DIRECTORY OF GRANT AWARDS
2026 GRANT CYCLE
APRIL 2026

NEW JERSEY COMMISSION ON BRAIN INJURY RESEARCH

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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 2026 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, 25 South Stockton Street, Trenton, New Jersey, 08625. The Commission's office can be reached by telephone at 609-913-5010, 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.

NEW JERSEY COMMISSION ON BRAIN INJURY RESEARCH

GRANT AWARDS

INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR26IRG003
Martin Yarmush, M.D., Ph.D.
Rutgers, The State University of New Jersey
$540,000

Project Title: *Targeted Mitochondrial Transplantation as a TBI Therapeutic*

Our goals are to halt secondary injury by repairing the blood-brain barrier (BBB) at its source—by restoring mitochondrial function within endothelial cells, the keystone element of BBB structure and function

Traumatic brain injuries are widespread and impose significant burdens on the healthcare system. As a result of both primary and secondary traumatic brain injury, the blood brain barrier is impaired, allowing toxic and inflammatory molecules to infiltrate the neural microenvironment, perpetuating neurodegeneration and neuroinflammation. Mitochondrial dysfunction at the blood brain barrier occurs within hours of TBI and is a key initiator of the downstream secondary traumatic brain injury effects, highlighting it as a key therapeutic target.
These secondary effects contribute to injury progression and patients' neurological symptoms ranging from headaches and blurry vision or more severe cognitive and motor impairments. Current treatments focus on managing symptoms and reducing downstream neuroinflammation with anti-inflammatory drugs, but they are unable to curtail the early and continued influx of harmful molecules through the impaired blood brain barrier, which drives secondary TBI. Therefore, early intervention aimed at repairing the blood brain barrier is crucial. This study aims to investigate the therapeutic potential of attenuating mitochondrial dysfunction in in vitro

endothelial barriers, the main component of the blood brain barrier, and in in vivo murine models of traumatic brain injury. Mitochondrial dysfunction can be attenuated via mitochondrial transplantation, which has successfully recovered endothelial barrier injuries of the heart, lungs, and kidneys. We will improve upon previous approaches by developing antibody containing mitochondrial vesicles that will specifically target endothelial barrier cells, leaving bystander cells unaffected. Successful completion of these studies will provide evidence for a new therapeutic target, mitochondrial dysfunction, to restore the endothelial barrier in post-traumatic brain injury blood brain barrier breakdown.

Contact Information

Martin Yarmush, M.D., Ph.D.
Rutgers, The State University of New Jersey
599 Taylor Road
Piscataway, New Jersey 08854
(732) 445-6530
yarmush@soe.rutgers.edu

INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR26IRG004
Rachel Navarra, Ph.D.
Rowan University
$540,000

Project Title: *A non-stimulant based approach to restore executive dysfunction and catecholamine imbalance following repetitive mild traumatic brain injury*

This project examines whether atomoxetine restores prefrontal catecholamine signaling, mitigates neuropathology, and improves executive function in male and female rats after repetitive mild TBI, providing mechanistic insight into targeted recovery strategies.

Traumatic brain injury (TBI) is a major public health concern, with repeated mild TBIs (rmTBIs) common among athletes, military personnel, and other at-risk populations. While single mild TBIs are often considered minor, repeated injuries can lead to long-term cognitive problems, including difficulties with decision-making, planning, and adapting to new situations, abilities collectively known as "executive functions." For those at risk of rmTBI, such executive deficits compromise academic, occupational, and social performance, severely disrupting pre-injury routines, with recovery influenced by the number of prior injuries and individual variability. These deficits are thought to arise from disruptions in chemical messengers, called catecholamines, in the prefrontal cortex of the brain. Currently, there are no evidence-based treatments to restore these functions after rmTBI.

