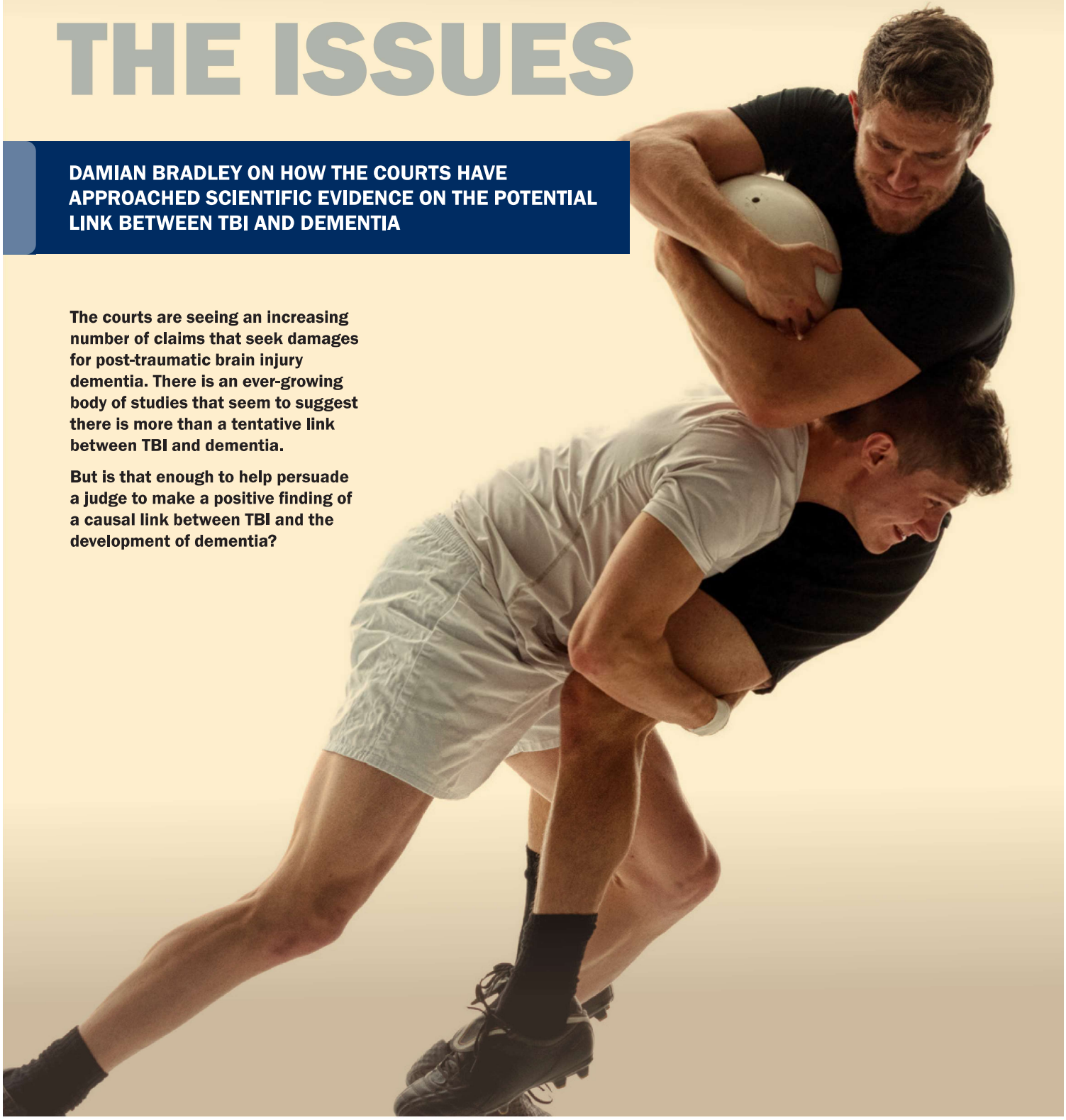
TACKLING THE ISSUES

DAMIAN BRADLEY ON HOW THE COURTS HAVE APPROACHED SCIENTIFIC EVIDENCE ON THE POTENTIAL LINK BETWEEN TBI AND DEMENTIA



The courts are seeing an increasing number of claims that seek damages for post-traumatic brain injury dementia. There is an ever-growing body of studies that seem to suggest there is more than a tentative link between TBI and dementia.

