Written Testimony

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Hearing before the House Judiciary Committee

Legal Issues Relating to Football Head Injuries

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Mr. Chairman and Members of the Committee:

Thank you for the invitation to testify today on legal issues relating to football head injuries. My name is Dr. Ann McKee. I am an associate professor of Neurology and Pathology at Boston University School of Medicine, and I am the Director of the Neuropathology Laboratory for the New England Veterans Administration Medical Centers at the Bedford VA Medical Center, the Director of the Brain Banks for the Boston University Alzheimer's Disease Center, the Framingham Heart Study, and the Centenarian Study, and I am a co-director for the Center for the Study of Traumatic Encephalopathy at Boston University.

I received my medical degree in 1979, and I am board certified in both Neurology and Neuropathology. I have extensive experience in neuropathology of neurological disease and have written extensively on the neuropathology of many neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, and Frontotemporal Dementia, as well as normal aging. For the past 23 years, I have been studying the brains of individuals after death and correlating the pathological findings to the patient's clinical symptoms during life. I have examined thousands of brains, brains from people in all walks of life including brains from individuals who have lived to be well over the age of 100. In addition, for most of my professional career, I have been focused on *tau* protein, a protein that becomes toxic when abnormally phosphorylated and builds up in the brains of patients with some neurodegenerative diseases, including Alzheimer's disease, but is found only in very limited quantities in the brains of normally functioning people.

In January of 2003, as part of my work with the Boston University Alzheimer's Disease Center and the Bedford VA, I examined the brain of a man who died at the age of 72 after 15 years of severe dementia requiring institutionalization. The man had been a

world champion boxer and had been clinically diagnosed with Alzheimer's disease beginning at the age of 58. However, when I looked at his brain on post-mortem examination, I found that there was absolutely no evidence of Alzheimer's disease; there was no evidence of *beta amyloid*, a protein that accumulates in the brain in people with Alzheimer's disease and is thought by many to be the cause of Alzheimer's disease. Instead, the brain of this world champion boxer showed a massive build-up of the toxic form of tau protein as neurofibrillary tangles (NFTs) and glial tangles throughout his brain. The neurofibrillary and glial tangles were also distributed in a unique pattern, a pattern not found in any neurodegenerative condition except Chronic Traumatic Encephalopathy, or CTE. In CTE, tau protein builds up in individual nerve cells and prevents them from making normal connections with other nerve cells, eventually killing the cells. In this man's brain, there were massive numbers of NFTs and glial tangles, so many in fact that you could see the abnormalities on the glass slides without the use of a microscope, as you can see in the right panels of Figure 1. There is tremendous accumulation of tau protein that appears as a brown pigment. All the brown pigment you see is abnormal, please compare what you see on the right to the brain of a normal 65 year old man on the left, all the slides are prepared and stained in exactly the same way, and there is absolutely no brown pigment visible in the normal individual. When you look at the brain microscopically as in the lower panels, you can see that many individual nerve cells of the boxer contain NFTs – they are found in nearly every nerve cell and there are almost no normal appearing cells. This individual, a former professional boxer, was clinically diagnosed with Alzheimer's disease during life, but the disease that actually caused his tragic 15 year decline in intellect and eventually killed him, was CTE, a disorder that would have been entirely prevented if he hadn't suffered repeated head injury in his younger years as a boxer.

My second case of CTE came in 2004, again when I was examining the brain of a man who had been clinically diagnosed with Alzheimer's disease when he was alive. When I looked at the slides, I immediately realized that the changes found in this individual were nearly identical to those that I had found in the world champion boxer, but in this case, the medical records did not indicate that he had ever had any head injury. So I called the patient's daughter, and sure enough, it turned out that the man had been a professional boxer during his twenties. Again, his post-mortem examination indicated that his functional deterioration, dementia and placement in a nursing home were not due to Alzheimer's disease, but instead due to CTE, a disorder that could have been entirely prevented. Over the ensuing years, I examined several other cases of CTE in professional boxers, all with a similar appearance and pattern of abnormalities.

