California Legislature

Assembly Committee on Arts, Entertainment, Sports and Tourism

2023 Youth Tackle Football

Improving Athlete Safety

Committee Members

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Laura Friedman
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California State Assembly

ARTS, ENTERTAINMENT, SPORTS, AND TOURISM

1020 N Street, Suite 152
(916) 319-3450

SHARON QUIRK-SILVA
CHAIR

Informational Hearing:
Youth Tackle Football – Improving Athlete Safety

October 3, 2023 - 11:00 am to 1:00 pm
State Capitol – Room 444 – Sacramento, California

Opening Remarks

Honorable Sharon Quirk-Silva, Chair
Members of the Committee

I. Latest research and medical concerns on concussions and head impacts

Dr. Chris Nowinski, PhD, Co-Founder and CEO, Concussion Legacy Foundation
Dr. David Camarillo, PhD, Associate Professor of Bioengineering, Mechanical Engineering and Neurosurgery at Stanford University
Dr. Stella Legarda, MD, President, California Neurology Society (CNS)

II. Impacts of safety measures from AB 1 (Cooper) of 2019

Steve Famiano, California Youth Football Alliance
Ron White, California Youth Football Alliance

III. Athletes and family impacted by CTE and head impacts

Jan Franklin, Jason Franklin's mother, Los Angeles
Mike Haynes, President, Mike Haynes and Associates, Pro Football Hall of Fame Class of 1997 (Player)
Pamela Thakur, Thakur Law Firm

Public Comments

Closing Remarks

Informational Hearing

Assembly Committee on Arts, Entertainment, Sports, and Tourism
Assemblymember Sharon Quirk-Silva, Chair

Youth Tackle Football – Improving Athlete Safety

SPEAKER BIOGRAPHIES

Dr. Chris Nowinski, Co-Founder and CEO, Concussion Legacy Foundation

Chris Nowinski, PhD, is co-founder and CEO of the Concussion Legacy Foundation, a non-profit organization leading the fight against concussions and Chronic Traumatic Encephalopathy (CTE) and dedicated to improving the lives of those impacted.

An All-Ivy Harvard football player-turned WWE professional wrestler-turned neuroscientist, Chris discovered the concussion crisis the hard way. A 2003 kick to the chin in a WWE match ended his career, causing Post-Concussion Syndrome and sending him to the office of his eventual CLF co-founder Dr. Robert Cantu.

Chris realized this lack of awareness among athletes, coaches, and even medical professionals not only cost him his career, but also threatened the health and well-being of athletes of all ages. The lessons Chris learned from Dr. Cantu lit a spark that inspired him to commit his life to serving patients and families affected by brain trauma. You can watch his TED Talk about this journey here.

Dr. Nowinski earned his doctorate in Behavioral Neuroscience from Boston University School of Medicine and has authored more than 30 scientific publications. VICE Sports called him "the man most responsible for making CTE part of the national conversation," and Sports Illustrated said, "It is Nowinski's figure which looms behind the doctors and the headlines and the debate roiling over sports' newfound commitment to minimizing head trauma." Nowinski serves on the NFL Players Association Mackey-White Health & Safety Committee, the Ivy League Concussion Committee, the Positive Coaching Alliance National Advisory Board, and as an advisor to All-Elite Wrestling.

https://concussionfoundation.org/about/staff/chris-nowinski

David Camarillo

David B. Camarillo is Associate Professor of Bioengineering, (by courtesy) Mechanical Engineering and Neurosurgery at Stanford University. Dr. Camarillo holds a B.S.E in Mechanical and Aerospace Engineering from Princeton University, a Ph.D. in Mechanical Engineering from Stanford University and completed postdoctoral fellowships in Biophysics at the UCSF and Biodesign Innovation at Stanford. Dr. Camarillo worked in the surgical robotics industry at Intuitive Surgical and Hansen Medical, before launching his laboratory at Stanford in

2012. His current research focuses on precision human measurement for multiple clinical and physiological areas including the brain, heart, lungs, and reproductive system. Dr. Camarillo has been awarded the Hellman Fellowship, the Office of Naval Research Young Investigator Program award, among other honors including multiple best paper awards in brain injury and robotic surgery. His research has been funded by the National Institutes of Health, National Science Foundation, Department of Defense, as well as corporations and private philanthropy. His lab's research has been featured on NPR, the New York Times, The Washington Post, Science News, ESPN, and TED.com as well as other media outlets aimed at education of the public.

https://profiles.stanford.edu/david-camarillo

Maris "Stella" B. Legarda, MD

Dr. Stella Legarda is a board certified pediatric neurologist, clinical neurophysiologist, and epilepsy specialist practicing in Monterey, California since 2013. Dr. Legarda has held past academic appointments at the University of Florida College of Medicine (as Assistant Professor) and at Georgetown University College of Medicine (as Associate Professor). In 2013 Dr. Legarda left academia to join her current multispecialty group at Montage Health in Monterey where her research interests in applied neuromodulation compel her practice to include adults with epilepsy and other acquired central nervous system disorders (traumatic brain injury, stroke, Parkinson disease, encephalopathies, and dementias). She has peer-reviewed publications on a range of subjects namely epilepsy, clinical neurophysiology, concussion, headache, Parkinson disease, and the value of infralow frequency (ILF) brain training in neurology practice. Dr. Legarda penned a chapter: "Remediating Brain Instabilities in a Neurology Practice" in the second edition of 'Restoring the Brain' (Ed. Kirk, H, 2020). In 2020 she received two Physician Scholar grant awards from the Montage Health Foundation to evaluate the treatment response of patients with post-concussion syndrome to ILF neuromodulation. Dr. Legarda is currently President of the California Neurology Society.

https://californianeurologysociety.org/board-of-directors

Steve Famiano

Steve Famiano is a parent, coach, administrator, and advocate with more than 20 years of experience helping develop and grow the sport of youth tackle football in Southern California and across the State. He helped to take a financially struggling program in his local area of Apple Valley, Ca to one of the strongest and most successful programs in Southern California for almost 18 years. During this time, he co-founded a youth football conference consisting of 12+ teams representing the High and Low Desert regions. City teams which included his local league, Victorville, Yucca Valley, Desert Hot Springs, Hesperia, and more. This conference, with Steve's leadership, became one of the first in California to utilize online player safety tracking and player safety statistics, eliminating the use of paper records.

In 2018, Steve stepped away from local youth tackle football to help create the Save Youth Football - California Coalition, a term coined by Steve that is now used by other States as a

cohesive name for those groups looking to preserve youth tackle football across America. In 2019, Steve continued his work in the sport by co-founding the California Youth Football Alliance, a registered California non-profit dedicated to honoring, improving and advancing the sport across the State. Steve and his partners joined with former Assemblyperson Jim Cooper to craft and pass the Nation's first and only youth tackle football safety bill, The California Youth Football Act (AB 1, Chapter 158, Statutes of 2019). Steve also consults leagues, coaches and parents looking for direction on the best practices of the sport to include the implementation of AB 1.

https://www.cayfa.org/our-board

Ron White

Ron White is the President and co-founder of the California Youth Football Alliance. He also founded the Golden Empire Youth Tackle Football, Inc. in 1998, and has acted as the group's President and Commissioner. He co-founded the California Save Youth Football Coalition, was a sponsor and consultant on the California Youth Football Act, and consulted on bills on interscholastic athletic emergency action plans and heat illness. Mr. White was a member of the Kern County Concussion Consortium from 2016-2020, a football coach in the Kern High School District from 1991-1995, and was a California Pop Warner coach and board member from 1985-1986.

"The purpose of the California Youth Football Alliance is to advance the great sport of youth tackle football relentlessly and continually by honoring our past, improving our present, and advancing our future so that more generations of student-athletes, coaches, and communities can experience the intellectual, emotional, social, and physical developmental benefits of the sport."

https://www.cayfa.org/our-board

Jan Franklin

Jan's son and only child Jason Franklin was a scout team linebacker for Arizona State University. He was known by friends and teammates for his work ethic, attitude, and dedication. While in college, Jason suffered three documented concussions. After college, Jason became increasingly angry, agitated, and began distancing himself from others. In 2018, Jason took his own life at age 26. After his death, a coroner who was familiar with Jason's football history suggested that his family donate his brain to the Boston University Brain Bank for analyisis, where Dr. Ann McKee diagnosed him with stage 2 (of 4) CTE. The Franklin family shares Jason's story in order to raise awareness for CTE and their "I Got You" Day, which honors Jason's legacy of helping others.

https://concussionfoundation.org/personal-stories/legacy-stories/jason-franklin

Mike Haynes

Mike Haynes was inducted into the professional football hall of fame in 1997. He was a three-year All-WAC star at Arizona State, was the New England Patriots' first selection in the 1976 NFL Draft.

Haynes relied on his speed, quickness and range to become both a premier defensive back and an outstanding punt return specialist. He enjoyed a sensational rookie year with the Patriots with eight interceptions and an AFC-leading 608 yards on 45 punt returns. That year, Haynes gave the Patriots their first-ever touchdowns on punt returns with 89-yard and 62-yard returns. He earned a Pro Bowl invitation as a rookie, the first of nine Pro Bowl bids.

Haynes, who was born on July 1, 1953, in Denison, Texas, recorded 28 interceptions and 1,159 yards on 111 returns, a 10.4-yard average during his seven years with the Patriots. He started his career with 58 consecutive starts before being sidelined with a rib injury late in 1979. Haynes played the last five games in 1983 and then started and had one interception in the Raiders' Super Bowl XVIII victory.

In seven seasons with the Raiders, Haynes returned only one punt, but he added 18 interceptions to give him a career total of 46 which were returned for 688 yards and two touchdowns, including a team-record 97-yard return against Miami in 1984. Haynes was an All-Pro choice in 1977, 1978, 1982, 1984 and 1985 and an All-AFC pick eight times.

https://www.profootballhof.com/players/mike-haynes/

Pamela Thakur, Esq.

Founder and Managing Attorney of the Thakur Law Firm, APC. With more than 18 years of experience, Pamela maintains a diverse practice that includes complex litigation in the areas of employment, business law, family law, personal injury, and real estate, as well as transactional services, tax and estate planning and healthcare law. She has been selected as a Super Lawyers "Rising Star" every year since 2016 and a Super Lawyer for litigation from 2020-2023.

In her experience as a litigator, Pamela has been fortunate to represent many people who were harmed and unable to find redress for their injuries on their own. A few years ago, Pamela had the opportunity to represent former UCLA football players who suffered injuries that were significantly exacerbated by the conduct of the UCLA coaching staff. Specifically, the players were pressured to continue to play or ignored when they voiced concern of their physical health, ridiculed for complaining of their injuries, discouraged from seeking medical attention, and compelled to return to play before their injuries had healed, all under the guise of a culture that purported to instill grit in players by playing through the pain. As a result, some of the players came to experience symptoms consistent with CTE, which unfortunately cannot be definitively diagnosed until an autopsy is conducted on the corpse, but which commonly affects football players. The cases all settled before trial.

https://www.thakurlawfirm.com/pamelatahimthakur

Recent Legislative Background on Youth Tackle Football and Concussion/Chronic Traumatic Encephalopathy (CTE):

- In 2016, Governor Jerry Brown signed Assemblymember Kevin McCarty's AB 2007 (Chapter 516, Statutes of 2016) that requires a concussion and head injury information sheet to be signed and returned by the athlete and athlete's parent or guardian before an athlete begins practice or competition in one of 27 sports offered by youth sports organizations, and proscribes return to play protocols for concussed athletes.

- In 2018, Assemblymember McCarty authored AB 2108 that would have prohibited any person who is not at least 12 years of age from playing tackle football with a youth sports organization. The bill was withdrawn by the author prior to its hearing in the Assembly Arts, Entertainment, Sports, and Tourism Committee.

- In 2019, the Legislature passed Assemblymember Jim Cooper's AB 1 (Chapter 158, Statutes of 2019) which was signed into law by Governor Gavin Newsom. This bill established the California Youth Football Act, with the purpose of establishing comprehensive safety measures for youth tackle football. Provisions of the bill included limits on the number of full contact practices per week, the amount of time per practice that can be full contact, specify the training programs that must be completed by coaches and administrators on tackling and blocking as well as concussion and head injury education, and regulate equipment inspections and recertification. The bill passed out of both the Assembly and Senate with no NO votes.

- In 2022, the Legislature passed AB 1348 (McCarty) that would have required the Surgeon General to convene a Commission on CTE and Youth Football to investigate issues related to the risks of brain injury associated with participation in youth football, and to provide recommendations to the Governor and Legislature on strategies to reduce this risk, including the minimum appropriate age for participation in youth tackle football. The bill was vetoed by Governor Newsom, who stated that the effectiveness of the California Youth Football Act, which took effect in January 2021, had not been sufficiently assessed and that more research is needed to better understand current safety measures and the risks.

- In 2023, Assemblymember McCarty introduced AB 734, which like AB 2108 in 2018, would prohibit youth sports organizations or youth tackle football leagues from allowing participants younger than 12 years of age. The bill was made into a two-year bill in the Assembly Arts, Entertainment, Sports, and Tourism Committee, and is the reason for the committee's hearing on the safety of youth tackle football.

CONCUSSION LegacyFoundation

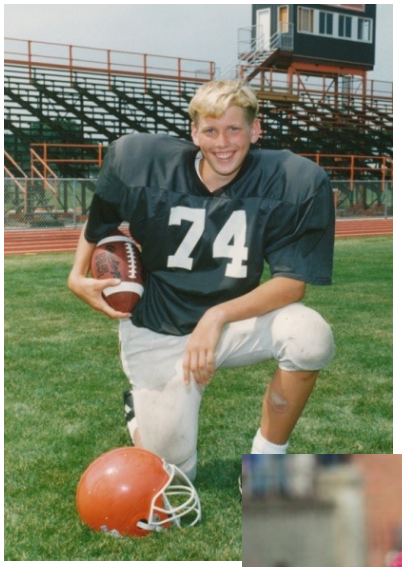
We Need an Age Minimum to play Tackle Football

Chris Nowinski, PhD

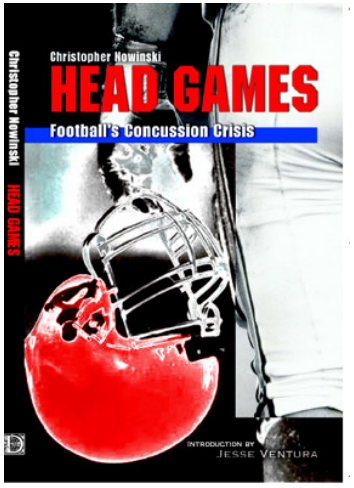
nowinski@concussionfoundation.org

Chris Nowinski, PhD

- Founding CEO, Concussion Legacy Foundation
- Co-founder, UNITE Brain Bank at Boston University
- Co-founder, Australian Sports Brain Bank at the University of Sydney
- Advisor:
  - NFL Players Association
  - Ivy League
- PhD, Behavioral Neuroscience, Boston University School of Medicine, 2017



I am Trying to Prevent CTE in Football Players



- In 2003, a concussion changed my life and inspired me to start researching concussions & CTE
- I have been an advisor to the **NFL Players Association** since 2010 and an advisor to the **Ivy League** since 2011.
- In both instances I convinced them to minimize hitting in practice to reduce CTE risk.
- Rather than minimize hits for kids, I think we shouldn't allow it up to a certain age.

Dedicated to Chris Eitzmann

- Chris was the captain of the 1999 Harvard football team and played 3 years in the NFL.

We were College Roommates

- Chris played 11 years of football overall.

He Left Four Children without Their Father

- A Dartmouth MBA and successful investment manager, Chris developed alcoholism in his late 30's and drank himself to death at 44. He had stage 2 CTE. Our interventions did not save him.

My Perspective

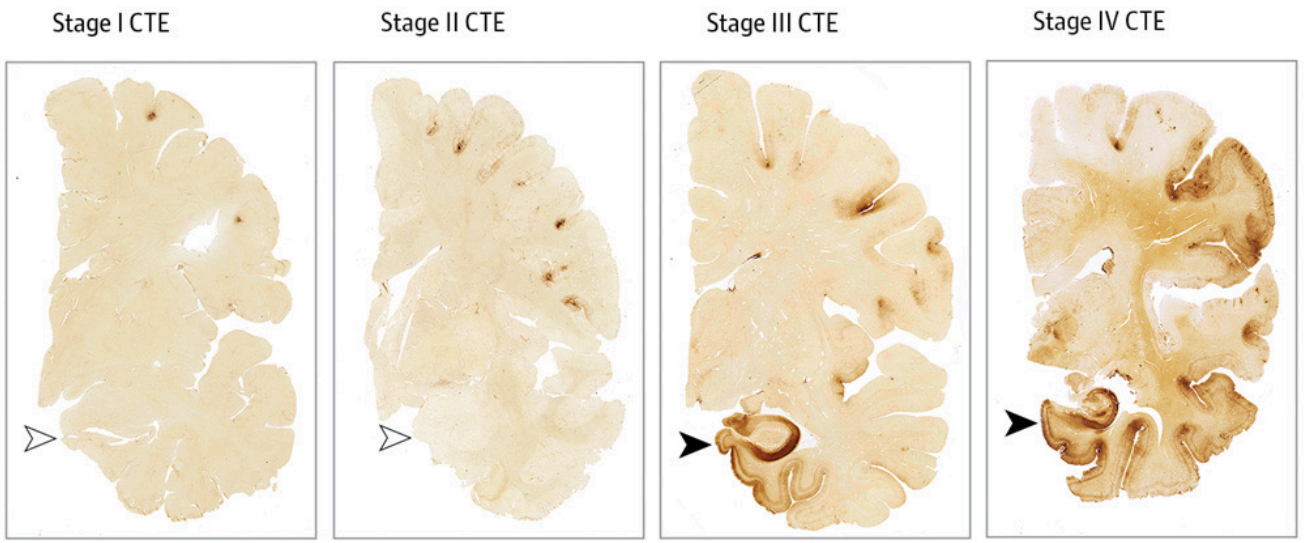
1. Tackle football has many benefits for players
2. Tackle football causes CTE
3. The more tackle football you play, the greater your risk of CTE
4. In our culture, children cannot provide informed consent
5. AB1 will not prevent CTE
6. Tackle football is the only team sport without an age minimum
7. The tackle football industry cannot reform itself

What is Chronic Traumatic Encephalopathy (CTE)?

1. CTE is a neurodegenerative disease caused by brain trauma that causes problems with **cognition, mood, behavior,** and **sleep**, and results in dementia.

2. Studies suggest **0-1% of the population** has CTE, but it is frequent in former contact sport athletes.

3. CTE cannot yet be diagnosed accurately nor effectively treated in the living – but CTE is entirely preventable.



Repetitive Head Impacts, like in Football, Cause CTE

Applying the Bradford Hill Criteria for Causation to Repetitive Head Impacts and Chronic Traumatic Encephalopathy

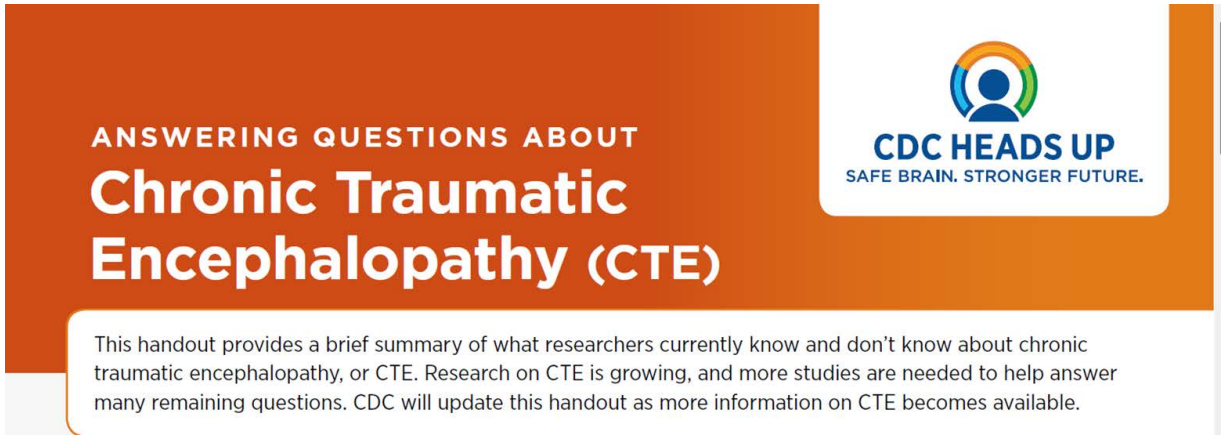
NOT concussions

Christopher J. Nowinski[1], Samantha C. Bureau[1], Michael E. Buck, Maurice A. Curtis[4], Daniel H. Daneshvar[5,6,7], Richard L. M. Faull[4], Elisa L. Hill-Yardin[12,13], Helen C. Murray[4], Alan J. Pearce[14], Cather, Adam J. White[15,16], Adam M. Finkel[17†] and Robert C. Cantu[1,18,19†]*

frontiers in Neurology

Published July 22, 2022

CONCUSSION LegacyFoundation



- The US Centers for Disease Control and Prevention, the world's greatest public health organization, concluded rTBI causes CTE in a 2019 fact sheet
- "The research to-date suggests that CTE is **caused** in part by repeated traumatic brain injuries, including concussions, and repeated hits to the head, called subconcussive head impacts."

NINDS Confirmed Causation on October 5th, 2022

- **Chronic traumatic encephalopathy (CTE)** is a delayed neurodegenerative disorder that was initially identified in postmortem brains and, **research-to-date suggests, is caused in part by repeated traumatic brain injuries.**

National Institute of Neurological Disorders and Stroke

https://www.ninds.nih.gov/current-research/focus-disorders/focus-traumatic-brain-injury-research

CTE Odds are Related to the Number & Strength of Head Impacts

nature communications

Article

https://doi.org/10.1038/s41467-023-39183-0

Leveraging football accelerometer data to quantify associations between repetitive head impacts and chronic traumatic encephalopathy in males

Received: 21 September 2022

Accepted: 30 May 2023

Published online: 20 June 2023

Check for updates

Daniel H. Daneshvar[1,2,3], Evan S. Nair[4], Zachary H. Baucom[4,5], Abigail Rasch[4], Bobak Abdolmohammadi[4], Madeline Uretsky[4], Nicole Saltiel[4], Arsal Shah[4], Johnny Jarnagin[1], Christine M. Baugh[6,7], Brett M. Martin[4,5], Joseph N. Palmisano[4,5], Jonathan D. Cherry[4,8,9,10], Victor E. Alvarez[4,9], Bertrand R. Huber[4,8,9,10], Jennifer Weuve[11], Christopher J. Nowinski[4,12], Robert C. Cantu[4,12,13,14], Ross D. Zafonte[1,2,3,15], Brigid Dwyer[4,16], John F. Crary[17], Lee E. Goldstein[4,16], Neil W. Kowall[4,16], Douglas I. Katz[4,16], Robert A. Stern[4,13,16,18], Yorghos Tripodis[4,5], Thor D. Stein[4,8,9,10], Michael D. McClean[4,5], Michael L. Alosco[4,16], Ann C. McKee[4,8,9,10,16] & Jesse Mez[4,16,19]

CONCUSSION LegacyFoundation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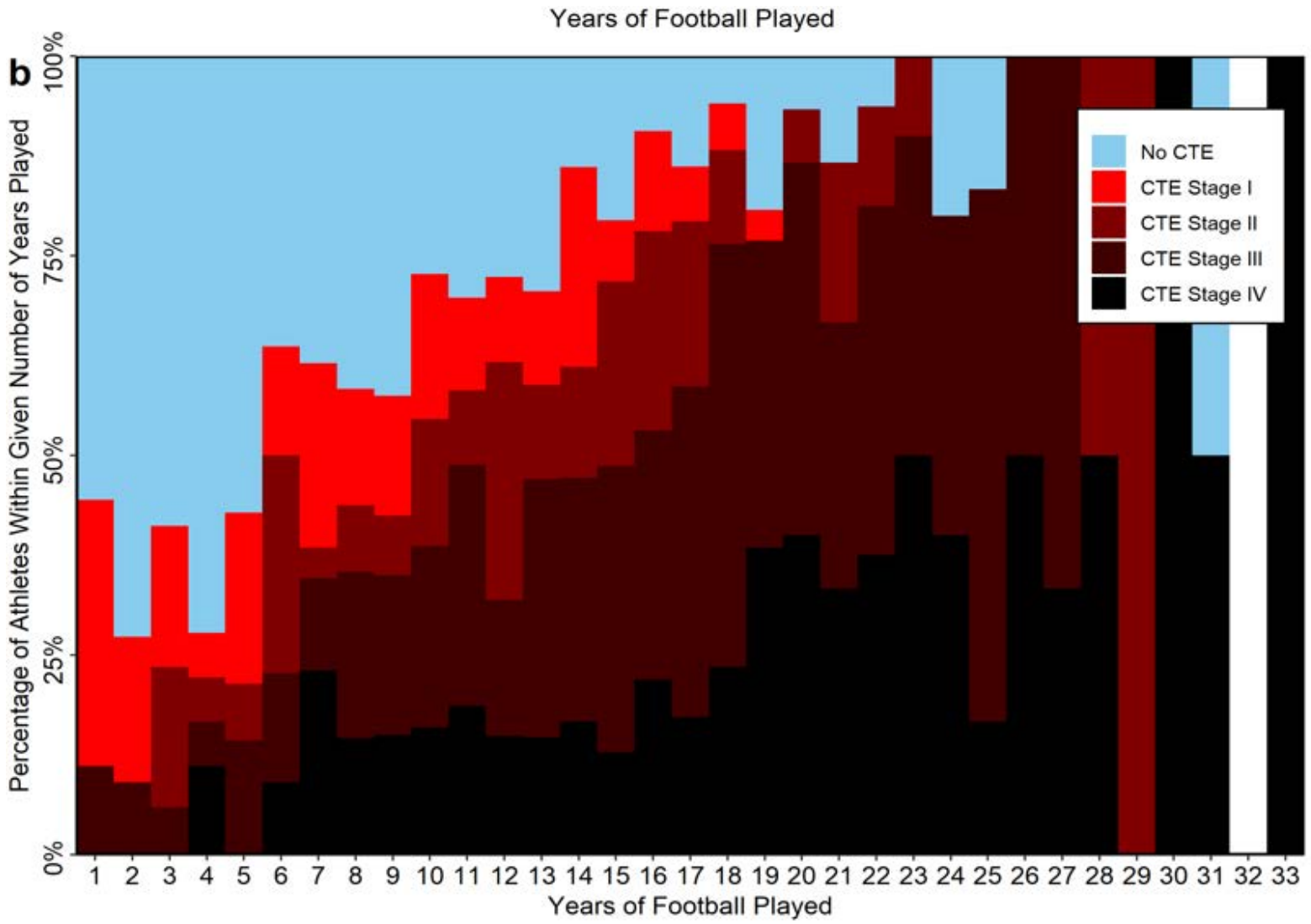
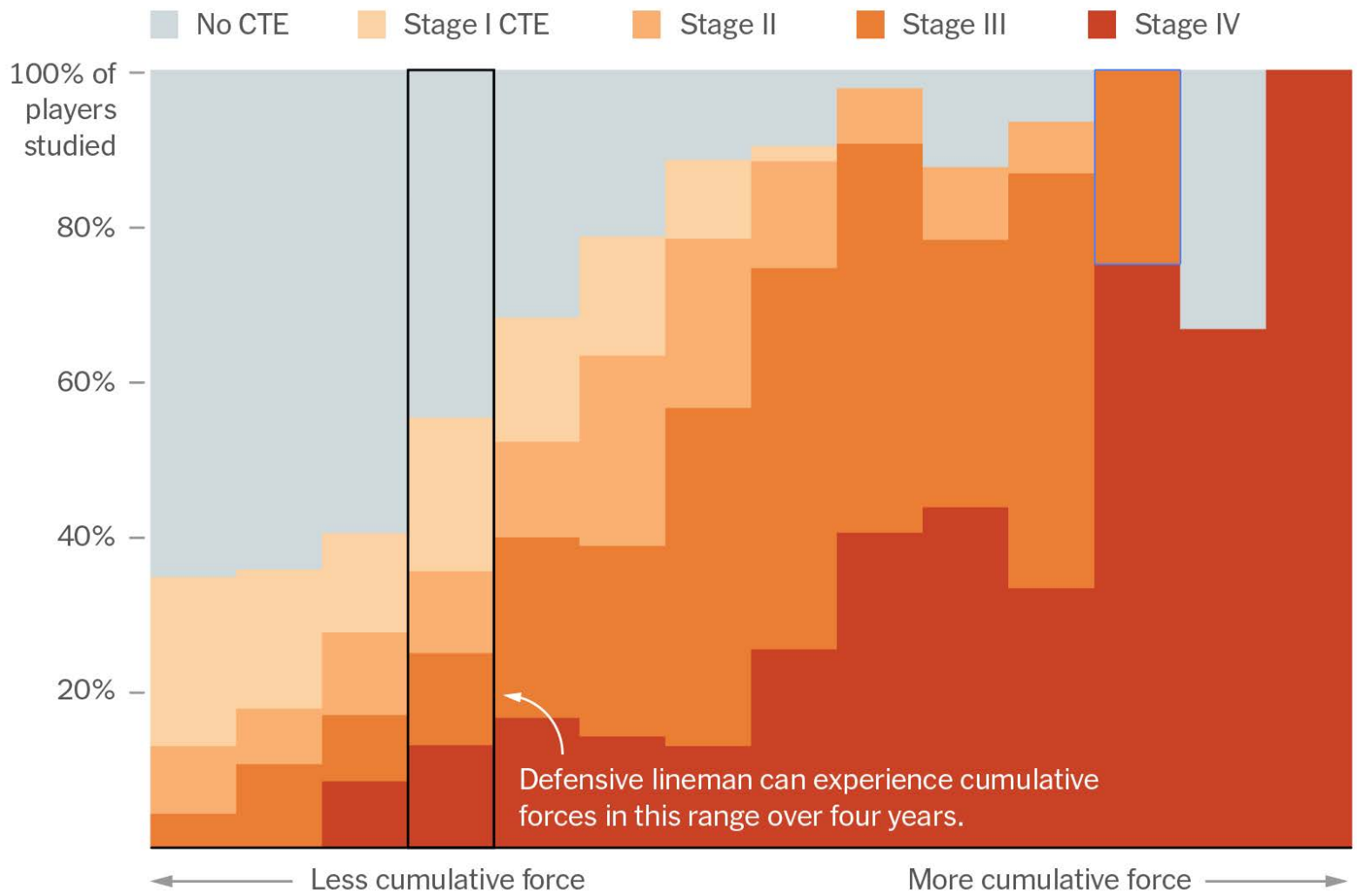

Years of Football Played
b
Percentage of Athletes Within Given Number of Years Played
100%
75%
50%
25%
0%
No CTE
CTE Stage I
CTE Stage II
CTE Stage III
CTE Stage IV
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33
Years of Football Played


No CTE
Stage I CTE
Stage II
Stage III
Stage IV
100% of players studied
80%
60%
40%
20%
Defensive lineman can experience cumulative forces in this range over four years.
Less cumulative force
More cumulative force

Kids Hit Hard

Article https://doi.org/10.1038/s41467-023-39183-0

Table 7 | Position exposure matrix of weighted average annual exposures aggregated from previously published helmet sensor studies

	Number of hits per season			Linear acceleration (g)			Rotational acceleration (rad/s^2)		
	Youth	High school	College	Youth	High school	College	Youth	High school	College
Overall	206.4	538.7	526.0	20.6	26.5	20.6	1203.4	1898.4	1574.2
DL		782.3	840.9		25.8	21.0		1801.3	1806
DB		316.6	371.6		28.5	20.2		1957.4	
LB		460.2	539.0		27.3	22.3		1870.4	2071.7
OL		734.4	814.6		25.8	21.0		1777.5	1782
QB		320.2	209.4		26.8	21		1476.4	
RB		475.1	421.7		27.7	21.8		1807.7	1878.8
TE		517.4	599.2		27.1	31.0		1625.8	1815.7
WR		301.9	313.9		28.8	19.5		2223.8	

Source data are provided as a Source Data file

DL defensive line, *DB* defensive back, *LB* linebacker, *OL* offensive line, *QB* quarterback, *RB* running back, *TE* tight end, *WR* wide receiver

Kids Can Hit Hard

Football/CTE relationship is similar to smoking/lung cancer

- Od
  -

RESEARCH ARTICLE

Duration of American Football Play and Chronic Traumatic Encephalopathy

Jesse Mez, MD, MS,[1,2,3] Daniel H. Daneshvar, MD, PhD,[1,4] Bobak Abdolmohammadi, BA,[1,2] Alicia S. Chua, MS,[1,5] Michael L. Alosco, PhD,[1,2] Patrick T. Kiernan, BA,[1,2,6] Laney Evers, BA,[1,2] Laura Marshall, BA,[1,2] Brett M. Martin, MS,[1,7] Joseph N. Palmisano, MS,[1,7] Christopher J. Nowinski, PhD,[1,8] Ian Mahar, PhD,[1,2] Jonathan D. Cherry, PhD,[1,9,10] Victor E. Alvarez, MD,[1,9,10] Brigid Dwyer, MD,[2,11] Bertrand R. Huber, MD, PhD,[1,2,9,10] Thor D. Stein, MD, PhD,[1,3,9,10,12] Lee E. Goldstein, MD, PhD,[1,2,12,13,14] Douglas I. Katz, MD,[2,11] Robert C. Cantu, MD,[1,2,8,15,16] Rhoda Au, PhD,[1,3,17,18] Neil W. Kowall, MD,[1,2,9,12] Robert A. Stern, PhD,[1,2,15,17] Michael D. McClean, MS, ScD,[19] Jennifer Weuve, MPH, ScD,[18] Yorghos Tripodis, PhD,[1,5] and Ann C. McKee, MD[1,2,3,9,10,12]

Hsieh LJ, Begg CB.
03 Mar 19;95(6):470-8.

