From my Desk

2013 Moody Prize Winner Jennie Ponsford, Ph.D.

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Correction

In the article Charting a Course for Long-term Care (Vol. 7, Issue 2), under the section “First Party Special Needs Trust,” the last sentence of the second paragraph should have read “The liability proceeds would be directed into the trust, and Medicaid would not consider those assets for eligibility purposes.” The word “not” was omitted in the print version. The correct version of the article can be found in the digital edition at www.biausa.org/brain-injury-publications.htm.
Any brain injury — whether mild, moderate, or severe — can change the way a person moves, thinks, acts, and feels. Like the injury itself, impairments can be focal, affecting a specific lobe of the brain, or diffuse, affecting multiple functions. Some injuries result in chronic conditions or speed up the normal aging process. For some people, a brain injury can cause or accelerate other diseases, like epilepsy and Alzheimer’s disease.

Recently, Dr. Ramon Diaz-Arrastia, director of clinical research at the Center for Neuroscience and Regenerative Medicine and a member of the Brain Injury Association of America’s board of directors, dubbed this as the “Golden Era of Brain Injury Research.” He did so based on President Obama’s announcement of a major federal effort to advance human brain research that could be used to treat a range of neurological conditions, including traumatic brain injury. The President included $110 million in funding for the “Brain Research through Advancing Innovative Neurotechnologies” program (known as the BRAIN Initiative) as part of his proposed FY 2014 budget for the National Institutes of Health. The main goal of the BRAIN Initiative is improved diagnosis and treatment for all brain disorders.

Right now, bench scientists and clinical researchers are making steady progress toward improved treatment in the minutes and hours after injury and through the rehabilitative process. For example, we are learning the therapeutic window may be longer than we thought, the spleen plays an important role in stem cell therapies, and the timing and intensity of exercise after injury may be a key to recovery. The Brain Injury Association of America is pleased to report on research progress in this issue of THE Challenge! These advances give us great hope that someday we will find a cure for brain injury. As always, we are deeply grateful to the authors who contributed to this issue.

In the meantime, I salute our affiliates for the terrific work they do each day and thank the advertisers who help make THE Challenge! possible. We are fortunate to have such generous individuals and corporations that support the Brain Injury Association and our mission to advance prevention, research, treatment, and education to improve the quality of life for all people affected by brain injury.

Susan H. Connors, President/CEO
Brain Injury Association of America
Dr. Jennie Ponsford is the 2013 recipient of the Robert L. Moody Prize for Distinguished Initiatives in Brain Injury Research and Rehabilitation. She is currently Professor of Neuropsychology and convenor of the doctoral program in clinical neuropsychology at Monash University as well as director of the Monash-Epworth Rehabilitation Research Centre in Melbourne, Australia. Dr. Ponsford has been an inspiration to many of us working in brain injury rehabilitation.

Throughout her career, which now extends over 30 years, Dr. Ponsford has been a consummate clinical neuropsychologist and an esteemed teacher and leader in brain injury research. There is scarcely an area related to recovery and rehabilitation after brain injury that Dr. Ponsford has not investigated, in many cases contributing a definitive study to the topic area. She has authored more than 120 peer-reviewed publications as well as books, book chapters, and other publications. She has contributed substantially to defining the care and services that we can offer to individuals with brain injury through cognitive and behavioral rehabilitation, family care and services, and vocational services, and has also conducted investigations of genetics and premorbid factors that require consideration in planning rehabilitation for individuals with brain injury. All of her research has been clinical and applied. Her research has always focused on interventions and factors that may contribute directly to an improved quality of life for individuals with brain injury and their close others.

Examples of Dr. Ponsford’s research contributions early in her career include a seminal study of
the evaluation of an intervention to improve attention after brain injury (Ponsford & Kinsella, 1988). This study was one of the first to carefully investigate attentional remediation after brain injury and to raise concern that early intervention for attention may not significantly improve on spontaneous recovery. That concern was later endorsed in the recommendations of the systematic reviews of cognitive rehabilitation from the American Congress of Rehabilitation Medicine (ACRM) Brain Injury Interdisciplinary Special Interest Group.

Dr. Ponsford has continued to contribute to our understanding and ability to consider a wide range of factors affecting the outcome of brain injury, including sexuality (Ponsford, 2003), family functioning and emotional factors (Ponsford & Schönberger, 2010), and genetics (Ponsford et al., 2011). In addition to expanding our knowledge about moderate-severe brain injury, Dr. Ponsford has also contributed to our appreciation of the effects of mild brain injury. For example, a recent controlled study of adults provided important documentation of subtle effects of mild traumatic brain injury (TBI) that are frequently dismissed as psychological or motivational but are apparent relative to orthopedic controls (Ponsford et al., 2011b).

Dr. Ponsford has investigated the effectiveness of both programmatic rehabilitation interventions (Ponsford et al., 2006) as well as more specific interventions for common problems after brain injury, such as sleep disturbance and fatigue (Ponsford et al., 2012). In the former comparative effectiveness study, Dr. Ponsford found that community-based rehabilitation and facility-based rehabilitation resulted in similar employment and independent living outcomes for matched samples of individuals post-TBI. In this study, she was also able to identify differences between the two approaches in specific functional outcomes and costs of care. In the latter study, she described the interactions between sleep, fatigue, emotional disturbance, pain, and hormonal changes. She and her colleagues used these analyses to lay the foundation for future treatment research in both pharmacologic and behavioral interventions for sleep disorders that are prevalent after brain injury.

Besides her personal contributions to improving the care and treatment of individuals with brain injury and their families, Dr. Ponsford has contributed to the training of several generations of students. As her students have progressed in their own careers, they have also contributed substantially to the body of knowledge in brain injury rehabilitation. Dr. Ponsford has been an active collaborator with colleagues in the United States and throughout the world. She works with the consumer-based brain injury society in Australia as well as with other global professional associations including the International Brain Injury Association, the World Federation for NeuroRehabilitation, and the International Neuropsychological Society. She and her students regularly present at conferences sponsored by these and other professional and consumer brain injury organizations. She has served as an officer in many of these organizations as well as on the editorial boards of several prominent journals in our field including the Journal of Head Trauma Rehabilitation, NeuroRehabilitation, Neuropsychological Rehabilitation, and the Journal of the International Neuropsychological Society.

Dr. Jennie Ponsford has grown up with this field and contributed substantially to its development throughout that span of time. She is highly respected by her colleagues in the United States and worldwide. She is among a small band of individuals who have devoted the entirety of their lives and careers to advancing our understanding of how to deliver the best rehabilitation and care to individuals with brain injury and their families.

The Moody Prize is presented annually by the University of Texas Medical Branch School of Health Professions and the Transitional Learning Center of Galveston. The prize, named for Robert L. Moody, is administered by a board of governors; candidates are considered by a panel of experts. Criteria include the total impact of a candidate’s work, achievements and unique contributions, and recognition by peers within the scientific or rehabilitation communities.

References:


The brain is the most complex organ of the body and repairing it after injury is challenging. To address this challenge, research scientists are working to increase our understanding of brain function and to develop better diagnostic tools and treatments for the millions of people worldwide living with traumatic brain injury (TBI). The National Institute of Neurological Disorders and Stroke (NINDS) is the lead institute at the National Institutes of Health (NIH) for TBI research and supports a range of high quality research on this topic.

Understanding reasons for good and poor recoveries after TBI is an important step toward developing better treatments, and several years ago NINDS supported a study to address this question. The study was unusual in that it evaluated thousands of cases of severe TBI by combining data retrospectively from several clinical trials. Such large data sets were previously unavailable, and the study successfully identified several major factors associated with recovery. However, some research questions could not be fully addressed because the original studies did not include the data needed to answer them. It became clear that many important questions about recovery after TBI would need to be addressed in new studies. Moreover, investigators also discovered that retrospectively comparing data from multiple studies was excruciatingly difficult and time consuming because the same types of information were coded in different ways. For example, some studies reported a subject’s educational status as “none, grade school, high school, college, or graduate school,” while other studies reported “total years of school.” Hundreds of these types of examples existed, and investigators spent years figuring out ways to make the studies comparable and then reformatting the data. There had to be a better way to combine information from multiple studies!
The better way is the International TBI Common Data Elements Project, a collaboration among the NIH, Department of Defense (DoD), National Institute on Disability and Rehabilitation Research, Department of Veterans Affairs, and Centers for Disease Control and Prevention to standardize definitions and protocols for TBI research. Rather than retrospectively reformatting and harmonizing data after a study ends, the goal of the International TBI Common Data Elements Project is to standardize data collection at the beginning. The project includes hundreds of scientific experts from around the world who serve on working groups and steering committees to develop recommendations for collecting data in a uniform manner. The standardized data recommendations are referred to as common data elements (CDEs).

Currently, more than 900 CDEs for TBI research exist and are available on multiple websites, including the NINDS Common Data Elements website (www.commondataelements.ninds.nih.gov/tbi.aspx). A small number of the CDEs are so commonly used that they are called “CORE CDEs” and recommended for use in virtually all TBI research studies. Other CDEs are recommended for use depending on the type of study, such as epidemiology, acute-hospital, rehabilitation, or mild TBI/concussion. Some of the CDEs are relevant to subjects of all ages, and others are specific to children or adults.

Following the development of the TBI CDEs, the next major step was to try them out to see if they could actually work in a research study conducted across multiple centers on a wide range of subjects. To give them a test-run, four TBI hospitals collaborated to collect data on more than 650 subjects with TBI, with injuries ranging from mild to severe, in a study called TRACK-TBI. The TRACK-TBI study revealed that the TBI CDEs had a few minor issues, but overall they were usable. In addition, the CDEs accelerated the research as the data were collected in just two years. Most of the data are still being analyzed; one early publication demonstrated that an MRI of the brain was more predictive of recovery outcomes at three months than a conventional CT scan. Although previous smaller studies had shown similar findings, the large number of subjects in the TRACK-TBI study greatly increased the clinical significance and impact.