I met Chris Nowinski in the summer of 2007 and through Chris's efforts in early 2008, I had my first opportunity to examine the brain of a retired professional football player. It was the brain of John Grimsley, a former linebacker for the Houston Oilers who had died of an accidental gunshot wound while cleaning his gun at the age of 45. According to his wife, he was concussed 3 times during his college football years, and at least 8 times during his NFL career, however, only one "cerebral concussion" was medically confirmed. He was never formally diagnosed with post-concussion syndrome and never sought medical attention for residual cognitive and behavioral difficulties. There was no history of ever losing consciousness for more than a few seconds and he never required being carried off the field or hospitalization. He never took any performance-enhancing drugs or used illicit drugs. He was a nonsmoker and there was no known family history of dementia.

According to his wife and close friends, he began showing changes in his behavior and cognitive decline at age 40. He developed difficulties in short-term memory, attention, concentration, organization, planning, problem-solving, judgment, and the ability to juggle more than one task at a time. For example, he would ask the same questions repeatedly over the course of the day and he would ask to rent a movie that he had already seen. He had difficulty assembling his tax records, shopping alone, and understanding television. His symptoms gradually progressed and became quite severe by the end of his life. He also developed a "shorter and shorter fuse" and would become angry and verbally aggressive over seemingly trivial issues.

When I first looked at his brain (it had been previously dissected by the coroner), I didn't see any gross changes. Yet when the microscopic slides were prepared, they showed the exact same pattern of changes that I had found in the brains of the boxers with CTE. There were large numbers of tau containing neurofibrillary tangles throughout all parts of the brain and there was absolutely no evidence of beta amyloid protein or Alzheimer's disease. The brain of this 45 year old husband and father, at the prime of his life, showed profound neurofibrillary degeneration, changes of CTE that were identical in nature to the changes I found in the brains of the boxers, but were now in a football linebacker some 30 years younger. In John Grimsley's brain, there were striking changes in regions of the brain controlling personality and behavior, such as the frontal lobes, profound changes in the areas controlling impulsivity and rage behavior such as the amygdala, and severe changes in anatomic structures that are responsible for memory, such as the hippocampus, mammillary bodies and thalamus. In Figure 1, the brain of John Grimsley is seen in the middle; in the top middle panel, you can see severe tau deposition in the frontal lobe and microscopically; in the bottom middle panel, you can see numerous nerve cells containing tau and NFTs.

