**DIRECTORY OF GRANT AWARDS
2016 GRANT CYCLE**

NEW JERSEY COMMISSION ON BRAIN INJURY RESEARCH

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JUNE 2016

NEW JERSEY COMMISSION ON BRAIN INJURY RESEARCH

This data was compiled in compliance with the New Jersey Commission on Brain Injury Research's statutory mandate, N.J.S.A. 52:9EE-1" …to compile a directory of brain injury research being conducted in the State."

The information contained within this directory is not all-inclusive. The research projects and researchers listed in this directory are all based in the State of New Jersey and have applied to and received funding during the fiscal year 2016 grant cycle. The research projects are not categorized, or listed in any particular order.

This directory is not a complete listing of all scientific research being performed within the State of New Jersey due to the proprietary nature of the research being conducted at various institutions throughout the State. In addition, institutions are not obligated to share their research information with the New Jersey Commission on Brain Injury Research.

Please feel free to contact the New Jersey Commission on Brain Injury Research at P.O. Box 360, 369 S. Warren Street, Trenton, New Jersey, 08625. The Commission's office can be reached by telephone at 609-633-6465, by fax at 609-943-4213, or by e-mail at NJCBIR@doh.nj.gov.

For information on the New Jersey Commission on Brain Injury Research's grant award process, grant applications and deadlines, please see: www.state.nj.us/health/njcbir.

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NEW JERSEY COMMISSION ON BRAIN INJURY RESEARCH

GRANT AWARDS

INDIVIDUAL RESEARCH GRANT RECIPIENTS:

CBIR16IRG017
Vijayalakshmi Santhakumar, M.D., Ph.D.
Rutgers University Biomedical & Health Sciences
$534,800

Project Title: *Does Enhanced Dentate Neurogenesis Promote Early and Long Term*
                 Circuit Dysfunction and Brain Injury

This multidisciplinary study will determine whether early abnormal increase in hippocampal neurogenesis after brain injury contribute to long-term neurocognitive decline and risk for epilepsy. Traumatic brain injury (TBI) is a major risk factor for several chronic disorders such as temporal lobe epilepsy and memory and cognitive dysfunction. The incidence of TBI from falls, traffic accidents, assault, combat injuries, and sports is constantly on the increase. Recent reports estimate over 1.7 million TBI cases in the US each year resulting in an annual economic burden beyond $60 billion.

Neurogenesis in the adult hippocampus replaces degenerating neurons with new neurons which are essential for maintaining proper memory and cognitive functions. As we age there is a decline in hippocampal neurogenesis which results in age associated memory and cognitive deficits. Brain injury rapidly increases generation of new neurons which has been proposed to help recovery of memory function. However, it has been shown that new neurons born after seizures tend to be abnormal and contribute to excessive excitability and epilepsy.

The proposed study will determine what happens to the pool of neural stem cell precursors and newly born neurons from the point of injury at time points over the course of several months after injury to determine if injury-induced increase generation of new born neurons leads to long term accelerated cognitive decline observed after brain injury. Additionally, the study will determine if new neurons born after injury contribute to early deficits in memory and long term increased risk for seizures. Thus, the proposed study will provide novel insights into the role of neural stem cells in neuropathology and neurological recovery after traumatic brain.

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CBIR16IRG032
David Margolis, Ph.D.
Rutgers University
Cell Biology & Neuroscience
$540,000

Project Title: *Role of Cortical Network Plasticity in Recovery from Traumatic Brain Injury*

This project uses novel in vivo imaging methods in mice to investigate brain plasticity underlying recovery from traumatic brain injury, and to screen candidate drugs to improve recovery.

Traumatic brain injury is a significant public health problem that has devastating cognitive and behavioral consequences in those affected. There is a critical need to discover treatments that improve recovery of brain function after injury. We still know too little about how brain activity changes in response to the initial injury and how these changes cause lasting detrimental effects on mental function and behavior. Research has shown that large brain networks are affected by injury, even in cases of focal head trauma. This is because brain areas are highly interconnected. Indeed, connections between brain areas are key for proper brain function. Therefore, it is critically important to understand how brain networks change their function over time after injury, and how this goes hand in hand with behavioral recovery. Some individual's recovery is faster than others. If we can show how brain networks account for behavioral and cognitive recovery, it might be possible in the future to change the function of those networks through drugs or rehabilitation to improve behavior and cognitive performance.