Lastly, there is the question of how to put the TBI CDEs into practice. It’s one thing to develop CDEs and another thing to actually use them. Fortunately, there are two major activities underway to promote the use of the TBI CDEs and data sharing. One is the NIH and DOD collaboration to provide a Federal Interagency TBI Research (FITBIR) Informatics System (fitbir.nih.gov). FITBIR provides a database for TBI research at no cost to the research scientists, as well as an electronic data capture tool, and a platform for collaboration and data sharing. FITBIR uses the TBI CDEs for its data dictionary. The second major activity is the International TBI Research (InTBIR) Initiative. InTBIR is a collaboration among the NIH, European Union Research Directorate, and Canadian Institutes of Health Research to support international team science to study 10,000 children and adults with TBI. InTBIR researchers will use the TBI CDEs in their studies to facilitate data sharing and analysis toward the development of better diagnostic tools and more effective treatments.

In summary, the International TBI Common Data Elements Project built a foundation for the FITBIR Informatics System and the InTBIR Initiative, and together all of these projects will help us to get farther, faster on understanding TBI and identifying ways to promote recovery.
Since its establishment, the Center for Neuroscience and Regenerative Medicine (CNRM) has brought together more than 200 federal investigators to focus on traumatic brain injury (TBI), particularly as relevant to the military service members in the national capital area. CNRM is a collaboration between the Uniformed Services University and National Institutes of Health (NIH). Intramural investigators at NIH have outstanding technical expertise that was not previously being applied to the needs of military service members. Department of Defense (DOD) investigators have benefited from core scientific resources within the CNRM that enable long-term gains not previously possible, such as the development of a brain bank and a biospecimen repository.

Interdisciplinary approaches have generated much stronger research efforts by bringing together teams across institutions. For example, investigators working in pre-clinical models of TBI can use neuroimaging for MRI and PET to determine mechanisms of dysfunction resulting from specific forms of TBI. The development of a brain bank will enable these plans to be validated at an early stage relative to the mechanism of damage observed in the human spectrum of injury. This information guides the strategies in clinical trials for patient selection and therapeutic targets. The diagnosis of a given patient is improved by the coordination of information from neuroimaging, neuropsychological and cognitive testing, and biomarkers data; these parameters also optimize treatment selection for a given patient and demonstrate an impact on patient outcomes. This interdisciplinary approach is developed and in practice for patients in ongoing CNRM studies. CNRM clinical studies at the NIH Clinical Center can accept civilian or military participants, which complements the studies carried out at Walter Reed National Military Medical Center and other military treatment facilities. For more information, go to braininjuryresearch.usuhs.edu.

Hyperacute MRI studies have already identified new features in patients with TBI that were not recognized with the standard of care CT imaging. Specifically, two distinct features were found in a high proportion of patients with brain injuries and indicate brain trauma. One feature is enhancement of the dura matter covering the brain tissue. A second feature is the presence of linear hyperintensities on MRI consistent with bleeding within the brain tissue, particularly extending across gray to white matter borders. These CNRM studies were led by Dr. Larry Latour of National Institute of Neurological Disorders and Stroke (NINDS)/NIH and take advantage of the expertise of these investigators with hyperacute imaging of patients with strokes that is now being applied to patients with TBI. Because there are more than 100 ongoing CNRM projects, it is difficult to communicate the contributions of each to the pre-clinical through clinical stages of development that are needed to improve patient outcome. This study is provided as one that is important and far forward in the clinical progression of research applications.

CNRM currently has 17 active clinical research studies, with several new studies starting in the coming months. These studies will help develop more effective assessments of TBI.

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The Center for the Study of Traumatic Encephalopathy (CSTE) was created as a partnership between Boston University School of Medicine and the Sports Legacy Institute. The goal of the CSTE is to learn more about the long-term effects of repetitive brain trauma, like that experienced by contact sport athletes and military veterans, through clinical and neuropathological research. The CSTE is particularly focused on understanding Chronic Traumatic Encephalopathy (CTE), a distinct neurodegenerative disease caused, in part, by repetitive brain trauma.

The CSTE has the largest CTE brain bank in the world, which is housed at the Bedford Veterans Administration (VA) Medical Center in Bedford, Mass. Led by Dr. Ann McKee, the VA CSTE Brain Bank has been instrumental in understanding CTE. Her team has found that this disease, which was once thought only to affect boxers, can be present in a wider variety of individuals exposed to repetitive brain trauma (e.g. American football players, hockey players, military service members, etc.). In their most recent paper, Dr. McKee and colleagues published the most comprehensive case series of neuropathologically confirmed CTE to date. This landmark paper described CTE in 68 cases of individuals exposed to repetitive brain trauma and defined a system for classifying the severity of disease.

The CSTE’s clinical research is led by Dr. Robert Stern. Dr. Stern and colleagues recently published an important paper describing the clinical presentation of CTE. Based on interviews with family members of individuals whose brains were donated to the VA CSTE Brain Bank, Stern and colleagues assert that there are two distinct clinical presentations of CTE: one that begins earlier in life with mood/behavior symptoms and one that begins later in life with cognition symptoms. This increased understanding of the signs and symptoms of CTE during life is critical to moving toward clinical diagnostic criteria for diagnosing CTE during life.

Dr. Stern is also the principal investigator on the first-ever NIH R01 research grant dedicated to studying CTE. This research study, called the DETECT study (Diagnosing and Evaluating Traumatic Encephalopathy using Clinical Tests), is about halfway to completion, and the ultimate goal of this study is to understand the clinical presentation and possible diagnostic biomarkers of CTE.

Additionally, Dr. Stern leads the LEGEND study (Longitudinal Examination to Gather Evidence of Neurological Disease) and the Brain Donation Registry. The Brain Donation Registry allows living athletes to pledge to donate their brain to the VA CSTE Brain Bank upon their death. The LEGEND study is a longitudinal, telephone- and computer-based research study that follows current and former athletes of all levels and sports. While it is known that not all people exposed to repetitive brain trauma get CTE, what differentiates those who do and do not get the disease is unknown. The LEGEND study aims to understand the other possible risk factors for CTE, including possible genetic risk factors that may play a role in the disease.

The VA CSTE Brain Bank team continues to examine the brains of individuals who have passed away for evidence of CTE and other neurodegenerative diseases.
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New Strategies for Treatment Show Promise for Memory and Motor Function Improvement

By Kathryn E. Saatman, Ph.D., University of Kentucky, Chandler Medical Center, Spinal Cord & Brain Injury Research Center and Departments of Physiology and Neurosurgery

Trauma to the head can initiate a complex series of damaging events in neurons, the electrically excitable cells of the brain. One very early event is a change in calcium levels within these cells.

Although calcium ions are critical for signaling in healthy cells, excessive levels of ionic calcium inside neurons can activate enzymes called calpains. Calpains are proteases, a type of enzyme that damages proteins.

Persistent activity of calpains impairs neurons and eventually results in neuron death. Under normal circumstances, calpain activity in the brain is held in check by a protein called calpastatin. Because traumatic brain injury (TBI) causes such excessive calpain activity, calpastatin levels appear to be insufficient to protect the brain.

In two related studies published in the past year, we demonstrated that increasing levels of calpastatin in the brain protects several key proteins in the brain and improves both coordinated motor function and memory after TBI (Schoch et al., 2012; Schoch et al., 2013). Genetically altered mice that had high levels of calpastatin showed less breakdown of various types of proteins including a structural protein, an ion channel protein, and a protein implicated in nerve growth.

(Continued on pg. 25)
Traumatic Injury to the Developing Brain of Young Children
Developing a Temporal Road Map for Emerging Cognitive and Social Deficits

By Linda Noble-Haeusslein, Ph.D., University of California at San Francisco

Young children who sustain traumatic brain injury (TBI) often experience problems with learning and memory. This can interfere with academic performance and impede their ability to progress with their peers. Equally important, young children with TBI are at risk for developing social dysfunction — the inability to develop proper social skills — which can interfere with establishing social bonds with peers and within the community as a whole.

During normal development, the immature brain undergoes remarkable physical and biochemical changes to become the complex adult brain. Injury to the brain as it is undergoing these processes disrupts function and interferes with the development of normal age-appropriate behaviors.

Therapies for a child with a brain injury must be multi-targeted, offering protection to the damaged brain and providing support for proper brain development. Designing such therapies is dependent, in part, upon the identification of behavioral benchmarks that underscore proper brain development. Thus, our experimental studies have focused on defining these benchmarks.

Our laboratory developed a model of TBI in mice at an age that approximates a human toddler. We confirmed that TBI in this model resulted in cognitive deficits associated with both learning and memory, and these deficits only became apparent as the brain aged after the injury. Such findings underscore the impact of injury on those developmental processes that define cognitive function.

The concept of “emergence” of behavioral deficits is also evident in our most recent studies. We began to construct a “social map” that defines the emergence of social behaviors during normal brain development and how and when this map may be altered in response to TBI. Relatively little research has addressed the emergence of social dysfunction that can shape the ability of a child with a brain injury to develop interpersonal skills that are essential for functioning in the outside world.

As a first step toward developing this map, we have taken advantage of the published literature on experimental models of autism, a disorder of brain development that is characterized by impaired social interactions and verbal and non-verbal communication. Scientists have developed a series of behavioral tools that underscore social deficits in mouse models that mimic this human brain disorder.

From this research, we have adapted many of these tools to our model of developmental TBI, focusing on metrics of social investigation including social recognition, social interaction, and social dominance. These tests were all based upon how animals physically interact with one another in uniquely defined social settings.

