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FIELD HEARING

LEGAL ISSUES RELATING TO FOOTBALL HEAD INJURIES, PART II

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WRITTEN STATEMENT

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MIKE WEBSTER, THE FATHER OF CHRONIC TRAUMATIC ENCEPHALOPATHY [CTE]

Good afternoon Chairman Conyers, Ranking Member Smith, and members of the Committee.

I discovered the first case of footballer's dementia in Pittsburgh Steelers Hall of Famer Mike Webster. I performed an autopsy on Mike Webster in 2002 when he died suddenly at the age of 50. Mike Webster's life after retirement from football was marred by progressive symptoms of dementia, major depression, mood disorders, drug abuse and violent/criminal tendencies.

Surprisingly his brain at autopsy appeared normal by naked eye examination. In spite of his brain appearing normal, I performed extensive tissue analysis of his brain using sophisticated tissue technology, which revealed a unique type of dementia. This instigated my definition of a new disease, which I called Chronic Traumatic Encephalopathy [CTE].

Prior to Mike Webster, there was no defined disease entity in football players known as CTE. The lesson Mike Webster taught us was that brains of contact sports athletes may appear normal by naked eye examination, by routine radiological examination using computerized axial tomography [CT] scans or by routine magnetic resonance imaging [MRI]. At this juncture, CTE can only be diagnosed definitively by tissue analysis at the cellular level using specialized proteomics technology. Frequently sufferers of CTE are told by their doctors that they do not have any substantive clinical evidence of dementia to support a confirmatory diagnosis of footballer's dementia.

THE BRAIN INJURY RESEARCH INSTITUTE [BIRI] AND CTE

Since Mike Webster, my colleague and I, Dr. Julian Bailes, Chairman of the Department of Neurosurgery at West Virginia University School of Medicine, have examined over twenty brains of amateur and professional contact sports athletes. We have done so as part of the group we co-founded: the Brain Injury Research Institute, based at the West Virginia University School of Medicine.

Our cohort at this time, in part includes three high school football players, eight professional football players, four professional wrestlers, one professional mixed martial arts fighter and two professional boxers. We have identified incipient CTE in an 18 year old high school football player. We have also identified CTE in two WWE wrestlers. We have examined the brains of a variety of subjects of repetitive brain injuries ranging in age from 2 years old to 89 years old. I have also identified CTE changes in the brain of a Vietnam War veteran diagnosed with lingering post traumatic stress disorder.

At the Brain Injury Research Institute, we have identified subtypes of CTE, which we expect will be published soon in a top scientific journal. By the end of 2010, we expect to have published eight papers on CTE, presenting to the scientific community and the general public what we have deciphered thus far on the disease, how doctors can diagnose it and how families can recognize the symptoms as they emerge.

Perhaps more importantly, I am very happy to announce that my colleagues and I at the Brain Injury Research Institute are currently engaged in research on treatments for this condition. These include

moving forward on studies of possible pharmacologic means for preventing and even curing CTE. We are also researching on a genotypic distribution of CTE sufferers to possibly identify a genetic proclivity for CTE.

Dr. Julian Bailes, a preeminent neurosurgeon and director of BIRI, is leading the expansion of our research efforts in evaluating, diagnosing, monitoring and advising living amateur and professional football players, who may be suffering from CTE, with the objective of developing clinical interventions for CTE. We need the support of the federal government, state governments, other governmental agencies, foundations and individuals to continue advancing this field of study, since the sequelae of sub-concussions and concussions are emerging as major public health threats of the 21st century.

CTE, SUB-CONCUSSIONS AND CONCUSSIONS

The concept of permanent brain damage and dementia following repeated blows to the head is a very well established and generally accepted principle in medicine. The first cases of dementia and brain damage in contact sports athletes were first described in boxers and the disease was named dementia pugilistica. Dr. Harrison Martland, a forensic pathologist, like myself, and the chief medical examiner of Essex County, Newark, New Jersey, described dementia pugilistica in 1928. However, it was not until we examined the brain of Mike Webster in 2002 did we identify the tissue evidence of a similar disease in football players, which we have named CTE.

