DIRECTORY OF GRANT AWARDS
2024 GRANT CYCLE

NEW JERSEY COMMISSION ON BRAIN INJURY RESEARCH

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MARCH 2024

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 2024 grant cycle. The research projects are not categorized or listed in any 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 609-913-5010, 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

PILOT RESEARCH GRANT RECIPIENT:

CBIR24PIL001
Stella Elkabes, Ph.D.
Rutgers- New Jersey Medical School
$180,000

Project Title: *The risk of multiple sclerosis in adults who sustained repeated concussions during adolescence: pre-clinical studies in a new mouse model.*

The present application will establish and validate a new mouse model to investigate the mechanisms by which repeated concussions during adolescence increase the risk and severity of multiple sclerosis and its comorbidities including cognitive decline, anxiety, depression, and epilepsy in the adult, with the goal of identifying and testing new therapeutic targets.

Traumatic brain injury (TBI) is a major public health concern as it leads to permanent disability. Mild TBI/concussion constitutes the majority of all TBI cases. Young athletes in contact sports and military personnel exposed to explosive devices in combat zones often sustain repeated concussions. Repeated concussions during adolescence and young adulthood predispose the individual to neurological and neuropsychiatric disorders later in life. Multiple sclerosis (MS) is among these neurological diseases. The risk and prevalence of MS is increased in adults that sustained repeated concussions during adolescence. MS is a debilitating disease of the central nervous system that can lead to paralysis. Comorbid conditions including cognitive dysfunction, anxiety, depression, and epilepsy are also observed in some individuals with MS and significantly interfere with day-to-day activities, reducing quality of life.

The changes that occur in the brain following repeated concussions, which increase the vulnerability to MS and MS-associated comorbidities, have not been defined. The present application proposes investigations to fill this knowledge gap. The first goal is to establish and characterize a novel mouse model that combines repeated mild traumatic brain injury (rmTBI)/concussions) during adolescence with MS-like disease, namely, experimental autoimmune encephalomyelitis (EAE), in adulthood. Currently, there is no animal model to study the long-term effects of rmTBI on the onset, course, and severity of EAE and EAE-associated cognitive deficits, mood dysfunction, and epilepsy. The development and validation of a reliable and reproducible animal model is pivotal to unravel the link between rmTBI and EAE, identify new therapeutic targets, and test novel therapies.

The new mouse model will be used to decipher the metabolic, cellular, and molecular alterations that occur in the brain following rmTBI and their causative role in the subsequent development of EAE and its comorbid conditions. To this end, the adenosine system will receive particular attention since adenosine is an essential compound involved in many biological events and perturbations in adenosine metabolism have been implicated in the pathology of TBI, MS, cognitive and mood impairment, and epilepsy. Synthetic modulators of adenosine metabolism

will be tested to determine whether they hold promise as agents that prevent the onset and progression of EAE and EAE-associated comorbid conditions. It is anticipated that the findings will pave the way for future comprehensive investigations to discover and test novel therapeutic targets to alleviate the adverse and long-lasting effects of repeated concussions. The findings could also be relevant to other neuropathological conditions that develop following rmTBI.

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PILOT RESEARCH GRANT RECIPIENT:

CBIR24PIL002
Christoph Buettner, Ph.D.
Rutgers Biomedical and Health Sciences
$180,000

Project Title: *Lipolytic dysregulation in traumatic brain injury*

The proposed studies aim to assess the extent and role of lipolytic dysregulation in driving neuroinflammation and related cognitive and behavioral sequelae of traumatic brain injury.

Traumatic brain injury (TBI) bears vast economic consequences for New Jersey due to several physical, mental and cognitive disabilities in addition to the personal toll on individuals, their families, and their communities. Even in mild forms, TBI can lead to several long-term sequelae such as anxiety, depression, and memory loss, for which treatment options are limited. Persistent brain inflammation is thought to underlie these sequelae and is thus a promising target for therapy. However, what drives the chronicity of brain inflammation long after TBI is incompletely understood. A core interest of ours is to understand how impaired brain control of metabolism contributes to glucose and lipid toxicity which drive brain inflammation. Our proposal is based on four key findings. After mild TBI, rats develop long-lasting glucose intolerance, a form of prediabetes or high blood sugar after a meal. Second, high blood glucose or increased release of fatty acids from adipose tissue induce brain inflammation even in animals that did not suffer TBI. Third, treatment with a drug that normalizes blood glucose improves brain inflammation as well as memory and anxiety in rodent models of TBI. Fourth, suppression of adipose tissue lipolysis improved brain inflammation in chronic conditions such as obesity and Alzheimer's disease. These findings suggest that metabolic dysregulation is a promising target to treat brain inflammation and possibly related complications after TBI. The proposed studies will examine the role of lipid toxicity in driving brain inflammation and related complications after TBI. The support by NJCBIR will allow to obtain important preliminary data to understand how to target metabolic dysregulation, specifically as it relates to lipid metabolism and lipotoxicity, for the treatment of chronic TBI complications.

