ tests impaired), 25 (14.7%) had 1 domain impaired and 20 (11.8%) of the football players had 2 or more domains that were impaired.

Discussion

This study examined the neuropsychological test performance of 170 male former college ($n=59$) and professional ($n=111$) football players (ages 45–74), most of whom had subjective cognitive concerns. Impairments were identified using established normative data that account for age, sex, and education. Episodic memory was the most frequently impaired cognitive domain, particularly memory of unstructured verbal information (i.e., NAB List Learning). Compared with unstructured verbal stimuli, learning and recall of contextual verbal stimuli (i.e., Craft stories) and visual information (i.e., BVMT-R figures) were better but impairments still frequent. Other domains with impairments included attention and psychomotor speed (i.e., Trail Making Test Part A) and set-shifting and mental flexibility (Trail Making Test Part B). With the exception of Trail Making Test Part B, performances on tests of executive functions and on visual-perceptual abilities were otherwise preserved.

The results of this study have several implications. Previous research has shown that more than one-third of NFL retirees report being “extremely concerned” about memory and thinking skills [36]. A majority of this sample also had subjective cognitive concerns. Our finding that performance on memory tests was the most frequently impaired is similar to other neuropsychological studies of former NFL players [3]. The mean performance of the word list learning test was at a level of impairment comparable to what is seen in patients with mild cognitive impairment (MCI) [37]. This finding, in combination with less significant reductions on scores on

psychomotor speed, confrontation naming, and semantic fluency suggests a neuropsychological profile that resembles an amnestic form of MCI in this sample of former college and professional football players with a mean age of 58, similar to what has been suggested by other investigators [38].

The 2021 NINDS Consensus Diagnostic Criteria for TES include impairments in episodic memory and/or executive functions as core clinical features [10]. One surprising result from this study was that, with the exception of Trail Making Test Part B, performance on tests of executive functions was relatively preserved. While this finding might provide additional support for a neurocognitive profile consistent with amnestic MCI, it might also be an effect of some of the well-known limitations in the neuropsychological assessment of executive functions. Studies that have examined many of the most commonly used tests of executive functions find only modest correlations among the tests suggesting that these functions are difficult to measure as they do not combine neatly into a unitary factor [39]. There are also indications that tests of executive functions often fail to correspond to behavioral ratings of dysexecutive behavior, raising questions about the ecological validity of the measures [40]. In theory, one would expect individuals with the “neurobehavioral dysregulation” of TES to be impaired most specifically on measures of impulsive responding. There was no evidence of impairment in this study on tasks like the Golden Stroop Color Word Interference measure, a well-known index of cognitive impulsivity.

This study included former college and professional football players. There was no statistically significant difference (with consideration of multiple comparisons) between the former college and professional American football players across any of the neuropsychological tests, but there were trends for worse performance in former professional football players. Former professional American football players, and primarily former NFL players, have been the focus of studies on the long-term neuropsychological consequences of American football play [3]. This is one of the first studies to feature middle aged to older adult former college football players without subsequent professional experience or other RHI exposure after college. From a public health perspective, it is critical to elucidate the long-term health outcomes of college football players given that approximately 800,000 student athletes have played college football in the USA since 1960, 250,000 of whom are currently older than 60 years of age [41]. Moreover, a recent health outcome survey study found a significantly higher prevalence of cognitive impairment disorders in former college football players compared to the general population, a finding similar to previous studies of former NFL players [41].

A challenge in the field of neuropsychology is the appropriate selection of normative data to derive standardize scores to establish levels of impairment. Here, normative data used included those from the specific test manuals, as well as from the NACC for UDS measures. A majority of normative data accounted for age, sex, and education. However, there were variations in normative adjustments across tests that could have influenced impairment rates by test and domain. Race-based norming was *not* performed. Race-based norming has been incorporated into the training and practice of neuropsychology since at least the 1990s (e.g., Heaton Norms) based on the assumption that race may be a proxy for socioeconomic factors associated with cognitive function. For people who identify as Black, race-based norming results in a stricter threshold needed to be designated as cognitively impaired compared with Whites. The differential treatment of Blacks when scoring and interpreting neuropsychological tests has been a controversial practice [42]. Recently, the NFL ended its use of race-based neuropsychological test norms to determine monetary compensation as part of the NFL Concussion Settlement. The use of race-based norms as part of a rigid algorithm that is void of clinical judgment to determine compensation perpetuates systemic racial injustice and inequity [43]. Prior to consideration of normative data, a majority of neuropsychological tests were developed in White populations, placing Black Americans at initial disadvantage from the beginning. A study is currently underway that is modeling the neuropsychological differences by race in this sample, along with relevant psychosocial, socioeconomic, social, and health factors that might explain observed differences.

There are limitations to the present findings. The asymptomatic unexposed men were required to have no reported symptoms to be eligible for the DIAGNOSE CTE Research Project. While recruitment of the former football players was not based on symptomatic status, most have subjective cognitive (and neuropsychiatric) concerns. This design is appropriate for biomarker development but it limits meaningful comparisons and interpretations on neuropsychological measures between groups as any observed differences could be biased by our recruitment methods. For this reason, statistical comparisons of the former American football players and the asymptomatic unexposed men were not performed [20]. The use of normative data circumvents limitations of study design and informs on rates of neuropsychological impairments among former elite football players. Our ability to make inferences on whether impairments are from pathology and a function of exposure to RHI is challenging given the recruitment design, lack of biomarkers, and in the context of the test performance of

the asymptomatic unexposed men. Although impairments were generally infrequent in the unexposed men, approximately 25% and 21% were impaired on BVMT-R Learning Trials and NAB List Learning Long Delay Recall trial, respectively. While the presence of neurological disease in this group cannot be ruled out, it might also be a function of the number of neuropsychological tests administered [31, 44–46]. In the present battery of close to 15 separate correlated indices, rates of impairments in the entire sample might be inflated due to type I error. We also excluded eight participants who had evidence of suboptimal performance on 2+ performance validity tests, based on the revised Slick criteria [33]. Four participants in the sample had suboptimal performance on the TOMM Trial 2 but not on any of the remaining validity indices. We acknowledge that failure of just one validity test can be indicative of invalidity and could have contributed to inflated impairment rates [32]. However, the use of 2+ tests to define invalidity is more stringent, followed recommended guidelines, and performance invalidity rates in this sample were overall low and did not influence the results.

The current study did not include a disease comparison group (e.g., Alzheimer's disease), which is needed to determine the specificity of the observed neuropsychological profiles and facilitate differential diagnosis. The sample includes individuals who volunteered to participate in research. Most of the male former football players had concerns about their cognitive function, mood, and/or behavior. External validity to the general football population, as well as to women and other athlete populations is limited. We used 1.5 SD below the normative mean to define impairment, a generally accepted convention [14, 17]. We recognize that a continuum exists. Finally, cognitive function was measured using traditional paper-and-pencil tests that might have lacked adequate sensitivity to capture certain impairments. The absence or low rate of impairments in certain domains (e.g., executive functions, visuospatial abilities) might be related to measurement. While digital phenotyping currently lacks clinical applicability, it is an exciting avenue of future research.

Conclusions

In this sample of 170 male former elite American football players, a comprehensive neuropsychological assessment revealed most frequent impairments in learning and recall for unstructured verbal stimuli. Continued efforts are needed to characterize the neuropsychological profile of individuals exposed to RHI to assist neuropsychologists and other clinicians in disease detection and differential diagnosis. Additional research that includes a disease comparison (e.g., Alzheimer's disease) and

examines causes of neuropsychological impairment in this population is needed. Development of tests sensitive to the specific executive functions disturbed in this population is also an important target for future research. Such development should include and extend beyond traditional paper-and-pencil tests which might not be adequate for the identification of certain impairments in this population.

Abbreviations

AD: Alzheimer's disease; BVMT-R: Brief Visuospatial Memory Test-Revised Version; CTE: Chronic traumatic encephalopathy; FTD: Frontotemporal dementia; MCI: Mild cognitive impairment; NAB: Neuropsychological Assessment Battery; NACC: National Alzheimer's Coordinating Center; NFL: National Football League; PET: Positron emission tomography; RHI: Repetitive head impacts; SD: Standard deviation; TES: Traumatic encephalopathy syndrome; TOMM: Test of Memory Malingering; UDS: Uniform Data Set; WRAT-4: Wide Range Achievement Test, 4th Edition.

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s13195-022-01147-9>.

Additional file 1: Supplemental Table 1. Self- and Informant-Reported Symptomatic Status of Participants at Time of Study Screening. **Supplemental Table 2.** Baseline Neuropsychological Test Performance of the Asymptomatic Unexposed Men. **Supplemental Table 3.** T-Score Distributions of Baseline Neuropsychological Test Performance of the Asymptomatic Unexposed Men. **Supplemental Table 4.** Baseline Neuropsychological Test Performance of the Former College and Professional Football Players. **Supplemental Table 5.** T-Score Distributions of Baseline Neuropsychological Test Performance for Former College and Professional Football Players.

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MLA, WBB, and RAS designed the study and drafted the manuscript. MLA, WBB, SVP, JC, YT, and MM analyzed and interpreted the data as well as assisted

with the presentation of the data. YT, JNP, BM, KH, and SVP assisted with data set creation and management. Roles in data acquisition included MLA, WBB, SJB, JVW, SVP, CHA, LJ, MLM, JLC, EMR, MES, and RAS. JLC, EMR, MES, and RAS conceived the overall study design of the DIAGNOSE CTE Research Project. All authors critically read and helped to revise the manuscript and assisted with the interpretation of the data. All authors approved the submitted version and agree to be accountable for the work.

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Availability of data and materials

The datasets generated and analyzed during the current study will be available in the Federal Interagency Traumatic Brain Injury Research (FITBIR) repository, <https://fitbir.nih.gov>. Datasets will also be available through a data-sharing portal for the DIAGNOSE CTE Research Project, <http://diagnosecte.com>. It is also anticipated that study datasets will be available in the Global Alzheimer's Association Interactive Network (GAAIN) repository, <http://www.gaan.org>.

Declarations

Ethics approval and consent to participate

All Participant Evaluation Sites received approval by their respective Institutional Review Boards. All participants provide written informed consent during their baseline and follow-up study visits.

Consent for publication

Not applicable.

Competing interests

CHA consulted for Avion, CND Life Sciences, Jazz, and Precon Health. LJ is Editor-in-Chief of the *Journal of Neuro-Ophthalmology* and is a paid consultant to Biogen (Cambridge, MA, USA). CB receives research support from the Ultimate Fighting Championship, Top Rank promotions, Haymon Boxing, Las Vegas Raiders, and Professional Bull Riders. He is a paid consultant for Aurora Concussion Therapy Systems, Inc. (St. Paul, MN). RA is a paid consultant to Biogen (Cambridge, MA, USA) and serves on the Scientific Advisory Board of Signant Health (Blue Bell, PA). WBB provides expert witness testimony in legal cases involving concussion and CTE. DWD reports the following conflicts: consulting: Amgen, CapiThera, Cerecin, Ceruvia Lifesciences, Cooltech, Ctrl M, Allergan, Abbvie, Biohaven, GSK, Lundbeck, Eli Lilly, Novartis, Impel, Satsuma, Theranica, WL Gore, Genentech, Nocira, Perfood, Praxis, Pfizer, AYYA Biosciences, and Revance; honoraria/expense reimbursement/royalties: American Academy of Neurology, Headache Cooperative of the Pacific, MF Med Ed Research, Biopharm Communications, CEA Group Holding Company (Clinical Education Alliance LLC), Teva (speaking), Amgen (speaking), Eli Lilly (speaking), Lundbeck (speaking), Vector psychometric Group, Clinical Care Solutions, CME Outfitters, Curry Rockefeller Group, DeepBench, Global Access Meetings, KLJ Associates, Academy for Continued Healthcare Learning, Majallin LLC, Medlogix Communications, Medica Communications LLC, MJH Lifesciences, Miller Medical Communications, Synapse, WebMD Health/Medscape, Wolters Kluwer, Oxford University Press, and Cambridge University Press; non-profit board membership: American Brain Foundation, American Migraine Foundation, ONE Neurology, Precon Health Foundation, International Headache Society Global Patient Advocacy Coalition, Atria Health Collaborative, Domestic Violence HOPE Foundation/Panfila, CSF Leak Foundation. Research Support: Department of Defense, National Institutes of Health, Henry Jackson Foundation, Sperling Foundation, American Migraine Foundation, and Patient Centered Outcomes Research Institute (PCORI); stock options/shareholder/patents/board of directors: Ctrl M (options), Aural analytics (options), ExSano (options), Palion (options), Healint (options), Theranica (options), Second Opinion/Mobile Health (options), Epien (options/board),

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Positive Effects of Religion and Social Ties on the Health of Former NFL Athletes

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Abstract

This study explores the relationship between religious service attendance, social ties, and health among former NFL players, a population with relatively high levels of religious attendance who endure physically demanding occupations. Research shows that frequent religious service attenders tend to have better health, partly because of social connections formed through religious involvement. We analyzed a sample of 1029 former NFL players. Consistent with previous research, bivariate and multivariate OLS regression models show that frequent religious attenders have statistically significantly better self-rated health. However, this relationship is moderated by social ties. Respondents who scored lower on the social ties index exhibited a stronger significant relationship between frequent religious attendance and health; those scoring higher on the social ties index exhibited no relationship between frequent attendance and health. Future research should examine how benefits of religious attendance vary depending upon strength of social relationships.

Keywords Social ties · Self-rated health · Religious service attendance · NFL athletes

Introduction

A large body of research shows that religion is positively associated with a range of favorable health outcomes. While religious participation takes many forms, among all measures of religion thought to benefit health, attendance at religious services is the most powerful predictor of health and mortality (George et al., 2002; Koenig et al., 2012; Rogers et al., 2010). One pathway through which religious attendance

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influences health is via social relationships, which provide social support, social control, and social capital (Idler, 2014). Research outside the study of religion shows that close relationships, or “strong” ties with primary groups, such as family, friends, and significant others, are reliable predictors of better health (Cohen, 2004). However, secondary relationships that form within groups and organizations, characterized by more formality and less intimacy and considered “weak ties,” are another potential mechanism linking social ties and health (Thoits, 2011). To date, much of the research on religious service attendance considers religion as a source of strong ties (Ellison & George, 1994; Lim & Putnam, 2010). Research is lacking on how religious service participation, in the absence of strong social ties, might yield a strong health effect (McCullough & Laurenceau, 2005), in light of evidence of the “strength of weak ties” for a range of social outcomes (Granovetter, 1973; Tindall & Malinick, 2017). Thus, we examine how the effects of religious service attendance vary across levels of relational closeness and frequency of contact with friends and family as determinants of physical health among former National Football League (NFL) players. In particular, we find that religious service attendance is most consequential for overall health among players reporting less frequent contact and closeness of relationships with friends and family. This finding is important because it points to religious practices and institutions as a potential health benefitting resource for individuals who have few, or altogether lack, strong social ties.

Former NFL players are an ideal focus for the study of religion and health because they endure extraordinary levels of physical wear and tear during their playing years but also have high levels of religious participation. These men experience remarkable rates of injury, with an estimated league-wide average of over 1,500 injuries during the regular season (Deubert et al., 2016). Notably, former players report pain at a rate three times greater than the general population (Cottler et al., 2011; Schwenk et al., 2007). Yet despite the accumulation of physical injury from their careers, former NFL players report better self-rated health than the general population of men in the United States (U.S.) (Weir et al., 2009). In addition, former NFL players show significantly higher rates of religious service attendance compared to men in the U.S. general population (Weir et al., 2009). This is also true for players during their careers, despite common media portrayals (Carter & Carter, 2014). Our analysis comparing age and race subgroups of former players shows higher rates of frequent religious attendance in younger age cohorts. Former NFL players over age 65 are more similar to their counterparts in the U.S. population for whom religious service attendance tends to be higher (Cupery, 2020). Other research finds that former elite athletes more generally are twice as likely to rate themselves as very religious compared to all U.S. men (Desmarais-Zalob, 2014). We explore the relationship of religion to players’ self-rated health.

Association of Religious Involvement and Health

Research consistently demonstrates the favorable effects of religious service attendance on health and longevity (George et al., 2002; Suh et al., 2019), overall mortality (Krause, 1998, 2006), an array of mental and physical health indicators (Carr,

2003; Hill et al., 2016) and self-rated health (Doane & Elliott, 2015; E. L. Idler et al., 2009; Krause, 2006). Religious involvement has also been found to promote increased happiness (Stark & Maier, 2008), life satisfaction ((Ellison & Gay, 1990; Krause, 2003), and general well-being (Hummer et al., 1999; Krause, 2004; ; ; Ellison and Hummer, 2010).

One mechanism by which religious involvement affects health is through social ties. Over four decades of research have provided evidence for the beneficial link between social ties and health (Berkman & Glass, 2000; Heaney & Israel, 2002; House, 1981; House et al., 1988; Thoits, 2011; Wiliams & House, 1991). Generally, having more or closer ties is predictive of better health, compared to having fewer or weaker ties, even without accounting for the positive or negative nature of the ties. Previous research suggests that religious involvement can be a source of strong ties through the provision of social support, since frequent attendance typically lends itself to forming close connections with other congregation members (Ellison & George, 1994; Lim & Putnam, 2010). However, not all relationships formed through religious association can be assumed to be strong ties.

Social connections established through religious involvement may include qualitatively weaker ties that people would not include when asked about their close relationships, yet may still be consequential to their state of affairs as originally described by Granovetter (1973). According to Fingerman and Tennant (2016), more casual acquaintances established through formal group participation can serve a variety of important functions, such as providing a sense of familiarity, predictability, or comfort, or substituting in other ways for more intimate relationships that individuals may be lacking, especially later in life (Fingerman, 2004, 2009; Fingerman et al., 2011; F. R. Lang & Fingerman, 2003). Such social connections may be increasingly important in the context of declining institutional involvements (Putnam, 2000), where people have fewer sources of weak ties.

While prior research has generally viewed social ties as playing a mediating role between religious involvement and health, a contingent relationship is also possible. Chan et al. (2019), in a study of purpose in life among adults, found that religious beliefs were associated with greater sense of purpose only for socially-disconnected people. Along similar lines, it may be that weak ties gained through religious involvement play a compensatory role for the health of people with a dearth of strong social ties. Our study explores this possibility by examining whether health benefits are associated with religious service attendance for ex-NFL athletes with varying closeness and sources of social ties.

Methods

Data and Sample

We analyzed data from a survey of former NFL players to understand the relationship between their religious service attendance, social ties, and self-rated health. Data come from the NFL Player Care Foundation Study of Retired Players (Weir et al., 2009), a stratified random sample of retired players with vested rights

in the NFL's pension system. Vested players in this sample have typically had a minimum of 4 years of active League play. The Foundation provided a complete listing of 6983 vested retired players (as of 2006–2007) from which a stratified random sample of 1625 players was selected for telephone interviews conducted by survey staff of the Institute for Social Research at the University of Michigan. Many components of the interview were drawn from established national health surveys conducted by telephone, such as the National Health Interview Survey, to allow comparison with general population survey data for other men of the same age and race. Stratification of the NFL study participants was based on age group and whether a player was on disability pension from the NFL. The field period was November to December, 2008, with 1,063 completed interviews for a final response rate of 65.4%. All respondents provided written informed consent, and the study protocol was approved by the University of Michigan Institutional Review Board (IRB).

Measures

Self-rated Health

The major outcome of interest was respondents' subjective self-rated health status. The question asked "Would you say your health in general is excellent, very good, good, fair, or poor?" The variable was reverse-coded so that a higher number indicated better health (range 1–5). A single self-rated health measure is widely used to indicate general health status and is a significant predictor of objective health status, physical functioning, physical symptoms, mental health, psychological well-being, longevity, and use of health services (Idler & Cartwright, 2018; Marsden & Campbell, 2012). Although self-rated health measures have been shown to have high validity and reliability (Schnittker & Bacak, 2014), Zajacova and Dowd (2011) raise concerns about the reliability of such measures, in particular the tendency for less consistent reporting among survey respondents who report their health to be either good, very good, or excellent. However, the slippage they identify is mostly concentrated among respondents without a bachelor's degree. Since 78% of the former NFL player sample has a bachelor's degree, the use of self-rated health is warranted in this population.

Religious Service Attendance

Respondents were asked "How often do you attend religious services?" A 10-point ordinal response scale ranged from 1 = "Never," 2 = "Less than once a year," 3 = "Once a year," 4 = "Several times a year," 5 = "Less than once a month," 6 = "Once a month," 7 = "Two or three times a month," 8 = "Nearly every week," 9 = "Once a week," and 10 = "More than once a week." As an indicator of

frequent religious attendance, we created a dichotomous variable with 0 = 1 to 7 and 1 = 8 to 10.

Spirituality

Respondents were asked “How spiritual would you say you are—very spiritual, fairly spiritual, not too spiritual, or not spiritual at all?” Response categories were 1 = “Very spiritual,” 2 = “Fairly spiritual,” 3 = “Not too spiritual,” and 4 = “Not spiritual at all.”

Religiosity

Respondents were asked “How religious would you say you are—very religious, fairly religious, not too religious, or not religious at all?” Response categories were 1 = “Very religious,” 2 = “Fairly religious,” 3 = “Not too religious,” and 4 = “Not religious at all.”

Social Ties

We selected indicators corresponding to Granovetter’s (1973) conception of tie strength to create an index of social ties based on responses to questions about connectedness in relationships with family members and friends (Marsden & Campbell, 1984). For each set of relationships, questions assessed sense of closeness by asking “How close do you feel toward your [family members/friends]?” The 4-category response scale for these two questions ranged from “Not close at all” to “Very close.” Two questions asked about frequency of contact with family and friends. The question for family asked “How often do you see, write or talk on the telephone with family or relatives who do not live with you?” with a 6-category response scale ranging from “Hardly ever or Never” to “Nearly everyday.” The question for friends asked “How often do you see, write or talk on the telephone with your friends?” had a 7-category response scale ranging from “Never” to “Nearly everyday.” Responses were recoded so that a higher value corresponded to a higher rating of ties, standardized using z-scores so that the items with greater scale range do not contribute more variance and summed to create an index.

Ability to Get Out

The survey included the Nagi scale of self-reported physical functioning (Nagi, 1976). Two items in this scale serve as indicators of the respondent’s perceived ability to get out to do a range of activities. These two questions were “By yourself, and without any special equipment, how difficult is it for you to” (a) get out to things like shopping, movies, or sporting events? and (b) participate in social activities such as visiting friends, attending clubs and meetings, going to parties? The response format included 0 = “Can’t do at all,” 1 = “Very difficult,” 2 = “Somewhat difficult,”

3 = "Only a little difficult," and 4 = "Not at all difficult." A small number of respondents said "do not do." We coded these responses as "1" for "get out to do things like shopping," and "2" for "participate in social activities," because it is more likely that some physically capable people avoid leaving the house for social activities than for shopping. We averaged the two items to create a new variable with a range of 0 to 4, measuring general ability to get out and do things.

Sociodemographic Characteristics

Sociodemographic controls included: respondent age in years (range 27 to 91); dichotomous indicators for white race (95% of non-white are black), college graduate (yes/no), and currently married (yes/no). Total household income and net household wealth were computed based on a series of questions on various sources of income (including wages, royalties, dividend income, pension income, etc.) and assets (including value of home, vehicles, investments, retirement accounts, etc., minus any debts). These values were reported in 2008 dollars in the table of descriptive results. The natural log of total assets and total income were used in the regression models.

Analysis

Descriptive statistics were generated on all study variables. Bivariate relationships between each study predictor (or series of indicator variables) and self-rated health were conducted using ordinary least squares (OLS) regression with the R-square ratio used to describe the amount of variance each study variable accounted for on its own. We conducted a series of linear regression models. Model 1 examined the relationship between frequent religious attendance and self-rated health in the full sample, accounting for the effect of social ties and the sociodemographic controls (college graduate, continuous age, race, total income (logged), total wealth (logged), current marital status, and religious affiliation). Model 2 added the ability to get out to address a possible confounder of the relationship between religious attendance and health; people who have difficulty leaving home may have poor health and be less likely to attend religious services.

To explore the interplay between social ties and religious attendance in relation to self-rated health, we divided the sample at the median value of the social ties index (514 cases above and 515 below), and created four indicator variables representing a 2×2 matrix of higher/lower scores on the social ties index and frequent/infrequent religious service attendance. We included three of the four resulting indicator variables in the model, leaving as a reference group those who scored lower on the social ties index and were not frequent religious attenders. This set of indicator variables replaced the measures of frequent religious attendance and the social ties index in Models 3 and 4, and allowed us to examine the joint effects of frequent religious attendance and social ties. This is mathematically equivalent, with the same coefficients and p-values, to an interaction between two dichotomous variables.

We included a dichotomous indicator for missing cases on the question regarding religious service attendance. This variable was coded 1 for those who were missing data on the religious attendance question and 0 for all other cases. This allows us to retain another 34 respondents who had complete data on all other study variables. Because we used a dichotomous measure of frequent religious service attenders, we can include another indicator measure related to attendance, and thus keep a greater portion of the original sample. The coefficient on *frequent attender* is still interpreted compared to infrequent attenders, so the results on this variable of interest were not affected.

Sensitivity Analyses

To assess concerns that our use of the missing-data indicator variable for religious attendance might affect the coefficients on the Social Ties/Religious Attendance indicator variables in Models 3 and 4, we also estimated these models excluding the 34 cases of missing data on the religious attendance question, with resulting sample size of 995.

To check that the results in Models 3 and 4 did not depend on dividing those scoring lower vs. higher on the social ties index precisely at the median, we estimated those models that divided the social ties index at multiple points near the median.

The OLS regression models above assume that the dependent variable is quantitative and continuous. Self-rated health could reasonably be considered as an ordinal variable, since we do not know that the points on the 5-point scale are equal. To check whether this impacts our results, we estimated ordinal logit regression models predicting the same dependent variable.

Finally, we also evaluated the relationship of player self-assessments of “religiosity” and “spirituality” with self-rated health, as controls to ensure that religious service attendance was not functioning as a proxy for other aspects of religious experience.

Results

Descriptive statistics on all study variables are reported in Table 1. The mean on the self-rated health assessment was 3.24 (range 1–5), where 3 is “good” health overall. Likewise, the indicator of functioning concerning the ability to get out and about had a mean of 3.57 (range 0–4). The percentage of those reporting frequent religious attendance (nearly every week or more) was 41.35 percent. The mean on the social ties index was 0.08 (range –13.14 to 3.65); without weighting, the mean was 0.00.

Table 2 presents correlations between all study variables. Correlations of particular interest were between frequent religious attendance and ability to get out ($r=0.086, p<0.01$), social ties and ability to get out ($r=0.229, p<0.001$), and frequent religious attendance and social ties index ($r=0.043, p=0.168$). It is important to note that frequent attendance and the social ties index were neither strongly

Table 1 Descriptive statistics on all study variables (unweighted $n=1,029$)

Variable	Mean	Median	Std. Dev	Min	Max
<i>Dependent Variable</i>					
Self-Rated Health	3.24	3	1.20	1	5
<i>Main Independent Variables</i>					
Frequent Religious Attender	41.4%	–	–	0	1
[attend missing indicator]	3.1%	–	–	0	1
Social Ties Index	0.08	0.53	2.68	-13.14	3.65
Able to Get Out	3.57	4	0.79	0	4
<i>Demographic Variables</i>					
College Graduate	77.8%	–	–	0	1
Currently Married	78.6%	–	–	0	1
White	53.7%	–	–	0	1
Age (years)	49.69	48	13.67	27	91
Total Income (logged)	10.78	11.35	2.74	0	15.26
Total Assets (logged)	13.00	13.65	3.41	0	19.12
Total Income (\$1000 s USD)	157.6	85	317.2	0	4260
Total Assets (\$1000 s USD)	2846	848	11,400	0	202,000

correlated with each other, nor did they compete for variance explained in their relationships to self-rated health. In regression models not presented here, leaving out frequent attender or social ties index did not result in any change to the coefficient of the other variable.

Table 3 reports the bivariate and multivariable associations between study variables and the outcome, self-rated health. The first two columns display the regression coefficient for the bivariate association, with the associated r-squared value. All study variables except Age and Total Income were statistically significantly associated with self-rated health at p-values of 0.05 or lower. Model 1 reports the multivariable associations in the full sample with all study variables except ability to get out. Frequent religious attendance and social ties were significantly positively associated with self-rated health. In Model 2, ability to get out was a large and statistically significant correlate of self-rated health and accounted for part of these associations. Yet even accounting for ability to get out, frequent religious attendance remained a statistically significant predictor of self-rated health in Model 2. The social ties index dropped to marginal significance ($p=0.0791$) in Model 2.

Models 3 and 4 use a set of indicator variables to examine potential interaction between frequent religious service attendance and social ties, whether the association of one of these variables with self-rated health depends upon the level of the other. Compared to the omitted reference group who scored lower on the social ties index and were not frequent attenders, the other three combinations of attendance and social ties exhibit significantly better self-rated health. This remains the case in Model 4, when controlling for ability to get out and is also the case in the bivariate model which includes no control variables. Not only do the three groups shown exhibit significantly better self-rated health than the reference group, the averages

Table 2 Correlations (Pearson's r) between all study variables (unweighted n = 1,029)

	Self-Rated Health	College Graduate	Age	White	Total Income (logged)	Total Assets (logged)	Currently Married	Frequent Religious Attender	[attend missing]	Social Ties Index	Able to Get Out
Self-Rated Health	1										
College Graduate	0.1449*	1									
Age	-0.0098	0.0786*	1								
White	0.0794*	0.1686***	0.3354***	1							
Total Income (logged)	0.0522+(0.094)	0.046	0.0810**	0.1040***	1						
Total Assets (logged)	0.1552***	0.1240***	0.0126	0.2296***	0.2376***	1					
Currently Married	0.1051***	0.1476***	0.0598+(0.055)	0.1540***	0.0573+(0.066)	0.1981***	1				
Frequent Religious Attender	0.1005**	0.0344	0.0172	0	0.0694*	0.022	0.1990***	1			
[attend missing]	-0.002										
Social Ties Index	0.1710***	0.0433	-0.0368	0.0055	-0.0561+(0.072)	0.0231	-0.1492***	1			
Able to Get Out	0.5251***	(0.669)	(0.165)	(0.238)	(0.860)	(0.460)	(0.000)				

Two-tailed p value in parentheses, *p < 0.05, **p < 0.010 ***p < 0.001

Table 3 Bivariate and multivariable OLS weighted regression of study variables on self-rated health

Variables	Bivariate Coefficient	Bivariate R-squared	Model 1	Model 2	Model 3	Model 4
College graduate	0.418*** (3.06e-06)	0.021 (0.000188)	0.335*** -0.00281 (0.321)	0.138+ (0.0799)	0.329*** (0.000254)	0.132+ (0.0954)
Age	-0.000856 (0.755)	0.000 (0.191*)	-0.00362 (0.149)	-0.00275 (0.336)	-0.00275 (0.336)	0.00397 (0.114)
White	0.191* (0.0108)	0.006 (0.0229+)	0.0791 (0.326)	0.0287 (0.684)	0.0701 (0.387)	0.0194 (0.783)
Total income (logged)	0.0546*** (5.69e-07)	0.024 (0.0944)	0.0360*** (0.00182)	0.0162 (0.111)	0.0404*** (0.000488)	0.0174+ (0.0854)
Total assets (logged)	0.307*** (0.000737)	0.011 (0.259)	0.105 (0.759)	-0.0252 (0.232)	0.112 (0.232)	-0.0291 (0.722)
Currently married	0.0771*** (2.90e-08)	0.030 (0.000114)	0.0644*** (3.03e-06)	0.0216+ (0.0791)		
Social Ties Index	0.250** (0.000253)	0.010 (0.00725)	0.205** (0.0459)			
Frequent Religious Attender	.790*** (0)	0.276 (0)	0.749*** (0)			
Able to Get Out	0.488*** (3.12e-06)	0.028 (0)	0.134* (0.134*)			
Higher Social Ties, Frequent Attender	0.393*** (0.000317)		0.420*** (6.64e-05)			
Lower social ties, frequent attender			0.351** (0.00121)			
Higher social ties, not frequent attender	0.408*** (2.60e-05)		0.346*** (0.000317)			

Table 3 (continued)

Variables	<i>Bivariate Coefficient</i>	<i>Bivariate R-squared</i>	Model 1	Model 2	Model 3	Model 4
Constant	2.380*** (0)	—	-0.0760 (0.757)	2.163*** (0)	—	-0.219 (0.370)
Unweighted Observations	1029	1029	1029	1029	1029	1029
R-squared	0.073	0.290	0.290	0.065	0.065	0.292

Two-tailed p -value in parentheses, * $p < 0.05$, ** $p < 0.010$ *** $p < 0.001$

Fig. 1 Average self-rated health score for religious attendance/social ties subgroups

		Religious Service Attendance	
		Frequent	Not Frequent
Social Ties Index	Higher Score	3.42 N = 238	3.34 N = 276
	Lower Score	3.33 N = 189	2.94 N = 326

of their self-rated health are statistically indistinguishable from each other. In the bivariate model, the average self-rated health of the lower social-ties/infrequent attender group is approximately 1/3 of a standard deviation below the other three groups. Figure 1 shows the average self-rated health scores for each cell in the 2 × 2 matrix of religious service attendance and social ties subgroups, highlighting the lower average self-rated health of the reference group.

Results of the sensitivity analyses showed that the findings were robust on each count. The models excluding the 34 cases of missing data on the religious attendance question showed substantively equivalent findings to the full-sample models presented, showing that our method of including missing data does not affect the findings in Models 3 and 4. Similarly, the self-rated health of the group that scored lower on the social ties index and were not frequent attenders at religious services remained significantly lower than the other three groups, with similar coefficient differences, when the variables were constructed with multiple cut-points near the median of the social ties index. The ordinal logit regression models had the same findings of significance, direction and effect size as the more straightforward OLS linear regression models. Finally, we present final models without the measure of self-rated religiosity and spirituality. Neither variable showed a statistically significant relationship to self-rated health, nor did their inclusion in the multivariable regression change the significance level or coefficient size on frequent religious attendance. This suggests that frequent religious service attendance is not functioning as a statistical proxy for general religiosity or spirituality.

Discussion

This study is based on data collected from former National Football League athletes who are vested for retirement, covered by disability assistance or are actively participating in the League's retirement pension system. Consistent with prior research, we find that religious service attendance is positively and significantly associated with better health in the full sample – even when controlling for ability to get out and do things. These findings are in line with existing literature that identified positive associations between religion, social ties, and self-rated health (Doane & Elliott, 2015; Heritage et al., 2008; Idler et al., 2009; Warren-Findlow et al., 2013; Zhang, 2017).

The most striking finding was the interplay between frequent religious service attendance and social ties in their respective associations with self-rated health. Frequent attendance was associated with significantly better health only for respondents in the lower half of the social ties index, and higher scores on the social ties index were associated with better health only for people who were not frequent religious service attenders. While previous studies have examined the relationship between social ties and health, to the best of our knowledge, none have documented the role played by religious service attendance in potentially ameliorating the negative health effects of having fewer or weaker social ties. According to Fingerman and Tennant (2016) as we age, "the social world of later life is rife with seemingly meaningless social ties" to more or less anonymous actors embedded in a mosaic of steady routines that lend continuity and predictability, such as consistent religious service attendance. Perhaps the "acquaintances" gained through going to worship, where members may have never or rarely gotten together outside of service, serve a special beneficial function by transcending the criteria of a stranger but not quite reaching the intimate nature of a friendship or intimate tie (Fingerman, 2004). Such affiliation can serve to create familiarity and stability, generate valuable ritual, and thereby provide comfort (Fingerman, 2004, 2009). A weak, or peripheral, tie may serve a similar function as a strong tie when an intimate tie is not available (Fingerman et al., 2011; E. L. Idler, 1987; Idler et al., 2009; Krause, 1998, 2006; Maselko et al., 2012). It is possible that these more casual acquaintances benefit health in the absence of closer relationships.

Frequent religious service attendance may thus play a compensatory role for individuals with less close and/or less frequent social ties by providing social relationships that they may otherwise lack. This implies that, as primary social ties diminish, religious service attendance becomes more important to health, or for people who have otherwise stronger social relationships, religious involvement may have less room to affect their health. This is not to say that weaker ties serve separate functions from strong ties, as Granovetter (1973) shows. Rather, in our analysis, the relationships through religious service attendance may have some of the same health benefits as strong ties, but these are only noticeable for people with fewer close ties.

Another contribution of this study is its attempt to account for the effects of reverse causation, whereby people with significant health challenges tend to have more difficulty leaving their homes and are therefore less likely than healthier people to attend religious services (George et al., 2002; Idler, 1987; Idler et al., 2009; Krause, 1998, 2006; Maselko et al., 2012). In cross-sectional data without the ability to correlate changes over time, it is important to consider this form of "health selection." We addressed this concern by operationalizing the concept of health selection (Idler, 1987; Maselko et al., 2012). Those who attend religious services are likely to be healthy enough to be active outside their homes. We controlled for health selection, to the extent that is possible in cross-sectional data, by including the measure of ability to get out. The addition of this variable to the regression models diminished the coefficient relating frequent religious service attendance and self-rated health by about one-third, although the positive relationship remained statistically significant. From this, we infer most of the relationship between religious attendance and self-rated health cannot be explained in terms of physically limited people

being unable to attend religious services, suggesting that religious attendance itself is more influential in this relationship. It is also possible that religious attendance may positively impact health enough to increase some people's ability to leave their homes. But it is unlikely that would account for much of the relationship between ability to get out and health, since ability to get out statistically explains far more variance in self-rated health than does frequent religious attendance.