We began by asking a very basic question: When do social deficits become apparent after injury to the developing brain? We found that mice subjected to TBI at the same stage as a human toddler did not show signs of social deficits at adolescence; social deficits became prominent when these animals reached adulthood. The emergence of these social deficits occurred at the same time as the appearance of cognitive deficits. We believe that these adverse long-term outcomes are a result of failure to acquire new psychosocial skills in response to developmental demands.

We are currently using a technology that allows us to record vocal communication between mice. These vocalizations change depending upon different experimental/social contexts. To date, no studies have applied this technology to developmental models of TBI, nor have there been any attempts to link this form of communication to social behaviors.

We believe that this new tool, together with our other behavioral measures of social interactions, will define a “social map” in mouse models that will guide us in discovery of new therapies intended to rescue the brain from the emergence of problematic behaviors.
The Interaction of the Spleen and Potential Stem Cell Therapies for TBI

By Charles S. Cox, Jr., M.D., University of Texas Medical School at Houston, Department of Pediatric Surgery

Our laboratory focuses on the pre-clinical, translational, and early (Phase 1 and 2) clinical trials studying the potential therapeutic effects of adult stem cells in traumatic brain injury (TBI).

Initially we pursued the idea that the use of stem cells could replace or regenerate injured neurons and their supporting cells in the injured brain, but subsequent information has largely shown this is not the case. Nevertheless, we repeatedly observed beneficial effects from these adult stem cells when administered intravenously into a laboratory model of TBI, despite the lack of integration of the cells into the brain.

Interestingly, cell tracking studies noted that when the adult stem cells were administered to an experimental subject who had sustained TBI, these cells were sequestered in the lungs and the spleen. This basic finding led us to pursue the idea that these cells exerted their effect by interacting with the immune cells already located in the lung and spleen and creating a “downstream” effect.

In a study of brain injury/stroke conducted by Drs. Willing, Pennypacker, Ajmo, and Hearn, we saw that the spleen played an important role in the immune response to neurological injury. Namely, after stroke, there was a loss of spleen volume accompanied by an outpouring of inflammatory cells that could worsen the deleterious effects of a stroke by increasing inflammation. We repeated these studies in the laboratory and found an almost identical loss of splenic mass/volume.

We were amazed to find that although removing the spleen blunted the inflammatory response to TBI, the spleen itself was necessary in order for adult stem cell therapies to be protective, because adult stem cells that migrated to the spleen served to protect the brain and release anti-inflammatory molecules called cytokines.

That finding didn’t really explain how the stem cell treatment after TBI protects the brain, however. We began to explore the idea that the activation of a certain type of brain cell called microglia might be a factor. Microglia are immune reactive cells in the brain that constantly monitor the status of the central nervous system (CNS). When these cells sense an injury or infection, they are activated to change shape and convert to a

(Continued on pg. 34)
TBI: Delayed Intervention May Still Provide Effective Protection

By Shruti V. Kabadi, Ph.D., David J. Loane, Ph.D., Alan I. Faden, M.D., Department of Anesthesiology, Center for Shock, Trauma and Anesthesiology Research (STAR), University of Maryland School of Medicine, Baltimore, M.D.

Although it is traditionally accepted in the brain injury research field that damage can only be reversed with an intervention within a few hours of trauma, our studies suggest that there may be a substantially longer therapeutic window for the treatment of TBI.

References:

Recent experimental and clinical evidence suggests that traumatic brain injury (TBI) leads to changes in responses of genes and genetic material inside our cells, including those related to inflammation in the brain. Using experimental models of TBI, our laboratory has demonstrated that trauma to the brain initiates a series of harmful events that begin seconds after the injury and can continue for years, leading to long-term tissue destruction.

TBI should not be viewed as a static, acute neurodegenerative disorder. Both human and animal studies have indicated that TBI leads to long-term inflammation as a result of the activation of immune cells in the brain (microglia). This inflammation plays a significant role in worsening TBI-associated tissue injury and behavioral dysfunction.

Microglia, which control inflammation in the brain and spinal cord, act as the first form of immune defense in the central nervous system (CNS). Increased microglial activation in the brain has been observed in post-mortem studies of survivors of brain injuries up to 16 years after injury. Our published studies have presented the first detailed, comprehensive account of the progressive and sustained nature of microglial activation and associated brain tissue injury continuing up to 4 months after TBI in the lab (Kabadi, S.V., et al., 2012a; Kabadi, S.V., et al., 2012b; Byrnes, K.R., et al., 2012).

Although the traditionally-accepted protocol in the administered brain injury research field is that the resulting damage can only be reversed with an intervention within a few hours of trauma, our studies suggest that there may be a substantially longer therapeutic window for the treatment of TBI.

More recently, we demonstrated that because inflammation is activated for an extended period of time after TBI, delayed treatment with a single dose of a novel anti-inflammatory compound, (RS)-2-chloro-5-hydroxyphenylglycine (CHPG), at one month after experimental TBI significantly reduced chronic microglial activation and associated neurological degeneration and behavioral deficits (Byrnes, K.R., et al., 2012). These findings not only introduce a novel treatment approach for brain trauma but also significantly extend the currently accepted therapeutic window to protect brain tissue following TBI.

In addition to targeting the inflammatory component of TBI using drug-based strategies, we have also begun to evaluate the potential of non-pharmacological approaches, such as physical activity and exercise, for the treatment of TBI. Our ongoing research underscores the critical importance of timing of exercise initiation after TBI and its association with chronic inflammation in the brain. Interestingly, we have observed that exercise initiation beginning at five weeks after TBI lessened progressive inflammation and improved cognitive ability (Piao, C.S., et al., Neurobiol Dis).

This work further challenges the widely held view that effective treatment to reduce tissue injury and loss requires early intervention. It also supports our hypothesis that beneficial treatment, even when administered at a delayed time point following TBI, can make a substantial difference.
A brain injury often begins when the head is struck by an object or is violently shaken by an outside force. The brain immediately begins to respond to the injury: Cell signals of injury must be controlled, damaged components must be cleaned up, and normal brain function must be restored. Various parts of the brain contribute to these functions after injury.

Although medical studies in the laboratory often focus on single genes or proteins, brain injury and the recovery from it primarily affect neural circuits, the same circuits responsible for brain-related behaviors. Damage to and recovery (reorganization) of brain circuits would disrupt function and elicit neurological symptoms that are characteristic of brain injury.

In animal models, as well as studies of human brain injury, inflammation — much like the swelling around a bee sting — is turned on in injured areas to clean up the damage and then begin repair to the best of its ability. Brain microglia cells are the dynamic sentries of the inflammatory response that patrol the brain for injury signals. In response to injury signals, microglia adapt rapidly by changing shape to meet the functional demands. Activated microglia cells change from a tumbleweed shape to a ninja throwing star shape and then work to remove cellular debris, regulate inflammatory processes, and promote tissue repair.

Our innovative research looks into a largely unstudied, century-old description of rod-shaped microglia. These were first described in the late 1880s by the renowned neuropathologist Franz Nissl and then by Ramón y Cajal and colleagues in the early 1920s. They observed these rod-shaped microglia in cases of paresis, typhoid, syphilis, and chemical exposure. Although recent medical interest has diverged from rod microglia, our recent research has uncovered massive groups of rod-shaped microglia in the brain after TBI. But what are they doing there?

In our laboratory, our diffuse brain injury model serves as a tool to investigate the role of rod microglia in brain injury. The neurons and the rod microglia, but we do not know if these signals lead to further damage or promote repair.

• Rod microglia do not appear in the brain immediately after injury; they slowly take shape over the first week following the TBI. This tells us that the signals generated by brain injury need to reach a specific threshold before microglia take on a rod shape. Because we find rod microglia only in diffuse brain injury, it is likely that only the injuries that damage the brain without destroying it can promote their formation.

• Rod microglia line up next to long “branches” of neurons. They form trains, one after another, along the whole length of a neuron. This tells us that there is communication between the neurons and the rod microglia, but we do not know if these signals lead to further damage or promote repair.

• Rod microglia form in the same brain regions that play a role in light and noise sensitivity following TBI. This sensitivity emerges in a delayed manner, indicating that the brain must undergo some progressive change. Perhaps rod microglia are emerging in the appropriate brain region and are responsible for sensory sensitivity through circuit reorganization.

As our research progresses in this area, our goal for ongoing studies is a detailed understanding of rod microglia and their involvement in circuit reorganization after TBI. We are resurrecting the investigation into the unfamiliar rod microglia, which could help to identify an unappreciated aspect of injury-induced signaling involved in circuit reorganization after TBI.

The long-term impact of these findings could lead to biomarkers for the detection or therapeutic targets for the intervention of rod microglia to alleviate harmful neurological consequences of TBI.
The Problem of Concussion Diagnosis

From physicians to parents, everyone can agree that diagnosing concussion is challenging. From our bioengineering perspective, concussion, or mild traumatic brain injury (mTBI), is essentially a “signal-to-noise” problem; the signal-to-noise ratio is generally very low. There are many small “signals” (such as brief memory loss), and these differ from injury to injury, both within an individual and from person to person. Different people’s symptoms after the same injury are mixed and not often the same. While this mixture of symptoms is also a problem in more severe forms of TBI, the signal is inherently larger than in mTBI, making diagnosis less problematic.

The problem of mTBI diagnosis is made more difficult by the variety of injuries that can cause a mTBI. For example, concussions can result from falls, football hits, or blast exposure. The resulting symptoms from each of these injuries may be different but could just as easily be very similar. As we can see, the variety of concussion signals stems from variability in the injury mechanism (i.e. type) and the injury response (i.e. symptoms). Concussion diagnosis requires accurate detection of these small and variable signals.

There is also a considerable amount of additional “noise,” which stems from an individual’s general health, fitness level, genes, diet, developmental stage, and so on. It is very difficult to pick out small signals from the noise of normal functioning within a person. There is also person-to-person variability that can be considered noise when trying to find assessment methods that can be applied to more than one person.