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FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR24FEL003
Shradha Suresh
Rutgers, the State University
$168,000

Project Title: *IL-6 as a therapeutic target for the treatment of TBI*

The focus of the project is to identify how changes to IL-6 levels mediate TBI-associated secondary injury and cognitive deficits.

Traumatic brain injury (TBI) results in severe neuronal damage by disrupting neural circuitry and promoting neuroinflammatory and neurodegenerative pathways. TBI is characterized by primary injury, resulting from the mechanical force applied to the head, and by secondary injury, which leads to neuronal dysfunction. Hence, patients with TBI display significant learning, memory, and cognitive deficits, resulting in a poor quality of life [1]. TBI is also associated with an increased risk for the development of various diseases, including Alzheimer's disease, Parkinson's disease, stroke, and cognitive disorders [1-3]. Naïve CD4+ cells differentiate into different T cell subtypes under the influence of specific cytokines. TBI results in an imbalance of Th1/Th2 and Th17/Treg cells, such that there is an increased presence of Th2 and Th17 cells. The altered ratio of T cell subtypes directly correlates to an increased presence of pro-inflammatory cytokines [5]. The cumulative effect of the build-up of these pro-inflammatory cells and cytokines is the breakdown of the blood-brain barrier (BBB) and recruitment of inflammatory T cells into the CNS [6]. IL-6 is a pleiotropic cytokine that triggers a pro-inflammatory immune response [7]. However, the pleiotropic activity of IL-6 is contingent upon the formation of the IL-6/IL-6R complex and subsequent binding to gp130 [5]. Hence, we hypothesize that interfering with the formation of the IL-6/IL-6R complex by using an anti-IL6R antibody (MR16-1) will suppress neuroinflammation by re-establishing the ratio of Th17/Treg cells, which will consequently allow for the maintenance of an intact BBB [8-11]. As such, we propose the administration of MR16-1 as a way to promote recovery from TBI-induced deficits in the controlled cortical impact (CCI) animal model of TBI. The antibody will be injected intraperitoneally, and as indicated in previous studies using MR16-1, will interfere with the formation of the IL-6/IL-6R complex and suppress the subsequent activation of inflammatory signaling pathways [12, 13]. If this study proves successful, we will have identified a novel therapeutic approach with translational applications in humans. Considering the association between IL-6-mediated neuroinflammation and other debilitating neurodegenerative diseases, our results will aid in developing a novel approach for treating TBI and these additional pathologies.

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FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR24FEL007
Victoria Stiritz
Rutgers Biomedical and Health Sciences
$168,000

Project Title: *Impulsivity After Head Trauma – Exploring the Effects of mTBI on Negative Urgency and Inflammation*

This study aims to model negative urgency through operant avoidance and examine the effect of mild traumatic brain injury (mTBI) on its expression and the neurochemical changes that may be contributing to this phenomenon.

Suicide rates among veterans have climbed notably higher than that of civilians over the past 20 years. Suicidality is complex, with a plethora of factors contributing to its development. Impulsivity has been identified as a key risk factor yet is multifaceted itself. Negative urgency is a dimension of impulsivity that encompasses experiencing strong impulses during negative emotional states. Given that military personnel are subject to periods of prolonged or extreme stress, studying how and why negative urgency may be exacerbated in these circumstances can lead us to new developments to keep our troops and veterans safe. Another key risk factor of suicidality is mild traumatic brain injury (mTBI). Impulsivity has also been included in the psychological sequelae of mTBI. Despite this overlap, the potential causal relationship between mTBI and the development of impulsivity has yet to be elucidated. This study aims to examine if the intersection between mTBI and suicidality is by fostering negative urgency due to inflammation in select brain regions previously associated with impulsivity. Briefly, after learning to lever-press to avoid shock, rats will sustain a single mTBI using the lateral fluid percussion injury model. After a short recovery period, performance on the task will be assessed at 1 week, 1 month, 3 months, and 6 months post-injury. Negative urgency is expected to be expressed as greater non-reinforced lever pressing. Inflammation in regions associated with impulsivity such as the raphe nuclei, locus coeruleus, and ventral tegmental area will be examined at the same time points. Upon completion of this study, we expect to gain powerful insight into the neurobiological mechanisms underlying behavioral changes experienced after mTBI, as well as develop a novel paradigm to model impulsivity.

