

Introducing the International Women in Multiple Sclerosis network

In recent years there has been a conscious global commitment to address challenges related to gender parity across many industries and working environments. Within the field of neuroscience, neuroimmunology, and neurology, efforts are ongoing to promote diversity and foster innovation, both in the clinical arena and in academic venues. The International Women in Multiple Sclerosis (iWiMS) network has spearheaded meaningful change by creating a supportive and inspirational community for women and minorities working in the area of multiple sclerosis and related disorders. In 12 short months, iWiMS has grown into a group of over 250 members, representing 25 countries around the world. The iWiMS community branches across a wide spectrum of interests, drawing members from basic science through to clinical research communities, from medical and scientific trainees to specialist consultant-neurologists and senior principal investigators, as well as members working in allied health.

Our purpose is to create mentorship opportunities and foster international research collaboration, with a focus on supporting young investigators and emerging leaders. We aim to connect academics and clinicians from around the world, enabling them to share and learn from each other's scientific and clinical experience. iWiMS strives to improve overall parity and enhance the visibility of women and minorities in our field, to help them achieve leadership positions in scientific organisations and health-care settings. We also encourage participation in scientific programmes through award nominations, speakers' invitations, and journal editorships, and provide opportunities to participate in grant panels and peer review processes.

We have already made headway on a number of our goals, by establishing an executive committee that coordinates the multifaceted efforts of the group, and by creating a website that serves to communicate our objectives and activities. Moreover, the iWiMS mentorship programme was launched in February, 2019, bringing together 100 members, connecting mentors, mentees and peer supports from around the globe. We have organised our first, globally accessible, web-based epidemiology conference, allowing for all to attend without incurring costs, compromising family time, or increasing our carbon footprint. We have created a number of clinical and research-specific interest groups designed specifically to allow new clinical and academic collaborations to emerge within our global community. We have established a nomination committee tasked with identifying award opportunities and nominating candidates for these. We are proud that two iWiMS members were recently awarded major accolades including the Barancik Prize for Innovation in Multiple Sclerosis Research, and the John Dystel Prize. Furthermore, we have reached out to a number of scientific organisations to partner with them to increase the number of women on executive committees and speakers' panels, thereby starting to address gender parity, diversity, and visibility.

We encourage the entire MS community to join us in this supportive and inclusive endeavour. We believe that the iWiMS model could be easily exported to other scientific fields.

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**Vilija G Jokubaitis, Anne-Katrin Pröbstel, Georgina Arrambide, Fiona Costello, Emmanuelle Waubant vilija.jokubaitis@monash.edu*

Department of Neurosciences, Monash University, Melbourne 3004, VIC, Australia (VGJ); Department

of Neurology, Alfred Health, Melbourne 3004, VIC, Australia (VGJ); Weill Institute for Neurosciences and Department of Neurology, University of California, San Francisco, San Francisco, CA, USA (A-KP, EW); Departments of Medicine and Biomedicine, Neurologic Clinic and Policlinic, University Hospital Basel, University of Basel, Basel, Switzerland (A-KP); Servei de Neurologia/ Neuroimmunologia, Centre d'Esclerosi Múltiple de Catalunya (Cemcat), Barcelona, Spain (GA); Vall d'Hebron Institut de Recerca, Hospital Universitari Vall d'Hebron, Universitat Autònoma de Barcelona, Barcelona, Spain (GA); Department of Clinical Neurosciences, University of Calgary, Calgary, AB, Canada (FC); and Benioff Children's Hospital, University of California San Francisco, San Francisco, CA, USA (EW)

First report the findings: genuine balance when reporting CTE

William Stewart and colleagues¹ (March 1, p 231–33) offer an asymmetric “call for balance”¹ when reporting the relationship between repeated head trauma and chronic traumatic encephalopathy (CTE). They misconstrue the term “first, do no harm” as a call for inaction and the one-sided reporting of important uncertainties. By contrast, public health professionals and, we hope, most physicians understand that the scientific and ethical imperative is to reduce net harm to patients and to society, and that demands concern for the consequences of both harmful reassurances and harmful actions.² Gauging the net harm of any clinical communication or policy action regarding the risk of CTE in retired American football players requires consideration of the dangers of needless worry, but balanced against the potential harm of failing to provide patients with a complete appraisal of the likely cause of their symptoms and of the possible consequences of repeated head trauma.

For current players, the dilemma of choosing between cutting short a successful career versus sustaining additional dangerous exposures and perhaps raising their risks of a neurological disease might be an even more poignant balancing act. For society

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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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*Adam M Finkel, Kevin P Brand, Arthur L Caplan, John S Evans, Paul R Wolpe
adfinkel@umich.edu

Department of Environmental Health Sciences, University of Michigan School of Public Health, Ann Arbor, MI 48109-2029, USA (AMF); Telfer School of Management, University of Ottawa, Ottawa, ON, Canada (KPB); Division of Medical Ethics, New York University School of Medicine, New York, NY, USA (ALC); Harvard T.H. Chan School of Public Health, Boston, MA, USA (JSE); and Center for Ethics, Emory University, Atlanta, GA, USA (PRW)

- 1 Stewart W, Allinson K, Al-Sarraj S, et al. Primum non nocere: a call for balance when reporting on CTE. *Lancet Neurol* 2019; **18**: 231–33.
- 2 Sokol DK. “First do no harm” revisited: Following the dictum means balancing moral principles. *British Med J* 2013; **347**: f6426.
- 3 Mez J, Daneshvar DH, Kiernan PT, et al. Clinicopathological evaluation of chronic traumatic encephalopathy in players of American football. *J Am Med Assn* 2017; **318**: 360–70.
- 4 Binney ZO, Bachynski KE. Estimating the prevalence at death of CTE neuropathology among professional football players. *Neurology* 2019; **92**: 43–45.
- 5 Finkel AM, Bieniek KF. A quantitative risk assessment for chronic traumatic encephalopathy (CTE) in football: how public health science evaluates evidence. *Hum Ecol Risk Assess Int J* 2018; published online Apr 29, 2018. DOI:10.1080/10807039.2018.1456899.

- 6 Brand KP, Finkel AM. A decision-analytic approach to addressing the evidence about football and CTE. *Semin Neurol* 2019. DOI:10.1055/s-0039-1688484.
- 7 Moore A. Bad science in the headlines: who takes responsibility when science is distorted in the mass media? *EMBO Rep* 2016; **7**: 1193–96.

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