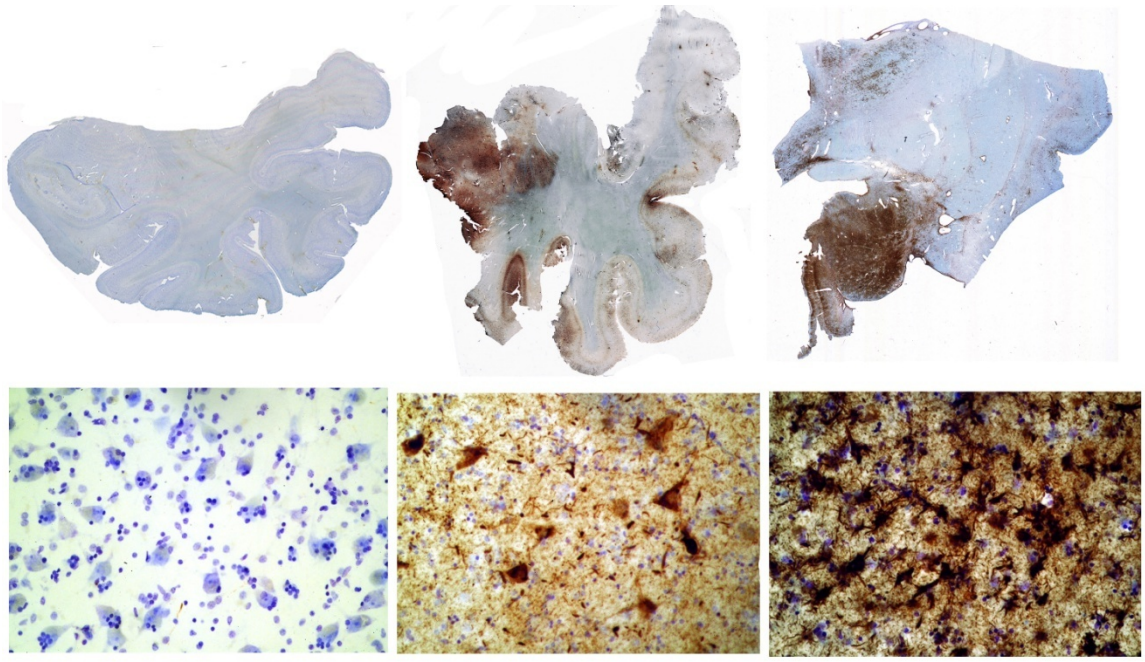

Figure 1

In a normal 45 year old, absolutely none of these changes would be found. Indeed these changes would not be found in a normal 65 year old, 85 year old or 110 year old.

The next football player's brain that I examined was that of Tom McHale, a 45 year old retired offensive lineman for the Tampa Bay Buccaneers. He was a husband and father of 3 young boys. After a 3 year decline in his ability to make sound business decisions, increasing apathy, depression, and memory loss, he died as a result of substance abuse. His wife did not know of any reported formal concussions during his year as a lineman. His brain too showed profound tau immunoreactive neurofibrillary degeneration in areas controlling memory, impulsivity, organization and problem solving (as you can see in Figure 2) and again with no evidence of any other disorder other than CTE.

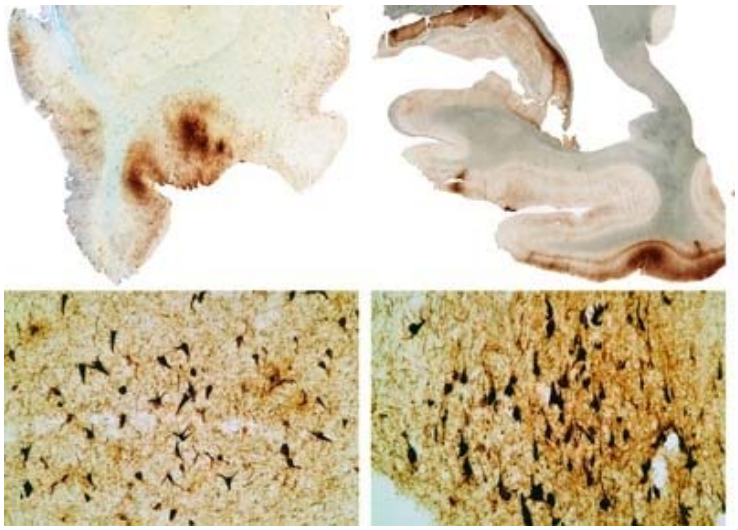

Figure 2

The third brain of a professional football player I examined was that of Wally Hilgenberg, a 66 year old former linebacker who died from complications related to Amyotrophic Lateral sclerosis or Lou Gehrigs disease. He played 16 seasons with the Minnesota Vikings and had at least 10 concussions, including losing consciousness on 1 or 2 occasions. He began showing slow and steady cognitive decline at the age of 56. His cognitive difficulties were manifest mainly by "not understanding things at a deeper level" and he had difficulties with executive functioning, including worsening organization and

planning skills. His cognitive decline progressed with worsening memory and language functions. In his last year, he stopped being able to read and was completely unable to learn how to operate an assistive communication device, even using the simplest level of commands. Inspection of his brain showed damage to the frontal cortex in a pattern that suggested it had been used as a battering ram, and the interior spinal fluid spaces were enlarged suggesting that the volume of the brain had declined. Microscopically the brain was densely riddled by tau containing NFTs and glial tangles throughout the cerebral cortex, basal ganglia, thalamus, and brainstem in the unique pattern that defines CTE, and again, in the complete absence of Alzheimer's disease and beta amyloid. Furthermore, the damage found in his brain was far greater in density and the damage was much more widespread than anything that I have ever found in Alzheimer's disease or any of the other common neurodegenerative disorders.