Concussion Assessment Methods

Concussion screening tools fall into several categories:

- neurological functional assessment (such as cognitive, sensory, and motor function);
- noninvasive imaging (such as structural and functional imaging);
- measurement of brain signals (such as electroencephalogram);
- detection of chemical markers of injury in body fluids (such as blood biomarkers); and
- biomechanical sensing (such as accelerometers or pressure gauges).

So how do we select the best diagnostic tools? Can we have a single measurement, or do we need a variety of tests and checklists? Regardless of the type and number of tools, there are a few criteria that should drive the selection.

Screening tools need sensitivity (precision) to detect the concussion signal. Concussion shares many symptoms with other conditions; therefore, quality diagnosis also demands specificity (accuracy). In other words, we don’t want the diagnostic tool picking up the noise and mistaking it for the signal. The tool should also be relatively short, objective, and practical in busy settings such as athletics and the military.

DETECT: A Novel Tool for Assessing Cognitive Impairment

Our research group has chosen to focus on cognitive functions, such as memory, reaction time, and processing speed, using tests that are sensitive to concussive symptoms. We have developed an immersive platform on which shortened neuropsychological tests are delivered without external distractions (Barker et al, 2007). The portable system, called Display Enhanced Testing for Cognitive Impairment and Traumatic Brain Injury — DETECT (patent pending) — provides an immediate objective score that is indicative of cognitive function (and possible impairment). Since cognitive deficits are often a component of concussion, DETECT can be used as an unbiased tool for concussion assessment.

For reliable screening, a tool must be clinically validated using sound research methods. We have shown that DETECT is as sensitive as traditional tests in evaluating cognitive impairment (Wright et al, 2010, 2011).

While it has the potential to be used as an independent tool, we also envision that DETECT can be combined with other screening tools to both develop even more sensitive diagnostic criteria (through cross-validation) and be part of an effective concussion assessment battery. To this end, we are combining DETECT with helmet accelerometers to assess the cognitive state in football players during play. We will determine the correlation between cognition and impact mechanics and improve detection of small neurological signals by filtering the noisy signal by biomechanical criteria.

(Continued on pg. 33)
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APRIL 1, 2013 — JUNE 30, 2013

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Until recently, many clinicians and scientists studying traumatic brain injury (TBI) paid relatively little attention to biomarkers. A biomarker can be as simple as checking temperature to indicate an infection or as complex as testing levels of glucose in blood for diabetic patients. There are many biomarkers that detect proteins in the blood that provide clues to the health of vital organs like the heart, liver, and kidneys.

Severe chest pains and the potential of a heart attack represent a common example of how biomarkers are used to improve diagnosis and treatment. A patient experiencing chest pain may not be having a heart attack but could be suffering from acid reflux, anxiety attacks, or pulmonary emboli — all of which require different medical interventions. Blood samples are quickly taken and samples are analyzed within 30 minutes to assess the levels of troponin, a protein found in heart cells. During a heart attack, levels of troponin are elevated in the blood. If the levels are sufficiently elevated, doctors can confidently rule out other causes and treat the patient for a heart attack.

Unfortunately the brain is the only major organ for which no blood biomarkers are routinely considered in clinical practice. CT scans or MRIs and functional neuropsychological assessments have been used as indicators, but these are not objective because they require some sort of interpretation and are not based on quantitative data. After a TBI, components of brain cells can leak into the surrounding fluid, and these cellular components are potential biomarkers that find their way into the blood where they can be measured.

Why are no blood-based biomarker tests currently being used to diagnose TBI? Historically, scientists have thought that a barrier between the blood and brain strictly regulates the movement of proteins and other substances from the brain into the blood stream. This blood-brain barrier was thought to make the measurement of changes in the brain following injury impractical. We now know that the blood-brain barrier is open following injury.

Studies have shown that biomarkers of damage to brain cells are elevated within hours following even mild TBI, and these biomarkers show greater and more prolonged elevations following moderate and severe TBI. Many investigators are conducting clinical studies of biomarkers of acute brain injury. Studies in adults and children are looking at sports concussions, mild and moderate TBI in the emergency room, and babies suffering from abusive head trauma (also known as shaken baby syndrome).

Cost is another major factor; industry figures estimate it costs between $100 million and $200 million to develop tests for biomarkers that could be approved by the FDA for clinical use.

Banyan Biomarkers, Inc. is currently conducting the only clinical trial seeking to secure FDA approval of a detection assay of biomarkers for mild and moderate TBI. The assay would be used in conjunction with other clinical information as an aid in the evaluation of patients 18 years or older with suspected mild or moderate TBI within 12 hours of injury to assist in determining the need for a CT scan of the head. The ALERT Study (www.biausa.org/alert-tbi) will enroll 2,000 patients and is expected to be completed early in 2014. Following trial completion, Banyan will analyze the data and file for FDA approval.

Biomarkers show promise for additional applications to diagnose and treat TBI. Biomarkers can provide critical information to more rapidly assess the effects of potential therapies for TBI and to rationally stratify patients with different types of brain injury in clinical trials of these therapies. Biomarkers have already proven successful in providing more accurate prediction of outcome following severe TBI, and they hold similar promise for predicting prolonged deficits and return to play following mild TBI and sports concussion, respectively.

Chronic biomarkers represent an exciting new area of investigation. TBI is best understood as a disease rather than an acute process. We now know that TBI is a risk factor for later neurodegenerative diseases such as Chronic Traumatic Encephalopathy, Alzheimer’s disease, and Parkinson’s disease. Studies are being conducted to discover and validate biomarkers of chronic pathological processes following acute TBI and their relationship to subsequent neurodegeneration.
Improvised explosive devices (IEDs) figure prominently in the arsenal of terrorists and insurgents around the world, and U.S. Military personnel have been facing an increasing risk from explosions during Operation Iraqi Freedom (OIF) and Operation Enduring Freedom (OEF). Blast injuries have accounted for almost 80 percent of casualties. Consequently, the incidence of traumatic brain injury (TBI) due to blasts has also risen. Because of advances in protective body-armor, soldiers are surviving explosions that would have been lethal previously. One result is that TBI has emerged as the signature wound of the global war on terrorism.

An explosion can cause injury via multiple, but fundamentally different, physical mechanisms. Primary injury is caused by the interaction of the blast shock wave with the brain and body. A shock wave is characterized by a disturbance in the air that travels at supersonic speeds with a massive increase in pressure, density, and temperature. Secondary blast injury is caused by shrapnel and other projectiles, leading to penetrating injuries. Tertiary injury is caused by acceleration and/or deceleration of the brain and body, leading to inertial-driven and blunt traumas. Quaternary injuries include those from all mechanisms not enumerated above, including burns, infections, and toxic gas.

Of these mechanisms, primary injury from the shock wave is the only mechanism unique to blast. Although penetrating, inertial-driven, and blunt (impact, concussive) TBI have been studied in detail for decades, primary blast TBI (i.e. TBI due purely to the shock wave) has received considerably less attention until recently. As bioengineers who are part of a multi-university research initiative among University of Pennsylvania, (David Meaney), Duke University (Dale Bass), and Columbia University (Barclay Morrison), funded by the Army Research Laboratory, we have been attempting to address important questions about primary blast injury in the context of TBI. By taking a multi-scale approach from the single cell level, through the tissue level, to the whole organism level, we have shown that a shock wave is capable of causing brain damage even when isolated from secondary or tertiary blast injury mechanisms.

Using our multi-scale approach to isolate different parts of the central nervous system, our current efforts are focused on identifying which components are being damaged and the biomechanical thresholds for that damage. We are finding that different components of the brain become injured at different blast severities. Not only do nerve cells die, but the blood brain barrier, a thin layer of endothelial cells that lines the capillaries and prevents blood constituents from entering the brain, becomes damaged and leaky. If certain blood constituents enter the brain, they can adversely affect its function or cause cell death.

We are finding that at even lower blast severities and in the absence of cell death, the electrical activity of neuron function in brain tissue is disrupted. Our results suggest that the blast shock wave alone is sufficient to cause changes in the function of the neural networks that make up the brain, a finding that may provide a physical basis for the long-term changes in the neuropsychiatric status of those suffering the after-effects of blast TBI. Our findings complement those in the more established studies of penetrating, inertial-driven, and blunt TBI.

Our ongoing work will have major implications for strategies aimed at both prevention and treatment of blast TBI. By determining the thresholds for damage to the most vulnerable components of the brain, we will be able to provide design criteria to helmet manufacturers. Armed with this quantitative data, they will be able to design new helmets and body armor to mitigate the precise aspect(s) of the shock wave responsible for damage. By providing higher-performance safety equipment, the incidence of blast TBI can be reduced. In terms of treatment, understanding which components of the brain are most susceptible may influence the choice of therapies to test for treatment of blast TBI. For example, damage to the blood brain barrier may require a different treatment than one aimed at preventing neuronal cell death. Depending on the severity of the blast, a combination of therapies may be necessary when multiple components of the brain are damaged. Our work may help to prioritize the therapies best used to treat blast victims.
The Brain Injury Association of America (BIAA) continues to advocate for access to care and Traumatic Brain Injury (TBI) Act reauthorization as Congress debates across-the-board spending cuts during the summer months. BIAA is committed to showing Congress that brain injury is a serious public health problem that will not be ignored. As important decisions are made throughout the fall, BIAA will be educating Congress about the importance of access to care for people with brain injury.

