Dr Rowena Mobbs MBBS PhD FRACP
Neurologist and Senior Lecturer
Macquarie Medical School
3 Technology Place
Macquarie University
NSW 2109

27 January 2023

Committee Secretary
Concussions and Repeated Head Trauma in Contact Sports
Senate Standing Committees on Community Affairs
PO Box 6100
Parliament House
Canberra ACT 2600

Dear Chair,

I welcome the Australian Federal Government's Inquiry into sports-related concussion and its long-term ramifications, including chronic traumatic encephalopathy or CTE. When we think of dementia, we often think of those in older age, but I care for athletes in their 30s, 40s and 50s who, due to expected neurological injury, struggle to work, struggle to parent, and struggle to maintain their marriages. Their families, including young children, are exposed to the behavioural ravages of CTE, to impulsivity, rage and violence. Some are now entering residential care, and CTE inflicts a prolonged period of suffering over 15, 20 or 25 years until death. Many go without a diagnosis or specialist care. Some have entered into suicide.

But, we need sport. Having represented Australia in two sports, I emphasise that sport leads to a longer life span through its vascular and social benefits. Neurologically, however, sport can lead to CTE as early as 25 years old, as in the case of one American bull rider[1]. I make this submission on behalf of my 180 patients of concern, many of whom have experienced over 100 concussions and thousands of subconcussions.

In 1994, almost 30 years ago, the National Health and Medical Research Council (NHMRC) reported that boxing and football-related head injuries can be associated with long-term neurological effects. Yet, in my opinion, our community has turned a blind eye to systematic concussion. The absence of mandatory reporting on concussion, neurological care after concussion, and stories of returning to the field too early are harrowing. Furthermore, the dearth of meaningful, fully independent, and appropriately funded research has represented a dark chapter in Australian sport.

I submit the following five recommendations for your consideration:

1. The Federal Government instigates revised and regularly reviewed guidelines on head and neck injury in sport.
2. The Federal Government funds longitudinal research on patients with existing symptoms of CTE, extending to postmortem neuropathological confirmation.

3. The Federal Government mandates that sporting organisations must adhere to a Code of Conduct to:
    a. Implement a public register of suspected and confirmed player concussions.
    b. Monitor the total duration of exposure to head contact (akin to radiation monitoring).
    c. Annually review the register and amend protocols and game rules accordingly.
    d. Fund players to attend an independent neurological assessment after a concussion.
    e. Establish subspecialist concussion and CTE clinics for athletes most at risk of CTE; reasonably those entering university or professional level or accumulating more than 10 years of contact sport; for neurological baseline followed by 5-yearly volumetric MRI brain and review.
    f. Require players, coaches, administrators, and club doctors to undertake annual formal education on concussion.
4. The Federal Government periodically reviews the success of the Code of Conduct approach and, if unsuccessful, works with the State Government to enact Work Health and Safety laws pertaining to sporting associations.
5. The Federal Government undertakes public awareness campaigns informing young children about their brains, older children about brain health for life, and adults about detecting concussion and CTE.

The Federal Government is to be commended for its concern regarding concussion and CTE in sport, and I hope this submission assists the committee with its deliberations.

Yours sincerely,

Dr Rowena Mobbs

Background

Presently, I am a consultant neurologist, MBBS BMedSci(Hons1) PhD FRACP and hold an associate clinical lecturer position at Macquarie University Hospital with a combined clinical and academic program. I work within the private system as a subspecialist cognitive neurologist at Macquarie Neurology and as Director of my private clinic, Harbour Neurology Group. I diagnose and manage all manner of cognitive issues, such as dementia, stroke, epilepsy, encephalitis, concussion and migraine, assisting over 200 patients with cognitive disorders annually. I have experience in managing a cohort of 180 patients with suspected traumatic encephalopathy syndrome (TES), 52 confirmed with probable TES on cognitive testing. TES is the in-life syndrome of CTE. This is arguably Australia's largest clinical cohort, as I am unaware of any other medical research program with double figures, let alone triple figures, of patients with CTE symptoms. I see a range of patient presentations of mild traumatic brain injury (mTBI) from acute concussion, postconcussion syndrome, postconcussion headache through to CTE. I perform research into traumatic brain injury and was previously Clinical Co-Director of the Australian Sports Brain Bank (ASBB), established in 2018, and now Director of the Australian CTE Biobank (ACB), Macquarie University, established in 2022. I have been a qualified medical practitioner since 2005, a neurologist since 2015 as a fellow of the Royal Australasian College of Physicians, and am a member of the Australian and New Zealand Association of Neurologists (ANZAN). In 2011, I obtained a PhD in spinal injury and neurodegeneration.