Our work uses a mouse model and cutting-edge neuroscience methods to test how brain networks respond to and recover from traumatic brain injury. Our team uses imaging to visualize brain activity of specific types of neurons across the entire cerebral cortex, and quantitative methods to measure how connections between brain areas change over time. Using our novel approach, the experiments aim to identify the mechanisms and timing of the brain's recovery from injury and to relate brain changes in the same subjects to cognitive and behavioral recovery. We believe our system will enable rapid screening of drugs that have potential therapeutic value for treating traumatic brain injury, with clinical implications within New Jersey and beyond.

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CBIR16IRG025
Michael F. La Fountaine, EdD, ATC
Seton Hall University
$526,549

Project Title: *Evaluating the Role of Baroreceptor Sensitivity in the Post-Concussive*
Symptomatic Milieu

The project measures changes to baroreceptor sensitivity following a concussion; information useful in identifying those with reduced ability to accommodate situations where blood pressure increases.

As public awareness of concussion grows and evidence demonstrates that repeated head impacts contribute to long-term risk of neurological damage, a shift in focus to assist our understanding of what a concussion does to clinically accessible processes in the body is necessary. By gaining this insight, we may be able to better appreciate the breadth of the clinical consequences that an injury and its symptomatic limitations impose. Although numerous assessment scales are available for the immediate evaluation and diagnosis of injury, the fact remains that much is still to be learned about how concussions affect body systems and reduce functional capacity for a brief time, especially among those with optimal functional performance (i.e., athletes). In evaluating how a concussion affects the brain's ability to coordinate responses, it may be important to include assessments that involve physical challenges where responses are made in proportion to the magnitude of a challenge. Deficits that may exist as a result of an injury, and its severity, would be expected to resolve on repeat evaluation as the injury "heals." Therefore, an approach to evaluate the brain's ability to coordinate these responses is a logical progression to facilitate this understanding.

The current proposal tests this approach by exploring changes to the arterial baroreflex during rest and physical stress on repeated evaluations over the first week following a concussion injury. The arterial baroreflex is a mechanism in the cardiovascular system under nervous system control that monitors blood pressure changes (both increases and decreases) with compensations to heart rate on a beat-to-beat basis. This reflex helps protect against large fluctuations in blood pressure which may contribute to syncope (i.e., fainting due to low blood flow to the brain) or pressure-related damage to end-organs (i.e., the brain) and in the context of the current proposal, exercise intolerance when the injury pervades.

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CBIR16IRG041
A.M. Barrett, Ph.D.
Kessler Foundation
$534,926

Project Title: *Improving Medication Self-Administration and Health after Brain Injury*

We model an innovative care pathway, by combining medication self-administration assessment we developed under Commission pilot funding, with an evidence-based reminder protocol.

After a traumatic brain injury, self-administering medication is a daily activity that provides a foundation for recovery, health and function. Taking medications incorrectly (medication self-administration or MSA errors) affects competence and dignity, but also strongly predicts the skilled help a person with brain injury will need after hospital discharge.

In this project, our interdisciplinary research group will extend our research on methods to identify and predict MSA errors over three years of intervention. We will add video call reminders, which others demonstrated improves medication adherence in a different clinical population. Participants with TBI will receive these video call reminders at the actual time when medication doses are scheduled to be taken. We will compare this approach to usual and standard care (no reminders) and automated texts, which were recently shown to enhance cardiac medication compliance. Impact of the three interventions will be compared based on participant's percentage of MSA errors identified on computerized pill bottle monitoring. These "Fitbits for medication accuracy" allow us to follow how participants take each medication dose throughout the six months they are enrolled in the study. We will also follow on a monthly basis whether participants are re-hospitalized, how caregivers assess their functional status, and how they self-rated quality of life.

Most people who are undergoing neurorehabilitation are completely unaware when they cannot perform MSA. We hope the assessment and intervention care pathway outlined in this study would help support a complete process that is patient-oriented, but yet does not require patient initiation or self-advocacy. We further hope this project will launch a model for further evaluation and demonstration, which would become part of standard neurorehabilitation care and accelerate brain injury recovery.

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FELLOWSHIP RESEARCH GRANT RECIPIENTS:

CBIR16FEL013
Przemyslaw Swiatkowski
Rutgers University
Cell Biology & Molecular Biology
$100,500

Project Title: *The Role of Cypin in Recovery of Electrophysiological and Cognitive Functions Following Traumatic Brain Injury*

Elucidating therapeutic targets for manipulation of the molecular mechanisms guiding protein recycling vital for formation of long-term memories in the brain after traumatic brain injury.