### TBI Act Reauthorization 2013

The summer months are an excellent opportunity to meet with your members of Congress to discuss their support for H.R. 1098 — the TBI Act — while they are in their district offices. The TBI Act was introduced by Representatives Bill Pascrell (D-N.J.) and Tom Rooney (R-Fla.), co-chairs of the Congressional Brain Injury Task Force, in March of this year. This bill would reauthorize the current programs relating to TBI and would also move the state grant and protection and advocacy grant programs from the Maternal and Child Health Bureau to another agency with the U.S. Department of Health and Human Services. The move acknowledges the impact of TBI across the age span, including older adults and returning service members/veterans. The Congressional Brain Injury Task Force recommends relocating the program to better coordinate with federal agencies regarding long-term services and supports available to individuals with other disabilities, particularly the long-term services and supports provision of the Affordable Care Act. BIAA thanks Reps. Pascrell and Rooney for introducing this important legislation.

BIAA, along with the National Association of State Head Injury Administrators (NASHIA) and the National Disability Rights Network (NDRN), have met with the staff of Senator Tom Harkin (D-Iowa), chair of the Senate Health, Education, Labor and Pensions (HELP) committee, to discuss introduction of the TBI Act in the Senate. Chairman Harkin and Ranking Member Lamar Alexander (R-Tenn.) plan to introduce a companion bill this fall.

The TBI stakeholders, BIAA, NASHIA, and NDRN, have also held meetings with Congressional offices to ask legislators to cosponsor the TBI Act. These offices include Rep. Frank Pallone (D-N.J.), Rep. Joe Pitts (R-Pa.), Rep. Michael Doyle (D-Pa.), Rep. Adrian Smith (R-Neb.), Rep. Mike Rogers (R-Mich.), Rep. Scott Perry (R-Pa.), Rep. Lee Terry (R-Neb.), and Rep. Cathy McMorris-Rodgers (R-Wash.). All meetings have been positive and legislative staff understands the urgency to reauthorize the TBI programs.

On August 5, 2013 a letter from the American Congress of Rehabilitation Medicine, BIAA, Friends of the TBI Model Systems, NASHIA, the North American Brain Injury Society, and the United States Brain Injury Alliance was sent to Sens. Harkin and Alexander stating consensus recommendations on the reauthorization of the TBI Act of 1996.

### Appropriations

In May, BIAA submitted written testimony to the Senate Appropriations Subcommittee on Labor Health and Human Services and Related Agencies for Fiscal Year (FY) 2014. Written testimony to the House Appropriations Subcommittee on Labor Health and Human Services and Related Agencies was submitted in March. Both letters proposed funding increases for TBI Act programs and the TBI Model Systems. To read BIAA’s testimonies, please visit www.biausa.org/biaa-legislative-priorities.htm.

### National Defense Authorization Act

The U.S. House of Representatives passed two amendments introduced by Rep. Bill Pascrell, Jr., concerning TBI in the military. As a result of the wars in Iraq and Afghanistan, approximately 176,000 men and women have sustained a TBI. The amendments were included as part of the FY2014 National Defense Authorization Act to aid the diagnosis and treatment of what has been dubbed as the “signature injury” of the wars in Iraq and Afghanistan.

The first amendment urges the Secretary of Defense to issue a report originally mandated in the FY2013 National Defense Authorization Act in a timely manner. This report must identify any gaps in services and treatments for soldiers with TBI, include a plan for addressing any gaps or redundancies, and identify an official to lead the implementation of any changes. The second amendment mandates a report on how the Secretary of Defense will identify, refer, and treat traumatic brain injuries with respect to members of the Armed Forces who served in Operation Enduring Freedom or Operation Iraqi Freedom prior to June 2010.
By amy c. colberg, m.a., Director of Government affairs, Brain injury association of america

Department of Labor, Department of Health and Human Services & Department of Education Appropriations

The Senate Appropriations Committee approved a FY2014 spending bill for labor, health, and education programs. The bill passed with a 16-14 party line vote. The Labor-HHS-Education bill would provide $783.4 billion for the respective departments, including $164.3 billion in discretionary funding.

The committee provided $9,760,000 for the TBI program. The program supports grants to states for coordination and improvement of services to individuals and families with TBI. Such services can include: pre-hospital care, emergency department care, hospital care, rehabilitation, transitional services, education, employment, long-term support, and protection and advocacy services. The committee also encouraged CDC to consider supporting multidisciplinary approaches to early identification and treatment of persons with TBI.

The legislation would fully fund President Obama’s request of $5.2 billion for the Centers for Medicare and Medicaid Services to carry out health care reform. The legislation would also fund the National Institutes of Health (NIH) at $31 billion, an increase of $307 million. This level will allow NIH to allocate $40 million for the new Brain Research through Application of Innovative Neurotechnologies (BRAIN) Initiative. The bill also included $7 million for the Centers for Disease Control (CDC).

Other highlights of the appropriations bill include:

- Elder Falls: In 2010, 2.3 million older adults were treated in emergency departments after a fall, with more than 662,000 of these patients hospitalized. Many falls are preventable with lifestyle interventions and appropriate physical supports. In a new initiative, the bill includes $10 million to research and implement evidence-based approaches to preventing elderly falls.

- Special Education: The bill provides $11.7 billion, an increase of $125 million, under section 611 of Part B Grants to States, for educating students with disabilities. The bill also includes $463 million, an increase of $21 million, to support statewide systems of coordinated and early intervention services for children with disabilities 2 years old and younger, as well as their families; and nearly $70 million, an increase of $20 million, to support research on how people with disabilities learn and how best to meet their educational needs.

- Sports-Related Injuries: The committee is concerned about the number and severity of injuries related to sports activities at every age and experience level, from professional to programs for children. While physical activity is an important part of a healthy lifestyle and should be promoted as a national public health goal, the number of injuries, particularly those related to brain injury and concussion, is a matter of grave concern. The committee believes that new developments in sports safety equipment can reduce the number of injuries to participants in sports activities. In addition, safer technologies that have already been implemented in one sport can be applied to safety equipment in other sports, yielding widespread improvements in safety and injury reduction. For example, the committee is aware of new designs for football helmets aimed at reducing or eliminating the injuries that contribute to concussions and traumatic brain injuries. The committee encourages CDC to test and improve these new helmet designs in cooperation with academic centers, sports sanctioning organizations, and equipment manufacturers.

Rehabilitation Research Legislation Introduced in the U.S. Senate

Sens. Mark Kirk (R-Ill.) and Tim Johnson (D-S.D.) introduced S. 1027 on May 22, 2013 to improve, coordinate, and enhance rehabilitation research at the National Institutes of Health (NIH). The bipartisan legislation would implement some of the recommendations raised in the Final Report of the Blue Ribbon Panel on Medical Rehabilitation Research at NIH.

(Continued on pg. 24)
BIAA is a founding member of the Disability and Rehabilitation Research Coalition (DRRC), which submitted recommendations to the Blue Ribbon Panel. As reported in the spring 2013 edition of THE Challenge!, BIAA met with Sen. Kirk and Sen. Johnson’s staffs in February of this year to discuss the importance of access to care for individuals with brain injury.

HELP Committee Passes Reauthorization of the Workforce Investment Act, S. 1356

Before heading home for August recess, the Senate Health Education Labor and Pensions (HELP) Committee passed the reauthorization of the Workforce Investment Act (WIA). The WIA Reauthorization has been overdue since 2003. The bill passed through committee by an 18-3 vote, and will now be considered by the full Senate. In the bill, the National Institute on Disability and Rehabilitation Research (NIDRR) will be moved to the Administration on Community Living (ACL) in the Department of Health and Human Services (HHS). NIDRR is renamed the National Institute on Disability and Independent Living Research (NIDILR) and will administer the TBI Model Systems program. There are 16 TBI Model Systems throughout the United States that provide care and collect information on individuals with traumatic brain injury during the acute hospital stay and in the community after discharge.

Title V of WIA reauthorizes the Rehabilitation Act, including vocational rehabilitation (VR) programs. According to the committee press release, the updates to Title V are aimed at making sure that young people with disabilities have increased preparation and opportunities for competitive, integrated employment. The bill requires state VR agencies, in conjunction with local educational agencies, to make pre-employment transition services available to students with disabilities. Updates to the bill also focus on creating better alignment of government programs at the national level that are focused on employment and independent living for people with disabilities.

MedPAC report to Congress

In May, the Medicare Payment Advisory Commission (MedPAC) reported its recommendations to Congress for reforming outpatient therapy services in Medicare. While BIAA appreciates MedPAC’s efforts to put forth meaningful reform proposals, we believe some of the panel’s final recommendations would significantly reduce access to vital rehabilitation care and do little to ensure appropriate payment for services or minimize inappropriate utilization. Despite serious concerns with many of the recommendations, BIAA is pleased by the Commissioners’ recognition that a hard cap on outpatient therapy without an exceptions process would create enormous barriers to care for tens of thousands of people across the country. However, MedPAC’s recommendation to lower the annual allowable limits for outpatient therapy services from $1,880 to $1,270 is particularly troubling to the brain injury community. Under such a proposal, Americans most in need of rehabilitation therapy, such as individuals who have sustained a brain injury, would undoubtedly, see their recovery efforts halted by delays in care and barriers to access. BIAA urges Congress to reject this approach. BIAA also shared these concerns in a letter to the chairman of MedPAC.

Future Care Coalition

The Future Care Coalition is an alliance of disability, patient, consumer, and provider organizations led by BIAA and the United Spinal Association that is committed to protecting the health and financial security of current and future Medicare beneficiaries who have incurred an injury, disability, or chronic condition as the result of an accident. The goal of the Future Care Coalition is to ensure that the voice of this population is represented and the Medicare rights of those who may have received a financial settlement in response to an accident, injury, or illness is preserved.

During the summer months the Future Care Coalition met with the Office of Financial Management in the Centers for Medicare & Medicaid Services and the White House to discuss the future Medicare beneficiaries’ issue. Both parties were receptive to the coalition’s concerns. The Future Care Coalition plans to meet with the Centers for Medicare and Medicaid Services Administrator Marilyn Tavenner this fall to share its concerns regarding the future medical issue. To learn more about the FCC, please visit: www.futurecarecoalition.com.