This project tests whether atomoxetine (ATX), an FDA-approved, non-stimulant medication used to treat ADHD, can improve executive function after repeated head injury. ATX increases the levels of key catecholamines, norepinephrine and dopamine, in the prefrontal cortex. This mechanism is hypothesized to improve executive impairments following rmTBI, by restoring chemical balance, reducing injury-related brain inflammation, and protecting against progressive brain damage. Using a validated rodent model of rmTBI, we will assess ATX's effects on two specific executive functions, cognitive flexibility and risk/reward decision-making. We will also examine sex-dependent responses and underlying brain pathology.

The proposed research aligns directly with NJCBIR priorities by advancing understanding of how repeated mild brain injuries cause cognitive deficits, evaluating a mechanistic-based treatment that may prevent secondary brain damage, and promoting recovery of function. The project leverages interdisciplinary collaboration among established experts in brain injury, behavioral neuroscience, and neurochemistry, establishing a translational platform to guide clinical interventions. Findings will inform treatment strategies, rehabilitation approaches, and evidence-based guidelines to improve independence, quality of life, and long-term outcomes for individuals living with repeated mild TBI.

Contact Information

Rachel Navarra, Ph.D.
Rowan University
2 Medical Center Drive
Stratford, New Jersey 08084
(856) 566-6819
navarra@rowan.edu

INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR26IRG013
Mauricio Lillo, Ph.D.
Rutgers Biomedical and Health Sciences
$540,000

Project Title: *Cx43 Hemichannels as a Therapeutic Target in TBI*

This project investigates how connexin 43 hemichannels initiate calcium influx that drives pannexin-1 activation, blood–brain barrier disruption, and cognitive decline after traumatic brain injury, testing whether genetic or pharmacological blockade of hemichannel–pannexin coupling preserves endothelial integrity, cerebral blood flow, and neurological function, thereby defining an unexplored therapeutic target for TBI.

Traumatic brain injury (TBI) is a major public health problem and one of the leading causes of long-term disability worldwide. Each year, millions of people sustain a TBI, and many survivors face persistent challenges such as memory loss, difficulty concentrating, mood changes, and problems with daily activities. These long-term effects do not only impact patients but also place a tremendous burden on families, caregivers, and the healthcare system. Currently, there are no effective treatments to directly prevent or repair the brain damage caused by TBI, making the search for new therapeutic strategies urgent and essential.

A major contributor to the lasting effects of TBI is damage to the blood–brain barrier (BBB). The BBB is a protective layer of specialized blood vessel cells, called endothelial cells, that regulates what substances can enter the brain from the bloodstream. Under healthy conditions, the BBB blocks harmful toxins, excess immune cells, and other damaging molecules from reaching brain tissue. After a TBI, however, the BBB often becomes "leaky," allowing harmful substances to pass into the brain. This leakiness triggers inflammation, swelling, and injury to neurons, which together contribute to memory deficits and other cognitive problems. Protecting the BBB is therefore considered a critical step toward improving recovery after TBI.

Our research focuses on understanding the molecular "gateways" in endothelial cells that cause the BBB to fail after injury. We have discovered that connexin 43 hemichannels—small pores in the endothelial cell membrane—play a crucial role in initiating BBB breakdown. When inflammatory signals such as the cytokine TNF-a are released after TBI, these hemichannels open and allow calcium ions to flood into the cells. This abnormal calcium entry then activates another pore-forming protein, pannexin-1. Once active, pannexin-1 channels release ATP, a signaling molecule that further amplifies inflammation, weakens the BBB, and sustains brain injury. In essence, connexin hemichannels "unlock" the pathway that sets off a damaging cycle of calcium influx, pannexin activation, inflammation, and BBB leakiness.

Importantly, we have shown that blocking connexin hemichannels with a specific inhibitor, called Gap19, prevents much of this harmful signaling. In laboratory experiments, Gap19 reduced calcium entry, pannexin-1 activation, and barrier leakage in endothelial cells. In animal models, it preserved BBB integrity and reduced brain inflammation. We have also generated unique genetic mouse models where connexin hemichannels are selectively impaired, but gap junction communication (important for normal brain function) is preserved. These mice show less BBB damage and improved outcomes after TBI, supporting the idea that connexin hemichannels are a promising therapeutic target.