Our findings may have implications for assessing and addressing the well-being of former NFL athletes. First, when we examined the matrix of relations between variables, a profile emerged of a subset of NFL retirees that may be in particular need of care. Not only does their health suffer along multiple dimensions, but they tend to be more socially isolated as well. This study suggests that for this sub-group, religious attendance can function as a social resource that aids in producing better self-rated health. Given high levels of religiosity among former NFL players, connecting players with religious communities should be seen as one strategy among others that can be used to address the needs of those with more severe health challenges. It is possible that religious involvement is particularly helpful within the masculine context of football. Similar to military veterans (Nieuwsma et al., 2013), it is possible that former NFL players disproportionately avoid seeking professional medical help because of the associated stigma of weakness, but religious involvement allows support and outlet that may meet similar needs.

Future research should aim to more firmly establish the factors that explain why the benefits of religious attendance only accrue to those with weaker social ties, similar to Idler's (2014) assertion that religion is an especially important social determinant of health for vulnerable populations. One explanation might be that acquaintances formed in religious congregations substitute for closer relationships where they are lacking, making the advantages redundant, and therefore not significant for those who have more and closer relationships. This topic deserves further research attention and replication attempts in other populations.

It would also be helpful for future research to clarify the role played by religious and spiritual beliefs (e.g., views of God) and practices (such as prayer), in linking religion to health for those who are otherwise less socially-connected. Similar to the previously-mentioned findings of Chan et al. (2019) about sense of purpose, religion may benefit health through what Pollner referred to as "divine relations" (1989:92). Pollner found that experiences of relationship with the divine were among the strongest predictors of well-being, even when controlling for religious service attendance and social integration. Research into mental health outcomes found that the direction of the relationship with prayer varied with respondents' forms of attachment to and imagery of God (Bradshaw et al., 2008; Ellison et al., 2014).

Study Limitations

An important limitation is that the measures available in this study to characterize the social relationships of former NFL players were missing important dimensions, especially the number of social ties as well as the quality of relationships in terms

of instrumental and emotional support. Prior research indicates that these are also important dimensions that can affect health (House et al., 1988). It would also be of interest to have information on these dimensions in relation to social connections within the religious organization.

Two other limitations are more obvious. First, the cross-sectional data used in this study is limited in ability to assess causal relationships. Second, as in most U.S.-based data, “religious service attendance” largely measures attendance at Christian services and is not necessarily applicable to other religions. Future research could test our findings using longitudinal data and in samples with other religions.

Conclusion

Using data on former NFL athletes, we found that frequent religious service attendance was associated with better self-rated health and are confident that this relationship was not a selection effect. Unlike prior studies of which we are aware, we found that frequent religious service attendance was associated with self-rated health only for people with less close and/or less frequent contact with friends and family. These findings suggest that health-related outreach to former professional athletes, and potentially other populations, should take into account the role religious involvement can play as a resource for individuals otherwise lacking in social ties.

Acknowledgements Tim Cupery, PhD, is Assistant Professor of Sociology at California State University – Fresno, where he teaches courses on Quantitative Methods, Religion, and Sports. He studies cross-national and individual aspects of religion with links to identity and political conflict, health, and sports. Much of his current research examines the health and well-being of former professional athletes.

Author Contributions All authors contributed to the study. Survey design and data collection were performed by David Weir, Keith Whitfield, and James Jackson. Data analysis was performed by Tim Cupery. Conceptualization, feedback on analysis, writing, and draft preparation were performed by TC, EB, RWT, AS, TR, and KV.

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Data Availability The NFL Player Care Foundation Study of Retired Players is limited-access and is housed in a secure data enclave at the Institute for Social Research at the University of Michigan.

Code Availability These analyses were conducted using Stata SE, version 16. No custom software or code was used in this research.

Declarations

Conflict of interest There are no conflict of interest disclosures to report.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of HELSINKI and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants involved in the study. The study was approved by the University of Michigan IRB.

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The structural and social determinants of Alzheimer's disease related dementias

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Abstract

Introduction: The projected growth of Alzheimer's disease (AD) and AD-related dementia (ADRD) cases by midcentury has expanded the research field and impelled new lines of inquiry into structural and social determinants of health (S/SDOH) as fundamental drivers of disparities in AD/ADRD.

Methods: In this review, we employ Bronfenbrenner's ecological systems theory as a framework to posit how S/SDOH impact AD/ADRD risk and outcomes.

Results: Bronfenbrenner defined the "macrosystem" as the realm of power (structural) systems that drive S/SDOH and that are the root cause of health disparities. These root causes have been discussed little to date in relation to AD/ADRD, and thus, macrosystem influences, such as racism, classism, sexism, and homophobia, are the emphasis in this paper.

Discussion: Under Bronfenbrenner's macrosystem framework, we highlight key quantitative and qualitative studies linking S/SDOH with AD/ADRD, identify scientific gaps in the literature, and propose guidance for future research.

KEY WORDS

ADRD, Alzheimer's disease, classism, dementia, Ecological Systems Theory, genderism, macrosystem, racism, sexism, social determinants of health, structural determinants

Highlights

- Ecological systems theory links structural/social determinants to AD/ADRD.
- Structural/social determinants accrue and interact over the life course to impact AD/ADRD.
- Macrosystem is made up of societal norms, beliefs, values, and practices (e.g., laws).
- Most macro-level determinants have been understudied in the AD/ADRD literature.

1 | BACKGROUND

The incidence and prevalence of Alzheimer's disease (AD) and AD-related dementias (ADRD) are unequally distributed among individuals and communities. Persistent AD/ADRD disparities have been observed among racialized groups, sexual and gender identity groups, and persons with varying socioeconomic resources. In 2020, the prevalence of AD was approximately two times higher among persons racialized as Black/African American than those racialized as White and 1.6 times higher among cisgender women than cisgender men.^{1,2} While evidence of AD/ADRD disparities is firmly established for some groups, research on the upstream factors that influence these disparities remains scarce.

Social determinants of health (SDOH) are the "conditions in the environments where people are born, live, learn, work, play, worship, seek health care, and age."³ Such conditions include economic stability (e.g., employment opportunities), social and community context (e.g., violence and social cohesion), access to and quality of health care, educational/occupational settings (e.g., school/workplace safety), and community/neighborhood-built environments (e.g., transportation, housing, and food access). These factors are often differentially distributed across populations through intentional systems of social, economic, political, and historical power structures that lead to poorer health outcomes among historically marginalized communities and sustained health disparities between hegemonic and nonhegemonic groups. Therefore, some scholars prefer the term "structural determinants of health" to signify the pervasiveness and deliberateness with which policies, societal norms and structures, and governing processes result in inequities in the distribution of power, resources, opportunities, and SDOH.⁴ The accumulated impact of structural and social determinants (henceforth S/SDOH) and associated inequities persists over the life course, negatively affecting physical health, mental health, brain health, and health-related behaviors, which can ultimately increase the risk, disparities, and inequities associated with AD/ADRD.

Theory-driven work will be needed in the future to elucidate the roots of AD/ADRD disparities.

Investigating the relationship between S/SDOH and AD/ADRD requires the use of a theoretical framework precisely attuned to the complexities of this pathway. Although many ecological frameworks for health exist,⁵ most prior research failed to operationalize and test S/SDOH, instead emphasizing intermediary factors like individual-level socioeconomic status. Recently, several researchers have begun to explicitly investigate the associations between S/SDOH and outcomes associated with AD/ADRD.^{5–9} A 2020 study of the Wisconsin Alzheimer's Disease Research Center cohort found that participants who lived in the most disadvantaged neighborhoods (bottom 20% score on the Area Deprivation Index vs. remaining 80%) had 4.1% lower hippocampal volumes.¹⁰ However, the number of rigorously published studies is limited, and among these, S/SDOH concepts and measures are differentially defined.

Frameworks like the Conceptual Framework for Action on SDOH by the World Health Organization and the Public Health Framework for Reducing Health Inequities by the Bay Area Regional Health Inequities Initiative outline structural determinants,⁵ like governance,

RESEARCH IN CONTEXT

1. **Narrative Review:** The authors searched health and social science databases such as PubMed and Web of Science for literature reviews and seminal papers examining associations between each structural/social determinant of health and Alzheimer's disease (AD) and AD-related dementias (ADRD).
2. **Interpretation:** Powerful structural and social factors influence AD/ADRD risk and risk factors across the life course, but how these forces operate and interact, causing disproportionate disease burden among vulnerable and marginalized populations, has not been adequately examined.
3. **Future Directions:** This paper describes macrosystem factors (e.g., racism, sexism, classism, and homophobia) hypothesized to influence AD/ADRD-associated outcomes and highlights preliminary studies that provide evidence for associations between these factors and AD/ADRD.

social and public policies, and societal values, as overarching factors that influence SDOH and, ultimately, impact health behaviors, health, and wellbeing. However, Urie Bronfenbrenner's ecological systems theory¹¹ explains pathways from and interactions between multiple systems on health that best demonstrate the complexity of S/SDOH as an exposure on AD/ADRD. To inspire greater inclusion of S/SDOH in AD/ADRD research, we highlight Bronfenbrenner's theory as a guiding framework.¹¹ In this article, we emphasize what Bronfenbrenner termed the "macrosystem" as the realm of power systems that drive S/SDOH and are the root cause of health disparities.

In this article, we highlight quantitative and qualitative studies linking S/SDOH with AD/ADRD, identify scientific gaps, and propose guidance for future research. Unfortunately, there are insufficient studies on this topic to conduct a systematic review or a meta-analysis. Thus, consistent with a narrative review, we aim to provide a broad overview with select illustrative examples to offer a standardized way to conceptualize S/SDOH globally, in the context of AD/ADRD.

1.1 | A global perspective

While much of the literature included in this article is situated in the United States (US), the impact of S/SDOH on AD/ADRD is global. In 2019, around 57.4 million people were living with AD/ADRD worldwide, and the number is projected to increase to 152.8 by 2050.¹² Early historical events like the Silk Road, Transatlantic Slave Trade, Great Depression, and World Wide Web have shaped the world in which we live into an ever-interacting global enterprise. From European colonization to pandemics, the geographic borders of nations do



FIGURE 1 Relationships among systems within Bronfenbrenner's ecological systems theory.

not prohibit the expansion of social phenomena. For instance, the same European countries that colonized the Americas, filtering their perspectives into the fabric of modern American nations like the US, were the same countries that colonized nations in Asia, Africa, Australia, and the Middle East—imposing their Euro-centric values, ideas, and practices that shaped these countries, which continue to shape and create disparities and inequities in these nations in the 21st century. Therefore, systems of power and discrimination are not exclusive to the US. Efforts to examine S/SDOH in different contexts should strongly consider how determinants may or may not apply in other regions of the world.

2 | METHODS: A GUIDED FRAMEWORK

A conceptual framework that links S/SDOH to AD/ADRD outcomes must allow for the complex interplay between power structures; the social, political, and physical environments; and human behavior, which combine to influence epigenetics, physiology, and disease risk factors and outcomes. Bronfenbrenner's theory¹¹ provides a useful approach to understand the relationships between different S/SDOH in the context of AD/ADRD (Figure 1). It delineates five major systems that were originally postulated to influence human development: micro, meso, exo, macro, and chrono. The Microsystems indicate the settings that individuals directly interact with (e.g., home, school). The mesosystem comprises the interactions between Microsystems, and the exosystem involves the indirect factors that influence health (e.g., climate change). The macrosystem is made up of societal norms, beliefs, values, and practices (e.g., laws) that directly influence Microsystems. Lastly, the chronosystem is composed of exposures to S/SDOH that occur throughout the life course or human development. Although a prior publication on AD/ADRD incorporated Bronfenbrenner's theory, it was used to frame a discussion of a new data collection instrument and did not provide details on key aspects of the ecological system.¹³ We add to this literature by building a comprehensive examination of the

macrosystem because of the weight of its influence on all the other systems including the Microsystems that influence brain health and AD/ADRD.

2.1 | Macrosystem

Bronfenbrenner describes the macrosystem as the outermost level, encompassing "patterns of ideology and institutional structure characteristics of a particular culture" (p. 9).¹¹ Embedded within the ideologies and structures of the macrosystem is the global phenomenon of grouping persons. Whether grouping is justified for science (e.g., taxonomy) or the allocation of resources, assigning values to groups and differentially providing resources to those groups leaves a biopsychosocial mark on individuals and communities in ways that are recognizable in their health.

Macrosystem structures are intimately related to social stratification. Examples of ways we categorize persons include phenotypic characteristics, perceived gender (based on perceived body parts and gender expression), cognitive and physical functioning, sexual orientation/activity, religious affiliation/practice, country of origin, and language and accent. Projects like the Human Genome Sequencing Project indicate that categories like "race" are not reflected in our genes. Similarly, assessments of cognitive and physical functioning suggest that differences in ability are more closely aligned with social categories (e.g., racialized group, socioeconomic status).¹⁴ Such science would indicate that it is the value assigned to social groups and the subsequent allocation of resources to these groups that is the leading cause of health inequities. The science would also contend that the patterns of ideology that inspire the assignment of value to social groups is consistent across the globe and informs the institutional structures we live within.

Subsequently, Bronfenbrenner's definition of the macrosystem bears weight in the application of the role of structural bias—capitalism, racism, sexism and genderism, and other biases—in

AD/ADRD morbidity and mortality. Bronfenbrenner also included public policy as a component of the macrosystem that determines the everyday properties of the micro-, meso-, and exosystems by directing behavior and development (p. 9).¹¹ Hence, as a working definition of the macrosystem for AD/ADRD research, we propose the following definition:

The macrosystem refers to bias-based value systems that consistently underlie a culture as a whole and are expressed and transmitted intergenerationally through exo-, micro-, and meso-systems through public policies that steer institutional and individual biases across time.

It is important to distinguish the exo-, micro-, and meso-systems as the actors of bias, executing the values set by the macrosystem. The dissemination of macrosystem values may be reflected downstream in AD/ADRD disparities. These values may also be observed in the micro- and mesosystems themselves. An example of this is the 2020 Lancet Commission on Dementia Prevention, Intervention, and Care that identified 12 modifiable risk factors (accounting for 40%) across the life course.¹⁵ Many of these risk factors (e.g., social isolation, physical inactivity, and air pollution) are contingent upon the S/SDOH within the micro- and mesosystems. However, when those S/SDOH are ultimately structured by the macrosystem (e.g., residential segregation and education),¹⁶ then it is necessary to examine the upstream contributors of disparities. In what follows, we provide examples of *bias-based value systems* contained within the macrosystem and the degree to which they have been examined in published AD/ADRD research.

2.2 | Interaction between micro- and macrosystems

According to Bronfenbrenner, "a microsystem is a pattern of activities, roles, and interpersonal relations experienced by the developing person in a given setting with particular physical and material characteristics" (p. 22).¹¹ An example of a microsystem is an individual's neighborhood, where people may travel to various destinations, seek health care, and engage in socializing and physical activities. The ability to live in certain neighborhoods (freedom of choice) and the availability of resources and opportunities (e.g., health care) in each neighborhood are influenced by macrosystem factors like structural racism and classism. For instance, the macrosystem dictates healthcare access by establishing clinical care settings in areas outside of disadvantaged neighborhoods, delaying appointments and referrals, and preventing access to affordable treatments for marginalized groups.¹⁷ Similarly, macrosystem factors influence access to greenspaces like parks, gardens, open spaces, and other public or privately owned land that is composed of trees, flowers, grasses, shrubs, and other plants. Greenspaces provide opportunities for social engagement, physical activity, and clean air that improve mental health, cognitive activity, and the microbiome/immune system.¹⁸ Investment priorities into new greenspaces are often driven by macrosystem factors like societal values, power structures, and longstanding policies and practices. As a result, historically marginalized communities continue to experience reduced access to greenspaces and tree canopy and, in turn, have

reduced opportunity to experience those spaces' health-promoting benefits.¹⁹ Table 1 provides a list of Microsystems influenced by the macrosystem that merit further research.

2.3 | Interaction between exo- and macrosystems

Climate change is an example of the indirect influence of the exosystem on health. Scientists worldwide have collectively cited anthropogenic climate change as the single greatest threat to the survival of numerous species, stability of multiple ecosystems, and overall planetary health.²⁰ Climate change is driven by a complex interaction between excess pollution from fossil fuel use, deforestation, livestock farming, and ozone depletion—all arising from human activity. It is also deeply influenced by macrosystem values (e.g., neoliberalism, free-market economics) that have led to global pollution and jeopardized the planet's sustainability. S/SDOH are impacted by adverse weather patterns (e.g., wildfires, hurricanes, floods, heat waves, delays in seasonal cycles) as well as plans and policies, or lack thereof, to combat projected climate change. Severe and unpredicted weather events resulting from climate change are associated with varying access to potable water, food, and homes/shelters to sustain life, particularly for the most vulnerable populations (e.g., children, older adults, immigrants, refugees, those with chronic conditions).²¹ Educational institutions are often disrupted during inclement weather/disasters, jeopardizing learning for marginalized populations already experiencing a lack of access to schooling. In addition, policies promulgated to address air pollution and climate change (e.g., congestion charging and low-emission vehicles) can result in differential access to affordable housing and transportation.²¹ The ensuing climate change has also engendered new public health and humanitarian crises by wiping out crops, reducing economic gross domestic product, igniting inflation, and increasing the risk of and morbidity and mortality from infectious (e.g., vibrio) and noncommunicable diseases (e.g., pulmonary and cardiovascular conditions).²² Residents in low- to middle-income countries and marginalized US communities are often too under-resourced to respond to acute crises, which, in combination with other adverse exposures from the macrosystem, exacerbates the chronic stressors the residents endure. Marginalized and vulnerable populations already experience health inequities, and, with ecosystem exposures like climate change, they face multidimensional deprivation.²³ Thus, climate change influences multiple AD/ADRD risk factors through its impact on S/SDOH.

3 | RESULTS: A GLOBALIZED MACROSYSTEM

3.1 | Capitalism and other social, economic, and class-based biases

Capitalism, the predominant economic system across the world, has led to the concentration of wealth and power in the hands of an elite few. Fundamental cause theory asserts that the link between

TABLE 1 List of relevant microsystems and seminal literature on AD/ADR or associated outcomes.

Section	Seminal literature
Neighborhood/community	
Food access and environment	McMichael AJ, McGuinness B, Lee J, Minh HV, Woodside JV, McEvoy CT. Food insecurity and brain health in adults: A systematic review [published online ahead of print, 2021 May 28]. <i>Crit Rev Food Sci Nutr.</i> 2021;1-16. doi: 10.1080/10408398.2021.1932721 Tani Y, Suzuki N, Fujiwara T, Hanazato M, Kondo K. Neighborhood food environment and dementia incidence: the Japan gerontological evaluation study cohort survey. <i>Am J Prev Med.</i> 2019;56(3):383-392. doi: 10.1016/j.amepre.2018.10.028
Pollution	Weuve J, Bennett EE, Ranker L, et al. Exposure to air pollution in relation to risk of dementia and related outcomes: an updated systematic review of the epidemiological literature. <i>Environ Health Perspect.</i> 2021;129(9):96001. doi: 10.1289/EHP8716 Fuller R, Landrigan PJ, Balakrishnan K, et al. Pollution and health: a progress update [published correction appears in Lancet Planet Health. 2022 Jun 14;]. <i>Lancet Planet Health.</i> 2022;6(6):e535-e547. doi: 10.1016/S2542-5196(22)00090-0
Greenspace	Besser L. Outdoor green space exposure and brain health measures related to Alzheimer's disease: a rapid review. <i>BMJ Open.</i> 2021 May 3;11(5):e043456. doi: 10.1136/bmjopen-2020-043456 PMID: 33941628; PMCID: PMC8098949. Zagnoli F, Filippini T, Jimenez MP, Wise LA, Hatch EE, Vinceti M. Is Greenness Associated with Dementia? A Systematic Review and Dose-Response Meta-analysis. <i>Curr Environ Health Rep.</i> 2022 Jul 20. doi: 10.1007/s40572-022-00365-5. Epub ahead of print. PMID: 35857243.
Recreation	Buettner LL, Langrish S. Rural vs. urban caregivers of older adults with probable Alzheimer's Disease: perceptions regarding daily living and recreation needs. InCaregiving–Leisure and Aging 2020 Mar 24 (pp. 51-65). Routledge. Stephen, R., Hongisto, K., Solomon, A., & Lönnroos, E. (2017). Physical Activity and Alzheimer's Disease: A Systematic Review. <i>The journals of gerontology. Series A, Biological sciences and medical sciences,</i> 72(6), 733-739. https://doi.org/10.1093/gerona/glw251
Transportation	Toepper M, Falkenstein M. Driving fitness in different forms of dementia: an update. <i>J Am Geriatr Soc.</i> 2019;67(10):2186-2192. doi:10.1111/jgs.16077 Babulal GM, Williams MM, Stout SH, Roe CM. Driving outcomes among older adults: a systematic review on racial and ethnic differences over 20 years. <i>Geriatrics (Basel).</i> 2018;3(1):12. doi:10.3390/geriatrics3010012
Housing	Coley RL, Leventhal T, Lynch AD, Kull M. Relations between housing characteristics and the well-being of low-income children and adolescents. <i>Dev Psychol.</i> 2013;49(9):1775-1789. doi:10.1037/a0031033 Okoye SM, Fabius CD, Reider L, Wolff JL. Predictors of falls in older adults with and without dementia [published online ahead of print, 2023 Jan 12]. <i>Alzheimers Dement.</i> 2023;10.1002/alz.12916. doi:10.1002/alz.12916 Wang K. Housing instability and socioeconomic disparities in health: evidence from the U.S. economic recession [published online ahead of print, 2021 Nov 26]. <i>J Racial Ethn Health Disparities.</i> 2021;10.1007/s40615-021-01181-7. doi:10.1007/s40615-021-01181-7
Poverty	Kalaria RN, Maestre GE, Arizaga R, et al. Alzheimer's disease and vascular dementia in developing countries: prevalence, management, and risk factors [published correction appears in <i>Lancet Neurol.</i> 2008 Oct;7(10):867]. <i>Lancet Neurol.</i> 2008;7(9):812-826. doi:10.1016/S1474-4422(08)70169-8 Trani JF, Moodley J, Maw MTT, Babulal GM. Association of multidimensional poverty with dementia in adults aged 50 years or older in South Africa. <i>JAMA Netw Open.</i> 2022;5(3):e224160. Published 2022 Mar 1. doi:10.1001/jamanetworkopen.2022.4160
Policing and incarceration	Policing & traumatic brain injury: Kaske EA, Cramer SW, Pena Pino I, et al. Injuries from less-lethal weapons during the george floyd protests in minneapolis. <i>N Engl J Med.</i> 2021;384(8):774-775. doi:10.1056/NEJM2032052 Incarceration & cognitive impairment: Cox RJA, Wallace RB. The role of incarceration as a risk factor for cognitive impairment [published online ahead of print, 2022 Sep 25]. <i>J Gerontol B Psychol Sci Soc Sci.</i> 2022;gbac138. doi:10.1093/geronb/gbac138

(Continues)

TABLE 1 (Continued)

Section	Seminal literature
Neighborhood deprivation	Kind AJH, Buckingham WR. Making neighborhood-disadvantage metrics accessible - the neighborhood atlas. <i>N Engl J Med.</i> 2018;378(26):2456-2458. doi:10.1056/NEJMmp1802313
	Powell WR, Buckingham WR, Larson JL, et al. Association of neighborhood-level disadvantage with Alzheimer Disease neuropathology. <i>JAMA Netw Open.</i> 2020;3(6):e207559. Published 2020 Jun 1. doi:10.1001/jamanetworkopen.2020.7559
Workplace/occupation	
Workplace/occupation	Huang LY, Hu HY, Wang ZT, et al. Association of occupational factors and dementia or cognitive impairment: a systematic review and meta-analysis. <i>J Alzheimers Dis.</i> 2020;78(1):217-227. doi:10.3233/JAD-200605
	Parker SK, Ward MK, Fisher GG. Can high-quality jobs help workers learn new tricks? A multidisciplinary review of work design for cognition. <i>Academy of Management Annals.</i> 2021;15(2):406-454. doi:10.5465/annals.2019.0057
Income	Marden JR, Tchetgen EJ, Kawachi I, Glymour MM. Contribution of socioeconomic status at 3 life-course periods to late-life memory function and decline: early and late predictors of dementia risk. <i>Am J Epidemiol.</i> 2017;186(7):805-814. doi:10.1093/aje/kwx155
	Yaffe K, Falvey C, Harris TB, et al. Effect of socioeconomic disparities on incidence of dementia among biracial older adults: prospective study. <i>BMJ.</i> 2013;347:f7051. Published 2013 Dec 19. doi:10.1136/bmj.f7051
School	
Literacy	Arce Rentería M, Vonk JMJ, Felix G, et al. Illiteracy, dementia risk, and cognitive trajectories among older adults with low education. <i>Neurology.</i> 2019;93(24):e2247-e2256. doi:10.1212/WNL.0000000000008587
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Education	Eng CW, Glymour MM, Gilsanz P, et al. Do the benefits of educational attainment for late-life cognition differ by racial/ethnic group?: evidence for heterogeneous treatment effects in the kaiser healthy aging and diverse life experience (KHANDLE) study. <i>Alzheimer Dis Assoc Disord.</i> 2021;35(2):106-113. doi:10.1097/WAD.0000000000000418
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socioeconomic position and health outcomes like AD/ADRd exist when the health benefits of money, knowledge, prestige, power, and social connections are made inaccessible to marginalized groups.²⁴ Despite the world's immense resources, capitalism incentivizes small groups of individuals, families, and communities to hoard wealth and resources, producing inequities in S/SDOH as well as health outcomes.²⁵ While no AD/ADRd research directly investigates the role of capitalism and clas-

sism in disease etiology, some aspects of capitalism and classism have been studied. These studies provide some understanding of the role of capitalism and classism as manifested through an individual's socioeconomic experience—education, employment, income, and housing. Inequitable distribution of socioeconomic assets, including early-life educational attainment and later-life wealth, explains racialized group disparities in dementia more substantively than do cardiovascular

risk or health behaviors.²⁶ Some features of occupation (e.g., manual vs. mental work, shift work) are known to be associated with risk for AD/ADRD.^{27–29} Research on individuals experiencing poverty and homelessness also suggests that they have greatly increased risk for AD/ADRD over the life course.³⁰ Hence, employing a S/SDOH framework to examine capitalism and classism at a macrosystem level would reveal that racial capitalism via occupational segregation increases AD/ADRD disparities over time (e.g., corporate oligarchies of today vs. robber barons of the 19th century).³¹ The natural experiment of capitalism and classism on AD/ADRD could be captured in modern research by comparing the impact of privatized health care with national universal health care programs.³² Though with limited access to monetary resources that can increase opportunities for better quality of life (e.g., nutrient-rich foods, adequate housing, health care, health insurance, transportation, low-pollution neighborhoods with greenspace and recreation, internet), marginalized groups will continue to experience the greatest burden of AD/ADRD without economic intervention. This is an underexplored area of AD/ADRD that merits attention.

3.2 | Structural racism and other social, cultural, and ethnic-based biases

Structural racism refers to a social system that assigns individuals to racialized categories (i.e., race) based on phenotypic characteristics like skin tone, hair texture, and the structure of eyes, nose, cheekbones, and lips.³³ After assignment (i.e., the process of racialization), opportunities and access to resources are structured based on the value assigned to each racialized category. Due to the legacy of European colonization, for many global contexts, persons racialized as White receive more opportunities and resources.³⁴ In the US, structural racism expressed via residential segregation indicates that living in areas where a majority of residents are racialized as non-Hispanic Black is associated with increased dementia risk.³⁵ This is due to residential segregation's ability to reduce or eliminate access to good-quality S/SDOH, like education, employment/jobs, nutrient-rich foods, health care, transportation, spaces for safe physical activity within neighborhoods, and housing, among other factors. Cognitive outcomes are also linked with structural racism as the odds of reporting poor subjective cognitive function doubles when women racialized as Black are exposed to racism within five or six microsystems—workplace, housing, police, courts, schools, health care—compared to one or two.³⁶ In these examples, structural racism directs S/SDOH to differentially treat and provide access to resources to persons racialized as Black. There are examples where persons racialized as Indigenous, Asian/Asian American, Latinx/a/o, or Middle Eastern experience similar discrimination, though this literature is underexamined in AD/ADRD research.

Other related biases operate similarly to racism. While “race” is a social construct not based in biology, ethnicity—also a misconstrued concept—refers to a shared cultural background between individuals. In the US, persons racialized as Latinx/a/o are considered an ethnic group because such persons trace their closest ancestors to a coun-

try in parts of North America, Central America, or South America. Ethnicity in this context implies region of birth or immediate ancestry within a fixed timeframe (i.e., since European colonization) as the defining characteristic of belonging to a group. Similarly, in India, there are distinct levels of social stratification ascribed to persons at birth that were mapped to one's place or status within the society or community. The social hierarchy, referred to as a Scheduled Caste system (including Scheduled Tribes) in India, shares functions similar to the racial hierarchies found in other parts of the world. The historical practice of discriminating against minoritized groups, or “backward” castes as they are called in social surveys (e.g., Dalits), results in the same disproportionate access to resources like drinking water, education, employment opportunities, housing, income, marriage, kinship, and political participation.^{37,38} Many other countries have similar social hierarchies with varying terms to describe ethnic groups, social classes, and, in many contexts, races. These distinctions are meaningful to note when conducting AD/ADRD research where values are assigned to groups and opportunities are unevenly distributed. Where social inequities exist, so too will AD/ADRD disparities.

3.3 | Xenophobia and related biases (e.g., against migration status, language, and accent)

Xenophobia and related biases describe the ways social systems assign group membership and associated attitudes and stereotypes to people based on their country of origin. Identifiers of national difference are often inferred from migration status, fluency in the majority group's primary language, and/or the presence of an accent. In addition to general suspicion and other negative responses, these basic identifiers may undeservedly illicit³⁹ negative encounters and experiences of acculturation, leading to restricted access to resources that increases AD/ADRD risk.⁴⁰ An example of this is disproportionate access to health care, which can result in delays in timely diagnosis and/or limited access to specialists and caregivers.⁴¹ For example, migrants experience factors throughout all stages of migration that may impact their health in both acute and long-term ways. Understanding acculturation in context⁴² includes acknowledging, among other things, changes in social networks, potential exposure to trauma (for refugees in particular), or discrimination and stigmatization that may occur through interactions with immigration enforcement and other members of the majority group's society more broadly.⁴³ These experiences shape health and wellbeing, including cognitive health, regardless of health insurance status or access to care.^{44,45}

When accessing care, there is a dearth of clinicians and/or interpreters to provide medical assessments in the non-majority group language. Specific to cognitive aging and AD/ADRD risk, cognitive tests developed in one language are often translated into and used in another language without proper validation or cultural consideration. Such lack of consideration may lead to confusion, frustration, and/or emotional distress, all of which negatively impacts not only cognitive performance but also trust in medical providers. Large-scale epidemiological studies are beginning to investigate the construct validity and

reliability of commonly used assessments.^{46,47} In addition to cognitive protocols for the investigation of risk for and development of dementia, more work is needed in clinical settings where individual performance is critical to accurate identification of AD/ADRD and dementia staging. Furthermore, although accents play a powerful role in the categorization groups, less work has been done to understand how their presence may impact outcomes associated with AD/ADRD, either directly or indirectly.^{48,49}

3.4 | Religious bias

Religious bias or discrimination describes the prejudicial treatment of individuals based on religion or belief. It includes stereotyping individuals based on assumptions made about belonging to a religious group.⁵⁰ In many parts of the world, religious freedom is threatened by national, institutional, and religious policies that play a significant role in interstate conflict, social upheavals, civil wars, and the persecution of minoritized religious groups.^{51,52} Religious bias has existed for centuries across the globe, including the persecution of Jewish and Christian people by the Romans, the Holocaust, Uighurs in China, and attacks on the Rohingya people in modern-day Myanmar.⁵³ These and other examples illustrate the global and historical magnitude of external, often colonial, forces imposing religious homogeneity and persecution on minoritized religious groups.⁵⁴ Religious bias can be accompanied by capitalism, xenophobia, and other structural biases that have severe consequences for minoritized religious groups' ability to access land, programs, education, health care, healthy foods, and other life-sustaining resources.⁵⁵ In the US, anti-Semitism and Islamophobia are on the rise^{56,57} and contribute to psychosocial stress associated with poor health outcomes that may also lead to increased AD/ADRD risk.

Although religious bias has not been formally studied in AD/ADRD research, it is a form of structural bias that hinders access to S/SDOH, imposes violence on minoritized religious groups, and restricts power to such groups majoritized groups.⁵⁸ Further, religious bias embedded in the implicit biases of healthcare providers, government officials, and other authority figures heightens religious discrimination, which is commonly associated with poor mental health, maladaptive coping behaviors, and health care-seeking behaviors.⁵⁹ Therefore, religious bias may play a role in the development of AD/ADRD and the health care of persons with AD/ADRD.

3.5 | Structural genderism and related biases (e.g., transphobia and sexism)

Structural genderism describes the ways social systems support and reinforce a binary view of human bodies. Genderism assumes that the human body is sexually dimorphic ("female" and "male") and, further, that social roles can be ascribed to these bodies (i.e., a person with XX chromosomes is assumed to have breasts and take on the social role of a cisgender female). This biologized-gender binary starts at birth

with sex assignment and continues throughout the life course, with society encouraging its constituents to conform to one gender role, appearance/expression, identity, and set of behaviors based on how body parts are externally perceived.^{60–63} Structural genderism encompasses transphobia (discrimination against transgender persons) and sexism (discrimination against cisgender females) by acknowledging there is an overarching bias against persons who do not conform to a society's biologized-gender binary (i.e., discrimination against non-binary persons and gender-diverse groups).

At the macrosystem level, genderism and its many forms (e.g., transphobia, sexism) are embedded in microsystems, resulting in widespread discrimination across S/SDOH. Institutions like prisons and long-term care centers alike have gender-segregated structures that impede and complicate healthcare access for gender-diverse groups, including delaying diagnosis and quality care for persons with dementia. Transgender adults who experience healthcare discrimination may be more likely to endorse experiencing cognitive decline.⁶⁴ Though, like with other S/SDOH, there is a dearth in the literature of studies exploring the association between structural genderism and AD/ADRD.

Though meaningful differences exist, studies examining subjective cognitive decline, which may be one of the first clinical manifestations of AD/ADRD,⁶⁵ indicate sexual and gender minority (SGM) adults may have a higher prevalence of subjective cognitive decline than non-SGM adults and may be more likely to experience related functional limitations.^{66,67} However, AD/ADRD risk factors and other health inequities are not uniformly experienced across SGM communities.⁶⁸ An analysis of 2015 Medicare claims data revealed that transgender adults aged 65+ had a higher prevalence of dementia compared to cisgender adults (18% vs. 12%).⁶⁹ Transgender adults from racialized and minoritized groups may also be more likely to experience greater subjective cognitive decline than cisgender adults from racialized and minoritized groups and transgender adults racialized as White.⁶⁶

Unfortunately, AD/ADRD research contributes to the bias-based practices that reinforce structural genderism. These practices include the systematic and purposeful omission of demographic measures used to identify gender-diverse persons and participants within a healthcare system or research study.⁷⁰ When included, gender-diverse persons are often lumped together with sexual minorities (SGM) or some variation of lesbian, gay, bisexual, gay, transgender, intersex, queer, questioning, asexual (LGBTIQQA). This makes it impossible to distinguish the effects of structural genderism on gender diverse persons from homophobia and related biases (see following discussion).

Scientific participation in structural genderism also restricts the discourse on the role of sexism on AD/ADRD to biological differences between cisgender females and males.^{71–74} Though differences exist on how older adults protect their cognitive health, reduce their risk for cognitive decline, and manage cognitive changes,⁷⁵ sexism as a determinant of chronic stress and access to health-sustaining resources is not discussed. Much of the literature on structural genderism is severely limited by poorly defined approaches to measuring sex and

gender and the underrepresentation and historical exclusion of gender diverse groups.⁷⁶⁻⁷⁹

3.6 | Homophobia and related biases (e.g., biphobia)

Homophobia represents emotional responses, negative attitudes (i.e., myths, stereotypes, assumptions, biases), and discriminatory behaviors toward same-sex relationships, attractions, behaviors, and/or identities. Related terms for homophobia include "homosexism," "heterosexism," "sexual prejudice," and "biphobia." Homophobic experiences may lead to stress in sexual minorities (lesbian, gay, bisexual, queer, as well as additional identities), which increases risk for physical and mental health disparities. Past research on outcomes associated with AD/ADRD found that sexual minorities, or lesbian, gay, bisexual, queer, and additional identities, experienced greater subjective cognitive decline and multimorbidity, including depression and functional limitations, than heterosexual adults.^{65,80} These findings reveal a pattern of poor health consequences for sexual minorities that, like other minoritized groups, may accumulate over the life course to influence AD/ADRD risk and have dire consequences for older adults seeking to age in a safe environment.

