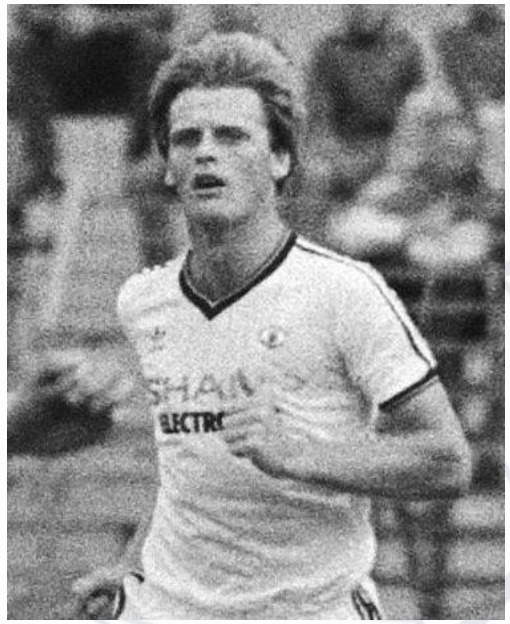


Traumatic brain injury in sport

Craig Murray KC
Professor Willie Stewart

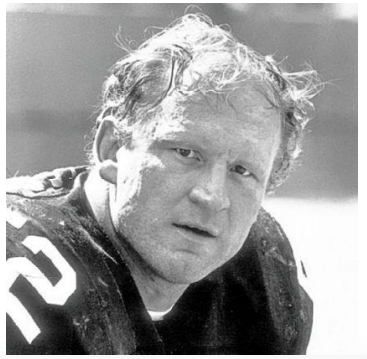
20th November 2026

Inquest touching the death of Gordon McQueen



Punch drunk syndrome / dementia pugilistica / chronic traumatic encephalopathy (CTE)





SPECIAL REPORT

CHRONIC TRAUMATIC ENCEPHALOPATHY IN A NATIONAL FOOTBALL LEAGUE PLAYER

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OBJECTIVE: We present the results of the autopsy of a retired professional football player that revealed neuropathological changes consistent with long-term repetitive concussive brain injury. This case draws attention to the need for further studies in the cohort of retired National Football League players to elucidate the neuropathological sequelae of repeated mild traumatic brain injury in professional football.

METHODS: The patient's premortem medical history included symptoms of cognitive impairment, a mood disorder, and parkinsonian symptoms. There was no family history of Alzheimer's disease or any other head trauma outside football. A complete autopsy with a comprehensive neuropathological examination was performed on the retired National Football League player approximately 12 years after retirement. He