Traumatic brain injury (TBI) is caused by head trauma and displacement of the brain within the skull. Neurons, which are the principal cells of the brain, are damaged due to physical impact against the skull and/or any foreign object. Often neurons die, leading to motor and cognitive deficits. Although the effect of TBI on learning and memory has been extensively characterized, there is no effective treatment to improve cognitive functions affected by TBI. It is critical, therefore, to identify a way of protecting neurons responsible for learning and formation of memories after TBI.

Cytosolic PSD-95 interactor (cypin) is a protein that has been extensively studied and described by our laboratory. Our animal studies show that decreasing amounts of cypin present in neurons improves learning and memory following TBI in mice. I predict that cypin plays an important role in learning and memory, and downregulating its expression, via genetic manipulation, will improve cognitive functions after TBI. This study will provide new insights into the molecular mechanisms of how TBI affects learning and memory and explore possible therapies.

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CBIR16FEL009
Anna Giarratana
Rutgers University Biomedical & Health Sciences
Robert Wood Johnson Medical School
$100,500

Project Title: *Effect of Genetic Polymorphisms on Recovery and Treatment after Traumatic Brain Injury*

This project will assess how genetic differences effect recovery after traumatic brain injury (TBI) and explore possible treatment methods. Traumatic brain injury is a serious and potentially life threatening clinical problem. An estimated 12,000 to 15,000 TBIs occur in New Jersey alone, 1,000 of which are fatal. In addition, TBI can cause severe lifelong disability. Currently, 175,000 New Jersey residents live with disability due to TBI, a number that grows every year. TBI begins with an initial primary injury caused by events such as falls, vehicle accidents, or sports collisions. The primary injury sets off a cascade of events that initiates a secondary injury which may be responsible for the long-term neurological disabilities associated with TBI. While safety precautions that limit the occurrence of TBI are certainly very important, once a primary insult has occurred, the goal of researchers should be to investigate the best method of preventing the secondary insult from occurring and thereby prevent the development of long-term disabilities that stem from TBI.

One promising way to better manage treatment of TBI would be to stratify patients into risk categories based on their own genetic makeup, and even tailor treatment to patients who have varying genotypes. Clinicians have long noticed that certain patients recover better than others after TBI, and determining what makes some patients more susceptible than others is a vital step in identifying high risk patients who are more likely to have a severe reaction and lifelong disability due to a TBI.

Genetic polymorphisms, or differences in DNA between people, are an important facet of what gives people genetic diversity. Two specific polymorphisms that have been suggested to confer higher risk for poor recovery following TBI according to preliminary clinical studies on humans are Apolipoprotein E4 (ApoE4) and Brain-derived neurotrophic factor (BDNF) Val66Val. However, the initial studies have not been in complete agreement because it is hard to be consistent in trials that use human subjects due to the heterogeneous nature of the injuries they have suffered and their complex genetic background. That is why it is vital for us to do experimental mouse studies where we can limit the confounding genetic factors and create a homogeneous and replicable injury.

Our study will investigate the effect of the ApoE4 and Val66Val polymorphisms in genetically engineered mice on recovery following a repeated, mild TBI, which mimics the pattern that is shown in human sports injuries. We will look at cellular and behavioral outcomes following the TBI in order to determine if the ApoE4 and Val66Val polymorphic mice show worse recovery

than the ApoE3 and Val66Met polymorphic mice. If we find that these specific polymorphisms do confer greater risk, it will be very useful to know in populations such as student athletes. A simple blood test will be able to determine if someone is at a higher risk for suffering damage after injury, allowing parents and athletes to make informed decisions about their participation in sports, and reinforcing the importance of immediate treatment following concussions. We will also examine biomarkers in serum and cerebrospinal fluid as well as use magnetic resonance imaging to identify indicators of injury and repair that correlate with the cellular and behavioral changes we observe. The biomarkers we identify will allow physicians and patients to monitor the injury and recovery process in a quantifiable way to improve diagnosis and decisions about return to play.

Finally, we will look at possible treatments for TBI, to investigate if we can improve recovery in patients with these high-risk genetics. Specifically, we will investigate the use of human mesenchymal stem cells (hMSCs) which can be derived from adult bone marrow and have been shown to improve outcome in spinal cord injury. In summary, this study will explore the ways to best manage and treat patients who have TBI in an effort to reduce the burden that New Jersey and its citizens currently suffer due to TBI.