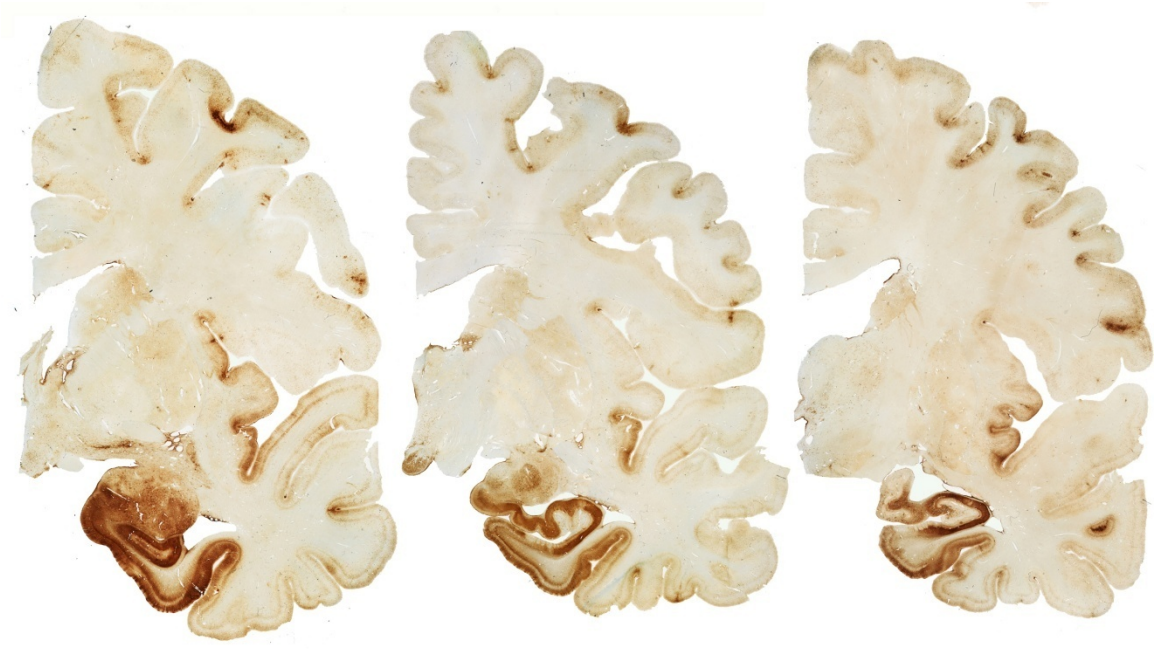

Figure 3

The fourth, fifth and sixth brains from former NFL football players that I examined, including one individual who took his own life, all showed the same distinctive, characteristic changes of CTE. The seventh brain of a former NFL player I analyzed was that of Louis Creekmur, a former offensive lineman for the Detroit Lions and an eight-time Pro Bowler. Louis Creekmur played ten seasons for the Lions, and was famous for suffering at least thirteen broken noses and 16 concussions. Beginning at the age of 58, he began to show increasing cognitive and behavioral difficulties including memory loss, problems with attention and organization, and outbursts of anger and aggression. He died from complications of dementia at the age of 82. The brain of Mr. Creekmur showed extensive damage including marked shrinkage of medial temporal lobe structures that control memory, shrinkage of the frontal and temporal lobes, and marked dilation of the spinal fluid cavities that line the brain's interior. There was widespread and severe tau deposition as NFTs throughout the frontal and temporal lobes, amygdala, hippocampus, thalamus and brainstem in the unique pattern that is only found in CTE. In Mr. Creekmur's case, the abnormalities were profound, they were severe, and they paralleled the changes found in the world champion professional boxer. Mr. Creekmur was also a member the NFL's Plan 88. Yet again, there was absolutely no evidence of Alzheimer's disease or any other neurodegenerative disorder, and the findings indicated that if Mr. Creekmur had not sustained repetitive head trauma during the play of football, he would be alive and well and enjoying his family and grandchildren today.