The goal of this project is to understand exactly how connexin hemichannels and pannexin-1 channels interact to damage the BBB after TBI, and to test whether blocking this interaction can protect brain function. In our first aim, we will study brain endothelial cells in the lab to determine how connexin

hemichannels control pannexin activation during inflammation. We will measure calcium dynamics, pore activity, and ATP release using advanced imaging and electrophysiology techniques. In our second aim, we will test whether blocking hemichannels improves memory and learning after TBI in mice. We will assess BBB integrity using imaging and dye-based assays, measure brain blood flow, and evaluate cognitive recovery using maze-based learning tests.

By clarifying how connexin hemichannels act as the "trigger" for pannexin-driven barrier disruption, this research will identify a new pathway that can be targeted to protect patients after TBI. Unlike current therapies, which mainly treat symptoms, targeting this mechanism has the potential to directly preserve BBB function, reduce brain inflammation, and improve memory recovery.

Contact Information

Mauricio Lillo, Ph.D.
Rutgers Biomedical and Health Sciences
185 South Orange Avenue
Newark, New Jersey 07103
(201) 687-2397
mlillo.gallardo@rutgers.edu

INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR26IRG015
Sridhar Kannurpatti, Ph.D.
Rutgers Biomedical and Health Sciences
$509,888

Project Title: *Sequentially timed treatments to improve brain concussive injury recovery*

TBI patients have a long rehabilitation timeline and currently with no viable treatment either for the early stages or the late stages. There is a need to develop TBI therapeutics not only for the early stage of rehabilitation soon after injury, but also for the late stages of several months to a year after injury. Proposed preclinical TBI study will develop a dual therapeutic regimen (provided during early and late-stages with two promising and novel agents) for improved TBI rehabilitation.

The Center for Disease Control has estimated five-year traumatic brain injury (TBI) outcomes where 22% died, 30% became worse, 22% stayed the same, while only 26% showed some form of improvement. Most mild to moderate TBI (mmTBI) patients survive, but individuals with mmTBI often do not get appropriate clinical attention. In long term follow up studies (up to 5 years after injury), mmTBI patients have been found to be significantly neglected during early rehabilitation (first few months after injury) with only 21% receiving any form of clinically structured rehabilitation (2). Lack of early and intermediate-stage rehabilitation along with no viable therapeutic interventions leads to memory problems, decreased processing speed, irregular sleep, fatigue, impulsivity, anxiety and depression. Progress is being made in developing neuroprotective strategies to reduce brain injury and several FDA approved drugs are being investigated; however, clinical trials are still in the early stages and paradigms are lacking to treat both the early-stage complications as well as delayed neurodegeneration. Addressing this critical gap, this study proposes to evaluate two treatments provided sequentially after mmTBI. The first therapeutic will be administered beginning several hours after the mmTBI and then daily for the following week. This therapeutic, kaempferol, is a natural flavonoid that is found in a variety of plants that include kale, spinach and broccoli. Our published and preliminary data show that kaempferol, has great promise as a therapeutic in two different rodent models of mmTBI where it promotes neuronal survival and improves brain structural integrity and neurological function. The second therapeutic to be evaluated is a protein that is naturally produced by the brain in response to injury called Leukemia Inhibitory Factor (LIF). But the brain only produces this protein for a short period of time after injury. In ongoing studies, we have shown that providing nose-drops containing LIF during the chronic stage of recovery (6-8 wks) from a mmTBI prevents axonal injury, improves brain structural integrity and improves neurological function. Studies suggest that these therapeutics act through different mechanisms, which allows the prediction that they can be used with therapeutic benefit as early and late-stage therapeutics over the mmTBI rehabilitation timeline. Magnetic resonance imaging (MRI) based approaches that can be used for individuals in the mmTBI spectrum will be used in combination with histological, biochemical and behavioral measures so that our work can readily advance into potential clinical trials.

