Chronic Traumatic Encephalopathy - CTE
What is it?
How should we manage it?

Ken Quarrie
Chief Scientist
New Zealand Rugby

Job chance to prevent injuries

By Hayden Meikle

Rugby

Dunedin's Ken Quarrie will combine his passion for rugby with a desire to reduce the number of serious injuries in the sport when he joins the staff of the New Zealand Rugby Football Union next month.

Quarrie (32) has been appointed NZRFU injury prevention co-ordinator and will be based at the union's head office in Wellington.

He and wife Nicola leave Dunedin for the capital tomorrow.

Quarrie has been based in Dunedin since 1986 and has completed a master of physical education (biomechanics) degree at the University of Otago.

He spent seven years with the university's rugby injury and performance project and worked as a fitness trainer for the Harbour club and Otago.

"I love rugby and obviously the prevention of serious injuries brought about by rugby is also something I'm passionate about," Quarrie said yesterday.

"Having had first-hand experience with injured players, I know what an impact injuries are having on the game."

The old-fashioned perception that rugby players should play on while injured was slowly dying, Quarrie said. "I'm pretty sure people are realising that top players simple can't perform if they are injured.

"I'm looking forward to liaising with people at all levels and hopefully reducing the level of rugby-related injuries."

Quarrie played premier club rugby for Kaikorai and University B in the late 1980s and early 1990s.

His new role has been created following a partnership between the NZRFU and ACC.