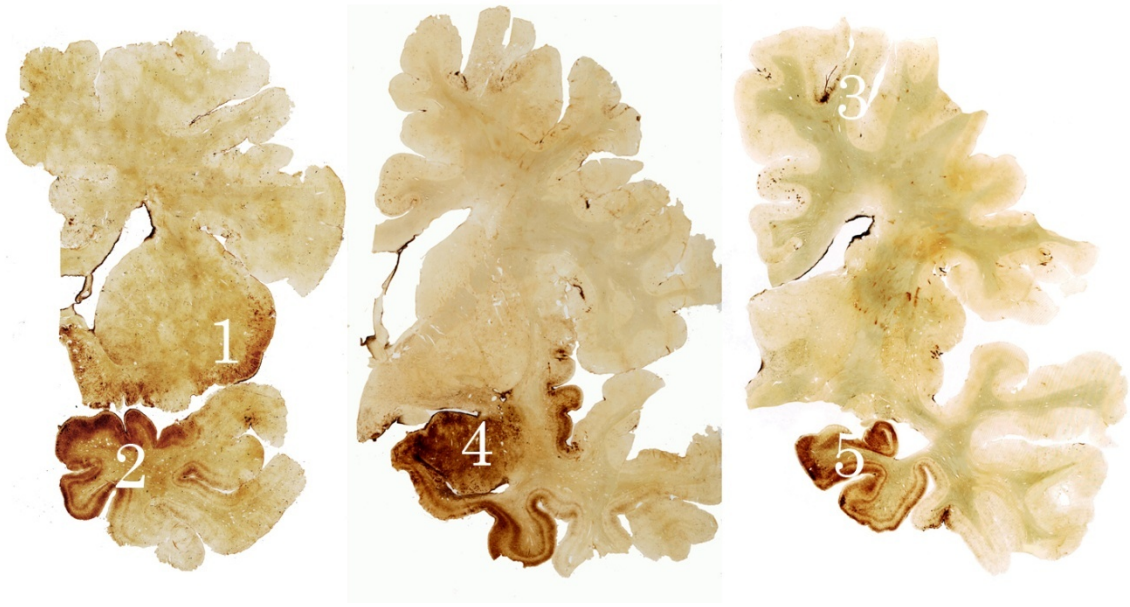

Figure 4

I have also examined the brain of a former college football player, Mike Borich, a former wide receiver for Snow College and Western Illinois University who died at the age of 42 after a several-year period of increasing irritability, aggressive and violent outbursts, and drug and alcohol abuse. His brain, too, showed CTE affecting widespread parts of his cerebral cortex and deep brain nuclei. Brains from 3 other college football players showed similar changes.

Lastly, I have had the opportunity to examine the brain of a high school football player who died at the age of 18. He had played football and other sports for 4 years and suffered several concussions. The brain of an 18 year old should be pristine, there should be no abnormalities anywhere, no abnormalities whatsoever. But in the brain of this young man, a brain that should be entirely normal, there were spots of undeniable pathology. They were 4 areas of damage in the frontal lobe that you could see even looking at the slides with your naked eye (Figure 5). In those areas, there were hundreds of degenerating nerve cells containing tau neurofibrillary tangles and disordered nerve

cell processes. Even in this 18 year old high school student, with only a few years experience playing football, there were signs of the earliest stages of CTE. Had he lived longer, this 18 year old would have almost certainly developed the same full blown CTE that we found in the other college and professional football players.