Initially, I was appointed by A/Prof Michael Buckland as Clinical Co-Director of the ASBB, having attended the launch and through my unpaid affiliation with the University of Sydney. I have seen CTE patients from this time. I sought to establish a clinical program of research linked to the neuropathology program. In 2019 I was appointed Conjoint Senior Lecturer with Macquarie University to establish a concussion and CTE clinical research program. Although my appointment with the ASBB was retracted, I worked towards continued collaboration with the ASBB. I have continued to seek seed grant funding and succeeded in obtaining philanthropic donations.

For clarity, I will refer to 'CTE' rather than 'probable TES' in describing my patient's clinical diagnosis. I note that doctors have long held the ability to diagnose 'dementia pugilistica' or CTE in boxers purely based on clinical suspicion according to their training, and that CTE is commonly described in neurology textbooks. The claim that CTE cannot be diagnosed in life is a later phenomenon through the lens of modern neuroimaging available for other dementias. Just as we diagnose Alzheimer's disease clinically over time, I am confident in assessing patients as having suspected CTE. Interestingly, both Alzheimer's disease (AD) and CTE were discovered around a century ago, but the high incidence of AD accounting for approximately 60-70% of dementia has fuelled the development of clinically useful biomarkers (currently around 90% sensitive and specific). CTE research is now booming secondary to feverish media interest and increased scientific awareness, and this will likely generate the development of imaging or other biomarkers within the next decade. The absence of a 100% perfect biomarker for CTE is no different to Alzheimer's disease, in which confirmation is ultimately attained using postmortem neuropathology. In other conditions, such as cancer, tissue biopsies can achieve 100% certainty during life.

Concussions and repeated head trauma in contact sports at all levels, for all genders and age groups, with particular reference to:

a. the guidelines and practices contact sports associations and clubs follow in cases of player concussions and repeated head trauma, including practices undermining recovery periods and potential risk disclosure;

We must never blame the concussed athlete themselves, just as we never blame the sick for being sick. The head-injured player cannot be expected to know what is right, and repeated suggestions by the sports that the education of players to detect and manage their own concussion will solve the issue are flawed.

A national push to institute standardised and accessible head injury in sports guidelines was made in 1994 by the Australian government's NHMRC publication entitled *Football Injuries of the Head and Neck[2]*. This involved medical representatives from the four major football codes and the launch was well attended by the media. A convenient supplementary small booklet was published and the pocket-sized *Concussion; Notes for Referees, Umpires and Coaches*, presumably to be distributed amongst the sporting community.

The Canberra-based core panel included Dr Ray Newcombe, neurosurgeon (Chair. My father), Dr Robert Reid, Sports Physician, Dr Robert Smethills, medical officer (ACT Rugby Union), Ms Virginia Dove, Public Relations Officer (NHMRC), Ms Claire Brady (Executive Secretary) and Ms Sandra Twist (Executive Secretary). On the Medical Consultant Panel were Dr John Crompton, neuroophthalmologist, Dr Paul Curtin, plastic and reconstructive surgeon, Dr Nathan Gibbs, sports physician, medical officer (Australian Rugby League), Dr James Harrison, epidemiologist (National Injury Surveillance Unit, Australian Institute of Health and Welfare), Dr Siri Kannangara, rheumatologist, medical officer (Australian Soccer Federation), Dr Geoffrey Klug, paediatric neurosurgeon, Dr Paul McCrory, sports physician, medical officer (Australian Football League), Dr Glen Merry, neurosurgeon (Chair, Trauma Committees of Royal Australasian College of Surgeons and Neurosurgical Society of Australasia), Dr Cate Storey, neurologist (Australian Association of Neurologists - now ANZAN), Dr Geoffrey Vanderfield, neurosurgeon, medical officer (Australian Rugby Football Union), Dr Roger Vanderfield OBE (member of the International Rugby Football Board, former international referee) and Dr John Yeo AO, spinal injury rehabilitation.

Chapter 2 discussed concussion (mild head injury), including acute care, stating that 'whilst concussed, athletes should not continue participation', and referred to concussion sequelae, 'if the injury is repeated, long-term disability may result', and, 'If there are further head injuries, the loss may be cumulative and eventually there may be sufficient impairment of function for everyday activities to be affected'. However, a degree of controversy within the scientific literature was noted. At that time, rugby union was said to have a mandatory stand-down period of three weeks after a concussion, which, compared to today's standards, is a remarkably conservative approach. An air of caution was also conveyed by the suggestion that 'it may be reasonable to consider withdrawing a player from contact sport for the rest of the season if there has been a second

moderate or severe concussion'. The NHMRC football report was contemporaneous with the NHMRC's, 'Boxing Injuries'[3], which explored concussion and its management, as well as "Chronic Encephalopathy" in reference to CTE.