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FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR24FEL009
Brandon Vaglio
Rutgers, the State University
$168,000

Project Title: *Engineered Exosomes as a Therapeutic for Secondary Traumatic Brain Injury*

Engineered exosomes with a neuronal targeting peptide and loaded with siRNA will be used to knockdown the protein Preso and destabilize the NMDAR/PSD-95/nNOS complex as a therapeutic for secondary traumatic brain injury.

Traumatic brain injury (TBI) is a leading cause of death and disability that can result from motor vehicle accidents, falls, and violent assaults. While public understanding of TBI focuses on the physical trauma that initiates the injury, there are many biochemical processes that can continue for months after the injury and can worsen the symptoms. These processes are encompassed under the term secondary TBI. In order to restore quality of life for TBI patients (including preserving cognitive and motor functions), therapeutic and rehabilitative treatments must target the different secondary TBI mechanisms. The protein Preso was identified in 2008 and has been implicated in playing a role in glutamate-induced excitotoxicity, a secondary TBI process that contributes to increased neuronal death. While researchers have demonstrated improved neuronal survival by knocking down the expression of Preso in TBI models, there are large gaps in knowledge related to the functional effects of Preso knockdown as a TBI therapeutic. We seek to knockdown Preso with the utilization of a short interfering RNA (siRNA) construct for Preso that has been loaded in exosomes, extracellular vesicles secreted by cells that are suitable for targeted drug delivery. The Firestein lab has established a protocol to extract exosomes that have a modified surface for neuronal targeting. We will test the siRNA loaded exosomes with cortical cultures and in a mouse TBI model to understand whether Preso knockdown preserves neuronal network firing, maintains baseline cognitive behavior, and impacts other secondary TBI mechanisms that induce neuronal death.

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FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR24FEL012
Seanna Kelly
Rutgers, The State University
$168,000

Project Title: *The role of immune response and glial subtypes on TBI-induced sleep changes.*

This project leverages a calibrated, closed-head injury model in Drosophila for candidate screening of genes involved in regulating sleep, lifespan, and healthspan in neurons and glia after traumatic brain injury.

Traumatic brain injury (TBI) is an umbrella term for head injuries caused by external forces or mechanisms including falls, motor vehicle accidents, and sports accidents. TBI survivors experience many long-term symptoms, including sleep disorders such as insomnia, hypersomnia, sleep apnea, and narcolepsy, which can persist for months or years after injury. Sleep is essential to human health, and sleep disorders are a contributing factor in memory issues, mood disorders, headaches, metabolic syndrome, all of which are also common post-TBI symptoms. While advances in medicine have increased survival from primary TBI, there are no pharmacological options to treat the chronic secondary pathologies present in the growing population of TBI survivors. This proposal seeks to identify genetic changes in the brain after TBI that drive sleep disorders. Identifying these genes, and which type of cells they act in, will allow development of targeted therapeutics to improve sleep after TBI, and potentially ameliorate other post-TBI pathologies. To identify these genes, I will use the fruit fly, Drosophila melanogaster. Fruit flies have been used for fundamental discoveries in behavioral genetics for nearly a century. Genetic and cellular similarities between flies and mammals mean that genes discovered in flies have similar roles in mammalian systems. We will use a novel approach to administer TBI to large numbers of flies, which will allow us to screen a library of genetically altered flies to determine the role of each gene in sleep changes after injury. Using fly genetic tools, we can also determine whether these genes act in neurons, the electrical signaling cells of the brain, or in glia, the support cells that regulate neuronal activity. There are many types of glial cells in fly and mammalian brains, each of which serves unique functions. We know that activation of a key genetic signaling pathway, called AP-1, occurs in glia after TBI in both flies and humans and contributes to changes in brain function and survival. However, which specific glial cell types this occurs in is unknown. I will identify which kinds of glia show AP-1 activation after injury in flies and determine whether activation of AP-1 in each glial type affects sleep and survival. This will allow better identification of both the roles of glia in sleep after TBI and further characterization of key genes that may serve as therapeutic targets to treat post-TBI sleep disorders. Successful completion of this proposal will provide insight into the molecular mechanisms causing post-TBI sleep disruptions. These findings can be further used for the development of targeted therapeutics to alleviate sleep disruption in TBI patients, increasing their quality of life.