AB1 WILL NOT prevent CTE

- AB1 covers helmet recertification, concussions, opioids, heat illness, EMTs, first-aid, etc.

- The ONLY aspects of AB1 that could possibly address CTE risk (which is never mentioned as a goal in the bill) are:
  1. The “limit” is a guideline of two practice sessions of 30 minutes involving full-contact **(this is more than NFL players hit)**
  2. Coaches much receive annual certification on teaching “safer” blocking and tackling to “remove the head from contact”

- Is fewer hits the right answer to a threat to their long-term health?
  - If it were, why don’t we limit 9-year-olds to one pack a day of smoking?

There is No Miracle Technology Solution to CTE

Helmets will not solve CTE

Car bumpers have not solved injuries from car accidents

The best answer for young children is:

STOP HITTING KiDS IN THE HEAD

When is it OK to start hitting kids?

Almost every major contact sport has changed so children don't receive preventable repetitive head impacts.

Almost.

AGE	5	6	7	8	9	10	11	12	13

Learn how we can save youth football at FlagFootballu14.org

What's the Right Age?

- Science doesn't directly answer policy questions. We generally decide on risk/benefit, like driving, or age of consent, like indoor sun tanning.

- California was the first state to ban **indoor tanning** for minors, now there are 20 states with laws. Outdoor tanning is still legal.

The New York Times

'You're 5 Years Old. Wow!' Child Is Stopped on Highway Headed for California

The boy was driving to California to buy a Lamborghini, the Utah Highway Patrol said.

Share full article

Why is Football an Outlier

- Unlike USA Hockey, US Soccer, and USA Lacrosse, no governing body can influence national rules for youth football.

- At a 2012 Aspen Institute panel discussion on the future of football, Pop Warner executive director Jon Butler said that if Pop Warner only offered flag football, “90 to 95 percent of our members would drop out” and play for independent teams “because whether it be kids or parents, they want to play tackle football.”

- It was discussed that if Pop Warner said no tackle until age 10, it would create a void to offer tackle for 5 to 9-year-olds that would be filled by a competitor, and the players would remain with the competitor, effectively putting Pop Warner out of business.

Offering Solutions

- Jon Butler, the longtime executive director of Pop Warner, recently said,

> "First, we have a duty to football. This means too much to people and has for so long we can't turn our back on it. We figured out tobacco. We figured out asbestos. We'll figure this out."

- We figured out tobacco and asbestos by making it ***illegal to expose children to them.***

- If we allow children to start playing tackle football at age 5, and if love the game and succeed and play in the NFL, the majority will have CTE. What kind of a prize is that?
  - If we start later, players may retain the benefits without losing so much to CTE

Flag Football is an Age-Appropriate Introduction to Football

- A CDC study found youth tackle players receive nearly 50x as many head impacts as flag.
- All the benefits without the risk for CTE.

My Perspective

1. Sports have many benefits
2. Tackle football causes CTE
3. More tackle football = more CTE
4. **Children cannot provide informed consent**
5. AB1 will not prevent CTE
6. Tackle football is the only team sport without an age minimum
7. The tackle football industry cannot reform itself

CONCUSSION LegacyFoundation

Please develop an age minimum for participation in tackle football.

Chris Nowinski, PhD
CEO, Concussion Legacy Foundation
nowinski@concussionfoundation.org

If you are a former football player struggling with suspected CTE, please reach out to the CLFHelpLine.org or help@concussionfoundation.org

5 Trick Questions Designed to Trivialize CTE Risk

These questions have been put forth by people associated with sporting bodies as a reason to not accept causation. These anecdotes display a lack of knowledge about understanding causation.

	Trick Question	Scientific Answer
1	"What about those people who have had substantial exposure to RHI but never developed CTE"?	The very definition of "risk" ensures that some individuals will escape the consequences of risky exposure. Many people smoke cigarettes and never develop lung cancer. "Risk" and "certainty" are entirely different concepts.

Adapted by Nowinski CN & Finkel AM from Brand KP, Finkel AM. A Decision-Analytic Approach to Addressing the Evidence About Football and Chronic Traumatic Encephalopathy. Semin Neurol. 2020 Aug;40(4):450-460.

5 Trick Questions Designed to Trivialize CTE Risk

These questions have been put forth by people associated with sporting bodies as a reason to not accept causation. These anecdotes display a lack of knowledge about understanding causation

	Trick Question	Scientific Answer
4	"What about those who had symptoms but were found not to have had CTE?"	Many symptoms are nonspecific. Memory problems, depression, etc. have multiple causes. It doesn't mean that CTE can't also cause them.

Adapted by Nowinski CN & Finkel AM from Brand KP, Finkel AM. A Decision-Analytic Approach to Addressing the Evidence About Football and Chronic Traumatic Encephalopathy. Semin Neurol. 2020 Aug;40(4):450-460.

Novel Mental Health Disorders After Concussion

1. A Canadian study of 152,321 pediatric concussions (age 5-18) versus matched control orthopedic injuries found the children with concussions are:
   - 39% more likely to have a mental health diagnosis
   - 49% more likely to engage in self-harm
   - 47% more likely to have an inpatient psychiatric hospitalization

Ledoux AA, Webster RJ, Clarke AE, Fell DB, Knight BD, Gardner W, Cloutier P, Gray C, Tuna M, Zemek R. Risk of Mental Health Problems in Children and Youths Following Concussion. JAMA Netw Open. 2022 Mar 1;5(3):e221235. doi: 10.1001/jamanetworkopen.2022.1235. PMID: 35254429; PMCID: PMC8902648.

110 of 111 NFL Players had CTE

JAMA | Original Investigation

Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football

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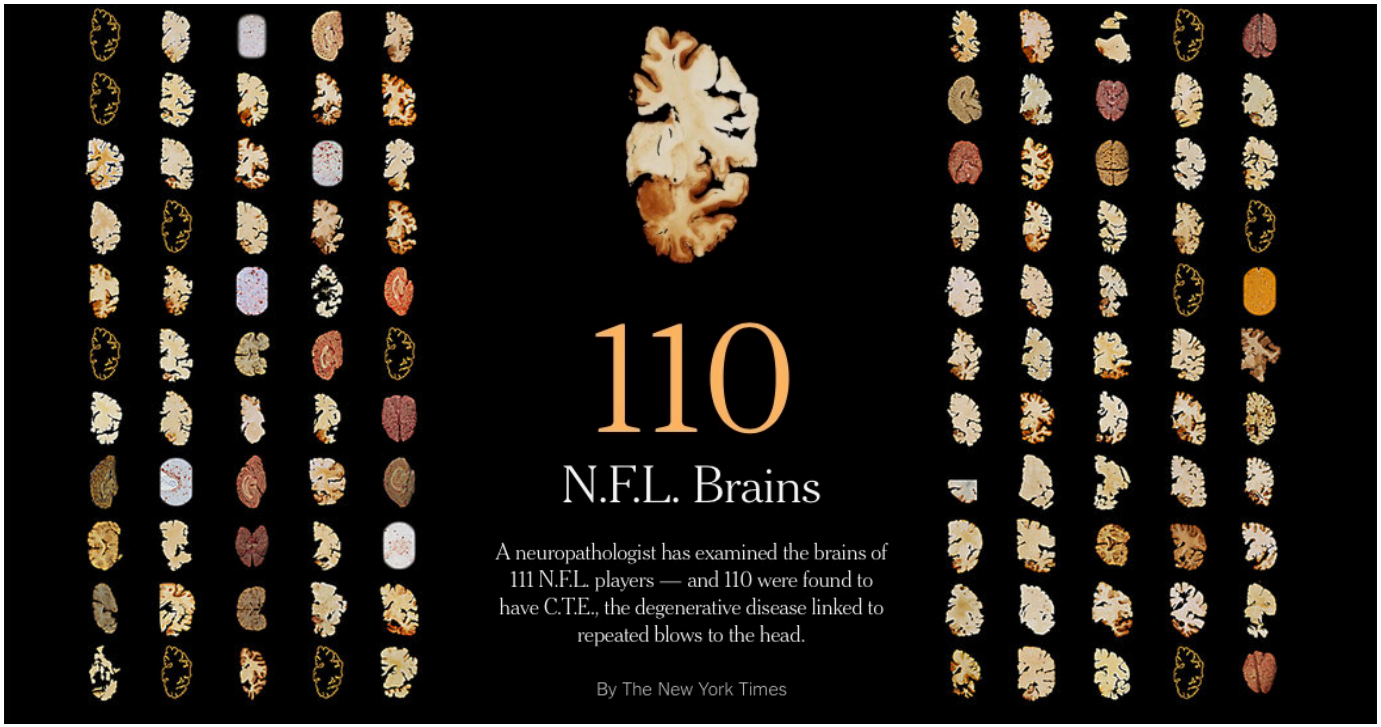
This 2017 JAMA study on CTE in football was the decade's most widely read and impactful study in neurosciences.

The accompanying New York Times multimedia feature (right) showed the damage.



Flag Football Under 14: An Education Campaign for Parents

White Paper by Chris Nowinski, Ph.D., and Robert Cantu, M.D.

If you are a parent considering enrolling your child in football, the Concussion Legacy Foundation strongly recommends you delay enrolling them in *tackle* football until the age of 14. Americans have played tackle football nearly 150 years, but only in the last 15 years has the scientific community begun investigating its long-term effects on the brain. The initial research is so concerning that we launched the Flag Football Under 14 campaign (FFu14) to educate parents so they can make an informed decision. This campaign builds on the recommendation made by Dr. Robert Cantu, who first advocated this policy in his 2012 book Concussions and Our Kids. Until tackle football is proven safe for the developing brain, we urgently recommend parents only enroll their children in flag and other non-tackle versions of football before age 14.

Youth Tackle Football is a Modern Experiment

Delaying the introduction of tackle football until 14 is not as radical as it may seem. While tackle football has existed for 150 years, modern youth tackle football has only existed for 50 years. When Pop Warner "youth" tackle football began, its youngest players were 15.[1] It was not until after the legendary coach's death in 1954 that Pop Warner Little Scholars, Inc., became a corporation that nationally promotes 5-year-olds playing tackle football. Adoption was slow, but by 1970, there were a few thousand Pop Warner youth tackle football teams across the country. Players from this era have begun dying, and their families are donating their brains to science due to concerns of how football affected their brains and behavior. The results of these studies should give us pause as to whether it is appropriate for children to suffer hundreds of head impacts a year playing youth tackle football.

Research on CTE has Changed our Recommendation

When CLF began researching chronic traumatic encephalopathy (CTE) in 2007, we did not make any recommendations on participation in youth tackle football because there was not yet enough science. This is no longer the case.

CTE is a progressive, degenerative brain disease caused by repeated head impacts, like those incurred in boxing or tackle football. CTE was originally called "Punch Drunk" because it was first observed in boxers in the 1920s. CTE starts as small lesions around blood vessels in the brain, but it progresses even after affected football players retire and stop receiving head impacts. As players age and the disease spreads, CTE attacks parts of the brain responsible for memory, judgment, mood, and control of behavior. The end stage of CTE can look like Alzheimer's disease and usually requires nursing home care because patients can no longer function independently and eventually lose the ability to speak, stand, or feed themselves.

There Is No More Debate: CTE is Caused by Repeating TBIs

While you may be told there is a debate in the medical community on whether causation is proven, remember that every doctor has an opinion, but not every doctor is an expert at interpreting research and understanding what qualifies as proof of causation. The independent experts in the United States government, including the **National Institutes of Health, Centers for Disease Control and Prevention, and the lead expert for the Department of Defense say CTE is caused by repeated TBIs.**

> "The research to-date suggests that CTE is caused in part by repeated traumatic brain injuries, including concussions, and repeated hits to the head, called subconcussive head impacts."[2]
> ***-US Centers for Disease Control and Prevention.*** *Answering Questions about CTE.*

> "CTE is a delayed neurodegenerative disorder that was initially identified in postmortem brains and, research-to-date suggests, is caused in part by repeated traumatic brain injuries"
>
> **- Dr. Nsini Umoh, M.D.,** *Program Director for Traumatic Brain Injury, National Institute of Neurological Disorders and Stroke, a division of the National Institutes of Health*

> "CTE is only seen in the setting of repeated head trauma. At the end of the day, this is produced by head trauma. I'm sorry, that's what all the research says."[3]
>
> ***- Daniel Perl, M.D.*** *Director of CTE Research, Dept. of Defense, Uniformed Services University*

CTE can only be confirmed through the post-mortem examination of the brain, and because it was never looked for, it was not found in a football player until 2005. Since then, it was diagnosed in 110 of the first 111 NFL players studied at Boston University, including Ken Stabler, Dave Duerson, John Mackey, and Aaron Hernandez. Unfortunately, CTE has also been found in college and high school football players, including athletes as young as 17. CTE is not seen in the normal population that has not been exposed to repeated head impacts.[4]

Clinicopathological Evaluation of CTE in Players of American Football [5]

Highest Level Played	Share with CTE
Youth	0 of 2 (0%)
High School	3 of 14 (21%)
College	48 of 53 (91%)
Semiprofessional	9 of 14 (64%)
Canadian Football League (CFL)	7 of 8 (88%)
National Football League (NFL)	110 of 111 (99%)

*Published in the Journal of the American Medical Association, July 2017

CTE is not just a football problem. In addition to boxing, CTE has been diagnosed in athletes who participated in ice hockey, soccer, rugby, baseball, bull riding, professional wrestling, and mixed martial arts. However, football is where the most evidence exists, as two-thirds of globally diagnosed CTE cases are American football players.

A 2023 study from the Boston University CTE Center confirmed CTE begins early in life when exposure to head impacts begins early in life. Among 92 football players who **died before the age of 30**, the

majority had CTE, including 30% who did not play after high school. In those cases, the cause of their CTE was **pediatric repeated traumatic brain injuries.**

CTE in Athletes who Died Before Age 30

Highest Level Played	Share with CTE
Youth*	2 of 7 (29%)
High School	14 of 45 (30%)
College	21 of 26 (81%)
Professional	11 of 12 (92%)
Total	**48 of 90 (53%)**

***in this study, the youth players with CTE also played other contact sports

CTE Odds May Increase by 30% Per Year

A 2019 study led by Boston University researchers and published in the *Annals of Neurology* medical journal analyzed 266 deceased football players, 223 of whom had CTE and 43 of whom did not. The researchers concluded the risk and severity of developing CTE is not correlated to number of concussions, but instead to the number of years playing tackle football.[6]

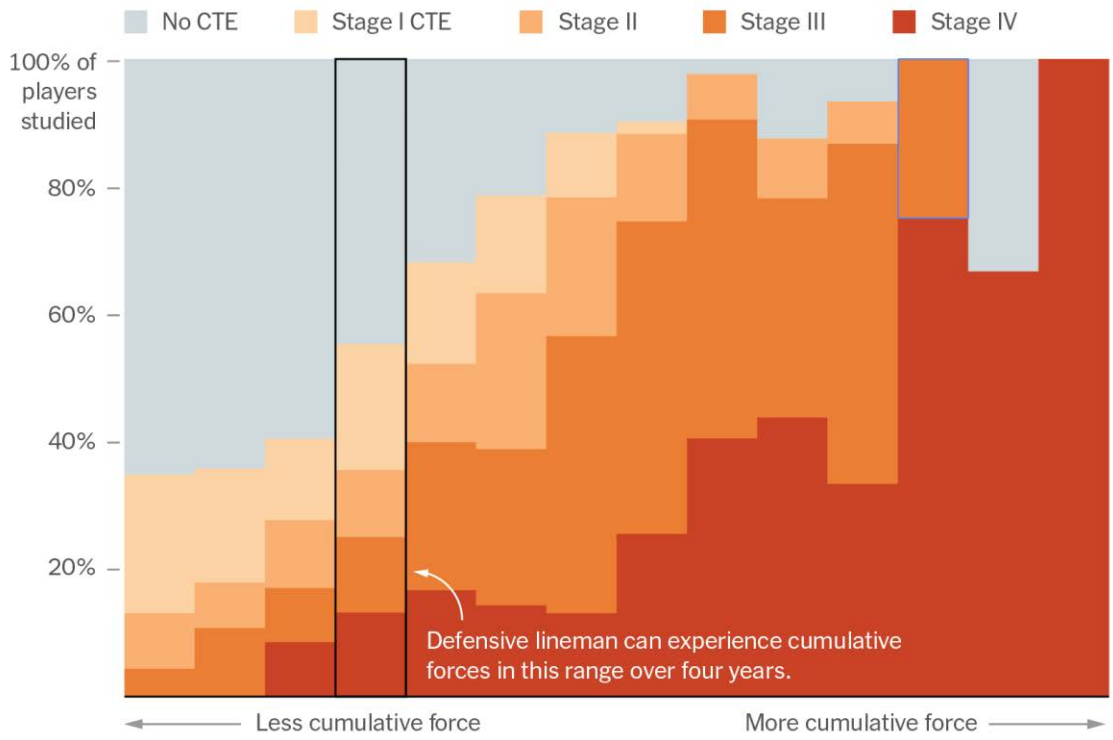
Researchers found the risk of CTE increases by 30% every year of tackle football, and it doubles every 2.6 additional years. (We don't know the risk of developing CTE after one year of playing tackle football, but it's likely very low.) This relationship between years of tackle and risk of CTE is of similar strength to the link between years of smoking and risk of lung cancer.

The link is so profound that a high school football player who starts tackle football at age 5, instead of age 14, has an incredible 10 times the risk of developing CTE.

Based on this data, if we were to successfully convince all parents to delay enrolling their children in tackle football until age 14, **we would expect to prevent more than 50 percent of future CTE cases.**

FFu14 Is Not about Concussions, but Nonconcussive Head Impacts

While years of playing football increases one's odds of developing CTE, years are a crude marker for repetitive head impacts. More recent studies provide strong evidence that the number and strength of head impacts received while playing better determine who develops CTE.[7] A study of 631 deceased football players revealed the odds of developing CTE increase by about 21% per 1,000 football head impacts and are even more strongly correlated when you increase the strength of those head impacts.



Estimated cumulative force of head hits for 631 former football players *(Image from the New York Times). Researchers in a new study estimated the cumulative force of head impacts absorbed by 631 former football players. Players who absorbed the most cumulative force from head hits had the worst forms of CTE.*

Concussions are not unique to football, and as the NFL commissioner has pointed out, "Concussions can happen in a variety of activities."[8] But this campaign is not focused on concussions. Literature reviews support that a single concussion, in the absence of repetitive brain trauma, *does not* consistently appear to be a major risk factor for CTE or related neurodegenerative diseases.[9] The 2019 *Annals of Neurology* study and subsequent studies that found a strong correlation between years of tackle football and risk of CTE found **no correlation between concussions and risk of CTE.**

However, tackle football does pose a unique risk to young brains. A CDC study found the median youth tackle football player received 378 head impacts per season, while the median flag player receives 8.

Tackle football is dangerous for the brain because it requires both tackling and blocking. Both activities cause regular head impacts **even when proper form is used**. Studies show tackle football causes more repetitive head impacts than any other youth team sport.

Some parents and coaches argue that children need to learn to tackle young in order to tackle safely. First, there is no evidence for this. Second, if this were true, we would also let children drive cars at 6 years old with the goal that they will drive more safely as adults. Of course, we don't let children do that because we know they are not cognitively mature or physically coordinated enough to drive safely. We believe we should use the same rationale when considering the age at which we ask children to tackle other children.

Children have Biological Disadvantages

Head impacts are more dangerous for children than they are for adults. Children are at a disadvantage playing tackle football for two main reasons:

1. Brain development
2. The Bobblehead Effect

Brain development. In the pre-adolescent and adolescent years (age 8 to age 13), the brain undergoes dramatic changes and maturation that are responsible for the transition from child to adult brain function.[10] Among the neurodevelopmental milestones identified in this population, three stand out for football players:

1. White matter development: Myelin is a fatty substance that surrounds axons (the long connections from one nerve cell to another), giving the white appearance of the white matter. Myelin acts like the insulation around wires in electrical systems, protecting cells and increasing the speed of communication between cells. The peak rate of nerve cell myelination occurs at ages 11 and 12. Without adequate myelin, axons can be more easily injured or destroyed, slowing the speed of brain processing.
2. Grey matter development: Important brain structures, including those controlling memory and emotion, reach their peak in size between ages 8 and 12, building trillions of connections to improve functioning.
3. Peak cerebral blood flow: Blood flow to the cerebrum (the largest and most important part of the brain) supplies the nerve cells with the nutrition and energy needed for healthy functioning. This blood flow peaks between ages 10 and 12, reflecting the tremendous growth and maturation of many regions of the brain.

The Bobblehead Effect. Youth football players are slower and smaller than adults, which can make head impacts in youth tackle football appear inconsequential, like a pillow fight.

However, using helmet sensors, researchers unexpectedly discovered that a youth player experiences head impacts that rival those of college football players.[11] [12] [13]

Experts in human development and biomechanics have suggested these head hits are similar for three reasons:

1. Children have dramatically larger heads relative to the rest of their body than adults.
2. A child's football helmet may be 10 percent of their bodyweight. (That would be equivalent to an NFL lineman wearing a 30-pound helmet!)
3. Children have smaller, weaker necks relative to adults.[14]

When combined, these three elements create a perfect storm. The reason our eyes deceive us is that it's not the speed a youth football player brings to the tackle that causes the severity of the head impacts; it is the fact they cannot slow their proportionally giant, heavy head down after it's been impacted, creating a Bobblehead Effect.[15]

Other Youth Contact Sports Have Changed

Football is not the only sport which should change to eliminate repetitive head impacts for children prior to 14. The Concussion Legacy Foundation advocates for all sports to have a modified youth version that does not involve preventable, repeated head impacts. Many sports have already changed:

- US Youth Soccer eliminated heading for players under age 11 in 2015, and limits heading in practices for 11 to 13-year-olds. (Learn about our Safer Soccer campaign.)
- The Football Association (UK) banned heading before 12 in 2022.
- Rugby Canada banned tackling before 11 in 2022.
- USA Hockey eliminated checking for 11 and 12-year-old players in 2011, introducing intentional contact at age 13.
- USA Lacrosse changed its rules for U14, now penalizing any check involving the head and any stick-to-head contact.

Youth football is the largest contact sport that has failed to implement a policy protecting young athletes from repetitive head impacts. Therefore, each year, about 2 million youth tackle football players are estimated to suffer nearly *a billion* combined head impacts during games and practices.

The Perceived Benefits of Tackle May Not Outweigh the Risks

It is undeniable that there are risks to hitting a child in the head 378 times each season in youth tackle football. Are there benefits?

First, the discussion must be appropriately framed. There are clear health benefits to exercise, and there are character-building benefits to team sports. No one questions this. However, there is no data to support that tackle provides better exercise than flag or greater soft benefits of team sports than flag, which is also a team sport.

In a debate of tackle versus flag, because both sports offer exercise, there is no use for a discussion of obesity or other health risks that come with inactivity.

Through this framework, we believe we cannot overlook the absurdity of hitting a child in the head hundreds of times simply because they happen to be getting exercise.

Star football coaches and players agree

Scientists are not alone in advocating for flag football under age 14. There is a growing contingent of football legends —both players and coaches — who advocate for delaying tackle until 14. They argue the

formula to become the best tackle football player at the high school, college, and professional level means passing on youth tackle football.

> "They don't need a helmet. They can play flag football. And with flag football you can get all the techniques. Why do we have to start with a 6-year-old who was just potty trained a year ago and put a helmet on him and tackle? . . . We'll eventually get to tackling."
>
> –***John Madden**, Pro Football Hall of Fame Coach and Broadcaster*
>
> †
>
> "I just don't think [tackle football] is worth the risk. I think they can have more fun playing flag football and developing the skills that if they ever want to transition, they can do it."
> **- *Drew Brees**, Future Hall of Fame Quarterback*
>
> †
>
> "I always encourage youngsters in America to play soccer. I think every American boy should play soccer till the eighth grade, then they should play football – American football."
> **- *Jim Harbaugh**, Head Coach, University of Michigan*

Waiting until high school to play tackle football did not hold back some of the greatest players in NFL history. In fact, the top five NFL players of all time waited until 14 to play tackle football: **Jerry Rice, Jim Brown, Lawrence Taylor, Walter Payton,** and **Tom Brady**. We assembled the Flag Football Under 14 All-Time Greatest Team, composed entirely of players who did not start tackle football until high school, to illustrate that it is better to wait.

Summary

In addition to NFL players and football coaching icons supporting the merits of Flag Football Under 14, public opinion is clear. A recent poll showed 4 out of 5 adults believe that tackle football is not appropriate for children under age 14. [16] The emerging consensus among football legends, American adults, and a growing number of leading neuroscientists reflects the impact of recent CTE research. Eliminating tackling in youth football, opting instead for flag or other non-tackle versions under age 14, will allow our children to enjoy the sport and continue to develop their athletic and academic skills without putting their brains — and futures — at risk.

We encourage parents and families to become educated on CTE and continue to follow advancements in our understanding so they can make an informed choice, as the wrong choice can have life-long consequences.

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Improving Brain Safety in Youth Football

October 2, 2023

Sacramento, CA

David Camarillo, PhD

Associate Professor of Bioengineering,

Neurosurgery and Mechanical Engineering (by courtesy)

Stanford University Schools of Engineering and Medicine

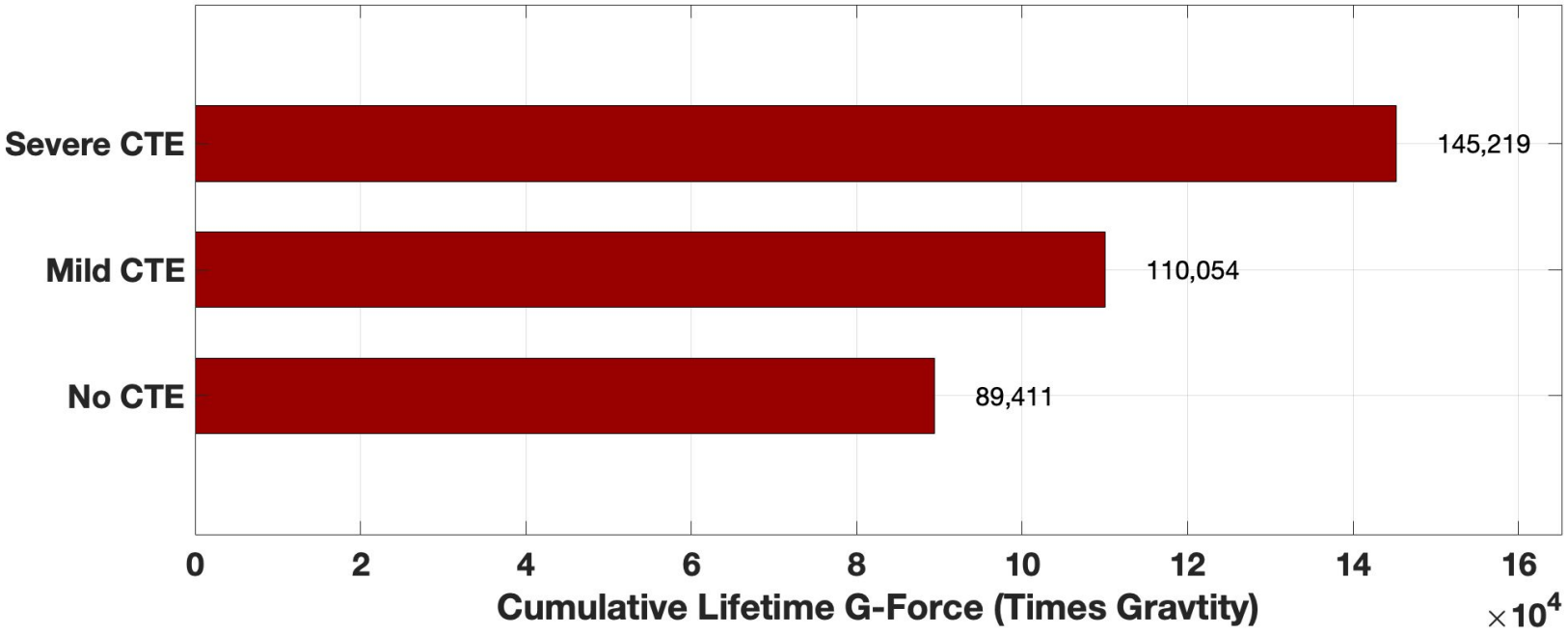
Stanford University

Does youth football cause brain damage?