Subsequent publications on concussion management have been haphazard and more often formulated by the individual sports internally. Shifting concussion policies in some football codes now allow players to return at 11 or 12 days, which, if concussed on a Thursday to Sunday, allows play in time for the following fortnight's games. In medicine, it is highly irregular to select a period of recovery outside of a weekly or decimal period, such as 5 or 10 days.

National protocol inconsistency began to improve with a leading paper published in 2019 by the Australian Institute of Sport, Australian Medical Association, Australasian College of Sport and Exercise Physicians, Sports Medicine Australia (SMA) Concussion in Sport Australia Position Statement[4]. The change from 'If in doubt, sit it out' to 'If in doubt, sit them out' was a worthy attempt to engender the expertise of spectators, club staff and referees. The Blue Card system, first trialled in Australia in 2017 by the Australian Rugby Union, has been valued for its simple messaging and widespread uptake. The $340,000 Concussion and Brain Health (CBH) Project 2021-2024, led by the AIS in collaboration with the University of Canberra, Research Institute for Sports and Exercise, the University of Newcastle, and Hunter Medical Research Institute is a good initiative; however, this appeared to have been awarded through a closed, non-competitive grant.

b. the long-term impacts of concussions and repeated head trauma, including but not limited to mental, physical, social and professional impacts;

If we consider that all of us will experience some head impacts during our lives, and half of us will experience a concussion, the difference to that of a boxer with a calculated 20,000 head hits over a career is obvious, but the difference to that of a committed amateur football player is less so. In the absence of a biomarker indicating the severity of or recovery from mild traumatic brain injury, one's exact burden of repeated head trauma forms a spectrum that is difficult to quantify across a lifetime. Individual confounding factors and genetic susceptibility contribute to the complexity. One study of high school American football players estimated 24 head impacts per game of at least 14g-force[5]. CTE has been identified in the four major football codes; Australian rules, rugby union, rugby league and soccer[6–9]. Other sports such as cycling, horse racing, extreme sports, rodeo riding, mixed martial arts, wrestling and ice hockey are implicated in having a risk of CTE. We do not yet understand how many people in Australia have CTE, but based on US research, it is estimated that at least 9.6% of the NFL population is affected, perhaps rising to one in five if brain donation is high[10]. Given the popularity of contact sport in Australia, with over 100,000 rugby league registrants yearly, many of whom have played for over ten years, one would estimate that thousands, possibly tens of thousands, may be affected.

The risk of CTE would appear to follow a dose-response and double for every 2.6 years of play in American football[11]. A UK study in rugby union has identified a two-fold risk of neurodegenerative disease in an amateur and professional mixed cohort[12] and a three-fold risk in former professional soccer players[13]. A higher risk of motor neurone disease was also noted in the rugby study[12]. In my clinical experience, the most

severe patients tend to be boxers, often displaying earlier Parkinsonism alongside cognitive decline. Football players seem to develop later Parkinsonism, but nonetheless carry a typical burden for CTE of at least 20-100 concussions, some with more than 100 concussions and over 30 years of exposure in their risk profile. This is well in excess of the 5-year higher grade level considered a risk under research criteria for CTE[14]. It is important in taking a history of CTE risk to ask at what age play commenced, until when, and if there were any missed seasons. It is also important to provide the patient a correct definition of concussion beyond a simple, “knockout, K.O.”, and to help patients understand that it extends to confusion, dizziness, nausea and vomiting, headache, tinnitus, sleep disturbance, mood disturbance and other features before they nominate their total concussion history.

Strong circumstantial evidence suggests that repetitive traumatic brain injury causes CTE[15]. The link has been newly recognised by the NIH National Institute of Neurological Disorders and Stroke, which recently stated that ‘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’. A landmark study of 202 American football players found that 87% of the brains obtained were CTE positive, including 110 of 111 former National Football League players[16]. In Australia, the Australian Sports Brain Bank (ASBB) has reported that 12 out of 21 (57%) former athletes had CTE, with 6 of the 12 dying by suicide[17]. These studies acknowledge the potential biases within their select cohorts, and it is difficult to determine the true prevalence of CTE across individual sports. It is clear that some players are prone to more easily sustained and prolonged symptoms after repeated concussions in a ‘crescendo’ effect, and some will be prone to develop CTE due to complex genetic and environmental factors. In determining an individual’s risk of CTE, health practitioners should enquire about the presence of cognitive decline, the total number of years played, mood and behavioural profile, confounding factors and supportive criteria such as Parkinsonian features.