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FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR24FEL013
Srinivasa R Gandu
Rutgers, the State University
$307,080

Project Title: *The role of cypin in regulating ubiquitin and ubiquitin-like post-translational modifications in recovery after TBI*

This study aims to understand how post-translational modifications affect the synaptic proteins in recovery after a TBI and the role cypin has in regulating these PTMs to strengthen neuronal connections.

Traumatic brain injury (TBI) is caused by direct or indirect impact on the head, resulting in brain movement that leads to neuronal injury and damage. TBI occurs in two phases, a primary or mechanical phase followed by prolonged activation of glutamate receptors, leading to a secondary or excitotoxic phase. TBI affects neuronal connectivity, which leads to cognitive deficits over time, and ultimately, causes neuronal death. Multiple TBIs are implicated in a number of neurodegenerative disorders. In this proposal, we will study how ubiquitin (Ub) and ubiquitin-like (Ubl) proteins post-translational modifications on synaptic proteins change after suffering a brain trauma. We will use cellular and animal models to determine changes to the levels of free Ub and Ubl and Ub- and Ubl-tagged proteins and how these changes correlate to neuronal recovery following a TBI. We will also study the effects of overexpression of the cytosolic PSD-95 interactor (cypin), a key regulator of changes to the Ub- and Ubl-tagging of synaptic proteins. Cypin has a neuroprotective function after TBI, and thus, our study will help in identifying potential therapeutic targets to aid in neuronal recovery following a TBI.

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FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR24FEL015
Aubree Alexander
Kessler Foundation
$260,216

Project Title: *The PUPIL (Play, Understand, Partner, Interact, and Learn) Program: Developing a caregiver curriculum and game to supplement an evidence-based cognitive rehabilitation protocol*

The current study aims to supplement an existing, evidence supported cognitive rehabilitation intervention with a newly developed, stakeholder-guided, game and caregiver psychoeducational curriculum (The PUPIL Program) that strives to increase use of cognitive strategies in daily life and to foster caregiver engagement in cognitive rehabilitation initiatives.

The current study addresses the need for accessible and affordable cognitive rehabilitation interventions that engage and train caregivers alongside individuals with traumatic brain injury (TBI). In the current proposal, a diverse group of stakeholders guides development of the PUPIL (Play, Understand, Partner, Interact, and Learn) program. The PUPIL program is an add-on module to an existing cognitive rehabilitation intervention, with demonstrated efficacy, called the Kessler Foundation Modified Story Memory Technique (KF-mSMT®). The PUPIL program includes an educational caregiver curriculum and a board game and encourages individuals with TBI, and their caregivers, to practice using cognitive strategies while playing a game. The ultimate goal is to engage caregivers in cognitive rehabilitation, educate caregivers about cognitive strategies learned in treatment, and teach ways to increase strategy use in daily life. The study has three phases. In Phase 1, diverse groups of stakeholders will provide feedback on the proposed PUPIL program. In Phase 2, information obtained in Phase 1 will be used to refine and further develop the PUPIL program. Phase 3 includes a preliminary evaluation of the PUPIL program, together with the KF-mSMT® and those who participate in the initial evaluation of the program will be interviewed about their experiences with, and opinions of, the PUPIL program.

The proposed project has the potential to develop an intervention that can benefit individuals with brain injury and their family members and/or caregivers. The project supports the education and training of caregivers and offers accessible, affordable, and engaging activities to practice cognitive strategies at home.

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INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR24IRG005
Bonnie L. Firestein, PhD
Rutgers, The State University
$540,000

Project Title: *A noninvasive AAV-based therapeutic for treatment of TBI*

We will intravenously administer novel engineered adeno-associated viral (AAV) vectors to target overexpression of cypin in the brain of mice with TBI to promote functional recovery.