First author	Year published	Primary study site	Total *N*	Played before age 12	Age (years)	Sample/ outcome measures	AFE binary vs. continuous	Positive Findings
Stamm (80)	2015	BU CTE Center	42	21	$M = 52$, SE = 1	Former NFL Players (DETECT)/Neuropsychological Testing	Binary	Yes
Stamm (81)	2015	BU CTE Center	40	20	$M = 52$, SD = 6	Former NFL Players (DETECT)/Neuroimaging (DTI; Corpus Callosum)	Binary	Yes
Schultz (82)	2018	BU CTE Center	86	Not reported	$M = 55$, SD = 8	Former NFL Players (DETECT)/Neuroimaging (Thalamic Volume)	Continuous	Yes
Kaufmann (83)	2021	BU CTE Center	63	Not reported	$M = 56$, SD = 8	Former NFL Players (DETECT)/Neuroimaging (Cortical Thickness)	Continuous	Yes
Alosco (92)	2017	BU CTE Center	214	101	$M = 51$, SD = 13	Former Amateur and Professional Football (LEGEND)/Neuropsychological Testing and Self-Report Measures	Binary and Continuous	Yes
Montenigro (93)	2017	BU CTE Center	93	Not reported	$M = 47$, SD = 14	Former High School and Collegiate Football (LEGEND)/Neuropsychological Testing and Self-Report Measures	Binary	No
Alosco (94)	2018	BU CTE Center	211	84	$M = 63$, SD = 18	Deceased Former Amateur & Professional Football (UNITE)/Post-Mortem Neuropathology, Symptoms, Age of Onset of Problems	Binary and Continuous	Yes
Solomon (91)	2016	Vanderbilt University	45	Not reported	$M = 47$, SD = 9	Former NFL Players/Neuroimaging, Neuropsychological Testing, Self-Report Measures	Continuous	No
Roberts (71)	2019	Harvard University	3,506	Not reported	$M = 53$, SD = 14	Former NFL Players (Football Players Health Study)/Self-Report Measures	Binary and Continuous	No
Iverson (86)	2020	Harvard Medical School	123	62	$M = 45$, SD = 6	Former high school football players/Self-Report Measures	Binary and Continuous	No
Iverson (85)	2021	Harvard Medical School	186	87	$M = 52$, SD = 11	Former high school football players/Self-Report Measures	Binary and Continuous	No
Bryant (122)	2020	Cleveland Clinic Lou Ruvo Center for Brain Health	Active fighters (n = 442); Retired fighters (n = 64)	Not reported	Active fighters M = 29 SD = 5; Retired fighters; $M = 48$, SD = 10	Male and female licensed professional fighters (boxers, mixed martial artists, and martial artists); Professional Fighters Brain Health Study/Neuroimaging, Neuropsychological Testing, Balance, Self-Report Measures	Continuous	Yes
Hunzinger (121)	2021	University of Delaware	1,034 (Active and Retired)	753	$M = 32$, SD = 11	Current and former male and female rugby players/Self-Report Measures	Binary and Continuous	No

Iverson et al., *Front Neurol.* 2021

Head acceleration is associated with disease severity



Article https://doi.org/10.1038/s41467-023-39183-0

Leveraging football accelerometer data to quantify associations between repetitive head impacts and chronic traumatic encephalopathy in males

June 2023 · Nature Communications 14(1)
DOI: 10.1038/s41467-023-39183-0

Daniel Daneshvar · Evan S. Nair · Zachary H. Baucom
Jesse Mez

The Guardian Cap: Promising, but controversial

NFL says positions wearing Guardian Caps saw 52% decrease in concussions

NBC Sports

By Michael David Smith • Published August 16, 2023 11:44 AM

guardiansports.com

No reduction in acceleration found on field

S.I. : Concussions II | Published: 14 March 2023

Padded Helmet Shell Covers in American Football: A Comprehensive Laboratory Evaluation with Preliminary On-Field Findings

Nicholas J. Cecchi, Ashlyn A. Callan, Landon P. Watson, Yuzhe Liu, Xianghao Zhan, Ramanand V. Vegesna, Collin Pang, Enora Le Flao, Gerald A. Grant, Michael M. Zeineh & David B. Camarillo

RESEARCH ARTICLE | SEPTEMBER 21 2023

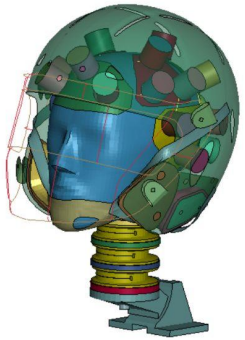
Preliminary Examination of Guardian Cap Head Impact Kinematics Using Instrumented Mouthguards

Kristen G Quigley; Dustin Hopfe, MS, LAT, ATC; Madison Fenner; Philip Pavilionis, MS, ATC; Vincentia Owusu-Amankonah1e; Arthur Islas, MD; Nicholas G Murray, PhD

J Athl Train (2023)

Reducing acceleration reduces concussions

Liquid Simulation Model



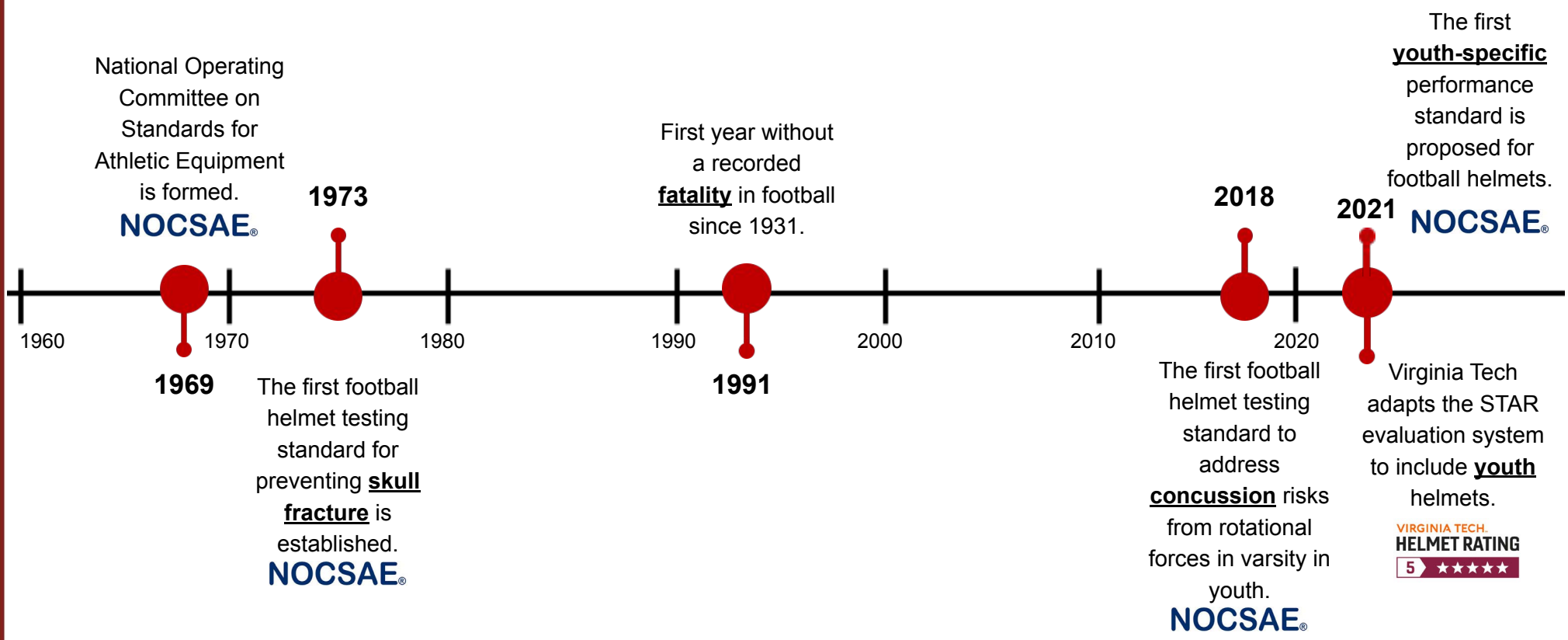
*Circle size does not indicate player-plays for liquid simulation data

Cecchi et al., *Front Bioeng Biotechnol.* 2023.

NFL Field Data

Bailey et al., *Ann Biomed Eng.* 2020

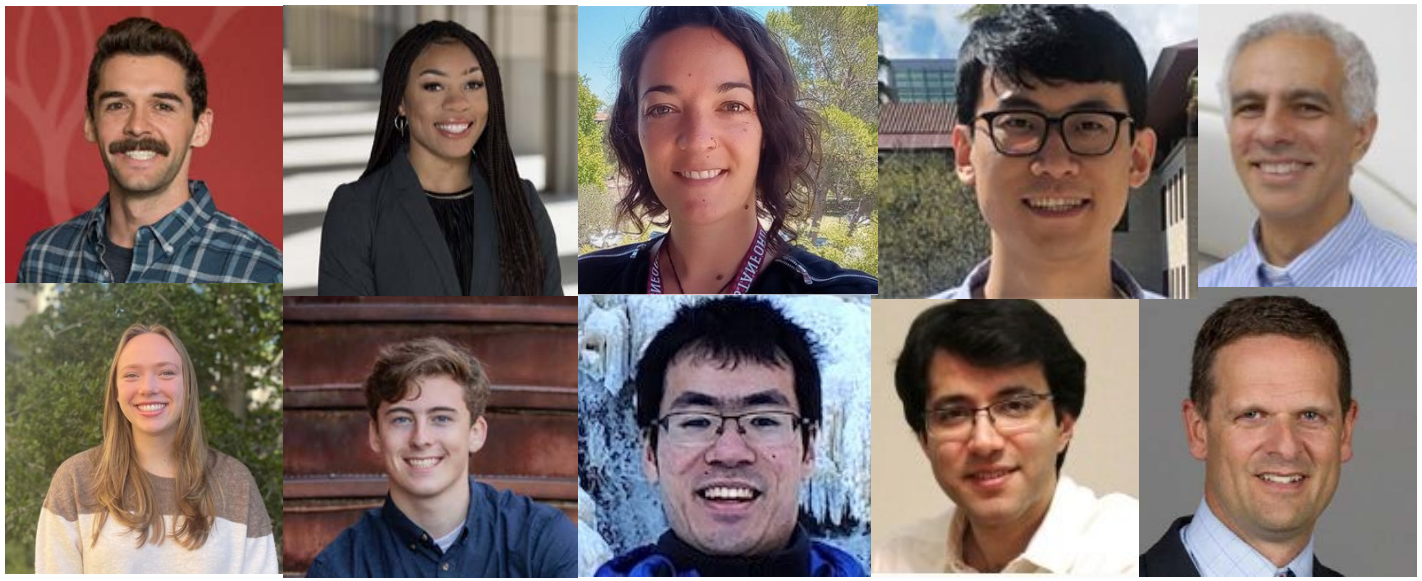
Youth helmets have been tested like adult helmets



Youth football can be made safer.

- Negative brain health effects of youth football are unclear
- Reducing head accelerations may prevent CTE
- Helmet innovations have been focused on adolescents/adults
- Youth helmet safety ratings need development
- Government Regulations improve safety

Acknowledgements



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Finite element evaluation of an American football helmet featuring liquid shock absorbers for protecting against concussive and subconcussive head impacts

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Introduction: Concern has grown over the potential long-term effects of repeated head impacts and concussions in American football. Recent advances in impact engineering have yielded the development of soft, collapsible, liquid shock absorbers, which have demonstrated the ability to dramatically attenuate impact forces relative to existing helmet shock absorbers.

Methods: To further explore how liquid shock absorbers can improve the efficacy of an American football helmet, we developed and optimized a finite element (FE) helmet model including 21 liquid shock absorbers spread out throughout the helmet. Using FE models of an anthropomorphic test headform and linear impactor, a previously published impact test protocol representative of concussive National Football League impacts (six impact locations, three velocities) was performed on the liquid FE helmet model and four existing FE helmet models. We also evaluated the helmets at three lower impact velocities representative of subconcussive football impacts. Head kinematics were recorded for each impact and used to compute the Head Acceleration Response Metric (HARM), a metric factoring in both linear and angular head kinematics and used to evaluate helmet performance. The head kinematics were also input to a FE model of the head and brain to calculate the resulting brain strain from each impact.

Results: The liquid helmet model yielded the lowest value of HARM at 33 of the 36 impact conditions, offering an average 33.0% (range: −37.5% to 56.0%) and 32.0% (range: −2.2% to 50.5%) reduction over the existing helmet models at each impact condition in the subconcussive and concussive tests, respectively. The liquid helmet had a Helmet Performance Score (calculated using a summation of HARM values weighted based on injury incidence data) of 0.71, compared to scores ranging from 1.07 – 1.21 from the other four FE helmet models. Resulting brain strains were also lower in the liquid helmet.

Discussion: The results of this study demonstrate the promising ability of liquid shock absorbers to improve helmet safety performance and encourage the development of physical prototypes of helmets featuring this technology. The implications of the observed reductions on brain injury risk are discussed.

KEYWORDS

concussion, brain injury, hydraulic shock absorber, helmet, brain strain, finite element modeling

Introduction

Sports and recreation can present a considerable risk of brain injury, with an estimated 1.6 to 3.8 million mild traumatic brain injuries, or concussions, occurring annually in the United States as a result of participation in these activities (Langlois et al., 2006). American football, in particular, presents a significant risk for concussion relative to other sports (Lincoln et al., 2011; Kerr et al., 2019). At the elite level of play (i.e., the National Football League (NFL)), one study found that the risk of concussion between the 2015 and 2019 seasons was 7.4% per player per season on average (Mack et al., 2021). In another study, single season concussion risks in youth, high school, and collegiate American football were found to be as high as 3.53, 9.98, and 5.54% per player per season, respectively (Dompier et al., 2015). Sport-related concussion can be followed by physical, behavioral, somatic, and cognitive symptoms (McCrory et al., 2017) that can last from the span of days, weeks, or even months as post-concussion syndrome (Broshek et al., 2015). Furthermore, while more research is required, several studies have found associations between concussion history and later life cognitive impairment and neurodegenerative disease development (Manley et al., 2017). Aside from the health risks, concussion can also have negative effects on athlete performance, career longevity, and salary earnings for professional American football players (Navarro et al., 2017). Therefore, concussion prevention remains a prevalent focus of sport policy changes and protective equipment innovations in the sport.

Even in the absence of diagnosed concussions, American football athletes are prone to sustaining repeated, subconcussive head impacts during their regular practices and competitions (Karton et al., 2020; Cecchi et al., 2021; Choi et al., 2022; Marks et al., 2022). Subconcussive head impacts, broadly, are those which are not of great enough magnitude to result in a clinically diagnosed concussion, but may still contribute to detectable short or long-term health effects (Mainwaring et al., 2018). The accumulation of subconcussive head impacts has been associated with both acute and chronic neurological consequences (Mainwaring et al., 2018), including development of neurodegenerative disease (Russell et al., 2021). Further, in American football players specifically, subconcussive head impacts have been associated with various indicators of brain changes, including those observed via imaging studies (Davenport et al., 2016; Foss et al., 2019), changes in oculomotor function (Joseph et al., 2019), and changes in levels of serum biomarkers of brain injury (Joseph et al., 2018). Therefore, attenuation of subconcussive impacts, in addition to concussion prevention, has become a recent priority in improving long term athlete brain health.

In an effort to reduce the risk of head and brain injury, protective helmets are required to be worn at all levels of American football competition. Helmets have evolved dramatically since their first introduction to the sport (Levy et al., 2004; Viano and Halstead, 2012) and presently consist of a variety of shock absorbing technologies with differing mechanisms for energy absorption and force attenuation (Hoshizaki et al., 2014; Dymek et al., 2022). Notably, some modern technologies include viscoelastic foams, buckling beams and structures, gas chambers, and 3D printed lattices. Helmet impact velocities range considerably in American football (Bailey et al., 2020a); to best protect an athlete from concussive and subconcussive head impacts, a helmet should be designed such that it can meaningfully attenuate impacts of both low and high velocities. However, current testing standards and rankings for helmets place emphasis on the ability of helmets to attenuate the magnitude of impacts associated with diagnosed concussions or more serious injuries (i.e., skull fractures) (Rowson and Duma, 2011; Bailey et al., 2020b; NOCSAE, 2021). While it appears that progress has been made in reducing the incidence of concussion and skull fractures as a result of some of these testing protocols (Viano and Halstead, 2012; Bailey et al., 2020a), a concern exists that these protocols may result in helmets only being optimized for peak performance upon high velocity impacts representative of concussions. An ideal helmet shock absorber would perform optimally across the entire range of impact velocities that an athlete is exposed to, inclusive of both concussive and subconcussive impacts.

An ideal shock absorber performs fundamentally different from existing foams, solid structures, and gas-based technologies. In practice, many of these existing technologies exert a force based on the amount they are compressed, leaving them prone to high spikes in reaction force when they reach maximum compression or "bottom out" (Fanton et al., 2020). The impact force profile of an ideal shock absorber, on the other hand, displaces through its full stroke at a constant force, which scales based on the speed and energy of an impact speed (Baumeister et al., 1997; Spinelli et al., 2018; Vahid Alizadeh et al., 2022). Research has suggested that, in the context of a protective helmet, a constant force profile is optimal for preventing brain injury (Vahid Alizadeh et al., 2021). While gases and solids have yet to achieve such a response that is capable of scaling with impact velocity, prototype and computational models of liquid shock absorbers have demonstrated promising results on achieving a force profile near to that of an ideal shock absorber (Fanton et al., 2020; Vahid Alizadeh et al., 2021; Vahid Alizadeh et al., 2022).

In addition to being used for modeling of shock absorbing technologies, finite element (FE) modeling has been used to investigate brain injury risk and advance the state-of-the-art of helmet technology (Dymek et al., 2022). Validated FE models of helmets and laboratory testing equipment have been developed to simulate laboratory impact tests of helmets (Bustamante et al., 2019; Corrales et al., 2020; Decker et al., 2020; Giudice et al., 2019; Giudice et al., 2020; Vahid Alizadeh et al., 2021), enabling researchers and helmet manufacturers to rapidly iterate designs and estimate the performance of helmets before engaging in large scale manufacturing. Further, the head kinematics resulting from these simulated impacts can be used as input to validated FE models of the human head and brain to estimate the brain strain resulting from impacts (Madhukar and Ostoja-Starzewski, 2019). Use of these computational tools can reduce cost, time, and difficulty in bringing new helmet technologies to market and aid in identifying the most promising technologies to prevent brain injury. In a previous study, an FE model of a helmet design utilizing theoretically idealized liquid shock absorber elements suggested a dramatic reduction in concussion risk was possible with this technology, but a method of manufacturing such a helmet was not explored and performance under lower velocity impacts was not studied (Vahid Alizadeh et al., 2021). Development of further, more advanced FE models of liquid shock absorbing technology

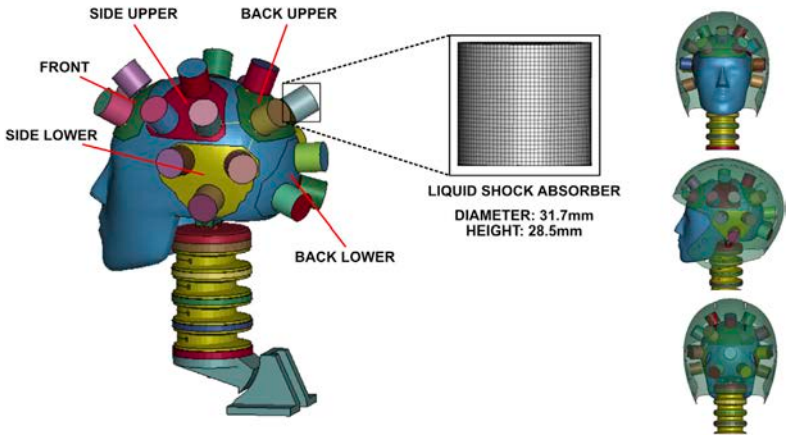

FIGURE 1
Arrangement of liquid shock absorbers in helmet finite element model.

could enable full helmet systems to more quickly reach consumers and reduce injury risk among the American football athlete population.

The objective of this research study was to use FE modeling to investigate the potential for a liquid-based shock absorber to improve the ability of American football helmets to attenuate the severity of both concussive and subconcussive head impacts in American football. To do this, we developed a helmet assembly with liquid-based shock absorbers integrated throughout it that would be feasible for physical re-creation. Further, we used a FE model of the human head and brain to determine not only the effect of this technology on brain injury risk metrics based on head kinematics, but also on the resulting brain strain from the simulated impacts.

Materials and methods

Liquid shock absorber design

The liquid shock absorber utilized throughout this study was modeled as a cylinder that can decelerate an impact mass in its axial direction (Figure 1), which was inspired by the cylindrical liquid shock absorber originally modeled by Vahid Alizadeh et al. (2022). In an impact, the top surface of the cylinder is pressed downwards, and the liquid inside is forced to flow through the orifice. The liquid passing through the orifice yields a pressure drop before and after the orifice, defined by Eq. 1:

$$\Delta p = \frac{\rho Q^2}{2C_d^2 A_o^2} \tag{1}$$

Where ρ is the fluid density, Q is the volumetric flow rate, C_d is the orifice discharge coefficient, A_o is the orifice area, and Δp is the pressure differential, $p - p_o$, where p is the fluid pressure inside the liquid shock absorber and p_o is the atmospheric pressure downstream of the orifice. Because the pressure at the outlet of the orifice is the same as the atmospheric pressure, the inner pressure of the shock absorber is higher, which can yield a resultant force to decelerate an impact mass. Furthermore, it should be noted that the pressure drop is also decided by the orifice area, which indicates that the resultant force can be easily tuned by modifying the orifice area.

A plain-weave high-strength fabric is used as the side wall of the shock absorber to constrain localized bulging and avoid rupturing of the side wall during impact while also allowing the shock absorber to be fully compressed. Carbon fiber shells are used for the top and bottom end caps of the cylinder to provide lightweight, rigid constraints at both ends of the shock absorber, enabling it to hold its cylindrical shape during impact. Water is used to fill the shock absorber and dissipate impact energy. The fabric side wall and carbon fiber end caps are nonporous, and the connection between the fabric and the end caps are modeled as sealed connections; therefore, fluid can only be discharged through the orifice. The orifice area is tuned to optimize helmet performance, which will be introduced later.

The height of the shock absorber was set to 28.5 mm, which was chosen to ensure that it could fit within the space between the scalp of the head and the shell of an American football helmet. To avoid axial buckling during impact, a larger diameter of the cylindrical shock absorber is better. However, smaller diameters are also favorable, considering that the shock absorbers are distributed across the scalp, which is a curved surface. Therefore, based on these two considerations, a diameter slightly larger than the height (31.7 mm) was adopted. A thickness of 0.6 mm was adopted for the side wall fabric, and a thickness of 2 mm was used for both end caps.

The liquid shock absorber was modeled as quadrilateral shell elements in LS-DYNA. Because the fabric can be bent freely, the side wall of the shock absorber was modeled by fully integrated Belytschko-Tsay membrane shell elements, which only have one integration point in the thickness direction and neglect the bending effect. Furthermore, multiple material angles were included to represent the different weaving directions of the fibers. *MAT_FABRIC was used to model the fabric material, which has a density of 777 kg/m^3, longitudinal modulus of 76 GPa, transverse modulus of 0.6 GPa, and shearing modulus of 0.8 Gpa (Vahid Alizadeh et al., 2022). The side wall was meshed by 1,292 elements with an average length of 1.5 mm. The

top and bottom caps were modeled by Belytschko-Tsay shell elements with two nodes in the thickness direction. The *MAT_ELASTIC was adopted to model the material, which has a density of 2000 kg/m^3, Young's modulus of 200 GPa, and Poisson ratio of 0.23. A cap was meshed by 292 elements with an average length of 1.5 mm. The side wall and end caps were connected by sharing nodes.

The effect of the orifice was modeled by *AIRBAG_LINEAR_FLUID_ID, which exerted pressure on the internal surface of the shock absorber according to the mass rate of the flow (Eq. 2):

$$\dot{m}_o = sign(\Delta p) C_d A_o [2\rho \Delta p]^{1/2} \quad (2)$$

As mentioned earlier, Δp is the pressure differential, $p - p_o$, where p is the pressure inside the shock absorber and p_o is the atmospheric pressure downstream of the orifice, or the back pressure. This back pressure plays the role of returning the fluid to the shock absorber after an impact has occurred. Upon the completion of impact loading, the pressure inside the shock absorber, or the upstream pressure, is lower than the back pressure and this negative pressure differential returns the fluid back into the shock absorber.

An internal incompressible fluid with water properties was used as the liquid inside the shock absorber (bulk modulus is 2.2 Gpa, density is 1,000 kg/m^3), and a discharge coefficient of 0.7 was used (Fanton et al., 2020; Vahid Alizadeh et al., 2022). The water was modeled as a control volume and no mesh was associated with the fluid part, but the inertia of the liquid was included. The orifice area is a key parameter determining the impact dissipation performance of the helmet. Orifice areas ranging from 10 to 200 mm^2, in increments of 5 mm^2, were adopted homogeneously for all shock absorbers in the helmet model and subjected to the NFL helmet test protocol (Bailey et al., 2020b, details of this test protocol are described in the following *Helmet Performance Testing* section). The Helmet Performance Score (HPS, see the calculation of HPS in the following *Helmet Performance Testing* section) was calculated for every orifice area to represent the risk of brain injury when the player is wearing that helmet. The HPS was found to increase when the orifice was too small (<40 mm^2) or too large (>70 mm^2) and remained relatively constant within these bounds (Supplementary Figure S1). Therefore, we adopted an orifice area of 59 mm^2, which corresponded the lowest level of HPS when evaluating HPS in orifice area increments of 1 mm^2 between 40 mm^2 and 80 mm^2 (Supplementary Figure S2). It should be noted that small variations could be observed between simulations of the same orifice area when evaluated in the two separate optimizations, likely due to small numerical errors.

Helmet assembly

A previously validated, open-sourced FE model for the Vicis Zero1 helmet (Giudice et al., 2020) was used as the base model for our full helmet assembly, and we modified the VICIS Zero1 helmet to test the helmet performance improvement afforded by integration of liquid shock absorbers. The original Vicis Zero1 dissipates impact energy by the buckling of elastic beams, which were removed. The material of the outer helmet shell was changed to a carbon fiber (with the same properties as the shock absorber end caps). The carbon fiber was substantially stiffer than the original helmet material and, therefore, could involve more shock absorbers during the impact due to its limited local deformation. The remaining parts of the helmet were kept the same as the original helmet model: the original facemask, chin pad, and chin strap were adopted in the liquid helmet model.

To integrate the cylindrical liquid shock absorbers into the full helmet system, we first fixed three shock absorbers onto a triangular bottom shell made of 2 mm thick carbon fiber (forming a "tripad"). To fix a shock absorber on the bottom shell, a set of 8 nodes evenly distributed about the circumference of the bottom cap of the shock absorber were rigidly connected to 8 nearby nodes in the bottom shell by *CONSTRAINED_NODAL_RIGID_BODY, which constrains every degree of freedom of nodes to be the same. We used seven tripad assemblies spread out throughout the helmet to protect the head from impacts at various locations: one at the front, one at the lower back, one at the upper back, and a lower and upper side tripad on each side of the helmet (Figure 1). This resulted in 21 liquid shock absorbers integrated throughout the helmet. For each of the tripads, the geometry of the bottom carbon fiber shell was decided by the skull, such that the surface of the Hybrid III ATD headform was extracted and cut into the triangular shells. Then, the triangular shells were offset against the skull as the bottom shell for the tripads. The exact shape of the shock absorber tripad shell and the location of shock absorbers within each tripad assembly varied such that shock absorbers would be distributed evenly around the skull and symmetrically with respect to the sagittal plane of the head. Finally, adjacent tripads were connected by elastic beams with a cross-sectional area of 19.6 mm^2. A relatively soft material (Young's modulus: 10 GPa, density: 1,631 kg/m^3, Poisson ratio: 0.4) was used for the elastic beams, allowing the tripads to shift for better fitting to the head. The total mass of the helmet was determined to be 2.03 kg.

Helmet Performance Testing

The single-precision solver of LS-DYNA (ls-dyna_smp_s_r1010_x64_redhat5_ifort160) was used to perform all helmet simulation experiments. The efficacy of the liquid helmet was tested with a validated, open source model of a linear impactor and 50th percentile male Hybrid III anthropomorphic test device headform (Giudice et al., 2019). The model used was made to replicate the equipment used in the NFL's Helmet Test Protocol (Bailey et al., 2020a). In these tests, a Hybrid III headform is equipped with a helmet and is impacted at a controlled speed by a ram. The impactor consists of a hard end cap and a soft foam, which is meant to represent another helmeted head. The total mass of the impactor is 15.6 kg. The headform was connected to a 50th percentile male Hybrid III neck, which was fixed to a plate that can only slide freely in the direction of the impact ram. The relative location between the impactor and headform, as well as the angles of the headform, could be adjusted to achieve different impact locations and directions. A set of gyroscopes and accelerometers were modeled at the center of the headform to measure the six-degree-of-freedom head kinematics (i.e., angular velocities and linear accelerations) resulting from impacts. The whole testing process has been modeled in FE (Giudice et al., 2019) and is also used in evaluating physical football helmets (Bailey et al., 2020b).

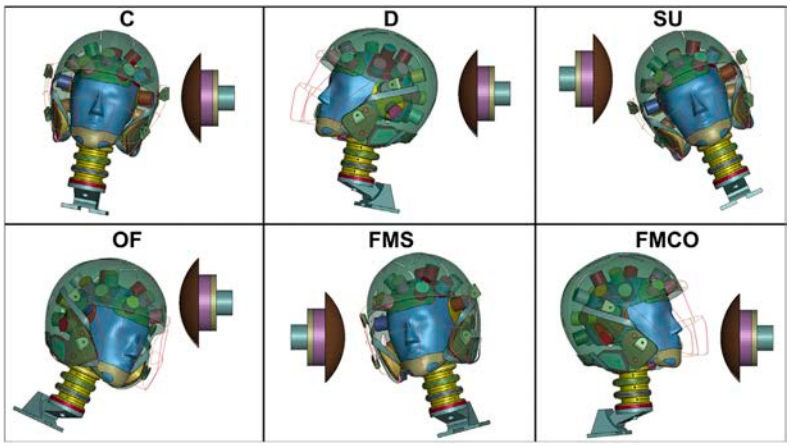

FIGURE 2
Impact testing locations. C: Side, D: Oblique Rear, SU: Side Upper, OF: Oblique Front, FMS: Facemask Side, FMCO: Facemask Central Oblique.

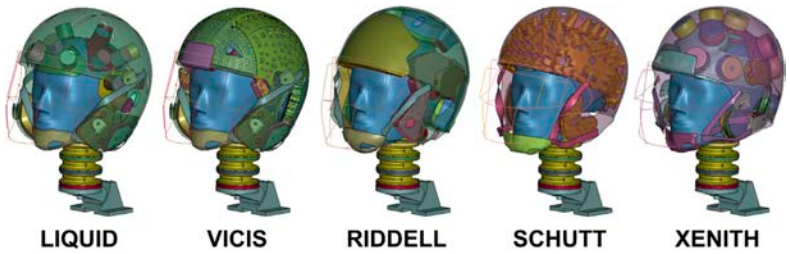

FIGURE 3
Finite element models of American football helmets tested in simulation. Each model is fit to the 50th percentile male Hybrid III head and neck finite element model. Helmet shells are set to 50% transparency for visualization of interior shock absorbing technology.

Testing was completed according to the NFL's Helmet Test Protocol. In this test protocol, there are six impact locations: Oblique Front (OF), Side (C), Side Upper (SU), Oblique Rear (D), Facemask Side (FMS), and Facemask Central Oblique (FMCO) (Figure 2) and three impact speeds: 5.5, 7.4, 9.3 m/s (Bailey et al., 2020a). Considering that this test protocol was developed primarily to replicate concussive head impacts, three lower speeds (1.6, 3.4, 5.0 m/s) representative of average impact velocities (inclusive of injurious and noninjurious impacts) (Bailey et al., 2020b) were also included for testing, and are referred to as subconcussive impact tests. In addition to the liquid helmet model designed in this study, four previously created, open-source football helmet FE models were also tested under the same impact conditions, including: the Riddell Revolution Speed Classic, the Vicis Zero1 (Giudice et al., 2020), the Schutt Air XP Pro (Decker et al., 2020), and the Xenith X2E (Corrales et al., 2020) (Figure 3). The masses of each of these helmets were measured in LS-DYNA as 1.86, 2.12, 1.71, and 1.74 kg, respectively.

Similar to the NFL Helmet Test Protocol, Head Injury Criterion (HIC) (Versace, 1971) and DAMAGE (Gabler et al., 2019) and HARM (Bailey et al., 2020a) were calculated for all impact conditions. These metrics were calculated using the head kinematics filtered with a cut-off frequency of 300 Hz. In three impact cases, the VICIS helmet model erroneously terminated prior to the completion of the impact event and therefore these cases have lower values of injury risk metrics than if the simulation had completed in full; these cases are labeled in the appropriate figures. The percentage reduction in HARM afforded by the liquid helmet relative to each existing helmet model was calculated. A Helmet Performance Score was calculated for each helmet using a weighted sum of HARM values from the concussive impact tests, with each HARM value being weighted according to the NFL's Helmet Test Protocol (Bailey et al., 2020b).

In addition to these kinematics-based metrics, we also calculated the expected strain on the brain tissue to show how different helmet technologies affect the human brain. In this study, the Global Human Body Model Consortium (GHBMC) head and brain model was used (Mao et al., 2013). The double-precision solver of LS-DYNA (ls-dyna_smp_d_r1010_x64_redhat5_ifort160) was

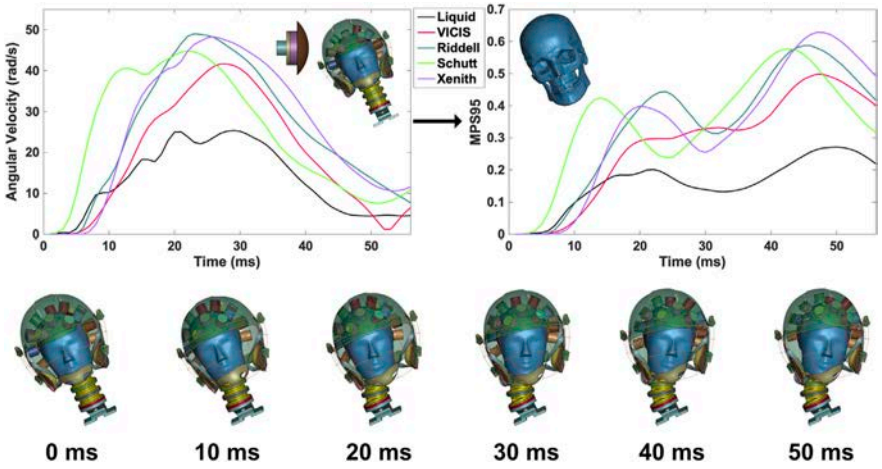

FIGURE 4
Example of angular velocity time histories from helmet impact simulations (top left) that are used as input to the GHBMC head and brain model to compute 95th percentile maximum principal strains (top right). The headform and helmet assembly at various time points during the impact process are shown (bottom). All data shown is from the Side Upper impact location at 9.3 m/s.

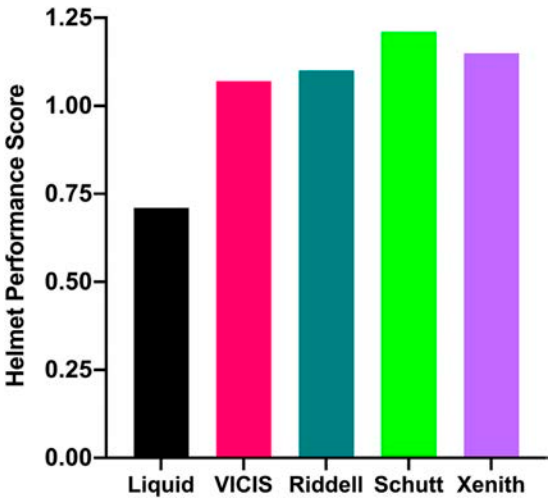

FIGURE 5
Helmet Performance Scores calculated from the NFL Helmet Test Protocol's concussive impact tests for each of the five helmets tested.

used to perform all brain strain simulations. Its skull was modified to act as a rigid body. The filtered head kinematics were assigned to the rigid skull as the loading to deform brain tissue, and the 95th percentile maximum principal strain (MPS95) across the whole brain was calculated for every time point of each simulation (Figure 4). The 95th percentile of maximum principal strain across the whole brain was used instead of the maximum principal strain to avoid the influence of extremely high values caused by numerical errors. Then, peak values of MPS95 across the full time history of each impact were recorded to represent the severity of brain deformation.