Cognitive, emotional and behavioural decline are archetypal characteristics of dementia. Cognitive decline relates to impaired memory, time and place orientation, attention, calculation, judgement, planning, visuospatial functioning and language. Emotional and behavioural decline relates to depression, anxiety, irritability, lability, impulsivity, criminality, anger, panic, obsessionality, confabulation, sleep-wake reversal, impaired insight, self-medicating behaviour, following behaviour and psychosis. In younger patients, emotional and behavioural decline often occurs before cognitive decline. These symptoms often co-occur in older patients[18] but every case is unique. Although the listed features can technically occur in other subtypes of dementia, CTE displays distinguishable clinical nuances, and I convey this after treating an estimated 1000 patients with vascular dementia, 500 with Alzheimer’s disease, 100 with Lewy Body dementia, 50 with frontotemporal dementia, 50 with alcohol-related dementia, 50 with normopressure hydrocephalus, 30 with Parkinson's plus disorders across my medical career. CTE is also often a long haul process of 10-25 years progression, in contrast to Alzheimer’s disease (6-10 years), frontotemporal dementia (6-8 years), Lewy body disease (5-7 years) and Parkinson’s plus disorders (5-7 years). Perhaps the main comparable subtypes as far as long duration dementia are vascular dementia, alcohol-related dementia and normopressure hydrocephalus, however, these have differing neuropsychological or radiological profiles. Dementia is Australia’s leading cause of death and determining potentially preventable forms and cures is important.

CTE is predominantly a younger onset subtype of dementia, and many of my patients are in the prime of their life, aged in their 30s, 40s, 50s and 60s. CTE may go undetected simply because medical practitioners or family do not suspect dementia in such a young individual or because of its very slow manifestation and occult nature. With traditional medical teaching that CTE is seen only in boxers, we as medical practitioners have been blinkered in our ability to diagnose it during life in other circumstances. Considerations include risks from other sports, previous assaults, domestic violence-related head injury, or military injury, including that related to blasts and parachuting (often a fall with head impact to the ground). In addition, because of partially intact social and linguistic pathways, patients with CTE can appear normal on the outside. However, in the words of my neuropsychology colleague Associate Professor Jennifer Batchelor, “once you scratch the surface, things are very clearly abnormal”, and neuropsychology testing is often revealing.

Our team has identified episodic memory impairments in line with international research that are an early feature, amongst other temporal and frontal changes. We have noticed patchy low metabolism and brain shrinkage on imaging. Our group is performing exploratory research on novel tau markers in CTE and continuing to monitor their path, all despite marginal seed funding.

c. the long and short-term support available to players affected by concussion and repeated head trauma;

Given that CTE is a cruel dementia that steals away the subtle emotional and behavioural cognition of a person, some of our patients have been managed under long-term mental health care for years prior to being diagnosed with a neurological disorder. Cognitive doctors such as neurologists, neuropsychiatrists or geriatricians work collaboratively with neuropsychologists to detect subtle forms of dementia so that treatment can be targeted, prognosis estimated, and research developed. We monitor patients yearly to confirm our hypotheses and to detect or treat any mimics. Sometimes dementia subtypes overlap, and we are used to evaluating what proportion of cognitive impairment may be due to other dementia or to other life factors such as substance use, depression, anxiety, posttraumatic stress disorder, intellectual baseline, developmental abnormalities, or malingering. Complexity is the dementia specialist’s everyday experience, and we are well-placed to assist in diagnosing and managing CTE.

Although CTE has its own distinct character, every individual with CTE is different. Some, who perhaps are more gentle in disposition, do not display the typical rage attacks described in CTE. Some may be physically aggressive and agitated ‘at the drop of a hat’, in response to minimal triggers such as queueing, stopping in traffic or walking around children’s clothes being left on the floor. Indeed, our patient’s partners and children within the family unit have sustained and are at risk of domestic violence from those with CTE, continuing a cycle of head injury burden. Some of my CTE patients are now entering dementia-related agitation and frailty, supported by the Aged Care system, the National Disability Insurance Scheme, or the Department of Veterans’ Affairs.

Although incurable, CTE management is available to modify outcomes like any other subtype of dementia. I have found in my own practice that there is a good response to mood-stabilising agents such as lamotrigine or sodium valproate in those with rage attacks. Sometimes low dose antipsychotic therapy is used to settle agitation and improve sleep. Antidepressant and antianxiety medication, along with counselling, is

considered. Melatonin and the benzodiazepine class can be used to settle REM Sleep Behaviour Disorder, whereby the patients act out violent dreams due to their dementia, presenting a risk of unintended harm to self and partner. Donepezil, an agent often used in Alzheimer's disease, appears to take effect to improve memory in some patients.