When I brought these findings to the attention of the NFL and the WWE, their responses were not supportive, to say the least; which is why I applaud the work of Chairman Conyers and this Committee. It is clear to me that without your intervention, there would have been no meaningful action to address the safety of athletes.

Because as we all know, the current issue at stake is not the debate whether CTE exists, but what we can all do together to help protect athletes and eventually develop a cure for CTE. I would laud the NFL for their recent pronouncements on CTE, which I must say were very much more encouraging than their pronouncements in 2002, 2005 and 2006, when we identified CTE in Mike Webster, Terry Long and Andre Waters. However, we must not make the mistakes of the past. Our approach and management of the sequelae of repetitive brain trauma in sports must be based on sound scientific principles interpreted by knowledgeable doctors who are adequately credentialed in sports medicine and neurological medicine.

The current focus has primarily been on concussions, while we are not yet paying enough attention to sub-concussions. There is a broad spectrum of acceleration-deceleration injuries to the head; unfortunately a concussion may represent one end of the spectrum. Sub-concussions or blows to the head, which may not manifest with immediately incapacitating symptoms and signs are equally as important as concussions in the patho-etiology of CTE. For every one documented concussion, there may be tens to hundreds of sub-concussions. Hundreds to thousands of sub-concussions can equally result in permanent damage to the brain cells on the cellular level in at least a proportion of the population. We know that only about 20% of amateur and professional boxers will manifest the most severe forms of CTE/ dementia pugilistica with multi-domain and global impairment of brain

functioning. A much larger proportion of individuals, we believe, will manifest less severe forms of CTE with single to several domain impairment of brain functioning like memory problems, mood disorders and major depression.

Concussions and Return to Play Guidelines

We must also consider the neuropathology of traumatic brain injury on the cellular and sub-cellular levels while developing models for the management of concussions in sports. Concussions to the brain cause cellular injuries to the cell membrane and cytoskeleton. These injuries result in the upregulation of specific genes, and the accumulation of certain types of proteins and peptides in the brain, for example Amyloid Precursor Protein [APP]. APP begins to accumulate noticeably in brain cells and nerve fibers about one to three hours following a concussion. APP disappears about three months following a concussion.

In considering this specific piece of neuroscientific information, one may question the basis for keeping a concussed player out of play for anytime shorter than three months. While a concussed player may be symptom free on the gross functional level after several weeks of sustaining a concussion, APP accumulation in the brain tells us that the player's brain may not have recovered after several weeks. The big question, we should then ask ourselves, based on the science, is whether one week, two weeks, or even several weeks are sufficient post-injury intervals for a player to return to play?

Concussions and the Brains of Children

When we are born, our brain weighs about 350 grams; at about one to two years of age, the brain attains about 75% of the adult size. The brain reaches 90% of its adult size at the fifth year and 95% by ten years old. The brain attains adult size by the seventeenth or eighteenth year largely due to continued myelination of nerve fibers. Before the age of eighteen, the human brain remains a developing brain.

Expectedly, injury to the developing brain of a child is more likely to result in more deleterious and more serious adverse outcomes than the developed adult brain. This means that the brains of children who play football are more vulnerable to the repeated impacts, sub-concussions and concussions intrinsic to the game of football. This means that a child who plays football may be precluded from attaining the full capacity of his cognitive and intellectual functioning as an adult. How can we translate and apply this piece of neuroscientific information to the administration and management of football in children?

Understanding the Pathology of CTE, Concussions, and Sub-Concussions

A forensic pathologist described dementia pugilistica in boxers. Another forensic pathologist described CTE in football players and wrestlers. The works of these two forensic pathologists underscore the vital role forensic pathologists and medical examiners play in further elucidating CTE and the effects of concussions and sub-concussions. One of the greatest problems we have encountered at BIRI is the

reluctance of forensic pathologists to save whole brains for comprehensive examination with the misunderstanding that tissue analysis for CTE diagnosis is primary research and not disease diagnosis.

Tissue analysis for diagnosis of CTE in the brain of a deceased athlete is similar to tissue analysis for diagnosis of Alzheimer's Disease in the brain of any deceased individual. Diagnosis of Alzheimer's Disease is not primary research and does not require the consent of any next-of-kin, and similarly diagnosis of CTE should not be classified as primary research and should not require the consent of any next-of-kin. The judgment and decision whether to perform tissue analysis for CTE diagnosis should be that of the forensic pathologist performing an autopsy as part of differential diagnosis and determination of cause and manner of death.