Contact Information

Sridhar Kannurpatti, Ph.D.
Rutgers Biomedical and Health Sciences
185 South Orange Ave
Newark, New Jersey 07101
(973) 972-5188
kannursr@njms.rutgers.edu

INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR26IRG025
Denise Krch, Ph.D.
Kessler Foundation
$497,607

Project Title: *A Dose Optimization Trial of Wonderworks, a Web-based Treatment for Impaired Attention after TBI*

This methodologically rigorous, dose-optimization randomized controlled trial will define the optimal therapeutic protocol for Wonderworks, an innovative, web-based intervention for attention deficits after TBI, by modeling the dose-response curve over 12 sessions to provide the essential data required for a future, large-scale efficacy trial.

Traumatic brain injury (TBI) is a major public health challenge, impacting approximately 175,000 New Jersey residents, with an additional 12,000 new injuries occurring each year. For many survivors, the long-term consequences include persistent and disruptive deficits in attention. These challenges can severely impact a person's ability to live independently, maintain employment, and manage a household. Everyday situations that require mental flexibility - such as following a complex recipe while holding a conversation or navigating a busy grocery store - can become overwhelming. While clinic-based therapies exist, they often involve burdensome travel and use simplified drills that do not fully prepare individuals for the complex cognitive demands of the real world.

This project will conduct a dose-optimization trial of Wonderworks, an innovative, web-based training program designed to improve attention skills after TBI. Wonderworks is a comprehensive, clinician-guided therapy that addresses the limitations of traditional approaches. It uses an engaging, game-like virtual environment that mimics the demands of a real-world job, requiring individuals to practice dividing their attention and alternating their focus. The program is more than just software; it includes personalized feedback and strategy training from a dedicated clinician, who helps participants transfer these new skills to their specific daily life challenges through practical homework. The software's advanced algorithm also continuously adjusts the difficulty to match each user's abilities. To ensure equitable access for all participants during the trial, loaner laptop computers and internet hot spots will be provided to those who need them.

The primary goal of this study is to answer the critical question: "What is the optimal amount of this therapy?" To do this, we will recruit 60 individuals with TBI from the Kessler Foundation. To ensure a fair and unbiased comparison, participants will be randomly assigned to one of two groups. One group will complete the 12-session Wonderworks program, while a second group will participate in a Holistic Cognitive Education program, which provides general information about brain health. By measuring the Wonderworks group's progress at multiple time points with a high-precision eye-tracking test - an advanced technology that provides an objective window into the brain's attention skills, allowing us to see subtle cognitive improvements that might otherwise be missed - we will map their rate of improvement. This analysis will allow us to identify the optimal "dose" needed to achieve the maximum therapeutic benefit. The results of this study will provide the essential roadmap for delivering this promising new tool to New Jersey residents in the most effective and efficient way possible.

Contact Information

Denise Krch, Ph.D.
Kessler Foundation
120 Eagle Rock Avenue
Suite 100
East Hanover, New Jersey 07936
(973) 324-8392
dkrch@kesslerfoundation.org

INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR26IRG026
David J Margolis, Ph.D.
Rutgers, The State University of New Jersey
$536,724

Project Title: *Investigating neural plasticity in the dorsal striatum for multi-symptom recovery from traumatic brain injury*

This project uses mouse models to investigate the role of the striatum in recovery from traumatic brain injury.

New therapies based on understanding the brain's response to traumatic brain injury (TBI) are urgently needed. In this collaborative project, four neuroscientists aim to discover how a brain region called the striatum responds to TBI and how its function can be harnessed for recovery. The PIs have expertise in powerful methods for measuring and manipulating brain function and behavior in mouse models and will take a multipronged approach to investigating the integration of signals in the striatum before and after TBI. The results will set the stage for developing new therapies with potential for better recovery from the debilitating effects of TBI.