Results

Overall, the liquid helmet yielded the lowest Helmet Performance Score (0.71), compared to the other four helmet models (VICIS = 1.07, Riddell = 1.10, Schutt = 1.21, Xenith = 1.15) (Figure 5). The liquid helmet yielded the lowest value of HARM at 33 of the 36 tested impact conditions, with 3 velocities at the FMCO location being the only conditions in which the liquid design did not outperform all of the existing helmet models. Relative to all helmet models, HARM reductions afforded by the liquid helmet averaged 32.0% (range: -2.2%–50.5%) at concussive velocity impact cases and averaged 33.0% (range: -37.5%–56.0%) at subconcussive velocity impact cases (Figure 6).

MPS95 values for each helmet at each impact condition can be found in Figure 7A, 7B, for the concussive and subconcussive impact tests, respectively. HIC, DAMAGE, and HARM values for each helmet at each impact condition can be found in Supplementary Figures S3, S4, for the concussive and subconcussive impact tests, respectively.

Discussion

In this study, we developed a FE model of a helmet featuring liquid shock absorbing technology with the goal of attenuating the severity of concussive and subconcussive head impacts in American football. Compared with four existing FE helmet models, our simulation results demonstrate that liquid shock absorbers have the potential to provide a considerable reduction in kinematics-based brain injury criteria across a wide range of impact velocities and impact locations. Using a FE model of the human head and brain to investigate the effects of the liquid helmet on resulting brain strains also suggested that dramatic attenuations of impact severity could be achieved, which could be meaningful when considering brain injury risk.

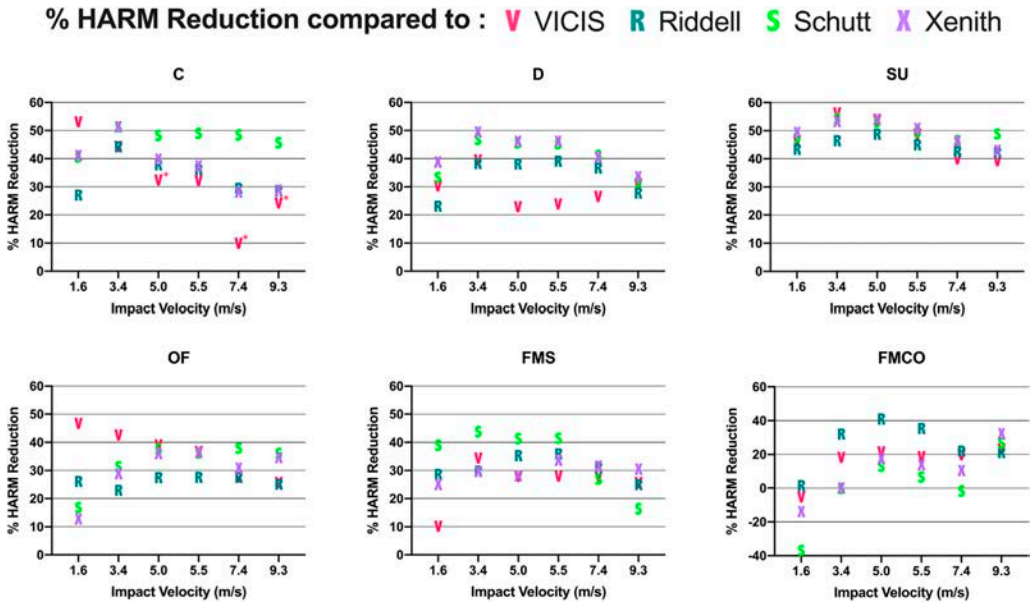

FIGURE 6
Reductions in HARM values afforded by the liquid helmet, relative to each existing helmet model at each impact condition. Positive values indicate that the liquid helmet had a lower value of HARM relative to a given helmet, while negative values indicated that the liquid helmet had a higher value of HARM. Asterisks denote instances in which impact simulations of the VICIS helmet model erroneously terminated prior to the completion of the impact event. C: Side, D: Oblique Rear, SU: Side Upper, OF: Oblique Front, FMS: Facemask Side, FMCO: Facemask Central Oblique.

The liquid helmet model we developed yielded a substantially lower value of Helmet Performance Score than any of the other FE helmet models tested, reducing Helmet Performance Score by approximately 34% compared to the best previously existing FE helmet model. Helmet Performance Score is the primary metric by which helmets are ranked for safety according to the NFL (Bailey et al., 2020a), and it is used to create an annual ranking of helmets that is published to all NFL athletes and the general public each year. Helmet Performance Score is an aggregate metric that combines the performance of helmets at various impact conditions by weighting their value of HARM based on relative risk of concussion incidence and then summing them together. The highest weighted impact location in the Helmet Performance Score is the Side Upper location, due to its high incidence of concussion in elite game play (Lessley et al., 2018). The liquid helmet model performed particularly well at this location, offering improvements in HARM by a range of 39%–50% across all concussive impact velocities, thus driving a lower Helmet Performance Score. However, the liquid helmet did not seem to compromise performance at concussive velocities at any location as a result of this highly improved location, offering HARM reductions at all but one of the concussive impact conditions (FMCO 7.4 m/s, 2% HARM increase relative to the Schutt helmet). Helmet Performance Score, and the HARM, HIC, and DAMAGE values from which it is derived, may be meaningful metrics to consider when attempting to reduce injury incidence on the field; a recent study found that Helmet Performance Score calculated from laboratory tests of helmets was associated with on-field concussion incidence in NFL game play (Bailey et al., 2020b). According to the data provided in that study, a Helmet Performance Score of 0.71 would represent a substantial improvement upon existing helmets. It should be noted that Helmet Performance Score is designed specifically for the NFL, and further investigation is warranted to determine how our findings would translate to a metric like Helmet Performance Score that was made for the youth, high school, and collegiate levels of game play. Nonetheless, other studies have found that helmets yielding improved laboratory performance (as defined by reductions in kinematics-based brain injury risk metrics) have been associated with reductions in on-field concussion incidence (Collins et al., 2006; Rowson et al., 2014) and preservation of brain white matter (Diekfuss et al., 2021) at levels of game play lower than the elite, professional level.

In American football, the number of subconcussive impacts athletes sustain far outweighs the number of diagnosed concussions in the sport (Cecchi et al., 2021; Choi et al., 2022; Marks et al., 2022). However, despite the prevalence of low velocity impacts on the field, helmets for this sport undergo limited testing for their performance upon low velocity impacts. The accumulation of subconcussive impacts has been associated with functional and microstructural changes in the brains of male athletes (Mainwaring et al., 2018), and in some cases has been associated with the development of neurodegenerative disease (Russell et al., 2021). In our study, we found that at different impact locations, the relative performance of existing helmets often changed according to the impact velocity, with some dramatic changes in relative performance at extreme ends of the tested velocities (e.g., Schutt helmet at FMS location). This suggests that the shock absorbing technologies in the existing helmets may benefit from improvements to ensure a reliable impact response across the wide range of low- and high-velocity impact conditions. Similar to concussive impacts, the liquid helmet

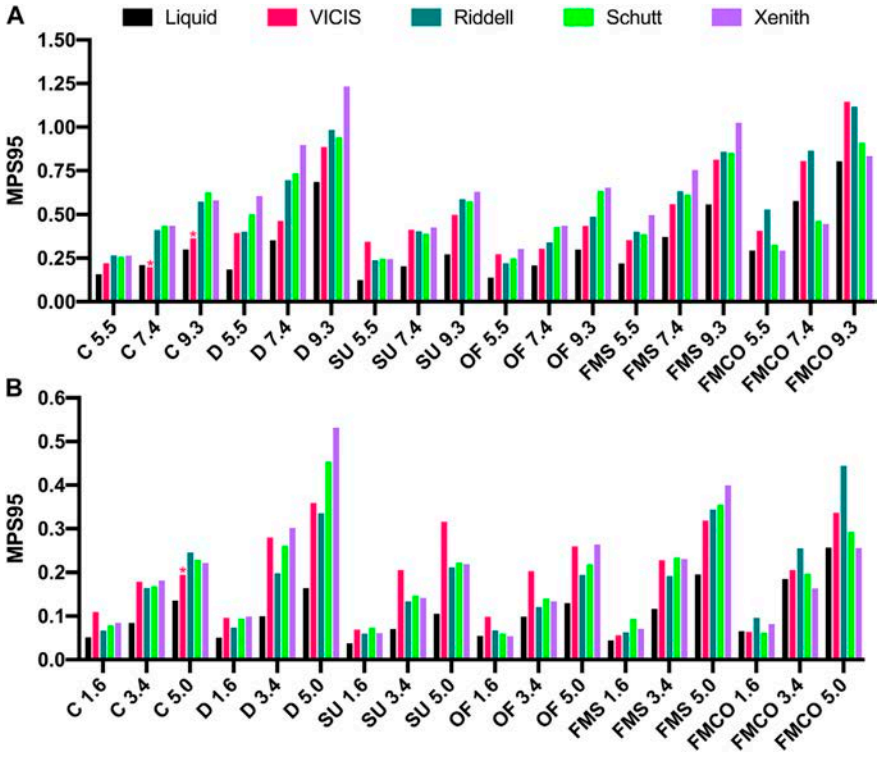

FIGURE 7
95th percentile maximum principal strain calculated via the GHMBC head and brain model for **(A)** concussive and **(B)** subconcussive impacts. Asterisks denote instances in which impact simulations of the VICIS helmet model erroneously terminated prior to the completion of the impact event. C: Side, D: Oblique Rear, SU: Side Upper, OF: Oblique Front, FMS: Facemask Side, FMCO: Facemask Central Oblique.

we developed offered large reductions in kinematics-based brain injury risk metrics at nearly all subconcussive impact conditions. These reductions could be meaningful for athlete brain health, as the magnitude of head kinematics resulting from subconcussive head impacts has been associated with various indicators of brain structure and function in American football athletes (Joseph et al., 2018; Joseph et al., 2019; Bartsch et al., 2020). The ability of the liquid technology to outperform existing helmet models across such a wide range of impact velocities (1.6 m/s to 9.3 m/s) is likely owed to the ability of liquid-based shock absorbers to scale their force response with impact velocity, rather than displacement as many foam and gas shock absorbers do (Vahid Alizadeh et al., 2021). In this respect, the liquid shock absorbers do not have to be tuned for a single velocity or a narrow range of velocities, and can perform efficiently across many impact velocities. Also similar to the concussive impacts, the only conditions in which the liquid helmet did not outperform all existing helmet models was at the FMCO location. At this impact location, the chinstrap is in tension and a portion of the impact force is transferred to the facemask. The Schutt helmet performed best of all the helmets at the 1.6 m/s FMCO impact condition, suggesting it had a facemask and chinstrap assembly that was optimized for such impacts. Low velocity impacts to the front of the facemask are particularly common for linemen (Bailey et al., 2021). Future designs of liquid shock absorbers and other helmet technologies should not be limited to interior shock absorbers only, and should consider the facemask, chin strap (Spinelli et al., 2018), and other regions of the helmet for potential safety improvements.

Kinematics from the simulated impacts were used as input to the GHBMC FE model of the human head and brain (Mao et al., 2013) in order to calculate brain strains. Similar to the kinematics-based brain injury risk metrics, MPS95 was reduced in nearly all of the concussive and subconcussive impact conditions. These reductions could be clinically meaningful in improving brain health in American football players. Using the GHBMC FE model and a combination of human and primate brain injury data, Wu et al. (2022a) proposed a 50% risk threshold for mild traumatic brain injury of 0.360 MPS95. In our dataset, eleven of the concussive NFL impact conditions resulted in all four of the existing FE helmet models exceeding this threshold. However, the liquid helmet only resulted in an MPS95 value of 0.360 or greater in five of the impact conditions, suggesting that utilizing a liquid helmet could result in a substantial reduction in expected concussions in the NFL helmet test. Using different brain injury criteria and a different helmet test methodology, a previous FE model of a football helmet featuring liquid shock absorbers came to similar conclusions that expected concussions could be dramatically reduced relative to the same open-source FE helmet models (Vahid Alizadeh et al., 2021). For milder head impacts, recent data has proposed that blood brain barrier disruption can occur in sports in the absence of any diagnosed brain injury (O'Keeffe et al., 2020).

Although a strain threshold for blood brain barrier disruption based on the GHBMC model has not yet been proposed, Shreiber et al. (1997) proposed a maximum principal strain threshold of 0.188 for blood brain barrier disruption based on FE modeling of a rat brain. In our subconcussive impact tests, the four existing helmet models exceeded an MPS95 value of 0.188 in eight of the impact conditions, while the liquid helmet model only exceeded this value in two of the impact conditions. While these results are encouraging, they should be interpreted with caution, as definitive thresholds for mild traumatic brain injury and blood brain barrier disruption remain elusive, and thresholds based on strain are model-dependent.

Previous research has demonstrated that collapsible liquid shock absorbers are capable of providing a near-ideal force profile that scales with impact velocity (Fanton et al., 2020; Vahid Alizadeh et al., 2022). The liquid shock absorber utilized in this study was inspired by a previous cylindrical shock absorber designed by Vahid Alizadeh et al. (2022), which exerts a reaction force on an impact mass while a fluid contained within the shock absorber is passed through an orifice. However, various material properties, the external dimensions, and orifice size were modified from this previous model for the purposes of integration to a football helmet. Although other designs of liquid shock absorbers, such as "volcano" or telescoping designs, have been shown to yield higher force efficiencies than a cylindrical design in uniaxial, individual unit tests (Fanton et al., 2020; Vahid Alizadeh et al., 2022), it is unclear if these results would translate to a full helmet system. The efficiency of those designs is highly dependent on a precise, variable contact area, which may not be achieved when multiple shock absorbers are engaged in an oblique impact, for example, as would be experienced in a full helmet. Further, one of the purposes of our study was to design a FE model of a helmet that could reasonably be translated to a physical prototype; manufacturing such precise, variable contact areas with a concave side wall out of soft, collapsible fabrics could prove to be difficult.

There are a number of design aspects that should be considered in future FE helmet models featuring liquid shock absorbers. First, while the present study demonstrates the potential improvements afforded by liquid shock absorbers being incorporated throughout an entire helmet, benefits could still be granted by targeting a specific location of a helmet or combining liquid technology with other foams or structures. For example, the American football helmet industry is moving towards position-specific helmets (Lessley et al., 2020); designing a single helmet zone featuring liquid shock absorbers that is targeted towards attenuating frequent impacts unique to a specific position could enable a quicker path to commercialization while maintaining meaningful benefits for athlete safety. Further, our optimization of the orifice area in each liquid shock absorber was homogeneous throughout the entire helmet. Future designs could explore tuning orifice areas and geometries for each specific impact location and could optimize each orifice based on the level of play or player position. An exploration of varying shock absorber geometries and varying materials used for the tripad shells and elastic connections between shells would also be worthwhile. Above all, a liquid shock absorber has yet to be implemented into a physical embodiment of an American football helmet. Any future design directions should consider the product comfort, fit, robustness, and manufacturing costs that are standard and expected in the helmet industry. While we strived to create a helmet architecture that could be recreated in a physical prototype, not all of these aspects of design were heavily considered in the model's design.

Several limitations exist in the present study. First, time has passed since the FE models of the existing helmets were developed. New helmets have been manufactured that, in their physical embodiments, have outperformed the helmet models that were tested in FE, both in the NFL Helmet Test Protocol and other test methodologies. It remains unknown how much of an improvement our liquid helmet model would have offered if FE models of the latest, top-ranking helmets were available. Further, despite being modeled into the physics of the liquid shock absorber, we did not consider the actual mechanism which would return liquid to the shock absorber after an initial impact. American football helmets are built to withstand multiple impacts, with reconditioning of helmets typically occurring every one or two years. Therefore, a liquid shock absorber design would likely need to meet these needs of the football helmet industry by including a reservoir that contains and returns the fluid to the shock absorber after an impact; without a fluid return mechanism, the performance of the helmet would decrease substantially upon subsequent impacts. Inclusion of a fluid return reservoir may increase the mass of the helmet substantially. Despite this, increased mass has been associated with improved helmet performance in impact testing (McIver et al., 2023), so we do not anticipate that such a modification would have negatively influenced helmet performance. However, it remains unclear how much helmet mass, rather than the physics of the liquid shock absorber, may have contributed to the improved performance of the liquid helmet model relative to the Riddell, Schutt, and Xenith helmet models, which all had lower masses than the liquid helmet. Regardless, the liquid helmet still demonstrated considerable improvements in performance over the VICIS helmet, which had a greater mass than the liquid helmet, indicating that mass alone did not dictate helmet performance. Further, while increasing helmet mass seems to be associated with improved brain safety according to impact tests, it remains unclear how an increase in helmet mass would affect neck injury risk and athlete comfort. Head shapes and sizes vary amongst athletes, and will therefore likely place a different amount of prestress or pressure on the shock absorbers within a helmet. Similar to the other FE models and previous evaluations of these FE models, we did not consider this potentially meaningful aspect of the helmet's response to impact (Giudice et al., 2020). Finally, the GHMBC model used to calculate brain strains is limited in that it represents only a 50th percentile male. The benefits offered by liquid technology could vary among different brain sizes, which have been shown to yield different levels of injury risk under similar loading conditions (Wu et al., 2022b). The GHBMC is also just one of several currently available FE models of the human head and brain, with some research suggesting that other FE models more accurately represent human brain displacement measurements than the GHBMC (Miller et al., 2017).

Overall, the present study proposes a plausible architecture for a full helmet system featuring liquid shock absorbers. The results of FE simulations with this helmet and other helmet models suggest that liquid technology has the potential to lower the risk of football-related brain injury by attenuating kinematics-based brain injury criteria and brain strains across a wide range of impact velocities. These findings support the future fabrication of helmets featuring liquid shock absorbers to validate these simulation results.

Data availability statement

The raw data supporting the conclusion of this article will be made available by the authors, without undue reservation.

Author contributions

All authors contributed to conception and design of the study. HV developed the finite element model of the helmet. NC, HV, and YL optimized the model's performance. NC and YL performed the simulation experiments. NC and YL wrote the first draft of the manuscript. NC, HV, and YL wrote sections of the manuscript. All authors contributed to the article and approved the submitted version.

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Conflict of interest

DC has a financial interest in Savior Brain, Inc.

The remaining authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fbioe.2023.1160387/full#supplementary-material

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Youth Tackle Football and the Developing Brain

2015

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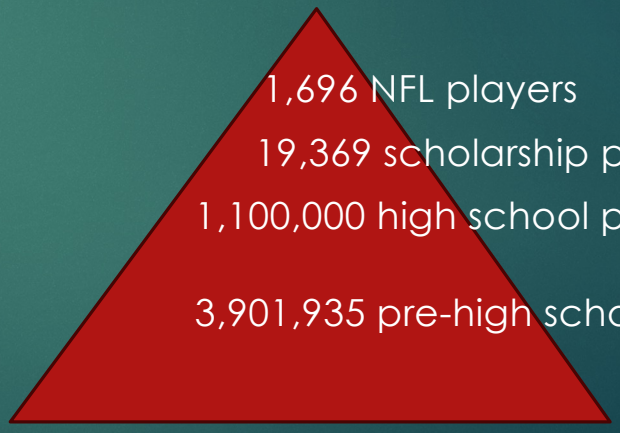
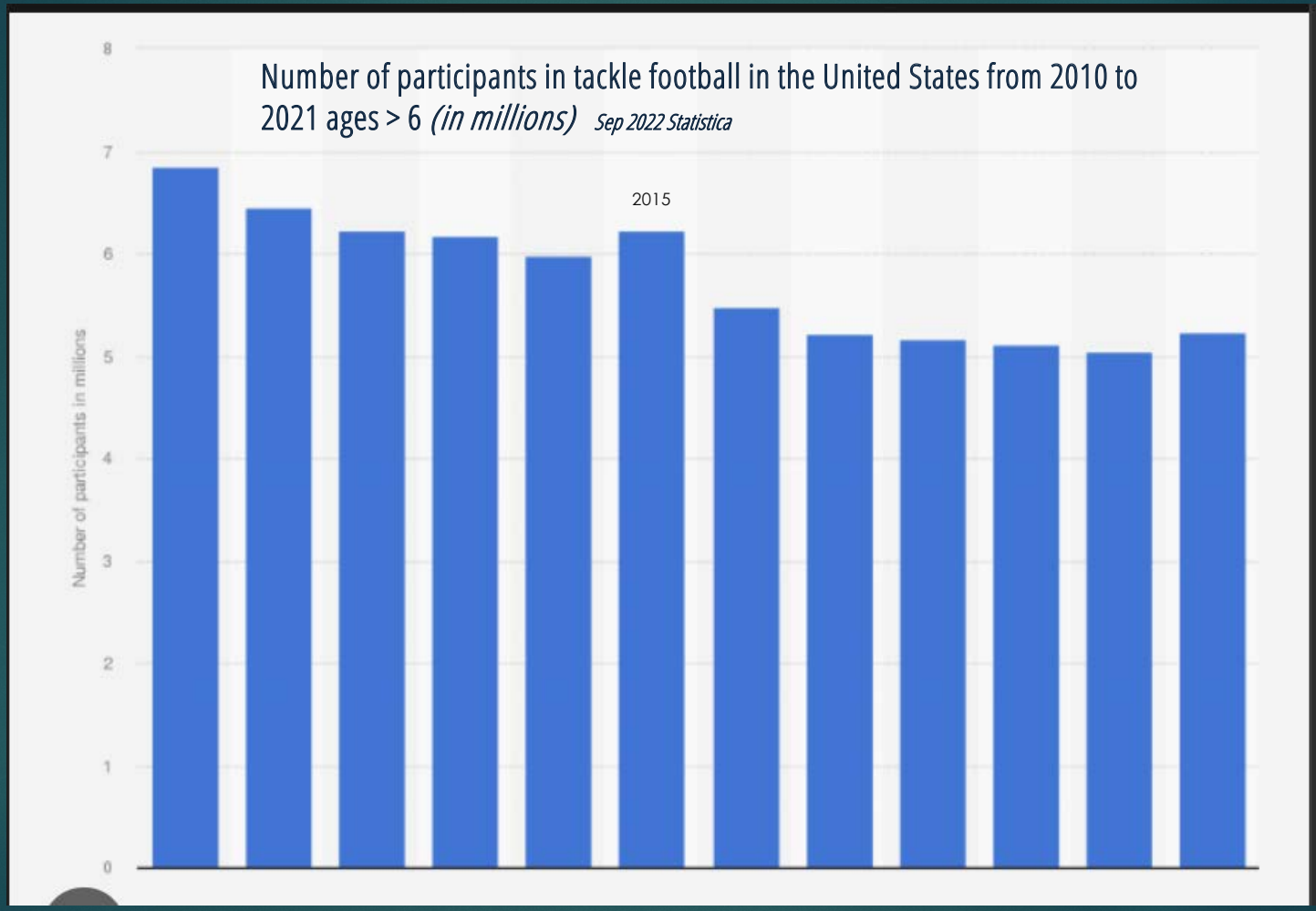
Pediatric Neurologist
President
California Neurology Society



5,023,000 total tackle football players >6 years old


Number of participants in tackle football in the United States from 2010 to 2021 ages > 6 (in millions) Sep 2022 Statistica
Number of participants in millions
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5
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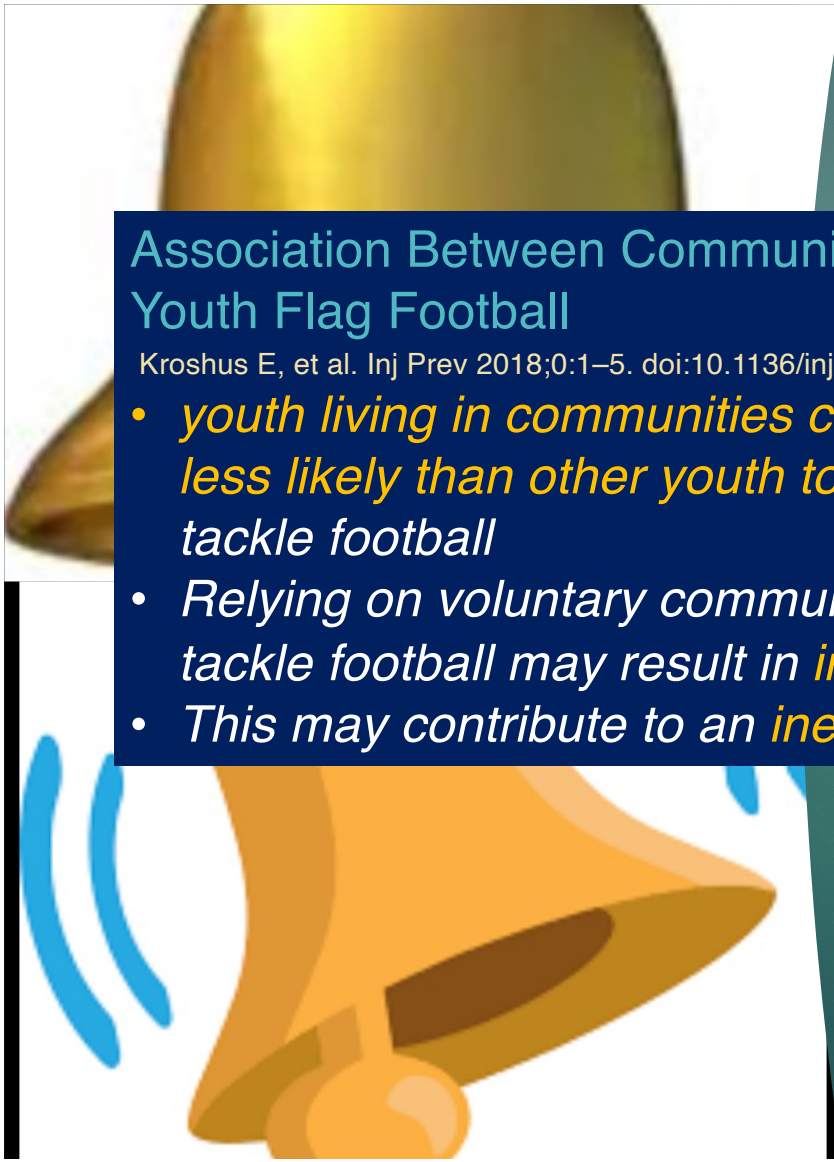
2015



BACKGROUND

Association Between Community Socioeconomic Characteristics And Access To Youth Flag Football

Kroshus E, et al. Inj Prev 2018;0:1–5. doi:10.1136/injuryprev-2017-042677

- *youth living in communities characterized by low educational attainment are less likely than other youth to have the option of a lower contact alternative to tackle football*
- *Relying on voluntary community-level adoption of lower contact alternatives to tackle football may result in inequitable access to such sport options*
- *This may contribute to an inequitable burden of brain trauma from youth sport*

- Youth athletes who play Tackle Football sustain 23 times more high-magnitude impacts per athletic exposure than a child who plays Flag Football *Waltzman et al, Sports Health* 13(5):454-462

High Magnitude Impacts may lead to Mild Traumatic Brain Injury (mTBI) and Concussion

m(TBI)

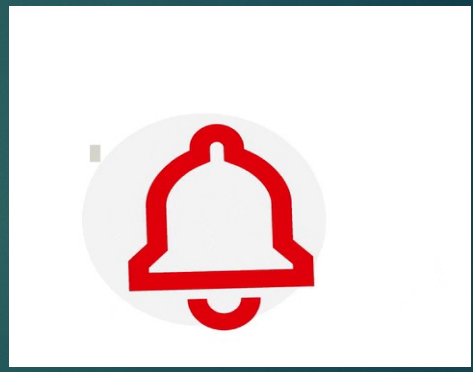
Single or **Repetitive** injury resulting in transient change of brain function *w/wo diagnosis of concussion*

- Repetitive injury → CTE (chronic traumatic encephalopathy)

concussion

Single or **cumulative** mTBI causing change in the child's:

- Behavior
- Learning ability
- Emotional well being
- Sleep
- Relationships

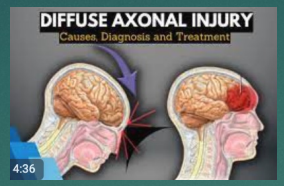


WHAT HAPPENS IN THE BRAIN

INJURY

- Concussion symptoms are clinical manifestation of microstructural brain injuries : diffuse axonal injury (includes *demyelination*)

- Axons are brain fibers in the **white matter** that **network** all brain regions

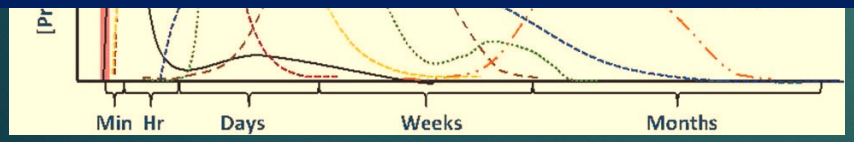


DEVELOPING BRAIN

- Genetic influences predominate in earlier years (0-6 years)
- White matter *myelination* is rapid in the first 5 years of life
- After age 5 *refinement and remodeling* occur *throughout childhood*, *adolescence* and into *young adulthood*

→ white matter development proceeds in response to environmental conditions ("white matter plasticity").

- **Normal white matter development** is positively correlated with educational & vocational attainment and favorable environmental influences
- ***Slower or impaired* white matter development** is associated with early deprivation and negative environmental influences



Wang et al (2013) from Bramlett & Dietrich J Neurotrauma 32:1834–1848 (2015)

mTBI as a risk factor for frontotemporal dementia.
Rosso et al., 2003; Kalkonde et al., 2012

RIVERMEAD POST-CONCUSSION SYMPTOMS QUESTIONNAIRE (RPQ)

Patient _____ DOI _____ Today's Date _____

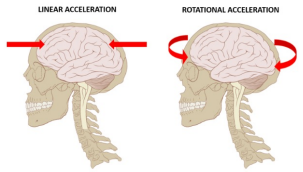
After a head injury or accident some people experience symptoms which can cause worry or nuisance. We would like to know if you now suffer any of the symptoms listed below. Compare yourself now with how you were before the accident and circle the number closest to your answer.