Around 30% of our cohort have chronic migraine due to head and neck injuries, and around 60% of these patients benefit from standard chronic migraine therapy available only in neurology clinics, including CGRP antagonists and botulinum toxin therapy. Females often sustain a more severe and prolonged concussion, and a higher incidence of migraine may be a factor. Chronic migraine may mentally cloud patients, even in the absence of a headache, and treating this disorder is important to understanding baseline cognition and whether a patient has CTE. Similarly, obstructive sleep apnoea due to musculature or weight around the neck during sleep can contribute to cognitive impairment but is amenable to treatment. Optimising the management of comorbid medical conditions, maintaining exercise and social activity, and ensuring a nutritious diet can all improve well-being and cognitive performance. Physiotherapists provide vital concussion and posttraumatic migraine relief alongside falls prevention and socialisation for isolated patients. Environmental approaches to reducing cognitive burden and increasing memory reminders using the expertise of occupational therapists are important.

Emotional and behavioural modification from an expert allied team, including neuropsychologists, clinical psychologists and neuropsychiatrists, is often beneficial. The support of services such as Dementia Australia is invaluable. We have developed an embraced program called Concussion Connect, which allows carers space to talk amongst themselves in one room, and patients in another, led by neurologists and neuropsychologists. This approach allows patients and carers to overcome their sense of being overwhelmed and isolated. Carers have expressed gratitude that they could reach out for support and the empathic approach to services offered.

e. the role of sports associations and clubs in the debate around concussion and repeated head trauma, including in financing research;

The link between CTE and repeated head injury was more frankly discussed in the NHMRC boxing report than in the football report, but both stated the need for research. The football report called for the establishment of a 'National Head and Neck Injuries Register', and the boxing paper suggested that '...research programs should be introduced with mandatory long-term follow-up'. Whilst prospective, long-term studies of well players is important, the delayed nature of CTE, sometimes many years after head injury, means that we should study older, symptomatic patients as well. In retrospective studies, confounding factors can be statistically adjusted for. The four major football codes are sizable commercial enterprises, and it is reasonable to assume that they may seek to help fund independent CTE research to build evidence as quickly as possible.

The reason for rescinding the two NHMRC guidelines and the apparent absence of a review process does not appear to have been made public. The NHMRC has a long history of producing robust and regularly reviewed medical guidelines. Some of the panel who participated in generating these guidelines are either recently retired or still practising within sporting organisations, affording an opportunity for new discussion. One

committee member (McCrory) is under allegations of plagiarism in scientific research, calling into question the Concussion In Sport Group (CISG) guidelines of which he was a founding member and Chair.

Recommendation 1: The Federal Government instigates revised and regularly reviewed guidelines on head and neck injury in sports

Despite the rise of concussion and CTE awareness in the US from 2005, when Omalu published the first case of CTE in American Football, and in particular the National Football League player settlement of US $765m in 2013, the Australian response across amateur sport has been slow. A general sporting culture of playing on despite concussion has continued over the years, in line with community perceptions that hard-played football is exciting football. We now have an opportunity to correct this.

Recommendation 2: The Federal Government funds longitudinal research on patients with existing symptoms of CTE, extending to postmortem neuropathological confirmation.

Positive action on acute concussion management has been seen in the past five years. Since the ASBB launched in 2018, there has been rising public awareness and concern as demonstrated by sustained media attention; however, there is a notable absence of direct study of CTE by sporting codes. Research programs led by the sports have tended to study those with a single concussion or asymptomatic controls rather than those with declared symptoms of CTE. For this reason, we directly offered to the NRL, AFL, Rugby Australia and Football Australia to consider studying our unique cohort.

The current Concussion in Sport Australia Position Statement does little to address CTE. It states that ‘There is currently no strong evidence clearly linking sport-related concussion with chronic traumatic encephalopathy’, and offers broad brush calls for greater research. Given that the results of large-scale retrospective studies and guidelines on CTE monitoring and management will likely take 5-10 years, and longer for prospective studies, I recommend that the interim position of the sports be more conservative and that they immediately enter a phase of harm minimisation on CTE.

f. the lack of a consistent definition of what constitutes ‘concussion’;

Both NHMRC publications highlight that the term concussion ‘means different things to different people’, and little has changed in this regard. Concussion definitions typically refer to immediate, transient neurological symptoms due to direct or indirect head impacts representing neuronal dysfunction. The patient may have difficulty understanding or recalling their own concussive symptoms, and many symptoms can be delayed by hours, days, weeks or even months. Difficulties arise in correlating the term ‘mild traumatic brain injury’ (mTBI), which is generally accepted to indicate pathological processes secondary to mechanical trauma, such as inflammation or cell death, that are not evident in neuroimaging. Pathological processes in mTBI may be triggered regardless of whether or not a patient has symptoms.