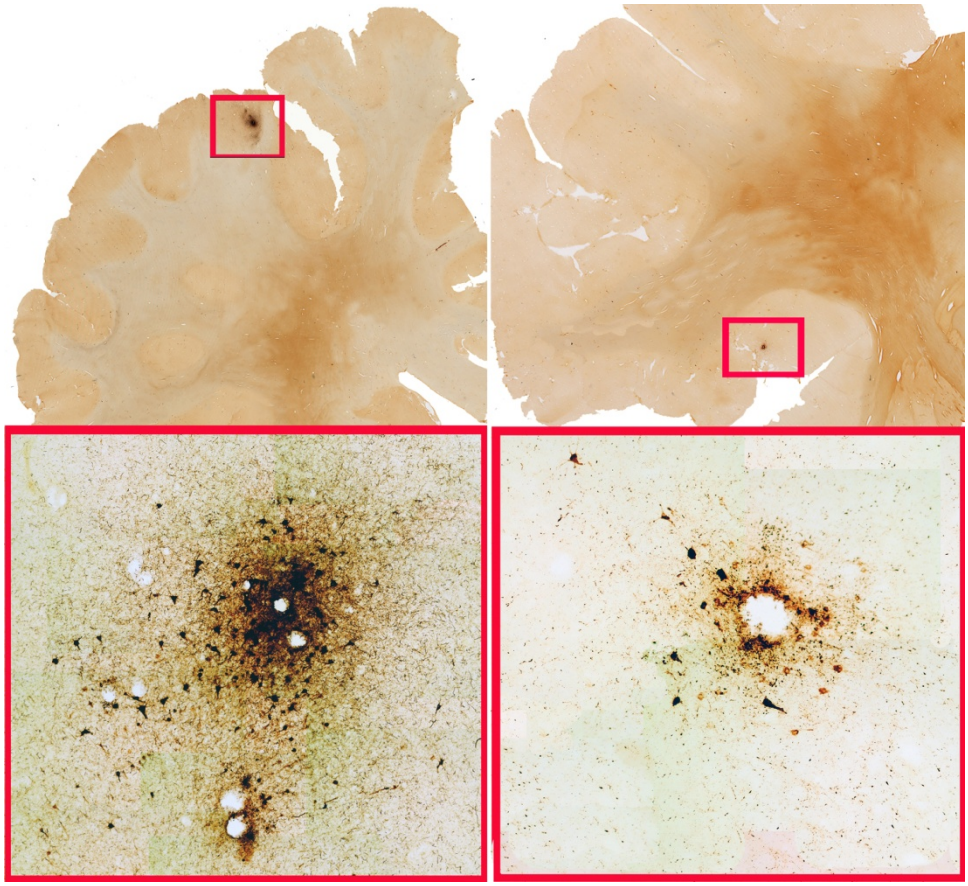

Figure 5

I have now examined the brains of 7 former NFL players, and 4 college layers, and all 11 have shown profound and widespread changes of CTE. I have also found CTE in a college level player and the earliest signs of CTE in a high school football player. I realize that this is just a handful of cases, so – so what? -what can you say from just 11 cases? Well, I can say that for the past 23 years, I have looked at thousands of brains, from individuals from all walks of life, of all ages, and during the past 20 years, I have primarily focused on abnormalities of tau protein. But I have only seen this unique

pattern of changes, in this severity, in individuals with a history of repetitive head trauma, including boxers and football players. These changes are dramatically *not normal* -there is no way these pathological changes represent a variation in normal that we find under a bell shaped curve. We have found these changes in every professional football players' brain that has come into my laboratory at the BU Center for the Study of Traumatic Encephalopathy and I have never seen this in 20 plus years of examining brains. I have had colleagues of mine from other institutions – leading neuropathologists from Harvard and Mt. Sinai—independently examine these brains, and they have come up with the same diagnosis as I had, CTE. I know that the argument is often made that there are hundreds of thousands of former football players, including former professional football players, with no signs of any cognitive decline or memory loss or personality change, but what I don't understand is why are we expecting that this exposure to repetitive head trauma will have 100% penetrance into the population and cause disease in every football player? Do we expect 100% of cigarette smokers will develop lung cancer? Do we expect 100% of children who play with matches or even chain saws will get hurt? No. Even if the percentage of affected players is 20%, or 10%, there are still thousands of kids and adults out there, right now, *playing football at all levels* -who will eventually come down with this devastating and debilitating disorder. And as a doctor and as a mother, I think this calls for immediate action. We need to take radical steps to change the way football is played and we need to make those changes today.

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Co-Principal Investigators: Ann McKee and Robert Stern

Type of Grant: Supplement to P30 Center Grant (N. Kowell, P.I); P30-AG13846

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Co-Principal Investigators: Ann McKee and Robert Stern

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