3.7 | Ableism

Ableism is reflected in stigma toward individuals with disabilities, including individuals with AD/ADRD and their caregivers. AD/ADRD stigma can lead individuals, including one's social network and healthcare professionals, to patronize, stereotype, isolate, or discriminate against individuals with AD/ADRD.⁸¹ AD/ADRD stigma may represent a barrier to healthcare service utilization and has been observed even at preclinical stages of the disease,^{81,82} with worse prognosis associated with higher levels of AD/ADRD stigma.⁸³ Beyond stigma and discrimination is the sheer lack of consideration and accommodation for persons with physical, cognitive, or developmental disabilities. A useful example is the principle of universal design, in which our built environments (e.g., building entrances, bathrooms, floors, sidewalks, and public spaces) are made accessible to the maximum extent possible through intentional designs that accommodate all individuals, regardless of ability. While universal design is used periodically in the US and has been used to help design care homes for persons with AD/ADRD globally,⁸⁴ numerous examples demonstrate how our built environments are not planned and designed for the safety, wellbeing, and health of older adults, who often experience decreased abilities in vision, hearing, and mobility.^{85,86} Universally designed built environments would increase accessibility to health-promoting opportunities (e.g., places for physical activity, jobs, education) for all people and, ultimately, may help protect against AD/ADRD by allowing individuals to live as independently and safely as possible. However, this interventional and policy research has not been explored in the context of AD/ADRD.

3.8 | Ageism

At the macro level, ageism reflects cultural values enacted through social institutions that diminish the value of older adults as integral members of a population.⁸⁷ In modern society, with technological and medical advances as well as rapid globalization, old age is no longer valorized and associated with exceptional survival and wisdom but is instead perceived as a source of frailty, economic expenditure, morbidity, and disability.⁸⁷⁻⁸⁹ Age is the strongest risk factor for AD/ADRD, and, consequently, those with dementia often experience a double stigma from ageism and ableism.⁸⁸ Maltreatment and dismissal of persons living with AD/ADRD adversely impact their social engagement, self-esteem, self-efficacy, and ability to access quality health care.⁹⁰ Ageism is often pronounced for groups from marginalized experiences, like those racialized and minoritized, perceived as cisgender women, with fewer socioeconomic resources, and/or houseless.⁹¹ Despite the inextricable link between age as the biggest risk factor for AD/ADRD, few studies explore the role of ageism in the quality of life and functional ability of persons living with dementia. Therefore, intervention and clinical trial research that reduces the mental, physical, and social needs of older adults living with dementia—as well as participatory research and community engagement—can aid medical professionals and caregivers in eliminating ageism.

3.9 | Intersectionality

Racism, classism, ableism, sexism and genderism, and other bias-based values are social mechanisms that have been consciously and subconsciously developed to validate and justify inequities within societies.⁹² Due to the similarity in deployment of these bias-based systems, intersectional oppression often occurs. With origins in Black feminist theory, intersectionality is a theoretical and analytical framework that captures cross-cutting experiences and considers how interlocking systems of oppression, associated with social hierarchy, compound and create disadvantage.⁹³ Several studies applied an intersectional lens to examine how interrelated systems of oppression confer risk for AD/ADRD and may lead to disparities that disproportionately disadvantage marginalized communities. For example, gendered racism^{94,95}—the simultaneous experience of racism and sexism—was associated with a higher number of cognitive complaints in US women racialized as Black.⁹⁶ Another US-based study revealed meaningful differences at the intersection of racism and sexism in biological aging, an emerging risk factor for AD/ADRD.⁹⁷ A large-scale study in the UK saw significant intersectional effects of gender, socioeconomic status, and racialized group on biological markers of aging. This study reported that groups racialized as Black and other racialized and minoritized cisgender men with fewer educational opportunities had higher systolic blood pressure compared to other groups.⁹⁸ Similarly, a population-based study in Malaysia found the prevalence of cognitive impairment was highest among older adults racialized as Malay who were more economically disadvantaged compared to older adults racialized as Malay who were not as economically disadvantaged.⁹⁹ These studies

highlight the importance of an intersectional approach in advancing our current understanding of AD/ADRD disparities globally. While the body of literature utilizing an intersectional framework remains scarce, this framework provides a critical opportunity to simultaneously analyze multifaceted power structures and processes that produce and sustain inequities in AD/ADRD and related outcomes.¹⁰⁰

4 | DISCUSSION

4.1 | Interaction between chrono- and macrosystems across life course

A life course approach to studying AD/ADRD is valuable for understanding how complex and varying genetic, environmental, behavioral, and social factors interact as we develop and age, leading to disparate health outcomes. Recognition of the importance of sensitive periods and latency in the etiology of chronic disease is not new. The fetal origins of the adult disease hypothesis, which has most commonly been associated with cardiovascular and metabolic diseases, is one of many theories that highlight the critical importance of understanding and measuring experiences well before disease onset.¹⁰¹ Given the interrelationship of cardiovascular and brain health, many of these same pathways are relevant for understanding the development of dementia.

A life course approach uses a multidisciplinary framework to elucidate how sensitive periods and latency affect dementia risk and exacerbate disparities and can help us understand how structural biases set the trajectory of risk and protective factors across the life course of an individual or community.¹⁰² Recent literature suggests early-life exposure to structural racism via residential segregation puts persons racialized as Black at disproportionate risk for AD/ADRD and is associated with lower cognitive function later in life.^{16,35,103} There is also strong evidence suggesting that cumulative socioeconomic experiences due to an intersection of capitalism, racism, and likely other macrosystems are prime indicators of AD/ADRD risk for marginalized populations.^{104,105} Such literature provides a blueprint for how to think about macrosystem exposures across the chronosystem.

4.2 | Scientific gaps, challenges, and future directions

Some scientists do not yet fully appreciate how contextual factors, like structural biases, influence disease mechanisms. However, without exploring the role of societal policies and discriminatory practices on the development and experience of AD/ADRD, these scientists introduce tremendous bias into their research designs and analyses. Consideration of S/SDOH is necessary to advance our understanding of AD/ADRD and develop effective prevention and treatment strategies. Funders, training programs, researchers, journal editors, and institutions must encourage inclusion of S/SDOH into every study of AD/ADRD.

Simultaneously, AD/ADRD research must work to refine definitions, methods, and instruments that capture the role of structural biases on health. Research is needed to capture how multidimensional structural racism, sexism, and genderism influence AD/ADRD across the life course.^{12,24} There is a need for a re-examination of how sex and gender are invoked in AD/ADRD so that gender is not limited to finite biologized categories. Methods, including measures, are needed to articulate how the intersection of structural biases operates and potentially accumulates to influence AD/ADRD etiology and patient outcomes. Culturally responsive instruments are needed to measure cognition and other important AD/ADRD outcomes across diverse cultures, linguistic backgrounds, and ways of life.

Key research questions cannot currently be addressed due to a lack of inclusivity of diverse populations in health research. This is certainly the case when examining the role of minority stress and discrimination due to xenophobia, religious bias, transphobia, and/or homophobia on AD/ADRD, cognitive impairment, and healthcare access.^{106–108} Perhaps these gaps can be filled by the robust scholarship of researchers on the margins of AD/ADRD research whose lived experiences aid in identifying salient exposures among populations more diverse than the usual AD/ADRD non-Hispanic White, educated, cisgender, heterosexual, and able-bodied samples. Therefore, attention must be paid to the research of scholars from countries that are not primarily White and high-income but have innovative and meaningful AD/ADRD research to share. Thus, there are transformative opportunities for AD/ADRD research that integrates diverse data from across settings, cohort studies with traditionally excluded or underrepresented populations, and community-based participatory research that closes the gap between research and community.⁷⁸

4.3 | Conclusion

Health and its associated outcomes are never individually centered but are products of exposure to certain risk factors in varying magnitude, from cells to cities, from neurons to nations. The projected growth of AD/ADRD cases by midcentury has expanded the research field and impelled new lines of inquiry into S/SDOH as fundamental drivers of disparities in AD/ADRD. The extant literature has only tangentially examined the residual impact of these accumulated structural biases on the risk of AD/ADRD. This narrative review provides a broad illustration for the potential relationships between S/SDOH and AD/ADRD. The macrosystem in Bronfenbrenner's ecological systems theory identifies powerful societal forces that determine the foundation for health outcomes through bias-based value systems that propagate discrimination and, subsequently, health disparities across the life course. The intersection of these bias-based values has an enormous impact on individual, neighborhood, and population health, which is typified in the study of AD/ADRD and the differential risk for marginalized groups. Measurement of the macrosystem will provide valuable insight into how AD/ADRD risk and outcomes may be exacerbated by structural biases and discrimination. Researchers, clinicians, and policymakers should be aware

of the macrosystem's role and how it influences AD/ADRD health disparities.

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CONFLICT OF INTEREST STATEMENT

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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PROTOCOL

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Definitions, terminology, and related concepts of “racial health equity”: a scoping review protocol

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Abstract

Background In the USA, access to quality healthcare varies greatly across racial and ethnic groups, resulting in significant health disparities. A new term, “racial health equity” (RHE), is increasingly reported in the medical literature, but there is currently no consensus definition of the term. Additionally, related terms such as “health disparities,” “health inequities,” and “equality” have been inconsistently used when defining RHE.

Methods The primary purpose of this scoping review is to investigate the current use and underlying concepts used to define racial health equity. The study will address two key questions: (1) “What terminology and definitions have been used to characterize RHE?” and (2) “What knowledge gaps and challenges are present in the current state of RHE research and theory?” The review will collect and analyze data from three sources: (1) websites from key national and international health organizations, (2) theoretical and narrative published articles, and (3) evidence synthesis studies addressing interventions targeting racial health equity and minority stakeholder engagement.

Discussion Defining “racial health equity” and related terminology is the first step to advancing racial health equity within the USA. This review aims to offer an improved understanding of RHE constructs and definitions, bringing greater unity to national racial health equity research efforts across disciplines.

Systematic review registration This protocol is registered with the Open Science Framework at <https://osf.io/7pvzq>.

Keywords Racial health equity, Health equity, Health justice, Scoping review, Landscape review, Racism, Discrimination, Health disparities, Definitions, Terminology

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Background

The concept of racial health equity (RHE) within the USA emerged in response to persistent disparities in health outcomes along racial lines. By 2003, the Institute of Medicine's "unequal treatment" [1] drew attention to the fact that individuals of color consistently experienced worse health outcomes and received lower-quality care than their white counterparts. The SARS-COVID-19 pandemic further exacerbated inequities along racial lines [2, 3], and global racial justice protests further drew attention to RHE as a research field and call to action. Specifically, RHE is a component of health equity, a variable concept that focuses on eliminating unfair disparities in health based on racial, environmental, socioeconomic, or structural factors beyond an individual's control.

In 2021, the White House released Executive Order #13,985 to advance health equity and to provide government support for people of color and others who have been historically underserved, marginalized, or affected by persistent poverty and inequality [4]. This order was extended in 2023 to establish equity-focused leadership plans within government agencies, creating economic rural opportunities and equity-focused urban developments, advancing civil rights, and promoting equity in data [5]. In the wake of these executive orders, government and private organizations, including the Robert Wood Johnson Foundation [6], and the United States Department of Health and Human Services [7, 8], have launched initiatives addressing racism and health inequities. Research studies specifically addressing racial health equity have also increased exponentially, while there were

0–2 articles per year containing RHE terms between the years 2008–2018, by 2022 there were 48 articles containing these terms (Fig. 1).

The effects of racism and discrimination are believed to be one of several social determinants of health (SDOH), which are non-medical factors that influence health outcomes. SDOH encompasses the environmental conditions in which people are born, work, live, and age [9]; 80–90% of a person's health and well-being is determined by these factors [10]. There are several conceptual models to explain key SDOH that are believed to underlie health inequalities and explain how racism, a social construct not influenced by biology, impacts health equity. These social determinants include structural determinants (e.g., governmental policies, housing availability, socioeconomic status), intermediary determinants (e.g., material conditions, food availability), and systematic barriers that underlie root or upstream causes of health inequities [11]. Systemic or implicit racism negatively and unfairly affects the health of racialized groups in the USA and perpetuates racial health disparities [12]. The effects of racism are thus included as a SDOH, with effects ranging from unfair barriers to employment and education, as well as access to healthcare [13, 14].

Despite the growing attention on health equity and the emergence of research to study health disparities, there is not yet a shared definition of terms, concepts, or conceptual frameworks. This may be due in part to the multidisciplinary nature of health equity research, with studies originating from investigators from both clinical and non-clinical backgrounds (e.g., anthropology, public

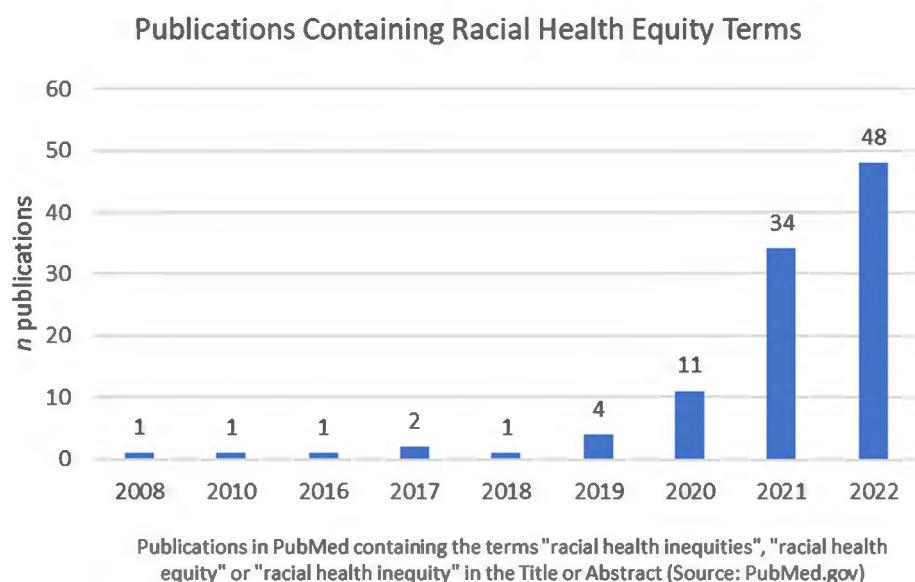


Fig. 1 Publications containing racial health equity (RHE) terms have greatly increased in recent years. Results include all publications within PubMed containing indicated terms within the title or abstract. Source: PubMed.gov. Data retrieved January 27, 2023

health, sociology, economics, epidemiology, and history). Additionally, three or more concepts underlie “racial health equity” (“racial,” “health,” and “equity”), which may be based on different constructs, values, or principles. For example, “health” can be further characterized into at least three domains, including physical, social, and mental well-being [15]. “Health equity” is also defined differently across health organizations and across research fields.

Interventions to mitigate health inequities vary across sectors, and the metrics of outcomes differ across disciplines. While many studies have been undertaken to reduce gaps in centering RHE, they may lack input and representation from other key disciplines outside of medicine, such as education and public health as well as from diverse stakeholders across hierarchy and settings whose input can inform the U.S. healthcare system for advancing racial health equity. A national consensus is lacking on what constitutes racial health equity. Clear and consistent descriptions of definitions, terms, constructs, and frameworks are needed to incorporate and integrate RHE concepts across organizations and disciplines to begin to establish a national consensus to center, guide, and advance RHE.

This protocol represents the first known effort to systematically catalog, evaluate, and map RHE terminology in its current and historical forms. We will use a multi-part search strategy to analyze the use of RHE terms within major health websites, theoretical articles, and evidence syntheses. Alignment and clarity of the current knowledge of RHE definitions and terms—with the understanding that these terms will continue to evolve over time—is a crucial first step to driving the development and implementation of relevant interventions for high-risk groups to achieve positive health outcomes.

Aims and objectives

In this study, we propose to conduct a landscape analysis, which is an evidence synthesis method that identifies trends, gaps, and opportunities within a

specified research field. Our analysis will collect definitions of “racial health equity” identified in key public health organization websites, original theoretical articles, narrative reviews, and recent evidence synthesis studies from medical and social science databases. Full inclusion and exclusion criteria are provided in Tables 1 and 2, and Supplemental Table 3.

Our RHE terminology study is part of a larger project, “Centering racial health equity in systematic reviews of interventions” conducted by the Cochrane US Network and funded by the Robert Wood Johnson Foundation which includes four scoping or landscape reviews including the present study. The remaining reviews focus on (1) stakeholder engagement [16], (2) logic models of RHE [17], and (3) healthcare interventions to promote equity in racialized populations [18]. To our knowledge, our terminology and definition study is the first to evaluate RHE constructs through systematic reviews and a landscape analysis. Our overarching goal is to determine how RHE constructs are used, defined, determined, and applied in the current state of knowledge by addressing the following key questions (KQs):

- KQ1: What terminology and definitions have been used to characterize racial health equity in the following:
 - (a) Public guidance documents, reports, and information content in websites produced by key public health organizations (e.g., government, private, non-profit) involved in guiding public health, medicine, and evidence-based practices in the USA
 - (b) Theoretical or conceptual original publications
 - (c) Evidence syntheses on interventions focused on addressing racial health equity?
- KQ2: What knowledge gaps and challenges are present in the current state of RHE research, practice, and theory?

Table 1 Inclusion and exclusion criteria for websites review (Aim 1)

	Inclusion criteria	Exclusion criteria
Website type	Well-established public health organizations (ex: CDC, WHO, NIH), organizations that guide and inform health care (ex: CMS), organizations with a focus on improving health and provide best practices (ex: Cochrane), non-profit health organizations (government, national, associations, professional societies)	For-profit health organizations (ex: pharmaceuticals), commercial websites, state health organizations, universities, hospitals
Setting	United states and organizations that guide US health care	Websites that are public but not in the USA or do not guide US health care
Date	No date restriction	N/A
Language	English	Other languages

Table 2 Inclusion/exclusion criteria for theoretical articles (SA 2)

	Inclusion criteria	Exclusion criteria
Article type	Primary research article Theoretical article Narrative reviews Editorials	Conference abstracts Video or other media Book chapters Dissertations Evidence syntheses
Content	Contains the definition of "racial health equity" or separate definitions of "health equity" within the context of race/ethnicity/minority communities Includes words of concept/definition/framework/terminology	No definition of "racial health equity," "health equity," or related terms Culture or acculturation focus The article focused on measuring outcomes without defining terms
Setting	No setting explicitly mentioned, or relevant to the USA (includes countries high on the human development index per WHO guidelines)	The article explicitly set within non-highly developed settings
Date	No date restriction	N/A
Language	English	Other languages
Accessibility	Full text available through the University of Colorado or Marymount University libraries	Full text unavailable through UC or MU libraries

To answer these questions, we propose the following Specific Aims (SA):

- SA 1: To identify and summarize RHE terminology used by key health organizations involved in guiding public health, medicine, and evidence-based practices
- SA 2: To identify and summarize RHE terminology and definitions from primary original, theoretical/conceptual articles
- SA 3: To identify and summarize RHE terminology and definitions from evidence synthesis studies
- SA 4: Summarize findings from Aims 1–3 to identify gaps and challenges in the current literature and to make recommendations for future research.

engine (Google) using the following search terms: "public" + "health" + "organizations" + "United States". Inclusion and exclusion criteria for websites is provided in Table 1. Websites to be analyzed will be limited to not-for-profit (e.g., Robert Wood Johnson Foundation), government (e.g., National Institutes of Health, Centers for Disease Control), academies (e.g., American Academy of Pediatrics), or evidence synthesis (e.g., Cochrane) organizations. Global organizations with relevance to the USA (e.g., World Health Organization) will be included. Excluded websites will include corporate or for-profit organizations, state-level government websites, and hospital or university websites. Additional websites fitting the inclusion criteria will be added based on input from the study team. Categories and corresponding numbers of websites to be included in our analysis are provided in Supplemental Table 1.

Definitions for terms including "race"/"ethnicity," "racism"/"discrimination," and "racial health equity"/"health equity" will be collected from each website, when present. Exact URLs, date of access, and any cited sources will be collected. The homepage, links to the different topics on the home page, and search functions will all be utilized to find definitions. Reports or links on the websites will also be searched for relevant definitions. Definitions will be reported as missing if they cannot be located after > 1 h of search time. All definitions will be collected in an Excel spreadsheet.

To assess the ease of locating RHE definitions on each website, we developed an ease-of-access website rating tool. Briefly, definition accessibility will be rated as "very easy," "easy," "medium," "hard," or "very hard" based on the location of the definition (homepage, external report, etc.), if present, and the time required to find the definition was not very long.

Justification and rationale

The study of racial health equity (RHE) is a burgeoning area with diverse, disparate definitions of key terms. Establishing consensus definition(s) of racial health equity will benefit community members, researchers, and policy makers by (1) allowing for precise measurements of intervention success against the shared definition, (2) allowing for the goals of equity research and policy to align to the shared definition, and (3) increasing the clarity of racial health equity research and policy language. Each of these benefits has the potential to enhance the impact of health equity interventions on racially minoritized populations.

Methods

Search strategy and data extraction

Review of health websites (Aim 1)

A list of major health websites with relevance to public health in the USA will be compiled from a search

Theoretical articles (Aim 2)

SA 2 and 3 will include systematic database searches for relevant literature. For SA 2, we will perform a search of theoretical and narrative articles with no restrictions on the date of publication. Details are provided below:

Search strategy MEDLINE (via Ovid MEDLINE® ALL, 1946 to present), Embase (via Embase.com, 1947 to present), Global Health (CABI), and PsycINFO (via Ovid, 1806 to present) will be used for the search strategy of primary articles and narrative reviews. The search will be developed and run by an experienced medical librarian. Subject headings and keywords will be used to search each database when available. The initial search strategy will be built in Ovid MEDLINE and then translated to additional databases. The MEDLINE search strategy for SA 2 is available in Supplemental Table 2.

Inclusion/exclusion criteria We will include theoretical and research articles in our definitions search under SA 2. This will include theoretical articles, primary research articles, narrative reviews, and editorials. Abstracts, dissertations, books, and other media will be excluded. Articles will be included if they have any definitions of “racial health equity” or “health equity” in the context of racial or ethnic health. We will not exclude articles based on date of publication or setting, unless the setting is explicitly mentioned as pertinent to non-highly developed settings. Searches will be limited to English language results. We will also perform hand searches for relevant gray literature under SA 2. Searches will be conducted via Google Scholar and will include additional narrative reviews, editorials, and/or book chapters following the inclusion/exclusion criteria. Full inclusion/exclusion criteria for SA 2 are provided in Table 2.

We will use the Covidence systematic review platform [19] to compile and screen articles for abstract and full-text review under the supplemental search. Covidence is a web-based collaboration software platform that streamlines the production of systematic and other literature reviews. A Covidence license is available to the study team through the University of Colorado Strauss Health Sciences Library. Two reviewers per title will review the title/abstracts for study inclusion, followed by a full-text review by two reviewers for final study inclusion. Any disagreements and conflicts will be resolved by discussion and consensus agreement.

Data extraction Key data extracted will include, but not be limited to author and journal information (e.g., article title, first and last author, countries of authors, areas of expertise, field of expertise, journal, journal

field), article information (e.g., purpose of review, focus of review, scope of review, key questions of review, article conclusions), and definitions (e.g., “health equity,” “race/racial/ethnicity,” “racial health equity,” and appropriate citations and page numbers of definitions). If present, we will also collect definitions and citations for related RHE terms, such as racial health *justice* or *-disparities*. We will use a REDCap database [20, 21], hosted at the University of Colorado Denver, to extract key information from our included studies (SA 2 and 3). REDCap (Research Electronic Data Capture) is a secure, web-based software platform designed to support data capture for research studies, providing (1) an intuitive interface for validated data capture, (2) audit trails for tracking data manipulation and export procedures, (3) automated export procedures for seamless data downloads to common statistical packages, and (4) procedures for data integration and interoperability with external sources. Data extraction will first be tested for congruity between two independent reviewers for at least 10 articles. Data will be extracted by one reviewer and independently verified by a second reviewer.

Evidence synthesis studies (Aim 3)

For SA 3, we will analyze definitions of health equity found within evidence syntheses identified by our team’s overlapping studies of racial health equity interventions within evidence syntheses and methods guidance documents. Evidence syntheses identified under these studies will be reviewed and included for data extraction if definitions of RHE terminology are present.

Search strategy Included evidence syntheses will be published in 2020 onward, while method guidance documents will have no date restriction. Title and abstract screening will be conducted in Distiller (DistillerSR. Version 2.35. DistillerSR Inc., 2023, to be accessed January–June 2023. <https://www.distillersr.com/>) in combination with simultaneous study searches of racial health equity interventions and methods guidance documents [16, 18]. These studies will include evidence syntheses of health interventions to promote health equity for racialized groups that were published since 2020. Multiple independent reviewers (two per title) will review titles and abstracts for potential inclusion, followed by a full-text review for a final determination of study inclusion. Any disagreements and conflicts will be resolved by discussion and consensus agreement. Three members of the study team will then review all articles included for definitions of racial health equity. We anticipate that many of these studies will include references to secondary studies for their definitions, and we will utilize our

team research librarian for assistance in pulling these referenced articles.

Data extraction Data will be extracted into an Excel and REDCap database, as in SA 2 (see above). Data extracted will include, but not be limited to author and journal information (e.g., article title, first and last author, countries of authors, areas of expertise, field of expertise, journal, journal field), syst evidence synthesis information (e.g. type of evidence synthesis, type of intervention, purpose of intervention, health condition targeted), definitions (e.g., “health equity,” “race/racial/ethnicity,” “racial health equity,” and appropriate citations and page numbers of definitions).

Statistical analysis Statistical analysis of SA 1–3 will be limited to summary and frequency statistics of the selected data. GraphPad Prism software (San Diego, CA, USA, www.graphpad.com) will be used to generate summary statistics and generate figures, when appropriate.

Quality assessment and risk-of-bias analysis Due to the nature of the articles we will be reviewing, we will not be conducting quality assessments or risk-of-bias analyses. At this time, we are not aware of any quality or risk-of-bias assessment tools designed for qualitative, theoretical articles that would report terminology information.

Thematic analysis Extracted definitions will be analyzed for recurring words using thematic analysis software. Word cloud software will also be used to visualize frequently used words or concepts.

Stakeholder involvement We recruited diverse leaders in global health equity to our team as regards cultural and linguistic background, country, area of expertise, and gender. Additionally, we recruited a talented advisory board with diverse areas of expertise in health equity. This advisory board helped ensure that research efforts were not duplicated and that relevant data was collected. We followed an inclusive process to design this protocol and incorporate feedback.

Discussion

Determining the current understanding of “racial health equity” is a first step towards promoting actionable and measurable goals to reduce health disparities within the USA. We anticipate that many websites will cite RHE terms but not provide explicit definitions. We also anticipate that most theoretical and systematic review articles that include definitions will cite a secondary source. Collecting data from these secondary sources will be

necessary for mapping definitions and identifying shared sources and concepts.

Our study has several limitations due to the nature of the definitions and linguistics, which are variable by field and constantly evolving. Our website search (SA 1) will be limited to major health organizations that appear in our search or are known to the study team, which will be influenced by our areas of expertise and biases. Our search will also be limited to one accession timepoint, and we acknowledge that definitions may be updated at any time. We will be transparent about this fact by collecting accession date information for all definitions. We will only review websites and articles in English; additionally, we will only collect evidence synthesis articles that have been published since 2020. We acknowledge that relevant articles may be missed, but we anticipate that we will be able to collect the most relevant definitions given the recent expansion of RHE terms in the literature (see Fig. 1).

Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s13643-023-02357-4>.

Additional file 1: **Table S1.** List of websites for review (SA 1). **Table S2.**

Keywords for theoretical article search (SA 2). **Table S3.** Inclusion/exclusion criteria for SA 3 according to PICOT guidelines. Inclusion criteria for systematic reviews is provided from parent study (*Centering Racial Health Equity in Systematic Reviews*) [14].

Acknowledgements

We thank Dr. Preeti Pushpalata Zanwar for her helpful discussions surrounding the scope of this project and our Advisory Board for providing helpful feedback on initial versions of this protocol. Study data will be collected and managed using REDCap electronic data capture tools hosted at the University of Colorado Anschutz Medical Campus [21, 22]. REDCap (Research Electronic Data Capture) is a secure, web-based application designed to support data capture for research studies, providing (1) an intuitive interface for validated data entry, (2) audit trails for tracking data manipulation and export procedures, (3) automated export procedures for seamless data downloads to common statistical packages, and (4) procedures for importing data from external sources.

External peer review

This protocol has been reviewed by steering committee members with the Robert Wood Johnson Foundation.

Authors' contributions

DR is the guarantor. PH provided guidance and oversight for all aspects of this project and the initial project design. ET provided the initial drafting of the manuscript. All authors edited the manuscript. MD assisted with the design of Aim 1 and ET assisted with the design of Aims 2–3. Additional project design and oversight were provided by VW, DF, AP, AR, NS, OD, CO, TD, MV, and DR. Project feedback and guidance were provided by TB and RT. CP created the search strategies and assisted with inclusion/exclusion criteria.

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Availability of data and materials

The data used for this study will be extracted from publications within the search databases as outlined in the “Methods” section. Extracted data will be made available via a University of Colorado library server.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Brain Health Registry Study Partner Portal: Novel infrastructure for digital, dyadic data collection

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Abstract

BACKGROUND: In Alzheimer's disease (AD) research, subjective reports of cognitive and functional decline from participant-study partner dyads is an efficient method of assessing cognitive impairment and clinical progression.

METHODS: Demographics and subjective cognitive/functional decline (Everyday Cognition Scale [ECog]) scores from dyads enrolled in the Brain Health Registry (BHR) Study Partner Portal were analyzed. Associations between dyad characteristics and both ECog scores and study engagement were investigated.

RESULTS: A total of 10,494 BHR participants (mean age = 66.9 ± 12.16 standard deviations, 67.4% female) have enrolled study partners (mean age = 64.3 ± 14.3 standard deviations, 49.3% female), including 8987 dyads with a participant 55 years of age or

Anna Aaronson and Miriam T. Ashford contributed equally to this study.

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older. Older and more educated study partners were more likely to complete tasks and return for follow-up. Twenty-five percent to 27% of older adult participants had self and study partner-report ECog scores indicating a possible cognitive impairment.

DISCUSSION: The BHR Study Partner Portal is a unique digital tool for capturing dyadic data, with high impact applications in the clinical neuroscience and AD fields.

KEYWORDS

aging research, Alzheimer's disease, Brain Health Registry, dementia, diversity, dyadic report, internet, internet registry, online, remote assessment, subjective cognitive decline

Highlights

- The Brain Health Registry (BHR) Study Partner Portal is a novel, digital platform of >10,000 dyads.
- Collection of dyadic online subjective cognitive and functional data is feasible.
- The portal has good usability as evidenced by positive study partner feedback.
- The portal is a potential scalable strategy for cognitive impairment screening in older adults.

1 | BACKGROUND

In cognitive aging and Alzheimer's disease and related dementias (ADRD) research, many clinical trials and observational studies require data collected from participant and study partner/informant pairs, also referred to as dyads. Study partners are often the participant's spouse, adult child, other family member, or close friend. Ideally the study partner knows the participant well enough to be able to provide insight into the current status of, and recent changes in, the participant's cognitive and functional abilities.^{1,2} Advantages of involving dyads in research include (1) efficient, accurate, and reliable data for assessing ADRD risk^{3,4}; (2) study partners may have insight into changes in complex activities of daily living, which are difficult to assess using neuropsychological tests^{1,2,5}; (3) study partner report is less affected by the participant's mood and declining awareness of their own abilities associated with dementia^{5,6}; (4) study partner involvement can help ensure participant safety and data integrity, and decrease study dropout.^{7,8}

Although most dyadic data are collected in in-clinic studies, dyadic data collection has been adapted recently to remote, unsupervised settings.^{5,9–12} The Brain Health Registry (BHR) Study Partner Portal is a novel and scalable platform to support remote collection of study partner data^{10,13} within the University of California, San Francisco Brain Health Registry. BHR is an online cognitive aging and ADRD-related research registry and cohort ($N > 92,000$ participants). Previous results support the preliminary feasibility of the BHR Study Partner Portal, and the validity of data collected.^{9,10,13,14} The goal of this exploratory study was to provide a detailed description of the dyads enrolled in the BHR Study Partner Portal, including their demographics, the subjective cognitive and functional data collected, and the dyad characteristics associated with study participation.

2 | METHODS

2.1 | Brain health registry (BHR)

The BHR is a public online registry. Adults (age 18+) can register, sign a digital informed consent, and complete online longitudinal tasks at 6-month intervals, including questionnaires and neuropsychological assessments.^{10,13} The BHR study is approved by the University of California, San Francisco Internal Review Board.

The BHR Study Partner Portal^{9,10} was launched in 2016 and is a novel, scalable, remote, online portal within the BHR. All enrolled BHR participants are presented with a "My Study Partner" questionnaire, which describes what a study partner is, and asks whether they have someone who could serve as their study partner. If a BHR participant indicates that they have one potential study partner, they are asked to enter this person's name and contact information. BHR then automatically sends the potential study partner an email describing the Study Partner Portal and asking them to join as the study partner for their associated BHR participant by following an email link. Potential study partners who click the link are directed to a page with more information about the Study Partner Portal and can register and sign an online informed consent. Consented study partners are considered enrolled. If the study partner does not respond to the initial invitation, three additional email invitations are sent 8, 11, and 21 days after the initial invitation. Participants are given an opportunity to confirm or change their study partner in annual follow-up visits to the BHR website. Study partners answer online questionnaires about the BHR participant and the study partner themselves, which take ≈ 30 minutes to complete. Questionnaires about the associated participant include a short health screener, and the following scales adapted for online use⁹: Everyday Cognition Scale (ECog),¹⁵ Functional Activities

Questionnaire (FAQ),^{16,17} Cognitive Function Instrument (CFI),^{18,19} and Mild Behavioral Impairment Checklist (MBI-C).^{20,21} Questions about the study partners themselves include demographics, relationship to BHR participants, and a short health screener. A unique identification code links data between the study partner and participant. Study partners and participants receive automated reminder emails to finish tasks, and to return for follow-up visits at 6-month intervals. Participants are not informed about the information that study partners provide.

For this study, study partners and BHR participants came from two sources: (1) enrolled in the BHR from the general public (April 2014 to September 2022; N = 10,113); or (2) first enrolled in the Imaging Dementia—Evidence for Amyloid Scanning (IDEAS) study^{9,22} and then invited to join BHR via email (February 2017 to February 2018; N = 381). IDEAS is a study with more than 18,000 participants age 65 years and older with Mild Cognitive Impairment (MCI) or dementia of uncertain etiology, with the goal to establish the clinical utility of a brain amyloid beta (A β) positron emission tomography (PET) scan. This analysis included all study partners and participants from both sources with data necessary to perform analyses.

2.2 | Participant metrics

2.2.1 | Demographic information

These analyses used the following study partner- and participant-reported demographic information: current age (continuous), gender (male, female, other, prefer not to say), years of educational attainment (continuous), ethnicity (Latino, non-Latino, declined to state), and race (Asian, African American/Black, Native American, Pacific Islander, Other, White, Multiple, and declined to state).

2.2.2 | Medical history

The following participant-reported medical history variables were included in the analysis: history of Alzheimer's disease (AD) ("Do you have any biological parents, full siblings, or biological children who have been diagnosed with AD?"), self-report of MCI ("Please indicate whether you currently have or have had any of the following conditions in the past: Mild Cognitive Impairment"), AD ("Please indicate whether you currently have or have had any of the following conditions in the past: Alzheimer's Disease"), dementia ("Please indicate whether you currently have or have had any of the following conditions in the past: Dementia").

2.2.3 | Self-reported memory concern

The analysis used both participant- and study partner-reported memory concern ("Are you concerned that you/[associated participant name] have/has a memory problem?")

RESEARCH IN CONTEXT

1. **Systematic review:** A review of the literature on dyadic subjective cognitive decline was conducted using electronic databases. Previous studies supported preliminary feasibility of the Brain Health Registry (BHR) Study Partner Portal, and the validity of the data collected.
2. **Interpretation:** Collection of remote, unsupervised, digital, online longitudinal subjective cognitive/functional data from a large cohort of dyads (participant, study partner pairs) is feasible. Dyads had high task completion and retention rates. However, characteristics associated with study partner enrollment, engagement, and retention levels highlighted selection biases for older and highly educated dyads. The BHR Study Partner Portal has good usability, as demonstrated by positive study partner feedback about their experience.
3. **Future directions:** Much needed efforts are underway and planned to increase the ethnocultural and socioeconomic diversity of dyads enrolled in the BHR. Important next steps are to look at the relationship between subjective and objective cognitive measures, and the contributions of dyad demographics to this relationship.

2.2.4 | Everyday Cognition Scale (ECog)

The ECog measures change in instrumental activities of daily living compared to 10 years before.¹⁵ ECog is completed separately by the participant and study partner, and includes questions related to six cognitive domains: Everyday Memory (e.g., remembering a few shopping items without a list), Everyday Language (e.g., forgetting the names of objects), Everyday Visuospatial Abilities (e.g., following a map to find a new location), Everyday Planning (e.g., thinking things through before acting), Everyday Organization (e.g., keeping living and workspace organized), and Everyday Divided Attention (e.g., the ability to do two things at once). ECog scores range from 1 to 4. BHR uses a version adapted for online use,⁹ with higher scores indicating a greater decline. The ECog is administered every 6 months in BHR. This analysis used baseline ECog scores. To identify participants with ECog scores falling into a range associated with possible MCI, we used cutoffs that were >1 standard deviation (SD) from the mean ECog scores of cognitively unimpaired participants in the Alzheimer's Disease Neuroimaging Initiative (ADNI) (Self-report ECog score mean = 1.34, SD = 0.31; Study partner-report ECog score mean = 1.17, SD = 0.26).²³ The cutoffs applied to define possible MCI were Self-report ECog score >1.65 or study partner report ECog score >1.43 .²³

2.2.5 | Additional subjective measures

The functional activities questionnaire (or FAQ) is a 10-item screening scale for evaluating instrumental activities of daily living (e.g., balancing a checkbook, writing checks, paying bills, and remembering appointments, family occasions, holidays, and medications) in research settings.^{16,17} Items are rated on a six-point scale (1 = Normal, 2 = Never did, but could now do, 3 = Never did, would have difficulty now, 4 = Has difficulty, but does by self, 5 = Requires assistance, 6 = Dependent). The Cognitive Function Instrument (or CFI) is a 14-item scale that assesses recent changes (compared to 1 year ago) in cognition and activities of daily living.^{18,19} Response options include Yes (1), Maybe (2), and No (3). The BHR Study Partner Portal uses online adapted versions of the FAQ and CFI reported by the study partner about the associated BHR participant. Baseline FAQ and CFI scores was used for this analysis.