The Convention on the Rights of Persons with Disabilities (CRPD)

BIAA has signed on to support the Convention on the Rights of Persons with Disabilities (CRPD), an international disability treaty that was inspired by U.S. leadership in recognizing the rights of people with disabilities. The CRPD is a vital framework for creating legislation and policies around the world that embraces the rights and dignity of all people with disabilities. The Americans with Disabilities Act (ADA) was the model for the CRPD, and the ADA’s values of independence and
respect and concept of reasonable accommodation are echoed throughout the treaty. The United States signed the CRPD in 2009. On December 4, 2012, the U.S. Senate considered the ratification of the CRPD but fell five votes short of the super-majority vote required to ratify a treaty. BIAA and its partners in the disability community remain committed to bringing the treaty up in the 113th Congress.

Happy Birthday to the Americans with Disabilities Act

On July 26, 2013, the Americans with Disabilities Act (ADA) celebrated 23 years! The ADA is aimed at eliminating discrimination against people with disabilities and ensuring equal opportunity for them in the workplace. The law was sponsored by Iowa Sen. Tom Harkin and Maryland Rep. Steny Hoyer and signed by President George H.W. Bush on July 26, 1990.

Although excessive calpastatin did not have a notable impact on the number of neurons that died in the cortex or hippocampus after a contusion brain injury, mice with excess calpastatin showed significantly improved ability to walk across narrow beams and to remember familiar objects, suggesting that calpain activity may be contributing to impaired brain function after TBI. Therapies designed to inhibit calpain may help to improve motor function, learning, and memory in TBI survivors.

We have also published provocative new findings using an alternative approach to treat brain injury. Instead of blocking the activity of a single, potentially damaging protein such as calpain, we are investigating the ability of insulin-like growth factor-1 (IGF-1) to promote a wide array of pro-survival, pro-growth, and pro-repair pathways to minimize brain damage.

IGF-1 is a hormone, primarily made in the liver, that is important for normal growth and development of the body. IGF-1 is also found at much lower levels in the adult brain, where it may protect neurons against death due to aging, disease, or injury. IGF-1 appears to play other beneficial roles in the brain, stimulating the creation of new neurons and protecting myelin, which insulates the neurons.

In a study published this year in *PLoS One*, we show that, when compared to normal (nontransgenic) mice, mice that have excess levels of IGF-1 in the brain after TBI have greater activation of the pro-survival protein Akt and fewer dying neurons in the hippocampus, a brain region critical for functions such as learning and memory (Madathil et al., 2013). Greater neuron survival in this region of the brain in mice with excess levels of IGF-1 was accompanied by significant improvements in memory ability and coordinated motor function over the first week after TBI.

This study suggests that boosting levels of IGF-1 in the injured brain may not only protect against neuron loss but improve cognitive and motor abilities following TBI.

References:


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The Brain Injury Association of America helps millions of people who never expect to be hurt.

HELP US HELP THEM
Please visit www.someoneyoulove.org to support our work
Focusing on Inflammasome and Brain Inflammation to Treat and Diagnose TBI

By Juan Pablo de Rivero Vaccari, Ph.D., W. Dalton Dietrich, Ph.D., and Robert W. Keane, Ph.D., Departments of Neurological Surgery and Physiology and Biophysics, Miami Project to Cure Paralysis, University of Miami Miller School of Medicine

Inflammation in the brain is a major cause of problems experienced after traumatic brain injury (TBI). A protein known as the NALP1 inflammasome appears to be a main contributor to inflammation in brain tissue after TBI.

There are different types of protein inflammasomes, including the NALP1, NALP2, NALP3, or AIM2 inflammasome. The components of the NALP1 inflammasome multiprotein complex are proteins that have been called NOD-like receptor protein-1 (NALP1), the apoptosis-associated speck-like adaptor protein (ASC), and the inflammatory protein, caspase-1.

The NALP1 inflammasome is present in nerve cells, and its activation results in the activation of caspase-1, a protein that causes cells to die. Caspase-1 is responsible for the release of chemicals called cytokines that are involved in causing inflammation. These cytokines are involved in several detrimental effects after injury to the central nervous system (CNS) including TBI, spinal cord injury, and stroke. For this reason, the inflammasome represents a great therapeutic target to improve outcomes after CNS injury by decreasing the severe and uncontrolled inflammatory response that is experienced by patients with brain injury.

Our laboratory has been successful using neutralizing antibodies against ASC and NALP1 in rodent models of spinal cord injury, brain injury, and stroke. These studies have shown that inhibition of the inflammasome after CNS injury results in decreased inflammation. As a result, these animals showed improvements in motor skills and cognition and showed a reduction in the amount of cell death that is caused by TBI. This treatment with neutralizing antibodies was given intravenously after injury, and current efforts are underway to develop similar neutralizing antibodies that can be tested in humans.

Currently, neutralizing antibodies — also called biologics — are successfully being used in the treatment of several inflammatory diseases such as arthritis or inflammatory bowel disease. Some of these biologics include Infliximab, Adalimumab, or Infliximab. These therapies do not target the inflammasome; they target proteins activated by the inflammasome. In terms of therapeutic efficiency, it is better to treat early inflammatory events such as the inflammasome than it is to treat inflammation caused by the release of inflammasome.

In light of the beneficial effects of the current approved biologics to treat diseases such as inflammatory bowel disease or arthritis, it is encouraging that a biologic that can target CNS injury could be developed. However, adequate clinical trials need to be carried out in order to test efficiency and safety when used in the CNS injury patient population. The main side effect of all biologics that have been approved by the federal drug administration (FDA) so far is immunosuppression, and this should be taken into consideration in the design of clinical trials.

An exciting area in the field of brain injury is the area of biomarker research. Biomarkers are indicators such as proteins that inform us of the biological status of the body. With adequate biomarkers, the hope for true personalized medicine can become a reality. Interestingly, the inflammasome proteins caspase-1, ASC and NALP1 can be potentially used as a biomarker of injury severity, and these proteins also offer a significantly improved way to predict how serious long-term dysfunction will be after TBI.

Patients who present with high levels of the inflammasome proteins caspase-1, ASC, and NALP1 in the cerebrospinal fluid (CSF) in the early period after TBI have poorer outcomes, as determined five months after injury based on the Glasgow Coma Scale (GCS), than patients who presented with lower levels of these proteins in their CSF. Therefore, if inflammasome proteins are low in the CSF of patients with TBI, these patients are expected to have better functional outcomes than patients who have higher CSF levels of these inflammasome proteins.

Combining the potential of inflammasome proteins to be used as biomarkers and the therapeutic potential of neutralizing antibodies against inflammasome proteins following CNS injury, one can expect to have new powerful biological tools (Continued on pg. 33)
The Brain Injury Association of California (BIACAL) was happy to announce the passing of SB 320 in the Senate Health Committee by a vote of 5-2 on May 1st. The bill then went to the Senate Appropriations Committee, where it was held over until January 2014. BIACAL will continue to work towards passing this bill out of committee early next year.

BIACAL is now accepting registrations for the 14th Annual Neuroscience of Brain Injury Conference: Research Informing Medical Treatment & Legal Practice. The conference will be held at the Silverado Resort & Spa in Napa on November 8-9, 2013. The legacy of the conference is to assemble the finest medical and legal practitioners from around the U.S. and this year continues that tradition. Individuals can register online at www.biacal.org and click on the conference graphic located on the lower left side of the home page. Sponsors and exhibitors are welcome. In addition to the two-day conference, an optional Wine Tasting/Dinner is planned for Friday evening. For more information about this conference or BIACAL, please go to the website at www.biacal.org.

The Brain Injury Association of Illinois (BIAIL) had a busy summer with camps and the annual golf outing. Former Chicago Bears player and BIAIL board member Hunter Hillenmeyer chaired the outing. Golfers were greeted by NBC5 news anchor Lauren Jiggetts, Bob Love of the Chicago Bulls, and former NFL lineman Reggie Smith. The golf outing gave BIAIL the opportunity to share our brain injury awareness and injury prevention message with golfers and guests.

The BIAIL summer camp program was a great success and lots of fun! The camps had a combination of returning and new campers who took part in a variety of activities. Our camp volunteers took time away from their clinical practice to share their expertise in therapeutic and cognitive activities during the camps. The BIA is a ThinkFirst Illinois chapter and will be utilizing BIAIL Peer Outreach Team members for presentations and community involvement. Peer Outreach training was provided during camp.

The BIAIL was asked by Governor Quinn to be present on July 16th at the signing of the “Employment First Act,” a new law that requires Illinois state agencies to make employment for people with disabilities a priority. The legislation was first proposed by Governor Quinn in his 2013 State of the State address and is part of his agenda to ensure all people have an opportunity to reach their full work potential. The signing took place during the Illinois Department of Human Services’ event to celebrate the 23rd anniversary of the passage of the Americans with Disabilities Act (ADA) of 1990.

Join us for “An Afternoon at the Races” on Saturday, September 28th at Arlington International Racecourse. This will be the last weekend of racing for the 2013 season, and is sure to be lots of fun with family and friends. Additional information can be found at the BIAIL website, www.biaill.org, and tickets can be purchased online.

The statewide Educational Conference will be held November 1-2, 2013, at the Drury Lane Conference Center. Our keynote presenters include Dr. Larry Zachary (University of Chicago), Dr. Julian Bailes (NorthShore HealthSystem), Dr. Tina Trudel (Lakeview Rehabilitation Center), Dr. James Young (Rush University Medical Center), and Dr. Anjum Sayyad (Marianjoy Rehabilitation Hospital), as well as additional presenters and topics. The exhibitor hall is a great place for our attendees to meet with businesses and service providers. The exhibitor hall also has a Brain Injury Research booth that attendees are able to visit and find current research opportunities that may be available. The conference is open to the public. The annual Concussion Clinic will be held on November 2nd. An ACBIS certification examination will also be offered during the conference.