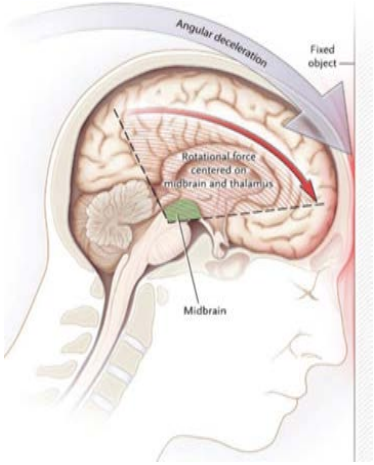
0 = Not experienced at all before or after the accident
1 = No more of a problem now than before the accident
2 = A mild problem for me now
3 = A moderate problem for me now
4 = A severe problem for me now

0 1 2 3 4	Headaches
0 1 2 3 4	Dizzy feelings
0 1 2 3 4	Nausea, upset stomach or vomiting
0 1 2 3 4	Noise sensitivity, or easily upset by loud noises
0 1 2 3 4	Sleep disburbance or disruption of sleep patterns
0 1 2 3 4	Fatigue, tiring more easily
0 1 2 3 4	Being irritable, easily annoyed or angered
0 1 2 3 4	Feeling depressed, tearful, crying easily or more emotional
0 1 2 3 4	Getting frustrated easily or being less patient with others
0 1 2 3 4	Poor memory or forgetting things
0 1 2 3 4	Difficulty concentrating
0 1 2 3 4	Taking longer to think
0 1 2 3 4	Blurry vision
0 1 2 3 4	Bright lights irritate or upset me, sensitive to bright lights
0 1 2 3 4	Double vision
0 1 2 3 4	Restlessness, have to move around, can't sit still
0 1 2 3 4	Other _____

Patient Signature _____ Doctor Signature _____

RPQ was originally published in the Journal of Neurology, Neurosurgery and Psychiatry in 1995 by King, Crawford et al from the Oxford Head Injury Service, Rivermead Rehabilitation Centre, Abingdon Road, Oxford, OX1 4XD, United Kingdom





Young athletes tend to have weaker necks and torsos = more susceptible

What happens in my clinic

Bigler 2008

Postconcussion symptoms (PCS)

- Follows head injury
- Usually resolve <3 months
- >95% normal routine CT/MRI

Persistent postconcussion symptoms (PPCS)

= *concussion symptoms persist beyond 3 months*

_ _ _

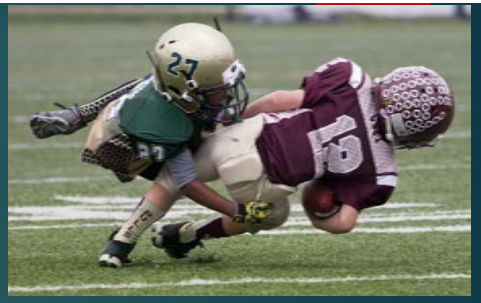
= up to 33% of **mTBI + head CT**

= up to 25% remain impaired at 12 months

Mcmahon et al, . *Neurotrauma* 31, 26–33. doi: 10.1089/neu.2013.2984

Further clinical investigations

Youth Tackle Football



Subconcussive Impact Exposure and White Matter Tract Changes over a Single Season of Youth Football

Radiology: Volume 281: Number 3—December 2016

- effects of subconcussive impacts on changes in specific white matter (WM) tracts as detected with MRI diffusion-tensor imaging
- statistically significant relationship between head impact exposure and WM changes ***in the absence of a clinically diagnosed concussion***

Age of first exposure (AFE) to American football and long-term neuropsychiatric and cognitive outcomes

Transl Psychiatry (2017) 7, e1236; doi:10.1038/tp.2017.197

- sample of **214 former American football players** compared longitudinally based on AFE <12 or >12
- those who began playing football before age 12 had
  - >2 X increased odds for clinically meaningful impairments in reported behavioral regulation, apathy and executive function
  - >3 X increased odds for clinically elevated depression scores
  - *independent of age, education and duration of football play

Younger AFE to football, in general, corresponded with worse behavioral regulation, depression, apathy and executive function, as well as increased odds for clinical depression and apathy

Age of First Exposure to Tackle Football and Chronic Traumatic Encephalopathy

Ann Neurol 2018;83:886–901

▶*Conclusion:* **Youth exposure to tackle football may reduce resiliency to late life neuropathology**

Table 3. Neuropathology Characteristics

	CTE+*	AFE < 12 (n = 84)	AFE ≥ 12 (n = 127)
Stage of CTE pathology, n (%)			
Stage I-II	61 (28.9)	31 (36.9)	30 (23.6)
Stage III-IV	150 (71.1)	53 (63.1)	97 (76.4)
Braak stage, n (%)			
Stage 0	45 (22.1)	28 (34.6)	17 (13.8)
Stage I	7 (3.4)	3 (3.7)	4 (3.3)
Stage II	23 (11.3)	9 (11.1)	14 (11.4)
Stage III	95 (46.6)	30 (37.0)	65 (52.8)
Stage IV	4 (2.0)	1 (1.2)	3 (2.4)
Stage V	13 (6.4)	0	13 (10.6)
Stage VI	17 (8.3)	10 (12.3)	7 (5.7)
CERAD, n (%)			
No neuritic plaques	128 (61.5)	60 (72.3)	68 (54.4)
Sparse neuritic plaques	52 (25.0)	13 (15.7)	39 (31.2)
Moderate neuritic plaques	16 (7.7)	5 (6.0)	11 (8.8)
Frequent neuritic plaques	12 (5.8)	5 (6.0)	7 (5.6)
ADNC, n (%)			
Not AD	118 (56.5)	59 (71.1)	59 (46.8)
Low ADNC	26 (12.4)	7 (8.4)	19 (15.1)
Intermediate ADNC	42 (20.1)	10 (12.0)	32 (25.4)
High ADNC	23 (11.0)	7 (8.4)	16 (12.7)
Lewy body, n (%) present*	48 (23.1)	17 (20.5)	31 (24.8)

After Life investigations

Table 1. Sample Characteristics of the Deceased Tackle Football Players

	CTE+ (N = 211)	No CTE (N = 35)	AFE < 12 (n = 84)	AFE ≥ 12 (n = 127)	*p-value
DEMOGRAPHIC/ATHLETIC					
Age of death, mean (SD) years	62.91 (17.91)	42.17 (22.40)	53.45 (16.91)	69.17 (15.72)	<0.001
Race, n (%) African American	41 (19.5)	4 (11.4)	19 (22.9)	22 (17.3)	0.32[b]
Education Level, n (%)					0.80[c]
Less than high school/some high school	2 (1.0)	4 (11.8)	1 (1.2)	1 (0.8)	
High school/GED	4 (1.9)	7 (20.6)	1 (1.2)	3 (2.4)	
Some college	47 (22.4)	11 (32.4)	20 (23.8)	27 (21.4)	
College degree	106 (50.5)	5 (14.7)	38 (45.2)	68 (54.0)	
More than college	10 (4.8)	0	4 (4.8)	6 (4.8)	
Graduate degree	41 (19.5)	7 (20.6)	20 (23.8)	21 (16.7)	
Years of football play, mean (SD)	15.07 (5.56)	8.07 (4.56)	16.73 (5.60)	13.97 (5.27)	<0.001
Decade of football play, mean (SD)[d]	5.78 (1.68)	7.74 (2.19)	6.42 (1.70)	5.36 (1.54)	<0.001
Age of first exposure to football, mean (SD)	11.84 (2.93)	11.26 (2.76)	8.79 (1.67)	13.86 (1.45)	<0.001
Highest level played, n (%) yes					<0.001[e]
High school	7 (3.3)	18 (51.4)	4 (4.8)	3 (2.4)	
College	64 (30.3)	8 (22.9)	37 (44.0)	27 (21.3)	
Semi-professional	2 (0.9)	4 (11.4)	1 (1.2)	1 (0.8)	
Professional	138 (65.4)	5 (14.3)	42 (50.0)	96 (75.6)	
Football primary position, n (%)					0.61[f]
Lineman (OL/DL)	72 (34.3)	10 (31.3)	27 (32.1)	45 (35.7)	
Linebacker	23 (11.0)	3 (9.4)	14 (16.7)	9 (7.1)	
Defensive back/safety	23 (11.0)	2 (6.3)	12 (14.3)	11 (8.7)	
Running back	34 (16.2)	1 (3.1)	9 (10.7)	25 (19.8)	
Quarterback	10 (4.8)	3 (9.4)	4 (4.8)	6 (4.8)	
Special teams	15 (7.1)	4 (12.5)	4 (4.8)	11 (8.7)	
Other/multiple	14 (6.7)	6 (18.8)	5 (6.0)	9 (7.1)	
Wide receiver	4 (1.9)	0	2 (2.4)	2 (1.6)	
Unknown	15 (7.1)	3 (9.4)	7 (8.3)	8 (6.3)	
Other contact sport history, n (%) any	34 (16.1)	8 (22.9)	14 (16.7)	20 (15.7)	0.86
Military history, n (%) yes	53 (25.2)	8 (22.9)	12 (14.3)	41 (32.5)	0.003
MEDICAL/LIFESTYLE, n (%)					
Heart attack	38 (21.2)	5 (16.7)	11 (15.3)	27 (25.2)	0.11
Hypertension	95 (53.4)	9 (30.0)	30 (42.9)	65 (60.2)	0.024
Elevated cholesterol	62 (35.2)	11 (36.7)	23 (32.9)	39 (36.8)	0.59
Diabetes	31 (17.2)	3 (10.0)	8 (11.4)	23 (20.9)	0.10
Stroke	23 (12.8)	4 (13.3)	3 (4.2)	20 (18.5)	0.005
Alcohol abuse	85 (40.7)	16 (45.7)	36 (43.4)	49 (38.9)	0.52
Illicit drug use	79 (37.6)	20 (57.1)	39 (47.0)	40 (31.5)	0.023
Steroid use	8 (5.8)	2 (10.0)	2 (3.6)	6 (7.1)	0.39
CLINICAL STATUS					
Age of cognitive symptom onset, mean (SD)	53.28 (17.12)	43.08 (20.48)	45.65 (16.14)	58.11 (15.99)	<0.001
Age of behavioral mood symptom onset, mean (SD)	46.28 (19.36)	35.54 (18.22)	38.97 (17.38)	50.96 (19.18)	<0.001
Tremor, n (%) yes	60 (28.7)	10 (28.6)	20 (24.1)	40 (31.7)	0.23
Falls, n (%) yes	73 (35.1)	9 (25.7)	20 (24.4)	53 (42.1)	0.009
Gait disturbance, n (%) yes	98 (46.9)	10 (28.6)	32 (38.6)	66 (52.4)	0.050

GET A HEADS UP ON

Football Helmet Safety

CDC HEADS UP
SAFE BRAIN. STRONGER FUTURE.

While there is no concussion-proof helmet, a football helmet can help protect your athlete from a serious brain or head injury. The information in this handout will help you learn what to look for and what to avoid when picking out a helmet for your football player.

Start with the Right Size:

BRING THE ATHLETE
Bring your athlete with you when buying a new helmet to make sure that you can check for a good fit.

HEAD SIZE
To find out the size of your athlete's head, wrap a soft tape measure around the athlete's head, just above their eyebrows and ears. Make sure the tape measure stays level from front to back. (If you don't have a soft tape measure, you can use a string and then measure it against a ruler.)

SIZES WILL VARY
Helmet sizes often will vary from brand-to-brand and with different models. Each helmet will fit differently, so it is important to check out the manufacturer's website for the helmet brand's fit instructions and sizing charts, as well as to find out what helmet size fits your athlete's head size.

Get a Good Fit:

GENERAL FIT
A football helmet should feel snug with no spaces between the pads and the athlete's head. The helmet should not slide on the head with the chin strap in place. If the helmet can be removed while the chin strap is in place, then the fit is too loose. Some helmets have a unique fitting system or use an air bladder system that requires inflation with a special needle to avoid puncturing the air bladders. You can find more information on fitting a football helmet on the manufacturer's website.

ASK
Ask your athlete how the helmet feels on their head. While it needs to have a snug fit, a helmet that is too tight can cause headaches.

HAIRSTYLE
Your athlete should try on the helmet with the hairstyle he will wear while at practices and games. Helmet fit can change if your athlete's hairstyle changes. For example, a long-haired athlete who gets a very short haircut may need to adjust the fit of the helmet.

COVERAGE
A football helmet should not sit too high or low on their head. To check, make sure the ear holes line up with the athlete's ears and the pad in the front of the helmet covers the athlete's head from the middle of his forehead to the back of his head.

VISION
Make sure you can see your athlete's eyes and that he can see straight forward and side-to-side.

CHIN STRAPS
The chin strap should be centered under the athlete's chin and fit snugly. Tell your athlete to open their mouth wide... big yawn! The helmet should pull down on their head. If not, the chin strap needs to be tighter. Once the chin strap is fastened, the helmet should not easily move in any direction, back-to-front or side-to-side. For helmets with a four point chin strap system, all four straps must be snapped and tightened as part of the fitting process.

Always follow the manufacturer's fitting instructions.

Take Care of the Helmet:

Athletes should NOT attempt to make any helmet or faceguard repairs themselves.

CHECK FOR DAMAGE
DO NOT allow your athlete to use a cracked or broken helmet or a helmet that is missing any padding or parts. For air bladder-equipped helmets, make sure to check for proper inflation. DO NOT alter, remove or replace padding or internal parts unless supervised by a trained equipment manager. Check for missing or loose parts and padding before the season and regularly during the season.

CLEANING
Clean the helmet often inside and out with warm water and mild detergent. DO NOT soak any part of the helmet, put it close to high heat, or use strong cleaners.

PROTECT
DO NOT let anyone sit or lean on the helmet.

STORAGE
Do not store a football helmet in a car. The helmet should be stored in a room that does not get too hot or too cold and where the helmet is away from direct sunlight.

DECORATION
DO NOT decorate (paint or put stickers on) the helmet without checking with the helmet manufacturer, as this may affect the safety of the helmet. This information may also be found on the instructions label or on the manufacturer's website.

Look for the Labels:

LOOK FOR A FOOTBALL HELMET WITH LABELS THAT:

- Say "MEETS NOCSAE Standard®"[1] as certified either by the manufacturer or by SEI[2]. That label means that the helmet model has been tested and meets NOCSAE performance and protection standards.
- State whether the helmet can be recertified. If not, look for the label that specifies when the certification to the NOCSAE standard expires.
- Specify how frequently the helmet must be reconditioned and recertified.
- Have the date of manufacture. This information will be helpful if the manufacturer has specified a useful life of the helmet, or has specified that the helmet may not be reconditioned and recertified, or if there is ever a recall on that particular model or year.

Know When to Replace a Football Helmet:

RECONDITIONING
Reconditioning involves having an expert inspect and repair a used helmet by: replacing missing parts, testing it for safety, and recertifying it for use. Helmets should be serviced regularly by a licensed NAERA2 member.

DO NOT allow your athlete to use a used helmet that has not been recently recertified for use by a NAERA reconditioner.

For a complete list of licensed recertifiers, visit the National Federation of State High School Associations (NFHS): https://www.nfhs.org/media/1018264/10-2020-nocsae-reconditioners.pdf.

Only companies that are licensed by NOCSAE® can recertify a helmet. All NAERA members are licensed by NOCSAE®".

10 AND OUT
Football helmets should be replaced no later than 10 years from the date of manufacture. Many helmets will need to be replaced sooner, depending upon wear and tear.

[1] National Operating Committee on Standards for Athletic Equipment: www.nocsae.org

[2] Safety Equipment Institute (SEI) is an independent and nationally accredited certification body: www.seinet.org

WHAT PARENTS
NEED TO KNOW ABOUT

BRAIN SAFETY AND YOUTH FOOTBALL

CDC HEADS UP

CDC

Football has the highest number of sports-related concussions and other traumatic brain injuries among youth.[1]

SOME PLAYS AND POSITIONS ON A FOOTBALL TEAM MAY PUT ATHLETES AT INCREASED RISK FOR CONCUSSIONS.[2]

63%

63% of concussions in high school football result from **tackling**.[2]

WHAT IS A CONCUSSION?

A concussion is a type of traumatic brain injury caused by a bump, blow, or jolt to the head, or by a hit to the body that causes the head and brain to move quickly back and forth. This fast movement can cause the brain to bounce around or twist in the skull, creating chemical changes and sometimes stretching and damaging brain cells.

What is a repetitive head impact?

A head impact is a bump, blow, or jolt to the head. A collision while playing sports is one way an athlete can get a head impact. When an athlete gets more than one head impact while playing a sport, it is called **repetitive head impact exposure**.

Do repetitive head impacts pose a health risk to my athletes?

While we know head impacts put an athlete at risk for concussion, more research is needed to understand if experiencing repetitive head impacts is harmful and how they may or may not affect the brain of a young athlete.

WILL A HELMET PROTECT MY CHILD FROM A CONCUSSION?

Helmets are not currently designed to prevent concussions. A properly fitted football helmet **can** help protect your child from the most serious types of brain or head injuries. Make sure your child wears a helmet that fits well and is in good condition.

PROPERLY FITTED HELMETS:

- Feel snug, leaving no spaces between the pads and the athlete's head.
- Won't slide on the head with the chin strap in place.
- Cannot be removed while the chin strap is in place.

Learn more and download the helmet fit app at **cdc.gov/HEADSUP**.

HOW CAN I KEEP MY CHILD SAFE?

Choose a non-contact or flag football program, especially for children ages 14 and younger.

A CDC STUDY COMPARING YOUTH TACKLE AND FLAG FOOTBALL FOUND:[3]

15x Tackle athletes had **15 times** more head impacts during a practice or game.

23x Tackle athletes had **23 times** more high-magnitude (hard) head impacts during a practice or game.

WAYS FOOTBALL PROGRAMS CAN PROMOTE SAFETY

CHOOSE A FOOTBALL PROGRAM THAT HELPS PROTECT YOUR CHILD

Look for a program that limits contact during practices, and ask the league or coach:

- How often do you do full-speed blocking or tackling drills?
- How much time to do you spend doing safe practice versus scrimmages and full-speed drills?
- Are certified athletic trainers available at games and practices?
- How do you teach and enforce fair play, safety, and sportsmanship?
- What is your protocol if you suspect a child sustains a concussion during a practice or game?

Talk to your child about the dangers of:[4]

- Hitting another child in the head.
- Using a helmet to collide with another child.
- Trying to injure or put another child at risk for injury.

[1] Sarmiento K, Thomas KE, Daugherty J, et al. Emergency Department Visits for Sports- and Recreation-Related Traumatic Brain Injuries Among Children - United States, 2010-2016. MMWR Morb Mortal Wkly Rep 2019;68:237-42.

[2] Marar M, McIlvain NM, Fields SK, Comstock RD. Epidemiology of concussions among United States high school athletes in 20 sports. The American journal of sports medicine 2012;40:747-55.

[3] Waltzman D, Sarmiento K, Devine O, et al. Head Impact Exposures Among Youth Tackle and Flag American Football Athletes. Sports Health. 2021; doi: 10.1177/1941738121992324.

[4] Collins CL, Fields SK, Comstock RD. When the rules of the game are broken: what proportion of high school sports-related injuries are related to illegal activity? Injury prevention 2008;14:34-8


CDC.GOV/HEADSUP
CDC HEADS UP
CDC

Concussion
INFORMATION SHEET

CDC HEADS UP
SAFE BRAIN. STRONGER FUTURE.

This sheet has information to help protect your children or teens from concussion or other serious brain injury. Use this information at your children's or teens' games and practices to learn how to spot a concussion and what to do if a concussion occurs.

What Is a Concussion?

A concussion is a type of traumatic brain injury—or TBI—caused by a bump, blow, or jolt to the head or by a hit to the body that causes the head and brain to move quickly back and forth. This fast movement can cause the brain to bounce around or twist in the skull, creating chemical changes in the brain and sometimes stretching and damaging the brain cells.

How Can I Help Keep My Children or Teens Safe?

Sports are a great way for children and teens to stay healthy and can help them do well in school. To help lower your children's or teens' chances of getting a concussion or other serious brain injury, you should:

- Help create a culture of safety for the team.
  - Work with their coach to teach ways to lower the chances of getting a concussion.
  - Talk with your children or teens about concussion and ask if they have concerns about reporting a concussion. Talk with them about their concerns; emphasize the importance of reporting concussions and taking time to recover from one.
  - Ensure that they follow their coach's rules for safety and the rules of the sport.
  - Tell your children or teens that you expect them to practice good sportsmanship at all times.
- When appropriate for the sport or activity, teach your children or teens that they must wear a helmet to lower the chances of the most serious types of brain or head injury. However, there is no "concussion-proof" helmet. So, even with a helmet, it is important for children and teens to avoid hits to the head.

Plan ahead. What do you want your child or teen to know about concussion?

How Can I Spot a Possible Concussion?

Children and teens who show or report one or more of the signs and symptoms listed below—or simply say they just "don't feel right" after a bump, blow, or jolt to the head or body—may have a concussion or other serious brain injury.

Signs Observed by Parents or Coaches

- Appears dazed or stunned
- Forgets an instruction, is confused about an assignment or position, or is unsure of the game, score, or opponent
- Moves clumsily
- Answers questions slowly
- Loses consciousness (even briefly)
- Shows mood, behavior, or personality changes
- Can't recall events *prior to* or *after* a hit or fall

Symptoms Reported by Children and Teens

- Headache or "pressure" in head
- Nausea or vomiting
- Balance problems or dizziness, or double or blurry vision
- Bothered by light or noise
- Feeling sluggish, hazy, foggy, or groggy
- Confusion, or concentration or memory problems
- Just not "feeling right," or "feeling down"

Talk with your children and teens about concussion. Tell them to report their concussion symptoms to you and their coach right away. Some children and teens think concussions aren't serious, or worry that if they report a concussion they will lose their position on the team or look weak. Be sure to remind them that *it's better to miss one game than the whole season.*

CONCUSSIONS AFFECT EACH CHILD AND TEEN DIFFERENTLY.

While most children and teens with a concussion feel better within a couple of weeks, some will have symptoms for months or longer. Talk with your children's or teens' healthcare provider if their concussion symptoms do not go away, or if they get worse after they return to their regular activities.

What Are Some More Serious Danger Signs to Look Out For?

In rare cases, a dangerous collection of blood (hematoma) may form on the brain after a bump, blow, or jolt to the head or body and can squeeze the brain against the skull. Call 9-1-1 or take your child or teen to the emergency department right away if, after a bump, blow, or jolt to the head or body, he or she has one or more of these danger signs:

- One pupil larger than the other
- Drowsiness or inability to wake up
- A headache that gets worse and does not go away
- Slurred speech, weakness, numbness, or decreased coordination
- Repeated vomiting or nausea, convulsions or seizures (shaking or twitching)
- Unusual behavior, increased confusion, restlessness, or agitation
- Loss of consciousness (passed out/knocked out). Even a brief loss of consciousness should be taken seriously

Children and teens who continue to play while having concussion symptoms, or who return to play too soon—while the brain is still healing—have a greater chance of getting another concussion. A repeat concussion that occurs while the brain is still healing from the first injury can be very serious, and can affect a child or teen for a lifetime. It can even be fatal.

What Should I Do If My Child or Teen Has a Possible Concussion?

As a parent, if you think your child or teen may have a concussion, you should:

1. Remove your child or teen from play.
2. Keep your child or teen out of play the day of the injury. Your child or teen should be seen by a healthcare provider and only return to play with permission from a healthcare provider who is experienced in evaluating for concussion.
3. Ask your child's or teen's healthcare provider for written instructions on helping your child or teen return to school. You can give the instructions to your child's or teen's school nurse and teacher(s) and return-to-play instructions to the coach and/or athletic trainer.

Do not try to judge the severity of the injury yourself. Only a healthcare provider should assess a child or teen for a possible concussion. Concussion signs and symptoms often show up soon after the injury. But you may not know how serious the concussion is at first, and some symptoms may not show up for hours or days.

The brain needs time to heal after a concussion. A child's or teen's return to school and sports should be a gradual process that is carefully managed and monitored by a healthcare provider.

To learn more, go to cdc.gov/HEADSUP

CDC HEADS UP

Discuss the risks of concussion and other serious brain injuries with your child or teen, and have each person sign below.

Detach the section below, and keep this information sheet to use at your children's or teens' games and practices to help protect them from concussion or other serious brain injuries.

○ I learned about concussion and talked with my parent or coach about what to do if I have a concussion or other serious brain injury.

Athlete's Name Printed:_____ Date:_____

Athlete's Signature:_____

○ I have read this fact sheet for parents on concussion with my child or teen, and talked about what to do if they have a concussion or other serious brain injury.

Parent or Legal Guardian's Name Printed:_____ Date:_____

Parent or Legal Guardian's Signature:_____

Revised January 2019

Jason Franklin

May 6,1992- July 14,2018

Early Life

Jason

Football Pictures

CTE Center Report

September 18, 2019

CTE CENTER REPORT

PATIENT'S NAME: Franklin, Jason

NEUROPATHOLOGY FINAL REPORT

1. Chronic Traumatic Encephalopathy (CTE): Stage II (out of 4 possible stages, with Stage IV most severe)

Comment: There is a small cavum septum pellucidum. The posterior corpus callosum in mildly thinned. Nine perivascular CTE lesions are identified in the frontal lobes – affecting primarily Rolandic, superior frontal and dorsolateral frontal cortices. Rare neurofibrillary tangles (NFTs) are also found in the temporal pole. There is mild neurofibrillary degeneration of the locus coeruleus. These changes conform to Chronic Traumatic Encephalopathy (CTE), Stage II (out of a possible IV, with IV being the most severe). In addition, there are prominent perivascular hemosiderin-laden macrophages with mild rarefaction of the subcortical white matter.

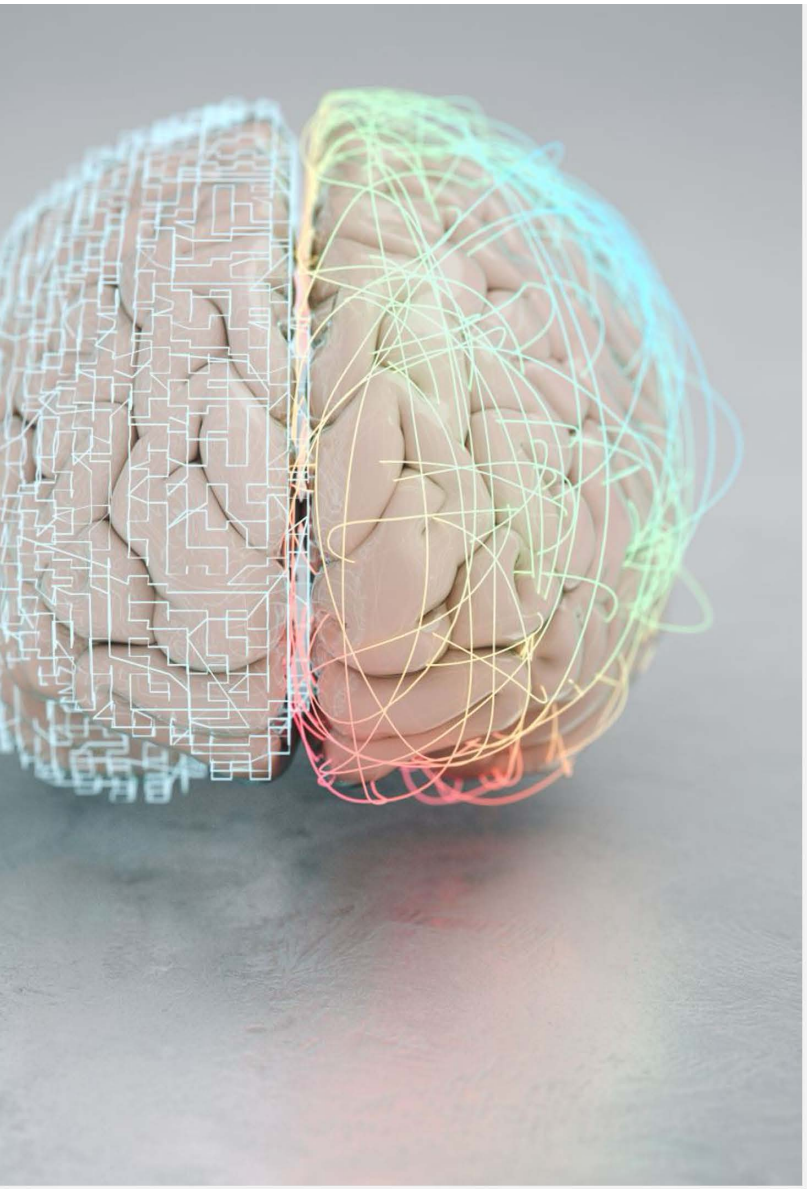
I Got You

THE IMPACT OF CHRONIC TRAUMATIC ENCEPHALOPATHY ON FAMILIES

Presented by:

Pamela Tahim Thakur, Esq.

THAKUR LAW FIRM, APC



WHAT IS CHRONIC TRAUMATIC ENCEPHALOPATHY (CTE)?

- **CTE is a brain disorder** generally caused by repeated head injuries – a common occurrence due the nature of American football
- **CTE causes degeneration** – the death of nerve cells in the brain – which results in memory loss, impulse control problems, aggression, depression, anxiety, suicidality, parkinsonism, dementia, etc.
- CTE worsens over time and can only be diagnosed postmortem
- CTE remains not well understood

Source: Mayo Clinic

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Los Angeles Times

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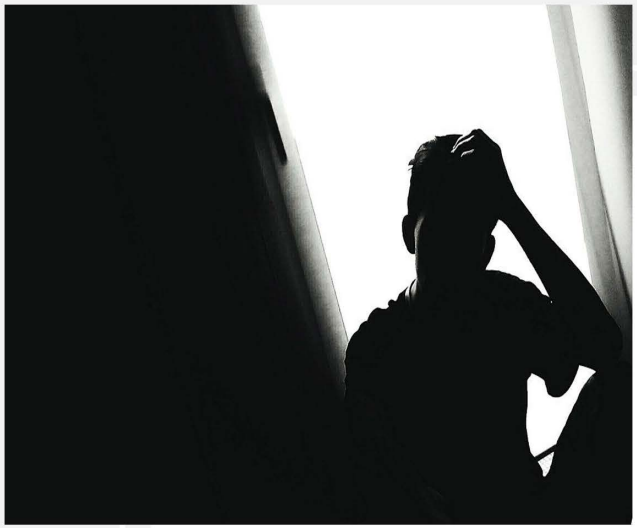
California home insurers plan return to fire zones under new deal

WHY AM I HERE?

- I represented four former UCLA football players against UCLA and the coaching staff, and the cases were highly publicized because these cases are novel and difficult to prove.
- The cases alleged that coaching staff ignored player concerns for their health, pressured players to play when injured, and ridiculed players who complained of their injuries, resulting in excessive concussions, early retirements, and significant injuries.
- Two of the players experienced symptoms consistent with CTE.
- As a result, the players' injuries were exacerbated, some suffered concussions, and two attempted suicide.

HOW HAS THE PLAYERS' CONDITION AFFECTED THEIR FAMILIES?



- Settlement of their cases has not cured the ongoing physical and mental health problems they face.
- Some of the players cannot control their violent urges and have ended up in jail.
- Some family members say that the players are no longer the same people as before.

CONCLUSION

- CTE irrevocably and tragically alters the life trajectory of those affected.
- No matter the damages obtained, California tort law is incapable of providing redress for the irreparable damage caused by CTE.
- Prevention through legislation is therefore the most proper way to address this serious problem.

They Played Football as Children. Now Their Families Mourn

They gave up the game after high school, but the damage seemingly was already done. Now, their families mourn and look for answers

Illustration by Karolis Strautniekas

Sep 9, 2023 10:00 am

S ince DuQuan Myers died six years ago, he’s come back in ways that are mysterious and magical and hard to explain. It started the day of his funeral, when the doves that were released over his coffin refused to fly home, perching instead in the oak tree just above his grave as if witnessing a visitation. Then there was that drive to El Paso, Texas, when — not once but twice

— the windshield wipers started swiping just as the satellite radio cut out midsong, switching to the tune Myers' sister Natasha had been listening to at the exact moment when her mom called with the news that her brother was gone. There was the way Myers' nephew — the one who was just a baby when Myers died — would sometimes point into an empty corner of a room and say "uncle." There were the Pokémon cards that Myers had loved as a child but hadn't played with in years turning up one by one in unexpected and impossible places, like the middle of a clean kitchen floor. There were the nights when Myers' mother, Letitia Wilbourn, would feel him, actually feel him, resting his head upon her shoulder, even though no one was there. But, of course, that's not right. There was someone there. He was there. She knew it. Such is the logic of grief.

Tragedy rips the grieving out of the storyline of their own life, denies them any sense of what the past meant or the future will hold. Wilbourn remembers few details from her son's funeral, but she does remember the doves. "That lady said, 'These birds have never done this. Never,'" she tells me, sitting on a small sofa in the darkened living room of her modest house in the suburbs of Fort Worth, surrounded by pictures and mementos from her son's short life: an infant footprint from the hospital where he was born, a class project he wrote about wanting to own a whale, a photo of him posing in his Little League uniform with a shy smile. "The lady was like, 'Was he a very gentle spirit?'"

DuQuan Myers *was* a gentle spirit. His family called him the "animal whisperer," the kid who loved to roam the farmland that still abutted their Echo Heights neighborhood and who would bring home all kinds of creatures he'd "rescued" — dogs, cats, turtles, frogs, lizards, toads, even once a snake that Wilbourn found, to her horror, in a candy jar. When his three older sisters squabbled, Myers was the one who always could diffuse the situation, the baby brother whose humor and charisma and cheerfulness were impossible to resist. It fit no known narrative that Myers would hurt any living thing, himself included. And yet on the evening of Feb. 17, 2017, Wilbourn had been driving home from her job as a cop for the Tarrant County Sheriff's Office when she received a phone call. On the other end, she could hear the keening sobs of the young woman Myers had dated off and on for years. "She said, 'Quan's dead,'" Wilbourn explains. "I was like, 'What?' She said, 'DuQuan's dead.'" Home alone in his apartment near Texas State, 26 years old and only months away from getting a degree in criminal justice, Myers had shot himself in the head.

Without thinking, wild with grief, Wilbourn turned her car toward San Marcos and drove 77 miles in 30 blind minutes before a friend called and persuaded her to pull over and turn around. Myers was gone. There was nothing to be done that night, which Wilbourn spent at home, alone, her phone switched off so nothing could puncture the cocoon of mourning and confusion. She'd spoken to Myers earlier that day, just before he'd begun his shift at FedEx. He'd seemed fine, his normal self. But had she missed something? She knew he had a gun, but he lived in Texas; everyone has one. She'd only discussed suicide with him once before, back when Chris Benoit, a wrestler Myers admired, hanged himself. Myers had said, "Oh, I'd never commit suicide. I love me way too much for that." Then he'd grinned at her with that way he had. "Look at how pretty I am." Now, her beautiful boy was gone.

In the car the next day, on the way to San Marcos to begin the procedural processes of death — closing up Myers' apartment, identifying his body, speaking to investigators who had no doubt that the gunshot wound was self-inflicted — Wilbourn was on a call with the medical examiner when, out of nowhere, she heard herself asking if they could save Myers' brain, if they could have it studied. "I just knew that the suicide didn't make sense, because he wasn't suicidal," she says, her voice measured, her Texas accent melodic. "I was just praying for answers, and when the phone rang, that's what came out. I wasn't thinking that anything was wrong with his brain or anything like that, I just knew something wasn't right. It's just the mother's instinct."

There was also, perhaps, a subconscious concern. Myers had played football for seven years, from sixth grade through his senior year of high school. In the time since he'd stopped playing, Wilbourn had learned about chronic traumatic encephalopathy — the degenerative brain disease that had plagued NFL greats like Mike Webster, Andre Waters, and Dave Duerson (who shot himself in the heart specifically so that his brain would be intact when studied). Like many people, she'd been alarmed by the reports of these famously tough athletes whose very toughness had been their undoing. Then again, she didn't actually think Myers fit the CTE profile: He had not played football past high school; he'd never had a concussion that she knew of; he was nothing like Aaron Hernandez or Junior Seau, men who'd grown angry and addled and violent before dying angry and addled and violent deaths. So it was a surprise, she says, when "the first thing out of my mouth was 'Can we donate his brain to Boston University?'" She looks down at the photos surrounding her. "I think that was just a God thing."