2.3 | Feasibility metrics

2.3.1 | Enrollment metrics

Enrollment metrics included in this analysis are the number of BHR participants who (1) Completed the "My Study Partner" questionnaire; (2) Identified a potential study partner; and (3) Indicated that they did not have a potential study partner. For those who invited a potential study partner, we report whether the invitation status was accepted, pending, or declined. We also report whether the study partner is active, defined as having started any questionnaire in the study partner module.

2.3.2 | Task completion and retention metrics

Completion metrics included whether study partners completed all Study Partner Portal tasks at least once (yes, no) and whether study partners completed the core questionnaire at least once (yes, no). The core questionnaire is the first questionnaire that study partners are asked to complete, and asks about demographic information, mood, health, medications, memory, and self-report diagnoses of MCI and AD for the study partner and participant. Retention metrics included completion of the core questionnaire at least twice (yes, no) and completion of study partner rated ECog at least twice (yes, no).

2.4 | Usability metrics

2.4.1 | Feedback questionnaire

While logged into the Study Partner Portal, study partners can at any time provide optional feedback about their experience by clicking on a widget labeled "Feedback." The widget directs study partners to an online questionnaire that includes the following questions: (1) "How easy was this site to use? From a 1–10 scale of not easy to very easy"; (2)

"How clear were the instructions for the site? From a 1–10 scale of not clear to very clear"; (3) "How accurately did we predict the time necessary to answer the questionnaires and take the tests?" (1 = It took a lot more time than expected – 5 = It took a lot less time than expected). The questionnaire also includes two questions with free-text response options ("What, if anything, was frustrating?" and "How can we improve?"). This analysis focused on the subset of study partners who provided this optional feedback, and used responses from the first time the questionnaire was completed (quantitative: N = 2034; qualitative: N = 735) the qualitative questions were analyzed using an open-coding approach and thematic analysis.²⁴ Blank responses were skipped.

2.4.2 | Statistical analysis

Descriptive information was tabulated for four samples: (1) all study partners, (2) all BHR participants with enrolled study partners, (3) study partners with an associated participant age 55 or older, and (4) BHR participants age 55 or older with enrolled study partners. Descriptive information was summarized using Ns and percentages for categorical variables (gender, race, ethnicity, subjective memory concern, family history of AD, BHR participant-reported MCI, AD) and means and SDs for continuous variables (age, education, study partner- and BHR participant-reported ECog scores, FAQ score, CFI score). In study partners with an associated BHR participant aged 55+, we estimated associations between study partner-reported ECog (outcome) and the following variables (predictors) using multivariable ordinary least-squares linear regression: participant-reported ECog; participant demographics (age, gender, education, race, and ethnicity); participant reported MCI, AD, and dementia; study partner demographics (age, gender, education, race, and ethnicity). Regression coefficients and 95% confidence intervals (CIs) are reported. In the same sample, multivariable binomial logistic regressions were fit to estimate associations between the study partner task completion and retention outcome metrics and demographic information. Each task completion and retention outcome metric was modeled separately. Predictors included either study partner- or BHR participant-reported demographic information (age, gender, education, race, and ethnicity). For all logistic regression models, odds ratios (ORs), 95% CIs, and p-values are reported. Analyses were done in SAS 9.4 (SAS Institute, Cary NC) and R.²⁵

3 | RESULTS

3.1 | Feasibility

3.1.1 | Study partner enrollment

Of 92,626 participants enrolled in BHR, a total of 25,374 (27.4%) indicated that they did not have a potential study partner in the "My Study Partner" questionnaire (see *Supplemental Material*). A total of 18,802 (20.3%) indicated that they had a potential study partner, who was then sent an invitation to join BHR. A total of 10,644 study partners

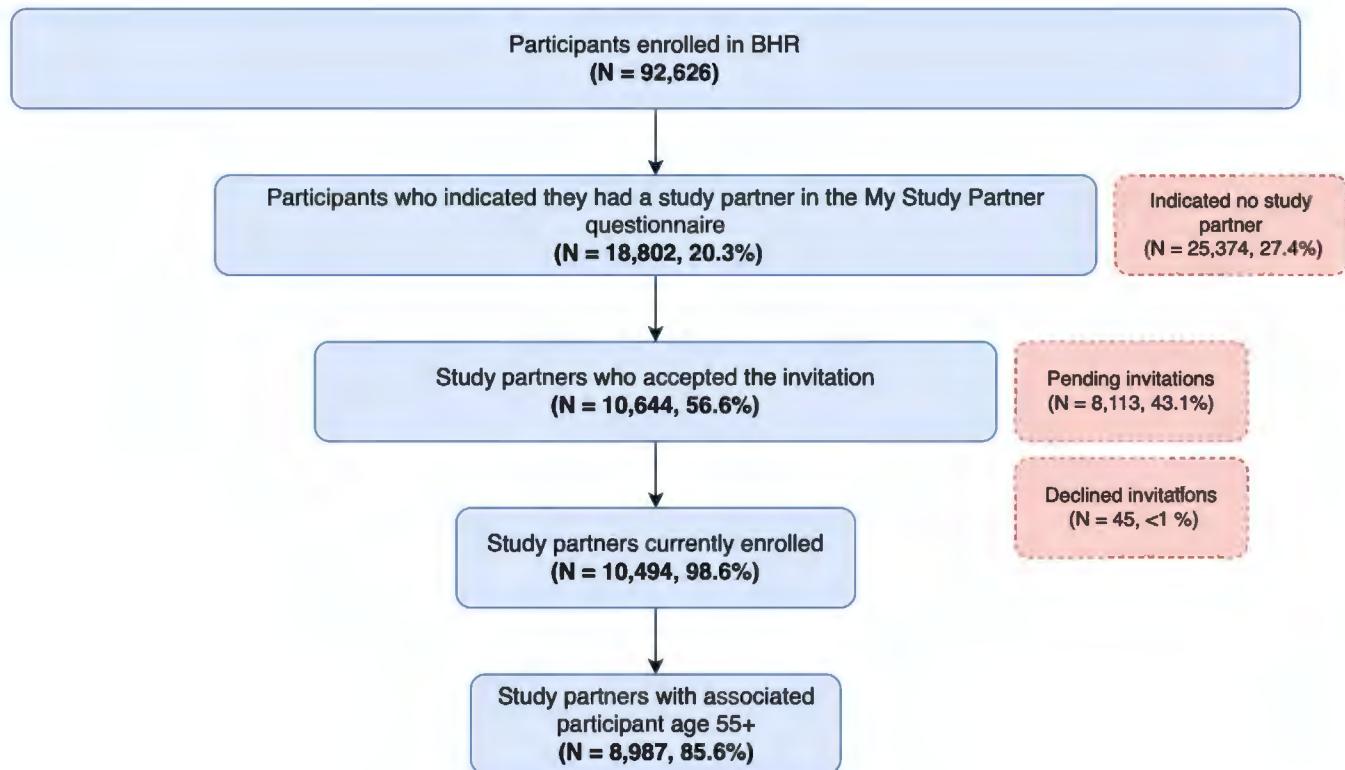


FIGURE 1 Study partner enrollment into the Brain Health Registry.

(56.6%) accepted the invitation, 45 (< 1%) declined, and 8113 (43.1%) invitations are pending, meaning the prospective study partner has not yet responded. A total of 10,494 study partners (98.6%) enrolled and signed an online consent. Of all enrolled study partners, 8987 (85.6%) have an associated BHR participant 55 years of age or older (Figure 1).

3.1.2 | Characteristics of BHR participants with study partners

Demographics of BHR participants with study partners are shown in Table 1. Compared to BHR participants ages 55+ who indicated that they did not have a potential study partner, participants with an enrolled study partner were significantly older (mean = 71.4, SD = 8.1 vs mean = 69.6, SD = 8.5 years of age) and had a higher educational attainment (mean = 16.6, SD = 2.3 vs mean = 15.6, SD = 2.5 years). Those with a study partner had a higher percentage of those identifying as White ($n = 8361$ [94.6%] vs $n = 49,665$ [81.3%]) and a lower percentage of those identifying as female ($n = 5844$ [66.1%] vs $n = 45,684$ [74.8%]). There were significantly higher percentages of participants with self-report MCI ($n = 894$ [10.1%] vs $n = 2893$ [4.7%]), AD ($n = 245$ [2.8%] vs $n = 467$ [0.7%]), and dementia ($n = 274$ [3.1%] vs $n = 687$ [1.1%]) in the group with a study partner versus the group without. For a more detailed comparison of these two groups, see Table S1.

3.1.3 | Characteristics of study partners

Study partners had an average age of 64.3 (SD = 14.25) and average educational attainment of 16.3 years (SD = 2.36). A total of 5174 (49.3%) identified as female, 9190 (87.6%) identified as White, and 437 (4.2%) reported Latino ethnicity. Study partners with an associated participant age 55+ were on average 67.1 years old (SD = 12.6) and had an average educational attainment of 16.3 years (SD = 2.37). A total of 4526 (50.4%) identified as female, 7950 (88.5%) identified as White, and 310 (3.4%) reported Latino ethnicity (see Table 2 for demographics of all ethnocultural groups).

3.1.4 | Task completion and retention

A total of 5370 (51.2%) study partners completed at least one longitudinal follow-up visit, and 4776 completed at least two instances of ECog. A total of 4741 study partners (52.8%) with participants age 55+ completed at least one longitudinal follow-up, and 4258 completed at least two ECog sessions (Figure 2). A total of study partners 6648 (74.0%) with participants age 55+ completed the entire study partner module at least once, and 7593 (72.4%) of all study partners completed the entire study partner module at least once. Fifty-three participants changed study partners over the course of their enrollment in BHR.

TABLE 1 Characteristics of all participants with study partners.

	N = 10,494 (all participants)	N = 8987 (participants age 55+)
Age, mean \pm SD (N) (min–max)	66.9 \pm 12.16 (N = 10,484) (22–90)	70.7 \pm 7.97 (N = 8987) (55–90)
Education in years, mean \pm SD (N) (min–max)	16.7 \pm 2.26 (N = 10,483) (12–20)	16.7 \pm 2.28 (N = 8985) (12–20)
Female, n (%)	7078 (67.4%)	5957 (66.3%)
Race, n (%)		
Black/African American	201 (1.9%)	162 (1.8%)
Asian	271 (2.6%)	190 (2.1%)
Native American	207 (2.0%)	164 (1.8%)
Pacific Islander	31 (0.3%)	21 (0.2%)
White	9832 (93.7%)	8507 (94.7%)
Other	253 (2.4%)	169 (1.9%)
More than one race	314 (3.0%)	233 (2.6%)
Latino ethnicity, n (%)	513 (4.9%)	350 (3.9%)
Subjective memory concern, n (%)	5311 (50.6%)	4690 (52.2%)
Self-report MCI, n (%)	806 (7.7%)	771 (8.6%)
Self-report AD, n (%)	214 (2.0%)	211 (2.3%)
Self-report dementia, n (%)	226 (2.2%)	221 (2.5%)
Family history of AD, n (%)	3888 (37.0%)	3523 (39.2%)

Abbreviations: AD, Alzheimer's disease; MCI, mild cognitive impairment; SD, standard deviation.

TABLE 2 Characteristics of study partners.

	N = 10,494 (all study partners)	N = 8987 (study partners of participants 55+)
Age, mean \pm SD (N) (min–max)	64.3 \pm 14.25 (N = 10,492) (18–90)	67.1 \pm 12.51 (N = 8985) (19–90)
Education in years, mean \pm SD (N) (min–max)	16.3 \pm 2.36 (N = 10,110) (12–20)	16.3 \pm 2.37 (N = 8648) (12–20)
Female, n (%)	5174 (49.3%)	4526 (50.4%)
Race, n (%)		
Black/African American	200 (1.9%)	155 (1.7%)
Asian	302 (2.9%)	219 (2.4%)
Native American	158 (1.5%)	124 (1.4%)
Pacific Islander	32 (0.3%)	25 (0.3%)
White	9190 (87.6%)	7950 (88.5%)
Other	271 (2.6%)	183 (2.0%)
More than one race	247 (2.4%)	183 (2.0%)
Latino ethnicity, n (%)	437 (4.2%)	310 (3.4%)
SP-report ECog score, M \pm SD (N) (min–max)	1.35 \pm 0.496 (1–4)	1.37 \pm 0.507 (N = 7315) (1–4)
SP CFI score, mean \pm SD (N) (min–max)	\pm 2.18 (N = 9747) (0–24)	1.46 \pm 2.23 (N = 8364) (0–14)
SP FAQ score, mean \pm SD (N) (min–max)	\pm 3.62 (N = 7472) (0–30)	1.24 \pm 3.75 (N = 6526) (0–30)

Abbreviations: CFI, Cognitive Function Instrument; ECog, Everyday Cognition; FAQ, Functional Activities Questionnaire; SD, standard deviation; SP, study partner.

3.1.5 | Characteristics associated with task completion and retention

Higher study partner age (OR = 1.013, CI = 1.008–1.017), higher study partner educational attainment (OR = 1.115, CI = 1.090–1.140), study partner female gender (OR = 1.215, CI = 1.076–1.373), higher participant age (OR = 1.006, CI = 1.001–1.011), and participant African

American/Black race (OR = 1.942, CI = 1.091 to 3.5) were significantly associated with a higher probability of study partner task completion. Study partner African American/Black race (OR = 0.386, CI = 0.234–0.636), study partner Asian race (OR = 0.614, CI = 0.440–0.857), study partner other race (OR = 0.507, CI = 0.360–0.714), and study partner multiple races (OR = 0.707, CI = 0.527–0.949) were significantly associated with a lower probability of study partner task completion.

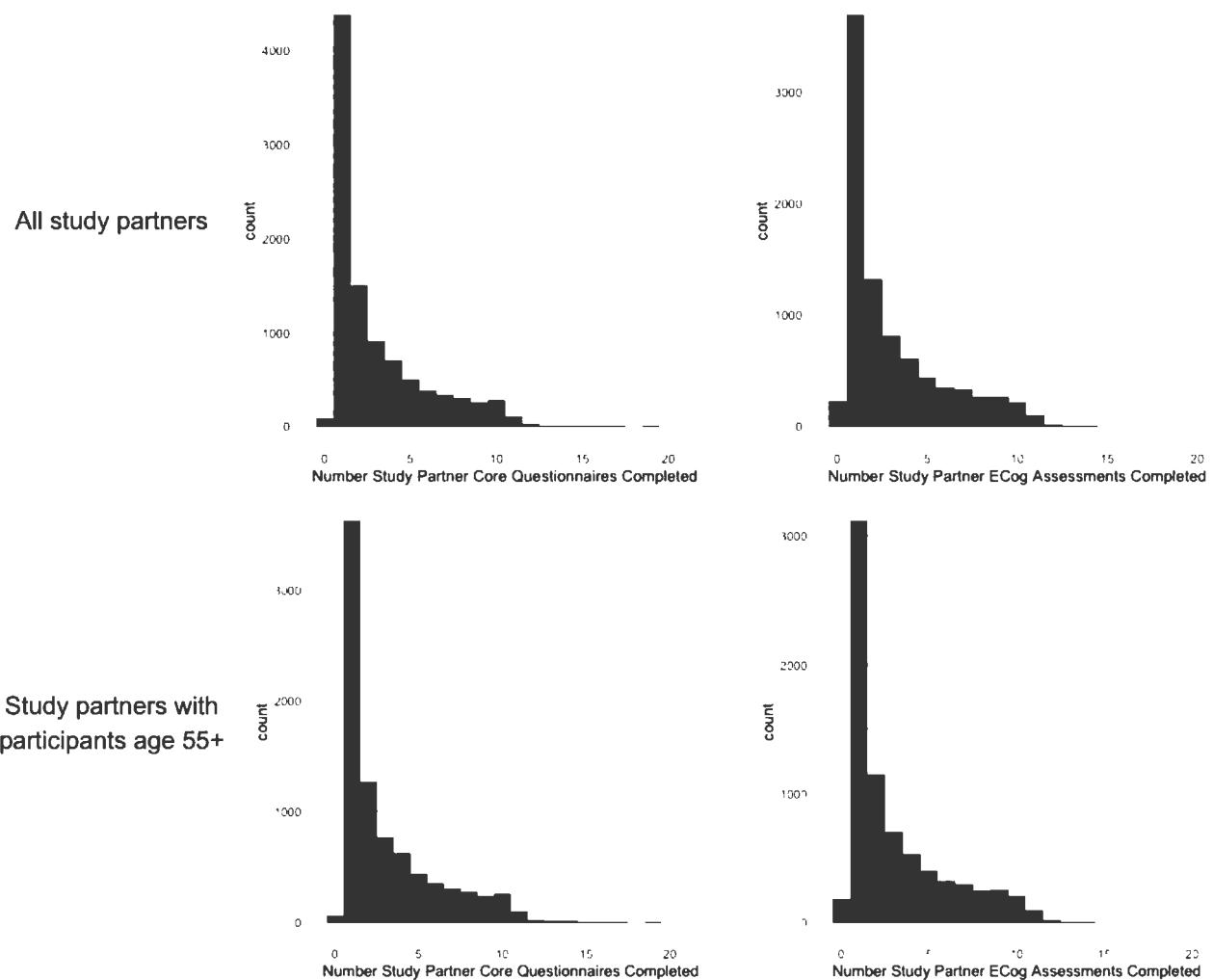


FIGURE 2 Top row: Study partner core questionnaire and Everyday Cognition assessments completed by all study partners. Bottom row: Core questionnaire and Everyday Cognition assessments completed by study partners with participants age 55+.

Higher study partner age (OR = 1.010, CI = 1.005–1.014), higher study partner educational attainment (OR = 1.075, CI = 1.053–1.099), study partner female gender (OR = 1.187, CI = 1.056–1.334), and higher participant educational attainment (OR = 1.027, CI = 1.005, -1.050), were significantly associated with a higher probability of completion of at least two study partner ECog sessions. Participant other race (OR = 0.610, CI = 0.374–0.995) and participant Latino ethnicity (OR = 0.734, CI = 0.557–0.968) were associated with a lower probability of completion of at least two study partner ECog sessions.

3.2 | Study partner- and self-report subjective cognitive and functional decline

3.2.1 | ECog scores

For study partners with participants age 55+, the mean study partner-report ECog score was 1.37 (SD = 0.507) and mean self-report ECog score was 1.47 (SD = 0.47). Using the established cut points, we found

that of 7315 participants age 55+ with available study partner-report ECog scores, 2038 (27.9%) were possibly impaired. A total of 2095 (24.2%) of 8652 participants with available self-report ECog scores were possibly impaired. For ECog results in the entire study partner cohort, see *Supplemental Results*.

3.2.2 | Relationship between self- and study partner-report ECog

Study partner- and self-report ECog scores were correlated for the entire study partner cohort ($r = 0.47, p < 0.001$) and for the age 55+ cohort ($r = 0.46, p < 0.001$) (Figure 3).

3.2.3 | Dyad characteristics associated with ECog scores

In study partners with associated participants age 55+, higher (worse) study partner ECog score was associated with a lower

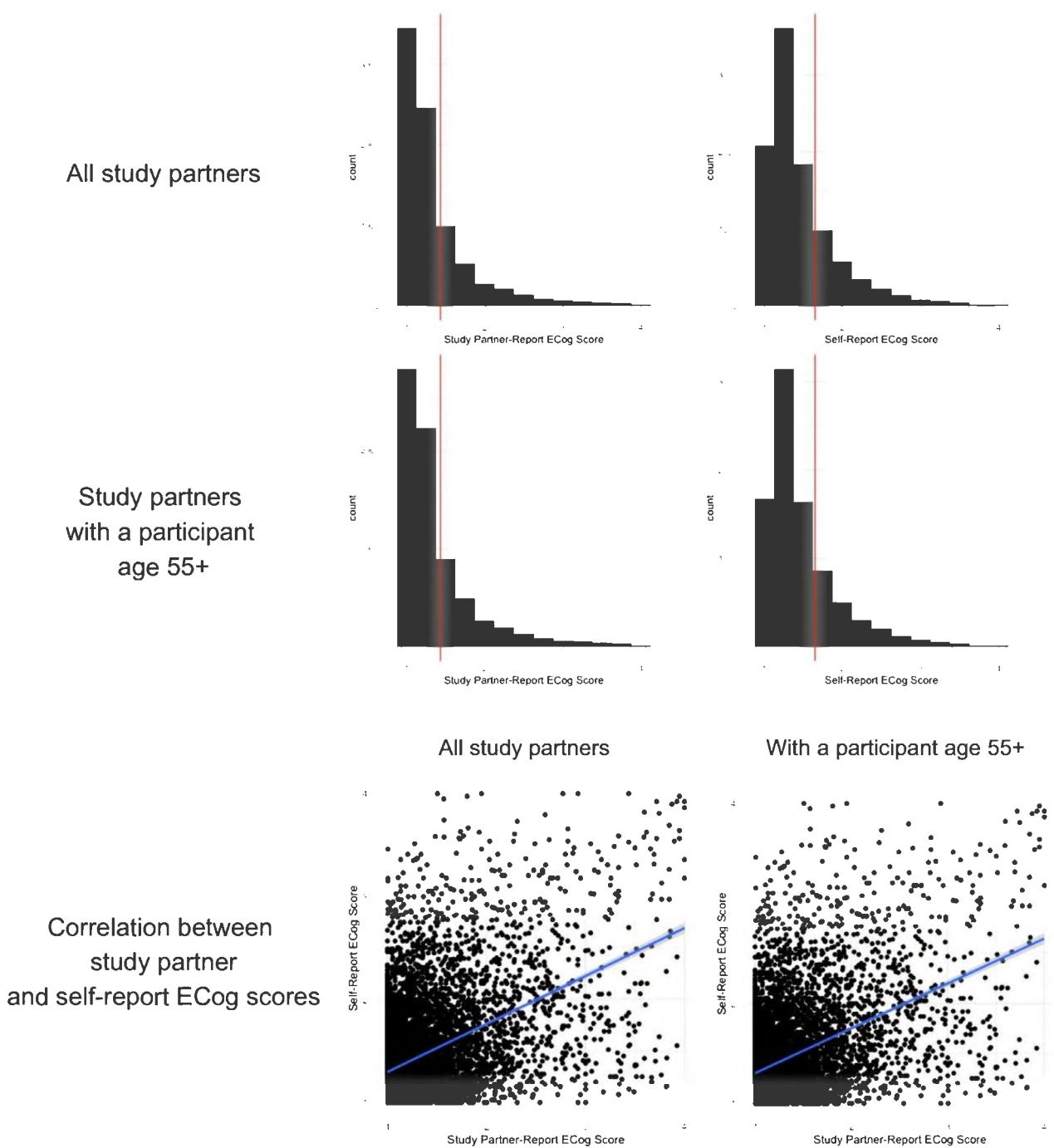


FIGURE 3 “Possibly impaired” cut points are represented by red vertical lines (1.43 for study partner–report scores and 1.65 for self-report scores). Top row: Self- and study partner–report ECog scores for all study partners and all associated participants. Middle row: Self- and study partner–report ECog scores all study partners with participants age 55+ and associated participants age 55+. Bottom row: Scatterplot of study partner– and self-report ECog scores in all participants and study partners 55+ and associated study partners.

study partner age in years, Native American study partner race, and study partner identification with multiple races. Higher study partner ECog score was associated with advanced participant age in years, lower participant educational attainment, male participant gender, and participant Native American race (Table 3).

3.3 | Usability of the BHR study partner portal

3.3.1 | Quantitative feedback

Study partners who answered the optional feedback questions about their experience ($N = 2034$; See Table S2) rated the portal as very easy

TABLE 3 Estimated regression coefficients and 95% confidence intervals from linear regression models fit to study partner ECog score.

Study partner characteristic	Study partner-report ECog score β (95% confidence interval)	Participant characteristic	Study partner-report ECog Score β (95% confidence interval)
Age in years	-0.003 (-0.004, -0.002)*	Age in years	0.013 (0.011, 0.015)*
Years of education	-0.005 (-0.010, 0.001)	Years of education	-0.016 (-0.022, -0.011)*
Gender		Gender	
Male	1.0 (reference)	Male	1.0 (reference)
Female	-0.011 (-0.043, 0.021)	Female	-0.093 (-0.125, -0.061)*
Race		Race	
African American/Black	0.106 (-0.045, 0.256)	African American/Black	-0.062 (-0.216, 0.092)
Asian	-0.037 (-0.133, 0.058)	Asian	0.006 (-0.098, 0.109)
Native American	0.278 (0.043, 0.513)*	Native American	0.317 (0.074, 0.560)*
Pacific Islander	-0.30 (-0.367, 0.306)	Pacific Islander	-0.240 (-0.666, 0.187)
White	1.0 (reference)	White	1.0 (reference)
Multiple	0.130 (0.037, 0.222)*	Multiple	0.051 (-0.032, 0.134)
Other	-0.015 (-0.136, 0.107)	Other	0.053 (-0.088, 0.193)
Ethnicity		Ethnicity	
Latino	0.016 (-0.068, 0.100)	Latino	-0.028 (-0.107, 0.051)
Not Latino	1.0 (reference)	Not Latino	1.0 (reference)

Note: * = $p < 0.05$.

to use (mean = 9.2, SD = 1.4) and the site instructions as very clear (mean = 9.2, SD = 1.3). Over half of the respondents (54.9%) indicated that it took them more time than expected to complete the tasks.

3.3.2 | Qualitative feedback

A total of 735 study partners answered one or both qualitative feedback questions. Answers fell into three overarching themes: task completion issues, content issues, and feedback that was not actionable (answers unrelated to questions, or study partners indicating that they had no feedback). For the task completion issue theme, technical issues and accessibility issues were identified as sub-themes. For the content issue, difficulties and design change requests were identified as sub-themes. See Table S3 for overarching themes, sub-themes, and a detailed description of each sub-theme.

4 | DISCUSSION

The main findings of this study are (1) Collection of remote, unsupervised, digital, online longitudinal subjective cognitive/functional data from a large cohort of dyads (participant–study partner pairs) is feasible. Dyads had high task completion and retention rates. However, characteristics associated with study partner enrollment, engagement, and retention levels highlighted important selection biases, including those for older and more highly educated dyads. (2) Greater subjective cognitive/functional decline (higher ECog scores) are associated with advanced dyad age, lower participant education, participant male

gender, and dyad Native American ethnocultural identity; (3) Approximately 25% of older adult participants in our cohort demonstrated evidence for possible cognitive impairment, based on ECog scores; (4) The BHR Study Partner Portal has good usability, as demonstrated by positive study partner feedback about their experience.

In terms of feasibility, we found that 74% of study partners with participants age 55+ completed all study questionnaires. Furthermore, in terms of longitudinal retention, we found that just over 50% of study partners completed at least one longitudinal follow-up visit. In comparison, in a recent analysis of the entire BHR participant cohort,¹³ we found that only 45% of participants completed at least two core questionnaires. This suggests that enrolled study partners have a high level of engagement, compared to BHR participants. This may be because initial enrollment in the Study Partner Portal is a sign of a high level of engagement, causing a selection bias for highly engaged participants. In addition, the entire study partner module is substantially shorter than the BHR participant module (30 min vs >1 h). In terms of usability, we found that, on average, study partners who answered optional questions about their study partner experience rated the BHR Study Partner Portal site as very easy to use and rated the site instructions as very clear. It is important to note that these feasibility and usability measures are limited by a biased sample that is disproportionately well educated and White.

Although we have previously analyzed engagement and retention of BHR participants, this is the first study to report these metrics specific to enrolled study partners. We identified study partner and participant demographic characteristics associated with higher levels of engagement and retention. In terms of engagement, study partner older age, female gender, and higher educational attainment, and

participant Black/African American ethnocultural identity were all associated with a greater probability of completing tasks. In terms of retention, higher study partner and participant educational attainment and study partner female gender were associated with a higher probability of longitudinal retention. Participant Other race and participant Latino ethnicity were associated with a lower probability of longitudinal retention. This is consistent with previous findings that many studies fail to adequately engage and retain non-White individuals, those with lower educational attainment, and male participants.^{26,27} These findings also are consistent with a previous analysis of the BHR cohort,²⁸ which found that non-White race, Latino ethnicity, and lower educational attainment were associated with decreased task completion.

The BHR Study Partner Portal is the largest study to our knowledge to include longitudinal measures of subjective cognitive and functional decline, with 56,839 instances of self-report ECog, 22,554 instances of study partner-report ECog, $n = 8480$ unique study partners providing ECog data, and $n = 4776$ study partners with longitudinal ECog data. We found that $\approx 25\%$ of older adult participants have ECog scores indicating the possibility of cognitive impairment within the MCI range.²³ Collection of dyadic subjective decline data in the Study Partner Portal represents a scalable, efficient strategy for screening older adults for cognitive impairment relevant to AD, especially in light of past work demonstrating a strong relationship between BHR study partner-report ECog scores and clinically confirmed MCI diagnosis.¹⁴ One of the main ways that BHR facilitates AD and related research is through referral of BHR participants to other studies, with more than 25,997 BHR participants enrolled in 30 different studies.¹³ Dyadic BHR data can be used in the future to identify participants to be referred to studies seeking older adults with MCI, including observational studies and treatment trials.

This large longitudinal data set, combined with demographic data, also provides a unique opportunity to identify dyad characteristics associated with subjective decline scores. We found that higher (worse) study partner ECog scores were associated with advanced participant age, lower participant educational attainment, male participant gender, study partner identification with multiple race categories, and study partner and participant Native American race. This finding contributes to a growing literature characterizing subjective decline in diverse populations.^{29–32} It suggests that dyad demographics can influence study partner reports of subjective cognitive decline and should be accounted for in future studies. An important next step is to look at the relationship between subjective and objective cognitive measures, and the contributions of demographics to this relationship. Others have found that this relationship is weaker in older adults from minoritized ethnocultural communities.²⁹ Another next step is to investigate the role of dyad relationship (i.e., spouse versus other type of study partner) in engagement and completion rates. Other research has demonstrated that spousal study partners are more willing to participate and have lower dropout rates compared to non-spousal study partners.^{7,33}

Limitations of our study include selection biases at multiple study stages, including BHR enrollment, study partner enrollment, and study

partner longitudinal retention. At each stage, we demonstrate a failure to adequately include and engage dyads from underrepresented ethnocultural groups and those with low education levels. There are numerous, complex factors contributing to the lack of inclusion of these groups in research, including failure of researchers to gain trust in these communities due to a legacy of unethical treatment, higher rates of comorbidities among these groups, failure of investigators to share information about studies with these groups, time burden of participation, issues with study design, and structural factors.^{34,35} Frameworks such as community-engaged research have shown promise for increasing inclusion and engagement of minoritized communities, such as Black and Latino individuals, in ADRD research.^{35–37} Several initiatives are now underway in BHR to improve participation of minoritized groups.

Furthermore, as with other online research studies, BHR, including the Study Partner Portal, has selection biases for those with adequate technology and internet access and literacy to complete online tasks remotely and unsupervised. These selection biases limit the generalizability of our results. Although the digital divide is narrowing for underrepresented populations, it still persists.^{38,39} We have begun multiple new initiatives aimed at including and engaging diverse dyads, which is a crucial next step in realizing the potential impact of this approach. An additional, more general limitation of relying on study partner data in AD research is that many older adults, especially those from underrepresented populations, do not have someone who is able to serve as their study partner.⁴⁰ This limitation has the potential to further exacerbate the selection biases we described.

In conclusion, the BHR Study Partner Portal is a novel, scalable approach to the collection of dyadic, subjective cognitive and functional data. This approach has many potential high impact applications in the clinical neuroscience, cognitive aging, and ADRD fields. The data collected can be used to characterize longitudinal subjective decline in a large cohort of adults who have extensive cognitive, health, and lifestyle data through their BHR participation. The entire de-identified dataset can be shared with other investigators, who can test their own hypotheses related to dyadic subjective measures. Enrolled study partners and their associated participants can be referred to other studies, facilitating recruitment and screening in many clinical research studies. The Study Partner Portal online infrastructure can be adapted for use in many different studies and settings, so that other investigators can collect and analyze dyadic data.

AUTHOR CONTRIBUTIONS

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CONFLICT OF INTEREST STATEMENT

Anna Aaronson, Chengshi Jin, Jessica Bride, Josephine Decker, Aaron DeNicola, Catherine Conti, Rachana Tank, Monica R. Camacho, Derek Flenniken, Juliet Fockler, Diana Truran, Aaron Ulbricht, and Maria C. Carrillo report no potential conflict of interest. Author disclosures are available in the [Supporting Information](#).

CONSENT STATEMENT

All participants and study partners in the Brain Health Registry provided informed consent by signing an electronic consent form.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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Regular Research Article

Childhood and Adulthood Trauma Associate With Cognitive Aging Among Black and White Older Adults

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ABSTRACT

Sociocontextual factors powerfully shape risk for age-related cognitive impairment, including excess risk burdening medically underserved populations. Lifecourse adversity associates with cognitive aging, but harms are likely mitigable. Understanding population-salient relationships and sensitive periods for exposure is crucial for targeting clinical interventions. **Objective:** The authors examined childhood and adulthood traumatic events in relation to cognition among Black and White older adults in the Health and Retirement Study (HRS). **Participants:** Participants ($N = 13,952$) aged 55+ had complete lifetime trauma and cognitive testing data at the 2006/08, 2010/12, and/or 2014/16 waves. **Measures:** Trauma indices comprised childhood and adulthood event counts. Outcomes included baseline performance and trajectories on the Telephone Interview for Cognitive Status. **Design:** Main and nonlinear trauma effects were modeled via linear regression, and overall contributions assessed with omnibus likelihood ratio tests. **Results:** Black participants ($N = 2,345$) reported marginally lower adulthood trauma exposure than White participants ($N = 11,607$) with no other exposure differentials observed. In White participants only, greater childhood trauma exposure predicted worse baseline cognition but slower change over time. Across race, adulthood trauma robustly associated with baseline cognition. Relationships were frequently

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nonlinear: low but nonzero trauma predicted highest cognitive scores, with much poorer cognition observed as trauma exposure increased. Relationships between adulthood trauma and trajectory were limited to the White sample.
Conclusion: Traumatic experiences, particularly in adulthood, may impact late-life cognitive health if not addressed. Findings highlight foci for clinical researchers and providers: adverse life events as a source of cognitive risk, and identification of community-specific resources that buffer behavioral, physical, and mental health sequelae of previous and incident trauma. (Am J Geriatr Psychiatry 2024; 32:373–385)

Highlights

- **What is the primary question addressed by this study?**

To identify community-specific relationships and sensitive periods that can facilitate targeted mitigation, we examined relationships between trauma during childhood and adulthood and cognitive outcomes among Black and White older adults.

- **What is the main finding of this study?**

Adulthood but not childhood trauma robustly associates with cognitive outcomes in both Black and White older adults, and relationships are frequently nonlinear.

- **What is the meaning of the finding?**

Clinical and/or community intervention on harmful sequelae of past and incident traumas may improve cognitive health in older populations.

OBJECTIVE

As the U.S. population ages, incidence and prevalence of Alzheimer's disease and related dementias (ADRD) increase; parallel increases in sub-clinical cognitive impairment correlate with additional adverse outcomes for older adults, including falls, loss of independence, and clinically presenting ADRD.¹ Fortunately, 30%–50% of global ADRD burden is modifiable;^{2,3} the physical, mental, and fiscal costs for patients, families, and communities highlight the need to identify malleable determinants of cognitive health. Growing evidence suggests exposure to stressors and stress-related sequelae represents one such modifiable source of risk,^{4–7} operating via environmental, behavioral, psychosocial, and biological pathways.⁸

Traumatic events are distinguished from other stressors by their severity. Like many ADRD determinants,² trauma-related risk processes may operate within sensitive periods of influence, when targeted risk reduction and access to clinical and community

supports will most substantially improve later-life cognitive outcomes. Substantial theoretical framing and empirical evidence establishes childhood as a sensitive period for long-term health detriment related to traumatic experiences, via persisting changes to physiological stress responses, and lasting impacts on social and economic trajectories.^{8,9} However, relatively few studies examine later-life cognitive outcomes in this context.¹⁰

An even smaller body of work focuses on cognitive impacts of trauma during adulthood, despite evidence suggesting stress operates on health throughout life. Adulthood objective and appraised stress associates with volumetric reductions in brain regions key for memory and executive function;¹¹ in experimental settings, introduction of a stressor temporarily reduces abilities in these domains.¹² Traumatic life events, even common experiences such as illness or death of a close family member, may also precipitate harmful ADRD risk factors for aging adults: poor management of vascular diseases, social isolation, depression, poor sleep, and even post-traumatic stress disorder.^{2,13–15}

There is particularly critical need to identify and mitigate salient cognitive risk factors in minoritized

populations including Black Americans, whose risk for ADRD and cognitive impairment is nearly twice as high as White peers¹⁶ and even more likely to be attributable to modifiable factors.³ Prevalence of modifiable excess risk results from structural, institutional, and interpersonal racism that perpetuates inequalities in neighborhood environments, educational and occupational opportunities, legal system contact, and healthcare access.^{17–19} Adverse social conditions expose the people living in those conditions to stressful events, and constrain the resources needed to cope with such events.^{9,20,21} Recent work suggests that stress exposures partially explain racial disparities in cognitive aging and may be a key determinant of cognitive health in older Black adults.^{4,5,22}

Recognizing the need to identify population-salient ADRD risk factors, and sensitive periods with distinct translational implications, the authors examined associations of childhood and adulthood trauma exposure with later-life cognitive test performance within Black and White samples of older adults. The authors used a within-group approach to account for experiences and exposures created through racialization across the life course.