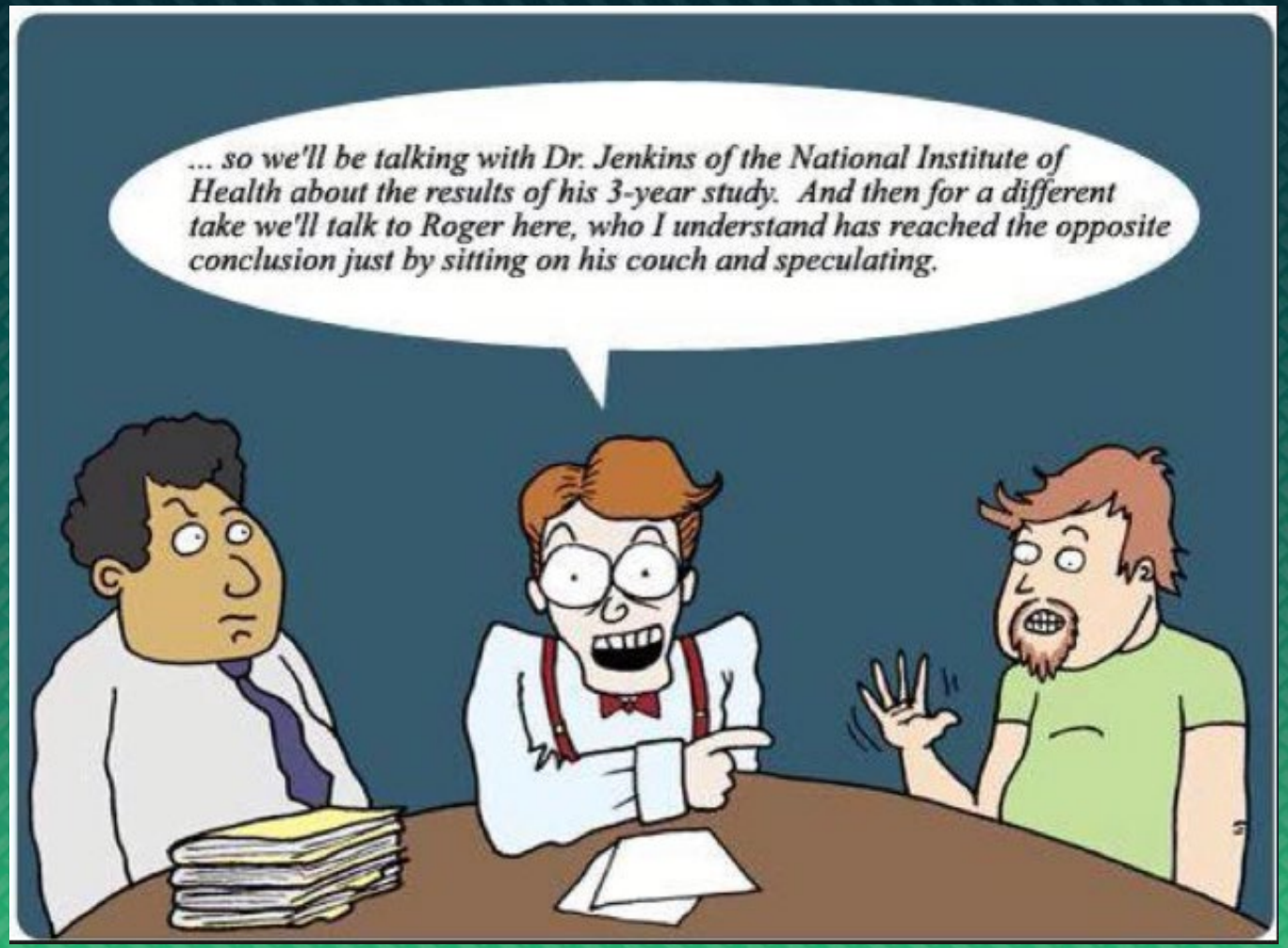
There are many ways of looking at CTE

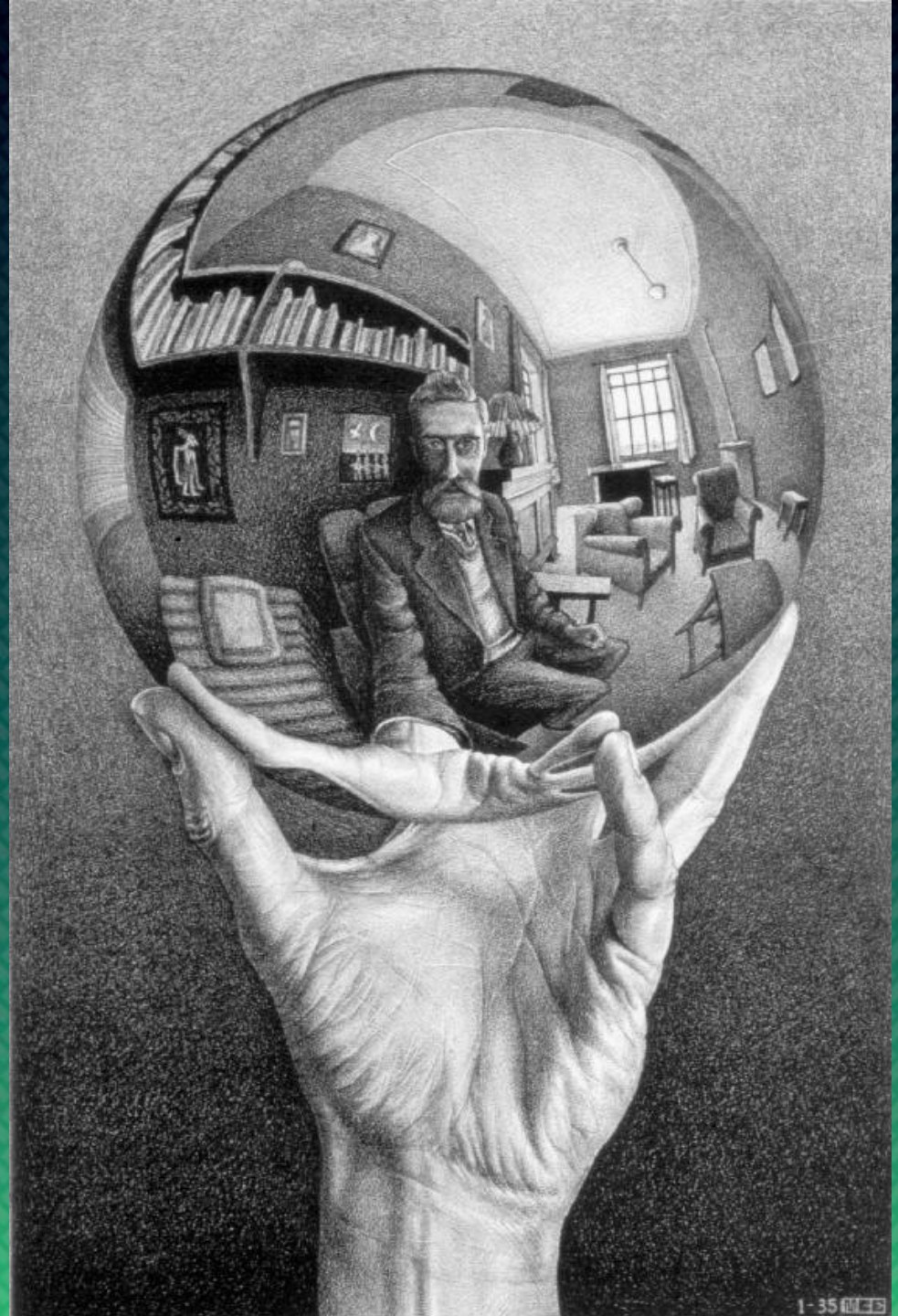
CTE is:

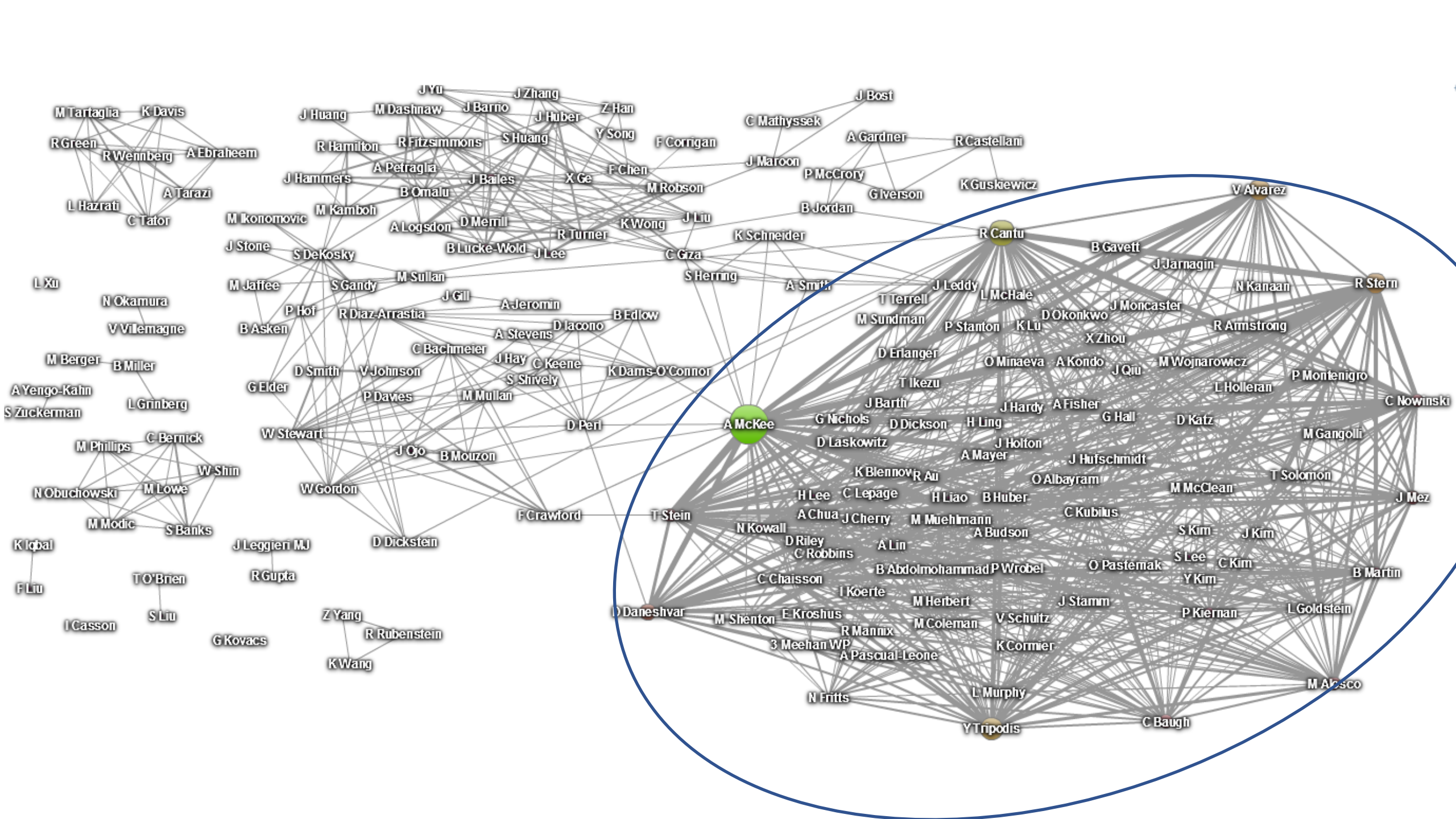
- A neuropathological diagnosis at autopsy
- An object of scientific study
- A cultural phenomenon
- A lived experience

Ventresca, 2019

Evidence in a ‘post-truth’ world – the problem of expertise



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C Baugh

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

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Today, CTE can only be definitively diagnosed following a neuropathological examination (31, 33). Because the National Institutes of Health recognizes consensus neuropathological criteria for CTE, but does not yet recognize clinical diagnostic criteria, this article examines the relationship between RHI and CTE neuropathology, and does not substantially explore the separate question of a causal relationship between CTE neuropathology and clinical symptoms (33).

- Nowinski et al., 2022

What is being claimed?