TECH
2

DuQuan Myers hadn't shown many symptoms — but was diagnosed with stage 1 CTE.

IT HAS BEEN ALMOST two decades since Pittsburgh Steelers Hall of Famer Mike Webster become the first NFL player to be diagnosed with CTE, after a heart attack at age 50 put an end to misery so intense that Webster — by then sleeping in his car because he couldn't remember where he lived — would shoot himself with a taser gun until he mercifully blacked out. Even then, CTE wasn't exactly a new discovery: A medical report from 1928 described boxers with "punch-drunk syndrome," later classified as dementia pugilistica. The first known use of the term "chronic traumatic encephalopathy" occurred in a neurology paper written in 1949. But Webster's 2005 diagnosis was a watershed moment, implicating the most popular sport in America, one grafted into the guts and glory of the American psyche — and one that millions of American kids were suiting up to play every season. So documentaries were made. Brain banks were established. Funding flowed. And as more and more brains of dead athletes were submitted for study, it became clear that, while CTE was a progressive disease, it was not the sole province of aging NFL players. It could show up in the brains of people in their teens and twenties, people who'd never played past the age of 18.

Even in CTE's earliest stages — and for reasons scientists are still exploring — its effects could be grave. "We're north of 1,300 brains, and while the overwhelming majority are from people who are older, we certainly have hundreds of brains of people in that younger age group," says Dr. Robert Cantu, co-director of the Center for the Study of Traumatic Encephalopathy, chair of neurosurgery at Emerson Hospital, and the nation's leading authority on concussions. "CTE hasn't directly killed anybody in their teens, twenties, thirties, forties, but we have a lot of people with CTE that have died at those ages. Almost all of them have died by suicide."

It is well-established that suffering even just one concussion is a risk factor for developing mental-health issues. The year after Myers' death, the *Journal of the American Medical Association* published a study that found that a person's risk of dying by suicide doubled after being diagnosed with a concussion. A 2021 study in the *Journal of Neurotrauma* followed patients for 180 days and found "significantly increased rates of depression, anxiety, or suicidal ideation following concussion" in those under 26 years of age. Another study, from 2022 and published in *American Journal of Physical Medicine & Rehabilitation,* found that 48 percent of all concussion patients were later diagnosed with depression, anxiety, panic disorder, post-traumatic stress disorder, bipolar disorder, or schizophrenia in the three years that they were tracked, and that "all of the mental illnesses this study chose to evaluate were present in a higher proportion of patients after a concussion than the general population."

But CTE isn't just about diagnosed concussions, most of which go on to heal with no obvious, lasting effects. It's about repeated, seemingly benign assaults to brain tissue, little "dings" that could occur dozens of times in a single game and that a player may not even clock but that nonetheless cause damage at the cellular level — microscopically torn blood vessels, disconnected neurons, jumbled tau proteins (the building blocks of the fibers that carry nutrients and messages from cell to cell). Given time, the brain can usually repair this damage — except when players don't even know there's been damage, and so don't grant themselves time to recover. In that case, injuries can compound until the brain is overwhelmed, until — unable to reconfigure themselves — the tau proteins essentially rot, creating toxic lesions that spread

deeper into the brain and even sometimes into the brain stem. This past June, the largest CTE study to date confirmed that the best predictor of future brain disease was not the number of diagnosed concussions a player had sustained but rather the cumulative force of all hits to the head throughout their career. In other words, a lot of little impacts could be as damaging as a smattering of major ones.

This means that when kids across America suit up this fall to square off on the gridiron, every hit will matter, every "ding" and every bell rung will count toward an unspecified number past which the brain might be permanently impaired. It means that no amount of concussion protocols can definitively stave off disaster, that some level of danger is lurking in every play. It means that America's greatest game is hurting our children in insidious and incalculable ways, and that addressing the issue might mean fundamentally changing the way we teach a game that has become fundamental to America's sense of self.

The scars on Barrett Callaghan's helmet were a source of pride — now, they're a painful reminder. *Photograph by Lyndon French*

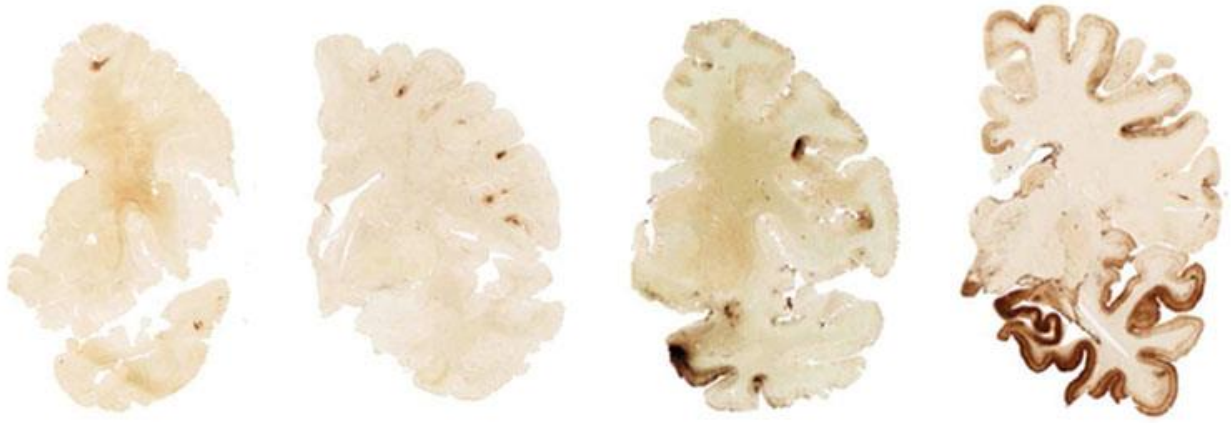
"It's a really profound problem to confront, especially for a sport that's been at the center of so much of our community and cultural life, as youth football is and has been," says Kathleen Bachynski, a professor of public health at Muhlenberg College and author of *No Game for Boys to Play: The History of Youth Football and the Origins of a Public Health Crisis*. Bachynski's research details a long history of medical objections that have done little to intercept the sport's

cultural dominance. In the 1890s, *The Chicago Tribune* announced that football would "physically ruin thousands of young men." In the early 1900s, the brutality of Ivy League games led them to be described as "boy-killing, education-prostituting, gladiatorial sport" (even Teddy Roosevelt intervened, leading to the limitation of certain plays). And throughout the 20th century, doctors continued to sound the alarm in publications like the *Journal of the American Medical Association* ("Football is no game for boys to play"), the *Journal of School Health* ("More concussions occur in football than is generally realized"), and the *American Journal of Public Health* ("[Children are] encouraged to addle their own brains with repeated concussions in such sports as football and boxing"). Meanwhile, as boxing declined in popularity, football's reach only grew. Youth leagues began mushrooming in the Sixties. Kids as young as five suited up. "It's really hard for us, societally, to confront something that we thought was great might not have been so great," says Bachynski of this cultural expansion. "But there are some health risks that we have decided even a fully-informed parent can't allow their kid to [take]. If you understand all the risks and harms of cigarettes, you cannot let your 12-year-old smoke a cigarette — that's not something you can choose as a parent. And we have to decide: 'Do repeated head impacts fall in that category?'"

For football's defenders, the answer is no. "I love this sport. I think it's America's greatest sport," says Dr. Julian Bailes, a former team physician for the Pittsburgh Steelers, a neurological consultant to the NFL Players Association, and the head of the medical advisory committee for Pop Warner, the country's largest youth-football league. Bailes knew Mike Webster personally and, having helped identify the clinical evidence of Webster's CTE (he's played by Alec Baldwin in the movie *Concussion*), is considered to be a pioneer in the field. He certainly acknowledges the cumulative nature of the risk when it comes to head impacts and CTE, but argues that the threshold for developing the disease would be unlikely to be met just by playing a few years of youth football. "I'm not pro-head injury or pro-concussion, but there's no conclusive evidence that if you play youth football it's going to lead to long-term brain damage," he tells me. "The risk occurs as [players] get into high school and college, and they are big and strong and fast and have 600 to a thousand hits a season. So in my opinion, it's a misdirected argument. There's probably 100 kids who get killed on bicycles every year. There's no kids dying playing Pop Warner football."

But science continues to raise red flags about the sport. A 2016 study published in *Radiology* found that playing just one season of youth football caused measurable changes in the white matter of children's brains as well as diminished brain function, even in players who had not displayed signs of having a concussion. Another, from 2018, showed that both years of football play and the age at which an athlete started affected the volume of the thalamus, the egg-shaped structure in the center of the brain that relays information from the body and has been associated with cognition and mood. "Tau is the sexy thing to talk about, so that's what people talk about," says Cantu of the changes to the brain that can occur from repetitive brain injury. "They think, 'You got CTE. That's bad.' Well, you can be very bad off without CTE, just from all those hits."

Dr. Ann McKee, the chief of neuropathology at the VA Boston Healthcare System, holds slides of a brain at her lab's brain bank in Bedford, Mass. *Shiho Fukada/The New York Times/Redux*



The stages of CTE, from 1 to 4. The damage at stage 1 may seem minimal but can have profound effects. *Boston University's Chronic Traumatic Encephalopathy Center*

According to Dr. Ann McKee, a neuropathologist and director of the CTE Center at Boston University — where she heads up the largest brain bank in the country — patients diagnosed with CTE almost always show other signs of brain damage, but those same injuries can still be present without the pathology of a progressive disease. "There are a lot of other changes that can cause very disabling symptoms, and they don't have to rise to the level of CTE," she says. "We're seeing changes in brain architecture, shrinkage of the brain, damage to brain structures,

inflammation, evidence of vascular disease — meaning the tiny blood vessels of the brain are probably injured and leaky."

All of these changes have the potential to affect cognition and mental health. And even if the effect is difficult to quantify, with more than 2 million kids playing tackle football every year, certain researchers question whether it has led to an epidemic of children whose potential is diminished, whose cognition might have been a little higher, whose chances of developing depression or dying by suicide might have been a little lower if they had never played a sport in which they had repeatedly sustained injury to their brains. "The novel mental-health-disorder research is actually becoming quite strong," says Chris Nowinski, a former Harvard football player and professional wrestler who went on to co-found the Concussion Legacy Foundation and get a Ph.D. in behavioral neuroscience after a concussion he suffered in the ring in 2003 kicked off a sleep disorder, a behavior disorder, and a throbbing headache that didn't let up for years. Now considered the public face of CTE awareness — he's the guy *Newsweek* called after Hernandez died by suicide in prison, and the person families typically contact if they want to donate the brain of a loved one suspected of having CTE — Nowinski not only believes that the connection between repetitive, low-level head impacts and mental illness is strong enough that developing a mood disorder could prove to be a risk factor for developing CTE, but he also believes that the connection is strong enough to be worrisome in its own right.

A study published in the *Journal of Neurotrauma* the month before DuQuan Myers' death found that cumulative head impacts, with or without diagnosed concussions, increased the risk of depression, apathy, and behavioral dysregulation. A study published just a few months after his death found that participants with CTE showed accumulations of tau lesions specifically "in brain regions that have been associated with … impulsivity, depressive symptoms, anxiety, and explosivity." Other studies have found football players to have higher rates of depression than the population at large. "Essentially," says Nowinski, "we're taking kids who have never had an anxiety or depression or self-harm issue, and they're getting it because of damage to their brain."

A MATTER OF HOURS after a bullet entered DuQuan Myers' brain, the local medical examiner in Lockhart, Texas, carefully, gingerly, removed it and submerged it in a formaldehyde solution. She then placed the container into a styrofoam box bound for Boston. Once the brain arrived at McKee's lab at the Boston University CTE Center, it was photographed and dissected. Half was snap-frozen and stored in perpetuity in a negative-80-degree freezer. The other half, the half that would be used to make the diagnosis, was put back in formaldehyde. Then the waiting game began.

Inside the skull of a living human, the brain is almost the consistency of jello, a miraculous mush of electrical charges. It took months for the formaldehyde to seep all the way in, hardening Myers' brain to a consistency that would allow it to be cut into eighth-of-an-inch slivers. Those slivers were then further dissected into hundreds of 10-micron slices, almost impossibly thin ribbons of neural matter that were stained and put into slides for McKee or one of her team to analyze under a microscope. Meanwhile, clinicians were rounding up medical records to determine Myers' medical past, his history of head impacts, and what symptoms he may have had. Both teams worked blindly: Neither would communicate about Myers' case until the clinicians had finished their research and the pathologists had made their determination.

In the spring of 2018, Letitia Wilbourn received a notification that McKee and her colleagues had come to a conclusion in her son's case. Not knowing what she expected or even what she wanted to hear, she arranged to take the call by herself, over her lunch break. "I didn't want to be home alone," she says. "I was carrying so much guilt with his suicide, thinking, 'Did I miss something? Did I do something? What caused it?' I had so much guilt."

On the call, McKee gently shared the diagnosis: Myers had had stage 1 CTE, characterized by one to three microscopic lesions. At this level, the lesions would have affected such a small percentage of Myers' brain that they may not have accounted for any changes in mood or behavior. But they would certainly have been proof of brain trauma that had been unable to fully heal. In fact, his brain also showed arteriolosclerosis (the thickening of small blood vessels), neuroinflammation, and loss of fibers in the white matter, Wilbourn was told. "Then they explained that there was nothing that I could have done to prevent what happened," she says. "They told me that there was no way for me to have known. It was an instant release. I actually had the best day I'd had since he'd died. And then came the guilt of having put him in football to begin with."

As gentle as he'd been off the field, Myers had been formidable on it, starting as both an offensive and defensive lineman despite his small size — only five feet five and 140 pounds by the end of high school. "He loved to tackle people, that's for sure," Wilbourn tells me. She thought of all those times when he had taken a brutal hit and she'd caught her breath, wondering if she should go down to the field and check on him but stopping herself with the reminder that parents weren't supposed to do that. She thought of that time he'd broken his leg during practice and been rushed to the hospital, how she'd thought that fractured fibula and tibia were the worst injuries of his life. She thought of the terrible headaches he'd have after practices, how she'd chalked them up to allergies. She remembered how in high school he'd complained about his vision and suddenly needed glasses when no one else in the family ever had. Now, on the phone with McKee, these things took on new meaning, the past reorganizing itself in her mind.

She'd been clear with the clinicians at Boston University: Myers' life was not without sadness. He and his girlfriend had recently broken up, though that wasn't unusual: They'd broken up and gotten back together more times than Wilbourn could count. His cousin had just passed away, shot by an ex in an act of passion. Like the rest of the extended family, Myers had been shocked and devastated, but seemingly not any more than anyone else. On the call with Boston University, these events were referred to as "triggers," not relevant to the official diagnosis, but certainly not irrelevant to what transpired.

"Often there's a very impulsive component to these suicides," McKee tells me when discussing Myers' case this past spring. "People with an injured brain have a lower ability to control their impulses. They can be fine one minute and then all of a sudden just pull the trigger or whatever they decide to do. Their judgment is off. There's a situation where something doesn't go well or they become depressed, and they don't have the checks and balances that a person who didn't have an injured brain [would have]. We all get depressed at some point, we all feel lousy, but the injured brain just can't stop it. They can't pull themselves back."

For McKee's team, Wilbourn's grief has become a familiar refrain. "That's really common, in my experience, to have just a random, totally impulsive act that just blindsides the family," McKee says. "That's not uncommon with these suicides."

Then again, she also has encountered plenty of cases in which the suicide is not unexpected. It's the end that some families have been expecting, warding off, fighting back for a long, long time.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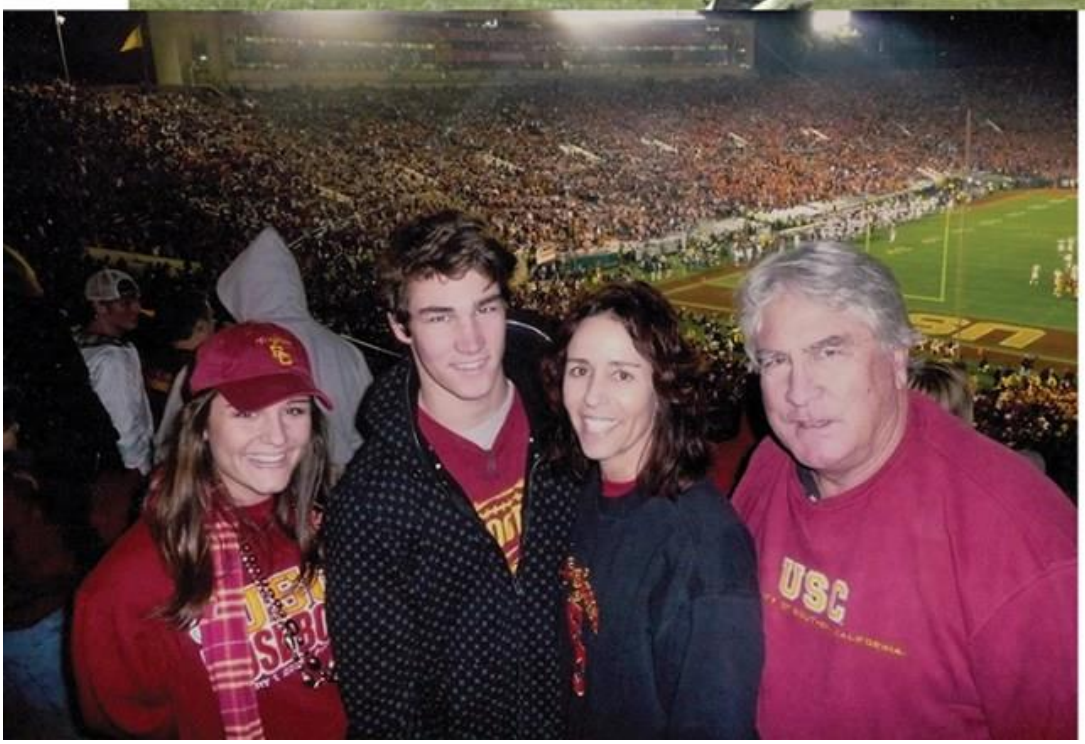
USC

Michael Bartlett felt his brain had somehow turned on him, and he grew moody. “He knew there was something wrong,” his mom, Susan, says.

AROUND THE TIME of his freshman year of college, Michael Bartlett — the person he was, the person he’d always been — started to disappear. It happened gradually, so slowly that at first it didn’t seem like much of anything at all, maybe just a bumpy transition into adulthood. Still, his parents, Larry and Susan, grew concerned: Michael had always been friendly and easygoing, with a smile so big it seemed to hijack his entire, handsome face. On the football field, he’d been the sort of solid kid others looked up to. Now, he seemed to frustrate more easily; now, he seemed a little aloof. A year or so into these changes, Larry and Susan took him for academic testing, hoping to figure out why their smart, quizzical son — the one who’d read philosophy as a child — was struggling so much with college. The results surprised them: They were told that Michael had a very high IQ but very low cognitive function. “She said it was like having a 400 horsepower engine in a car with no wheels,” Susan says of the report. “And we’re like, ‘What does that mean?’”

What it ultimately meant was a decade of misery for her family, a decade of Michael’s brain seeming to turn on him. He tried cognitive therapy. He tried antidepressants. He tried taking medication for ADD. When none of that helped, he self-medicated with alcohol. He dropped out of college, moving home to his parents’ house in La Mesa, California, isolating himself in his room, fully aware that not just his future but something even more intangible was slipping away. “He knew there was something wrong with his brain,” says Susan. “He kept saying, ‘There’s something wrong. This is my best tool. I cannot lose my brain. I can’t.’ He tried to figure out what was wrong: ‘Maybe I have this, maybe I have that. I don’t know what’s wrong.’” She doesn’t remember him ever mentioning CTE, though the thought may have occurred to him. He’d started playing football when he was nine, coming home so beat up that Susan remembers thinking, “‘You’re not going back to that.’ That’s the stupidest thing I ever saw.” But he did go back. By his early twenties, “he started to shut down a lot. I knew we were in terrible trouble, because he would say things like, ‘If I can’t get my brain right, I can’t live.’”

Eventually, it became clear that he didn’t intend to. He got a DUI. He tried to hang himself with a belt in his closet. When Michael was arrested for lunging at the gun of one of the cops who’d responded to a suicide call, Larry couldn’t help but realize that, mixed in with the hurt and sadness, he also felt a sense of reprieve. “The thing is, he wasn’t gonna die in jail, wasn’t gonna commit suicide if he stayed there,” he says, his voice breaking. Michael never had a girlfriend. He never had a job. Larry and Susan had no idea what to do. There was no family history of mental illness, no indications of past trauma; there was only the constant vigil their life became, trying to keep their son alive. “We had problems leaving the house at all,” says Susan. “Because we were always afraid [of what we’d find] when we came home.”

When it comes to mental illness, causation is difficult, if not impossible, to prove. Many mental illnesses present in young adulthood — and their causes are certainly multifactorial. But for a number of the scientists who study CTE and brain injury, the multifactorial nature of mental illness — the nature versus nurture of it — is exactly the point. No one can predict their genetic chance of developing a mental illness; no one can predict how those odds might change after hundreds or thousands of blows to the head. “In the concussion clinic that I run, all of our

clinicians doing intake evaluations ask, 'Have you ever been diagnosed or worked up for ADD, ADHD, any learning disability, any mood disorder, anxiety, depression, panic attack?'" Cantu says. "The reason that we do this is that whenever you have an underlying challenge to start with, brain injury will only aggravate it."

This is especially true while the brain is still developing. Though Michael Bartlett never played football past high school, he started at an age when the head is still relatively big compared with the body, and when the muscles of the neck have not been built up, meaning that even minor impacts can snap the head about. At nine, the brain is also less myelinated than it one day will be. "Myelin is the coating of nerve fibers," Cantu explains. "It's like the coating of a telephone wire. It not only improves transmission, but it also makes it a little harder to break, so the same degree of acceleration forces on youngsters' brains can do more damage to the axons than they would do to an adult."

Most importantly, according to Cantu, Bartlett began playing just before a crucial stage of brain development that, he says, occurs between the ages of 10 and 12. In these years, the circuitry is being honed — some connections between nerve cells drop out, and others are enhanced — creating the smoother neural pathways that not only allow for more efficient information processing but also dictate adult personality and intellect. "If you injure the brain during that period of time, you're essentially causing your genetic endowment not to occur, but some aberration of it," says Cantu. "And the aberration is not better."

Nor is it reversible. A 2017 study on 214 former football players found that those who had started tackle football before the age of 12 were twice as likely to have "problems with behavioral regulation, apathy, and executive function" and three times more likely to have "clinically elevated depression scores," even if they never played past high school. Another study from the following year found that participants with CTE who'd started playing tackle football before 12 developed behavioral and mood symptoms 13 years earlier than those who started at 12 or later, a pattern that was also observed in participants without CTE. (Bailes points out, however, that there are some studies that do not replicate these findings.) Younger age of first exposure to a collision sport — one in which contact doesn't just randomly happen but is part of every play in the game — has been associated with structural alterations in the thalamus (even when adjusting for years of play) and in the corpus callosum, the bundle of nerves that connects the brain's two hemispheres. And while damage may not become apparent until early adulthood, when cognitive demands could outpace the brain's diminished capabilities, or may even fly under the radar — how can one measure potential that's lost? — the bottom line, Cantu argues, is that "those who start young will have greater later-life chances to wind up with cognitive, behavioral, and mood issues — and if they get CTE, it'll be worse."

All of which could contribute to an explanation of what happened on July 11, 2019. Just after his 29th birthday, Michael Bartlett drove to an overpass, pulled his car over, climbed up on the concrete wall, and jumped 85 feet to the pavement below. Larry and Susan had been driving around La Mesa, frantically looking for him, when they saw the flashing red lights. "The police wouldn't even let me see him," Larry says. "All they would let me do is hug the body bag. I don't even remember the next two days. It was just like we were in a different world. And it's been that way ever since."

These days, when Susan feels like she's spiraling, she pulls out the report from Boston University, the one that diagnosed Michael with stage 1 CTE. "You know, when you're in a bad place, it's kind of helpful to see the medical answer," she explains. It doesn't change what happened, of course, but it achieves something: It solves the mystery Michael had been so desperate to solve when he was alive. For that, the Bartletts are grateful. "That's where our son exists today, in Boston," says Larry. "That's his contribution to this horrible disease. He contributed his brain."

THE CHICAGO SPRING RECEPTION hosted by the Concussion Legacy Foundation is a cascade of grief amid canapes. One evening this past spring, in a lovely room of the University Club with sweeping views of downtown, family members of the deceased or desperate stand up one by one to share their plight. There is the young son who speaks of his father's decline, the dad who wants to honor his child's memory, the teammate who wonders if his friend's struggles await him also. Toward the end of the impromptu speeches, Jan Callaghan falteringly makes her way to the center of the room. Her son Barrett's legacy story is on the CLF's website, alongside a photo of the football helmet he'd worn in middle school, the one scarred with deep gashes and gouges.


10

After high school, Barrett Callaghan lived in a daze. His parents later learned he had stage 1 CTE.

She'd pointed them out the day before, standing over the helmet as it rested on the dining-room table of her house in the Orland Park neighborhood of Chicago. There was a time when those gouges were a source of pride. Now, Jan shudders at the thought.

For years, hers had been a football family, their lives arranged around the endless drills and practices, their weekends spent driving to and from games, their laundry hamper full of grass-stained uniforms. When he was seven, Barrett saw his brother Richie play and begged his parents to find a team for him. Jan remembers getting a call from her husband during one of Barrett's first games: "He said, 'You've got to get up here. You're not going to believe this.'" As she approached the field, she searched out Barrett's little red uniform with the number 3. "And my jaw dropped as I watched him go from one end of the field to the other with the football, never stopping. As a young boy, he was a force. When that football hit his hands, there was no stopping him."

As Barrett got older, it became clear that he'd never play professionally, but it was also clear that football would organize the rhythms of his life. One of his sisters was a cheerleader. His dad, Rich, volunteered as a coach. "The slogan we had was 'Don't drop your bonnet,'" he says the day we meet, sitting at the kitchen table, his voice thick with sorrow. "'Tackle the right way, into the chest with your face guard up. Don't have your head down.'" Richie had heeded this warning. But Barrett was a different sort of player than his brother, one who made up for his small size with his tenacity. "He was a really tough player," Jan says. "He did play with his head."

There were a lot of tough players on the South Side of Chicago, where the kids attended Catholic school and where football came second to God, if only just. "They call it the black-and-blue South Side Catholic," says Rich. "Because the kids are black and blue." So it was hard to explain to football friends what was happening to Barrett in the final years of his life. People thought he was lazy when he dropped out of college and moved back home, unfocused when he couldn't hold down a job, losing the thread when he'd lash out aggressively for almost no reason. People distanced themselves. Jan would have to remind them, "This is Barrett we're talking about. You've known him forever. Do you think it was normal that he couldn't finish school?" She saw how he couldn't sleep, how embarrassed he was when he couldn't take in simple information or remember simple tasks, how he saw his brother and sisters building lives and careers and families and felt desperately left behind. When he came down to the kitchen one summer afternoon and told Jan he thought he had CTE, she had already come to the same conclusion. She assumed she'd go the rest of her life not knowing for sure. But when state troopers knocked on her door at 4 a.m. the morning of Sept. 26, 2021, and told her that Barrett had died in an accident, a head-on collision with a car going the wrong way on the highway, she had barely been able to even take in the news before she started making plans to have his brain donated.

As with smoking and lung cancer, there's no known threshold for developing CTE and no way to know an individual player's risk factors for being affected by it or other structural changes to the brain — no way to yet explain why some players are ravaged while others seemingly escape

harm. On average, those who play a collision sport for less than five years have a very small chance of getting diagnosed with the disease, but the risk goes up after that; at 15 years of play, the risk has been estimated to be 10 times higher. The location of the damage probably matters, too; some parts of the brain are "silent" and harm to them could pass unnoticed, while others are crucial to cognition and regulation of mood. And some people may just take longer to heal, making them more susceptible to outrunning their cognitive reserve. "Probably in some cases, the brain's regulatory systems are not very good — they are more fragile — so the brain cannot bring itself back to normality," says Dr. Lea Grinberg, a neuropathologist who studies the early stages of neurodegenerative disease and whose 2018 study on Alzheimer's found it to also be preceded by mood dysregulation.

Despite these unknowns, the diagnosis of stage 1 CTE has been enlightening, but further isolating for Barrett's family. "This woman whose husband took his life brought up this great point," Jan tells me. "She said, 'Everybody went on and on when he passed about what a hard, tough player he was and a great teammate. And then when I told everybody that he had CTE? Crickets. Nobody wants to talk about it.'" She looks down at her hands spread over the table. "It's like you're on an island," she continues. "I don't know if they just don't believe it, or they don't want their precious little football tarnished." Or maybe, she considers, they just don't want to have to face the possibility that the same fate could await their kid, which is a reservation she understands.

Even Rich had trouble accepting Barrett's diagnosis at first, though time and research have helped him come around. "There's clearly more than a smoking gun here," he says. "The gun's been fired." Jan nods in agreement: "You pay attention, or you don't."

IN RECENT YEARS, plenty of people have paid attention. According to the Sports and Fitness Industry Association, participation in tackle football for kids ages six to 12 has declined almost 29 percent since 2016, the year the NFL first publicly (and belatedly) confirmed a link between football and CTE — which has now also been found in the brains of hockey, soccer, and rugby players, as well as wrestlers, mixed-martial artists, and the first woman, Australian rules footballer Heather Anderson, who died last year at the age of 28 by an apparent suicide. Meanwhile, every state in the nation has passed laws aimed at protecting children who get concussions playing sports, and helmets have undergone overhauls in pursuit of the perfect padding. U.S.A. Football, which governs the amateur arm of the sport, has promoted "heads up" tackling and concussion training. Pop Warner introduced concussion guidelines in 2010 (including the mandate that anyone suspected of having a concussion must be cleared by a medical professional), banned full-speed head-on blocking and tackling drills in 2012, limited full-contact practices to 25 percent of the time and banned kickoffs (the game's most dangerous play) for athletes under the age of 11 in 2016, and got rid of the three-point stance (which lowers a player's head) in its younger divisions in 2019. (Changes have also been made to youth hockey and soccer.)

Yet many brain scientists and public-health officials question whether some of these changes are having a demonstrable effect, or even an effect at all, when it comes to the most insidious forms of brain injury. Many states' laws tend to deal with managing concussions after they happen, not preventing them. When it comes to helmets, improvements better protect the skull, but they do

not stop the brain from ricocheting inside of it, slamming against bone, torquing the brain stem; in fact, the more padding a helmet has — and thus the heavier it is — the more of a weapon it ostensibly becomes. A *British Journal of Sports Medicine* study from 2017 found that new protective equipment had no measurable effect on concussion prevention, much less on the prevention of non- or subconcussive blows. Leagues that use heads-up techniques have actually recorded slightly more concussions (though the increase was statistically insignificant).

According to Cantu and Nowinski, the only changes that demonstrably help are those that reduce contact overall, like the ones Pop Warner has implemented in the face of declining participation. Yet such changes are only as good as their enforcement, and while volunteer coaches may mean well — and may no longer subscribe to the myth that their job is to turn grade-schoolers into gladiators — they may also take false comfort in pronouncements that attempt to undermine the connection between CTE and collision sports and that are sometimes made by organizations with a financial stake in keeping that connection blurry. The International Consensus Conference on Concussion in Sport, an entity responsible for issuing the guidance that informs sports protocols across the globe — and one led by a number of scientists with ties to sports organizations — has consistently published statements that treat the science as unsettled, providing legal cover to conference sponsors like FIFA and World Rugby should they be accused of covering up known risks. In 2016, Pop Warner settled a case brought by the mother of a former player who died by suicide at age 25 and was later found to have CTE, opening the door to other potential lawsuits. ("At Pop Warner, there is nothing more important than the safety of our players, and since 2010, we have led the way in making the game of youth football a safer and better experience than ever before," the organization stated in response to the settlement.)

For whatever reason, there are certainly scientists who tend to focus not on what's known — that exposure to collision sports is the one constant in nearly all documented cases of CTE and that CTE is not the only negative neural outcome — but instead on what's unknown. "Nobody's looking at chess players to see if they die by CTE," says Dr. Michael Lewis, who specializes in preventive medicine and serves on the Pop Warner Youth Football Medical Advisory Board. While Lewis supports the changes Pop Warner has made, he also argues that they are solutions to a problem that may be overstated — or oversimplified by pointing a finger at collision sports. "I mean, I kind of look at it as the climate-change thing. Is the science settled on climate change? I don't think so. But what's the downside of cutting back on carbons? [When it comes to] getting your head hit or whatever, there's no downside to decreasing the exposures. We kind of look at it as, 'OK, the science is anywhere but settled, but let's be conservative and let's limit the amount of contact in practice.' Let me just say, it's not straightforward. It's the brain. It's complicated."