METHODS

Population and Setting

Data are from the Health and Retirement Study (HRS), a nationally representative panel study of the population in the United States over age 50. Each full wave is completed across 2 yearlong periods. Data are collected in home at the enrollment baseline interview and by telephone at each biennial follow-up wave. A self-administered questionnaire, including lifetime trauma assessment, is left with respondents at the end of baseline interviews. Data from waves 2006/2008 through 2014/2016 are included in analyses. Participants were included in a cross-sectional analysis of baseline cognitive test performance if they were aged greater than or equal to 55 years and had complete life trauma and cognitive data available from at least one wave (see Figure, Supplementary Digital Content 1, for CONSORT diagram). Participants were included in a longitudinal analysis if they had two or more waves of cognitive data available.

The HRS (NIA U01AG009740) is conducted by the University of Michigan.

Measures

Childhood traumatic events

Data on childhood traumatic events (CT) were drawn from responses to 11 items regarding lifetime trauma on the leave-behind questionnaire.²³ For participants who completed the questionnaire at two or more timepoints, first timepoint with complete trauma data was used. Four items asked specifically about childhood (e.g., Before you were 18 years old, were you ever physically abused by either of your parents?). Participants were also asked whether seven additional events occurred at any age (e.g., Were you the victim of a serious physical attack or assault in your life?) and, if yes, the most recent year that the event occurred. Age at event was calculated as the difference between birth year and year of event. A CT index score was computed based on number of “Yes” responses to childhood-specific items together with the number of “Yes” responses to any-age items reported as occurring before age 18 (potential CT index range: 0–11).

Adulthood traumatic events

Data on adulthood traumatic events (AT) were drawn from the seven any-age items described above. An AT index score was computed as total number of items with “Yes” response with age of event being 18 or older, (potential AT index range: 0–7).

Cognitive outcomes

Cognitive outcomes were assessed with the 27-item adapted Telephone Interview on Cognitive Status (TICS). The TICS provides a global cognition score as well as subscores for verbal learning and memory. For each measure, lower scores indicate poorer performance.

Self-identified race and covariates

HRS participants are asked to specify their race and Hispanic/Latino ethnicity (“Do you consider yourself Hispanic or Latino”) at their baseline

interview. The current study included those participants who identified as White/Caucasian or as Black/African American and excluded those who identified as Hispanic/Latino. Covariates included basic demographic characteristics, chosen a priori based on empirical correlation with both cognitive test scores and with self-reported experiences of trauma: age at interview, gender, and educational attainment. Age was calculated for each interview based on date of birth and analyzed as a continuous variable. Gender was self-reported as "male" or "female" and analyzed as a binary variable. Educational attainment was categorized as "under high school" (<12 years), "high school" (equal to 12 years), "some college" (greater than 12 years; less than 16 years), and "college grad" (≥ 16 years).

Analyses

In order to assess within-group relationships of childhood and adulthood trauma with cognitive outcomes, the analytic sample was stratified by self-identified race into two samples, non-Hispanic Black/African American (referred to from this point forward as Black) and non-Hispanic White (referred to from this point forward as White). Characteristics of Black and White participants were compared using χ^2 and Mann-Whitney U tests. In preliminary analyses, a priori modeling of trauma-cognition relationships revealed both linear and parabolic forms to be relevant. Accordingly, performance on each of three key cognitive scores (global cognition, immediate recall, and delayed recall) was examined with respect to two trauma terms: i) main effects for CT and AT index scores, and ii) a quadratic of the relevant trauma index score. Continuous trauma index scores were centered prior to squaring and analysis.

Two modeling setups were used. First, cross-sectional relationships between main and quadratic trauma effects and baseline cognitive performance were examined using linear regression, adjusting for baseline age, gender, and educational attainment. Second, to examine relationships between traumatic event indices and cognitive trajectory across time, a two-stage process was used. In the first stage, the authors calculated each participant's cognitive slope using linear regression with longitudinal cognition as the outcome, and longitudinal age as the only covariate. These calculated individual slopes were then

used as the outcome in models otherwise similar to the cross-sectional analyses, though age is not included given it was used / accounted for in the first stage.

"Omnibus" trauma and cognition

Wald tests assessed significance of main and quadratic pieces of the form separately. To assess if trauma indices, overall, associated with each outcome, likelihood ratio tests (LRT) were used to compare models with the linear and quadratic effects of CT and AT ("omnibus" trauma) to models without either.

Statistical significance was assessed at the 5% level throughout. For each inferential test type (main CT and AT effects, quadratic effects, omnibus tests), Benjamini-Hochberg corrections were performed to mitigate false discovery rate (FDR) at 5% across the 12 models. All analyses and graphics were conducted using R version 4.0.0 and utilized the *lme4*, *boot*, *tableone*, and *ggplot2* packages. Model diagnostics for residual trends, heteroscedasticity, outliers, residual distribution, random effects distribution, and residuals versus random effects trends revealed no issues.

RESULTS

Sample Characteristics

A total of 2,345 Black and 11,607 White participants were included in baseline cross-sectional analyses. A total of 1,510 Black and 8,434 White participants were included in cognitive slope analyses. Participant characteristics for the whole sample and the slope subgroup are presented in [Table 1](#). Black participants were slightly younger than White participants and more likely be female. Relative to White participants, a greater proportion of Black participants reported completing fewer than 12 years of education, and a smaller proportion reported completing at least 16 years. Average numbers of endorsed traumatic life events in childhood and in adulthood were low in both groups. White participants reported marginally more AT events than did Black participants but otherwise surveyed trauma exposure did not vary by race. While CT and AT events were only analyzed as cumulative scores in

TABLE 1. Participant Characteristics of Black and White Analytic Samples

Characteristic	Cross-Sectional Baseline Cognitive Performance Analyses			Cognitive Slopes Analyses		
	Black/African American	White	p	Black/African American	White	p
N	2345	11607		1510	8434	
Age, years (mean (SD))	64.73 (8.59)	68.07 (9.61)	<0.001	64.48 (7.55)	67.58 (8.64)	<0.001
Male (N (%))	834 (35.6)	4979 (42.9)	<0.001	505 (33.4)	3551 (42.1)	<0.001
Education (N (%))			<0.001			<0.001
<High School	679 (29.0)	1603 (13.8)		410 (27.2)	1036 (12.3)	
High School	711 (30.3)	4154 (35.8)		473 (31.3)	3059 (36.3)	
Some college	581 (24.8)	2758 (23.8)		380 (25.2)	2000 (23.7)	
College grad	374 (15.9)	3092 (26.6)		247 (16.4)	2339 (27.7)	
Childhood trauma index (mean (SD))	0.50 (0.80)	0.51 (0.78)	0.347	0.49 (0.78)	0.50 (0.77)	0.275
Adulthood trauma index (mean (SD))	1.03 (1.15)	1.05 (1.10)	0.046	1.03 (1.15)	1.05 (1.09)	0.139
Global cognition (mean (SD))	13.29 (4.36)	16.06 (3.97)	<0.001	13.73 (4.06)	16.48 (3.66)	<0.001
Immediate recall (mean (SD))	5.12 (1.53)	5.65 (1.56)	<0.001	5.24 (1.44)	5.80 (1.46)	<0.001
Delayed recall (mean (SD))	3.73 (1.86)	4.60 (1.88)	<0.001	3.90 (1.76)	4.78 (1.77)	<0.001

Statistical tests between racial groups' characteristics was conducted using Mann-Whitney U tests for continuous items, and χ^2 tests of association for differences in gender proportion (df = 1) and education level (df = 3).

line with accumulation-of-risk models,²⁴ the frequencies of endorsement for each individual event are provided (see Figure, Supplementary Digital Content 2, for sample proportions endorsing traumatic events in childhood and adulthood).

Trauma and Cognition

Relationships of main and quadratic effects for CT and AT indices with baseline cognitive performance in three domains are presented in Table 2. Omnibus effects, described here, are fully presented in supplementary materials (see Table, Supplemental Digital Content 3, for detailed model characteristics and results). Relationships between number of childhood and adulthood trauma counts and predicted level of performance for global cognition and memory subscores, by race, are shown in Figure 1. In the Black sample there were no statistically significant associations between CT index and baseline TICS performance in any domain. However, AT index associated with baseline level of performance in global cognition, $F(2, 2,345) = 8.37$, p less than or equal to 0.001 and on the delayed recall subscale, $F(2, 2,345) = 3.80$, $p = 0.02$. A comparison of trauma and age coefficients as they predict global cognition (see Table, Supplementary Digital Content 3, for covariate estimates) are helpful to illustrate clinical significance of effects and occasional nonlinearity of trauma-cognition relationships. In the Black sample, the addition of one traumatic event from a starting point of zero exposure is "protective" for global cognition, the cognitive

equivalent of 4.02 fewer years of chronological age. However, the addition of one event from a starting point of four events is equivalent to 4.02 additional years of chronological aging. For White participants, CT index associated with level of performance in global cognition, $F(2, 10,899) = 6.55$, $p = 0.002$ in White participants, but not memory subscores. Comparing trauma and age coefficients as they predict global cognition, an addition of one childhood event from zero exposure is equivalent to 1.11 additional years of chronological age while the addition of one event from a starting point of four events is equivalent to 2.04 additional years of chronological age. AT index associated with level of performance in global cognition, $F(2, 11,607) = 9.01$, p less than 0.001; immediate recall, $F(2, 11,607) = 12.00$, p less than 0.001; and delayed recall, $F(2, 11,607) = 6.25$, $p = 0.002$. An addition of one adulthood event from zero exposure is equivalent to 1.48 fewer years of chronological age while the addition of one event from a starting point of four events is equivalent to 0.95 additional years of chronological age.

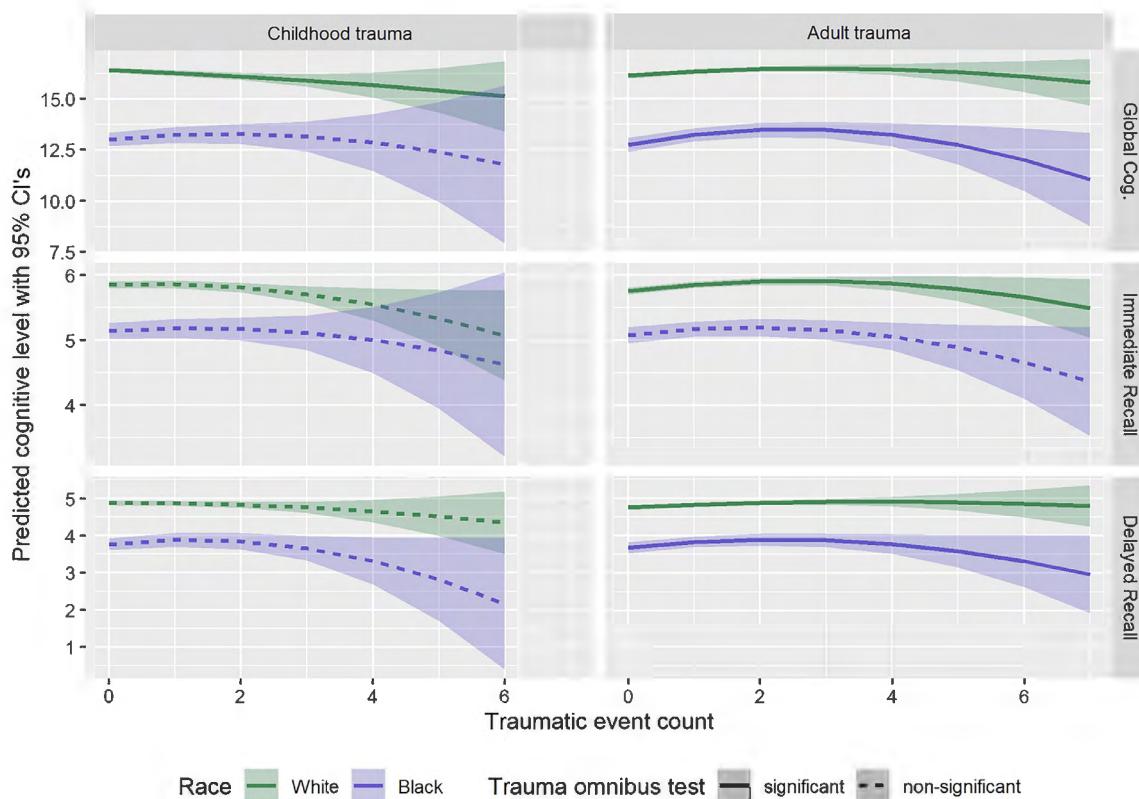
Relationships of main and quadratic effects for CT and AT indices with slope of cognitive test performance across time in three domains are presented in Table 3. Omnibus effects, described here, are also fully presented in supplementary materials (see Table, Supplemental Digital Content 3, for detailed model characteristics and results). There were no significant associations between CT or AT and cognitive slope for Black participants in any cognitive domain. Among White participants, CT index associated with

TABLE 2. Beta Coefficients and Confidence Intervals for Trauma-Baseline Cognition Regression Analyses

Race	Trauma Life Period	Model Outcome	Stress Coefficient Component	N	Coefficient Estimate	df	t Value	Estimate's 95% CI	p Value	Adj. p Value
Black	Childhood	Global cognition	Main effect	2008	0.208	2000	1.268	-0.11 to 0.53	0.2050	0.2734
			Quadratic effect	2008	-0.083	2000	-0.958	-0.25 to 0.09	0.3380	0.4506
		Immediate recall	Main effect	2008	0.043	2000	0.709	-0.08 to 0.16	0.4786	0.5743
			Quadratic effect	2008	-0.026	2000	-0.809	-0.09 to 0.04	0.4188	0.4569
		Delayed recall	Main effect	2008	0.120	2000	1.602	-0.03 to 0.27	0.1093	0.1639
			Quadratic effect	2008	-0.077	2000	-1.961	-0.15 to -0.01	0.0500	0.0999
	Adult	Global cognition	Main effect	2345	0.356	2337	4.070	0.18–0.53	<0.0001	0.0002
			Quadratic effect	2345	-0.121	2337	-2.948	-0.2 to -0.04	0.0032	0.0302
		Immediate recall	Main effect	2345	0.056	2337	1.724	-0.01 to 0.12	0.0849	0.1455
			Quadratic effect	2345	-0.032	2337	-2.107	-0.06 to -0.01	0.0352	0.0955
		Delayed recall	Main effect	2345	0.106	2337	2.668	0.03 to 0.18	0.0077	0.0185
			Quadratic effect	2345	-0.042	2337	-2.254	-0.08 to -0.01	0.0243	0.0955
White	Childhood	Global cognition	Main effect	10044	-0.140	10036	-2.115	-0.27 to -0.01	0.0344	0.0689
			Quadratic effect	10044	-0.015	10036	-0.390	-0.09 to 0.06	0.6962	0.6962
		Immediate recall	Main effect	10044	0.007	10036	0.257	-0.05 to 0.06	0.7971	0.8502
			Quadratic effect	10044	-0.027	10036	-1.813	-0.05 to 0.02	0.0698	0.1197
		Delayed recall	Main effect	10044	-0.006	10036	-0.189	-0.07 to 0.06	0.8502	0.8502
			Quadratic effect	10044	-0.016	10036	-0.886	-0.06 to 0.01	0.3755	0.4506
	Adult	Global cognition	Main effect	11607	0.156	11599	4.189	0.08 to 0.23	<0.0001	0.0002
			Quadratic effect	11607	-0.041	11599	-2.056	-0.08 to -0.01	0.0398	0.0955
		Immediate recall	Main effect	11607	0.073	11599	4.889	0.04 to 0.10	<0.0001	<0.0001
			Quadratic effect	11607	-0.022	11599	-2.805	-0.04 to -0.01	0.0050	0.0302
		Delayed recall	Main effect	11607	0.060	11599	3.333	0.03 to 0.10	0.0009	0.0026
			Quadratic effect	11607	-0.011	11599	-1.143	-0.03 to 0.01	0.2532	0.3798

All models additionally adjust for baseline age, gender, and educational attainment.

Adjusted p values were conducted using the Benjamini-Hochberg procedure across the trauma coefficient main effects and quadratic effects separately (12 items in each adjustment).
Bold indicates $p < 0.05$.

FIGURE 1. Baseline cognitive performance as predicted by trauma index scores.

Shaded regions represent the 95% confidence interval for the estimated relationship between trauma and cognitive test score. Predictions and CI's are calculated for age being set to the median age in the data (66 years old), and using the modes for gender (Female) and educational attainment (High school). Graphic panel columns represent the two different trauma measures (Childhood and Adulthood), and the graphic panel rows represent the three different cognitive test outcomes (Global Cognition, Immediate Recall, and Delayed Recall).

scores on global cognition, $F(2, 7,840) = 5.05, p = 0.006$ and immediate recall, $F(2, 7,840) = 5.07, p = 0.006$. In these same White participants, AT index associated with change in those same domains of global cognition, $F(2, 8,427) = 5.28, p = 0.005$ and immediate recall, $F(2, 8,427) = 6.62, p$ less than 0.01 (Fig. 2).

CONCLUSION

In this nationally representative sample of older adults, the authors examined relationships between experiences of childhood and adulthood traumatic events and cognitive outcomes in later life. The

authors conducted within-group analyses by race in accordance with disparities frameworks^{9,18} and evidence that measured and unmeasured differences in the lived experiences of Black and White older adults have implications for exposure distributions and magnitudes of effect.²⁵ To our knowledge, this is the first exploration of population-specific relationships of distinct sensitive periods for trauma exposure with cognitive health.

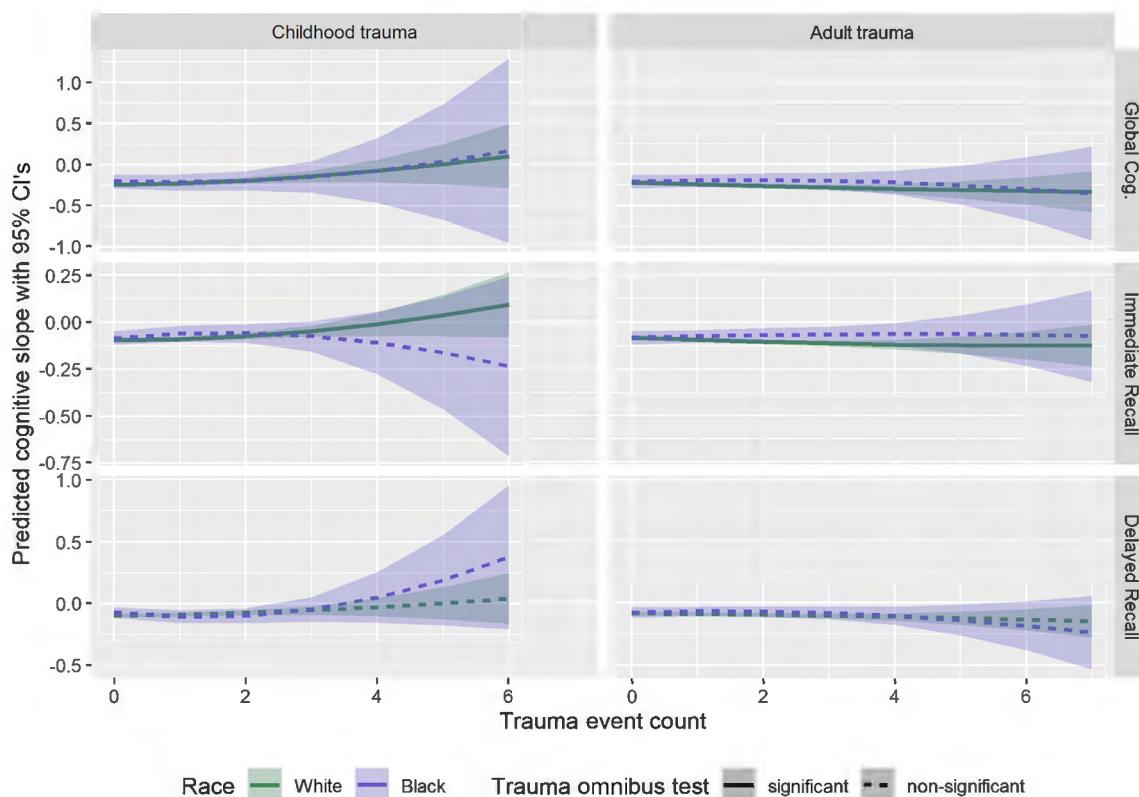
Our finding that Black and White participants reported similar exposure to traumatic events during childhood, with White adults reporting marginally greater exposure during adulthood, was somewhat surprising within a larger body of work

TABLE 3. Beta Coefficients and Confidence Intervals for Trauma-Cognitive Slope Regression Analyses

Race	Trauma Life Period	Model Outcome	Stress Coefficient Component	N	Coefficient Estimate	df	t Value	Estimate's 95% CI	p Value	Adj. p Value
Black	Childhood	Global cognition	Main effect	1372	-0.011	1365	-0.261	-0.09 to 0.07	0.7942	0.7942
			Quadratic effect	1372	0.015	1365	0.592	-0.03 to 0.06	0.5536	0.7583
		Immediate recall	Main effect	1372	0.022	1365	1.206	-0.01 to 0.06	0.2282	0.4564
			Quadratic effect	1372	-0.009	1365	-0.891	-0.03 to 0.01	0.3729	0.7583
		Delayed recall	Main effect	1372	-0.037	1365	-1.693	-0.08 to 0.01	0.0907	0.3628
			Quadratic effect	1372	0.022	1365	1.735	-0.01 to 0.05	0.0829	0.7583
	Adult	Global cognition	Main effect	1510	0.009	1503	0.414	-0.03 to 0.05	0.6788	0.7714
			Quadratic effect	1510	-0.006	1503	-0.570	-0.02 to 0.01	0.5687	0.7583
		Immediate recall	Main effect	1510	0.007	1503	0.773	-0.01 to 0.03	0.4398	0.5864
			Quadratic effect	1510	-0.001	1503	-0.265	-0.01 to 0.01	0.7909	0.8057
		Delayed recall	Main effect	1510	0.004	1503	0.376	-0.02 to 0.03	0.7071	0.7714
			Quadratic effect	1510	-0.005	1503	-1.029	-0.01 to 0.01	0.3039	0.7583
White	Childhood	Global cognition	Main effect	7847	0.019	7840	1.321	-0.01 to 0.05	0.1866	0.4564
			Quadratic effect	7847	0.008	7840	0.948	-0.01 to 0.02	0.3431	0.7583
		Immediate recall	Main effect	7847	0.006	7840	0.871	-0.01 to 0.02	0.3837	0.5756
			Quadratic effect	7847	0.005	7840	1.397	-0.01 to 0.01	0.1626	0.7583
		Delayed recall	Main effect	7847	0.008	7840	1.066	-0.01 to 0.02	0.2863	0.4908
			Quadratic effect	7847	0.003	7840	0.676	-0.01 to 0.01	0.4990	0.7583
	Adult	Global cognition	Main effect	8434	-0.022	8427	-2.703	-0.04 to -0.01	0.0069	0.0413
			Quadratic effect	8434	0.001	8427	0.246	-0.01 to 0.01	0.8057	0.8057
		Immediate recall	Main effect	8434	-0.011	8427	-3.185	-0.02 to -0.01	0.0015	0.0174
			Quadratic effect	8434	0.001	8427	0.576	-0.01 to 0.01	0.5645	0.7583
		Delayed recall	Main effect	8434	-0.005	8427	-1.262	-0.01 to 0.01	0.2068	0.4564
			Quadratic effect	8434	-0.001	8427	-0.332	-0.01 to 0.01	0.7402	0.8057

All models additionally adjust for gender and educational attainment.

Adjusted p values were conducted using the Benjamini-Hochberg procedure across the trauma coefficient main effects and quadratic effects separately (12 items in each adjustment).
Bold indicates $p < 0.05$.

FIGURE 2. Change in cognitive performance as predicted by trauma index scores.

Shaded regions represent the 95% confidence interval for the estimated relationship between trauma and cognitive test score. Predictions and CI's are calculated for age being set to the median age in the data (66 years old), and using the modes for gender (Female) and educational attainment (High school). Graphic panel columns represent the two different trauma measures (Childhood and Adulthood), and the graphic panel rows represent the three different cognitive test outcomes (Global Cognition, Immediate Recall, and Delayed Recall).

on racialized stress exposures. However, results were consistent with prior findings on traumatic event indices specifically, highlighting the importance of delineating distinct stress constructs. Several studies have reported on racial disparities in chronic stress and discrimination exposures among HRS participants,^{20,26,27} but in a sample of Black and White men drawn from the first cohort to complete the leave-behind questionnaires, those disparities did not extend to the surveyed childhood and adulthood traumatic events.²⁷ These findings are echoed in a latent cluster analysis of Adverse Childhood Experiences (ACEs) in another large cohort; among non-Hispanic Black adolescents, ACEs representing

socioeconomic adversity, not trauma, drove disproportionate overall prevalence of ACEs.²⁸

Childhood trauma significantly associated with cognition only in White participants, with relationships differing qualitatively for baseline cognition and for cognitive change. A primarily linear negative association with baseline performance on global cognition contrasts with an apparent protective effect on trajectory. Negative associations with baseline cognition echo prior work with other outcomes in the full, predominantly White HRS cohort. The few studies exploring surveyed childhood traumatic events as a predictor of health have reported associations between childhood trauma items and physiological

detriment including telomere attrition²⁹ and all-site cancer prevalence.³⁰ Slowed decline is not consistent with theories of accelerated aging or weathering under adverse conditions and in general, causal protective relationships between high exposure to childhood trauma and preserved late-life cognition are not plausible. Inconsistency could be explained by atypical resilience prevalent in a sample of older adults able to select into a longitudinal study. Stable TICS score over time may also reflect baseline scores that underestimate cognitive function in persons who have experienced social disadvantage, a systematic test bias common to cognitive screeners. Within the Black/African American sample, the type of traumatic experiences assessed (or absent) may again explain our wide confidence intervals and subsequently null findings for childhood trauma. Zhang et al. focused on an index of childhood socioeconomic disadvantage, and found that it associated with 12-year dementia risk in the full HRS sample and partially accounted for racial disparities therein.³¹ Meanwhile, another HRS study using a broad index of childhood stressors to predict functional limitations, inclusive of the trauma items in the current study, found, as the authors did, that associations observed in White participants were absent in Black participants.³² In other cohorts, absence of relationships or even positive relationships have been observed.^{33,34} A systematic review attributed inconsistencies in findings on childhood adversity as a determinant of later-life health to issues of internal validity, and potential for effect modification by other demographic characteristics such as age and gender.¹⁰ Notably, within the Black sample for this study, our wide confidence intervals and subsequent null findings for childhood trauma could be due to several factors including the relatively smaller sample of Black participants, the few participants reporting high numbers of traumatic events in this group, and the type of traumatic experiences assessed or absent.

That significant cognitive detriment was observed for both Black and White participants at higher exposures to adulthood traumatic events, with substantial quadratic effects driving particularly strong negative relationships in the Black sample, is not surprising. One prior study of lifetime accumulated stressors and cognitive aging in a much smaller and volunteer-based cohort described associations of adversity with cognition across both Black and White middle-aged

and older adults—but relationships were both greater in magnitude and expanded to more cognitive domains in Black participants, compared to White peers.⁵ Similar results have also been recently observed in much larger, population-based cohorts.^{22,35} The aforementioned HRS-based study on traumatic life events and men's functional health echo the current results as well; trauma exposure associates with functional health across racial groups but also partially accounts for racial disparities in functional limitations despite equivalent exposure, indicating greater magnitude of effect in older Black men.²⁷ The current study did not make between-race comparisons of the relationships between trauma and health, but prior findings on disparate magnitudes of effect can help guide mechanistic hypotheses and future research questions. Differential vulnerability to trauma in the absence of differential exposure is likely to be explained by the inequitable contexts in which traumatic events occur. Cumulative stress indices do not account for co-occurring race-based stressors such as everyday discrimination.²⁰ Additionally, if coping resources—emotional, instrumental, financial, physiological—are already strained by existing chronic adversity, then the impact of acute stressors is likely to extend and expand.²¹

In conjunction with a small but rigorous body of previous work, our findings also demonstrate the importance of testing for nonlinear health effects of social adversity. As noted, in both Black/African American and White samples, relationships between traumatic experiences in adulthood and cognitive test performance were modestly positive when traumatic events were present but rare. When these low-exposure groups dominate a sample, regression coefficients for linear terms are driven by that positive association, obscuring detriment occurring at high exposure. Nonlinear modeling of risk factors is uncommon in cognitive aging research. However, studies elsewhere have described these "U-shaped" relationships between cumulative adversity and health outcomes, wherein nonzero but low adversity scores predict better mental and physical function.^{36,37} A frequent hypothesis is that when rare, adverse experiences require adaptation that fosters personal resilience. Few studies report positive associations between adversity and cognitive outcomes, but protective effects of specific adverse childhood experiences, such as not having enough food to eat, being

underweight, and living with someone with mental illness, have been observed for the cognitive health of older ethnoracially diverse and Black cohorts.^{19,33} The authors note that the cumulative traumatic event exposures are right-tailed in our data, with most participants experiencing no or few events, and very few experiencing a large number of events. The relatively small (and thus more variable) number of high cumulative totals could ostensibly influence the nonlinear trends the authors observed. Additionally, if surveyed traumatic events represent a proxy for correlated, potentially unmeasured trauma and adversity, exacerbated associations at high cumulative totals would also be expected. The need for future studies of trauma and adversity, including nonlinear considerations, is clear.

Finally, our study is not the first to find that stress-related exposures associate primarily with a baseline level of cognitive health rather than with rate of decline across time. Our findings in this regard are similar to previous studies reporting that measures of adversity primarily associate with a reduction in level of cognitive health at an early point in the measured trajectory,³⁸ likely fomenting social disparities via a later-life “peak” closer to thresholds of impairment.

Limitations

Limitations in the described analysis draw attention to crucial future studies and research directions. First, our analyses did not include HRS participants who identified as Hispanic/Latino. Intersectionality between ethnicity and racialization requires additional within-group analyses that consider heterogeneity across culturally unique subgroups of broader Latino populations, as well as integration of factors such as nativity and immigration status. Additionally, as with many studies utilizing self-reported exposure data and cognition as an outcome, the potential for exposure misclassification is a concern, though the authors expect that underreporting should attenuate rather than inflate risk estimates in this study. Further, as discussed, selection and operationalization of distinct stress constructs inform results. In line with cumulative stress models, the authors focused on a count of life traumas within a relatively brief index. Trauma exposures were low; it is noteworthy that associations with cognitive health were detectable given this analytic constraint. Nonetheless there are

notable considerations for such models. All experiences are equally weighted, but though the nature of “traumatic events” relative to “stressful events” is in their seriousness and severity, there may still be age, period, cohort, or other axes of variation in both objective impact and in subjective appraisal. In Black populations in particular, event indices are likely to underestimate exposure²⁰ and to fail to account for racial trauma or the adaptation and coping strategies developed to thrive in a context of racism.³⁹ Complementary existing and future research guided by other life course models is needed. In a sweeping study of ethnoracial disparities in stress exposures and health outcomes, Sternthal, Slopen, and Williams describe unique implications for stress modeling, between and within populations.⁴⁰ For instance, lifetime cumulative models can help account for bias resulting correlations between stressors, including unmeasured stressors, that individuals are exposed to. In other cases, examining the population-specific importance of distinct events,¹⁴ or clusters or chains of events that tend to occur in conjunction with each other or sequentially^{28,32} can provide crucial information for screening and mitigation purposes, particularly within the clinical setting. Finally, as with all nonexperimental studies, residual confounding of trauma-cognition relationships may be present. In particular, early-life exposures such as childhood socioeconomic status, quality of education, and chronic stress potentially intersect in complex ways with the primary relationships of interest. These intersections are likely to vary with racialization.⁴⁰ Studies that model this complexity with intentionality can parse not only unique contributions but also mediation and moderation effects.

Implications for Clinical Research and Translation

Despite its limitations, this study adds to a growing body of literature suggesting accumulating life stressors can undermine successful aging in older populations including Black/African American communities. In these samples, at high levels of trauma exposure, one additional adulthood trauma is equivalent to between one (White participants) and four (Black participants) years of chronological aging. Black Americans have been historically underserved by healthcare institutions. Clinical, public health, and

health policy teams dedicated to equitably improving outcomes for older adults are uniquely situated to assess and mitigate health detriment associated with traumatic experiences given their close work with patients, families, and healthcare systems.

In terms of research implications, it is noteworthy that magnitude and even direction of effects for risk or protective factors in predominantly White samples cannot be assumed to hold for other populations; equity-focused gerontological science requires inclusive cohorts adequately powered for within-group analyses. Within and beyond race and ethnicity, this must include assessment of intersections between axes of both vulnerability and resilience. For instance, several studies have shown that trauma during early- and middle-adulthood associate with functional limitations in older age.^{23,32} If accommodations are absent, functional limitations and disability may compound health effects of accumulating stressors by constraining access to activities and resources that facilitate coping and recovery. Establishing population-specific physiological, psychological, behavioral, and environmental mediators and moderators will help to pinpoint salient intervention loci across stages of life and at all levels of function.

The role that clinical practices can play in mitigating health impacts of trauma is also increasingly clear. Traumatic experiences during childhood and adulthood have been robustly tied to disruption of brain-healthy behaviors, depressive symptoms, and symptoms of post-traumatic stress disorder in older adults. While studies of resilience—thriving under adverse conditions—often focus on individual-level traits, a more impactful approach from a clinical and public health perspective includes facilitating and sometimes forging connections between older adults and meaningful resources. Empirically, both clinical supports including medication and psychotherapy, and community assets including interpersonal relationships and access to religious services, have been found to help older adults recover following

traumatic psychosocial and physical stressors.^{15,41,42} While acute events themselves may not always be preventable, clinicians play an irreplaceable role in helping older patients and families to navigate coping with and recovering from those events, even—especially—in a context of more chronic adversity.

DISCLOSURES

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AUTHOR CONTRIBUTIONS

MZ was responsible for study concept and design, supervising analyses and interpreting data, and primary writing and preparation of manuscript. RWT contributed at all stages. AS provided data. DN performed data analyses. AS, LLB, DRB, DKR, RC, and DN contributed substantively to preparation of manuscript.

DATA STATEMENT

These data have not been previously presented orally or by poster at scientific meetings.

SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found in the online version at <https://doi.org/10.1016/j.jagp.2023.09.015>.

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Flortaucipir tau PET findings from former professional and college American football players in the DIAGNOSE CTE research project

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Abstract

INTRODUCTION: Tau is a key pathology in chronic traumatic encephalopathy (CTE). Here, we report our findings in tau positron emission tomography (PET) measurements from the DIAGNOSE CTE Research Project.

METHOD: We compare flortaucipir PET measures from 104 former professional players (PRO), 58 former college football players (COL), and 56 same-age men without exposure to repetitive head impacts (RHI) or traumatic brain injury (unexposed [UE]); characterize their associations with RHI exposure; and compare players who did or did not meet diagnostic criteria for traumatic encephalopathy syndrome (TES).

RESULTS: Significantly elevated flortaucipir uptake was observed in former football players (PRO+COL) in prespecified regions ($p < 0.05$). Association between regional flortaucipir uptake and estimated cumulative head impact exposure was only observed in the superior frontal region in former players over 60 years old. Flortaucipir PET was not able to differentiate TES groups.

DISCUSSION: Additional studies are needed to further understand tau pathology in CTE and other individuals with a history of RHI.