"CTE is clinically associated with symptoms of irritability, impulsivity, aggression, depression, short-term memory loss and heightened suicidality that usually begin 8-10 years after experiencing repetitive mild traumatic brain injury"

Mckee et al., 2013

Most common clinical features (>70%) in pathologically verified CTE

COGNITIVE	BEHAVIORAL	MOOD	MOTOR
Memory	Physical violence	Depression	Ataxia
Executive dysfunction	Verbal violence	Hopelessness	Dysarthria
Impaired attention	Explosivity	Suicidality	Gait impairment
Dementia	Loss of control	Anxiety	Tremor
Cognitive impairment	Short fuse	Irritability	Masked facies
	Impulsivity	Apathy	Rigidity
	Paranoia	Loss of interest	
	Rage	Fearfulness	

Montenigro et al. Alzheimers Res Ther 2014

What is being claimed?

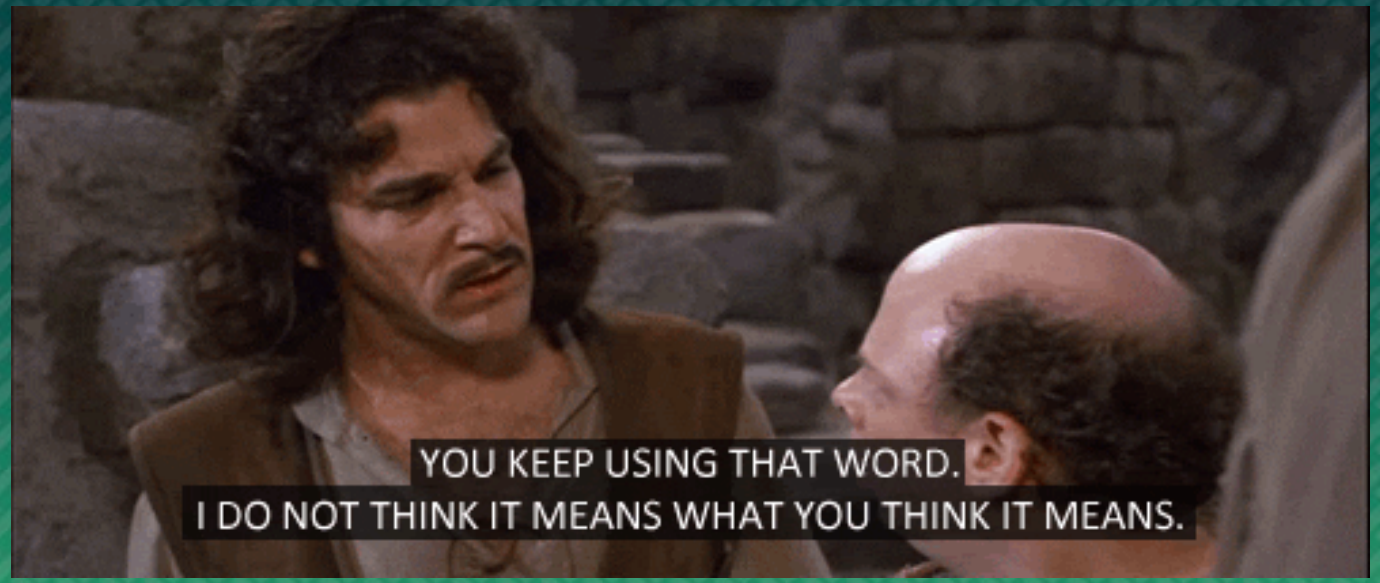
Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease caused by exposure to repetitive head impacts (RHI), such as those sustained in contact sports

Mahar, Alosco & McKee, 2017

What is being claimed?

*Baugh et al. 2014

…wait a minute…what do we mean by ‘cause’ again?



There is considerable scientific controversy regarding CTE as a distinct *clinical* entity

"It has not been established that the described tau pathology, especially in small amounts, can cause complex changes in behaviour such as depression, substance abuse, suicidality, personality changes or cognitive impairment"

Iverson et al. 2015

- Stewart et al. 2019 (61 authors unaffiliated with sports organisations).

Contrary to common perception, the clinical syndrome of CTE has not yet been fully defined, its prevalence is unknown, and the neuropathological diagnostic criteria are no more than preliminary. We have an incomplete understanding of the extent or distribution of pathology required to produce neurological dysfunction or to distinguish diseased from healthy tissue, with the neuropathological changes of CTE reported in apparently asymptomatic individuals. Although commonly quoted, no consensus agreement has been reached on staging the severity of CTE pathology. A single focus of the pathology implicated in CTE is not yet sufficient evidence to define disease.

- Stewart et al. 2019 (61 authors unaffiliated with sports organisations).

Unfortunately, the uncertainties around the clinical syndrome and the pathological definition of CTE are not acknowledged adequately in much of the current research literature or related media reporting, which at times has resembled science by press conference. Too often an inaccurate impression is portrayed that CTE is clinically defined, its prevalence is high, and pathology evaluation is a simple positive or negative decision. This distorted reporting on CTE might have dire consequences. Specifically, individuals with potentially treatable conditions, such as depression or post-traumatic stress disorder, might make decisions on their future on the basis of a misplaced belief that their symptoms inevitably herald an untreatable, degenerative brain disease culminating in dementia. We propose that the principle of, first, to do no harm, is used when communicating on CTE, whatever the platform.

- Response from Casper et al:

A recent Correspondence letter called for balanced reporting about CTE, but we are concerned that Stewart and colleagues ignore the troubling history of experts collaborating with for-profit organisations to foreground uncertainty and eventually forestall regulatory efforts, limit liability, and downplay harm. We contend that journalists should not seek balanced reporting, because doing so makes it harder for at-risk individuals to evaluate the dangers of CTE. There are hazards in the overstatement of risks, but understatement also brings hazards. Given the history of NFL-led attempts to downplay harm, a call for balanced reporting in this field can give undue credence to uncertainties.