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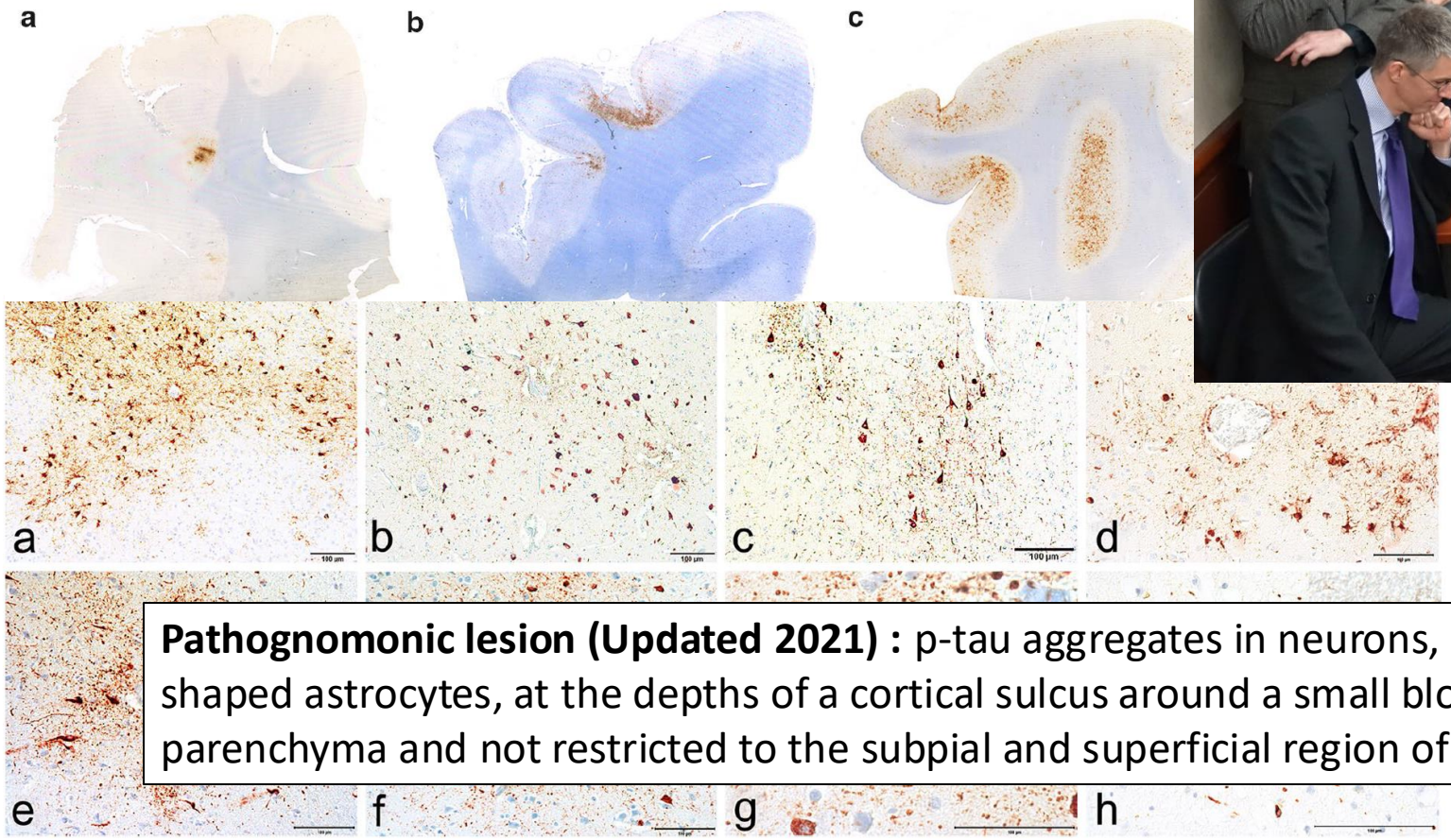
CrossMark

CONSENSUS PAPER

The first NINDS/NIBIB consensus meeting to define neuropathological criteria for the diagnosis of chronic traumatic encephalopathy

Ann C. McKee[1,2,3,4,5] · Nigel J. Cairns[6] · Dennis W. Dickson[7] · Rebecca D. Folkerth[8] · C. Dirk Keene[9] · Irene Litvan[10] · Daniel P. Perl[11] · Thor D. Stein[2,3,4,5] · Jean-Paul Vonsattel[12] · William Stewart[13] · Yorghos Tripodis[3,14] · John F. Crary[15] · Kevin F. Bieniek[7] · Kristen Dams-O'Connor[16] · Victor E. Alvarez[1,2,3,4] · Wayne A. Gordon[16] · the TBI/CTE group





Pathognomonic lesion (Updated 2021) : p-tau aggregates in neurons, with or without thorn-shaped astrocytes, at the depths of a cortical sulcus around a small blood vessel, deep in the parenchyma and not restricted to the subpial and superficial region of the sulcus