KEYWORDS

CTE, flortaucipir, football, PET, Tau

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1 | BACKGROUND

Chronic traumatic encephalopathy (CTE) is a neurodegenerative disease associated with exposure to repetitive head impacts (RHI), such as those experienced by contact/collision sport athletes and military combat veterans.^{1,2} CTE is defined neuropathologically by the presence of neurofibrillary tangles (NFTs) in neurons, around small vessels, and in an irregular pattern at the depth of the cortical sulci^{3,4} distinct from other tauopathy-related neurodegenerative diseases such as Alzheimer's disease (AD).^{3,5} The clinical presentation of individuals with neuropathologically-confirmed CTE, referred to as traumatic encephalopathy syndrome (TES),⁶ includes progressively worsening cognitive impairment (especially in episodic memory and executive functioning), neurobehavioral dysregulation (e.g., rage, short fuse, emotional lability), and in some instances, parkinsonism, and motor neuron disease.^{6–9} Currently, CTE can only be definitively diagnosed *post mortem* based on neuropathological assessment. The lack of well-validated *in vivo* biomarkers specific for CTE NFT hampers clinical detection and diagnosis during life.¹⁰ To address this challenge, the National Institute of Neurological Disorders and Stroke (NINDS) funded a multi-institutional and multidisciplinary study, referred to as the "Diagnostics, Imaging, and Genetics Network for the Objective Study and Evaluation of Chronic Traumatic Encephalopathy (DIAGNOSE CTE) Research Project" to develop methods to detect, characterize, and DIAGNOSE CTE by evaluating a cohort of former professional and college American football players and a comparison group of same-age men without exposure to contact/collision sports or history of RHI or traumatic brain injury (TBI).¹⁰

Since tau is the central pathology that defines CTE, *in vivo* biomarkers that can assess tau pathology are essential for its detection and characterization. Tau-specific Positron emission tomography (PET) has emerged as a promising technique to detect and quantify NFTs in AD and other tauopathies.^{11–13} The PET tracer [F18]-flortaucipir (FTP) was approved by the United States Food and Drug Administration (U.S. FDA) to measure NFTs in patients being evaluated for AD, a mixed 3-repeat-4-repeat (3R-4R) tauopathy. Although FTP has been found to have a high affinity for AD tau,^{14,15} its affinity for tau isoforms in other tauopathies is weaker, especially in 4R tauopathies, such as progressive supranuclear palsy (PSP) and corticobasal degeneration (CBD).^{16,17}

The detection of NFT in CTE has unique challenges. First, because tau deposition in earlier stages of CTE is patchy, with a focal sulcal depth distribution,^{4,18} the overall uptake across any specific region of interest (ROI) is expected to be modest at best. Second, although CTE is a mixed 3R-4R tauopathy like AD, there are changes to the ratio of 3R:4R tau isoforms across disease stages, between neuronal and glial tau,¹⁹ as well as across different regions.²⁰ Finally, growing research using cryo-electron microscopy has demonstrated that the molecular structure of tau filaments across different tauopathies are distinct,^{5,21,22} and the 3R tau isoform which FTP is more sensitive in detecting is more predominant at late-stage CTE. Despite these challenges, in a previous investigation, higher FTP uptake was

RESEARCH IN CONTEXT

1. **Systematic review:** Chronic traumatic encephalopathy (CTE) is a neurodegenerative disease associated with exposure to repetitive head impacts (RHIs) and defined neuropathologically by the presence of hyperphosphorylated neurofibrillary tangles. We previously reported in a moderate-sized cohort of former professional football players and controls that elevated tau can be detected by flortaucipir PET in the player group; however, another recent study failed to observe this. Here, in a larger cohort that also included former college players, we confirmed the modestly elevated flortaucipir uptake in former football players. The flortaucipir uptake was also associated with the estimated exposure to RHIs but only in participants 60 years and older. The flortaucipir PET was not able to differentiate clinical groups in former players.
2. **Interpretation:** Elevated flortaucipir uptake can be detected in patients with RHI exposure, although this requires a sufficient sample size. There is also a delay between RHI exposure and the accumulation of tau pathology, which contributed to the conflicting results in the literature.
3. **Future directions:** Future studies are needed to identify optimal CTE tau biomarkers, clarify the relationship between tau markers and different indicators of RHI in former football players and other groups, and the extent to which they predict subsequent clinical progression and *post mortem* CTE pathology.

observed in a group of 26 former National Football League (NFL) players (all with cognitive, mood, and behavioral symptoms; ages 40–69) compared to a control group of 31 same-age participants (all asymptomatic and without a history of TBI).²³ Association between FTP uptake and exposure to RHI as measured by the years of playing football was also observed. However, no association was found between FTP uptake and cognitive function or neuropsychiatric symptoms.²³

In this study, we examine further the ability of FTP PET to detect CTE tau pathology in former American football players and examine the relationships between FTP PET uptake and RHI exposure and clinical diagnosis. This study addresses previous limitations by including a much larger sample size, greater variability in RHI exposure (including in addition men who only played football up through college), greater variability in symptom severity in the former players (from asymptomatic to mild dementia), and an asymptomatic comparison group of same-age men without a history of playing contact/collision sports, other RHI exposure, concussion, or TBI.

TABLE 1 Summary of cohort characteristics

	PRO (N = 104)	COL (N = 58)	UE (N = 56)	p-Value
Age (years) Mean \pm SD (min, max)	58.7 \pm 7.9 (45,74)	53.1 \pm 7.3 (45,74)	59.5 \pm 8.4 (45,74)	<0.0001
Race (n [%] Black/African-American)	45 (43.2%)	11 (19.0%)	21 (37.5%)	0.008
APOE-e4 carriers (n [%])	30 (30%)	20 (35%)	10 (19%)	0.16
Education (years) Mean \pm SD (min, max)	16.6 \pm 1.2 (15,21)	16.9 \pm 1.4 (15,22)	17.5 \pm 3.4 (13,30)	0.04
MoCA total score Mean \pm SD (min, max)	24.4 \pm 3.5 (12,30)	25.2 \pm 3.2 (11,30)	26.6 \pm 2.3 (17,30)	<0.001

Note: One-way ANOVA was performed to examine continuous cohort variables for exposure group level differences, and χ^2 test was used to determine group difference for categorical variables, including percentage of Black or African American participants.

Abbreviations: APO-E, apolipoprotein E; COL, former college football players; MoCA, Montreal Cognitive Assessment; PRO, former professional football players; SD, standard deviation; UE, participants with no football exposure.

2 | METHODS

2.1 | Participants

The design of the DIAGNOSE CTE Research Project has been previously described.¹⁰ Briefly, the overall study enrolled 240 participants, ages 45–74, including 120 former professional football players (PRO) and 60 former college football players (COL) regardless of their cognitive and clinical status, and 60 control participants without RHI exposure, TBI, or combat military history and who denied symptoms at telephone screening (unexposed, UE). Specific inclusion and exclusion criteria are reported elsewhere.¹⁰ Participants were evaluated at one of four Participant Evaluation Sites: (1) Boston (Boston University Chobanian & Avedisian School of Medicine, with MRI scans conducted at Brigham and Women's Hospital); (2) Las Vegas (Cleveland Clinic Lou Ruvo Center for Brain Health); (3) New York (New York University Langone Health); and (4) Scottsdale/Phoenix (Mayo Clinic Arizona, with PET scans conducted at Banner Alzheimer's Institute in Phoenix, AZ). The study was approved by the Institutional Review Boards at all sites, and written informed consent was obtained for all participants. Eight participants who were part of our previous study²³ were excluded from this analysis to avoid circularity as the primary regions we examined as described below were defined statistically using data including these participants. Participants without FTP PET data were also excluded, resulting in a total of 218 participants (104 PRO, 58 COL, and 56 UE) included in this analysis (Table 1).

2.2 | Brain imaging

T1 MRIs were acquired on 3T Siemens Skyra scanners across the four study sites using MPRAGE sequence with 1 mm³ isotropic resolution.¹⁰ FTP PET data were acquired following a bolus injection of approximately 259 MBq (7 mCi) of the PET tracer on a PET/CT scanner (GE Discovery 710 or Siemens mCT) at one of the four participating sites in dynamic mode with 5-min frames. Most FTP scans were

acquired with an 80–100 min post-injection window while three participants only had data up to 90 min after injection. All PET data were reconstructed with CT-based attenuation correction and standard random and scatter corrections. The use of flortaucipir in this study was carried out through an Investigational New Drug (IND #131,391) from the U.S. FDA. FTP doses were requested through and provided at no cost by Avid Radiopharmaceuticals (Philadelphia, PA, USA). Quality control and imaging calibration procedures were completed prior to study initiation by Invicro (Needham, MA, USA) to certify the scanners used in this study at each site.

The T1-weighted MRI data were analyzed using FreeSurfer v6 (Martinos Center for Biomedical Imaging, Charlestown, Massachusetts, USA) to define anatomical regions of interest (ROIs) by the Brigham and Women's Hospital team. FTP PET analysis was conducted at Banner Alzheimer's Institute using an in-house pipeline.^{24,25} The analysis included scanner harmonization filtering to reach a common 8-mm resolution,²⁶ between frame motion correction, target frame summation, PET-to-MR coregistration, and regional standardized uptake value ratio (SUVR) extraction based on the FreeSurfer generated anatomical ROIs with bilateral inferior cerebellar cortex as the reference region.²⁷ Fully bias field corrected and intensity normalized T1-MRI from the FreeSurfer pipeline (T1.mgz) was also spatially normalized using the Statistical Parametric Mapping (SPM) to generate the individual-to-template space nonlinear transformation and transform PET data into the MNI template space. The FTP PET data in template space were also renormalized using cerebellum crus one region (5128 voxels) as the reference for prespecified regional analysis to be consistent with our prior work.²³ Three prespecified statistical ROIs, bilateral superior frontal region (2887 voxels), bilateral medial temporal region (1283 voxels), and left parietal region (252 voxels)²³ (referred to as prespecified statistical ROIs henceforth), were included as the primary ROIs. These prespecified statistical ROIs were voxel clusters defined in our previous study of 26 formal professional football players and 31 control subjects where former players had statistically significantly higher FTP SUVR than controls²³ (Figure S1). For 13 participants missing MR data, the summed FTP PET data were transformed to the

template space using a separate PET only SPM pipeline with a pre-established FTP template. For these participants, only the prespecified statistical ROI measures were extracted.

2.3 | Estimated cumulative RHI exposure and TES diagnostic grouping

In this study, the cumulative head impact index based on measurements of linear acceleration (CHII-G) was used to estimate the lifetime cumulative g-force that participants experienced due to football RHI.^{28,29} CHII-G is a composite score based on a combination of self-reported measures of exposure,²⁸ projected onto data aggregated into a positional exposure matrix (PEM) based on published helmet accelerometer studies, stratified by position and level of play.²⁹ All participants were diagnosed through multidisciplinary diagnostic consensus conferences using the NINDS Consensus Diagnostic Criteria for TES (including the Provisional Levels of Certainty for CTE Pathology).^{6,10} The consensus conference panelists included 16 clinician-researchers (all DIAGNOSE CTE Research Project investigators) representing multiple disciplines (neurology, neuropsychology, psychiatry, neurosurgery), from seven institutions. Panelists were presented with the participant's medical history (including neurologic and psychiatric); football history (and other RHI exposure); self- and informant-reported complaints of cognitive, mood, and/or behavior problems, as well as functional dependence status; neurological/motor evaluation findings; and standardized neuropsychological and neuropsychiatric test results (the specific tests and assessment methods have been described previously¹⁰). Based on this information, the panelists used the TES criteria and adjudicated the following diagnostic categories pertinent to the current study: (1) No TES, (2) TES with suggestive level of certainty for CTE pathology, and (3) TES with possible or probable level of certainty for CTE pathology. A diagnosis of TES⁶ requires (1) substantial RHI exposure from contact sports, military service, or other causes; (2) core clinical features of cognitive impairment (in episodic memory and/or executive functioning, substantiated by performance on formal neuropsychological testing, > 1.5 SDs below norms) and/or neurobehavioral dysregulation (e.g., rage, emotional dyscontrol, short fuse); (3) progressive course; and (4) that the core clinical features are not fully accounted for other neurologic, psychiatric, or medical disorders. The Provisional Levels of Certainty for CTE Pathology are determined based on specific RHI exposure thresholds, core clinical features (e.g., cognitive impairment is required for possible or probable levels of certainty), functional status, and additional supportive features, including delayed symptom onset after retirement from football, motor signs, and psychiatric features.⁶

2.4 | Statistical analysis

One-way analysis of variance (ANOVA) was performed to examine continuous cohort variables for exposure group-level differences, and

the χ^2 test was used to determine group differences for categorical variables, including the percentage of Black or African American participants.

Exposure group-level differences in FTP PET-measured tau pathology were examined in both regional and voxel-wise analysis. The primary regional analysis focused on the three prespecified statistical ROIs to replicate our previous findings and examine the relationship between FTP uptake and exposure to football. Exploratory analysis was also performed for a preselected subset of eight anatomical ROIs (to limit the impact of type I error due to multiple comparisons) that are known to be susceptible to tau pathology in aging, Alzheimer's disease, and/or CTE, including entorhinal cortex, parahippocampal gyrus, superior frontal cortex, superior parietal cortex, hippocampus, amygdala, inferior parietal cortex, and inferior temporal cortex. Group-level comparison of regional FTP SUVR adjusted for age and race was performed using analysis of covariance (ANCOVA) followed by post hoc pair-wise comparisons in the framework of ANCOVA.

For the voxel-wise analysis to examine the spatial extent of the FTP measured tau burden group differences, voxel-wise general linear model analysis on the spatially normalized FTP SUVR images was performed between the groups. The primary comparison was made between all former players (PRO+COL) and UE followed by additional comparisons for PRO versus UE and COL versus UE. To avoid potential biases caused by analysis variation, only those FTP scans with data over the full 80- to 100-min post-injection window that had successful FreeSurfer runs were included in the voxel-wise analysis. A Monte-Carlo simulation approach introduced previously²³ was used to assess the omnibus significance for the voxel-wise SUVR differences between groups. Referred to as the majority-count statistics (MCS) in this report, this statistical significance examination is a way to assess the significance free from the multiple comparisons concern. MCS capitalizes on the argument that the likelihood of observing wide-spread tau load increase in one contrast direction (e.g., higher SUVR in PRO than in UE) versus minimal load in the opposite direction (e.g., lower SUVR in PRO than in UE) should be very low if no group difference exists. In other words, the number of voxels where one group has higher SUVR than the other group should be statistically the same for the opposite direction if no group SUVR difference exists. A detailed description of MCS can be found in our previous study.²³ Additionally, we applied family-wise error (FWE) corrections to adjust for the voxel-wise multiple comparisons with localization power.

To investigate whether tau burden is associated with exposure to RHI, linear regression analysis was performed within the PRO+COL group with CHII-G as the response variable, regional FTP SUVRs for the three prespecified statistically defined ROIs as the independent variable, and age and race as covariates. To examine whether FTP PET can differentiate the two TES diagnostic groups described above in former football players, ANCOVAs were performed between the two groups for FTP SUVR in each of the three prespecified ROIs again with age and race as covariates. Exploratory analyses were also performed in the subset of former football players (PRO+COL) older than 60 ($N = 54$, 45 PRO, 9 COL) to examine the tau association with RHI exposure and its ability to differentiate clinical groups, as in our recent

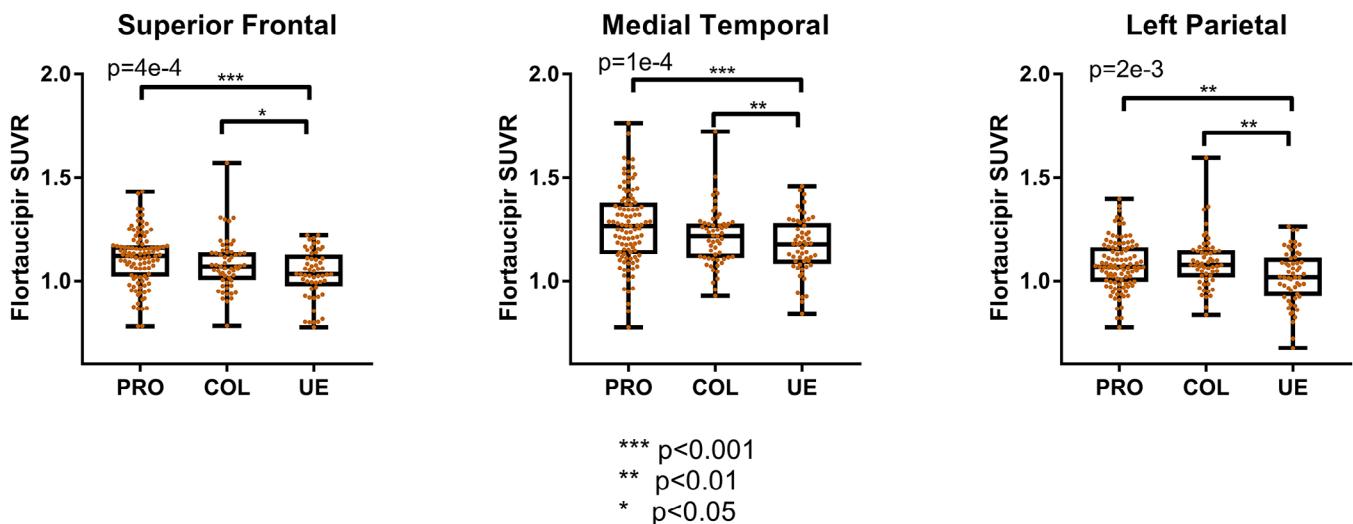


FIGURE 1 Comparison of flortaucipir SUVR in prespecified statistical ROIs among football exposure groups controlling for age and race. The *p*-value for the ANCOVA test was indicated for each plot, significance level of post hoc pair-wise comparison was also reported. ANCOVA, analysis of covariance; COL, former college football players; PRO, former NFL players; ROIs, regions of interest; SUVR, standardized uptake value ratio; UE, asymptomatic participants with no football exposure;

investigations of white matter changes in this cohort, increased white matter lesion was only observed in participants older than 60.³⁰

3 | RESULTS

Demographics for each of the three exposure groups are summarized in Table 1, example FTP SUVR images are shown in Figure S2. A statistically significant (ANCOVA $p < 0.01$) exposure group difference in regional FTP SUVR was observed for all three prespecified statistical ROIs controlling for age and race (Figure 1). In pair-wise post hoc comparisons, the PRO group had higher regional FTP SUVRs than the UE group for all three statistical ROIs ($p < 0.01$). The COL group also had higher regional FTP SUVRs than the UE group ($p < 0.05$). The combined group of all former football players (PRO+COL) also had higher FTP SUVRs than the UE group for all three regions ($p < 0.05$). No statistical differences were observed between the PRO and COL groups. In the exploratory analysis with anatomically defined regions, significant group difference in the same direction in the three-way comparison ($p < 0.05$) in FTP SUVR was observed in six of the eight preselected regions including entorhinal cortex, parahippocampal gyrus, superior frontal cortex, superior parietal cortex, hippocampus, and amygdala (Figure S3). The PRO group also had higher FTP SUVRs than the UE group in the same six regions in pair-wise comparisons ($p < 0.02$).

In the voxel-wise analysis, a total of 202 participants (99 PRO, 51 COL, and 52 UE) with consistent imaging data acquisition and pre-processing were included and controlled for age and race. The raw statistical significance (Z-score) map of the one-tailed test for the PRO+COL group having greater FTP SUVRs than the UE group is shown in the left panel of Figure 2 with a threshold of $p < 0.005$ not corrected for multiple comparisons which were dealt with using MCS. The map of the opposite direction is shown in the right panel. We observed

24016 voxels where the FTP SUVR was higher in former players than in UE, in comparison to only 1100 voxels where FTP SUVR was lower in former players than in UE. Given the difference in the number of voxels between the two directions, the omnibus significance assessed by MCS is $p < 0.001$. After FWE correction 43 voxels in the superior frontal cortex, medial temporal cortex, and precuneus remained significant in the expected direction while no voxels remained significant in the opposite direction. Similar findings were observed in the PRO versus UE and COL versus UE comparisons (Figure S4).

Regional FTP SUVR was not significantly associated with CHI-G in the PRO+COL group for any of the prespecified ROIs. However, in the sensitivity analysis, there was an association in the superior frontal region in PRO+COL participants over age 60 years ($p = 0.03$, Figure 3D). Regional FTP SUVRs were not significantly different between former players with a TES diagnosis from those who did not (Figure 4A-C). In addition, regional FTP SUVRs were also not significantly different between former players with a TES diagnosis at a suggestive level of CTE pathology and those who had a possible or probable level of CTE pathology (Figure 4D-F). The results were similar in the sensitivity analyses that only included participants over 60 years, and FTP SUVR was not able to differentiate TES diagnostic groups.

4 | DISCUSSION

In this study, we examined tau PET imaging with the FTP tracer in former professional and college American football players from the DIAGNOSE CTE Research Project cohort. Significantly higher FTP SUVR was observed in the former football players compared to the unexposed controls in all three prespecified ROIs (bilateral superior frontal, bilateral medial temporal, and left parietal), controlling for age and race. Higher FTP SUVR was also observed in six anatomically

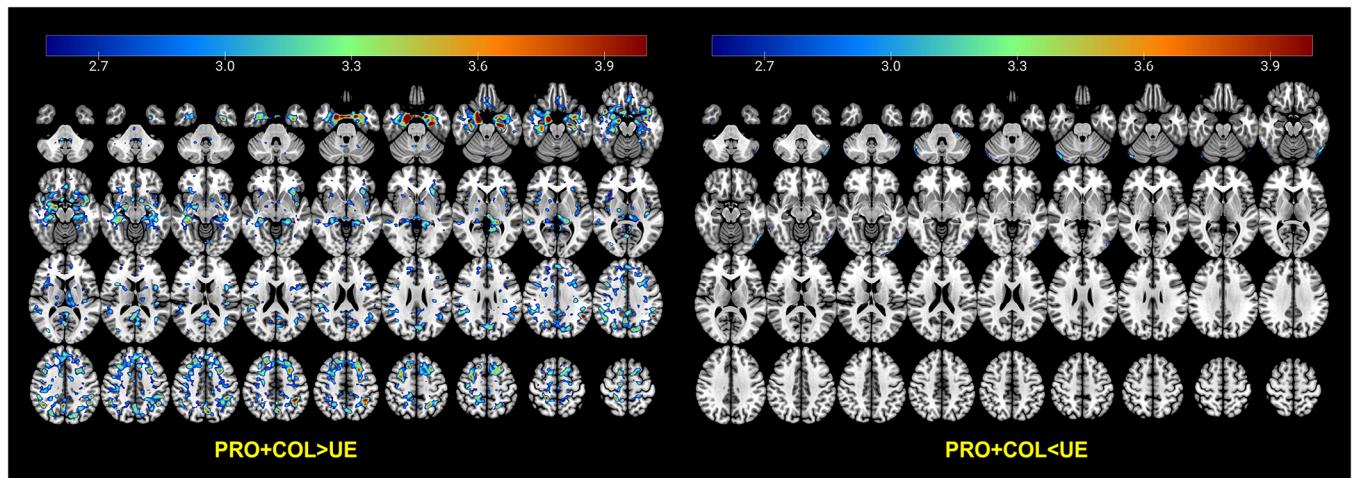


FIGURE 2 Voxel-wise comparison of FTP uptake between football exposure groups PRO+COL versus UE. Voxel-wise Z-score map for one-tailed test for each direction is shown at a threshold of $p = 0.005$ uncorrected for voxel-wise multiple comparisons which were dealt with using MCS, age and race are controlled for in all analyses. A total of 24,016 voxels were above this threshold in the expected direction (PRO+COL > UE) in contrast to 1100 voxels in the opposite direction (PRO+COL < UE). MCS analysis with $N = 1000$ iterations found overall significantly elevated FTP uptake in former American football players (PRO+COL) than control participants (UE) ($p < 0.001$). Additionally, 43 voxels remained significant after the FWE correction. COL, former college American football players ($N = 51$); FTP, flortaucipir; FWE, family-wise error; MCS, majority count statistics; PRO, former professional American football players ($N = 99$); UE, control participants not exposed to head injuries ($N = 52$)

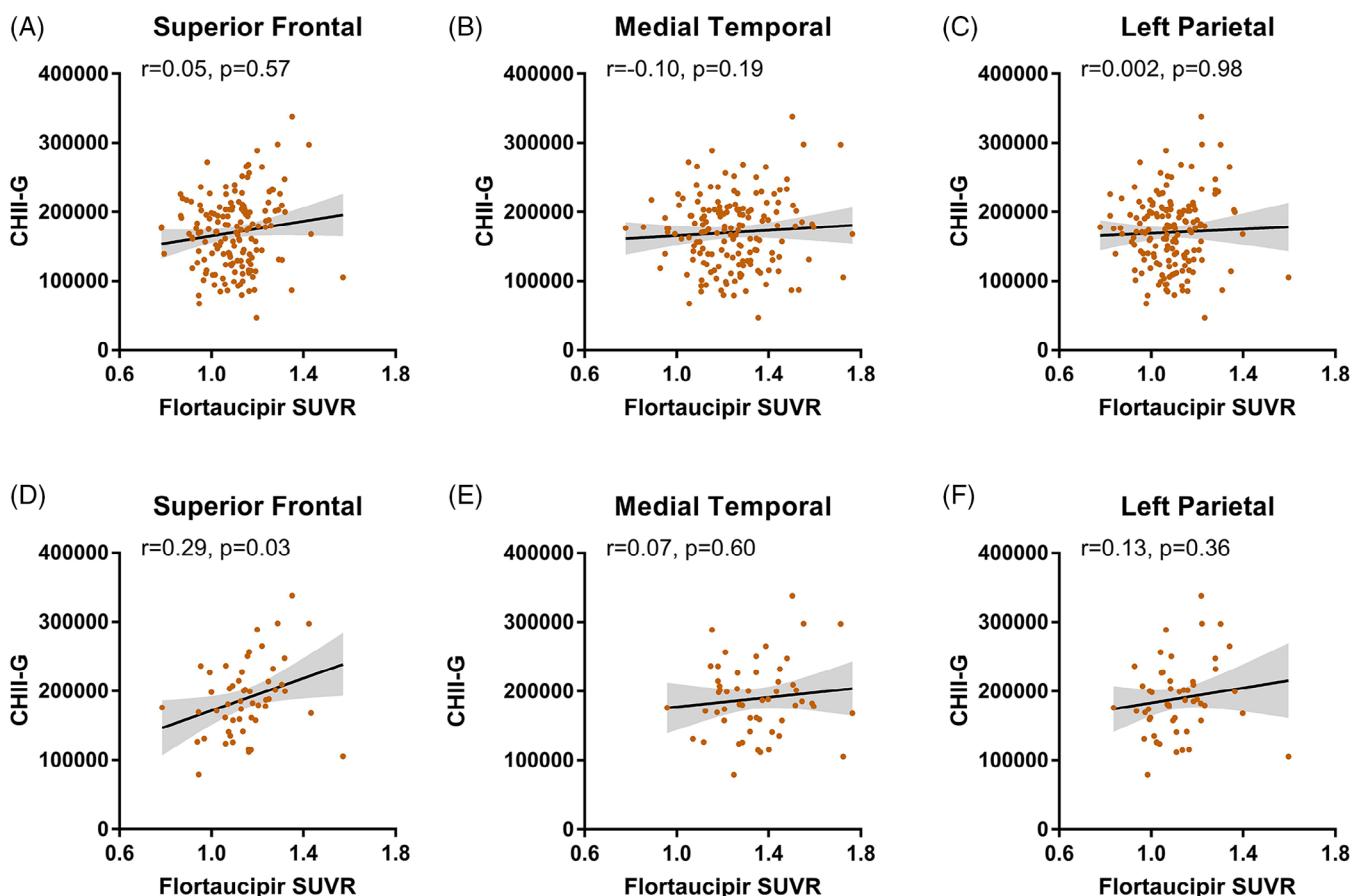


FIGURE 3 Association between regional FTP SUVR and CHII-G in the full COL+PRO group (A, B, C) and the subset of COL+PRO participants over age 60 years (D, E, F). All analyses controlled for age and race. CHII-G, cumulative head impact index based on measurements of linear acceleration; COL, former college football players; FTP, flortaucipir; PRO, former NFL players; SUVR, standardized uptake value ratio; UE, participants with no football exposure

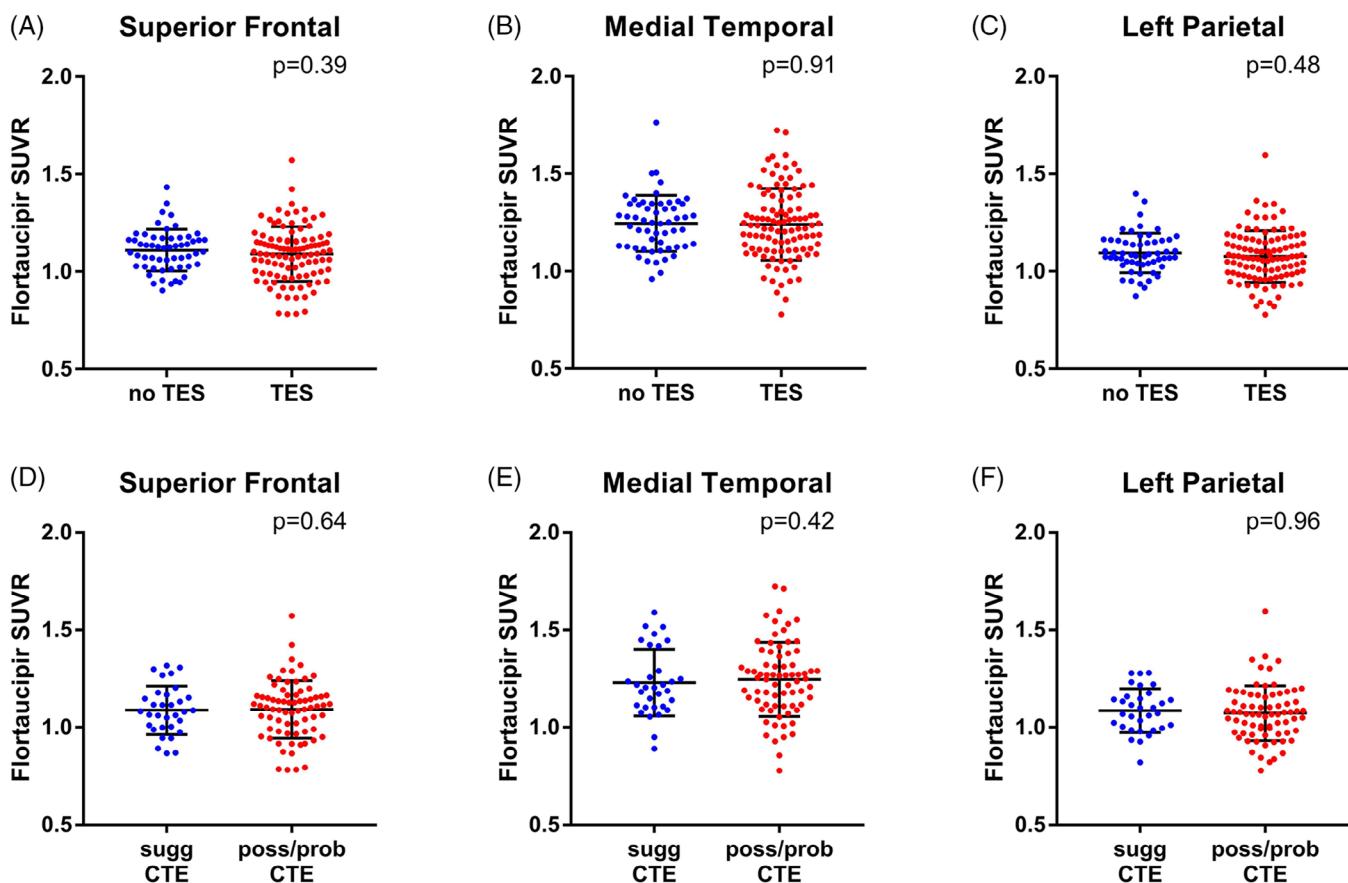


FIGURE 4 Comparison of FTP SUVRs in the prespecified regions between former American football players (PRO+COL) with or without a TES diagnosis (A, B, C); and comparison of FTP SUVRs in the prespecified regions between former players with a TES diagnosis and suggestive level of CTE pathology (sugg CTE) and former players with a TES diagnosis and possible or probable level of CTE pathology (poss/prob CTE) (D, E, F). COL, former college football players; CTE, chronic traumatic encephalopathy; FTP, flortaucipir; PRO, former NFL players; SUVR, standardized uptake value ratio; TES, traumatic encephalopathy syndrome

defined brain regions in former football players compared to controls. The voxel-wise analysis also revealed widespread elevation in FTP SUVR in the PRO group compared to the UE as well as for the combined player group versus UE. The spatial extent of the elevated FTP SUVRs included 39% of the voxels in the bilateral superior frontal region, 67% of voxels in the bilateral medial temporal region, and 25% of voxels in the left parietal region.

Tau burden measured by regional FTP SUVR did not show a significant association with estimated cumulative RHI exposure as measured by CHI-G in the full PRO+COL group. However, the association was significant when only former players older than 60 years were examined but only in the superior frontal region. This may suggest that there is a significant delay between exposure to RHI and the accumulation of tau pathology measurable by FTP PET, as would be expected in a progressive tauopathy. This may also reflect the specificity of FTP binding to 3R tau isoforms expected in later-stage CTE. FTP SUVR in our prespecified regions was not able to differentiate players who met diagnostic criteria for TES from those who did not. Similarly, there were no differences in FTP SUVR when comparing former players with lower versus higher levels of certainty of CTE pathology, based on the TES

criteria. These patterns remained the same when only older participants were examined.

The group level differences and the spatial patterns of elevated FTP SUVR observed in this study are in line with our previous study in a smaller cohort.²³ We extended the findings to demonstrate CTE-related tau pathology also affects former football players at the college level. The previous study reported the association of FTP SUVR with years of football play²³ which was not confirmed in our current study. Another recent study³¹ failed to observe differences in FTP SUVR between former professional football players and controls, and the discrepancy is attributable to the younger age and smaller sample size of that study given the moderate differences in FTP uptake and the delayed manifestation of tau pathology as we discussed earlier. A recent study of the incidence rate of CTE-related pathology in military personnel did not observe increased pathology in those who were exposed to blasts or other military-related TBI, while those with exposure to contact sports had a higher incidence rate of CTE pathology.³² This may be attributable to the fact that the exposure to head injury due to blasts or other military-related events was more likely to be incidental and less frequent than those playing contact sports.

In a small subset of six participants of this cohort, we have previously reported a moderately strong association between FTP SUVR and post mortem pathological measures of tau density.³³ Those findings, along with this current study suggest that FTP—as a first-generation tau PET tracer with substantial off-target binding—could be useful for detecting CTE-related tau pathology but may not be optimal. As described previously, although CTE is characterized by a mixture of 3R and 4R tau isoforms, their relative contribution to the overall tauopathy shifts from predominantly 4R in the early stages to 3R as the disease progresses²⁰ making FTP less optimal for detecting CTE related tau aggregates at the early stages and different tracers are likely needed for detecting CTE related tau pathology at different stages due to the stage related variability in the underlying tau pathologies.³⁴ Moreover, because early-stage CTE p-tau deposition is patchy, with a perivascular, sulcal depth distribution,^{4,18} tau tracer uptake across specific ROIs would not be expected to be as robust as is seen in early-stage AD pathology, for example.³⁵ This is also confirmed in this study where only patchy and moderately elevated FTP SUVR was observed in former football players and does not follow the typical spatial pattern of tau deposition in clinical/preclinical AD patients. Examination of other tau PET tracers^{36–38} is warranted, as is the development of tracers specifically targeting CTE tau based on knowledge gained through recent cryo-EM findings,^{21,22} as well as studies of molecular docking and dynamics simulations.³¹ PET imaging of other pathways such as neuroinflammation³⁹ in the individuals exposed to substantial RHI is warranted. Advanced quantification techniques^{40,41} may also improve the ability to use tau PET imaging to assess CTE-related tauopathies.

In summary, we confirmed our previous finding of modestly elevated PET measurements of tau tangle burden in empirically predefined ROIs in a larger number of former American football players and controls and related the findings to three levels of RHI exposure in the former professional players, former college players, and unexposed controls. Additional studies are needed to clarify the extent to which these or other measurements of PET or fluid biomarker measurements of tau tangle pathology are associated with different indicators of RHI former football players and other groups, the extent to which they distinguish between those who do or do not meet criteria for TES, and the extent to which they predict subsequent clinical progression and post mortem CTE pathology.

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CONFLICT OF INTEREST STATEMENT

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CONSENT STATEMENT

The study was approved by the Institutional Review Boards at all sites, and written informed consent was obtained for all participants.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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Clinical Outcomes and Tau Pathology in Retired Football Players

Associations With Diagnosed and Witnessed Sleep Apnea

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Abstract

Background and Objectives

Obstructive sleep apnea (SA) is common in older men and a contributor to negative cognitive, psychiatric, and brain health outcomes. Little is known about SA in those who played contact sports and are at increased risk of neurodegenerative disease(s) and other neuropathologies associated with repetitive head impacts (RHI). In this study, we investigated the frequency of diagnosed and witnessed SA and its contribution to clinical symptoms and tau pathology using PET imaging among male former college and former professional American football players.

Methods

The sample included 120 former National Football League (NFL) players, 60 former college players, and 60 asymptomatic men without exposure to RHI (i.e., controls). Diagnosed SA was self-reported, and all participants completed the Mayo Sleep Questionnaire (MSQ, informant version), the Epworth Sleepiness Scale (ESS), neuropsychological testing, and tau (flortaucipir) PET imaging. Associations between sleep indices (diagnosed SA, MSQ items, and the ESS) and derived neuropsychological factor scores, self-reported depression (Beck Depression Inventory-II [BDI-II]), informant-reported neurobehavioral dysregulation (Behavior Rating Inventory of Executive Function—Adult Version [BRIEF-A] Behavioral Regulation Index [BRI]), and tau PET uptake, were tested.

Results

Approximately 36.7% of NFL players had diagnosed SA compared with 30% of the former college football players and 16.7% of the controls. Former NFL players and college football players also had higher ESS scores compared with the controls. Years of football play was not associated with any of the sleep metrics. Among the former NFL players, diagnosed SA was associated with worse Executive Function and Psychomotor Speed factor scores, greater BDI-II

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scores, and higher flortaucipir PET standard uptake value ratios, independent of age, race, body mass index, and APOE $\epsilon 4$ gene carrier status. Higher ESS scores correlated with higher BDI-II and BRIEF-A BRI scores. Continuous positive airway pressure use mitigated all of the abovementioned associations. Among the former college football players, witnessed apnea and higher ESS scores were associated with higher BRIEF-A BRI and BDI-II scores, respectively. No other associations were observed in this subgroup.

Discussion

Former elite American football players are at risk of SA. Our findings suggest that SA might contribute to cognitive, neuropsychiatric, and tau outcomes in this population. Like all neurodegenerative diseases, this study emphasizes the multi-factorial contributions to negative brain health outcomes and the importance of sleep for optimal brain health.