Recording

Some Important Unanswered Questions Relating to CTE

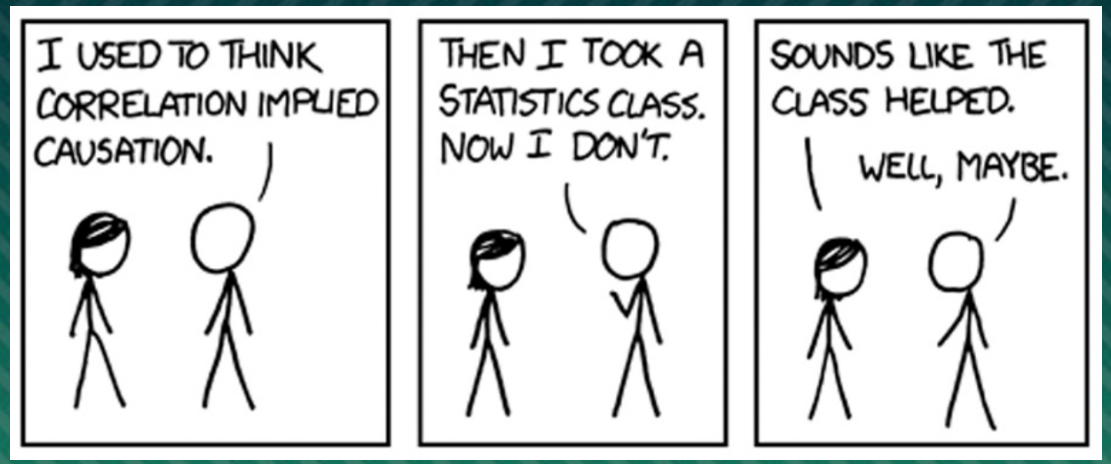
1. Prevalence
2. Genetic or other risk factors
3. Resilience factors
4. Clinical diagnostic criteria (likely high rates of misdiagnosis using the previous research criteria; research needed on the new criteria)
5. Extent to which the neuropathology causes specific clinical symptoms or problems
6. Extent to which the neuropathology is progressive
7. Extent to which the clinical features are progressive

Causality in epidemiology

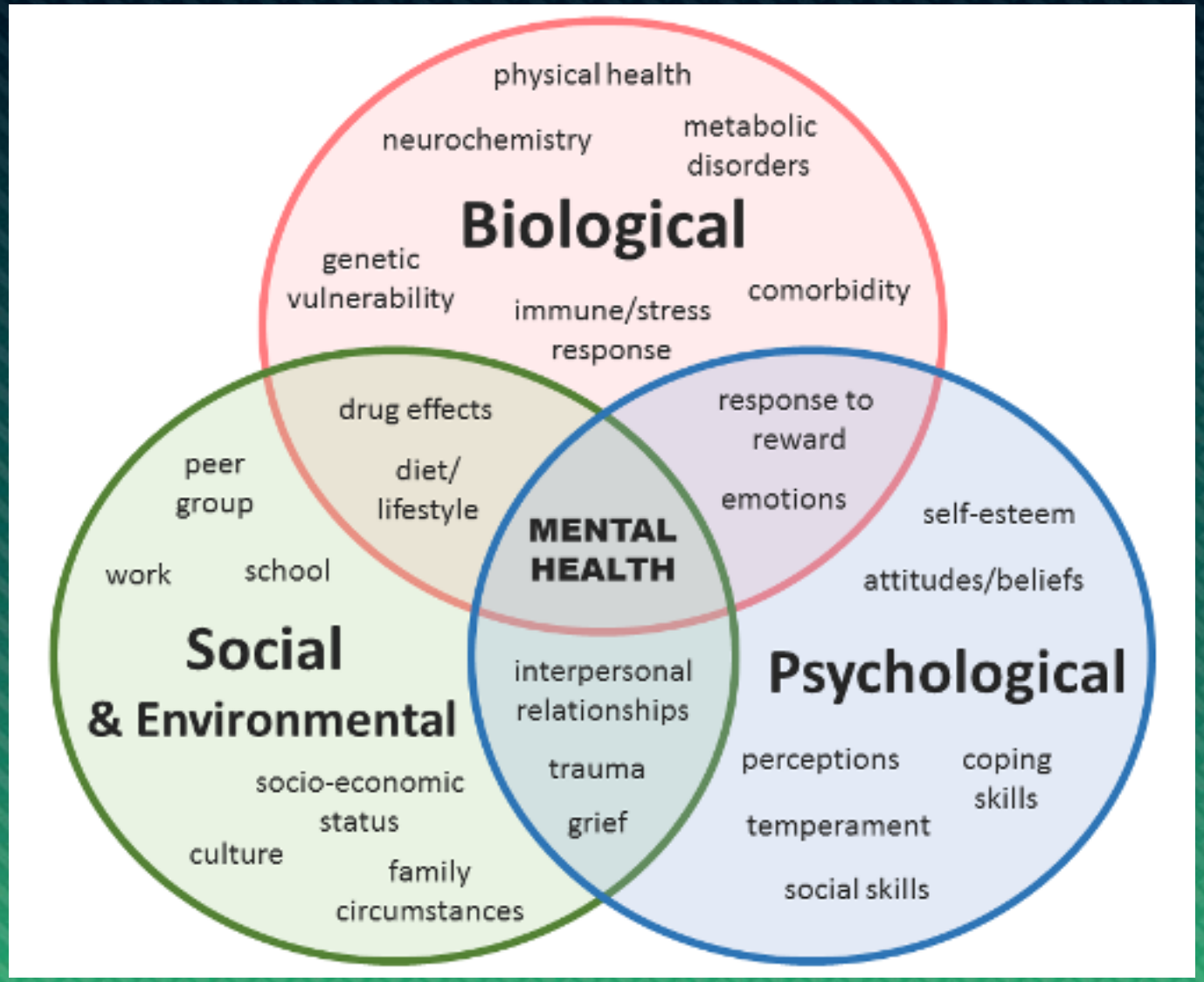
"No single, clearly articulated definition"

- Five categories:
  - Production
  - Necessary and sufficient
  - Sufficient-component
  - Counterfactual
  - Probabilistic

Parascandola & Weed, 2001



The ‘biopsychosocial’ model of health (Engel, 1977)