But is that enough to help persuade a judge to make a positive finding of a causal link between TBI and the development of dementia?

Examining the research

The concept of claiming provisional damages for post-traumatic brain injury dementia was introduced in *Mathieu v Hinds (1 Hinds) (2 Aviva Plc)* [2022] EWHC 924 (QB). In *Mathieu*, the claimant suffered TBI on a pedestrian crossing, after being struck by the defendant on a stolen moped.

The claimant was successful in that the Court ordered provisional damages for the claimant's lifetime with regard to the chance of his developing epilepsy. However, the claim for provisional damages for alleged increased risk of developing dementia due to TBI was unsuccessful.

Mrs Justice Hill confirmed that the claimant needed to prove that there was 'more than a fanciful chance' that the TBI would cause him dementia in the future; and that 'as a matter of generality the claimant was required to prove that a single TBI could cause dementia and such risk applied to him.'

In this case, the claimant made an excellent recovery from the TBI and was in the top 1% of patients with his level of brain injury; and so any general risk that might exist was likely to be lower in the claimant's case.

The range of studies before the court exploring the link between a single TBI and dementia were viewed as being in a mess, and the court was confounded by some of the conclusions reached. The court also relied on the Hicks meta-analysis study from December 2019, which criticised certain individual studies as being difficult to interpret and pointed to some weaknesses in the research. Criticism was made of self-reporting, the application of very small cohorts providing a very restrictive view, and an overreliance on the analysis of veterans.

The court did accept that there may be an association between a single TBI and dementia, but science did not particularly assist with providing a clear understanding of the causative route.

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A recent case

On 4 April this year a new decision on TBI-related dementia was handed down, in *The Executors of the Estate of the Late Geoffrey Charles Ivory v Swale Borough Council*. Here, the late Mr Ivory, aged 82, had fallen in a car park on 14 May 2014 after tripping over a raised kerb. He sustained injuries to his head and face.

There was no loss of consciousness recorded, and although dazed, he was discharged home the same night. In July 2014 Mr Ivory attended hospital with symptoms of a seizure and was later diagnosed with an acute on chronic subdural haematoma. Sadly on 25 January 2020 Mr Ivory died, and his executors took over proceedings.

The evidence for the case largely relied on history of family members as to the health, level of mobility and activities that the deceased was able to undertake prior to the head injury.

The picture painted by the family members was that Mr Ivory had been extremely active, had no health issues, and that he was able to undertake his own gardening. Since the accident his health had deteriorated and in particular, he underwent a step change decline in cognitive functioning after the seizure of July 2014.

At Trial this evidence was left in tatters. It emerged that Mr Ivory was suffering from several pre-accident complaints including diabetes, Chronic Obstructive Pulmonary Disease (COPD), glaucoma, mobility problems; and it had been suggested that he undergo a test for dementia, but he declined. It was also shown that Mr Ivory was paying a gardener to tend his garden.

A key factor was the dispute regarding the nature of the head injury. The defendant denied there was a TBI at all, as the main injury was to the cheek and there was no evidence of classic TBI symptoms.

Judge Parker determined that there was no loss of consciousness, and there was mild TBI at most. He also found that there was no step change in cognitive decline in July 2014 and no post-traumatic epilepsy.

The claimant's neurologist Dr Allder was cross-examined on the scientific research, and he conceded that 'the mechanisms by which TBI generates dementia are still being worked out'.

The Judge concluded that 'the current state of research does not establish that either a single mild TBI or a chronic subdural haematoma is likely to cause or accelerate dementia.