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CBIR16FEL010
Jessica Verpeut, Ph.D.
Princeton University
Neuroscience Institute
$201,744

Project Title: *Effects of Cerebellar Perturbation on Neocortical Adaptation to Post-Traumatic Brain Injury*

This project will analyze neural circuitry of the cerebellum and forebrain to determine alterations in dendritic growth and morphology with possible interventions following traumatic brain injury.

Chronic effects of traumatic brain injury (TBI) result from disruptions in neural circuitry. The forebrain is the primary region of injury during blunt force trauma and has long-distance connections with distal brain regions. Interactions between brain regions are typically studied by focusing on acute effects, such as diaschisis of blood flow and neural activity. This project seeks to probe these long-distance interactions in vivo using a focal-injury model of TBI, two-photon microscopy, and pharmacogenetic perturbation of neural activity. The result will be to understand long-term effects of TBI on both the directly injured region, as well as interactions with distal regions that may be involved indirectly.

The work will be organized into: (1) Quantifying the remodeling of the forebrain post-injury to the contralateral hemisphere of the forebrain or to the cerebellum by observing changes in neural circuitry in vivo, (2) Perturbing inhibitory signals from the cerebellum and analyzing contralateral forebrain neural circuitry after TBI. These studies will test the hypothesis that inhibitory signals from the cerebellum may modulate critical periods of development and neural plasticity. These windows may be reopened by turning off inhibitory signals from the cerebellum or contralateral forebrain. These studies will provide an understanding into the long term effects of TBI on neural circuitry and identify sites distal from the site of injury as potential targets for TBI intervention and rehabilitation.

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PILOT RESEARCH GRANT RECIPIENTS:

CBIR16PIL018
Bryan Pfister, Ph.D.
New Jersey Institute of Technology
$180,000

Project Title: *In Vitro Platform for Medium Throughput Study of Injury to Human Glutamatergic Cortical Projection Neuro*ns

Develop a multiwell in vitro platform to study mechanisms of traumatic brain injury in live human neurons and establish a technology to perform large-scale experimentation and drug screening.

Out of two million traumatic brain injuries (TBIs) a year only 500,000 are hospitalized; the remainder are those who do not seek medical attention or their unreported cases are managed with observation, rest and avoidance of repeated trauma. Identification of drugs to decrease the consequences of TBI or promote recovery is greatly needed. Currently, we are limited to investigating human TBI clinically in survivors, or pathologically using tissues available after a subject has died. Due to these constraints, the development of treatments presents formidable challenges and non-human models are required to explore mechanisms for treatment. Despite undeniable strengths, animal models are not practical for screening of biochemical libraries for their effects on trauma. The evaluation of new treatments could greatly benefit from an experimental system to study brain injury in live human neurons. The availability of such a platform on a high-throughput scale would allow the screening of compounds for their effects directly in human neurons.

Neuronal culture models have had significant impact by identifying important biological processes involved in TBI pathology. Currently these models are limited by experimentation of single samples, one-by-one, keeping productivity slow. The discovery of new treatments for many diseases, however, is accelerated with the use of high throughput tissue culture techniques. In this fashion, cell culture is more cost effective and rapid than animal models, providing an efficient method for screening for biomarkers and potential therapies. This proposal is to develop a neuronal culture platform to study the effects of potential treatments in differentiated human neurons and establish a technology to perform large-scale experimentation such as drug discovery. The unique characteristics of this model are its replication of injury and cell population that are directly relevant to human TBI that can be studied in a rapid fashion.

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CBIR16PIL015
David Shreiber, Ph.D.
Rutgers University
Biomedical Engineering
$180,000

Project Title: *Multi-Functional Biomaterials for Simultaneous Protection from Free*
                 Radicals and Targeting of Therapeutics after TBI

We will develop a biomaterial that protects neurons from damage from free radicals and polymerizes via these free radicals to localize delivery of neurotrophic factors.

TBI starts with physical trauma to the head, which is followed by a persistent multi-faceted secondary injury cascade that can continue to damage neurons long after the initial trauma. We believe that components of the secondary injury cascade can also be used to control the presentation of trophic factors that promote neuronal survival by reacting with a responsive carrier ma. Since the injury site has the largest concentration of these components, this would act to localize the delivery of therapeutic molecules at the injury site. In addition, the reaction of the responsive material with the different components may prevent the components from reacting with and damaging cells. Thus, if successful, our approach will improve neuronal survival in two ways: (1) protecting neurons from components at the injury site and (2) localizing and sustaining the presentation of trophic factors at the injury site. Both of these aspects of the biomaterial can potentially improve outcome following TBI. Together, we believe that this novel, multi-potent approach will significantly enhance neuronal survival following TBI. This Pilot Proposal describes a series of aims and objectives that are designed to generated proof of principle for the stimuli-responsive biomaterial. If successful, we will then move to establish efficacy in vivo and determine the best means of delivery.