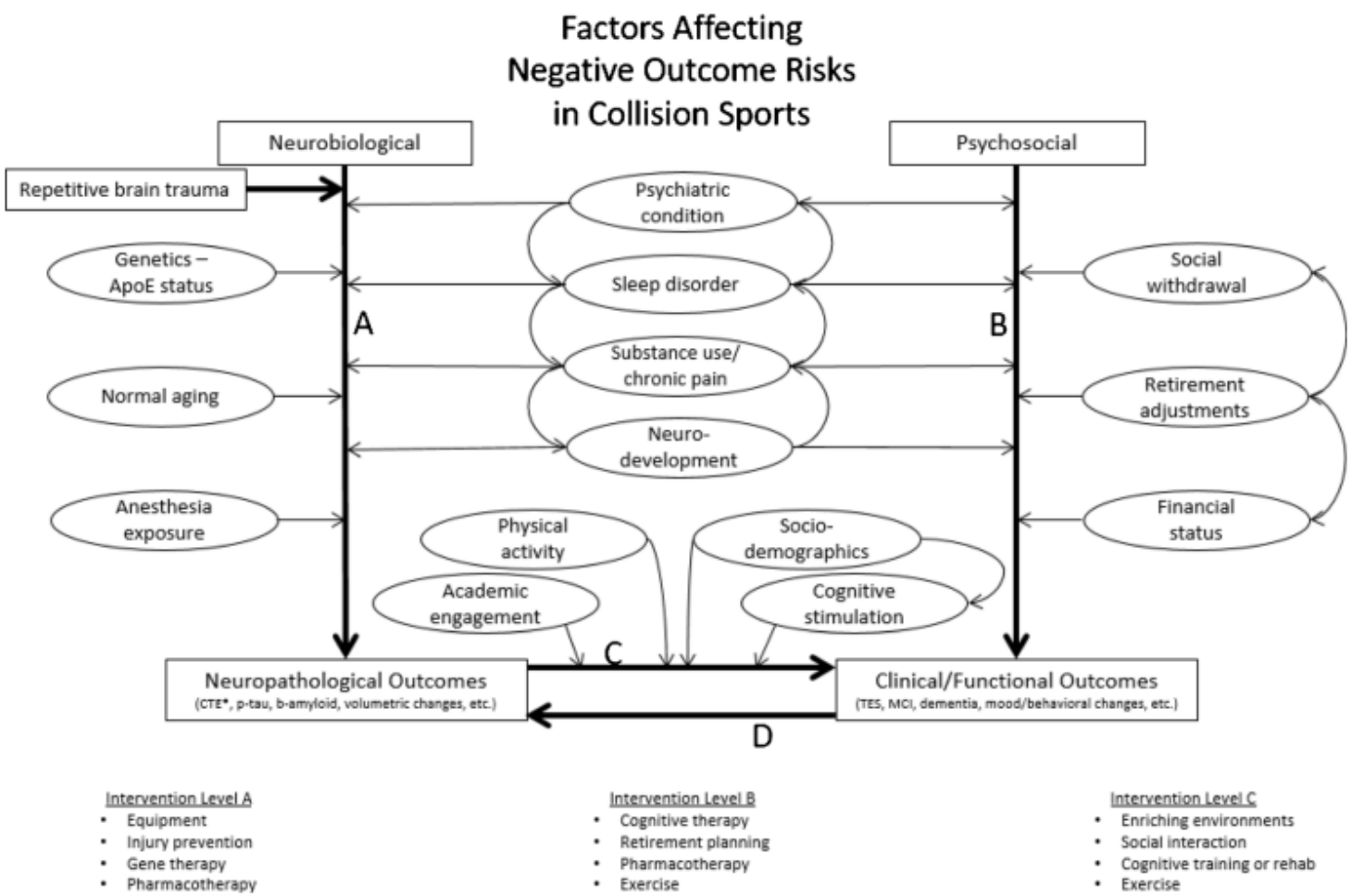


Fig. 1 Conceptual framework describing a non-exhaustive list of mediators and moderators of neuropathological and clinical outcomes of collision sport exposure. Evidence suggests repetitive brain trauma is a primary risk factor for developing neuropathology unique to CTE. *Arrows* between factors (*ovals*) indicate possible uni- and bidirectional influences amongst these variables. *Path A* includes the potential neurobiological mediators and moderators of pathological burden. *Path B* includes psychosocial factors directly influencing clinical/functional outcomes which over time, if untreated, may result in underlying neuronal reorganization or compound neuropathological outcomes (*Path D*). Path C describes factors that may modify the degree to which neuropathological changes ultimately manifest clinically (i.e., "cognitive reserve"). Possible treatment interventions may mitigate neuropathological outcomes by directly targeting neurobiological factors (Intervention Level A), prevent or minimize negative clinical/functional outcomes by directly targeting psychosocial factors (Intervention Level B), or mitigate the degree to which accumulated neuropathology translates to clinical outcomes (Intervention Level C). Research is essential for early identification of at-risk populations and improved understanding of appropriate intervention timing (e.g., before, during, and/or after playing careers). Abbrev: *ApoE* apolipoprotein E, *CTE* chronic traumatic encephalopathy, *p-tau* phosphorylated tau, *b-amyloid* beta amyloid, *TES* traumatic encephalopathy syndrome, *MCI* mild cognitive impairment *Research linking CTE to repetitive brain trauma, specifically, suggests mediating/moderating factors are not causally responsible for its unique distribution of neurofibrillary tangles

Chronic Traumatic Encephalopathy as a Preventable Environmental Disease

Michael E. Buckland[1,2], Andrew J. Affleck[1], Alan J. Pearce[3] and Catherine M. Suter[1,2]*

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In this Perspective we explore the evolution of our understanding of chronic traumatic encephalopathy (CTE) and its relationship with repetitive head injury. As with many neurodegenerative conditions, there is an imperfect correspondence between neuropathology and clinical phenotype, but unlike other neurodegenerative diseases, CTE has a discrete and easily modifiable risk factor: exposure to repetitive head injury. Consequently, evaluation of the evidence regarding exposure to repetitive head injury and CTE risk should be undertaken using public or occupational health frameworks of medical knowledge. The current debate over the existence of CTE as a disease of concern is fuelled in part by immediate medico-legal considerations, and the involvement of high-profile athletes, with inevitable media interest. Moving beyond this debate has significant potential to address and reduce disease impact in the near future, and provide novel insights into mechanisms underlying abnormal protein accumulation in CTE and other neurodegenerative diseases.

Keywords: concussion, tau, punch-drunk, neuropathology, dementia, neurodegeneration

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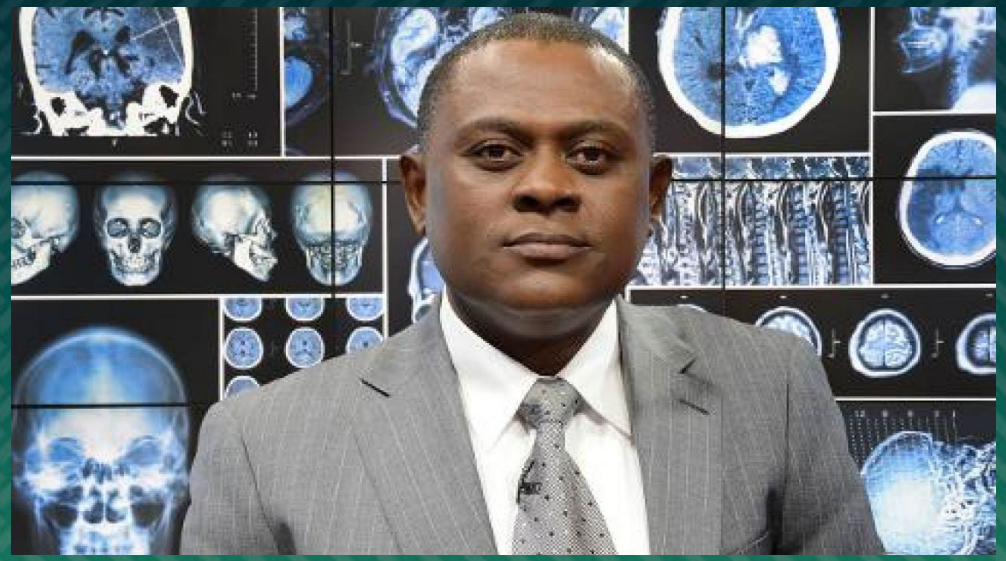
"A determination of causation between RHI (repetitive head impacts) and CTE has significant medico-legal consequences for professional and amateur sports, both in terms of liability and long-term viability," the study published in the Frontiers in Neurology journal concluded. "If CTE is environmentally caused, then settings with exposure to RHI, which could include participation in some sports, may become regulated by governmental organizations that oversee workplace and public safety, and individuals and organizations could become financially liable for the care of those who develop CTE."