In the absence of a clinically useful biomarker panel to confirm neural damage after head injury, the definition of concussion will remain problematic. Biomarker research has

progressed, and serological markers of mTBI are undergoing advanced clinical research. A conservative path would be to equate neurological symptoms with mTBI and to respect the potential for subconcussions to cause injury until these biomarkers emerge.

i. alternative approaches to concussions and repeated head trauma in contact sport, and awareness raising about its risks;

A Code of Conduct for sports will facilitate cultural change at all levels. This should include the need for external and independent neurological review for those with a risk of CTE. Video evidence in televised sports of concussion should, where possible, be made available to the practitioner. Consideration of the need for sideline or ringside neurology review independent of the clubs should be made, preferably using a panel of practitioners. Obtaining a consistency of independently determined and enforceable protocols under regular medical review is vital.

Recommendation 3: The Federal Government mandates that sporting organisations must adhere to a Code of Conduct to:

a. ***Implement a public register of suspected and confirmed player concussions.***
b. ***Monitor the total duration of exposure to head contact (akin to radiation monitoring).***
c. ***Annually review the register and amend protocols accordingly.***
d. ***Fund players to attend an independent neurological assessment after a concussion.***
e. ***Establish subspecialist concussion and CTE clinics for athletes most at risk of CTE; reasonably those entering university or professional level or accumulating more than ten years of contact sport; for neurological baseline followed by 5-yearly volumetric MRI brain and review.***
f. ***Require players, coaches, administrators, and club doctors to undertake annual formal education on concussion.***

Neurologists, especially cognitive neurologists, are well placed to help lead the establishment of concussion clinics nationally to monitor players. There are approximately 50 cognitive neurologists and 600 neurologists in total in Australia. Neurologists are experts in the detection and management of subtypes of dementia, including CTE, the treatment of overlapping migraine or epilepsy to head injury, and the management of neurological comorbidities in head-injured patients. Rehabilitation physicians, sports physicians, emergency physicians, occupational physicians, neurosurgeons and general practitioner specialists often have experience in managing concussive disorders.

There is a doubling of CTE risk every 2.6 years in NFL combined with slow progression in symptoms[11] with sensitivity and specificity maximised at 11 years of play. Another recent study found that college-level and professional football players have 2.38 (95% simulation interval (SI): 1.16, 5.94) and 2.47 (95% SI: 1.46, 4.79) times the risk of being diagnosed with CTE as high-school-level players[19]. Therefore, it may be reasonable to consider baseline MRI brain with volumetric studies and 5-yearly screening for

higher-risk players (nominally those entering college / university / professional levels or over ten years of play). Definitive biomarkers of concussion and CTE will be needed to best refine monitoring protocols.

The retirement of players due to concussive head injury burden or concern over CTE is a relatively new phenomenon in Australia. Foreseeing that many of my CTE patients present having had 30 years of contact and more than 50 concussions, I feel nervous about players continuing beyond age 30, particularly if they began at 5. Many former players say they love the game and would play again but might have retired earlier were they aware of CTE risk. A lower burden of head injury overall will equate to lower rates of CTE. A philosophical and individualised approach is helpful, acknowledging that, for some, retirement is a higher health risk due to the development of depression, drug use, financial strife or other negative social consequence. As is usual medical practice, a careful and informed conversation between the medical specialist and the patient allows the patient to be fully informed before making any decision.

I recommend the government establish pathways of subspecialist care for those with the highest risk of CTE in dedicated concussion and CTE clinics, involving neurologists and neuropsychologists to handle retirement decisions. Retirement from professional sports programs might be revised to incorporate a tailored plan of continued neurological care and well-being.

Recommendation 4: The Federal Government periodically reviews the success of the Code of Conduct approach and, if unsuccessful, works with the State Government to enact Work Health and Safety laws pertaining to sporting associations.

Compensation schemes similar to that seen in asbestos-related diseases should be considered, alongside a review of insurance, workers compensation, and occupational health and safety frameworks. Success of the Code of Conduct may be measured in reducing concussion rates and severity, or in improved athlete outcomes. Surprisingly, workers compensation and occupational health and safety laws do not apply to sports either on-field or off-field when they appear to apply to many other contractual arrangements and community associations.

Recommendation 5: The Federal Government undertakes a series of public awareness campaigns informing young children about their brains, older children about brain health for life, and adults about detecting concussions and CTE.