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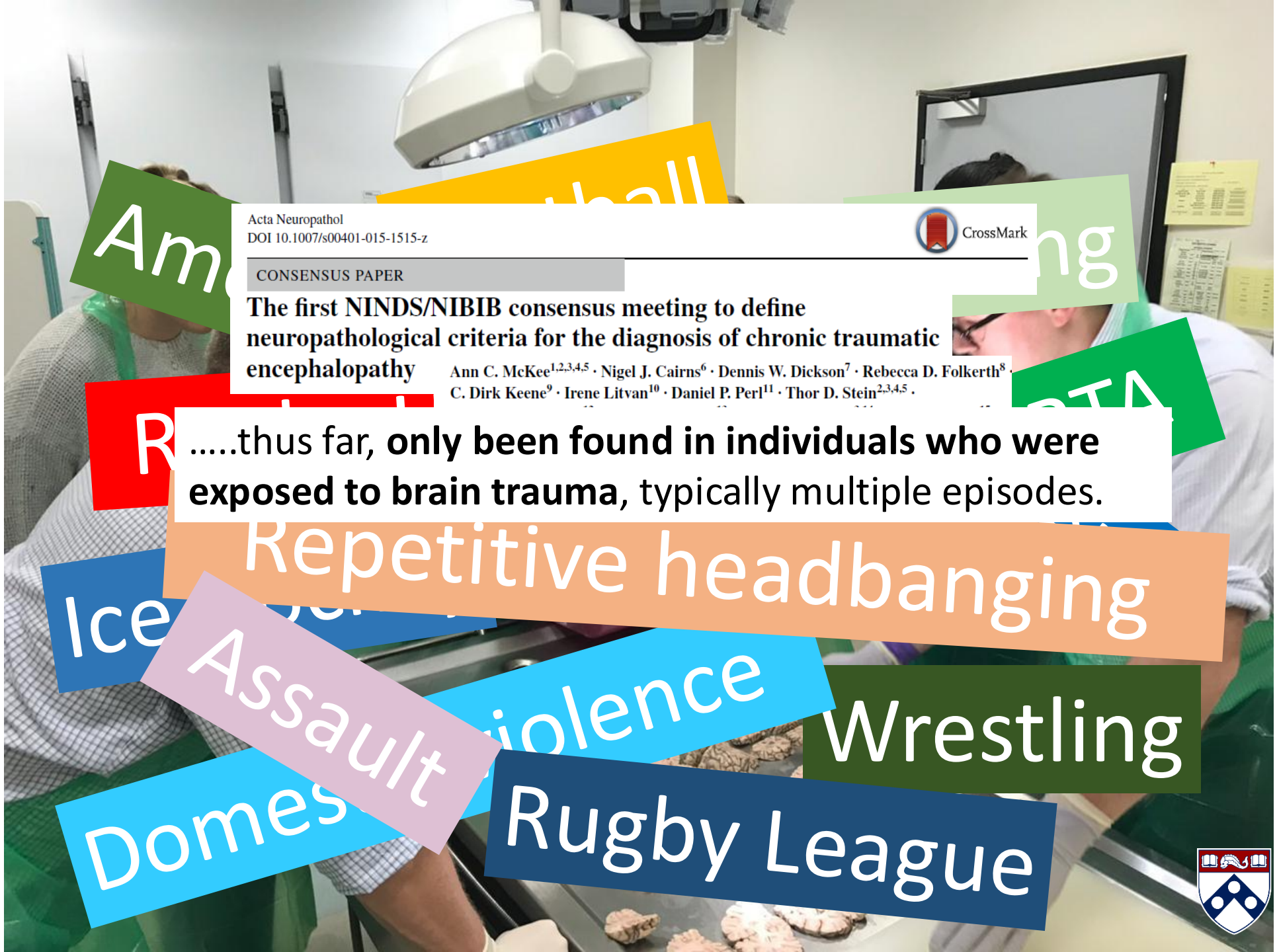

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Acta Neuropathologica
https://doi.org/10.1007/s00401-023-02576-y

CORRESPONDENCE

Check for updates

The perils of contact sport: pathologies of diffuse brain swelling and chronic traumatic encephalopathy neuropathologic change in a 23-year-old rugby union player

Edward B. Lee[1] · Claire Kennedy-Dietrich[2] · Jennian F. Geddes[3] · James A. R. Nicoll[4] · Tamas Revesz[5] · Douglas H. Smith[6] · William Stewart[2,7]

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- **23 year old** elite rugby union
- Tackle, brief loss of consciousness; **"concussion"**
- Played on, collapsed
- Transfer hospital
- Died ~24h later

- Very early stage CTE-NC

Acta Neuropathologica
https://doi.org/10.1007/s00401-023-02644-3

CORRESPONDENCE

Check for updates

Risk of chronic traumatic encephalopathy in rugby union is associated with length of playing career

William Stewart[1,2] · Michael E. Buckland[3,4] · Bobak Abdolmohammadi[5] · Andrew J. Affleck[3,4] · Victor E. Alvarez[5,6,7,8] · Shannon Gilchrist[1] · Bertrand R. Huber[5,6,7,9,10] · Edward B. Lee[11] · Donald M. Lyall[12] · Christopher J. Nowinski[5,13] · Emma R. Russell[1] · Thor D. Stein[5,6,8,9] · Catherine M. Suter[3,4] · Ann C. McKee[5,6,7,8,9,10,14]