'The additional feature relied upon by the claimant – post-traumatic epilepsy - has not been established on a balance of probabilities to be associated with, still less causative of dementia.'

Part of the scientific research relied on by the claimant's neurologist was carried out by Dr Shively. This was a meta-analysis of 15 studies that all intended to assess whether patients who had sustained head injury resulting in loss of consciousness were at greater risk of dementia.

In her analysis, Dr Shively opines that the 'best data indicate that moderate and severe TBIs increase risk of dementia between 2 and 4 fold. It is less clear whether mild TBI, such a brief concussion results in increased dementia risk, in part because mild head injuries are often not well documented and retrospective studies have recall bias.

'However it has been observed for many years that multiple mild TBIs as experienced by professional boxers are associated with a high risk of chronic traumatic encephalopathy (CTE), a type of dementia with distinctive clinical and pathologic features.'

Dr Shively also reports that 'another line of evidence indicating that neurodegeneration after TBI shares some features with AD (Alzheimer Disease) comes from imaging studies. Cerebral atrophy after TBI is not diffuse, but rather regionally selective.

'The regions of the brain that show most prominent atrophy after TBI, such as the hippocampus, amygdala, precuneus and parietal and frontal cortices, overlap closely (but not perfectly) with regions of predominant βamyloid deposition, decreased glucose use, and progressive atrophy in AD.

'These findings may relate to common molecular mechanisms shared between AD and TBI related neurodegeneration.'

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Scandinavian studies

It is surprising that neither the *Mathieu* case nor the *Ivory* case sought to adduce the nationwide cohort study of Nordström.

This research published in 2018 comprised all individuals in Sweden aged 50 years and above on 31 December 2005; amounting to over 3.3 million people. It tracked nationwide databases from 1964 until 31 December 2012 with diagnoses of dementia and TBI.

Similar to the findings of Dr Shively, single mild TBI showed a weaker association with dementia. The study concluded that the risk of dementia diagnosis risk decreased over time after TBI, but is still evident over 30 years after the trauma. The association was stronger for more severe TBIs and multiple TBIs, and it persisted after adjustment for familial factors.

There is also a study undertaken by Fann and Others in 2010 for Denmark with a cohort of almost 2.8 million which had similar findings. An additional marker was that the risk of dementia was highest in the first six months after TBI, with a hazard ratio of 4.06.

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The need for more research

Clearly, there is scope for further scientific studies to provide more clarity and assistance to the courts, enabling claimants to bring these types of claims for damages and provisional damages. As is evident from Dr Shively and others, there is the possibility of enhanced scanning technology that could assist in pinpointing the TBI-related neurodegeneration.

As technology continues to improve and regular scanning takes place of the population at large, it is likely that a more comprehensive radiological history can accurately plot the neurodegeneration and help prove any step change that is alleged.

The cases that have been put before the courts so far have had their deficiencies. A model case will be needed in order to prove the causative link between TBI and dementia. It is clear that neurological opinion alone is not enough to sway the judge. Any appropriate cases will also need the support of an epidemiologist and proper scrutiny of all appropriate studies to be relied on.

As for the next opportunity for the court to consider these issues; there is the group action involving England World Cup winner Steve Thompson, who together with 200 other sportspersons is bringing claims against World Rugby, the Rugby Football Union and the Welsh Rugby Union. These claims are for permanent brain damage due to concussions and sub-concussive injuries suffered during their playing career.

This type of approach was successful in a US class action against the NFL, achieving a settlement of circa $1 billion. Although CTE can only be diagnosed post-mortem, the history of exposure and increasing risk can be evaluated, and the scientific development research around the issues can pave the way for successful claims in the future.

If science can progress to the next level, where there is an established causal consensus between TBI and dementia - as has been achieved with TBI and post-traumatic epilepsy - then the courts must sit up, take note and be guided by such scientific developments when determining these types of claims.

Damian Bradley is a partner in the employer's liability team at Express Solicitors

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