Such a stance puts one of the doctors advising the nation's largest youth-football organization behind not just the NIH but even the NFL in acknowledging the connection between football and CTE. But one need look no further than the American Academy of Pediatrics to see the sway football has had over science. In 1957, the academy advised against collision sports for children ages 12 and younger, concluding that "body-contact sports, particularly tackle football and boxing, are considered to have no place in programs for children [of this age]." Since that time, and even as an abundance of evidence of brain trauma has come to light, the academy's stance has softened. In 2015, the AAP — the same group that recommends against body-checking in hockey in players under the age of 15 due to concussion risk — released a policy statement that

essentially sidestepped the issue when it comes to America's most popular game. It conceded that eliminating youth tackle football would "likely lead to a decrease in the incidence of overall injuries, severe injuries, catastrophic injuries, and concussions," but also argued that it would "lead to a fundamental change in the way the game is played."

In light of this trade-off, the AAP, the agency tasked with advising parents and pediatricians about reasonable risk, essentially passed the responsibility for that determination on to them: "Participants in football must decide whether the potential health risks of sustaining these injuries are outweighed by the recreational benefits associated with proper tackling." (When approached about this policy, a representative from the AAP informed *Rolling Stone* that it was "currently undergoing an update.")

Thus far, CTE can only be diagnosed postmortem, though this month, Bailes' research team will begin stage three of an FDA trial to diagnose the disease in living people using PET scans and MRIs. This would be a significant breakthrough, not just enabling those with the disease to get early intervention but also allowing for the possibility of treatment. "There's plenty of potential treatments," says McKee. "But it's hard to assess their efficacy without having a biomarker to follow to see if it's getting better. That's part of the problem right now: We can't diagnose it during life, so we can't follow a treatment to see if it's actually beneficial. But I think we're going to get there."

Until then — and in light of not just CTE but other potential cognitive and mental-health outcomes — many doctors and scientists argue that the best approach from a public-health perspective is to outlaw the collision part of collision sports, at least until children's brains have passed the period of rapid development in their tween years. The Concussion Legacy Foundation's "Stop Hitting Kids in the Head" campaign has encouraged parents to hold off on enrolling their kids in tackle football until they are 14. Cantu has called on the surgeon general to ban tackle football for children under that age, pointing out that all of the health and social benefits of a team sport can be retained in less-violent versions of these games (in fact, the Manning brothers famously didn't start playing tackle football until middle school). McKee goes even further, arguing that no one should play a collision sport until they reach the age of consent, until they are emotionally mature enough to weigh the risks themselves and physically mature enough to better withstand them. That may sound extreme to football diehards, but not to McKee, who spends her days looking at damaged brains and speaking to the devastated families who donate them. "Part of the reason I can't let this go is I know what it's doing to people," she tells me. "And I also know it's preventable."

A TEXAS THUNDERSTORM is brewing, the air growing hot and thick with the smell of the coming rain, on the recent Saturday morning when Letitia Wilbourn visits her son's grave. Under the shade of the oak tree where the doves once perched, she rearranges the plastic flowers and carefully sweeps dust off the gravestone with a little brush she keeps in the car for that purpose. Her daughter Natasha stands there and cries. Wilbourn tries to keep herself busy. "I promised Quan I wouldn't break," she says, walking back to her car. "I told him I will always try to smile when I'm discussing him."

Back home, Wilbourn sits on the floor of her living room, in view of Myers' football trophies, surrounded by his photos, down the hall from the room where his clothes still hang in the closet. The extended family has gathered today, assorted cousins, aunts, uncles, and grandchildren passing through the room and in and out of the house, a tight-knit, boisterous group of which Wilbourn seems to be the calm and stoic center. Life goes on, certainly, even if, in some ways, it now revolves around the memory of death. "If Quan was still here right now, then he probably would be dealing with something mentally in his head," Natasha offers haltingly. "Like, how would he be today? Would he be outside walking around with his shirt off? No shoes? Where would he be?"

Wilbourn's oldest child, Monique, nods. They all know the CTE narrative, the tragic way it unfolds. "What would have been the progression? Would we have to have been looking for him out in the streets?"

"I don't know if I would be happy seeing him here right now with dementia versus what happened," Natasha says, beginning to cry again. Wilbourn reaches for her daughter's hand. Still, she doesn't break.

There are times when she does. Sometimes, she'll be driving and pass a field with peewee players huddled together, their little helmets and bodies clashing under the Texas sun, and she won't be able to look away. "I pull over and pray for every kid out there," she tells me. "I pray, and I cry." When people ask about her son, she tells them about CTE. She implores parents to consider other sports. She wants her son's death — her own suffering — to have some purpose, some meaning. But, she tells me, "this is Texas, and football is God to some people."

As we're talking, a 12-year-old boy walks into the room and sits silently on the sofa. He's Wilbourn's nephew's child. He started playing tackle football this past year, despite his family's experience with CTE, despite Wilbourn's admonishments, despite the fact that his father sits nervously on the sideline of each game, watching his son's head and the movements of his body, looking for signs of disaster. This very afternoon, the boy is a witness to his family's grief, yet it won't stop him from playing this game of neural Russian roulette. What can Wilbourn do? He isn't her child. All she can do is pray that maybe, just maybe, this is a game he'll win.

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Leveraging football accelerometer data to quantify associations between repetitive head impacts and chronic traumatic encephalopathy in males

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Chronic traumatic encephalopathy (CTE) is a neurodegenerative tauopathy associated with repetitive head impacts (RHI), but the components of RHI exposure underlying this relationship are unclear. We create a position exposure matrix (PEM), composed of American football helmet sensor data, summarized from literature review by player position and level of play. Using this PEM, we estimate measures of lifetime RHI exposure for a separate cohort of 631 football playing brain donors. Separate models examine the relationship between CTE pathology and players' concussion count, athletic positions, years of football, and PEM-derived measures, including estimated cumulative head impacts, linear accelerations, and rotational accelerations. Only duration of play and PEM-derived measures are significantly associated with CTE pathology. Models incorporating cumulative linear or rotational acceleration have better model fit and are better predictors of CTE pathology than duration of play or cumulative head impacts alone. These findings implicate cumulative head impact intensity in CTE pathogenesis.

Chronic traumatic encephalopathy (CTE) is a neurogenerative disease identified in individuals with exposure to repetitive head impacts (RHI), including military veterans, victims of physical violence, and contact and collision sports (CCS) athletes, including American football players[1,2]. CTE is distinguished from other neuropathologies by the pathognomonic lesion of perivascular accumulation of hyperphosphorylated tau in neurofibrillary tangles (NFTs), typically at the sulcal depths of the cerebral cortex, as well as diffuse NFTs in medial temporal lobe, diencephalon, basal ganglia, and brain stem[3].

Effectively quantifying the RHI exposure needed to develop CTE has been limited and challenging. Importantly, self or informant reported symptomatic concussion has not been associated with the presence or severity of CTE pathology[1,4,5]. However, total years of football played, thought to serve as a proxy for RHI, demonstrated a

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significant dose-response relationship with both CTE presence and CTE severity[5]. The finding that years of play is positively associated with CTE pathology, whereas symptomatic concussion is not, suggests that other regularly occurring exposures, such as RHI, may have an instrumental role in influencing CTE development. However, the assessment of concussion is problematic, as concussion definitions and diagnoses have changed over time, and many athletes report vastly different numbers of concussions based on definitions applied[6]. This difficulty accurately quantifying concussions experienced is further compounded in studies that attempt to estimate concussion burden based on informant report.

American football provides a unique model to better understand the relationship between RHI and CTE. Athletes begin playing football at different ages and for different career durations. The average frequency of impacts that an athlete experiences over a given season, and the average intensity of those impacts, varies based on position and level played[7]. We hypothesized that these differences could be leveraged to investigate the relationships between RHI-related factors and CTE pathology, potentially with implications relevant for football players, as well as other contact sport athletes.

To estimate an athlete's RHI exposure based on football position, duration, and level of play, data can be obtained from helmet accelerometers. Helmet accelerometers measure the head impact count and acceleration across different levels and positions of football play[8–41]. Building on the use of exposure matrices to quantify risks associated with exposures in other domains, data from helmet sensors have been previously utilized to approximate the potential number of head impacts that a football player may have received[42–45]. This cumulative head impact index (CHII), which incorporates an individual's career length, positions, and levels of play, represents an athlete's estimated lifetime number of football-related hits to the head[42–45]. CHII has been linked to former athletes' mood and cognitive symptoms, cerebrospinal fluid total tau levels, and all-cause mortality, but the relationship to CHII and underlying pathology has not been previously studied[42–45]. Further, since the CHII was developed, more recent helmet sensor data from youth, high school, and collegiate football players have been published. These additional data (as well as older data) include estimates of average linear and rotational acceleration, measures of acceleration that were not included in the original CHII. Incorporation of these additional data may improve estimation of an athletes' exposure to RHI and, by extension, improve estimation of the effect of RHI on CTE risk.

In this study, we sought to update the previously published CHII with updated data. Based on the well-established use of job exposure matrices to retrospectively characterize exposure to occupational hazards among workers[46], we similarly developed a positional exposure matrix (PEM) to retrospectively characterize exposure to RHI among former football players. The PEM was derived from published helmet sensor studies that reported findings specific to position and level of play. We used this PEM to calculate CHIIs for deceased former football players whose brains were donated for neuropathological analysis. In addition, we expanded this PEM beyond number of impacts to include average acceleration of impacts, to allow for the calculation of estimated lifetime exposure to linear (CHII-G) and rotational (CHII-R) acceleration. We hypothesized that CHII, CHII-G, and CHII-R would be correlated with the presence and severity of CTE pathology, as well as NFT burden, and that models incorporating the intensity of impacts (CHII-G and CHII-R), would have better model fit and make better predictions of CTE presence, CTE severity, and NFT burden than models that do not incorporate these data (CHII and duration of play alone). We hypothesized that informant-reported concussion number and position would not be associated with CTE presence, CTE severity, or NFT burden.

Results

Brain donor characteristics

A total of 631 brain donors who played American tackle football were included in the study. Characteristics of all brain donors are presented in Tables 1–3. On average, athletes died at 59.7 years old (SD = 20.1) and played 12.5 years of football (SD = 5.9). 180 athletes did not have CTE, 163 had low-stage CTE (Stage I or II), and 288 had high-stage CTE (Stage III or IV). The absolute and relative distributions of athlete duration of play by CTE status/severity are presented in Fig. 1 (with distributions for exposure subgroups presented in Supplementary Fig. 1). The mean duration of play for those without CTE was 9.5 (SD = 5.3) years, for those with low-stage CTE was 11.6 (SD = 5.0) years and for those with high-stage CTE was 15.0 (SD = 5.7) years. The mean CHII, CHII-G and CHII-R for those without CTE was 4,515 hits (SD = 3,199), 87,489 total g-force (SD = 47,178) and 6.57×10^6 total rad/s^2 (SD = 4.35×10^6) respectively; for those with low-stage CTE was 5,553 hits (SD = 3,410), 107,650 total g-force (SD = 41,755) and 8.32×10^6 total rad/s^2 (SD = 3.72×10^6) respectively; and for those with high-stage CTE was 7,641 hits (SD = 3,870), 148,777 total g-force (SD = 52,557) and 12.26×10^6 (SD = 5.0×10^6) respectively. The three CHII measures were highly correlated (CHII vs. CHII-G: Pearson's correlation (t) (degrees of freedom, df: 654) = 0.76, 95%CI: 0.72–0.79, $p < 2.2 \times 10^{-16}$; CHII vs. CHII-R: t(df: 654) = 0.66, 95%CI: 0.61–0.70, $p < 2.2 \times 10^{-16}$; CHII-G vs. CHII-R: t(df: 654) = 0.95, 95%CI: 0.95–0.96, $p < 2.2 \times 10^{-16}$).

Informant-reported concussion number not associated with CTE status, CTE severity, or neurofibrillary tangle burden

Across all brain donors, there was no association between informant-reported concussion number and CTE status (odds ratio [OR] per concussion (df: 587) = 1.00, 95% confidence interval (CI) = 1.00–1.00; $p = 0.23$; Table 4). For athletes with CTE, there was no association between informant-reported concussion number and CTE severity (OR per concussion (df: 418) = 1.00, 95% confidence interval (CI) = 1.00–1.00; $p = 0.49$; Table 5). There was no association between informant-reported concussion number and cumulative neurofibrillary tangle (NFT) burden (mean difference in NFT burden index per concussion (df: 478) = 1.56×10^{-4}, 95%CI = -1.96×10^{-3}–2.28×10^{-3}; $p = 0.95$; Table 6). All estimates above were adjusted for age at death (models unadjusted for age are included in Supplementary Tables 1–3).

Position at highest level not associated with CTE status, CTE severity, or neurofibrillary tangle burden

Across a range of analyses, primary playing position had no meaningful association with CTE related outcomes. We observed no association between football position played at the highest level and CTE status with positions evaluated individually (all ps > 0.05, Supplementary Table 4) or dichotomously (i.e., speed vs non-speed, defined previously[47]; OR(df; 467) = 1.39, 95%CI = 0.89–2.16; $p = 0.15$, Table 4). For those athletes with CTE ($n = 362$), there was no association between position played at the highest level and CTE severity (individually: all ps > 0.05; Supplementary Table 4. Dichotomously: $p = 0.49$; Table 6). There was no association between position played at the highest level and NFT burden ($n = 385$; *individually*: all ps > 0.05 except running back $p = 0.002$, with OR = 0.59, 95%CI = 4.4–801.8; Supplementary Table 4; *dichotomously*: $p = 0.08$; Table 6). These analyses restricted to brain donors with a single known position at the highest level of play. All models were adjusted for age at death.

Duration and cumulative exposure measures associated with CTE status and severity

There was a significant association between duration of play, as well as the cumulative exposure measures, and CTE status (all ps < 0.001; Table 4), or severity (all ps < 0.001; Table 5), adjusted for age at death. Each additional year of play was associated with 15% increased odds of being diagnosed with CTE (df: 630, 95%CI: 1.11–1.19, $p = 9.4 \times 10^{-13}$) and,

Table 1 | Demographic characteristics of brain donors by CTE status

	No CTE (*n* = 180)	Mild CTE (*n* = 163)	Severe CTE (*n* = 288)	All (*n* = 631)
Age at death, mean (SD)	53.0 (21.8)	47.5 (19.0)	70.9 (12.2)	59.7 (20.1)
Age at death, median (IQR; range)	57 (36,49; 13,97)	47 (30,62; 17,89)	73 (65,79; 30,97)	65 (46,76; 13,97)
Cause of death (%)				
Accidental overdose	12 (6.9%)	10 (6.3%)	5 (1.8%)	27 (4.4%)
Cancer	11 (6.3%)	10 (6.3%)	23 (8.2%)	44 (7.1%)
Cardiovascular disease	29 (16.6%)	28 (17.5%)	47 (16.7%)	104 (16.9%)
Injury	6 (3.4%)	9 (5.6%)	5 (1.8%)	20 (3.2%)
Motor neuron disease	2 (1.1%)	7 (4.4%)	12 (4.3%)	21 (3.4%)
Neurodegenerative disease	48 (27.4%)	14 (8.8%)	141 (50.0%)	203 (32.9%)
Suicide	46 (26.3%)	48 (30.0%)	14 (5.0%)	108 (17.5%)
Other	20 (11.4%)	34 (21.3%)	35 (12.4%)	89 (14.4%)
Unknown	1 (0.6%)	0 (0.0%)	0 (0.0%)	1 (0.2%)
Race (%)				
Asian	1 (0.6%)	0 (0.0%)	0 (0.0%)	1 (0.2%)
Black/African American	21 (11.7%)	29 (17.9%)	51 (17.7%)	101 (16.1%)
Native American/Alaskan	2 (1.1%)	1 (0.6%)	1 (0.3%)	4 (0.6%)
Pacific islander	1 (0.6%)	1 (0.6%)	1 (0.3%)	3 (0.5%)
White	151 (84.4%)	127 (78.4%)	234 (81.3%)	512 (81.4%)
Other	3 (1.7%)	4 (2.5%)	1 (0.3%)	8 (1.3%)
Sex (%)				
Female	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Male	180 (100.0%)	163 (100.0%)	288 (100.0%)	631 (100.0%)
Education level (%)				
No high school	3 (1.7%)	0 (0.0%)	1 (0.3%)	4 (0.6%)
Some high school	10 (5.6%)	2 (1.2%)	0 (0.0%)	12 (1.9%)
High school/GED	21 (11.7%)	3 (1.9%)	4 (1.4%)	28 (4.4%)
Some college	47 (26.1%)	40 (24.7%)	46 (16.0%)	133 (21.3%)
College degree	56 (31.3%)	87 (53.7%)	150 (52.1%)	293 (46.5%)
More than college	6 (3.3%)	3 (1.9%)	22 (7.6%)	31 (4.9%)
Graduate degree	37 (20.6%)	27 (16.7%)	65 (22.6%)	129 (20.5%)

CTE chronic traumatic encephalopathy, *IQR* interquartile range, *SD* standard deviation

amongst those with CTE, 14% increased odds of being diagnosed with severe CTE (df: 450, 95% CI: 1.08–1.20, $p = 4.5 \times 10^{-7}$). Every additional estimated 1,000 head impacts was associated with 21% increased odds of being diagnosed with CTE (df: 630; 95%CI: 1.13–1.29, $p = 7.6 \times 10^{-9}$) and, among those with CTE, 13% increased odds of being diagnosed with severe CTE (df: 448; 95%CI: 1.05–1.22, $p = 9.2 \times 10^{-4}$). Every additional estimated 10,000 g cumulative linear acceleration to the head was associated with 20% increased odds of being diagnosed with CTE (df: 630; 95%CI: 1.15–1.25, $p = 2.0 \times 10^{-15}$) and, among those with CTE, 19% increased odds of being diagnosed with severe CTE (df: 450; 95% CI: 1.12–1.26, $p = 1.1 \times 10^{-8}$). Every additional estimated 1,000,000 rad/sec^2 cumulative rotational acceleration to the head was associated with 22% increased odds of being diagnosed with CTE (df: 630; 95%CI: 1.16–1.29, $p = 7.0 \times 10^{-15}$) and, among those with CTE, 20% increased odds of being diagnosed with severe CTE (df: 450; 95%CI: 1.13–1.28, $p = 1.7 \times 10^{-8}$).

CHII-G and CHII-R classified CTE status better than duration of play or CHII

Based on the Bayesian information criterion (BIC), there was very strong (ΔBIC > 10) evidence for improved model fit for models estimating the relationship between CTE status and either duration of play, CHII-G, or CHII-R, compared with the model using CHII. Additionally, there was very strong evidence (ΔBIC > 10) for improved model fit for models using either CHII-G and CHII-R over duration of play alone. There was no evidence of differences in model fit for the model incorporating CHII-G compared to CHII-R (ΔBIC < 2).

ROC analyses (Fig. 2A) indicated significant improvement in classifying CTE status for models using CHII-G (area under curve (AUC) =0.757, 95%CI: 0.715–0.798; $p = 9.5 \times 10^{-5}$) or CHII-R (AUC = 0.766, 95% CI: 0.725–0.807; $p = 1.5 \times 10^{-5}$), but not CHII (AUC = 0.698, 95%CI: 0.651–0.742; $p = 0.22$) compared to models using duration of play alone (AUC = 0.716, 95%CI: 0.675–0.760). There were similar significant improvements in classification using CHII-G ($p = 2.6 \times 10^{-4}$) and CHII-R ($p = 9.9 \times 10^{-5}$) compared to CHII. There were no significant differences between CHII-G and CHII-R in classifying CTE status ($p = 0.19$). Similarly, 10-fold cross validation analyses found lowest mean squared error (MSE) for CHII-R (MSE = 0.169) and CHII-G (MSE = 0.170), followed by duration of play (MSE = 0.177) and then CHII (MSE = 0.183).

CHII-G and CHII-R better at classifying CTE severity than duration of play or CHII

When restricted to individuals with CTE, the BIC provided very strong evidence (ΔBIC > 10) for improved model fit for models estimating the relationship between CTE severity and either duration of play, CHII-G, or CHII-R compared with the model using CHII. Additionally, there was strong evidence (ΔBIC > 8) for improved model fit for models using either CHII-G and CHII-R over duration of play alone. There was no evidence of differences

Table 2 | Repetitive head impact exposure characteristics of brain donors by CTE status

	No CTE (*n* = 180)	Mild CTE (*n* = 163)	Severe CTE (*n* = 288)	All (*n* = 631)
Number of reported concussions, mean (SD)	67.0 (283.4)	89.2 (211.0)	115.1 (350.8)	94.5 (301.2)
Concussion number, median (IQR; range)	15 (6,50; 0,3500)	20 (7,87; 0,1500)	25 (9,100; 0,5000)	20 (7,92; 0,5000)
Age of first exposure to football (SD)	10.9 (3.8)	10.2 (3.6)	12.1 (2.8)	11.3 (3.4)
Duration of football in years (SD)	9.5 (5.3)	11.6 (5.0)	15.0 (5.7)	12.5 (5.9)
Highest level of football play (%)				
Pre-high school	10 (5.6%)	6 (3.7%)	1 (0.3%)	17 (2.7%)
High school	60 (33.3%)	29 (17.8%)	8 (2.8%)	97 (15.4%)
College	61 (33.9%)	64 (39.3%)	81 (28.1%)	206 (32.6%)
Semi-professional	16 (8.9%)	7 (4.3%)	4 (1.4%)	27 (4.3%)
Professional	33 (18.3%)	57 (35.0%)	194 (67.4%)	284 (45.0%)
Position played at highest level (%)				
Defensive back	10 (5.6%)	21 (12.9%)	29 (10.1%)	60 (9.5%)
Defensive line	23 (12.8%)	26 (16%)	37 (12.8%)	86 (13.6%)
Kicker	1 (0.6%)	1 (0.6%)	0 (0.0%)	0 (0.0%)
Linebacker	14 (7.8%)	26 (16.0%)	33 (11.5%)	73 (11.6%)
Offensive line	25 (13.9%)	23 (14.1%)	50 (17.4%)	98 (15.5%)
Punter	0 (0.0%)	2 (1.2%)	0 (0.0%)	2 (0.3%)
Quarterback	10 (5.6%)	7 (4.3%)	13 (4.5%)	30 (4.8%)
Running back	10 (5.6%)	10 (6.1%)	52 (18.1%)	72 (11.4%)
Tight end	8 (4.4%)	4 (2.5%)	13 (4.5%)	25 (4.0%)
Wide Receiver	5 (2.8%)	5 (3.1%)	10 (3.5%)	20 (3.2%)
Multiple	64 (35.6%)	32 (19.6%)	48 (16.7%)	144 (22.8%)
Other	4 (2.2%)	1 (0.6%)	0 (0.0%)	5 (0.8%)
Unknown	6 (3.3%)	5 (3.1%)	3 (0.1%)	14 (2.2%)
Participation in other contact/collision sport (%)	62 (34.4%)	53 (32.5%)	55 (19.1%)	170 (26.9%)
Military history (%)	41 (22.9%)	24 (14.8%)	82 (28.5%)	147 (23.4%)
Military combat exposure (%)	7 (3.9%)	7 (4.4%)	7 (2.4%)	21 (3.4%)
Exposure indices				
CHII, mean (SD)	4,515 (3,199)	5,553 (3,410)	7,641 (3,870),	6,117 (3,815)
CHII, median (IQR; range)	3963 (2191,6220; 206,18222)	4613 (3227,7027; 206,18384)	6841 (4830,9521; 206,19240)	5381 (3445,6118; 206,19240)
CHII-G, mean (SD)	88,972 (47,178)	107,650 (41,755)	148,777 (52,557)	119,081 (55,451)
CHII-G, median (IQR; range)	89,411 (60757,110939; 4246,280130)	110,054 (82076,131468; 4246,233872)	145,219 (106432,189016; 4246,310242)	112,741 (83737,151860; 4246,310242)
CHII-R, mean (SD)	6.57×10^6 (4.35×10^6)	8.32×10^6 (3.72×10^6)	12.26×10^6 (5.0×10^6)	9.48×10^6 (5.19×10^6)
CHII-R, median (IQR; range)	6.28×10^6 (4.00×10^6,8.30×10^6; 0.25×10^6,29.9×10^6)	8.16×10^6 (5.81×10^6,10.4×10^6; 0.25×10^6,18.9×10^6)	12.0×10^6 (8.30×10^6,15.5×10^6; 0.25×10^6.34.7×10^6)	8.48×10^6 (6.21×10^6,12.69×10^6; 0.25×10^6,34.7×10^6)

CTE chronic traumatic encephalopathy, *IQR* interquartile range, *SD* standard deviation

in model fit for the model incorporating CHII-G compared to CHII-R (ΔBIC < 2).

ROC analyses (Fig. 2B) found significantly improved performance in classifying CTE severity for models using CHII-G (AUC = 0.717, 95% CI: 0.670–0.764; $p = 8.5\times10^{-7}$) or CHII-R (AUC = 0.726, 95%CI: 0.678–0.772; $p = 7.6\times10^{-6}$), but not CHII (AUC = 0.674, 95%CI: 0.620–0.725; $p = 0.75$) compared to models using duration of play alone (AUC = 0.668, 95%CI: 0.617–0.718). Similarly, there were significant improvements in classification using CHII-G ($p = 0.014$) and CHII-R ($p = 0.009$) compared to CHII. There were no significant differences between CHII-G and CHII-R in classifying CTE status ($p = 0.21$). Similarly, 10-fold cross validation analyses found lowest mean error for CHII-R (MSE = 0.134) and CHII-G (MSE = 0.134), followed by duration of play (MSE = 0.138) and then CHII (MSE = 0.144).

Duration of play, CHII-G, or CHII-R preferred over CHII for modeling NFT burden

In models adjusted for age at death, there was also a significant relationship between increasing NFT burden and increasing years of football played, in addition to all cumulative exposure measures ($n = 519$, all ps < 0.001, Table 6). The models incorporating duration of play, CHII-G, and CHII-R, had higher R^2s and showed very strong evidence for improved model fit (ΔBIC > 10, Table 6) compared to models incorporating CHII.

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Table 3 | Neuropathologic and clinical characteristics of brain donors by CTE status

	No CTE (*n* = 180)	Mild CTE (*n* = 163)	Severe CTE (*n* = 288)	All (*n* = 631)
Other neuropathologic diagnoses (%)				
Alzheimer's disease	39 (21.7%)	4 (2.5%)	56 (19.4%)	99 (15.7%)
Frontotemporal lobar degeneration	11 (6.1%)	11 (6.8%)	26 (9.1%)	48 (7.6%)
Lewy body disease	24 (13.4%)	13 (8.0%)	64 (22.2%)	101 (16.1%)
Motor neuron disease	0 (0.0%)	7 (4.3%)	13 (4.5%)	20 (3.2%)
Diagnosed dementia (%)	63 (36.7%)	45 (28.0%)	216 (76.3%)	324 (52.6%)

CTE chronic traumatic encephalopathy, *IQR* interquartile range, *SD* standard deviation

Sensitivity analyses: 1) after removing athletes who played multiple contact sports, 2) after removing athletes who had other neurodegenerative pathologies, and 3) without adjustment for age at death.

While all athletes included in the above analyses played football, many had other exposures to RHI besides football; 26.9% of athletes (*n* = 170) participated in contact sports besides football and 23.4% (*n* = 147) served in the military (Supplementary Fig. 1). Similar relationships as reported above were observed between exposure measures and CTE outcomes when restricted to athletes with no military history or other contact sport exposure besides football (Supplementary Tables 5 and 6).

Additionally, some athletes had other neurodegenerative diseases besides CTE; 15.7% of athletes had Alzheimer's disease (*n* = 99), 7.6%

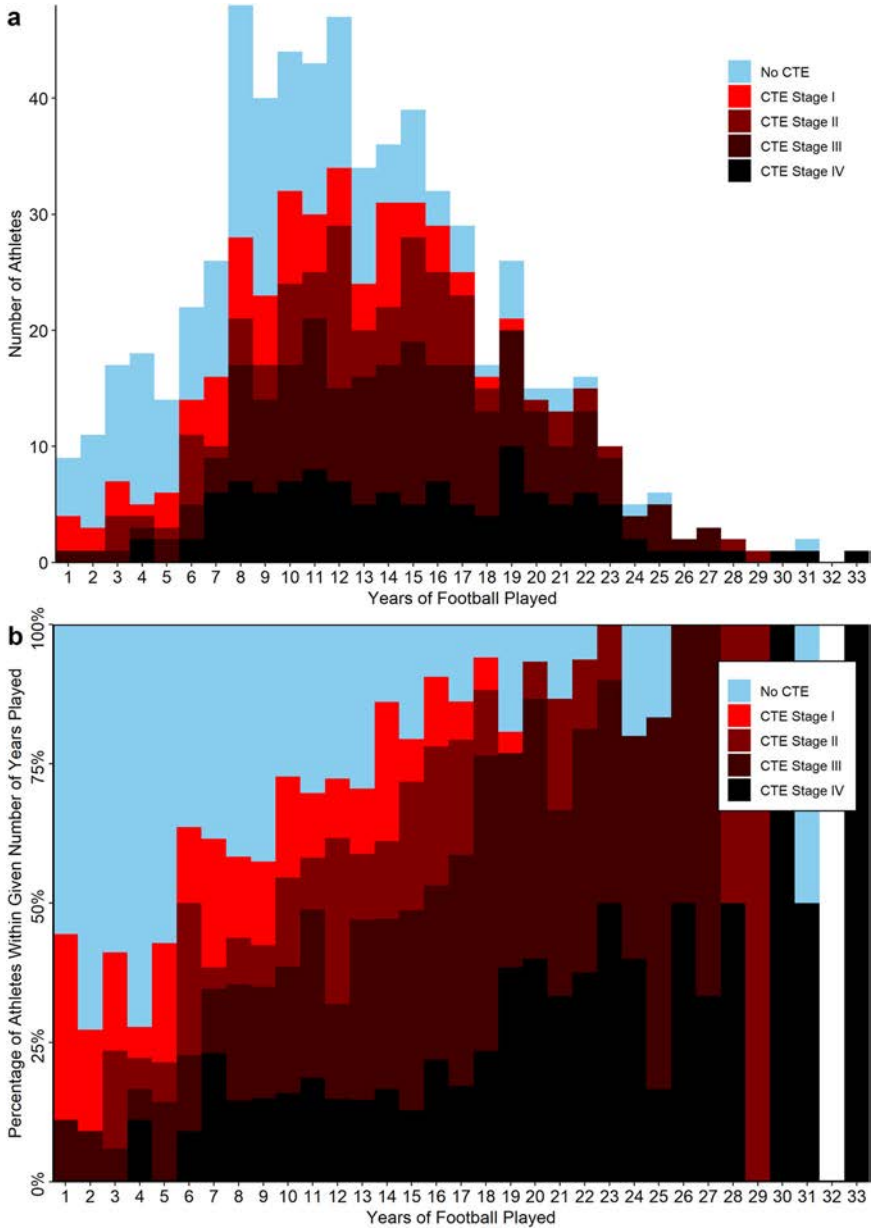

Fig. 1 | Athlete CTE Status by Years of American Football Played. Histograms (**a**) and percent distributions (**b**) for the study sample by duration of play. Of note: among the 69 athletes who played ≤5 years of football, 35 played another contact sport (13 of whom had CTE), 21 were in the military (7 of whom had CTE), and 9 were exposed to combat as part of military service (4 of whom had CTE). 47 of the 69 athletes had one or more of these exposures (16 of whom had CTE). Of the 69 athletes who played ≤5 years of football, 9 had CTE and none of these exposures. Please see Supplementary Fig. 1 for additional details. Source data are provided as a Source Data file. CTE chronic traumatic encephalopathy.