- 31 former rugby players
- 21 with CTE-NC; 10 no CTE-NC
- **~14% increase in CTE risk/ year played**

Localised pathology first detectable while still participating

Longer individual exposed to sport, greater risk of pathology

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Understanding repetitive head trauma in sport



Pathology

Multiple contact/ combat sports
- **specific pathology** identified at post-mortem, CTE-NC
  - thus far, only found where history **brain injury/ repetitive impacts**
  - **Increased risk with longer careers (exposure)**
- In addition, complex, **mixed pathologies of TReND**



This is fascinating, but you're describing brain pathology that might mean nothing at all.

Is there any evidence former contact sports athletes have altered risk of neurodegenerative disease?

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The NEW ENGLAND JOURNAL of MEDICINE

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Neurodegenerative Disease Mortality among Former Professional Soccer Players

Daniel F. Mackay, Ph.D., Emma R. Russell, M.Sc., Katy Stewart, Ph.D., John A. MacLean, M.B., Ch.B., Jill P. Pell, M.D., and William Stewart, M.B., Ch.B., Ph.D.

- 7676 former pro soccer v 23,028 matched controls
- Death certification data
- Risk of death due to **neurodegenerative disease ~3.5x higher than expected**
- Risk varies by disease subtype:
  - **Alzheimer's disease ~5x higher**
  - **Motor neuron disease ~4x higher**
  - **Parkinson's disease ~2x higher**

Table 2. Primary Cause of Death among Former Soccer Players and Matched Controls.

Primary Cause of Death	Former Soccer Players (N=7676)	Matched Controls (N=23,028)	Hazard Ratio for Death (95% CI)	P Value*
	number (percent)			
Any cause†	1180 (15.4)	3807 (16.5)	0.87 (0.80–0.93)	<0.001
Ischemic heart disease	173 (2.3)	568 (2.5)	0.80 (0.66–0.97)	0.02
Lung cancer	74 (1.0)	362 (1.6)	0.53 (0.40–0.70)	<0.001
Neurodegenerative disease	134 (1.7)	120 (0.5)	4.10 (2.88–5.83)‡	<0.001

‡ In an analysis that included adjustment for competing risks of death from ischemic heart disease and death from cancer, the subhazard ratio was 3.45 (95% CI, 2.11 to 5.62) (P<0.001).

Table 3. Mortality with Neurodegenerative Disease Listed as the Primary or a Contributory Cause.

Primary or Contributory Cause of Death	Former Soccer Players (N=7676)	Matched Controls (N=23,028)	Hazard Ratio for Death (95% CI)	P Value*
	number (percent)			
Any neurodegenerative disease	222 (2.9)	228 (1.0)	3.53 (2.72–4.57)	<0.001
Neurodegenerative disease subtype				
Dementia not otherwise specified	180 (2.3)	178 (0.8)	3.87 (2.86–5.24)	<0.001
Alzheimer's disease	64 (0.8)	47 (0.2)	5.07 (2.92–8.82)	<0.001
Non-Alzheimer's dementias	121 (1.6)	133 (0.6)	3.48 (2.42–5.00)	<0.001
Motor neuron disease	22 (0.3)	17 (0.1)	4.33 (2.05–9.15)	<0.001
Parkinson's disease	28 (0.4)	44 (0.2)	2.15 (1.17–3.96)	0.01

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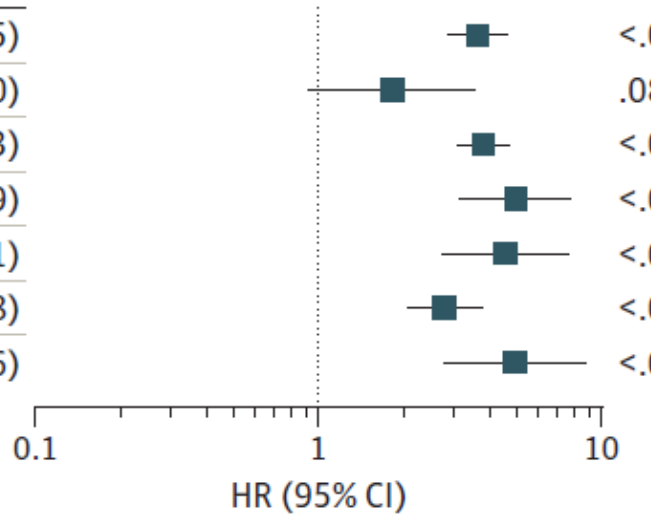
JAMA Neurology | Original Investigation