Forensic pathologists should be encouraged, possibly through legislation, to become more proactive in routinely identifying and diagnosing CTE in all autopsied deceased contact sports athletes. This practice can only generate more independent and objective data, which will enable multidisciplinary development of preventive and curative interventions for CTE and other types of sports related brain injury sequelae.

BIRI, NFL, Prevention and Possible Cure for CTE

Clinical bio-markers

Currently, there are no diagnostic clinical bio-markers for CTE, concussions or sub-concussions in the living patient. We should focus on developing a battery of objective quantitative bio-makers in the blood for sub-concussions, concussions and CTE in clinical pathology. Decades ago we did not have specific bio-makers for heart attacks; today fortunately, we have a bio-maker called Troponin-I for heart attacks. If your Troponin-I blood level was elevated beyond an established threshold it would be clinically diagnostic of heart muscle necrosis [heart attack]. Similarly if your prostate specific antigen [PSA] blood level was elevated beyond set thresholds, it would also be diagnostic of hypertrophy [enlargement] of the prostate and/or prostate cancer. These are all high through-put tests, which can be performed within minutes in very efficient analytical systems.

There is no reason why similar models cannot be developed for sub-concussions, concussions and CTE for the diagnosis, monitoring and management of traumatic brain injury in football and in all types of contact sports. Bio-makers would be superior, more efficient, more objective, more reproducible and more quantitative diagnostic tools than neuropsychiatric testing. While neuropsychiatric testing is useful, we should not stop at neuropsychiatric testing. We should work collaboratively and relentlessly in applying the science of proteomics to developing diagnostic bio-makers and diagnostic algorithms, which are specific for CTE and the sequelae of concussions and sub-concussions in sports.

Proteomics, peptides, proteins and possible cure for CTE, concussions and sub-concussions

Impacts to the head and body are intrinsic to the play of football and other types of high-impact contact sports. Rules changes may mitigate the prevalence of sub-concussions and concussions; unfortunately, it seems less likely that impacts to the head may be completely avoided in the game of football. The ultimate focus therefore should be on developing prophylactic and curative interventions, including

pharmacologic interventions, for both the acute, sub-acute, delayed, chronic and persistent sequelae of sub-concussions and concussions.

The propositional value of our work on CTE is that we have identified the proteins and peptides, which accumulate in the brain of CTE sufferers. We also know the proteins and peptides, which are involved in the pathogenesis of concussions and sub-concussions both in the acute and sub-acute post-injury periods. There are existing drugs, which we know can prevent the accumulations of these abnormal peptides and proteins, and the abnormal chemical reactions, which bring about these accumulations. If we can prevent the formation and accumulation of these abnormal peptides and proteins, there is a high scientific probability that we can cure CTE, acute and chronic sequelae of concussions and sub-concussions. We can develop laboratory based analytical systems and clinical trials to test these drugs for their ability to successfully interrupt the pathological cascades of CTE and the sequelae of concussions and sub-concussions.

Understanding traumatic brain injuries is a complex endeavor, which requires multi-faceted and multi-disciplinary efforts that cannot be provided by only a committee of doctors and other professionals. The NFL and other organizers of contact sports, at all levels, should finance studies and research by different, unrelated independent research groups and individuals across the United States including residents, fellows, doctoral and post-doctoral students. No single individual or group can provide all the innovative ideas, novel methodologies, original thought and intuition, which would be needed. Such research sponsorships should not be based on any affiliations, associations, preferences or pre-conditions. They must remain completely unbiased and transparent.

Elucidating and deciphering CTE and the sequelae of sub-concussions and concussions can only enhance the game of football and other high impact contact sports at all levels of play. It can only enhance the health, safety and quality of life of all players, their families and the general population. It can only reduce the unimaginable direct, indirect, tangible and intangible economic costs to these players and their families at all levels, the NFL, other organizers of contact sports and the society at large.

Thank you again for allowing me to speak with you and I will be pleased to answer any questions.