Upcoming dates are being scheduled for ACBIS trainings and examinations. Please contact the BIAIL office if you are interested in becoming a Certified Brain Injury Specialist.
Memorial Day, May 27th, marked the 26th anniversary of the major fundraiser for the Brain Injury Association of Kansas and Greater Kansas City: the Amy Thompson Run for Brain Injury. The Run is a Memorial Day fixture in Kansas City, uniting over 2,000 runners, walkers, survivors, and their families as they bring awareness to brain injury within the community. The event featured an 8K, 5K, and one mile Walk for Thought.

Increased efforts to raise awareness about brain injury saw a record number of participating Walk for Thought fundraising teams. It all took place in the scenic area off the well known Kansas City Plaza. Honorary Chair for 26 years is another Kansas City fixture, KMBC newscaster Larry Moore. Mr. Moore was also emcee for the morning festivities. This year’s honoree was Kansas City’s Dustin Criscione, who will be attempting to run across Kansas for brain injury awareness in September.

You can follow his quest on Facebook under Dustin’s Journey for Hope. Over $2 million has been raised since its inception in 1988.

The Brain Injury Association of Louisiana (BIALA) held its Annual Conference at the Embassy Suites Hotel in Baton Rouge with the theme “Creating Quality Connections.” Over 100 participants, including survivors, caregivers, and medical professionals, attended the two-day event that included presentations, testimonials, hands-on demonstrations, exhibitors, and a networking social.

We participated in the ThinkFirst Injury Prevention Foundation National Conference held in New Orleans. New Orleans Saints quarterback Drew Brees spoke to a group of students on the importance of wearing a helmet while riding a bike to protect from brain injuries. On June 22, we held the inaugural Wild Side Ride and Cook-Off fundraiser in Breaux Bridge. The family-friendly ride included a convoy of Jeeps through beautiful St. Martin Parish ending with a picnic party at Park Hardy. Participants were then treated to a rib and wing cook-off challenge, jambalaya, and live music. Special thanks to the Louisiana Jeepers organization and Courtesy Jeep in Breaux Bridge for sponsoring, organizing, and implementing the event. The event raised $7,000 through sponsorships, rider registrations, cook-off sales, and a silent auction. Brain injury awareness and prevention information was distributed to all participants.

BIALA continues to partner with the Louisiana Department of Health and Hospitals, Office of Aging and Adult Services, to operate a Resource Center through funding by the Traumatic Head and Spinal Cord Injury Trust Fund Program. The Resource Center provides a toll-free helpline, website, and a

Resource Directory of services to assist survivors and their families with transitional services, housing, transportation, education, and vocational rehabilitation. The Center also has a medical library with the latest research and advancements in science, information on local support groups, and resources on returning to work or school after injury.

Summer in Michigan has been very productive for the BIA of Michigan (BIAMI) on a number of fronts.

The ongoing movement by Michigan’s insurance industry and our Governor to drastically reform our no-fault insurance system has been on a temporary hiatus during the state legislature’s summer break, giving us a bit of breathing room to plan for the fall session. While our efforts to date in fending off the challenge have been successful, we anticipate a renewed effort by our opposition in September and are encouraging our members to help us continue the fight. In the meantime, both the BIA of Michigan and the broad-based Coalition Protecting Auto No-Fault (CPAN) continue to jointly host town hall meetings across the state to educate and inform the public about Michigan’s current auto no-fault law as well as to present both sides of this hotly contested issue.

The BIAMI held its first ever Legal Conference this past June, where over 160 participants learned about various legal issues affecting those with brain injuries. It was very well-received, with a wide range of law issues covered. Our East Golf Fundraiser took place in July, where over 210 golfers helped raise over $60,000 for the Association, while our West Golf Outing took place August 1 in Grand Rapids and was also a success.

Our Veterans Program is also in the midst of a busy summer, having held their third annual “Tips Up for the Troops” bass fishing tournament event on June 8th and is now preparing

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for both their “Putting for Prayers” golf outing in August and their second annual Veterans Conference in September, which provides information and assistance for veterans and their spouses in making a successful transition back into civilian life. The Veterans Conference takes place immediately prior to the Association’s 33rd Annual State Conference, the largest brain injury conference in the United States. Both events continue to grow in popularity and are expected to again draw a record number of attendees.

**NEBRASKA**

The Brain Injury Association of Nebraska (BIA-NE) board of directors announced Peggy Reisher, M.S.W. as the new executive director of the Brain Injury Association of Nebraska. She has worked for the BIA-NE as director of Programs and Services and as the director of the Nebraska Veterans Brain Injury Task Force Project for six years.

Her leadership has been the driving force in establishing the Nebraska Veterans Brain Injury Task Force, a group of representatives from civilian, military, and key government agencies working together to identify gaps in services for military members and veterans returning from war with brain injury and/or post traumatic stress disorder (PTSD). With the help of other task force members, she has taken an action-oriented approach to educating professionals, law enforcement, and community members about working with and understanding veterans’ experiences and brain injury, PTSD, and reintegration needs.

With a master’s degree in social work, Reisher worked for 14 years on the traumatic brain injury unit at Madonna Rehabilitation Hospital in Lincoln, Neb., where she helped patients and families identify community resources upon discharge from the hospital.

“While working with patients with brain injury and their families, I found it frustrating and frightening to tell them there were very limited brain injury services or supports as they transitioned from the hospital to their Nebraska community,” said Reisher. “I realized that it was going to take people like me, who could see the gaps in services across the spectrum, to be an advocate and voice for those with brain injury and their families.”

She then began to seek opportunities to become more involved, providing awareness and education about the needs of those with brain injury on a statewide and national level.

“As I take on the role of executive director for the BIA-NE, I do so with great pride and passion, for I sincerely know there is a need for those with brain injury to have a voice in Nebraska,” she said.

**NEW HAMPSHIRE**


As is customary, the winner of the Ellen Hayes award was announced and presented during the luncheon. The award is the highest honor BIANH bestows upon an individual for their outstanding efforts in support of individuals with brain injuries. This year, the Association’s board of directors voted to honor a survivor and a professional. Bud Elkind was honored as this year’s professional recipient and Michael Painter as the survivor recipient. Both of these individuals have worked tirelessly in support of brain injury survivors and their families.

BIANH now turns its attention to the 30th Annual Charity Golf Tournament on Wednesday, August 14, in Gilford. Over the past 30 years, many avid golfers and supporters of the BIANH have played in this charity tournament. All proceeds raised through the event are used to support programs focused on brain injury prevention and to provide emergency financial assistance to survivors and their families.

Two fall fundraisers are scheduled for BIANH: the 2nd Annual Heads-Up Half Marathon on Sunday, September 8; and the 27th Annual Walk-By-The-Sea that will occur on Sunday, October 6, 2013.

**OHIO**

Signed at the end of June, Ohio’s two-year Budget Bill relocates the state’s Brain Injury Program (BIP) and its Advisory Committee to a new administrative home at the Ohio Valley
Center for Brain Injury Prevention & Rehabilitation (OVC) within The Ohio State University Wexner Medical Center.

Housed within the Ohio Rehabilitation Services Commission (RSC) for all but the initial year following its creation, Ohio’s Brain Injury Program and Advisory Committee are set to be transferred to OVC by October 1, 2013. The BIP’s purposes, as described in state statute (ORC 3304.23), will remain unchanged. BIP’s focus is on promoting prevention, access to services and supports, education, and training benefiting Ohioans with brain injury and their families. The composition of the BIP’s Brain Injury Advisory Committee (BIAC) also will not change with the transfer and includes representatives from key state agencies as well as a seat designated for the Brain Injury Association of Ohio.

The need to relocate Ohio’s Brain Injury Program to a new administrative home became evident as increased federal restrictions were placed on the Program’s funds, limiting it’s use to vocational rehabilitation initiatives rather than across the full scope of the Program’s statutorily defined functions. Long-standing, highly valued services, including information and service linkage assistance provided through BIAOH’s Helpline and Community Support Network (field) offices, lost critical funding. In the summer of 2012, RSC Director Kevin Miller issued a memo pledging his commitment to minimize further disruption of supports for the brain injury community, and to work with BIAOH and BIAC to better position the Brain Injury Program to carry out its mission. Thereafter, the BIAC established an evaluation process to identify alternative administrative “home” agencies whose purposes, structures, and functions were a closer match with those of Ohio’s Brain Injury Program. After extensive deliberation, BIAC unanimously recommended transferring Ohio’s Brain Injury Program to Ohio State. BIAOH, instrumental in passage of the 1989 legislation creating the BIP and BIAC, supported BIAC’s recommendation.

In fact, according BIAOH’s long-serving executive director, Suzanne Minnich, “BIAOH’s Board was 100 percent behind transferring Ohio’s Brain Injury Program to OVC for its administration. Its faculty has great depth of knowledge in brain injury, from the clinical to research perspectives, and including policy, program development, as well as education and training. OVC’s strengths, together with the commitment, expertise, and resources of BIAC members, give us great hope for positive future developments for our members and constituents.”

**SOUTH CAROLINA**

The Brain Injury Association of South Carolina has had many reasons to celebrate this summer! On May 18th, BIASC and the South Carolina Association of School Nurses hosted a professional development training to increase school nurses’ knowledge regarding concussion management in the school setting. The training was provided by a South Carolina Developmental Disabilities Council grant, Heads Up! South Carolina. Eighty-eight school nurses across South Carolina attended the training.