Association of Field Position and Career Length With Risk of Neurodegenerative Disease in Male Former Professional Soccer Players

Emma R. Russell, MSc; Daniel F. Mackay, PhD; Katy Stewart, PhD; John A. MacLean, MBChB; Jill P. Pell, MD; William Stewart, MBChB, PhD

Figure 1. Player Position and Risk of Neurodegenerative Disease Among Male Former Professional Soccer Players Compared With a Matched General Population Control Group

Source	HR (95% CI)	*P* value
All players	3.66 (2.88-4.65)	<.001
Goalkeeper	1.83 (0.93-3.60)	.08
Outfield	3.83 (3.11-4.73)	<.001
Defender	4.98 (3.18-7.79)	<.001
Midfield	4.59 (2.73-7.71)	<.001
Forward	2.79 (2.06-3.78)	<.001
Multiposition	4.94 (2.79-8.76)	<.001



Risk of neurodegenerative disease diagnosis (death/ hospital attendance/ prescribing:

- varies by player position
  - Goalkeepers, around same risk as non-footballers
  - Outfield, around 4x higher
    - **Defenders, around 5x higher**
    - Forwards, around 3x higher
- **Increases with longer playing careers**
- Unchanged for players born 1910-1969 (playing to around 2000)

JAMA Network | Open

Original Investigation | Neurology

Health and Lifestyle Factors and Dementia Risk Among Former Professional Soccer Players

Emma R. Russell, PhD; Donald M. Lyall, PhD; Daniel F. Mackay, PhD; Kirstie Cronin, MSci; Katy Stewart, PhD; John A. MacLean, MBChB; Jill P. Pell, MD; William Stewart, MBChB, PhD

FIELD:LONG

Former professional footballers show **same, if not lower risk wider dementia risk factors** ie better overall physical and mental health

Table 2. Comparison of Rates of Dementia and Potentially Modifiable Risk Factors Between Former Soccer Players and Matched Controls

Risk factor	Participants, No. (%)		HR (95% CI)	*P* value[a]
	Former soccer players (n = 11 984)	Matched controls (n = 35 952)		
Dementia	434 (3.62)	453 (1.26)	3.02 (2.54-3.58)	<.001
Hypertension	884 (7.38)	2678 (7.45)	0.97 (0.88-1.06)	.51
Diabetes	511 (4.26)	2283 (6.35)	0.75 (0.67-0.85)	<.001
ARD	482 (4.02)	1778 (4.95)	0.77 (0.69-0.87)	<.001
Smoking	170 (1.42)	910 (2.53)	0.55 (0.46-0.66)	<.001
Depression	138 (1.15)	513 (1.43)	0.85 (0.69-1.04)	.12
Hearing loss	131 (1.09)	369 (1.03)	1.05 (0.85-1.28)	.66
Obesity	128 (1.07)	533 (1.48)	0.72 (0.59-0.87)	.001

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Understanding repetitive head trauma in sport





Pathology

Population

Multiple contact/ combat sports
- **specific pathology** identified at post-mortem, CTE-NC
  - thus far, only found where history **brain injury/ repetitive impacts**
  - **Increased risk with longer careers (exposure)**
- In addition, complex, **mixed pathologies of TReND**

Football/ rugby (and others)
- neurodegenerative disease risk **higher than expected**
- risk varies by:
  - **Disease subtype**
  - **Player position**
  - **Career length**
- **Wider health very much better in former athletes** (inc wider dementia risk factors)