Table 4 | Association between exposure measures and CTE status (absent versus present)

	Odds ratio (95% CI)	*p*-value	BIC	Cross-validation mean error	AUC	*p*-value[a]
Concussion number	1.00 (1.00–1.00)	0.23				
Position, non-speed vs speed[b]	1.39 (0.89–2.16)	0.15				
Duration of play per year	1.15 (1.11–1.19)	9.4×10^{-13}	685.3	0.177	0.716	
CHII per 1,000 hits	1.21 (1.13–1.29)	7.6×10^{-9}	705.0	0.183	0.698	0.22
CHII-G per 10,000 g	1.20 (1.15–1.25)	2.0×10^{-15}	665.9	0.170	0.757	9.5×10^{-5}
CHII-R per 1,000,000 rad/sec^2	1.22 (1.16–1.29)	7.0×10^{-15}	665.2	0.169	0.763	1.5×10^{-5}

Separate logistic regressions were run for each exposure measure due to multicollinearity to determine odds ratios, and *p*-values. For models with significant exposure measures, BIC and the mean error resulting from a 10-fold cross-validation analysis are reported to determine relative model performance. All models had the outcome of CTE status (absent vs present) and were adjusted for age at death

AUC receiver operating characteristics area under curve, *BIC* Bayesian information criterion, *CHII* cumulative head impact index representing estimated number of head impacts per donor per 1,000 hits, *CHII-G* cumulative head impact index representing estimated cumulative g-force experienced by each donor per 10,000 g, *CHII-R* cumulative head impact index estimated cumulative rotational force experienced by each donor per 1,000,000 rad/sec^2, *CTE* chronic traumatic encephalopathy

[a]AUC *p*-value represents results of bootstrap analysis with 2000 replicates drawn from the sample to determine if there was a true difference between the AUCs for models examining CTE status and duration of play compared to other exposure measures

[b]Presented as dichotomous non-speed (offensive and defensive lineman) vs speed (all other positions) for all football players with a single known position

Table 5 | Association between exposure measures and CTE severity (mild versus severe)

	Odds ratio (95% CI)	*p*-value	BIC	Cross-validation mean error	AUC	*p*-value[a]
Concussion number	1.00 (1.00–1.00)	0.49				
Position, non-speed vs speed[b]	1.37 (0.79–2.36)	0.26				
Duration of play per year	1.14 (1.08–1.20)	4.5×10^{-7}	399.9	0.138	0.668	
CHII per 1,000 hits	1.13 (1.05–1.22)	9.2×10^{-4}	418.2	0.144	0.674	0.75
CHII-G per 10,000 g	1.19 (1.12–1.26)	1.1×10^{-8}	390.2	0.134	0.717	8.5×10^{-7}
CHII-R per 1,000,000 rad/sec^2	1.20 (1.13–1.28)	1.7×10^{-8}	391.8	0.134	0.726	7.6×10^{-6}

Separate logistic regressions were run for each exposure measure due to multicollinearity to determine odds ratios and *p*-values. For models with significant exposure measures, BIC and the mean error resulting from a 10-fold cross-validation analysis are reported to determine relative model performance. All models had the outcome of CTE severity (mild vs severe) and were adjusted for age at death

AUC receiver operating characteristics area under curve, *BIC* Bayesian information criterion, *CHII* cumulative head impact index representing estimated number of head impacts per donor per 1,000 hits, *CHII-G* cumulative head impact index representing estimated cumulative g-force experienced by each donor per 10,000 g, *CHII-R* cumulative head impact index estimated cumulative rotational force experienced by each donor per 1,000,000 rad/sec^2, *CTE* chronic traumatic encephalopathy

[a]AUC *p*-value represents results of bootstrap analysis with 2000 replicates drawn from the sample to determine if there was a true difference between the AUCs for models examining CTE status and duration of play compared to other exposure measures

[b]Presented as dichotomous non-speed (offensive and defensive lineman) vs speed (all other positions) for all football players with a single known position

had frontotemporal lobar degeneration ($n = 48$), 16.1% had Lewy body disease ($n = 101$), and 3.2% had motor neuron disease ($n = 20$). Similar relationships as reported above were also observed when excluding athletes with any other neurodegenerative process (Supplementary Tables 5 and 6).

Adjusting for age at death may remove some of the variance in the neuropathological outcomes that the RHI exposures measures may explain. To investigate if this was the case, we re-ran our analyses without age in the models. Point estimates for the RHI exposure measures did not meaningfully change, but overall model fit and predictive power were mildly reduced (Supplementary Tables 1–3).

Discussion

We investigated which components of RHI exposure from football play may be implicated in CTE pathology. The PEM reported here adapts the use of exposure matrices from other disciplines, and builds upon the previous use of CHII to quantify exposure to RHI. To our knowledge, this is the first study to use a PEM to estimate the relationship between different forms of RHI exposure and CTE pathology.

This PEM is a tool for quantifying a football player's lifetime exposure to RHI, by estimating the cumulative number of head impacts as well as the cumulative linear and rotational accelerations associated with those impacts. We demonstrated an association between cumulative RHI exposure and CTE status, CTE severity, and NFT burden in football players. In general, model performance and fit using cumulative linear and rotational accelerations were improved compared to models with cumulative number of head impacts and years of play. Additionally, we found no relationship between informant-reported concussion number and CTE status, CTE severity, or NFT burden. Position played at the highest level was not associated with CTE status, CTE severity, or NFT burden, with the exception of a relationship observed between NFT burden, but not CTE status or severity, for running backs compared to offensive linemen.

These results provide additional evidence that repeated non-concussive injuries are associated with CTE pathology. This is in contrast to the emphasis on concussions that is often discussed in the medical and lay literature[48]. Further, these results suggest that models incorporating intensity of impacts (i.e., linear and rotational acceleration) have better model fit and are better at predicting CTE status and severity than models incorporating duration of play or number of hits to the head alone. These results, if validated, could be used to identify changes to policy or gameplay that might limit CTE risk by decreasing cumulative exposure, such as by limiting duration of exposure, the number of exposures, and the magnitude of those exposures.

This study also validates the use of a PEM to characterize the aspects of RHI exposure experienced by football players. Other outcomes besides CTE believed to be associated with RHI exposure, such as measures of diffuse axonal injury[49], neuroinflammatory markers[50,51], or other neuropathologic processes[52], could be evaluated in the context of exposures derived from the PEM to better understand the characteristics of the exposure most responsible for these changes.

Table 6 | Association between exposure measures and neurofibrillary tangle burden

	Mean increase in NFT burden per unit increase in respective measure (95% CI)	R^2	*p*-value	BIC
Athletes with all 11 brain regions available for analysis ($n = 519$)				
Concussion number	1.56×10^{-4} (-1.96×10^{-3}–2.28×10^{-3})	0.43	0.89	
Position, non-speed vs speed[a]	1.34 (−0.26–2.93)	0.35	0.10	
Duration of play per year	0.47 (0.36–0.57)	0.49	3.8×10^{-16}	3558
CHII per 1,000 hits	0.44 (0.26–0.62)	0.45	1.8×10^{-6}	3602
CHII-G per 10,000 g	0.51 (0.39–0.63)	0.49	2.6×10^{-16}	3557
CHII-R per 1,000,000 rad/sec^2	0.52 (0.39–0.65)	0.49	7.6×10^{-15}	3564

Separate linear regressions were run for each exposure measure due to multicollinearity to determine betas, R^2, and *p*-values. For models with significant exposure measures, BIC is reported to determine relative model performance. All models had the outcome of semi-quantitative NFT burden summed across 11 brain regions (0–33) and were adjusted for age at death. The sum score was based on neuropathologists semi-quantitative NFT burden on a 0–3 scale with increasing severity for 11 brain regions implicated in CTE: dorsolateral frontal cortex, middle frontal cortex, orbitofrontal cortex, hippocampus regions CA1, CA2, CA3/4, substantia nigra, amygdala, entorhinal cortex, inferior parietal cortex, and locus coeruleus. Results are presented for the 519 athletes with available tissue for all 11 brain regions

BIC Bayesian information criterion, *CHII* cumulative head impact index representing estimated number of head impacts per donor per 1,000 hits, *CHII-G* cumulative head impact index representing estimated cumulative g-force experienced by each donor per 10,000 g, *CHII-R* cumulative head impact index estimated cumulative rotational force experienced by each donor per 1,000,000 rad/sec^2, *CTE* chronic traumatic encephalopathy, *NFT* neurofibrillary tangle

[a]Presented as dichotomous non-speed (offensive and defensive lineman) vs speed (all other positions) for all football players with a single known position

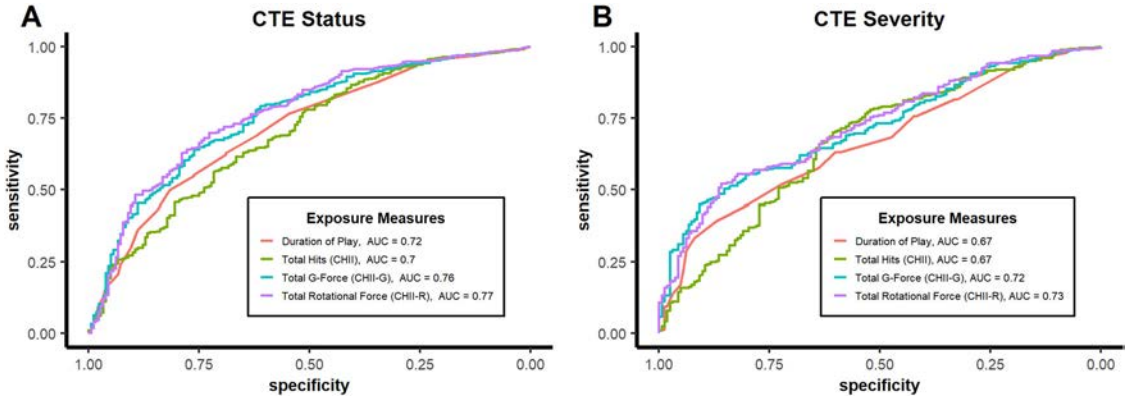

Fig. 2 | Performance of Exposure Measures as Classifiers of CTE Pathology. ROC Curves for Exposure Measures as Predictors of CTE Status (**A**) and CTE Severity (**B**). Source data are provided as a Source Data file. AUC area under the ROC curve, CHII cumulative head impact index representing estimated number of head impacts per donor, CHII-G cumulative head impact index representing estimated cumulative g-force experienced by each donor, CHII-R cumulative head impact index estimated cumulative rotational force experienced by each donor, CTE chronic traumatic encephalopathy, ROC receiver operating characteristics.

Several biomechanical factors may explain why models testing the association between cumulative acceleration and CTE pathology have better fit than models incorporating the number of head impacts alone. Increasing linear forces are associated with focal injuries[53]; greater forces may therefore result in more areas of localized damage[54]. Furthermore, rotational forces may result in increased shear-induced damage, particularly around small blood vessels and at the depths of the sulci[49].

This study utilized a PEM generated solely from available helmet accelerometer literature. Helmet accelerometer studies typically report results pertaining to head impacts greater than 10–15 g[35]. These thresholds were chosen to exclude incidental movements and jostling between the helmet and head[55], but there is no evidence that these thresholds are most relevant to CTE pathology[56]. Future work should explore different thresholds for counting hits, to further characterize the nature of head impacts most responsible for CTE pathology.

The position an athlete plays serves as a proxy for the RHI exposure experienced. The fact that position at the highest level was not associated with CTE status, CTE severity, or NFT burden (the latter for all positions but running backs), whereas cumulative measures incorporating position were, suggests that one single position inadequately represents an athlete's lifetime RHI exposure. Cumulative exposure measures appear to better approximate RHI exposure compared to single positions, likely because the former better accounts for the fact that football players change positions throughout their careers and often play multiple positions. Future measures should incorporate special teams participation (e.g. kickoff and punt), as this can vary by week and RHI from these positions may not be otherwise incorporated.

This study has several limitations. The study consists of a convenience sample of football playing brain donors, who tend to have greater exposure to RHI than the general population of football players. Even the athletes with lower years of exposure to football often had exposure to RHI from other sources, including other contact sports or military service, which were not characterized in this study. Along these lines, 82% of the subjects in this study played at the college level or above; predictions and thresholds from the present study would likely be most applicable to athletes with similar high levels of exposure. However, a substantial number of subjects had lower exposures ($n = 17$ with only youth participation and $n = 95$ only through high school), so we are not extrapolating to exposure ranges for which we have no data.

There are several sources of potential measurement error related to the cumulative RHI exposure measures: These measures were not observed directly while the donors were alive, but instead extrapolated from helmet accelerometer studies of other

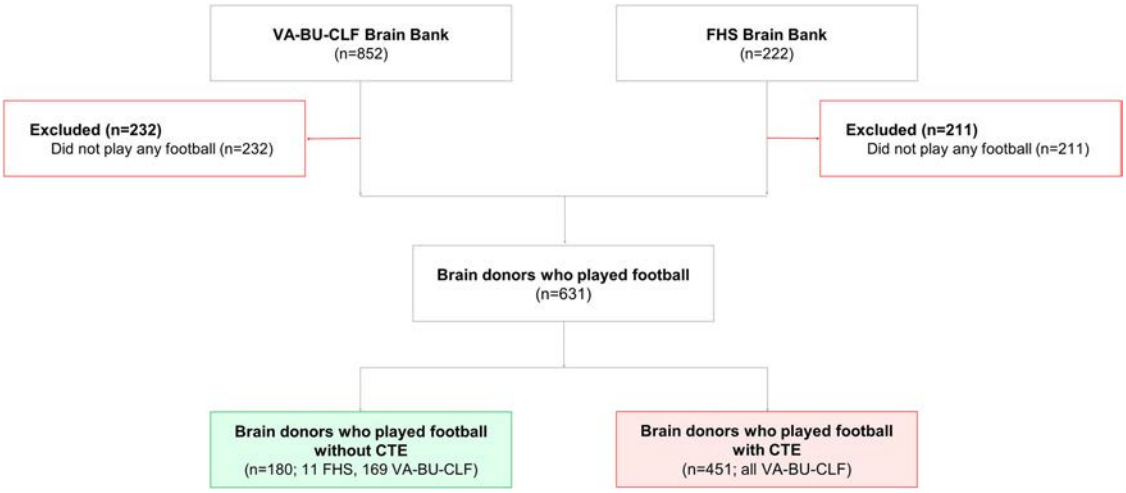

Fig. 3 | Study inclusion and exclusion criteria for FHS and VA-BU-CLF study brain donors. Source data are provided as a Source Data file. BU Boston University, CLF Concussion Legacy Foundation, FHS Framingham Heart Study, VA Veterans Affairs.

recent football players. Additionally, play style has changed over decades[57], and many of the donors played decades ago, but the extrapolated data do not reflect these changes. Some studies reported means while others reported medians and we considered them equivalently. Also, the helmet accelerometer technology and the minimum thresholds for measured hits (e.g., 10 g vs 15 g) differed across studies. These studies also reported averages across all athletes at a given level, but donors in this cohort, particularly those that ultimately played professionally, likely had different RHI exposures than an average athlete. Additionally, accelerometers in helmets may move independently from the head and may not accurately indicate acceleration experienced by the brain[58]. Because the professional football leagues have not made their helmet sensor data available, we extrapolated collegiate athlete exposure to professional athletes. Furthermore, athletes play multiple positions even within a given season; these analyses averaged up to two positions per season but may not accurately reflect uneven distributions of playing time for a given athlete. Special teams participation could introduce measurement error, as impacts during kickoffs and punts, which tend to be high magnitude, were not incorporated into the PEM-derived cumulative exposure measures. Even with several sources of potential measurement error, measurement error can be overcome with a sufficiently large sample size, provided there is not bias in the estimation. We do not have reason to suspect that measurement error differed by neuropathological status to introduce bias. Given that we were able to find a robust relationship between the estimated cumulative measures of RHI exposure and CTE pathology in the absence of a reasonable explanation for bias, it seems this sample size was sufficiently large to overcome potential measurement error. There are additional limitations. CTE diagnoses and staging may have been obscured by comorbid pathology. However, we would not expect this to bias the reported results as the pathologists were blinded to the athletic history, upon which the RHI exposure measures were based. Concussion number was also obtained retrospectively from informant report and validated with medical records where available; given changes in concussion diagnoses over decades[57], as well as recall bias and use of informant report, these reported concussion numbers may not accurately reflect concussion exposure. As a result, it is possible that a true relationship between number of symptomatic head impacts and CTE pathology exists, but that measurement error obscured this relationship. However, these difficulties in diagnosing concussions are neither new, nor resolved, so the present study demonstrates a potential means of estimating head impact burden that is robust to these issues.

This work utilized a PEM to better characterize which components of RHI exposure from football play may be implicated in CTE pathology. Models incorporating impact intensity, in addition to duration of exposure and number of impacts, improved model fit and prediction of CTE pathology compared with models without impact intensity. These findings provide insight into the pathogenesis of CTE, implicate potential targets for CTE interventions, and indicate that future studies may benefit from using PEM-derived measures of RHI exposure.

Methods

Brain donor recruitment

All participants were former football players from the Understanding Neurologic Injury and Traumatic Encephalopathy (UNITE) and Framingham Heart Study (FHS) Brain Banks (Fig. 3), which have been previously described[5]. Donors to the UNITE Brain Bank must have had a history of repetitive head impact (RHI) exposure through contact and collision sports (CCS), military service, or domestic violence. Recruitment to the UNITE Brain Bank began in 2008 and is ongoing; the present study included donors through 2020. Since tracking began in 2014, recruitment occurred in the following ways. Next-of-kin contacting the brain bank near or at the time of death was the most common recruitment method ($n = 433$). Other forms of recruitment included individuals joining the CLF Brain Donation Registry during life ($n = 18$), a medical examiner contacting the brain bank ($n = 33$), a CLF representative contacting the next-of-kin after the time of death ($n = 19$), and a consult request ($n = 1$). No sex or gender analyses were carried out as all football players in this cohort were male.

FHS is a prospective surveillance study tracking incident cardiovascular disease, stroke, and dementia. The study was established in 1948, enrolling a representative group from Framingham, Massachusetts and subsequently their children (Gen 2) and grandchildren (Gen 3), as well as ethnically diverse cohorts (Omni 1&2) to better capture changing demographics of Framingham. The FHS Brain Bank contains a voluntary subset of these cohorts, and began recruiting in 1997. Because the FHS is a community-based sample, players tended to be older and to have fewer years of American football play. Their addition resulted in more total players without CTE and in a similar distribution of age-at-death between players with and without CTE.

For both brain banks, prospective donors were excluded if there was a postmortem interval for donation greater than 72 hours, or if fragments or less than a hemisphere of tissue were received. Consent

from participant's next-of-kin was required to participate. Institutional review board approval was obtained through Boston University Medical Campus and Bedford VA Hospital.

Clinical data

For the UNITE Brain Bank, previously detailed methods for retrospective clinical data collection and comprehensive review of all relevant medical records were followed for all participants. A structured clinical history was obtained from informants, including a timeline of cognitive, behavioral, mood and motor symptomatology. Clinicians with expertise in neurodegenerative disease reviewed all cases to reach consensus on a dementia diagnosis based on DSM-IV-TR criteria. Clinicians and clinical research assistants were blinded to the neuropathological examination and findings[59].

For the FHS brain bank, clinical data were obtained prospectively as part of the FHS clinical assessment. Participants underwent cognitive assessment and those with suspected cognitive impairment were brought to a consensus meeting, during which it was determined whether the donor met criteria for dementia[60].

Contact sport and traumatic brain injury (TBI) history

Retrospective assessment of contact sport and TBI histories from informants were similar in both brain banks and have been described previously[1,5,59]. For each CCS sport exposure (football or otherwise), informants provided a history of levels played, years played at each level, and age of first exposure. Football position at each level was also recorded. In addition, athletic history for professional football players was verified with a comprehensive database maintained by Hidden Game Sports/24-7 Baseball LLC, which has been previously used for research purposes[1,5,61]. Informants were also asked if donors had any military service or combat exposure. Each donor's TBI history was queried from informants, including assessing the total number of concussions experienced. Following amendments made to study protocols, informants for donors included in the study after January 2014 were read a formal definition of concussion in advance of being asked how many concussions each donor experienced[6].

Neuropathological examination

Methods for brain tissue processing and evaluation have been described previously[1,4]. Neuropathologists were blinded to the donor's exposure history, all clinical history, and medical diagnoses. All pathologic diagnoses were reviewed by four neuropathologists (A.C.M., B.R.H., T.D.S., V.E.A.) and discrepancies were resolved by discussion. Gross and microscopic examination were conducted following previously published methods[62]. Well-established criteria were used for the neuropathologic diagnosis of neurodegenerative diseases, including Alzheimer's disease (NIA-Reagan criteria of high or intermediate likelihood)[63], Lewy body disease[64], frontotemporal lobar degeneration[65,66], and motor neuron disease[67]. CTE was diagnosed using NINDS/NIBIB neuropathological criteria[3]. Donors with CTE were staged (I to IV, in order of increasing severity) based on previously published criteria[4,68], which were later classified as low-stage CTE (stages I and II) and high-stage CTE (stages III and IV). Neuropathologists also recorded semi-quantitative NFT burden on a 0–3 scale with increasing severity across 11 brain regions implicated in CTE: dorsolateral frontal cortex, middle frontal cortex, orbitofrontal cortex, hippocampus regions CA1, CA2, CA3/4, substantia nigra, amygdala, entorhinal cortex, inferior parietal cortex, and locus coeruleus. Cumulative neurofibrillary tangle (NFT) burden was defined as the sum score across these 11 regions.

Helmet sensor study literature review and creation of the positional exposure matrix (PEM)

We created a PEM that quantified RHI features specific to football position and level of play, adapting the template of the job exposure matrix, which is used in the field of occupational health to retrospectively characterize exposure[46]. This involved three steps: (1) identifying published helmet accelerometer studies reporting hits per season, linear acceleration, and rotational acceleration specific to position and level of play; (2) abstracting measures of central tendency (e.g., mean or median hits per season) from each report; and (3) computing summary means of hits per season, linear acceleration, and rotational acceleration, specific to position and level of play, with each study's estimate weighted in proportion to its sample size.

First, data were compiled from previously published helmet accelerometer studies in football players that reported either the number of impacts sustained per season, average linear acceleration sustained each season, and/or average rotational acceleration sustained each season[8–41]. Specifically, a literature review was conducted using PUBMED to identify articles published prior to 2021 with the search terms: "head impact telemetry system," "football helmet accelerometer," "football helmet linear acceleration," and "football helmet rotational acceleration". These articles were reviewed and included in the positional exposure matrix (PEM) if they fit the following criteria:

- Head impacts were measured across *practices and games for the entire season*
- *Level of play* (youth, high-school, college) was identified
- *Mean or median* head impact frequencies, linear acceleration, or rotational acceleration were reported by *position played* (only for high-school and beyond, given that no studies reported results for youth by position played)
- Any impact event with a peak linear acceleration <10 *g* was excluded from analysis. A minimum *cutoff of 10* g *ensures* the elimination of nonimpact events (e.g., jumping) from the calculation of head impact frequency

Based on these criteria, 34 articles were identified.

We next compiled values that were either directly reported in a specific paper or that we derived from the data reported (e.g., a paper might have reported total participants and cumulative hits across all participants; mean frequency of hits per season was derived by dividing cumulative hits per season by number of participants). Values were derived using arithmetic if not directly reported. The contributing studies reported either the mean or median values, both were included in the PEM and treated similarly. When studies grouped multiple positions together in their results (e.g., simply reporting results for "speed" and "non-speed" positions), the aggregate information provided for each group was applied to all positions within that group, for that study. Cumulative head impacts, linear acceleration, and rotational acceleration for a single player across a single season were reported and derived if necessary. Mean or median impact frequencies, linear acceleration, and rotational acceleration across a single season were weighted by each study's sample size. These weighted averages are the impacts experienced per position per season at the different levels of play (youth, high school, college).

Finally, these data were used to develop the PEM. The PEM aggregated the weighted mean annual numbers and intensities (linear and rotational acceleration) of exposures to head impacts across all reported positions and levels of play. For any missing information in the PEM based on lack of available helmet sensor data (e.g., no reports of the rotational acceleration experienced by collegiate defensive backs), the average data for that level across all positions was used (e.g., the mean rotational acceleration across all collegiate positions weighted by study sample size). Because there is currently no helmet sensor data for semi-professional or professional football players, collegiate data from the PEM were used to approximate these levels of play. Table 7 provides a summary of values obtained from key variables in the PEM, with the corresponding data reported in Supplementary Source Data, per PRISMA guidelines[69].

Table 7 | Position exposure matrix of weighted average annual exposures aggregated from previously published helmet sensor studies

	Number of hits per season			Linear acceleration (g)			Rotational acceleration (rad/s^2)		
	Youth	High school	College	Youth	High school	College	Youth	High school	College
Overall	206.4	538.7	526.0	20.6	26.5	20.6	1203.4	1898.4	1574.2
DL		782.3	840.9		25.8	21.0		1801.3	1806
DB		316.6	371.6		28.5	20.2		1957.4	
LB		460.2	539.0		27.3	22.3		1870.4	2071.7
OL		734.4	814.6		25.8	21.0		1777.5	1782
QB		320.2	209.4		26.8	21		1476.4	
RB		475.1	421.7		27.7	21.8		1807.7	1878.8
TE		517.4	599.2		27.1	31.0		1625.8	1815.7
WR		301.9	313.9		28.8	19.5		2223.8	

Source data are provided as a Source Data file
DL defensive line, *DB* defensive back, *LB* linebacker, *OL* offensive line, *QB* quarterback, *RB* running back, *TE* tight end, *WR* wide receiver

Cumulative head impact indices (CHIIs)

The PEM was used to calculate three indices of exposure to head impacts over each athlete's lifetime. The indices, which correspond with the three head impact features in the PEM, were the estimated number of the impacts (CHII), the estimated total linear acceleration sustained (CHII-G), and the estimated total rotational acceleration sustained (CHII-R). For each athlete, CHII was calculated:

$$\mathrm{CHII} = \sum_{n=1}^{y} h_n \tag{1}$$

where y is the number of years the athlete played football and h is the number of head impacts an average athlete is exposed to annually according to the PEM, given the position and level that the athlete played, during year n. For example, an athlete who played one year of football in high school as a linebacker, one year of football in high school as a defensive lineman, and one year in college as a defensive lineman, would have (numbers from PEM in Table 7):

$$\begin{aligned}\mathrm{CHII} &= \sum_{n=1}^{y} h_n = h_1 + h_2 + h_3 \\ &= (\textit{mean weighted annual frequency of high school linebacker hits}) \\ &+ (\textit{mean weighted annual frequency of high school defensive lineman hits}) \\ &+ (\textit{mean weighted annual frequency of college defensive lineman hits}) \\ &= 460.2 + 782.3 + 840.9 = 2{,}083.4 \text{ estimated lifetime cumulative hits}\end{aligned} \tag{2}$$

Similarly, CHII-G was calculated:

$$\mathrm{CHII-G} = \sum_{n=1}^{y} h_n g_n \tag{3}$$

where g is the linear acceleration (measured in g-force) that an average athlete is exposed to annually according to the PEM, given the position and level that the athlete played, during year n. For the above example athlete:

$$\begin{aligned}\mathrm{CHII-G} &= \sum_{n=1}^{y} h_n g_n = h_1 g_1 + h_2 g_2 + h_3 g_3 \\ &= h_1 \times (\textit{mean weighted annual g-force of high school linebacker hits}) + h_2 \\ &\times (\textit{mean weighted annual g-force of high school defensive lineman hits}) + h_3 \\ &\times (\textit{mean weighted annual g-force of college defensive lineman hits}) \\ &= 460.2 \times (27.3) + 782.3 \times (25.8) + 840.9 \times (21.0) \\ &= 50{,}405.7 \text{ estimated lifetime cumulative g-force}\end{aligned} \tag{4}$$

Similarly, CHII-R was calculated:

$$\mathrm{CHII-R} = \sum_{n=1}^{y} h_n r_n \tag{5}$$

where r is the rotational acceleration (measured in rad/sec^2) that an average athlete is exposed to annually, according to the PEM, given the position and level that the athlete played, during year n. For the above example athlete:

$$\begin{aligned}\mathrm{CHII-R} &= \sum_{n=1}^{y} h_n r_n = h_1 r_1 + h_2 r_2 + h_3 r_3 \\ &= h_1 \times (\textit{mean weighted annual rad/sec2 of high school linebacker hits}) + h_2 \\ &\times (\textit{mean weighted annual rad/sec2 of high school defensive lineman hits}) + h_3 \\ &\times (\textit{mean weighted annual rad/sec2 of college defensive lineman hits}) \\ &= 460.2 \times (1870.4) + 782.3 \times (1801.3) + 840.9 \times (1806) \\ &= 3{,}788{,}580.5 \text{ estimated lifetime cumulative rotational acceleration}\end{aligned} \tag{6}$$

For athletes who played multiple positions during a given year, the respective exposure index for that year was the mean exposure index of the multiple positions. Previous iterations of the CHII asked athletes to estimate the percent of time that they played at any positions they played within a given season. However, in pilot testing, when informants were asked to estimate the percentage of time that their loved ones spent playing at a given position, informants consistently reported that they that they did not know. As such, the questions asking percent time played at each level were not incorporated into the present study.

Statistical methods

Separate logistic regression models were fitted to determine the association between each exposure measure (concussion number, position at highest level, duration of football play, CHII, CHII-G, and CHII-R) and CTE status (absent or present). Among those with CTE, we fitted parallel models using CTE severity (low or high) as the outcome. Because we sought information about individual-level prediction, receiver operating characteristics (ROC) curves were plotted for all significant exposure measures ($p < 0.05$) to observe the relationship between each exposure measure and both dichotomous CTE outcomes. The bootstrap approach was used to evaluate differences in discriminative power between models[70]. To further evaluate predictive ability of each model, a 10-fold cross-validation study was performed

for each significant exposure measure and both dichotomous CTE outcomes.

We fitted linear regressions to quantify the association of each exposure measure with NFT burden, a semi quantitative measure consisting of a zero to three score for each region, summed across 11 brain regions (total range: 0–33). These analyses were restricted to data from athletes with all 11 brain regions available for neuropathological evaluation (82.1%). Imputation was not performed for missing brain region data because we were sufficiently powered without imputation. Additionally, missingness was not associated with exposure measures after adjusting for age at death, so estimated effects were unlikely to be biased by differential missingness.

All models were adjusted for age at death given its known association with CTE pathology. For all models, Bayesian information criterion (BIC) was calculated to aid in comparison of model fits. To adjust for multiple analyses, the Bonferroni-adjusted significance level of 0.0083 was used. All data were collected and secured using Boston University Medical Center REDCap. All analyses were performed with R v4.0.5 or SPSS v.27.0.1.0. Given the high-profile and sensitive information for these donors, access to data is strictly monitored to ensure confidentiality. De-identified biospecimens and limited dataset clinical are available upon submission of IRB approved proposal and data use agreement. Please contact corresponding author to initiate this process. All code is available upon request to the corresponding author.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

The raw datasets generated during and/or analyzed during the current study are not publicly available due to potential ability to identify elite athletes based on exposure data. However, these data are available from the corresponding author on request and with relevant IRB approval. Source data are provided with this paper.

Code availability

All code used for the present project, as well as a sample dataset, has been provided and is available as the supplementary code file.

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Author contributions

All authors have agreed both to be personally accountable for their own contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which that author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature. D.H.D., J.W., Y.T., M.D.M., M.L.A., A.C.M., and J.M. made substantial contributions to the conception and design of the work as well as the acquisition, analysis, and interpretation of data, and have approved the submitted version. Z.H.B., J.J., C.M.B., B.M.M., J.N.P., C.J.N., R.C.C., R.D.Z., B.D., J.F.C., L.E.G., N.W.K., D.I.K., and R.A.S. have made substantial contributions to the analysis and interpretation of data and have approved the submitted version. E.S.N., A.R., B.A., M.U., N.S., A.S., J.D.C., V.E.A., B.R.H., and T.D.S. have made substantial contributions to the acquisition, analysis, and interpretation of data and have approved the submitted version.

Competing interests

D.H.D. serves as an expert witness in legal cases involving brain injury and concussion and serves as an advisor and options holder for StataDx outside the submitted work. C.J.N. is the cofounder and chief executive officer of the Concussion Legacy Foundation; reported nonfinancial support (travel reimbursement) from the NFL Players Association as a member of the Mackey-White Health & Safety Committee, WWE, and AEW (All Elite Wrestling); and serves as an advisor and options holder for Oxeia Biopharmaceuticals, PreCon Health, and StataDx outside the submitted work. R.C.C. reported royalties from Houghton Mifflin Harcourt; compensation for expert legal opinion to the National Collegiate Athletic Association and National Hockey League; consults for the Concussion Legacy Foundation; is senior advisor and paid consultant to the NFL Head Neck & Spine Committee; is a member of the Mackey-White Committee of the National Football League Players Association; is vice president of National Operating Committee on Standards for Athletic Equipment and chair scientific advisory committee and cofounder of Medical Director Concussion Legacy Foundation; and is on the Medical Science Committee for the National Collegiate Athletic Association Student-Athlete Concussion Injury Litigation. R.D.Z. receives royalties from Oakstone for an educational CD (Physical Medicine and Rehabilitation: A Comprehensive Review) and from demosMedical, part of Springer Publishing, for serving as co-editor of the text Brain Injury Medicine and serves on the Scientific Advisory Board of Oxeia Biopharma, BioDirection, ElMINDA, and Myomo. He is also PI on a grant entitled the Football Players Health Study at Harvard University, which is funded by the National Football League Players Association (NFLPA) and evaluates patients for the MGH Brain and Body TRUST Center, sponsored in part by the NFLPA, and serves on the Mackey-White Health and Safety Committee. L.E.G. is a paid consultant to Johnson & Johnson, Janssen Research & Development LLC, and Rebiscan Inc and has received funding from the WWE and Ivivi Health Sciences. D.I.K. reported royalties from Springer/Demos Publishing for a textbook on brain injury; serves as an expert witness in legal cases involving brain injury and concussion; receives a stipend from Encompass Health as program medical director for brain injury and chair of the annual Neurorehabilitation conference; and has received honoraria for a keynote address for the HealthSouth Annual Medical Directors Meeting. R.A.S. reported personal fees from Biogen and Lundbeck outside the submitted work; member of the Mackey-White Committee of the National Football League Players Association; receives royalties for published neuropsychological tests from Psychological Assessment Resources Inc; and is a member of the Board of Directors of King-Devick Technologies. M.L.A. receives royalties from Oxford University Press for a textbook outside the submitted work. A.C.M. is a member of the Mackey-White Committee of the National Football League Players Association and reports other funding from Buoniconti Foundation during the conduct of the study. No other authors have competing interests as defined by Nature Portfolio, or other interests that might be perceived to influence the results and/or discussion reported in this paper.

Additional information

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