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CBIR16PIL026
Joshua Berlin, Ph.D.
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$180,000

Project Title: *Cellular Events Precipitating Seizure-Like Neuronal Activity After*
                 Traumatic Stretch Injury to a Cortical Network Relevance for Therapeutics

This project seeks to investigate acute cortical neuron hyperexcitability following stretch injury as an experimental model for seizure activity following traumatic brain injury. Post-traumatic seizures is a serious morbidity for many people who experience traumatic brain injury. Current therapies are inadequate so new approaches are needed. To determine what novel therapeutic approaches might be successful, we would be greatly aided if we understood the processes that initiate the development of post-traumatic seizures. Unfortunately, current experimental systems miss many of the early events that set the injured brain onto the course to seizure activity.

Using a collaborative, interdisciplinary approach the principal investigators of this proposal have developed a novel in vitro model for seizure activity. We have developed data suggesting that this model system shares important properties with established traumatic brain injury models in which neuronal hyperexcitability is a common precursor to seizure development. Thus, we propose in this exploratory project to collect data that describe the changes in neuronal function occurring immediately following stretch injury. Towards this goal, we are asking specific questions about the behavior of the neurons in our model system to determine if and how they show hyperexcitable behavior, consistent with seizure activity.

The data derived from proposed experiments will allow us to define the basis for the early changes in neuronal function following injury, so that we can develop specific hypotheses to test in future experiments. In developing these data, we will necessarily employ experimental manipulations and pharmacological agents that target specific types of channels and receptors, thereby opening a window on potential approaches to minimize hyperexcitability after stretch injury and pointing the way towards future research to investigate therapeutic interventions to prevent seizures following traumatic brain injury.

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CBIR16PIL025
Helen Genova, Ph.D.
Kessler Foundation
$173,762

Project Title: *Examining Neural Changes Following an Emotional Processing Intervention*

This study will examine functional and structural changes following an intervention for emotional processing deficits in TBI using functional magnetic resonance imaging and diffusion tensor imaging.

Individuals with Traumatic Brain Injury (TBI) experience a number of physical and cognitive symptoms. Recent evidence suggests that in addition to these issues, a significant number of individuals with TBI have difficulty in emotional processing and social cognition. Specifically, some individuals with TBI have difficulty correctly identifying emotions from facial expressions. Deficits in emotional processing can have a significantly negative impact on social interactions, mood, and quality of life. Therefore, treatment of emotional processing deficits is critical to improving the lives of individuals with TBI.

The current study examines a treatment for emotional processing deficits in TBI. Specifically, this study examines the neural changes which occur as a result of an emotional processing intervention. Using functional magnetic resonance imaging (fMRI) and diffusion tensor imaging (DTI), the proposed study will examine both brain structure and function before and after the intervention. It is hypothesized that brain activity will increase following the intervention, especially in those brain regions involved in social cognition. Further, we expect that the integrity of white matter tracts connecting brain regions associated with social cognition will be improved following the intervention. The current proposal is an extension of a funded project which examines the efficacy of the emotional processing intervention on objective behavior.

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CBIR16PIL021
Mohammed Abdul Muneer Peringady, Ph.D.
New Jersey Institute of Technology
$177,469

Project Title: *A Therapeutic Approach to Alleviate Angiotensin II Induced Neurovascular*
Complications in Traumatic Brain Injury

This proposal is to undertake a therapeutic intervention to mitigate angiotensin II induced oxidative stress, hypertension, BBB impairment, neuroinflammation & neurodegeneration in blast induced TBI.

Traumatic brain injury (TBI) is characterized by physical brain injury that temporarily or permanently causes the disability by affecting cognitively, physically and emotionally. TBI causes approximately 1.7 million deaths and hospitalizations in the United States and about 20,000 people state wide annually.