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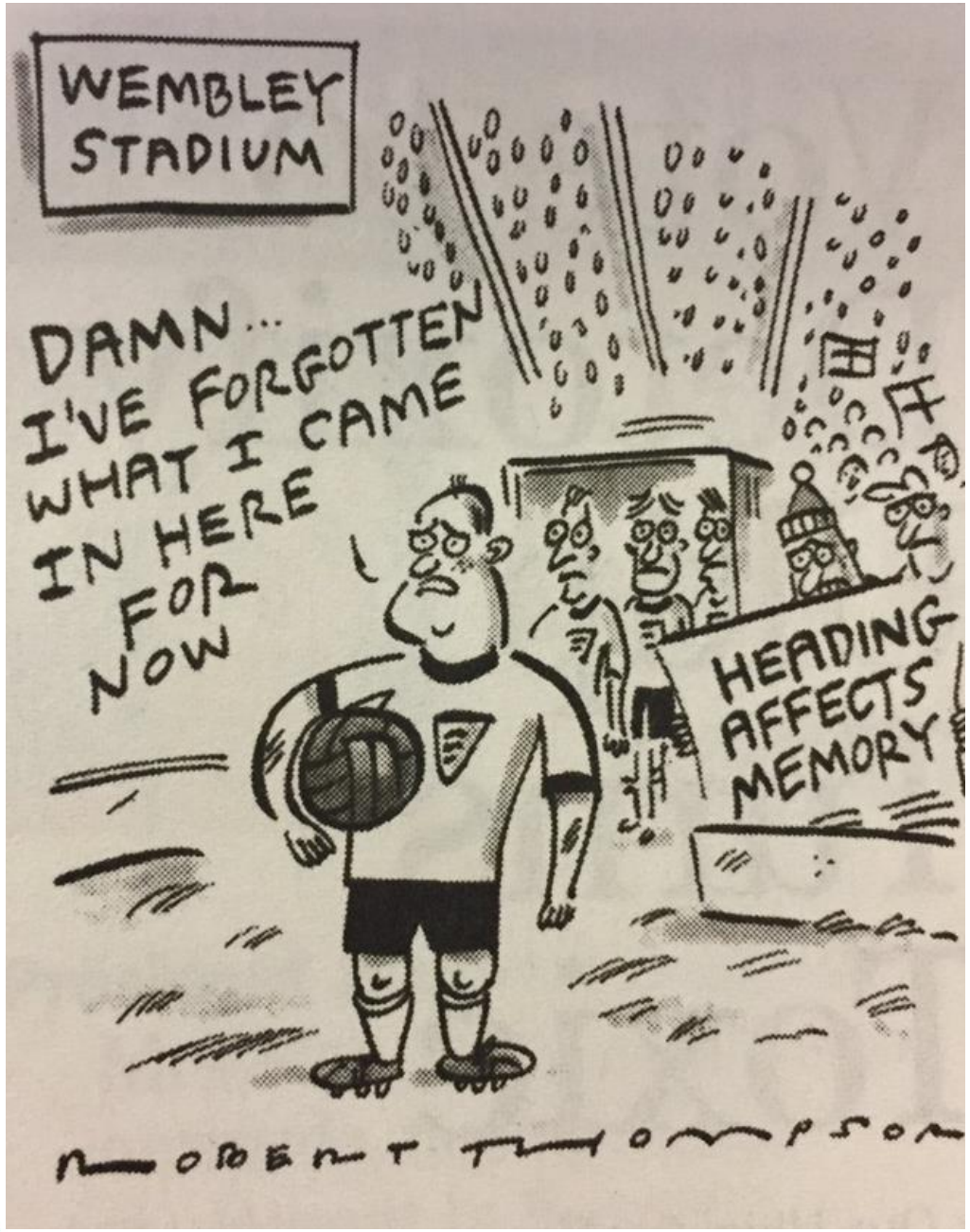
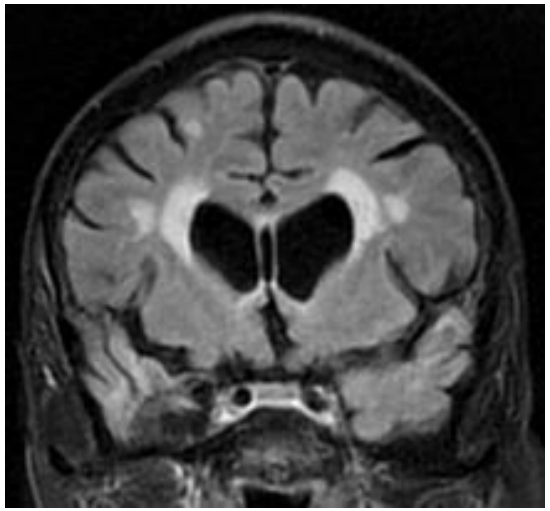


Of course, repeated knee injury leads to degenerative joint disease.