Contact Information

David J Margolis, Ph.D.
Rutgers, The State University of New Jersey
604 Allison Rd
Nelson B243
Piscataway, New Jersey 08854
(848) 445-9533
david.margolis@rutgers.edu

INDIVIDUAL RESEARCH GRANT RECIPIENT:

CBIR26IRG029
Pabitra Sahoo, PhD
Rutgers, The State University - Newark
$540,000

Project Title: *Regulation of Local App mRNA Translation by Axonal Stress Granules Post Traumatic Brain Injury*

In this proposal we aim to test how cortical TBI activates proNGF signaling to release App mRNA from BFCN axonal SGs to induce local synthesis of APP, which then promotes axon degeneration.

Traumatic brain injury (TBI) to the brain's cortex can damage neurons not only at the injury site but also in surrounding areas. This damage is linked to specific proteins that trigger a process that causes nearby neurons to die. One group of neurons, called basal forebrain cholinergic neurons (BFCNs), are particularly affected because they send long connections to the cortex. When these connections are injured, it can lead to the degeneration of the neurons themselves. Our research has found that after a brain injury, a protein called amyloid precursor protein (APP), which is linked to Alzheimer's disease, plays a crucial role in the death of these neurons. In healthy neurons, the instructions (mRNA) to make APP are stored in a dormant state, but after an injury, these instructions are activated, and APP is produced in large amounts. We are investigating how the injury turns on this process and causes damage. Our study will explore three key areas: (1) Define how G3BP1-containing axonal SGs regulate App mRNA storage and release in BFCNs (2) Identify the molecular mechanisms downstream of G3BP1 granules that determine BFCN survival (3) Test whether targeting G3BP1 granules protects BFCNs after TBI in vivo. By the end of this project, we hope to understand how brain injuries cause neuron death and identify potential ways to protect neurons after trauma.

Contact Information

Pabitra Sahoo, PhD
Rutgers, The State University - Newark
225 University Avenue
LSC-601C
Newark, New Jersey 07102
(973) 353-3493
pabitra.sahoo@rutgers.edu

FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR26FEL004
Xinnian Jiang
Rutgers, The State University of New Jersey
$168,000

Project Title: *Microneedle-Based Electrochemical Biosensing Platform for Monitoring of Traumatic Brain Injury Biomarkers*

Development of a microneedle-based electrochemical biosensing platform for continuous and minimally invasive monitoring of glutamate, lactate, and uric acid in interstitial fluid to enable objective assessment of traumatic brain injury rehabilitation.

Traumatic brain injury (TBI) is one of the leading causes of disability and death worldwide, affecting millions of people every year and creating long-term health, social, and economic burdens. Patients with TBI often suffer from memory loss, difficulties in movement, and other chronic complications that can persist for years. Unfortunately, current methods for monitoring recovery from TBI rely heavily on subjective scoring systems or intermittent imaging, which cannot capture the rapid chemical changes happening in the brain. As a result, clinicians lack objective tools to track rehabilitation in real time and to provide timely, personalized treatments. This project aims to fill this gap by developing a new wearable biosensing platform that can continuously and non-invasively monitor key brain-related chemicals in the fluid just beneath the skin. Using tiny microneedles that are virtually painless, the device will detect important biomarkers such as glutamate, lactate, and uric acid. These chemicals are known to reflect brain injury severity and the body's healing process, offering a direct window into what is happening inside the brain during recovery. By integrating electrochemical sensors with a wireless communication system, the platform will allow real-time tracking of brain chemistry in freely moving subjects. The research will begin with the design and testing of the microneedle-based sensors in the laboratory, followed by validation in animal models of TBI. Data from the device will be compared with established laboratory methods and behavioral assessments to ensure accuracy and reliability. If successful, this approach will provide the first minimally invasive, continuous, and objective system for monitoring TBI rehabilitation. The long-term vision of this work is to translate the technology from animal models to human patients, ultimately offering doctors a new tool to personalize treatment, evaluate therapy effectiveness, and improve recovery outcomes. Beyond TBI, the same sensing platform could also be adapted to monitor other neurological and metabolic disorders where biochemical tracking is crucial. By providing real-time insights into brain recovery, this project has the potential to transform how we monitor and treat TBI, reduce healthcare costs, and improve the quality of life for patients and their families.