Public health campaigns are important to address not only the risk of sport but the risk of leaving sport. I am aware of at least one football program that has been cancelled in a private school, and this causes me great concern. We are missing role models for good concussion care and monitoring in society. Education on brain injury should begin through youth sporting clubs or schools.

I recommend firstly, that the government establish a public health campaign on understanding and valuing the brain for young children as a basis for lifelong brain health. A model similar to Healthy Harold could be considered.

Secondly, I recommend a program on having a healthy brain for life through sport with avoidance of harm for older children. A model similar to Slip Slop Slap Seek and Slide messaging could be considered. This investment could lower rates of dementia overall through greater awareness of the negative effects of alcohol, smoking, and head injury, and the benefits of sleep preservation, eating well, monitoring vascular risk and maintaining social and physical activity for life.

Thirdly, a public awareness campaign for adults, including parents, coaches, commentators and spectators, could focus on recognising concussions and CTE. I developed the Concussion Big5 program to provide the public with the cardinal neurological signs of concussion in an easily recognisable format (www.concussion big5.au). These signs are within the realm of standard neurological practice to indicate a disturbance to the brain. The Big5 are; Slump (loss of consciousness) Sway (ataxia) Slow (bradykinesia) Stun (confusion) and Slur (altered responsivity) and two videos were produced; one with famous players and partners, and an animated cartoon. I have invested substantive personal funding (beyond $30,000) and many hours of my time in generating potential public health campaigns such as the Concussion Big5.

Concussion Connect, held every two months and now in its third year, has provided a sanctuary of education and support for patients and families facing possible CTE. Patients are supported in one room and carers in another by experienced neurology and neuropsychology teams in a confidential group-therapy program addressing the common symptoms of CTE. Our feedback has been excellent, with one patient saying, “It’s soothing when you know you are not the only one. Others don’t get it”, and another, "I can feel my confidence coming back in a different way that is safer for my family”. The program could be considered for expansion nationally, potentially in collaboration with Dementia Australia sites. Our team at Macquarie University have generated two national online seminars on concussion and CTE, created website material for the Australian CTE Biobank and Dementia Australia, spoken to schools, liaised with Sydney FC on campus, obtained support from The of Men of League, Equestrian Australia, Brain Injury Australia. We have sought communication with Snow Sports Australia, Racing NSW, and Racing Victoria and worked with overseas community advocates such as Judith Gates (Head for Change in the UK) and Chris Nowinski (Concussion Legacy Foundation). We have reached out to the Neurological Foundation and Auckland University for collaboration and co-launched with their New Zealand CTE Biobank. We have created podcasts and videos on concussion and CTE, available freely online.

j. international experiences in modifying sports for children;

Prevention is possible for many, even if Australians continue playing contact and collision sport. With a reduction in training burden, age of first contact play, an improved culture around concussion and CTE detection, the education of parents, commentators, club officials and referees, and a strictly held set of national guidelines, we will be well ahead internationally. We must commence measuring strength not in ‘body on the line’ related head injuries but in a culture of ‘best brain, best performance’ and head injury avoidance from the earliest years. We must endeavour to improve the brain health literacy of children as they develop in sports via an enduring public health platform that they may themselves teach in years to come.

There are valid pathways for maintaining the vascular benefits of sport with delayed head impacts in children. Modified games such as ‘tag’ or ‘flag’ for children should be

more widely adopted, and contact skills should be commenced at a later age. Commencement in adolescence is neurologically preferable to earlier childhood, recognising that the brain does not fully develop until the mid-20s; however, later may not be safer as far as learning conservative tackle methods and establishing correct motor memory. Athletes I have asked suggest age 12 as a good time to learn a safer tackling method and that age 14 is, arguably, too late.

In my opinion, boxing presents the highest risk for CTE, and should not commence until the medical age of informed consent at 16.

Input from the paediatric neurology community will be an important step for evolving recommendations in line with international standards. The Concussion Legacy Foundation in the US #tacklecanwait initiative recommends no tackling before age 14 on the basis of a ten times risk of CTE in those commencing American Football at age five versus age 14. The use of large helmets with weight and centrifugal forces is cited as a reason why it is hard to equate this data with Australian sports. I recommend this may be relevant for the under-14 age groups when the head is relatively larger than the body. However, in considering injury styles between the codes, an American football tackle may be as severe to a fall onto the head from marking the ball in AFL, a clash of heads in soccer at speed, a two-player tackle at a running individual in rugby league, or a forward taking out the legs of a rugby union player catching a high ball who impacts the ground head first. Until we gather more data, a no contact before age 14 may be the safest approach.

k. any other related matters.