Why is it so hard to get recognition that repeated TBI/ head impacts lead to degenerative brain disease?

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Understanding repetitive head trauma in sport

Is there an association between professional contact sport and neurodegenerative disease?

Yes, convincingly demonstrated in several cohorts.

Is the association likely to be causal?

Yes, particularly for repetitive head impacts and CTE-NC.

What about Alzheimer's, Parkinson's and MND specifically?

Less clear evidence, but very likely misclassified AD, PD etc in life and lack of autopsy confirmation confound data. MND is higher in elite sport, but may not be TBI/RHI related

Have alternative health, lifestyle and genetic explanations been adequately considered?

Substantially, but not entirely

Is current evidence sufficient to justify reducing avoidable head-impact exposure?

Unquestionably

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Introduction to CTE Litigation

Czernuska v. King [2023] EWHC 380

- "So far as the legal test is concerned, I endorse Mr Weir KC's basic proposition that, within the law of negligence, the test is whether the Defendant failed to exercise such degree of care as was appropriate in all the circumstances: this was the test endorsed in *Condon v Basi* [1985] 1 WLR 866 where the Court of Appeal adopted the formulation and approach of Kitto J in Rootes v Shelton."

- Rootes v. Shelton, 1968 Australia: "Non-compliance with ['the rules of the game'], conventions or customs (where they exist) is necessarily one consideration to be attended to upon the question of reasonableness; but it is only one, and it may be of much or little or even no weight in the circumstances."

NFL Settlements

- 1994 committee
- Death of Mike Webster
- PM by Bennet Omalu
- Boston CTE centre
- $765 settlement in 2013

Inquests

- A Coroner's investigation is not (and is not intended to be) "an exhaustive forensic inquiry of the kind that may be necessary in adversarial litigation" (*R (Wandsworth Borough Council) v. HM Senior Coroner for Inner West London* [2021] EWHC 801, per HHJ Teague QC at [29]).

- The test for causation of death was explained in the *Wandsworth* judgment as: "that the event or conduct said to have caused the death must have more than minimally, negligibly or trivially contributed to it." (at [32]).

- The relevant event "must make an actual and material contribution to the death of the deceased" (*R (Tainton) v. HM Senior Coroner for Preston and West Lancashire* [2016] EWHC 1396 (Admin), at [62]).

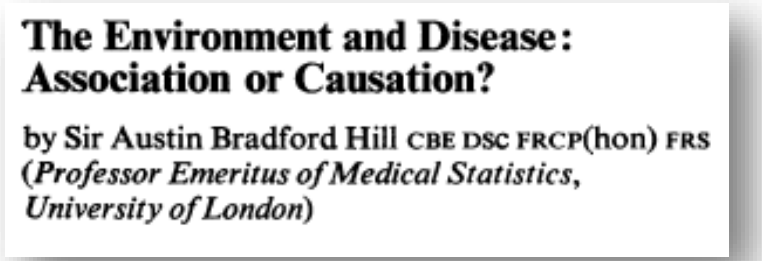
Watson v. Secretary of State for Work and Pensions [2025] UKUT 365

Hughes v.World Rugby [2026] EWHC 2149

Legal Causation

- Bradford Hill Criteria
  - strength of association,
  - consistency,
  - specificity,
  - temporality, and
  - biological gradient.

- *Simmons v. British Steel* 2004 SC (UKSC) 94



The Environment and Disease: Association or Causation?

by Sir Austin Bradford Hill CBE DSC FRCP(hon) FRS
(*Professor Emeritus of Medical Statistics, University of London*)

Considerations regarding evidence of causality. **Not** a checklist

- **Strength of Association:** NDD overall, 3.5x. CTE-NC, considerably higher; typical threshold accepted as 2-fold
- **Consistency:** FIELD data replicated across populations and sports; CTE-NC in multiple populations, all RHI/TBI exposed
- **Specificity:** Multiple dementia risks, only one for CTE-NC; general health better ie NDD outcome against trend (specific)
- **Temporality:** exposure many decades before outcome
- **Biological gradient:** longer careers or higher exposure positions = higher risk
- **Plausibility and Coherence:** underlying pathology of injury and subacute phases consistent with adverse brain health
- **Experiment**: animal models replicate some of observations; exclusion studies (reduce heading etc) by default.
- **Analogy:** Established consequences of boxing

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Discussion