Traumatic brain injury (TBI) is a leading cause of death and disability in the young and elderly, yet there remains no successful treatment strategy to improve outcome in TBI survivors. We identified a protein, called cypin, that protects cognition and accelerates recovery after TBI. Recently, we reported that targeting cypin therapeutically with small molecule activators improves neurocognitive outcome after TBI in mice. However, since the process of optimizing these molecules for eventual use in humans is lengthy, we propose to develop different types of therapeutics to increase cypin in parallel. In the current studies, we will develop a novel strategy to deliver DNA that encodes cypin specifically to the brain in mice. This will result in increased cypin levels only in the brain, and specifically in neurons, which would aid in recovery after TBI. Our studies are directly related to NJCBIR priorities since we are identifying therapeutic agents and targets for the treatment of TBI. Our ultimate goal is to develop a novel therapeutic for translation to patients who have experienced a TBI.

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INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR24IRG009
Peii Chen, Ph.D.
Kessler Foundation
$536,728

Project Title: *Time Course and Impact of Spatial Neglect on Brain Injury Recovery*

The study aims to establish the time course, clinical impacts, and neural mechanisms of spatial neglect in people who sustained traumatic brain injury.

Spatial neglect occurs in approximately 30% of individuals who receive inpatient rehabilitation care after traumatic brain injury (TBI). Through extensive work in neuropsychological and rehabilitation research in people with stroke, much has been learned about spatial neglect including its brain mechanisms, various symptom presentations, and impacts on mobility, self-care management, and real-life activities. However, the time course and clinical impact of spatial neglect on TBI recovery is unknown. This knowledge gap has hindered the progress in research as well as clinical care regarding TBI rehabilitation. The proposed study will close this knowledge gap and be the first prospective, longitudinal project to understand the behavioral and brain changes over time in individuals with spatial neglect post TBI.

The overall objective of the proposed project is to determine how spatial neglect affects functional improvement among people with moderate to severe TBI. We hypothesize that damage to the white matter tracts in the attention networks will be associated with spatial neglect in the TBI population, and reduced white matter integrity in inter-hemispheric tracts will be associated with chronic spatial neglect and poor functional recovery. The success of the proposed 3-year project will generate solid evidence of spatial neglect post TBI and encourage researchers and clinicians to investigate TBI-specific interventions for the disorder.

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INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR24IRG017
Anthony Lequerica, Ph.D.
Kessler Foundation
$435,689

Project Title: *Sleep-wake Objective MeasuremeNt Of adoLEscents Navigating TBI (SOMNOLENT study)*

This project will examine 2 promising indicators of sleep-wake cycle disturbance derived from wrist accelerometry to better understand their properties in an adolescent TBI rehabilitation setting.

Traumatic brain injury (TBI) often results in disruption of brain processes involved in the regulation of the sleep-wake cycle. In the inpatient rehabilitation (IR) setting after TBI, sleep aids are frequently prescribed, but there is less reliance on objective measures of the sleep-wake cycle to guide treatment. As the gold standard in sleep assessment, polysomnography (PSG), involving application of electrodes to scalp, face, chest, and legs, is costly and invasive and not feasible for routine use in the IR setting. Actigraphy, on the other hand, using a wrist-worn device, captures rest-activity cycles in a less expensive and less invasive manner. It records information that is analyzed by computer software and shown to differentiate sleep vs. wake with over 90% accuracy compared with the gold standard of PSG. Because it can be worn continuously over many days and nights, it is useful in the study of circadian rhythms that drive sleep-wake regulation. Two actigraphy indices show great promise for assessing sleep quality and sleep cycle regulation: the sleep fragmentation index (SFI) and rest-activity ratio (RAR). However, there is a scarcity of studies examining these indices in children, and research regarding how they relate to adolescents with TBI in the IR setting is almost nonexistent. The proposed study aims to examine these sleep-wake indices to gain a better understanding of their relevance among adolescents with TBI at a point in recovery where quality sleep is crucial both for neural repair and promoting greater efficiency during the day where patients are expected to work hard to achieve treatment goals. This study will set the groundwork for future studies examining their utility as a routine part of clinical care to identify adolescents in need of more extensive evaluation using PSG, as well as studies examining the effectiveness of treatments for post-TBI insomnia so that patients can make the most of their time in IR and improve long term outcomes.

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