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Cite this as: *BMJ* 2026;394:e100526
http://doi.org/10.1136/bmj-2026-100526

Chronic traumatic encephalopathy in former NFL players

New estimates strengthen evidence of CTE burden in former NFL players, while highlighting uncertainties

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Repetitive head impacts are common in contact and collision sports, and the potential long term effects of these on brain health have become an important public health concern.[1 2] Evidence from a cohort of nearly 20 000 former National Football League (NFL) players found neurodegenerative related mortality to be almost four times higher than expected in the general population, despite substantially lower overall mortality.[3] In the linked study, Daneshvar and colleagues (doi:10.1136/bmj-2026-100418) extend these observations by estimating the possible burden of chronic traumatic encephalopathy (CTE) among deceased former NFL players and by examining the association between advanced CTE and dementia.[4] Together, these findings help bridge epidemiological observations with neuropathological evidence.

Epidemiological studies showing increased neurodegenerative related mortality cannot determine the neuropathology underlying these deaths. CTE is a distinct neuropathological diagnosis associated with exposure to repetitive head impacts that currently can only be confirmed after death.[5 6] Previous brain bank studies have reported a high proportion of CTE among former contact sport athletes, including 110 of the first 111 former NFL players examined at Boston University's UNITE Brain Bank.[7] However, because brain donation is non-random and may be more likely when players had neurological symptoms during life, the proportions observed in this sample of NFL players cannot be directly interpreted as the prevalence of CTE among all former players.[8]

Daneshvar and colleagues recognise this limitation, and their work advances the evidence base in several important ways. Rather than reporting only the prevalence of CTE among brain donors, they identified a well defined source population comprising all former NFL players who died between 2008 and 2021 and linked this population with data from brain banks. This allowed the investigators to estimate plausible lower and upper bounds for CTE prevalence at death in all NFL players. They also combined neuropathological findings with clinician adjudicated dementia and death certificate data, thereby examining both the disease burden and its clinical relevance. At least 18.5% of players who died during the full study period, and 24.5% of those who died during 2016-21, had confirmed CTE; among donors, stage IV disease was associated with dementia.[4]

The study also illustrates why estimating the burden of CTE remains challenging. The source population is well defined through previous NFL participation, but ascertainment of CTE is conditional on two later events: death during the study period and brain donation for neuropathological examination. Conditioning CTE ascertainment on death and brain donation may result in selection bias because the donors may differ systematically from players whose brains were not examined. Although the authors used inverse probability weighting to account for measured reasons for donation, decisions to donate may also depend on unmeasured clinical, family, and societal factors, including cognitive or behavioural changes during life and awareness of CTE. Therefore, the reported prevalence range should be interpreted as plausible bounds under the assumptions made, rather than a precise estimate of the actual prevalence among former players. Furthermore, the observed association between stage IV CTE and dementia should be recognised as an association within the selected donor population rather than an unbiased estimate for all former NFL players.[4]

The clinical interpretation is also more complex than attributing dementia to a single neuropathological process. Coexisting neurodegenerative and vascular changes to the brain were common among donors, as is typical in dementia at older ages.[9 10] Nevertheless, dementia was present in 90% of donors with stage IV CTE, and advanced disease remained associated with dementia after adjustment for measured characteristics.[4] The study also highlights the limitations of mortality records: fewer than half of donors with clinician adjudicated dementia had a neurodegenerative condition recorded on their death certificate. This finding is consistent with previous studies showing substantial under-recording of dementia on death certificates,[11 12] and suggests that mortality studies may underestimate the broader clinical burden of neurodegenerative disease among former players.

No single study is likely to fully define the epidemiology of CTE or the contribution of playing in the NFL to the development of CTE. However, by combining a well defined source population with postmortem confirmation of CTE, Daneshvar and colleagues provide one of the most informative estimates to date of CTE burden among former NFL players. The next challenge is not simply to establish whether repetitive head impacts have long term neurological effects, but to quantify those consequences well enough to inform athletes, clinicians, sporting organisations, and policymakers. This information will require prospective, real time recruitment and follow-up of players from the onset of their careers, ideally starting from high school or college football teams, and potentially matched to a control group of non-football players with similar characteristics. Alongside this, there needs to be

improved measurement of cumulative head impacts, new approaches to identifying CTE during life, longitudinal measurement of potential time varying confounders, and continued efforts to reduce avoidable exposure while uncertainty remains. Above all, it is essential to regularly communicate the existing and emerging evidence on this issue to past, present, and prospective NFL players; any consent to play should be obtained with knowledge of the potential risks involved and the uncertainty and limitations of the current research evidence.

Competing interests: The BMJ has judged that there are no disqualifying financial ties to commercial companies. The authors declare the following other interests: TK and RDR are statistical editors for *The BMJ*. TK reports having received research grants from the German Federal Joint Committee and personal compensation from the North-East German Society for Gynaecological Oncology, AbbVie, BMJ Group, and Frontiers Media SA. RDR reports compensation from the BMJ Group and royalties for his textbooks on prognosis research in healthcare and individual participant data meta-analysis. RDR is supported by the National Institute for Health and Care Research (NIHR) Biomedical Research Centre: Birmingham, and is also an NIHR senior investigator. The views expressed are those of the authors and not necessarily those of the NHS, NIHR, or Department of Health and Social Care.

AI use: We used ChatGPT (OpenAI) during preparation of this work to improve clarity and wording of the text. The authors reviewed and edited all AI assisted content and take full responsibility for the final version.

Provenance and peer review: Commissioned; not externally peer reviewed.

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