Introduction

Obstructive sleep apnea (SA) is characterized by recurrent episodes of airway narrowing and collapse during sleep, leading to intermittent airflow limitation.¹ This results in snoring, snorting, and gasping for breath. Patients with SA present with daytime sleepiness, impaired cognitive test performance, and abnormal mood.² SA is common (30%–80% of older adults^{3,4}), yet frequently undiagnosed and even when diagnosed, frequently untreated.⁵

Compared with the general population, former American football players are likely at increased risk of SA given their demographics and body habitus. Prominent risk factors of SA include being overweight,⁶ having a large neck circumference, and being older and male.^{7,8} Players are also at increased risk to sustain traumatic brain injuries (TBIs), and TBIs have been associated with SA.⁹ Prior studies have shown symptoms of SA in 27% of former American football players, aged between 37 and 55 years, from the National Football League (NFL) Player Care Foundation Cardiovascular Health Screening Program.¹⁰ African American/Black race has also been reported as a risk factor of SA, although this is likely secondary to other health disparities related to racial inequities (e.g., access to health care and nutrition).^{11–13} Currently, approximately 70% of NFL players are Black.¹⁴ Of importance, the proportion of Black NFL players has changed over time because approximately 40% of NFL players were Black between 1959 and 1988.^{15–17}

The presence of SA in older adults has been associated with cognitive decline¹⁸ and dementia¹⁹ and thus might contribute to cognitive and neuropsychiatric symptoms that have been reported in older former American football players.²⁰ SA has also been associated with pathologic tau: This is observed in both objectively diagnosed SA and in those with sleep partners' report of witnessed apneas.²¹ While the link between SA and tau (using PET imaging) has been reported

in those with positive amyloid-beta status, i.e., those on the Alzheimer disease continuum,^{21,22} it has not been investigated in those at risk of primary tauopathies, such as chronic traumatic encephalopathy (CTE). CTE is a neurodegenerative tauopathy that can be diagnosed only neuropathologically at this time and has been identified at autopsy among those exposed to repetitive head impacts (RHI), commonly former football players and other contact sport athletes.^{17,23,24} Years of American football play has been associated with greater p-tau density at autopsy^{17,25} and greater flortaucipir binding on PET imaging in 26 former NFL players.²⁶ Thus, p-tau has an association with both SA and exposure to football in older symptomatic players. It is possible that SA may contribute to tau aggregation and associated symptoms in former American football players.

SA is treatable, and symptoms related to SA including depression²⁷ and memory²⁸ can be reversed even after a few months of treatment. Memory loss and mood symptoms are frequently reported in former football players and part of the suggested clinical manifestation of CTE.²⁰ Given its potential as a treatment target, we investigated diagnosed SA and its symptoms among former professional and college football players from the Diagnostics, Imaging, and Genetics Network for the Objective Study and Evaluation of Chronic Traumatic Encephalopathy (DIAGNOSE CTE) Research Project.²⁹ We examined (1) the frequency of SA in former players, (2) the association between SA and the reported major clinical manifestations of CTE, i.e., cognitive difficulties, depression, and behavioral dysregulation, and (3) the association between SA and tau (flortaucipir) uptake on PET. We conducted using a 2-tiered approach, given the potential importance of body mass index (generally higher in former football players) and apolipoprotein e E4 alleles (more frequent in African Americans but with distinct race-stratified impact on AD) in SA and AD risk; we ran models both without and with these variables. We also examined the influence of continuous positive airway pressure (CPAP) treatment on these associations. Because sleepiness is an important but nonspecific outcome of SA and can affect cognition and behavior, we also investigated self-reported sleepiness.

Methods

Participants and Design

Participants were enrolled in the DIAGNOSE CTE Research Project.²⁹ The objectives of DIAGNOSE CTE are to develop in vivo biomarkers for CTE, characterize its clinical presentation, and refine and validate clinical research diagnostic criteria. A total of 240 participants were enrolled, including 120 former NFL players, 60 former college football players, and 60 asymptomatic men who had no exposure to RHI or TBI. The former American football players spanned the symptom continuum, whereas the unexposed men were required to be asymptomatic. Detailed methodology of the

DIAGNOSE CTE Research Project including inclusion and exclusion criteria are published elsewhere.²⁹ The 240 study participants served as the primary sample for this study. However, the sample sizes for analyses varied because of missing data on study variables. Specific sample sizes for variables are described further and summarized in Table 1.

Evaluations took place at 1 of 4 US sites, including Boston University School of Medicine (with MRI conducted at Brigham and Women's Hospital), Cleveland Clinic Lou Ruvo Center for Brain Health in Las Vegas, Mayo Clinic Arizona (with PET imaging techniques conducted at Banner Alzheimer's Institute), and New York University Langone Medical Center. Baseline evaluations were conducted between September 2016 and February 2020. Participants completed a 2-day evaluation that consisted of a comprehensive clinical history, neurologic and neuropsychological examinations, flortaucipir PET for tau imaging, MRI, and participant-based and informant-based measures pertaining to sleep, mood, and behavior.

Standard Protocol Approvals, Registrations, and Patient Consents

All participants were required to have decisional capacity to participate in the study. All sites received approval by their Institutional Review Board, and participants provided written informed consent.

Measures

Sleep

Witnessed Apneas: Mayo Sleep Questionnaire (MSQ)

The MSQ was developed to screen for various sleep disorders associated with neurologic disease, including SA.³⁰ It is a 16-item measure that is completed by both the patient and the informant. The informant version is considered sensitive and specific when compared with polysomnography (PSG) findings.³⁰ Only informant results are reported here. There are 2 questions specific to apnea, and these include number 5, "Has the patient ever snorted or choked himself awake?" (we will refer to this as *snorted*), and number 6, "Does the patient ever seem to stop breathing during sleep?" (we will refer to this as *breathe*). For both questions, participant response choices are yes or no. Compared with PSG data in the validation cohort, affirmative answer to either question had a 61% sensitivity to SA as measured by PSG, and the specificity was 89% if both questions were negative.³⁰ In this study, the sample size (due to missing informant data) was 159 on the MSQ items due to missing informant responses ($n = 88$ former NFL players, $n = 42$ former college football players, and $n = 29$ asymptomatic unexposed men).

*Epworth Sleepiness Score*³¹ is a self-report instrument that asks people to rate their likelihood of falling asleep in 8 different activities. Participants rate this on a 0–4 scale, where higher scores reflect greater sleepiness. Sample size is reduced to 238 for this variable due to missing information for 2 participants.

Diagnosed SA and CPAP Treatment

Participants completed questionnaires about medical history, including the yes/no question, "have you ever been diagnosed with SA?" ($n = 236$ as 4 reported "unknown"). If there is an affirmative response, there is also the yes/no question "are you prescribed CPAP?" These variables were examined in this study.

Neuropsychological Assessment

Participants underwent a comprehensive in-person neuropsychological battery at baseline, the details of which are described elsewhere.²⁹ This battery included more than 15 individual tests that assess all major cognitive domains, including attention, visual scanning, and psychomotor speed; executive functions; learning and episodic memory (verbal and visual); language; and visuospatial abilities. Principal component analyses were used to create 5 cognitive domain factor scores, including (1) Verbal Learning and Memory; (2) Executive Function and Psychomotor Speed; (3) Visual Learning and Memory; (4) Number Span Forward and Backward; and (5) Verbal Fluency. These factor scores were used as outcomes in this study. For all factors, higher scores represent better performance.

Mood and Behavior Self-Report Questionnaires

We examined symptoms of neurobehavioral dysregulation and depression. Neurobehavioral dysregulation is a core clinical feature of the 2021 research criteria for traumatic encephalopathy syndrome (TES, the purported clinical syndrome of CTE), and symptoms of depression are a supportive feature. Neurobehavioral dysregulation was assessed by the informant version of the Behavior Rating Inventory of Executive Function—Adult Version (BRIEF-A).³² This is a well-validated 75-item measurement of executive function behaviors. Informants rated how often each behavior had been a problem on a 3-point scale (1 = never, 2 = sometimes, and 3 = often). Higher scores indicate greater dysfunction. The BRIEF-A yields 3 indices including the Behavioral Regulation Index (BRI). The BRI reflects items that assess an individual's ability to control impulses and self-monitor their behavior. BRI raw scores are converted to age-adjusted T scores, which served as an outcome. To assess symptoms of depression, we used the established self-report scale, the Beck Depression Inventory-II (BDI-II).³³ Higher scores reflect greater depressive symptomatology.

Tau PET

The use of flortaucipir in this study was performed through an Investigator Investigational New Drug (IND #131391) from the US Food and Drug Administration. Eighty minutes after a 370 MBq (10 mCi) bolus injection, the participant completed a continuous dynamic 20-minute brain scan (4 frames, 5 minutes each). Imaging calibration and quality control procedures were completed for all sites before study enrollment. Participants also underwent MRI. The structural MRI was used for coregistration of the PET scans in this study. Fully bias field-corrected and intensity-normalized T1-MRI from the Free-Surfer pipeline (T1.mgz) was spatially normalized using the

Table 1 Sample Characteristics

	Former professional players (n = 120)	Former college players (n = 60)	Asymptomatic unexposed participants (n = 60)	Effect ^a
Demographics				
Age, mean (SD) y	59.1 (7.8)	53.5 (7.7)	59.3 (8.3)	COL<PROF; COL<UE
Education, mean (SD) y	16.6 (1.1)	17.1 (1.0)	17.3 (3.4)	NS
Race, n (%)				
American Indian or Alaska Native	0 (0)	0 (0.0)	0 (0.0)	PROF>COL; UE>COL
Black or African American	52 (43.3)	11 (18.3)	24 (40.0)	
Native Hawaiian or other Pacific Islander	0 (0.0)	0 (0.0)	1 (1.7)	
White	66 (55.0)	48 (80.0)	35 (58.3)	
Multiple races	2 (1.7)	1 (1.7)	0 (0.0)	
Ethnicity, n (%)				
Hispanic or Latino	3 (2.5)	0	0	—
Athletics				
Duration of football play, mean (SD) y	18.0 (3.3)	11.5 (2.5)	—	<0.01
Position group at highest level of play, n (%)				
Offensive line	22 (18.3)	22 (36.7)	—	COL>PROF
Defensive lineman	14 (11.7)	5 (8.3)	—	
Offensive backs and receivers	36 (30.0)	14 (23.3)	—	
Linebackers	21 (17.5)	7 (11.7)	—	
Defensive backs	23 (19.2)	12 (20.0)	—	
Special teams	4 (3.3)	0 (0.0)	—	
Cardiovascular disease				
Body mass index, mean (SD)	32.0 (4.5)	33.8 (4.8)	30.8 (4.5)	COL>PROF; COL>UE
Systolic blood pressure, mean (SD) mm Hg	124.8 (11.2)	129.9 (13.8)	133.9 (13.3)	UE>COL; UE>PROF; COL>PROF
Diagnostic history of high cholesterol, n (%)	48 (40.0)	19 (31.7)	26 (43.3)	NS
Diagnosed or treated for hypertension, n (%)	53 (44.2)	25 (41.7)	26 (43.3)	NS
Diagnostic history of diabetes, n (%)	9 (7.5)	3 (5.0)	7 (11.7)	NS
APOE genotype				
ε4, n (%) present	33 (28.7)	20 (33.9)	11 (19.6)	NS
Sleep characteristics				
MSQ-snorted, n (%)	35 (39.8)	24 (57.1)	9 (31.0)	—
MSQ-gasping, n (%)	33 (37.5)	24 (57.1)	7 (24.1)	—
ESS, mean (SD)	8.7 (5.3)	8.8 (5.0)	5.9 (3.6)	—
Diagnostic history of SA, n (%)	44 (36.7)	18 (30.0)	10 (16.7)	—

Continued

Table 1 Sample Characteristics (continued)

	Former professional players (n = 120)	Former college players (n = 60)	Asymptomatic unexposed participants (n = 60)	Effect ^a
Prescribed CPAP, n (%)	31 (70.5)	14 (77.8)	8 (80.0)	—

Abbreviations: COL = College; ESS = Epworth Sleepiness Scale; MSQ = Mayo Sleep Questionnaire (informant); Prof = professional; SA = obstructive SA; UE = unexposed.

^aAnalysis of variance with Tukey post hoc compared the former professional football players, former college football players, and the asymptomatic unexposed men on continuous outcomes. The Chi-square or Fisher exact test (for cell sizes <10) were used for binary outcomes. For athletic characteristics, only the former professional and college football players were compared. Race was examined as a binary variable (Black vs non-Black) as was position played (lineman vs nonlinebacker). Group comparisons for the sleep characteristics are modeled in Table 2. Data in this Table are based on characteristics of the entire DIAGNOSE CTE Research Project sample. The MSQ is the informant version, and sample sizes range due to missing data and include the following: MSQ-snored, n = 130 former American football players, n = 29 unexposed; MSQ-Gasping, n = 130 former American football players, n = 29 unexposed; and ESS, n = 178 former American football players, n = 60 unexposed. Sample sizes also reduced for APOE, n = 174 former American football players, n = 56 unexposed.

Statistical Parametric Mapping software to generate the individual-to-template space nonlinear transformation and transform PET data into the Montreal Neurological Institute template space and renormalized using cerebellum crus 1 region as the reference. This allowed the extraction of the previously defined statistically defined regions of interest (ROI) through voxel-wise analysis, which were found to have elevated flortaucipir standard uptake value ratios (SUVRs) in former NFL players,²⁶ including bilateral frontal, left parietal, and bilateral medial temporal regions (referred to as prespecified statistical ROIs henceforth).

Sample Characteristics

Semistructured interviews were performed, supplemented by online questionnaires, to collect data on demographics (e.g., age, education, race, and ethnicity); clinical, athletic, military, and TBI history; and other variables not relevant to this study. An aliquot of whole blood collected during the baseline blood draw was used for apolipoprotein E (APOE) genotyping. Race and ethnicity were self-reported.

Statistical Approach

Analyses were performed to address 3 specific study questions that included the following: (1) How common are SA and its symptoms in participants in the DIAGNOSE CTE, i.e., former NFL players and former college football players? (2) Are SA and its symptoms associated with cognitive and neuropsychiatric function in former football players? and (3) Are SA and its symptoms associated with tau in former football players? To test each of these questions, multivariable linear regression models were performed. The first model compared each group of former football players (i.e., college and NFL players) and the unexposed asymptomatic men on each sleep symptom (i.e., diagnosed SA, or witnessed apnea snored, and difficulty breathing). We then tested the association between each sleep symptom and (1) each neuropsychological factor score and each neuropsychiatric scale and (2) flortaucipir uptake on PET. Models controlled for age and race (self-reported as Black or White, and we created an “other” group that combined the other race identifications that were infrequent for statistical purposes). As a sensitivity analysis, we added body mass index (BMI) and APOE e4 carrier status as covariates to each model. BMI is elevated in former football players,³⁴ an indicator of

vascular health, and when elevated is a risk factor of SA.⁷ APOE e4 status has been linked with SA risk³⁵ and tauopathies,³⁶ and these relationships are moderated by race.³⁷ Finally, among those with diagnosed SA, we added prescribed CPAP to each model to determine whether this moderated the impact of SA on the outcomes. For analyses that examined the association between the SA indices and the neuropsychological factor scores and neuropsychiatric scales, *p* values were false discovery rate (FDR) adjusted using the Benjamini-Hochberg procedure. A *p* value (or FDR *p* value) less than or equal to 0.05 defined statistical significance.

Data Availability

Datasets generated and/or analyzed during this study are available from the corresponding author on reasonable request.

Results

How Common Are SA Symptoms in Former Players, and Does This Differ by Level of Play or Duration of Exposure?

Sample demographic and symptom characteristics are listed in Table 1. Analysis of variance with Tukey post hoc compared the former NFL players, former college football players, and the asymptomatic unexposed men on Table 1 characteristics. The former college football players were younger, more likely to be White, and had a higher BMI compared with the former NFL players and the unexposed men. However, there were minimal differences between the former NFL players and asymptomatic unexposed men. Given the differences between former college and NFL players, which might affect several variables of interest, we examined the groups separately for all analyses.

Table 2 summarizes models examining group differences on sleep characteristics. Close to 37% (31.4%) of the former NFL players and 30% of former college football players were diagnosed with SA compared with 16.7% of the asymptomatic unexposed men. Of the former football players with SA, 42.4% of the former NFL and 33.33% of the former college players were prescribed CPAP compared with 28.6% of the asymptomatic unexposed men.

Table 2 Summary of Linear Regression Models Comparing Former American Football Players and Asymptomatic Unexposed Participants on Sleep Indices

Sleep variable	Model 1: Adjusting for age and race						Model 2: Adjusting for age, race, BMI, and APOE ε4					
	College			NFL			College			NFL		
	Est.	95% CI	p Value	Est.	95% CI	p Value	Est.	95% CI	p Value	Est.	95% CI	p Value
MSQ-snorted	0.97	-0.10 to 2.09	0.08	0.31	-0.61 to 1.28	0.52	0.77	-0.36 to 1.95	0.18	0.35	-0.59 to 1.35	0.47
MSQ-gasping	1.92	0.74 to 3.30	0.003**	0.59	-0.35 to 1.62	0.24	1.75	0.45 to 3.23	0.01*	0.55	-0.43 to 1.62	0.28
Diagnosed SA	0.66	-0.32 to 1.68	0.19	1.06	0.31 to 1.89	0.01*	0.30	-0.77 to 1.39	0.58	0.91	0.05 to 1.83	0.04*
ESS	2.99	1.21 to 4.76	0.001**	2.73	1.26 to 4.19	0.0003**	2.98	1.08 to 4.89	0.002**	2.31	0.81 to 3.82	0.002**

Abbreviations: BMI = body mass index; ESS = Epworth Sleepiness Scale; MSQ = Mayo Sleep Questionnaire; SA = sleep apnea.

Est (estimates) are unstandardized.

* $p < 0.05$. ** $p < 0.005$.

Former NFL Players

In models including age and race as covariates, we assessed group differences in each sleep symptom in reference to the asymptomatic unexposed men. Former NFL players were more likely to have diagnosed sleep apnea ($\beta = 1.06$, $p = 0.008$) and report higher levels of sleepiness ($\beta = 2.73$, $p = 0.0003$) but groups did not differ on witnessed apneas. Age was a significant predictor of snorted and sleepiness. Black race was associated with more sleepiness in the former NFL players. When BMI and APOE ε4 were added to the models, the differences remained with the former NFL players being more likely to have diagnosed sleep apnea ($\beta = 0.91$, $p = 0.04$) and to report higher levels of sleepiness ($\beta = 2.31$, $p = 0.003$) than the asymptomatic unexposed men. Black race remained as a predictor of higher reported daytime sleepiness ($\beta = -3.82$, $p = 0.02$) with the BMI and APOE covariates.

Former College Football Players

In models with former college football players and asymptomatic unexposed men, interruptions to breathing were more common in the former college football players ($\beta = 1.92$, $p = 0.003$), and they also reported more sleepiness ($\beta = 2.99$, $p = 0.001$). Age and race were not significant predictors of any sleep metric. When BMI and APOE ε4 were added to the models, the relationships remained with former college players being more likely to have interruptions to breathing ($\beta = 1.75$, $p = 0.012$) and greater sleepiness ($\beta = 2.98$, $p = 0.0003$).

Association Between Sleep Indices and Years of Football Play

Years of football play was not associated with any of the sleep metrics in either former college football players or former NFL players, with or without APOE and BMI included. For former NFL players, Black race was significantly associated with diagnosed SA ($\beta = -2.62$, $p = 0.009$) and sleepiness ($\beta = -2.89$, $p = 0.0046$) when years of play was included in the model, such that White players were more likely to have a diagnosis, and reported lower levels of sleepiness. Covariates

were not significant in the models with former college football players only.

Are SA Symptoms Associated With Cognition, Symptoms of Depression, and Neurobehavioral Dysregulation in Former American Football Players?

Former NFL Players

In the former NFL players, diagnosed SA was associated with lower scores (i.e., worse) on the Executive Function and Psychomotor Speed factor score ($\beta = -0.58$, $p = 0.3$) and higher BDI-II scores ($\beta = 4.86$, $p = 0.01$) on gasping. Higher ESS scores correlated with higher BDI-II ($\beta = 0.48$, $p < 0.005$; Table 3). With APOE ε4 and BMI in the models, the relationship between diagnosed SA and the Executive Function and Psychomotor Speed factor score was non-significant, but the effect size remained comparable with the main models ($\beta = -0.46$, uncorrected $p = 0.05$, FDR corrected $p = 0.24$). ESS remained associated with the BDI-II and BRIEF-A BRI ($\beta = 0.41$, $p = 0.01$, $\beta = 0.68$, $p = 0.03$, respectively). When CPAP treatment was added to the models, there were no longer significant associations between witnessed or diagnosed SA and the clinical outcome measures. ESS continued to be associated with BDI-II scores ($\beta = 0.67$, $p = 0.02$).

Former College Football Players

In the former college football players, snorted and gasping were associated with higher BRIEF-A BRI scores ($\beta = 11.88$, $p = 0.04$; $\beta = 12.24$, $p = 0.03$, respectively). Higher ESS scores correlated with higher (i.e., better) Visual Memory factor scores ($\beta = 0.08$, $p < 0.005$) and BDI-II scores ($\beta = 0.74$, $p = 0.01$). With APOE ε4 and BMI in the models, there were no significant relationships between witnessed or diagnosed SA and the neuropsychological factor scores, BRIEF-A BRI or the BDI-II. ESS continued to be associated with Visual Memory factor scores and BDI-II (Visual Memory: $\beta = 0.08$, $p < 0.005$; BDI: $\beta = 0.73$, $p = 0.01$). When CPAP treatment was added to the models, the only relationship that remained was between ESS and the BRIEF-A BRI ($\beta = 2.41$, $p = 0.04$).

Table 3 Association Between Sleep Indices and Clinical Symptoms in Former American Football Players

Measure	MSQ-snorted			MSQ-gasping			Diagnosed SA			ESS		
	Est.	95% CI	p Value	Est.	95% CI	p Value	Est.	95% CI	p Value	Est.	95% CI	p Value
Former college football players												
Verbal learning and memory	-0.35	-0.96 to 0.27	0.33	-0.17	-0.80 to 0.46	0.84	-0.07	-0.66 to 0.52	0.99	-0.02	-0.07 to 0.03	0.79
Visual learning and memory	0.33	-0.20 to 0.86	0.33	0.04	-0.50 to 0.59	0.87	-0.38	-0.87 to 0.12	0.33	0.08	0.04 to 0.12	<0.01*
Executive function and psychomotor speed	-0.32	-0.85 to 0.20	0.33	-0.11	-0.65 to 0.42	0.84	-0.43	-0.92 to 0.06	0.33	-0.02	-0.07 to 0.03	0.79
Number span forward and backward	-0.54	-1.11 to 0.04	0.33	-0.50	-1.08 to 0.09	0.47	-0.02	-0.63 to 0.58	0.99	0.00	-0.05 to 0.06	0.98
Verbal fluency	-0.12	-0.90 to 0.67	0.76	0.22	-0.57 to 1.00	0.84	0.01	-0.63 to 0.58	0.99	-0.01	-0.07 to 0.05	0.89
BRIEF-A BRI (informant version)	11.88	2.18 to 21.59	0.04*	12.24	2.57 to 21.91	0.03*	9.10	-0.69 to 18.88	0.14	0.79	-0.12 to 1.70	0.09
BDI-II	3.44	-3.07 to 9.94	0.29	1.03	-5.57 to 7.63	0.75	4.01	-2.28 to 10.30	0.21	0.73	0.18 to 1.29	0.02*
Former NFL players												
Verbal learning and memory	-0.32	-0.71 to 0.06	0.49	-0.13	-0.52 to 0.27	0.86	0.02	-0.34 to 0.37	0.92	-0.02	-0.06 to 0.01	0.35
Visual learning and memory	0.19	-0.24 to 0.62	0.66	-0.02	-0.45 to 0.42	0.94	0.07	-0.32 to 0.45	0.92	0.01	-0.03 to 0.04	0.69
Executive function and psychomotor speed	-0.02	-0.48 to 0.44	0.95	0.31	-0.15 to 0.76	0.86	-0.58	-0.99 to -0.16	0.03*	-0.01	-0.05 to 0.03	0.69
Number span forward and backward	-0.12	-0.55 to 0.31	0.73	0.17	-0.26 to 0.61	0.86	0.17	-0.22 to 0.57	0.75	-0.02	-0.05 to 0.02	0.68
Verbal fluency	-0.17	-0.56 to 0.23	0.66	-0.08	-0.48 to 0.32	0.86	-0.14	-0.51 to 0.23	0.75	-0.03	-0.07 to 0.00	0.35
BRIEF-A BRI (informant version)	5.81	-0.28 to 11.91	0.06	5.88	-0.29 to 12.04	0.06	0.59	-5.40 to 6.57	0.85	0.57	-0.01 to 1.15	0.05*
BDI-II	3.92	0.04 to 7.80	0.06	3.87	-0.06 to 7.79	0.06	4.86	1.67 to 8.06	0.01*	0.48	0.19 to 0.76	<0.01*

Abbreviations: BDI-II = Beck Depression Inventory-II; BMI = body mass index; BRI = Behavioral Regulation Index; BRIEF-A = Behavior Rating Inventory of Executive Function-Adult Version; ESS = Epworth Sleepiness Scale; MSQ = Mayo Sleep Questionnaire.

Linear regressions tested the association between each sleep index and each neuropsychological, mood, and behavior measure. Est (estimates) are unstandardized. p Values are false discovery rate adjusted using the Benjamini-Hochberg procedure. Neuropsychological measures are factor scores derived using principal component analyses.

*p < 0.05.

Are SA Symptoms Associated With Increased Tau in Former Football Players?

Former NFL Players

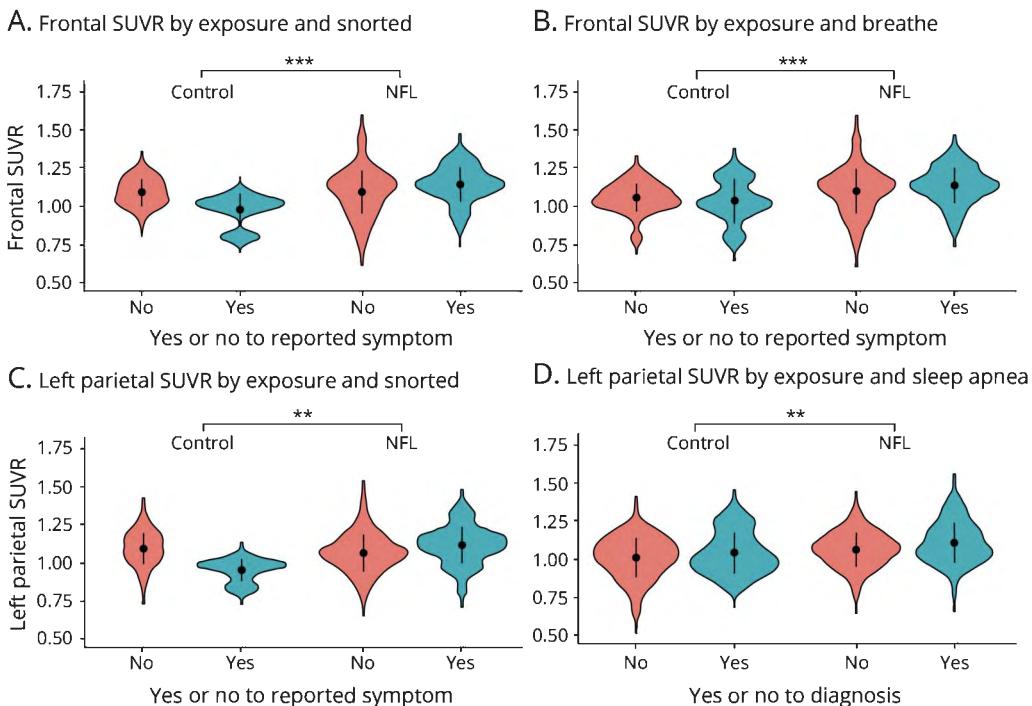
In the former NFL players, snorting was associated with greater frontal ($\beta = 0.08$, $p = 0.01$ Figure, A) and left parietal ($\beta = 0.07$, $p = 0.01$ Figure, C) flortaucipir SUVRs; gasping was associated with greater frontal SUVR ($\beta = 0.06$, $p = 0.04$ Figure, B); and diagnosed SA was associated with greater left parietal SUVR ($\beta = 0.05$, $p = 0.04$ Figure, D). With BMI and APOE ε4 in the model, the findings for snorted and frontal

and left parietal SUVRs remained significant ($\beta = 0.07$, $p = 0.01$, $\beta = 0.06$, $p = 0.03$, respectively). By contrast, the findings for gasping and diagnosed SA were no longer significant. However, effect sizes remained similar. When CPAP treatment was added to the model, none of the relationships with flortaucipir SUVR remained significant.

Former College Football Players

There were no significant sleep and tau PET associations among the former college players.

Figure Relationship Between Obstructive Sleep Apnea and Tau PET



Violin plots illustrating the frequency of yes/no answers for witnessed (A-C) and diagnosed (D) apnea in relation to tau PET SUVR in the frontal (A and B) and left parietal (C and D) regions where former NFL players with the diagnosed or witnessed apnea had a higher overall SUVR compared with former NFL players without the symptom/diagnosis or controls. When MBI and APOE status were added to the models, the findings for "snorted" (A and C) remained significant. NFL = National Football League; SUVR = standard uptake value ratio.

Discussion

This study examined rates of SA and its association with clinical outcomes and tau (flortaucipir) PET in former NFL players and former college football players. We present findings from these groups separately, and when controlling for BMI and APOE, most findings remained. First, among the former NFL players, findings included the following: (1) Compared with asymptomatic unexposed men, former NFL players were more likely to have diagnosed SA and to report higher levels of sleepiness; (2) Diagnosed SA was associated with worse executive function and processing speed (without BMI and APOE in the model) and significantly associated with greater symptoms of depression (with or without BMI and APOE in the model). Excessive sleepiness corresponded to greater neurobehavioral dysregulation; and (3) Uptake of tau (flortaucipir) PET in frontal and left parietal regions was associated with SA witnessed apneas. Adding CPAP use to our models removed the associations between SA symptoms and flortaucipir SUVRs and neuropsychological test performance.

Second, the pattern of findings in the former college football players were slightly different. Specifically, the associations were generally restricted to witnessed SA and greater neurobehavioral dysregulation and experiencing excessive sleepiness and more symptoms of depression. There was a paradoxical positive association between sleepiness and

visual memory, which is likely driven by hidden variables or a statistical artifact given that the directionality does not make sense clinically. Most notably, the associations between SA symptoms and tau PET SUVR were not significant in the former college football players. Given the known association between years of football play and pathologic tau,¹⁷ it is possible that tau was less extensive in the former college football players vs the former NFL players.

While we confirmed that SA is common in the age group studied (45–74 years), it was more frequent in former NFL players compared with our internal comparison groups (former college football players, asymptomatic unexposed men). Although an intuitive explanation would be the larger BMI of former NFL players, the findings for both diagnosed and witnessed SA remained after controlling for BMI. Although not measured, neck circumference might be a potential explanation given that this tends to be higher in football players and is a risk factor of SA.³⁸ There was also a higher proportion of Black participants among the former NFL players, and Black race was associated with diagnosed SA particularly when years of play was included in the models. This finding is consistent with reports of racial disparity in the instance of SA in nonplayer cohorts.³⁹ Factors associated with race might explain the observed effects in the former NFL players, and this deserves further attention as part of a separate study. Finally, there is existing evidence that TBI is associated with sleep disruption and SA, but the

biological mechanisms of such associations are unclear.⁴⁰ In our sample, years of American football play, a proxy for duration of exposure to RHI, were not associated with SA symptoms. These findings suggest exposure to RHI is unlikely to fully account for the higher rates of SA in former NFL players.

We found associations between SA and executive function and psychomotor speed and with symptoms of depression and neurobehavioral dysregulation in former NFL players.³⁸ There were no associations with memory. Impairments in executive function and memory and neurobehavioral dysregulation are core features of the TES research diagnostic criteria, the purported clinical syndrome of CTE.²⁰ Symptoms of depression are a supportive feature. While the sample studied is at risk of CTE given their exposure to repetitive head injuries,²¹ the presence and severity of underlying CTE pathology is uncertain. Similar to AD and other causes of dementia,⁴¹ SA and other modifiable risk factors likely exacerbate and/or independently contribute to cognitive impairments in people at risk of CTE. Given how common SA is in former American football players^{39–41} and that both mood²⁷ and cognition²⁸ can improve with SA treatment, SA is an important modifiable target for intervention in this population. Although the number of participants with untreated diagnosed SA in our cohort was low, controlling for CPAP treatment eliminated associations between SA and executive function and psychomotor speed.

Tau pathology has previously been associated with SA among people who were amyloid positive on PET.²¹ In this sample, tau PET was related to SA in the former NFL players. Specific effects were found for the left parietal and frontal ROIs, but not the mesial temporal region. This is interesting given the expected relationship between mesial temporal tau and AD pathology,⁴² even very early in the disease. This pattern suggests that the associations observed are not related to AD tau, which coincides with most of the sample being amyloid negative. As mentioned, it is also uncertain whether our findings relate to the presence of CTE p-tau. However, it is reasonable to hypothesize that SA might independently contribute to and/or exacerbate the development of p-tau pathology in people at risk for CTE. Although not the focus of this study, the frontal p-tau uptake might partly explain the observed association between SA and symptoms of neurobehavioral dysregulation and executive dysfunction. In this cohort, controlling for CPAP rendered the associations with tau PET findings nonsignificant.

We reported results for sleepiness, which can be associated with SA but is nonspecific, and common in the setting of depression, pain, and other symptoms often reported by former elite football players. Sleepiness was not associated with BMI, while witnessed and diagnosed apnea were. Sleepiness was greater in the former football players and associated with symptoms of depression and neurobehavioral dysregulation, but not cognitive function. The relationship

between sleepiness and depression and neurobehavioral dysregulation was independent of CPAP use. It was also not associated with flortaucipir uptake. Although sleepiness was common in the former football players, it had different clinical and biological correlates showed different than SA and could be related to other factors that will be explored in future analyses.

Our study is not without limitations. We did not perform objective sleep studies. While the measures that we used have substantial alignment with PSG,³⁰ future research on SA in former football players should include these gold standard measures. Since the inception of DIAGNOSE CTE, tau imaging has evolved. Flortaucipir is not the most sensitive to CTE p-tau pathology,⁴³ and later-generation tracers might better identify the kind of tau pathology that we might find in this cohort. Many of our findings seem to be specific to the former NFL players, but this could be related to statistical power, given there are twice as many former NFL players as college players in our sample. Last, the recruitment design of the DIAGNOSE CTE was designed to develop biomarkers for CTE. It involved recruitment of people based on exposure to RHI (i.e., former elite American football players) and symptoms (i.e., the unexposed men were required to have no symptoms). Given SA is associated with cognitive and neurobehavioral symptoms in the general population, it is possible that rates of SA and associated estimates were biased by the recruitment design.

Overall, our findings indicate that former American football players are at risk of SA, and this may be related to factors that extend beyond BMI. Moreover, SA was not related to years of play, arguing against a direct mechanistic relationship with exposure to RHI. Among former NFL players, SA was also associated with worse cognitive function and neurobehavioral dysregulation and with greater uptake of flortaucipir PET. Many of these associations diminished when accounting for CPAP treatment. As is true for all neurodegenerative diseases, this study shines a light on the multifactorial contributions to negative brain health outcomes that have been reported in former American football players. Specifically, this study emphasizes the important relationship between sleep apnea symptoms, which is treatable, and brain health.

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Effects of mTBI with loss of consciousness on neurobehavioral symptoms, depression, and insomnia in former collegiate and NFL football athletes

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ABSTRACT

Objective: Considering that diagnostic decisions about mTBI are often predicated on clinical symptom criteria, it is imperative to determine which initial presentation features of mTBI have prognostic significance for identifying those at high risk for long-term functional impairment.

Setting: Zoom interview Participants: Male, former NCAA Division I, and professional-level National Football League (NFL) athletes ($n = 177$) between the ages of 27 and 85 ($M = 54.1$, $SD = 14.7$).

Design: Cross-sectional case-control. Main Measures: History of mild TBI, history of loss of consciousness (LOC), depression symptoms, insomnia, neurobehavioral symptoms.

Results: Number of mTBI exposures did not predict neurobehavioral symptoms ($B = 0.21$, $SE = 0.18$, $p = 0.23$), but number of mTBI + LOC events did ($B = 2.27$, $SE = 0.64$, $p = <.001$). Further analysis revealed that the number of mTBI + LOC events predicted neurobehavioral symptoms indirectly through both depression ($B = 0.85$, 95% CI = [0.27, 1.52]) and insomnia ($B = 0.81$, 95% CI = [0.3, 1.4]). Further, the direct effect of mTBI + LOC events on neurobehavioral symptoms became non-significant when depression and insomnia were added to the model ($B = 0.78$, $SE = 0.45$, $p = 0.08$).

Conclusions: Findings support LOC at time of injury as an important predictor of long-term outcomes. Additionally, results suggest depression and insomnia as potential mediators in the association between mTBI + LOC and neurobehavioral symptoms. These findings provide justification for early depression and insomnia symptom monitoring following mTBI + LOC.