Public Health - scientists and advocates

- Science and advocacy are both important
- Establishing the evidence
- Communicating the evidence
- Driving change



“No child under the age of 18 should be permitted to play contact/collision sports”

“after only one season of rugby, your child may have suffered brain damage as the human brain does not have any reasonable capacity to regenerate itself.”

“Once you have suffered a concussion, it’s going to stay with you your entire life.”

Flag Football Under 14: An educational campaign for parents

If you are a parent considering enrolling your child in football, **the Concussion Legacy Foundation strongly recommends you delay enrolling your child in *tackle football* until the age of 14**. Research continues to show us that the long-term effects of repetitive brain trauma from tackle football can be catastrophic. Meanwhile, football experts - from coaches to Hall of Fame players - remind us that you don't need to start tackle young in order to become a great football player. Until tackle football is proven safe for the developing brain, we urgently recommend flag and other non-tackle versions of football before age 14.

As parents, you deserve to make an informed decision about your child's future health. That's why we launched Flag Football Under 14.

> To review the science behind Flag Football Under 14, read the White Paper.

ELSEVIER

Seminars in
Pediatric
Neurology

Youth Exposure to Repetitive Head Impacts From Tackle Football and Long-term Neurologic Outcomes: A Review of the Literature, Knowledge Gaps and Future Directions, and Societal and Clinical Implications

Michael L. Alosco, PhD,* and Robert A. Stern, PhD*,†

parents of children who play or played youth CCS that they or their loved ones are at risk for CTE or other neurologic or psychiatric consequences. Messaging that conflates "concussion" and "CTE" add to the confusion and fear, such that some clinicians are faced with children and/or their parents who fear that a single concussion may result in later-life dementia or suicide. The existing studies that examined youth tackle football and long-term neurologic outcomes have all been among tackle football players who went on to play in high school, college, and, in most studies, in the NFL. It is unlikely that exposure to RHI from youth tackle football alone would confer risk for a later-life dementia disorder. And, not everyone who plays tackle football, even at higher levels, will develop CTE. The threshold for the num-

TABLE 2 | Other important strength of association results.

Study	Outcome	Exposure	Hazard ratio	Notes
Lehman et al. (53)	Neurodegenerative COD	Former NFL v. general population	3.26 (1.9–5.22)	3.26 is biased low, given that SMR for all COD combined was 0.53 (a strong "healthy worker effect")—neurodegenerative COD were especially enriched in the NFL group
Venkataramani et al. (54)	Neurodegenerative COD	Former NFL v. former "replacement players" who played several games during the 1987 strike	4.09 (0.23–73.3)*	Non-significant OR. HWE minimized by using unexposed group fit enough to be on an NFL roster briefly; however, these (181) deaths are only those in each cohort who died in their 50s (about 5% of each group)
Mackay et al. (55)	Neurodegenerative COD	Former pro soccer players v. age/sex matched controls	4.1 (2.88–5.83)	
Nguyen et al. (56)	Neurodegenerative COD (underlying or contributing cause)	Former NFL players v. former Major League Baseball players	2.99 (1.64–5.45)	HWE eliminated by comparing athletes with/wo RHI. But only about 15% of this cohort had died as of study publication
Russell et al. (57)	Neurodegenerative COD	Former pro soccer players v. age/sex/SES matched controls	3.66 (2.88–4.65)	HR among defenders was >2.5× that among goalies, suggesting a gradient with RHI
Daneshvar et al. (58)	ALS diagnosis	All NFL players who debuted between 1960 and 2019 vs. general population rates	3.59 (2.62–5.69) (incidence ratio)	3.94 (2.62–5.69) (mortality ratio)

**Infinite raw OR (zero cases among "unexposed"); Haldane-Anscombe correction applied.*

All scientific work is incomplete—whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time.

Sir Austin Bradford-Hill (1965)

- “…there is no reason not to act and follow the precautionary principle, namely to remove contact, including the tackle from the school game, until the reintroduction of contact can be shown to be safe.”
- Pollock and Kirkwood, BJSM 2016

Does the scientific uncertainty around CTE & other late-onset neurodegenerative conditions require the application of the precautionary principle?

Yes

(probably)

Does application of the precautionary principle *require* that tackles should be removed from school rugby?

Does it imply that all contact sports should be banned for children under the age of 14?

I don’t believe so at present

Guidelines for Application of the Precautionary Principle*

Proportionality:	"Measures: must not be disproportionate to the desired level of protection and must not aim at zero risk"
Nondiscrimination:	"comparable situations should not be treated differently and…different situations should not be treated in the same way unless there are objective grounds for doing so"
Consistency:	"measures should be comparable in nature and scope with measures already taken in equivalent areas in which all the scientific data are available"
Examination of the benefits and costs of action or lack of action:	"This examination should include an economic cost/benefit analysis when this is appropriate and feasible. However, other analysis methods…may also be relevant"
Examination of scientific developments:	"The measures must be of a provisional nature pending the availability of more reliable scientific data"…"scientific research shall be continued with a view to obtaining more complete data"