On June 7, 2013, South Carolina’s Student Athlete Concussion bill, H. 3061, was signed by Governor Nikki Haley. The new law requires school districts in South Carolina to provide information fact sheets on concussions to all coaches, volunteers, student athletes, and parents or legal guardians; removal from practice or athletic competition if an athlete has sustained a concussion; and written medical clearance must be approved by a physician before a student athlete can return to play. This law went into effect immediately, which means student athletes will be subject to the new policy starting in the 2013-14 school year. BIASC is working hard to provide resources to school districts developing new concussion guidelines and policies. Also, BIASC has been approved to receive a grant from the Carolina Panthers to provide a continuing medical education (CME) webinar to physicians who treat youth and to provide educational opportunities for school professionals.

On June 11, 2013, the South Carolina Brain Injury Leadership Council (SCBILC) was signed into law as the statewide advisory body on brain injury. In 1996, Public Law 104-166, the federal Traumatic Brain Injury Act, designated that states create “state advisory boards.” A grant awarded to the South Carolina Department of Disabilities and Special Needs from the U.S. Department of Health and Human Services, Health Resources and Services Administration helped to firmly establish SCBILC. Although SC no longer receives federal funding, SCBILC has continued to work diligently to build service system infrastructure, organizational coordination, and collaboration to improve and expand comprehensive brain injury services.

We have just wrapped up our 2013 Statewide Life with Brain Injury Conference. It was held on July 12, 2013, in Columbia. Over 200 survivors, family members, and professionals came out to hear topics including “Practical Pointers to Securing Employment for Survivors” and “Functional Neuroanatomy as It Relates to TBI for Professionals.”

Now, BIASC is hard at work planning its 6th Annual Run for Thought 5K/1 Mile Walk, which will be held November 9, 2013, in Greenville. Check out our website, www.biausa.org/SC/, to learn more about the latest news and events from the BIASC!

**TENNESSEE**

The Brain Injury Association of Tennessee has collaborated with Ft. Campbell and the Epilepsy Foundation of Middle

(Continued on pg. 32)
and West Tennessee to start a monthly support group at Ft.
Campbell. This support group is for soldiers and veterans
with a brain injury and their families and meets on the second
Wednesday of each month.

We have set the date for our 2014 Annual Conference.
This will be held on Friday, March 28 at Trevecca Nazarene
University in Nashville.

Our second annual “Walk for Brain Injury” will be held
September 21, 2013. We are combining this year’s walk with
our Global Picnic. Registration is available at fundly.com/
walk-for-brain-injury-and-global-picnic.

**VIRGINIA**

The Brain Injury Association of Virginia (BIAV) hosted 72
campers this year at Camp Bruce McCoy in May and June.
Camp provides the opportunity for persons with brain injury
to socialize with one another and participate in a whole range of
activities from horseback riding and canoeing to fishing and arts
and crafts. Kathy Fedge, the mother of one of the campers,
said of her son Mac, “For Mac, camp is total freedom from his
brain injury. He truly blossoms when he goes to camp. And for
us, as caregivers, these two weeks allow us to renew ourselves.”
One of the counselors added, “[For us, camp] provides two weeks of
selflessness and service where we come together for this time
to create a mini family. This camp broadens people’s horizons
and makes us think beyond what we know.”

During the two weeks, State Senator Ken Alexander of Norfolk
and Delegate Ron Villanueva of Virginia Beach came to Camp
Bruce McCoy to meet with campers and counselors and to
gain a better understanding of our community and how BIAV serves
them.

Also in June, BIAV participated in a Richmond fitness festival called
RVA Streets Alive! This event brought sporting groups and health
professionals from around the metro Richmond area to participate
in activities and education and get Richmonders active and involved
in being healthy. BIAV partnered with Richmond Cycle Smart
and gave out brain helmets from the BIAA’s Lynn Chiaverotti
Memorial Fund while educating people on the importance
of wearing a helmet and how to cycle safely. It was a truly
amazing event for public awareness of our organization, and
the helmets were wildly popular!

In July, BIAV hosted Brain Injury Report Out Day in conjunction
with the Virginia Department of Aging and Rehabilitative
Services (DARS). This event provided critical information
about the tremendous strides made by the commonwealth
in establishing a network of services to meet the needs of
Virginians with brain injury, with a highlight on serving those
with concussions, while also noting several areas in which
improvement is still needed. This year, the focus was on the
risk and impact of sports concussion on young athletes. The
morning keynote speaker was Chris Nowinski, co-founder and
president of the Sports Legacy Institute (SLI) and co-director of
the Center for the Study of Traumatic Encephalopathy (CSTE) at
Boston University School of Medicine.

In August, BIAV hosted a donor recognition dinner, to give
thanks and appreciation for those donors who continue to
support our organization. Susan Connors, president and CEO
of the BIAA, and Mac Fedge, the camper mentioned earlier,
spoke to the group of donors to highlight the important role
that everyone plays not only in making the BIAV a success, but
how our donors contribute to the wider community.

We also held three neurobehavioral focus group discussions
in August. For many years, BIAV has worked collaboratively
with multiple constituencies to advocate with elected and
appointed policymakers to improve systems of care for those
with brain injury. While we have experienced some success at
expanding community based supports and services, Virginians
who experience behavioral challenges as a result of their brain
injury lack sufficient access to the care they require. It’s a
complicated issue with lots of moving parts, and due in part
to the lack of a Medicaid waiver for brain injury, licensing and
reimbursement restrictions, variations of policy within state
agencies and private providers, and inadequate facility based
short- and long-term care options.
The Difficulty of Concussion Diagnosis: Why It Is So Challenging and What We Can Do About It

(Continued from pg. 17)

The complexity of concussion makes identification of definitive symptoms (the true signals) challenging. It is likely that two or more screening tools will be necessary to increase sensitivity, specificity, and therefore, reliability.

Working together, we can solve the “difficult” problem of concussion diagnosis.

References:


Focusing on Inflammasome and Brain Inflammation to Treat and Diagnose TBI

(Continued from pg. 27)

to determine injury severity by looking at the protein levels of inflammasome proteins and according to those levels determine the adequate amount of antibody treatment to deliver. Moreover, knowing the levels of these proteins in healthy individuals or individuals with brain injury who have good outcomes after injury, the physician can use those protein levels as a therapeutic goal as well.

References:


CDC Challenges Kids and Teens to a Poster Design Contest!

Kids and teens can draw, paint, or use a computer to design a poster to help educate other kids and teens about concussion safety. One winner will be chosen in each category: Ages 5-8; Ages 9-12; Ages 13-15; Ages 16-18. The winner in each category will get one prize of $250.00. Kids and teens can send in posters June 12, 2013 through January 31, 2014. Judging will take place February 1st through 28th, 2014. Winners will be notified and prizes will be awarded by March 19, 2014.

Learn more at: www.beheadsup.challenge.gov

Tell Us What You Think of THE Challenge!
BIAA wants your feedback to help us improve THE Challenge! Let us know what you think at: www.biausa.org/survey

www.biausa.org/survey
and PTSD, improve brain imaging, and test new treatment strategies. Most CNRM studies involve advanced brain scanning. Experimental treatment studies include cognitive rehabilitation using a computer interface, therapeutic pharmacological treatments, aerobic exercise therapy, bright light therapy, and trans-cranial direct current stimulation.

For more information on CNRM Clinical Studies, please email CNRMstudies@usuhs.edu or call 855-TBI-CNRM (824-2676). Please also visit our websites below to learn more about CNRM and our various research activities:

CNRM: www.usuhs.edu/cnrm
CNRM Brain Injury Clinical Research: braininjuryresearch.usuhs.edu
CNRM Brain Tissue Repository: www.researchbraininjury.org

The Interaction of the Spleen and Potential Stem Cell Therapies for TBI

(Continued from pg. 14)

state that can worsen and/or extend the early inflammatory response to injury and further damage brain tissue that has been damaged but is not dead.

However, microglia can also revert to a “good” anti-inflammatory/remodeling status when under the influence of specific anti-inflammatory cytokines — like those released from the spleen after adult stem cell infusions. Through a sequence of laboratory TBI experiments, we demonstrated that various stem cell therapy approaches resulted in a decrease of the inflammatory response to TBI by changing microglia cells to the beneficial anti-inflammatory state in the injured brain.

We are now continuing our work in this area to determine whether the spleen is the critical link in the observed functional benefits of stem cell therapies. These data are important as they influence our understanding of the most beneficial and effective means of administering stem cell therapies. For example, injecting cells into the brain won’t likely be helpful if the benefit is from these cells collecting in the spleen.

Other studies have shown that inflammatory microglia are active for years after a TBI and that this activation correlates with neurological outcome. This may mean that our anti-inflammatory treatment time window for TBI is longer than we have classically thought. It remains to be determined if this approach can be translated into clinical studies.

BRAIN INJURY ASSOCIATION OF AMERICA
UPCOMING WEBINARS & LECTURES

September 10, 2013, 3 p.m. ET
Concussion: The Role of Imaging in Treatment
Barry Willer, Ph.D. and John Leddy, M.D., FACSM, FACP,

September 25, 2013, 3 p.m. EST
Outpatient, Home, and Community-based Services
Anne Marie McLaughlin, Ph.D.

October 17, 2013, 3 p.m. EST
Neuroendocrine Dysfunction following TBI
Lisa Kreber, Ph.D.

November 21, 2013, 3 p.m. EST
Rainbow U: A Case Study in Holistic Group-based Treatment
Heidi Reyst, Ph.D., CBIST

December 5, 2013, 3 p.m. EST
Anger Management (Caregiver Webinar)
Tessa Hart, Ph.D. and Monica Vaccaro, MS

December 12, 2013, 3 p.m. EST
Age-related Brain Disease
Tom Hall, MA

Registration for upcoming webinars, as they become available, can be filled out online in the Marketplace of the Brain Injury Association of America’s website at: www.biausa.org. Recordings of most webinars are available for purchase in the Marketplace as well.

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For more information on how to become part of Brain Injury Association of America Corporate Sponsors Program, please visit the sponsorship and advertising page at www.biausa.org or contact Susan H. Connors at 703-761-0750 or shconnors@biausa.org.