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as a whole, there are public health, economic, and other consequences of excessively sanguine policies, as well as of excessively precautionary ones.

Stewart and colleagues¹ also caricature reasoned concern about CTE. No researcher we are aware of has claimed that symptoms in someone with a history of repeated head trauma "inevitably herald an untreatable, degenerative brain disease".¹ Both the primary investigations³ and the few quantitative risk assessments of CTE to date^{4,5} have been careful to explain that the very high prevalence of CTE found in case series certainly overstates, due to recruitment bias, the incidence of CTE in retired football players. However, better evidence for alternative explanations will be necessary to refute the strong link between repeated head trauma and CTE. In particular, the authors express many concerns about errors in examining patients (whether retrospectively during research or prospectively in the clinical setting) and concluding that a particular patient had or has CTE, but they only warn against false positives, not false negatives.

In any event, the association between repeated head trauma and CTE would still be strong even if some or many of the positive attributions of CTE in the various case series were erroneous.

We applaud the notion of emphasising uncertainty about causality. However, the weight of evidence should not be warped by using fallacious arguments. Stewart and colleagues¹ claim that the neuropathological changes of CTE have been reported in "apparently asymptomatic individuals".¹ Although, in our view, neither of the articles they cite show that, their statement would be unremarkable even if true. The existence of people with particular lesions (or biochemical changes) and no symptoms does not in any way cast doubt on the ability of those same lesions to cause harm in others. Similarly, reports of some persons with an exposure and without

a particular disease, or without exposure but with the disease (as in lifelong smokers who died of food poisoning, or lung cancer in non-smokers), are completely compatible with a true statistical or causal association between an exposure and a disease. These logical fallacies, and others, are clouding the CTE literature.⁶

Researchers should be able to interpret uncertain evidence differently without necessarily being accused of malfeasance. Stewart and colleagues¹ cite an essay⁷ that condemns scientists for being "willing accomplices"⁷ to media-fueled fraud, such as occurred with the debunked link between vaccines and autism. Perhaps someday scientific consensus will have reasons to reject the ominous evidence implicating head trauma in CTE. If so, it will be in spite of, not thanks to, advocacy such as from Stewart and colleagues.¹

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In 1983, US Congressman Dennis Eckart asked Robert Patterson, a doctor from the American Medical Association, "can you equate for me the impact of a blow to a boxer's head with the force of impact in another sport...?" Patterson then described in congressional testimony how the American footballer "Frank Gifford was...knocked cold for 24 hours...The blow is the same...it's small, repetitive blows...it's this cumulative effect that [leads] to the punch-drunk syndrome".¹ Patterson made scientific findings from decades of previous research palatable for Congress, but conveying evidence became harder in the following decades. As the book *League of Denial* recounts, beginning in the 1990s, the National Football League (NFL) have sought to influence public perceptions of brain injury research.² However, since at least 1983, people who have played collision sports have died with chronic traumatic encephalopathy (CTE), including Frank Gifford.

A recent Correspondence letter³ 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.⁴ 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.⁵ 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.⁶ A well documented history of what we term ignorance by design exists

in debates about the consequences of lead exposure, tobacco use, and anthropogenic climate change. In each of these cases, pressures from lobbies led to overstatement of uncertainties at the expense of public understanding of documented risks. This strategy works, at least in part, because it resonates with scientists' tendency to err towards least drama, that is, to downplay risks because of cultural norms and place a high burden of proof on the individuals making claims. It also resonates with journalistic norms of objectivity, which are seen as requiring equal weighting for both sides in a debate, even when only one side has the weight of evidence.⁷ In brain injury research, there is cultural pressure to discount risk because the sports that put people at risk are popular.

Scientists need to convey evidence and uncertainties clearly, but not at the expense of public health, particularly when the price of excessive caution is the health of children and their heroes.

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