Many of our Australian contact sports are shared in New Zealand, and the opportunity to collaborate on these efforts is suggested.

References

1. Keene CD, Latimer CS, Steele LM, Mac Donald CL. First confirmed case of chronic traumatic encephalopathy in a professional bull rider. Acta Neuropathol. 2018;135: 303–305.

2. Head and Neck Injuries in Football: Guidelines for Prevention and Management. Publishing date: 1994 Status: Rescinded Reference number: SI2B Available in print: No - PDF only Further information: nhmrc.publications@nhmrc.gov.au.

3. Boxing Injuries.NHMRC Publishing date:1994 Status:Rescinded Reference number:SI1.

4. Elkington LJ, Manzanero S, Hughes DC. An update to the AIS-AMA position statement on concussion in sport. Med J Aust. 2018;208: 246–248.

5. Broglio SP, Martini D, Kasper L, Eckner JT, Kutcher JS. Estimation of head impact exposure in high school football: implications for regulating contact practices. Am J Sports Med. 2013;41: 2877–2884.

6. Pearce AJ, Sy J, Lee M, Harding A, Mobbs R, Batchelor J, et al. Chronic traumatic encephalopathy in a former Australian rules football player diagnosed with

Alzheimer's disease. Acta Neuropathol Commun. 2020;8: 23.

7. Buckland ME, Sy J, Szentmariay I, Kullen A, Lee M, Harding A, et al. Chronic traumatic encephalopathy in two former Australian National Rugby League players. Acta Neuropathol Commun. 2019;7: 97.

8. Lee EB, Kinch K, Johnson VE, Trojanowski JQ, Smith DH, Stewart W. Chronic traumatic encephalopathy is a common co-morbidity, but less frequent primary dementia in former soccer and rugby players. Acta Neuropathol. 2019;138: 389–399.

9. Ling H, Morris HR, Neal JW, Lees AJ, Hardy J, Holton JL, et al. Mixed pathologies including chronic traumatic encephalopathy account for dementia in retired association football (soccer) players. Acta Neuropathol. 2017;133: 337–352.

10. Binney ZO, Bachynski KE. Estimating the prevalence at death of CTE neuropathology among professional football players. Neurology. 2019;92: 43–45.

11. Mez J, Daneshvar DH, Abdolmohammadi B, Chua AS, Alosco ML, Kiernan PT, et al. Duration of American Football Play and Chronic Traumatic Encephalopathy. Ann Neurol. 2019. doi:10.1002/ana.25611

12. Russell ER, Mackay DF, Lyall D, Stewart K, MacLean JA, Robson J, et al. Neurodegenerative disease risk among former international rugby union players. J Neurol Neurosurg Psychiatry. 2022. doi:10.1136/jnnp-2022-329675

13. Mackay DF, Russell ER, Stewart K, MacLean JA, Pell JP, Stewart W. Neurodegenerative Disease Mortality among Former Professional Soccer Players. N Engl J Med. 2019;381: 1801–1808.

14. Katz DI, Bernick C, Dodick DW, Mez J, Mariani ML, Adler CH, et al. National Institute of Neurological Disorders and Stroke Consensus Diagnostic Criteria for Traumatic Encephalopathy Syndrome. Neurology. 2021. doi:10.1212/WNL.0000000000011850

15. Nowinski CJ, Bureau SC, Buckland ME, Curtis MA, Daneshvar DH, Faull RLM, et al. Applying the Bradford Hill Criteria for Causation to Repetitive Head Impacts and Chronic Traumatic Encephalopathy. Front Neurol. 2022;13: 938163.

16. Mez J, Daneshvar DH, Kiernan PT, Abdolmohammadi B, Alvarez VE, Huber BR, et al. Clinicopathological Evaluation of Chronic Traumatic Encephalopathy in Players of American Football. JAMA. 2017;318: 360–370.

17. Suter CM, Affleck AJ, Lee M, Pearce AJ, Iles LE, Buckland ME. Chronic traumatic encephalopathy in Australia: the first three years of the Australian Sports Brain Bank. Med J Aust. 2022;216: 530–531.

18. Stern RA, Daneshvar DH, Baugh CM, Seichepine DR, Montenigro PH, Riley DO, et al. Clinical presentation of chronic traumatic encephalopathy. Neurology. 2013;81: 1122–1129.

19. LeClair J, Weuve J, Fox MP, Mez J, Alosco ML, Nowinski C, et al. Relationship between level of American football playing and diagnosis of chronic traumatic encephalopathy in a selection bias analysis. Am J Epidemiol. 2022;191: 1429–1443.