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Introduction

Worldwide, mild traumatic brain injury (mTBI) affects approximately 42 million individuals annually and has become a pressing health concern over the past several decades (1,2). Diagnosing mTBI or ‘concussion,’ has become increasingly controversial, as there is no universal consensus on a definition. Generally, mTBI is characterized by head injuries that result in disrupted brain function as evidenced by altered mental status (e.g. confusion and disorientation), loss of consciousness (LOC) (<30 min), post-traumatic amnesia (<24 hours), and/or transient neurological dysfunction (e.g. headache and dizziness) (3). These clinical features are typically accompanied by minimal impairment on objective neurological measures, e.g. Glasgow Coma score of 13–15 (4). Within a sports context, the Concussion in Sport Group has developed a conceptual definition of sports-related concussion as a “traumatic brain injury caused by a direct blow to the head, neck, or

body resulting in an impulsive force being transmitted to the brain that occurs in sports and exercise-related activities (5). Clinical symptoms resulting from sports-related concussions may or may not involve loss of consciousness and will not present on standard clinical neuroimaging (computed tomography or magnetic resonance imaging T1- and T2-weighted images) as an abnormality (5). Research shows that while most post-concussive symptoms resolve after several months (6–9), a subset of individuals experience prolonged neurobehavioral symptoms that affect quality of life and daily functioning (e.g. headache, dizziness, fatigue, irritability, concentration difficulties, memory impairment, and disrupted emotional functioning) (10). Since mTBI diagnoses are often predicated on clinical symptom criteria, it is imperative to determine which initial presentation features of mTBI have prognostic significance for identifying those at high risk for long-term functional impairment.

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Football athletes

Due to frequent head collisions that occur in American football and the pressure to return to play after injury, identifying prognostic markers for mTBI-related health outcomes is particularly relevant in football players. Over 70% of college-level football players have experienced acute symptoms of mTBI at some point in their athletic career, while 39% report having sustained an mTBI with LOC (mTBI + LOC) (11). Within a professional American football sample, 58% of players returned to play less than seven days after LOC (12), and LOC at time of injury was strongly associated with poor cognition-related quality of life, even decades after retiring from play (13).

Repeated mTBI exposure

While most individuals recover from a single mTBI within four weeks, repeated mTBI exposure is associated with prolonged recovery and chronic symptoms in adolescent and young adult athlete populations (14). There is no clear evidence regarding changes in recovery time upon repeated mTBI in professional athlete populations. Although controversial, evidence suggests a link between repeated mTBI and chronic neurobehavioral symptoms. For example, in rodents, sustaining multiple mTBIs is linked with neuropathology that is distinct from isolated exposure (15–18), including increased long-term neuroinflammation and white matter degradation (16,19). In humans, athletes with a history of multiple mTBIs perform significantly worse on neurocognitive assessments (20–22) and are at much higher risk of long-term mild cognitive impairment when compared to a control group of athletes (23).

Loss of consciousness

While repeated mTBI exposures may have compounding effects, LOC at time of injury may independently impact long-term neurobehavioral outcomes (24–27). Although only a subset of mTBIs are associated with LOC, it is used as a diagnostic criterion to distinguish mTBI from moderate-to-severe TBI (28). LOC indicates greater severity of injury; neuroimaging reveals that mTBI + LOC has increased neuropathological biomarkers of dementia (e.g. amyloid- β load, gross infarctions; microinfarctions) (25) and decreased connectivity within prefrontal white matter tracts (26,27). Importantly, groups with more lifetime mTBI + LOC events show both decreased white matter connectivity and decreased cognitive flexibility on neurocognitive tests, indicative of long-term deficits in executive functioning (26). Although there is compelling evidence for the mechanisms by which mTBI + LOC may affect long-term clinical symptoms and recovery trajectory after mTBI, LOC's utility as a prognostic marker remains controversial (21,24). It is imperative to consider how LOC at time of injury may uniquely impact lifetime neurobehavioral symptomatology.

Depression and insomnia

Critically, mTBI exposure is also associated with long-term psychiatric symptoms. Sustaining one or more mTBIs increases risk of depression at >11 months post-injury (29,30), and those with a history of multiple mTBIs or mTBI + LOC have an even higher incidence of depression (31–37). The relationship between mTBI + LOC and increased depressive symptoms may be partly explained by decreased white matter connectivity in mTBI-exposed samples (26,27,34); a systematic review by Medeiros et al. (2022) report associations between DTI measures of white matter damage and depression severity after TBI (38). Major Depression is also a significant predictor of neurobehavioral symptoms (39,40), suggesting that neurobehavioral symptoms may be attributable to post-mTBI onset of depression. Despite these findings, the potential mediating effect of depression on the relationship between mTBI + LOC and long-term neurobehavioral symptoms has not been explored.

Nearly all patients with depression report sleep disturbance, and insomnia is associated with an increased risk for depression, suggesting a bi-directional relationship between the two (41). Long-term sleep disturbance is a prevalent sequelae following mTBI exposure (42), with exposure to mTBI + LOC further increasing risk (43–45). In adolescents, disrupted sleep following mTBI exposure is associated with a three-to-four-fold increase in recovery time (46). Insomnia has also been independently linked to deficits in cognition (47,48), including impairments in working memory and episodic memory (49). The impact of sleep disturbance on neurobehavioral symptoms has only briefly been examined post-mTBI, where sleep quality accounted for 32% of the variance in neurobehavioral symptoms post-injury (50). Pronounced sleep disruption after mTBI + LOC could explain the relationship between mTBI + LOC and long-term neurobehavioral symptoms. Prior research has documented the mediating effect of sleep quality on risk for depression in mTBI-exposed individuals (51), however, the effect of mTBI + LOC on this relationship remains unexamined.

It is important to note that many of the existing studies examining the relationship between mTBI and long-term depression, insomnia, and neurobehavioral symptoms are conducted in Veteran and community samples and may not generalize to athlete samples. Evidence suggests that the mechanism of injury (e.g. blast versus non-blast) and context of injury (e.g. in conjunction with combat-related trauma) may influence pathology and outcomes (52–54).

Current study

In the current study, we investigated the long-term impact of repeated mTBI with and without LOC on neurobehavioral symptoms in National Collegiate Athletic Association (NCAA) Division I former college-level and professional football athletes. Consistent with prior studies, we hypothesized that the number of mTBI + LOC events is associated with long-term neurobehavioral symptoms. Less is known, however, about whether the effects of mTBI + LOC events are still associated with neurobehavioral symptoms when controlling for the effects of depression and insomnia. Considering

depression and insomnia are directly associated with mTBI and are independently associated with neurobehavioral symptoms (43,55–57), we predicted that mTBI + LOC would predict neurobehavioral symptoms indirectly through both depression and insomnia. Indirect effects may suggest insomnia and depression as potential mediators in the relationship, although further longitudinal investigation is needed. The practical consequences of this investigation will guide the implementation of short-term screening of depression and insomnia in high-risk patients following sports-related mTBI, for example, in those who lost consciousness at time of injury.

Methods

Participants

A sample of 211 of Male former contact and non-contact athletes were recruited via social media, recruitment flyers, and recruitment e-mails to participate in a larger neuroimaging study. Participants were primarily contacted through NFL alumni chapters, university athletic departments, and NCAA football team offices. For the present study, 30 former NCAA Division I non-contact sport athletes were excluded from the sample. One hundred and seventy-seven former elite football athletes between the ages of 27 and 85 ($M = 54.1$, $SD = 14.7$) were included. The sample consisted of former collegiate (NCAA Division I; $n = 65$) and professional-level (National Football League [NFL]; $n = 112$) football players. Participants completed a health survey administered virtually over Zoom. The questionnaire assessed mTBI history and included several measures of general health outcomes.

All participants were fluent in English, had no history of neurological injury or disease and no substance use disorder. Participants provided informed consent, and all study procedures were approved by the Duke University Institutional Review Board and the George Washington University Institutional Review Board. For full inclusion and exclusion criteria, see Appendix A.

History of mTBI and mTBI+LOC

Five retrospective questions were used to determine (1) number of mTBI exposures and (2) number of mTBI + LOC events during each phase of the participant's athletic career (Table 1). Participants responded on a scale of 0–30 (number of mTBI exposures) for each question. Total number of sports-related mTBI exposures and mTBI + LOC events were determined by summing the response from these 5 questions, with total scores

ranging from 0 to 120 mTBI exposures ($M = 7.9$, $SD = 14.1$) and 0 to 8 mTBI + LOC events ($M = 1.1$, $SD = 1.5$), out of a possible 150. To address outliers in a limited sample size, we performed 90% Winsorization on both mTBI exposures and mTBI + LOC events, whereby all data below the 5th percentile were set equal to the 5th percentile (mTBI = 0, mTBI + LOC = 0), and data above the 95th percentile set to the 95th percentile (mTBI = 12.9, mTBI + LOC = 3.9) (58). A total of 26 mTBI observations and 14 mTBI + LOC observations were adjusted for Winsorization. After Winsorization, mTBI exposures ranged from 0 to 12.9 ($M = 5.3$, $SD = 4.42$), and mTBI + LOC events ranged from 0 to 3.9 ($M = 1.03$, $SD = 1.3$).

Neurobehavioral symptoms

The Neurobehavioral Symptom Inventory (NSI) is a 22-item questionnaire assessing the current severity of post-concussive symptoms associated with mTBI, including dizziness, poor concentration, and slowed thinking. All items were measured on a five-point scale, with self-reported symptom ratings between 0 (absent) and 4 (severe) (59). The NSI has excellent internal reliability ($\alpha = 0.93$) and includes a total score (sum of all items) and subscale scores for three symptom categories: cognitive, affective, and somatic (60). In addition, the Validity-10 scale can be derived to screen for potential overreporting of symptoms by summing ten NSI items rarely endorsed by individuals exposed to TBI (61). As recommended for clinical utility (62), we excluded participants with a Validity-10 score >18 ($n = 4$), which may suggest symptom exaggeration (63).

Depression

Current depressive symptom severity (symptoms experienced within 2 weeks of study participation) was measured using the Beck Depression Inventory II (BDI-II), a 21-item self-report questionnaire (64). Participants responded using a four-point scale (0 to 3), with 0 and 3, respectively, indicating lowest and highest severity of symptoms. Total severity scores were determined by summing the 21 items.

Insomnia

Current insomnia symptom severity (symptoms experienced within 2 weeks of study participation) was measured using a modified Insomnia Severity Index (ISI) (65). The ISI is a 7-item questionnaire measuring the severity of sleep disturbance and its related waking symptoms. Participants

Table 1. Health Survey questions on athlete sport-related concussion history

Question
History of mTBI
(1)'How many sports-related concussions did you have before high school?'
(2)'How many times did you lose consciousness ("knocked out") due to sports-related concussion?'
(3)'In college, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'
(4)'In the armed forces, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'
(5)'In your professional career, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'
History of mTBI+LOC

- (1)'Before high school, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'
- (2)'In high school, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'
- (3)'In college, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'
- (4)'In the armed forces, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'
- (5)'In your professional career, how many times did you lose consciousness ("knocked out") due to sports-related concussion?'



respond using a 5-point scale (0–4) (66,67). By error, a modified version of ISI was administered for all participants in this study, which included 6 of the 7 items [excluded item: *'To what extent do you consider your sleep problem to INTERFERE with your daily functioning (e.g. daytime fatigue, mood, ability to function at work/daily chores, concentration, memory, mood, etc.) CURRENTLY?'*]. Total severity score was determined by summing the 6 items.

Statistical analyses

Statistical modeling was carried out using R (v. 4.2.1). First, bivariate relationships between mTBI and mTBI + LOC with neurobehavioral, depression, and insomnia symptom severity were evaluated using linear regression. In all cases, diagnostic tests were conducted to ensure that all data met the necessary assumptions for linear regression. Descriptions and results of diagnostic tests can be found in Appendix B. To follow-up on significant effects of mTBI + LOC and neurobehavioral symptom severity, we then assessed mTBI + LOC as a predictor of neurobehavioral symptom severity, indirectly through depression and insomnia. Analysis was performed using a parallel structural equation model with the package *lavaan* (68). For all indirect and total effects, 95% confidence intervals (CI) were estimated. CIs excluding 0 were considered statistically significant. The structural equation model was tested using 10,000 bootstraps resampled with replacement using bias-corrected confidence estimate.

Results

Sample characteristics

In the total sample ($n = 177$), race/ethnicity composition was 32% Black, 61% white, 1% Hispanic/Latino, 1% Asian, 4% multi-racial, and <1% other or not reported. Ages ranged from 27 to 85 years ($M = 54.1$, $SD = 14.7$). All participants completed at least some college. For sample characteristics, see Table 2.

Results of one-way ANOVAs indicated neurobehavioral symptoms did not significantly differ by race ($f = .015$, $p = 0.9$). There were no associations between neurobehavioral symptoms and participant age. Neurobehavioral symptoms differed by at least two maternal education groups ($f = 4.04$, $p = 0.05$). Neurobehavioral symptoms differed between at least two participant education groups ($f = 10.34$, $p = 0.001$). Depressive symptoms did not significantly differ by race ($f = 0.23$, $p = 0.99$) or maternal education ($f = 0.24$, $p = 0.62$). There was a significant correlation between depressive symptoms and age ($r = -0.18$, $p = 0.01$). Depressive symptoms differed significantly between at least two participant education groups ($f = 4.7$, $p = 0.03$). Insomnia did not significantly differ by race ($f = 1.09$, $p = 0.36$), education ($f = 0.36$, $p = 0.54$), or maternal education ($f = 2.47$, $p = 0.12$). There was no association between insomnia and age ($r = -8.9e-4$, $p = 0.36$). For mean NSI scores by group, see Table 3.

Bivariate associations

To evaluate the direct effect of the number of mTBI exposures on neurobehavioral symptoms, we conducted a linear regression while covarying for age, race, education, and maternal education. When controlling for these demographic factors, the association between the number of mTBI exposures and neurobehavioral symptoms was not significant ($B = 0.21$, $SE = 0.18$, $p = 0.23$).

When the same linear regression was run to determine if number of mTBI + LOC events predicted neurobehavioral symptoms while covarying for age, race, education, and maternal education, and number of mTBI exposures, there was a significant effect of mTBI + LOC ($B = 2.27$, $SE = 0.64$, $p = <.001$).

We then conducted multiple linear regressions to explore the proposed structural equation model. As hypothesized, there were significant direct effects of number of mTBI + LOC events on depression ($B = 1.26$, $SE = 0.44$, $p = 0.005$), and insomnia ($B = 1.34$, $SE = 0.35$, $p = <.001$) when controlling for age, race, education, and maternal education. Additionally, both depression ($B = 0.91$, $SE = 0.09$, $p = <.001$) and insomnia ($B = 1.12$, $SE = 0.12$, $p = <.001$) significantly predicted neurobehavioral symptoms.

Indirect effects
Results of exploratory structural equation analysis suggest mTBI + LOC predicted neurobehavioral symptoms indirectly

Table 2. Sample demographics and symptom characteristics

	NCAA Division 1 (n = 69)	Professional (n = 108)	Total Sample (n = 177)
Age (SD)	47(15.4)	59(12.2)	54(14.7)
Race			
White	63.8%	57.9%	60.9%
Black	27.5%	34.6%	32.1%
Mexican	1.4%	-	<1%
Other Latino	-	<1%	<1%
Asian	1.4%	<1%	1.1%
More than one	2.9%	4.7%	4%
Other	-	<1%	<1%
Not Reported	2.9	1.7%	1.7%
Education			
Some college	1.4%	10.1%	6.8%
Associate's	-	<1%	<1%
Bachelor's	52.2%	55.6%	54.2%
Postgraduate	46.4%	33.3%	38.4%
Mom Education			
8 th grade or less	5.8%	10.2%	8.5%
Some HS	1.4%	4.6%	3.4%
HS graduate	28.9%	45.3%	38.9%
Some college	5.8%	8.3%	7.3%
Associate's	2.9%	7.4%	5.6%
Bachelor's	34.8%	12.9%	21.5%
Postgraduate	20.3%	11.1%	14.7%
LOCs (SD)	0.79(1.2)	1.3(1.6)	1.1(1.5)
mTBIs (SD)	6.9(14.1)	8.7(14.2)	7.9(14.1)
Winsorized LOCs(SD)	0.78(1.2)	1.2(1.3)	1.0(1.3)
Winsorized mTBIs(SD)	3.0(3.9)	5.9(4.6)	5.3(4.4)
NSI (SD)			
Affective	5.6(5.0)	4.3(4.4)	4.8(4.7)
Somatic	2.3(2.7)	3.5(4.6)	3.3(4.0)
Cognitive	2.8(2.8)	2.5(3.2)	2.6(3.1)
Total NSI	10.68(8.7)	10.3(11.1)	10.5(10.2)
BDI (SD)	8.2(7.3)	6.5(6.9)	7.2(7.1)
ISI (SD)	6.4(5.5)	6.5(5.9)	6.5(5.8)

Table 3. Demographic-level descriptive statistics for neurobehavioral, depression and insomnia questionnaires

	NSI(SD)	BDI(SD)	ISI(SD)
Race			
White	10.7 (10.5)	7.4(8.1)	6.1(5.6)
Black	10.5 (9.8)	7.1(5.6)	7.5(5.9)
Mexican	13.0 (NA)	9.0(NA)	5.0(NA)
Other Latino	2.0 (NA)	0.0(NA)	2.0(NA)
Asian	4.0 (5.7)	6.5(9.2)	1.5(2.1)
More than one	13.0 (12.7)	6.3(4.3)	8.3 (7.8)
Other	4.0 (NA)	3.0(NA)	0(NA)
Education			
Some college	14.2 (17.0)	9.7(8.9)	7.0(6.1)
Associate's	2.0 (NA)	4.0(NA)	0.0(NA)
Bachelor's	11.6 (10.2)	7.9(7.8)	6.8(5.8)
Postgraduate	7.9 (7.8)	5.7(5.9)	6.0(5.7)
Mom Education			
8 th grade or less	13.5 (12.4)	7.3(6.4)	8.0(7.8)
Some HS	10.3(8.0)	6.3(5.6)	6.5(7.6)
HS graduate	12.0(12.6)	7.1(7.9)	6.9(6.2)
Some college	10.3(6.5)	7.4(6.9)	6.1(5.4)
Associate's	5.8(4.3)	2.7(4.4)	7.4(6.3)
Bachelor's	8.1(7.0)	7.0(7.8)	5.5(4.4)
Postgraduate	10.0(8.6)	8.6(5.6)	5.7(6.7)

through both depression ($B = 0.85$, 95% CI = [0.27, 1.52]) and insomnia ($B = 0.81$, 95% CI = [0.3, 1.4]). Additionally, the direct effect of mTBI + LOC group on neurobehavioral symptoms became non-significant ($B = 0.78$, SE = 0.45, $p = 0.08$) when depression and insomnia were added to the model (Figure 1). Significant indirect effects suggest that insomnia and depression should be further explored as potential mediators in the relationship between mTBI + LOC and neurobehavioral symptoms, using longitudinal data.

Overlapping content between the BDI-II, ISI, and NSI questionnaires could contribute to associations between the measures. Specifically, the BDI-II and NSI both include items

about indecisiveness, fatigue, sadness, and irritability. The ISI and NSI both include an item about difficulty falling asleep. See Table 4 for correlations between measures. As a result, we ran further exploratory analyses using the three subscales of the NSI (affective, somatic/sensory, and cognitive) to examine more granular indirect effects.

Affective subscale

Results of exploratory mediation analysis revealed mTBI + LOC predicted affective neurobehavioral symptoms indirectly through both depression ($B = 0.39$, 95% CI = [0.15, 0.65]) and insomnia ($B = 0.44$, 95% CI = [0.17, 0.71]). The direct effect of mTBI + LOC on affective symptoms became non-significant when depression and insomnia were added to the model ($B = 0.24$, SE = 0.17, $p = 0.15$) (Figure 2).

Somatic/Sensory subscale

Results of exploratory mediation analysis revealed mTBI + LOC predicted somatic/sensory neurobehavioral effects indirectly through insomnia ($B = 0.25$, 95% CI = [0.06, 0.49]), but not depression ($B = 0.12$, 95% CI = [-0.04, 0.34]). In addition, the direct effect of number of mTBI + LOC events on somatic/sensory symptoms is non-significant ($B = 0.43$, SE = 0.25, $p = 0.8$) when insomnia was added to the model (Figure 3).

Cognitive subscale

Results of exploratory mediation analysis revealed mTBI + LOC predicted cognitive neurobehavioral symptoms indirectly through both depression ($B = 0.33$, 95% CI = [0.09, 0.65]) and

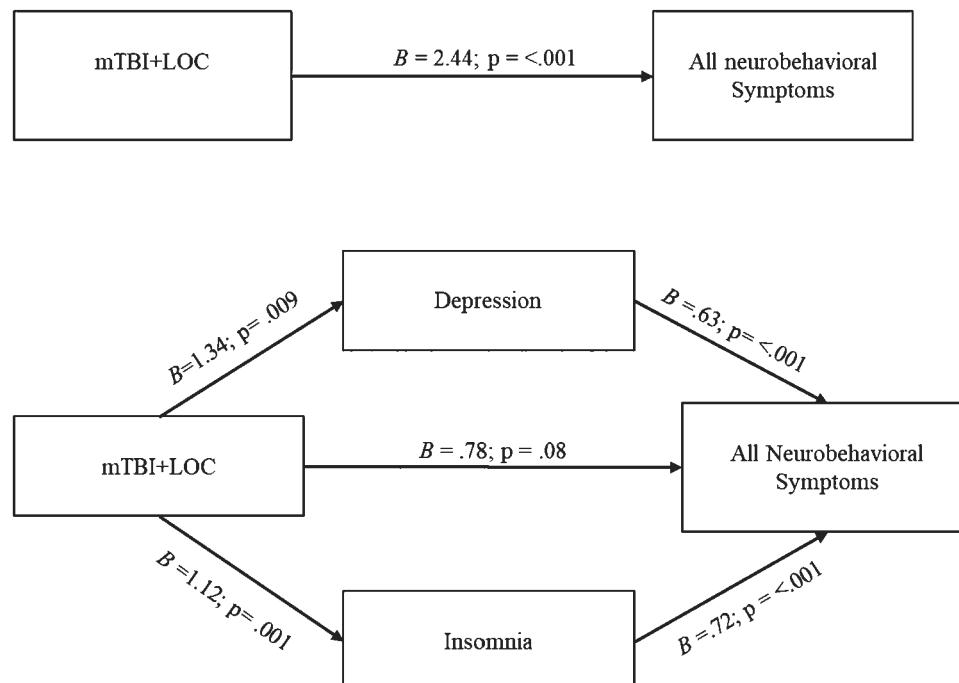


Figure 1. mTBI+LOC significantly predicts affective symptoms indirectly through depression ($B = 0.39$, 95% CI = [0.15, 0.65]) and insomnia ($B = 0.44$, 95% CI = [0.17, 0.71]).

Table 4. Correlations between symptom measures

		Correlations	1	2	3	4	5	6
1	NSI Total Score		—					
2	NSI Affective Subscale		.91**	—				
3	NSI Somatic/Sensory Subscale		.83**	.60**	—			
4	NSI Cognitive Subscale		.85**	.71**	.55**	—		
5	BDI-II Total Score		.65**	.68**	.33**	.68**	—	
6	ISI Total Score		.63**	.69**	.42**	.48**	.47**	—

**Correlation is significant at the 0.01 level (2-tailed).

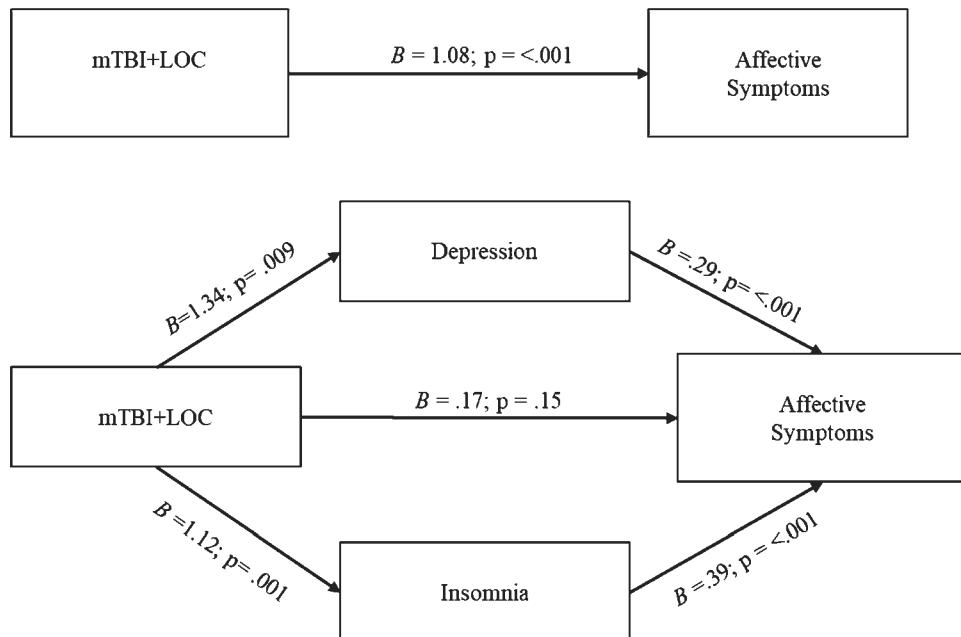


Figure 2. mTBI+LOC significantly predicts somatic/sensory symptoms indirectly through insomnia ($B = 0.25$, 95% CI = [0.06, 0.49]), but not depression ($B = 0.12$, 95% CI = [-0.04, 0.34]).

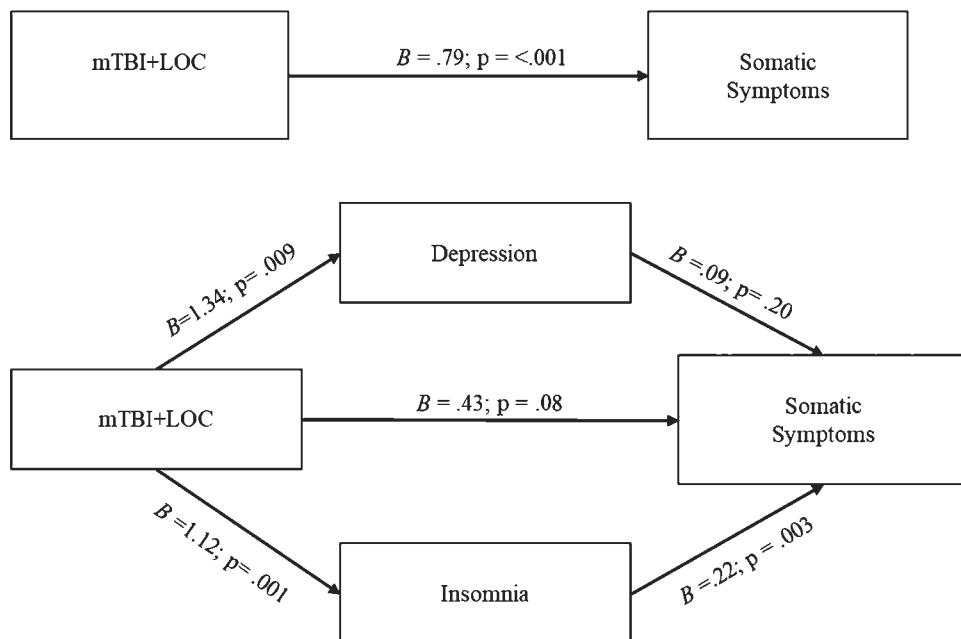


Figure 3. mTBI+LOC significantly predicts cognitive symptoms, indirectly through depression ($B = 0.33$, 95% CI = [0.09, 0.65]) and insomnia ($B = 0.13$, 95% CI = [0.04, 0.25]).

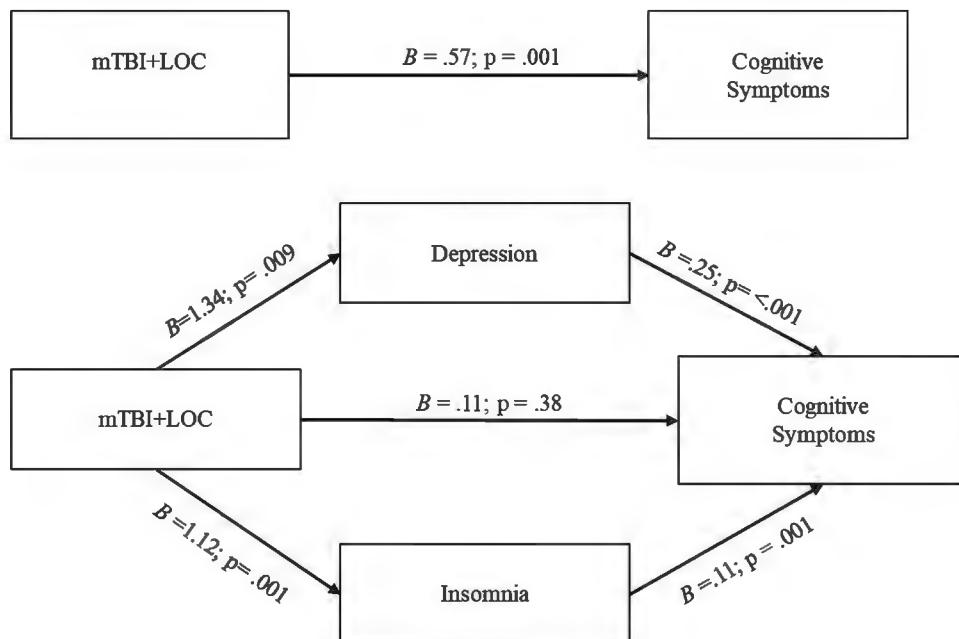


Figure 4. mTBI+LOC significantly predicts cognitive symptoms, indirectly through depression ($B = 0.33$, 95% CI = [0.09, 0.65]) and insomnia ($B = 0.13$, 95% CI = [0.04, 0.25]).

insomnia ($B = 0.13$, 95% CI = [0.04, 0.25]). In addition, the direct effect of mTBI + LOC on cognitive symptoms is non-significant ($B = 0.11$, SE = 0.13, $p = 0.38$) when depression and insomnia are added into the model (Figure 4).

Discussion

In a sample of former collegiate and professional football athletes, there was a direct effect of mTBI + LOC – but not mTBI – exposures on current neurobehavioral, depression, and insomnia symptoms. Further analyses revealed mTBI + LOC predicted neurobehavioral symptoms indirectly through both insomnia and depression. Additionally, the direct effect of mTBI + LOC on neurobehavioral symptoms became non-significant when depression and insomnia were added into the model. Indirect effects suggest that insomnia and depression may mediate the relationship between mTBI + LOC and neurobehavioral symptoms, however, we are limited by the cross-sectional nature of our data. In clinical science, mediation analysis on cross-sectional data is limited because causal inferences are not possible (69). Due to the range of time between mTBI exposure ($M = 29.07$, $SD = 13.91$) and mTBI + LOC exposure ($M = 32.85$, $SD = 15.19$) and the timeframe for reporting of depression, insomnia, and neurobehavioral symptoms, we can conclude that mTBI exposure precedes the symptoms reported in this study, providing strong justification for further mediation analyses. Future studies should examine longitudinal effects to provide more insight on the causality of these associations.

In exploratory analyses of subscale measures from the NSI, a number of mTBI + LOC events predicted cognitive and affective neurobehavioral symptoms indirectly through both depression and insomnia. In both models, the direct effect became non-significant when depression and insomnia were included in the model, suggesting a potential full mediation. In

addition, a number of mTBI + LOC events predicted somatic/sensory neurobehavioral symptoms indirectly through insomnia alone. It is important to note that these findings are specific to mTBIs that *include* loss of consciousness, as the number of mTBIs alone did not predict neurobehavioral symptoms.

Prior research reveals an association between mTBI + LOC and increased sleep disturbance, as well as mediating effects of mTBI-related sleep disturbance on depression symptoms (46). Our findings expand on the unique effect of mTBI + LOC on depression and insomnia symptoms, and their potential mediating influence on neurobehavioral symptoms. The neurocognitive effects of both insomnia and depression are often acute; neurocognitive effects of disrupted sleep may be reversed with as little as one eight-hour sleep period (70), while cognitive functioning typically improves following treatment for depression (71). This underscores the importance of frequent screening for depression and insomnia in patients who experience LOC at the time of mTBI, as they may be a useful target of intervention to alleviate neurobehavioral symptoms.

Understanding the neurophysiological mechanisms associated with these findings will provide further insight toward prevention of persistent post-mTBI symptoms. Sleep is regulated by complex neurophysiological mechanisms, with various neurohormones, neurotransmitters, and brain regions facilitating the sleep-wake cycle (57). This cycle is crucial for maintaining metabolic homeostasis, involving the glymphatic system which removes neurotoxic waste from intercellular space (72). It is possible that mTBI + LOC damages neural pathways crucial to this system, disrupting this maintenance cycle. Elevated biomarkers of neurodegeneration (neurofilament light) have been identified among sleep-disrupted mTBI patients in comparison to non-sleep-disrupted mTBI patients though the direction of this relationship remains unclear (73).

Although the neurobiological underpinnings of depressive symptoms are not fully understood, glymphatic dysfunction

from disrupted sleep may also influence depressive symptoms (74). Some studies suggest that depression is associated with increased inflammation, which is particularly relevant to the distinct inflammatory response associated with mTBI exposure (75,76). Our present study suggests a need for further investigation toward the neurophysiological effects that mTBI + LOC has on mechanisms for sleep regulation and depression.

We also found that insomnia and depression may fully mediate the relationship between mTBI + LOC and subjective cognitive post-concussive symptoms. This expands on prior findings that mTBI + LOC, especially in patients exposed to repetitive head impact, corresponds to worse prolonged cognitive function and increased depression severity (77). It is also imperative for future work to include more objective measures of cognition, although we excluded participants with possibly exaggerated symptoms based on established cutoffs (61), additional study is needed to determine whether this relationship is specific to subjective cognitive deficits or also translates to objective neuropsychological test performance.

Limitations

This study has several limitations. An item from the ISI was excluded, potentially compromising replicability. Additionally, while the ISI has been validated in clinical settings (78), polysomnography may be more reliable for evaluating sleep-related disorders (79).

We are further limited by the cross-sectional nature of our data and thus, claims regarding causality are not possible. These results are part of an ongoing five year, longitudinal effort to evaluate the effects of high-level football participation on brain health, while the present results are cross-sectional, they provide strong justification for investigating the mediational effects of depression and insomnia on the relationship between mTBI + LOC and long-term neurobehavioral symptoms. Future analyses within this cohort, as well as other large-scale prospective longitudinal studies (e.g. TRACK-TBI) will be valuable for more reliably mapping pathways between mTBI + LOC, depression and insomnia symptoms, and neurobehavioral symptoms (80,81).

While current neurobehavioral, depressive, and sleep-related symptoms were measured at time of participation, previous (pre-mTBI) symptomatology was not assessed, and thereby not controlled for, in the current study. History of neurobehavioral, depressive, and sleep-related symptoms, in conjunction with mTBI and mTBI + LOC events could differentially impact long-term outcomes. Additionally, other previous and current morbidities were not assessed. Evidence suggests that factors such as post-traumatic stress, psychiatric symptoms, and headaches/migraines can impact long-term outcomes following mTBI (82,83). While outside of scope for the current study, future studies may consider administering a battery of medical and psychiatric symptom assessments or accessing medical records in order to control for important pre-mTBI and post-mTBI morbidities.

Medical records were not accessed to confirm mTBI history in participants and the reliability of self-reported mTBI history is controversial. While it is well established that current

athletes are motivated to underreport or not disclose mTBI symptoms in order to return to play, it is unclear if retired athletes retrospectively underreport mTBI history. One study in retired NFL athletes found moderate reliability of self-reported mTBI history (with and without LOC) across multiple timepoints of instrument administration (84). To increase validity of self-report data, our study provided a definition of 'concussion' to all participants, as suggested by prior literature (85,86).

Additionally, non-sports related mTBI was not assessed in the study, and subjects may have had mTBI + LOC events from non-sporting events (e.g. motor vehicle collision) that were not included in the current analyses.

Despite limitations, the current study makes a novel contribution to our understanding of the relationship between mTBI + LOC and long-term neurobehavioral outcomes. Findings reveal that LOC at time of injury has prognostic significance for identifying those at high risk for long-term impairment. Further, this study is among the first to provide empirical support that depression and insomnia may mediate relationship between mTBI + LOC and long term neurobehavioral symptoms. However, further investigating the relationships amongst LOC at mTBI exposure, depression, insomnia, and neurobehavioral symptoms is necessary to inform clinical care for mTBI patients. Longitudinal analysis is needed to determine true statistical mediation in the relationship between mTBI + LOC and long-term outcomes. Future research should utilize psychiatric and medical assessments to control for comorbid symptoms. Examining potential sex differences is also needed, as women are underrepresented in sport-concussion studies but may have greater prevalence of sports concussion and subsequent impact on neurobehavioral symptoms and cognitive function (87).

Conclusions

Overall, these findings provide insight into the factors influencing post-LOC neurobehavioral symptoms. Consistent with prior research, we found that a history of mTBI + LOC is directly associated with increased neurobehavioral symptom endorsement at >11 months post-injury. However, there has been little research investigating the factors that may influence this relationship. In the current study, mTBI + LOC predicted total neurobehavioral symptoms indirectly through depression and insomnia. Notably, the direct effect of mTBI + LOC on total neurobehavioral symptoms became non-significant when depression and insomnia were added into the model. Overall, findings support early intervention and symptom monitoring for depression and insomnia following mTBI + LOC exposure, as these factors may be the key indicators of poor functional outcomes.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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