Individuals exposed to explosive blast are at increased risk for TBI. Military casualties have enhanced the incidence and morbidity associated with TBI, particularly due to blast exposure. Blast TBI (bTBI) results in many neurobiological disorders, most of them lead to permanent disability. The recent clinical and experimental reports show that blast waves cause short and long term neuropathologies. While the biochemical mechanisms are still elusive, we have been trying to find out the mechanism of bTBI caused by primary blast waves. In several other biochemical mechanisms Angiotensin II (Ang II) has been reported as a mediator of oxidative stress generation via activation/induction free radical generating enzymes NADPH oxidase (NOX) and inducible nitric oxide synthase (iNOS).

In this proposal, we bring a novel hypothesis that Ang II could induce oxidative stress, which contributes the BBB disruption, neuroinflammation and neurodegeneration mediated via TBF-beta1 and MMPs signaling pathways in bTBI. This will be the first study to attempt a therapeutic approach by blocking the binding of Ang II to its receptor, Ang T1 receptor using Angiotensin receptor blocker, Losartan to prevent or reduce the neurovascular complications due to bTBI. The study will create an impact for treating BBB damage, which leads to BBB leakage, neuroinflammation and hemorrhagic stroke.

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CBIR16PIL035
Davide Comoletti, Ph.D.
Rutgers University Biomedical & Health Sciences
$180,000

Project Title: *Neuronal Cell Surface Molecules that Stimulate Myelination*

Preliminary data show that CASPR2 stimulates oligodendrocyte-neuron interactions. We will study the molecular details of how oligodendrocytes myelinate neurons in development and after brain injury. Traumatic brain injury is different from other types of bodily injury because it can affect all aspects of our lives including our physical and mental abilities, and personality. Brain injuries heal more slowly than other injuries and often incompletely. Mechanisms of recovery remain uncertain and the consequences of similar injuries may be very different. The brain of healthy humans undergoes constant turn-over of the white matter (rich in myelin) such that myelin can be added to new neuronal projections and restored after mild traumatic lesions. In TBI, damaged axons need to be remyelinated to protect neurons and facilitate recovery of neuronal function.

Oligodendrocytes are the cells in the brain that recognize the axons of the neurons and wrap around them producing the myelin sheets. At the molecular level, only a handful of molecules have been found to stimulate oligodendrocytes to myelinate neurons in the central nervous system. The question of which molecules allow the oligodendrocytes to recognize, adhere and envelop the appropriate neurons is central to understanding how myelination occurs and how to enhance it (or protect) it after traumatic episodes. Our data suggest that a protein called CASPR2 is important in these initial steps of oligodendrocyte-neuron recognition and it is sufficient to trigger myelination through their association with a specific partner on the oligodendrocyte surface.

In this project we plan to use various assays to understand the mechanistic details of how CASPR2 triggers myelination. Knowing more about CASPR2-mediated myelination has the potential to translate rapidly into better tools to improve remyelination after TBI as CASPR2 may become a target for therapeutic interventions to help a subset of patients to cope with the disease. In the future, a clear understanding of the molecular mechanisms of oligodendrocyte-neuron recognition will be important to find strategies to maximize and stimulate remyelination.

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CBIR16PIL021
Kathy Chiou, Ph.D.
Kessler Foundation
$176,776

Project Title: *Mindful Mending: Evaluating the Effects of Mindfulness Meditation on*
                 Metacognition in Moderate to Severe Traumatic Brain Injury

This study evaluates the efficacy of a mindfulness meditation program in improving metacognitive functioning after moderate to severe TBI and identifies the neural mechanisms underlying this practice.

Metacognition refers to in-the-moment awareness of performance while completing a cognitive task and is a cognitive domain that has been shown to influence community re-integration and quality of life after TBI. Problematically, adults with TBI experience metacognitive deficits after injury, but treatment options to improve metacognition are currently limited. Recently, research has emerged showing that healthy adults improve their metacognitive functioning after engaging in mindful meditation. While these study findings are promising, it remains unknown whether the efficacy of mindfulness meditation in treating metacognition also generalizes to adults with moderate to severe TBI.

This study is a randomized clinical trial designed to determine whether the practice of mindfulness meditation improves metacognitive functioning after moderate to severe TBI. Additionally, functional magnetic resonance imaging (fMRI) will be used to document the effect of mindfulness practices on neural activation after injury. Documented patterns of activation will serve as biomarkers that will be critical to advancing our understanding of neuroplastic responses to treatment after TBI.

The evaluation of meditation mindfulness as an effective approach to improving metacognition after TBI provides the first step towards the development of rehabilitative interventions for a cognitive domain that is being increasingly recognized for its important influence on functional capacities after injury. Improving metacognitive functioning in individuals with TBI will ultimately increase their ability to function independently and improve their overall quality of life.

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