*EC Commentary, 2 February 2000

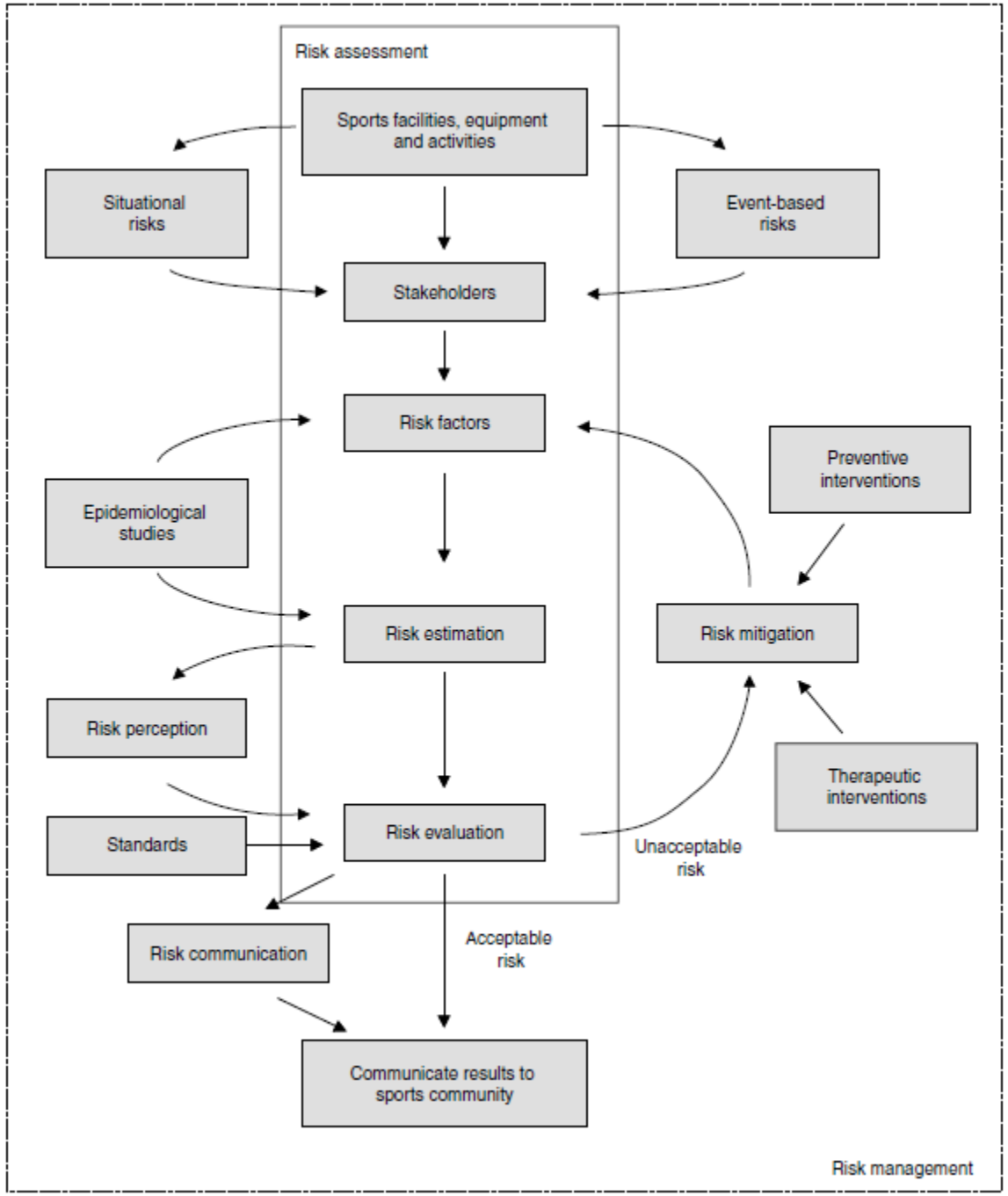


Fig. 1. A framework for risk management in sport.

Facts and values: on the acceptability of risks in children's sport using the example of rugby — a narrative review

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ABSTRACT

A clash of values has been identified between those who assert that:

1. all childhood injuries, regardless of origin, are inherently undesirable and should be prevented and;
2. those who believe that some measure of injury to children is an acceptable compromise for the physical benefits associated with physical activity and the development of abilities to appraise and deal with risks.

A debate regarding whether the tackles and collisions permitted in schools' rugby represent acceptable risks, and what steps should be taken if they do not, exemplifies the issue.

Questions regarding the magnitude of injury risks in sport are issues of fact and can be quantified via the results of injury surveillance studies. Risks are neither

BJSM, claimed that a 'complete ban on tackles is unnecessary and may be detrimental.'[4]

Pollock is a professor of public health research and policy, and in her 2014 book *Tackling Rugby: What every parent should know about injuries*[5] has provided a great deal of detail about why she believes a ban on contact should be implemented in schools' rugby. In her book, she stated:

> *Rugby is a high-impact collision sport that entails an expectation of some injuries. At what point do injuries become sufficiently serious and sufficiently frequent to be unacceptable?*[5]

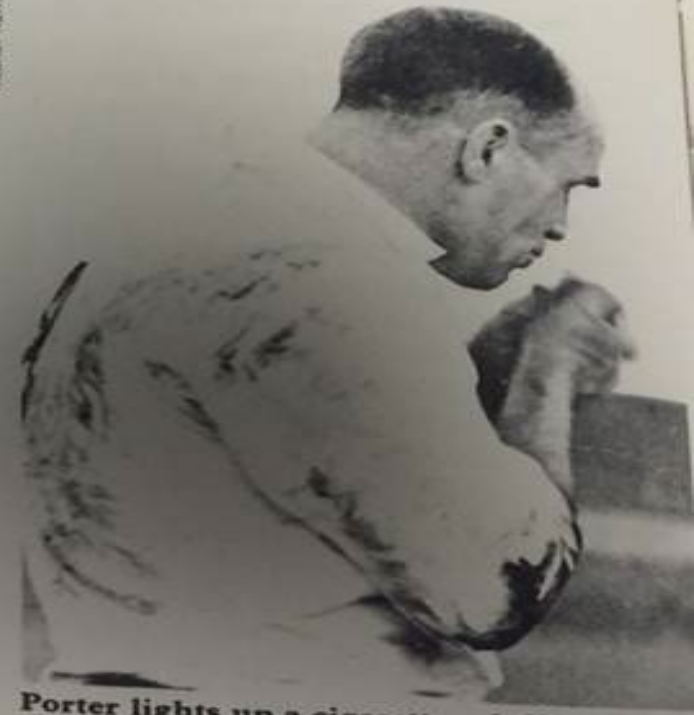
The question raised is a good one and applies to all sports and recreational activities for all ages, not just rugby. The purposes of this manuscript are to:

