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Concussion in Sport: It's time to drop the tobacco analogy

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Concussion in Sport: It's time to drop the tobacco analogy

The analogy between smoking tobacco and sport-related concussions (SRCs) was initially made in a US Congressional Committee of Inquiry in 2009.[1] The inference was one of institutional malpractice, with 'big tobacco' evoked to convey concerns that the NFL had manipulated scientific evidence for its own commercial ends. Subsequently, the analogy has been used to compare the causal relationships between smoking cigarettes and lung cancer and SRCs and Chronic Traumatic Encephalopathy (CTE).[2] It is our contention that the analogy (used in either sense) detracts from the complex concerns that now confront researchers and, as such, has become detrimental to advancing the development of science and potential solutions.

The smoking analogy of causation is problematic on four counts:

1) Smoking has little to commend it in relation to health benefits (although there is evidence of psychological benefits, an association with lower body mass index, and reduced incidence of Parkinson's), while sport, exercise and physical activity is a mainstay of global public health. Efficacy evidence for the health promoting properties of physical activity is compelling, and while each sport entails a distinct health cost-benefit balance, their conflation in public health messaging belies a recognition that the promotion of sport is necessary for society to achieve population level physical activity targets.[3]
2) Cigarettes are far more addictive than exercise or sport. The addiction properties of smoking mean that those who start the earliest are likely to have the greatest exposure to harm. Consequently, laws have historically prohibited the sale of cigarettes to children and in some jurisdictions (notably New Zealand and the UK) are being extended to adults. Conversely, public health bodies are particularly concerned about the propensity of children to drop out from sport during adolescence. The detrimental characteristics of exercise addiction are rare, and offset by the health benefits of continued engagement in physical activity across the life-course.
3) The aetiology of neurodegenerative conditions and lung cancer do not easily align. For instance, while the attributable risk of cigarette smoking for lung cancer is estimated to be between 85-90%,[4] the 2020 report of the *Lancet* Commission on dementia identified brain injury in mid-life as one of 12 potentially modifiable risk factors which, combined, constitute 40% of the overall known modifiable risk.[5] The same Commission cites smoking as a more significant risk factor for dementia (5%), than brain injury (3%).

4) The social impact of lung cancer and sport-related neurodegenerative conditions are starkly different in scale. WHO rank trachea, bronchus and lung cancers sixth among the leading causes of death (and indeed smoking is a prominent risk factor for the leading three: Ischaemic heart disease, stroke and Chronic Obstructive Pulmonary Disorder).[6] *Tobacco in Australia* claims that tobacco products kill half of all long term users.[7] Conversely the risks of developing sport-related neurodegenerative conditions are closely associated with (but not necessarily limited to) one relatively small but high-profile occupational group. Indicatively studies have found 5.0% of former professional footballers[8] and 11.4% of former rugby union internationals[9] to be diagnosed with neurodegenerative disease. Even amongst those who had fought for world heavyweight boxing titles, deaths from neurological disorders were found to be 15.5%.[10] While exposure to the highest levels of risk from cigarette smoking is open to almost all of the 1.14 billion smokers around the world,[11] only a tiny proportion of the world's population will ever become elite athletes and therefore most at risk of developing sport-related neurodegenerative illness.

We recognise that analogies are as much about invoking comparison as they are about precision. But the consequences of the use of the smoking analogy of causation are to oversimplify what is, in reality, a field of increasing complexity. For example, SRC researchers are no longer simply concerned with whether concussion causes CTE because evidence from former professional footballers has led researchers to suggest that longer-term neurodegenerative conditions primarily relate to the asymptomatic consequences of heading or subconcussive impacts.[8, 12] Not only have advocates of the smoking analogy begun to broaden the focus from concussion to repetitive head impacts (RHIs) (see e.g. Reference 2) concerns about outcomes have also widened from CTE to include Parkinson's, Motor Neuron Disease, Alzheimer's and other dementias. Evidence across these studies suggests that the heightened risk of these individual conditions varies, that the risk of Parkinson's may be reduced,[13] and that the risk of all-cause mortality may also be reduced.[9, 13] In contrast to smoking where human action, pathology and symptoms relatively closely align, even research seeking to establish a causal relationship between RHIs and CTE recognise the different challenges of linking CTE neuropathology with clinical symptoms.[2] Thus, while the science of smoking has continually highlighted the magnitude of a single causal factor (tobacco smoking) for a primary health outcome (lung cancer), the science examining the relationships between SRCs and neurodegenerative disease outcomes has become increasingly multi-faceted. The mounting evidence suggests that the time is right to broaden rather than narrow the parameters of the scientific enquiry into the causes of

neurodegenerative disease among athletes. The smoking analogy of causation works against this.

We acknowledge that the tobacco analogy of institutional malpractice has been an effective device for mobilizing critical voices and challenging the status quo. However, the original, institutional, orientation of the smoking analogy is problematic because, since 2009, SRC has become an international, cross-sport concern in which multiple governing bodies of sport have initiated their own and varied responses to the concussion crisis. Regardless of whether the NFL acted like 'big tobacco' as originally claimed in the 2009 Congressional Committee of Inquiry,[1] it is our view that 'big sport' is patently not monolithic in its response to SRC and longer term neurodegenerative conditions. For instance, the first study to established heightened risk among former professional football players was part-funded by the Football Association.

Moreover, in lacking precision and reducing complexity the smoking analogy of causation misses the primary function of analogy - i.e., to provide better understanding. Rather than pursue a single causal factor for one health outcome, the scientific advancement of the field – as set out, e.g. by the US Centers for Disease Control and Prevention - rests on discovering the 'potential risk factors for CTE, including the role of a person's sex, genetics, medical history, and environmental and lifestyle factors'; how the disease process begins; and 'Why some people with a history of long-term exposure to repeated head impacts develop CTE and others do not'.[14] Moving beyond the reductionist hypothesis of a single cause-effect, we must consider the heterogenous group of conditions and mechanisms, and ask how symptomatic and asymptomatic brain impacts in a particular occupational context, with a particular set of social and economic professional relations fosters a particular culture of risk, attitudes and behaviours. *The time for politically motivated analogies has now passed.* The development of coherent and pragmatic solutions requires effectively describing and theorising the complexity of the problems faced by an increasing range of sports people and sports institutions.

Competing interests

None

Contributorship

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Statement of interests

There are no competing interests

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