Match 40

NEW ZEALAND 13, GREAT BRITAIN 10

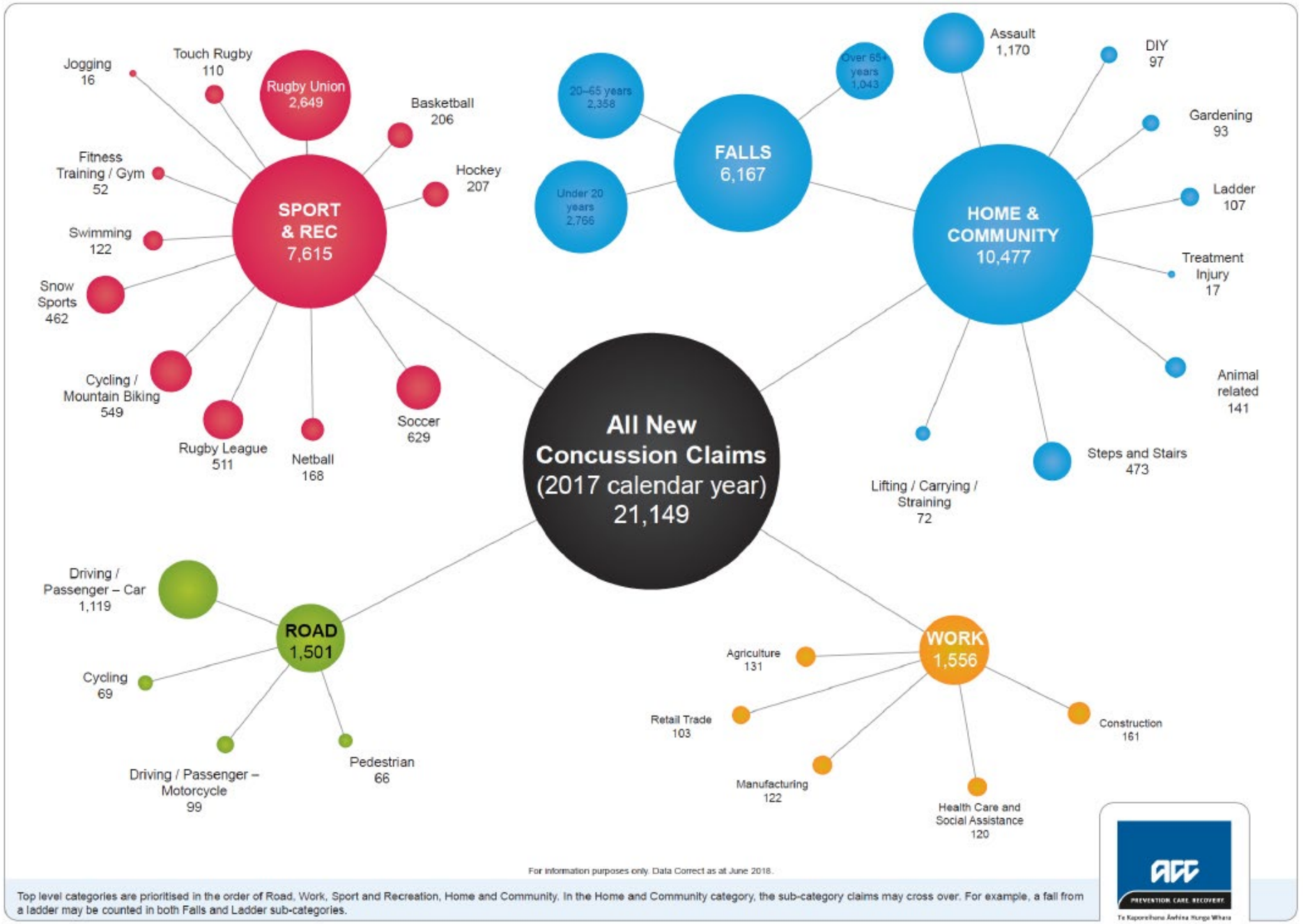
NZ RUGBY MUSEUM

WEEKLY NEWS



Porter lights up a cigarette at halftime.

Four pairs of arms reach for the ball in this classic line



Rugby Smart

NEW ZEALAND RUGBY

adidas

Concussion and chronic traumatic encephalopathy: International Rugby Board's response

Martin Raftery

THE MEDIA IMPACT

Concussion and the potential threat of associated chronic traumatic encephalopathy (CTE) have attracted unprecedented media attention. The public debate regarding the link between CTE and head injuries in sport is emotive as well as distracting. This media focus has been positive in that it has raised public awareness of concussion but the same media focus could have negative consequences by

- Reducing sports participation and undermining the health benefits of exercise;
- Forcing sports to adopt hastily developed and evidence deficient risk management strategies.

Dr Patricios and Dr Kemp have called for leadership from Rugby in this area and have also called for collision sports to unite and provide a unified, unemotive and consistent message regarding possible neurocognitive effects associated with concussion in sport.

The International Rugby Board (IRB) supports this call for collision sport unity and outlines below an overview of our risk management strategy related to concussion.

WHAT IS THE RISK?

The risk of long-term neurodegenerative illness following head injury is unknown.[1] This uncertainty is the fuel that fires the public debate. The lack of concrete evidence allows both sides to publically claim a position that is neither supported nor refuted by science.

What we do know is that a single moderate-to-severe traumatic brain injury (TBI) can lead not only to acute neurological deficits but also to long-term neurodegenerative issues, in 40–50% of patients.[2] We also know that *repetitive* head injuries in boxers and recently within other sports have been linked with long-term neurological sequelae.[3 4]

What we do not know is

Is there a link between concussion (mild TBI) and neurodegenerative disease?

At what level of repetitive head injury do these long-term neurological sequelae become evident?

Also unknown—what is the impact of individual susceptibility, mental illness, alcohol or substance misuse in the development of neurodegenerative complications following head injury?[1]

The IRB accepts that a risk is associated with head injury and concussion even if this risk is unquantifiable and unknown. As a responsible governing body the IRB will not delay action until irrefutable evidence is available but rather act prudently when evidence has been collected to an acceptable level. As such the IRB have developed a risk management strategy based on the current available evidence.

DEVELOPING CONSTRUCTIVE INTERVENTIONS

The IRB has a history of adopting an evidence-based risk management approach to assess and manage safety issues within their sport, an example being a change in scrum engagement Laws in 2007.[5] Recognising that the science around concussion is still evolving and evidence around concussion is incomplete, the IRB has based their risk management strategy on two recommendations proposed by McKee and Cantu. These strong proponents of the link between concussion and CTE suggest that the risk of CTE can be reduced by decreasing the number of concussions or mild traumatic brain injuries. They highlight that decreasing the number of concussions in sport can be accomplished by

A. Limiting exposure to head trauma;
B. Adhering to strict 'return to play' guidelines.[6]

LIMITING EXPOSURE TO HEAD INJURIES

Head contact in Rugby Union has been illegal since the inception of the game in the mid-1800s. Despite illegal contact being enshrined in the Laws of the Game, head injuries persist. The IRB acknowledges that limiting head trauma in Rugby is a key risk management strategy.

To confirm that exposure to head injury within the game has been limited as far as is practical, the IRB commissioned an independent game safety review. The goal of this review was to confirm that not only had Laws and Regulations been developed to minimise head contact events within Rugby but that these Laws and Regulations had been implemented and were being monitored and actioned. The monitoring and actioning of developed procedures is the critical component in obtaining positive outcomes from safety initiatives.

This safety review was able to confirm that Laws and Regulations had been developed, Match Officials had received directives related to prevention of head contact and that a significant component

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My perception of World Rugby's approach

1) Limit exposure to head acceleration events
2) Effective management of concussion
3) Maintain watching brief on evidence and make further changes if required

Is World Rugby’s approach to brain injuries consistent with the precautionary principle?


Proportionality
Scientific developments
Consistency
Tackle ban for school rugby?
Ban on all contact sports for under 14s
Examination of costs and benefits
?
Non discrimination

Conclusions

1) The precautionary principle does not (necessarily) imply that tackles should be banned in school rugby
2) World Rugby's position statement on CTE appears to be consistent with the precautionary principle

Conclusions

3) We need to understand more about the long-term risks **AND**

4) We need to communicate the risks to participants