Contact Information

Xinnian Jiang
Rutgers, The State University of New Jersey
599 Taylor Road
Piscataway, New Jersey 08854
(732) 558-7653
xinnian.jiang@rutgers.edu

FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR26FEL014
Tulika Das
New Jersey Institute of Technology
$307,080

Project Title: *Preventing Monocyte Infiltration in Repeated Low-Level Blast Traumatic Brain Injury Attenuate Injury Induced Behavioral Deficits and Chronic Neuronal Loss*

Repeated exposure to low level blast waves causes BBB damage allowing peripheral monocytes to enter CNS further upregulating inflammation mediated neuronal loss leading to chronic behavioral deficits severity of which varies with the increasing number of the exposure.

Blast induced TBI (bTBI) or what was known as "shellshock" during world war I and in 1990s and gained a new name "mild TBI" was still not officially accepted as distinct diagnosis till 2005 [12]. While mild TBI and concussion shares many similarities, bTBI is unique in ways how the injury impacts the brain tissue and the diffusive nature of the injury. When a shockwave of supersonic speed hits the brain, it induces rapid contraction and relaxation of brain tissue and its vasculature [13]. Repeated exposure to shockwave is an occupational hazard for service members and law enforcement personnels. Some even argue exposure to single shockwave can be regarded as repetitive due to the multiple factors leading to a cascade of events like primary intracranial pressure wave generation, movement of head and increase of pressure in the vascular system [14]. Despite growing number of studies, a lot is still unknown about bTBI, specifically repeated low intensity level bTBI (rLLB).

Primary damage of BBB has been shown to arise from the biomechanical loading of the pressure wave [7]. It is believed to initiate oxidative stress and serve as a signal for microglial activation at acute phase. In our lab previously, rats exposed to 180 kPa blast pressure wave demonstrated NOX-mediated oxidative stress as a major causative factor in BBB disruption at sub-acute stages following the injury [7, 15]. The mechanical trauma can also result in neuroinflammation where astrocytes and microglia initiate non-specific immune response by releasing damage-associated molecular patterns (DAMPs). Peripheral immune cells including monocytes and neutrophils in the inflammatory cascade migrate into the CNS, an immune privileged site, due to breach in BBB from the injury [16]. Monocytes upon entering CNS get activated into macrophage and share similar morphology as activated microglia. To distinguish between the monocyte derived macrophage (MDM) and activated microglia, a double transgenic CX3CR1GFP/+: CCR2RFP/+ mouse model has been generated, in which resident microglia are labeled with GFP and peripheral monocytes with RFP. Using these transgenic animals, an earlier study of single 180 kPa BOP injury showed microglial activation and monocyte infiltration as early as 4 hours post injury and lasted through 30 days post injury suggesting chronic inflammation [8]. This study also confirmed a decline in neurocognitive behavior following the injury.

The central hypothesis of this study is: Repeated exposure to low-level blast causes BBB damage allowing peripheral monocytes to enter the CNS exacerbating inflammation mediated neuronal loss leading to chronic behavioral deficits, which varies with the increasing number of exposures. Preventing BBB permeability with the membrane sealing agent P188 will reduce monocyte infiltration and the associated development of cognitive decline and neuronal loss.

Contact Information

Tulika Das
New Jersey Institute of Technology
323 Dr. Martin Luther King Jr Blvd
Newark, New Jersey 07102
(862) 315-1991
td256@njit.edu

FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR26FEL015
Li Ling Goldston
Rutgers, The State University of New Jersey
$147,000

Project Title: *Deferoxamine (DFO) Primed BDNF-Enriched Drug Loaded Extracellular Vesicles (EVs) from Neural Stem Cells for Enhanced Neural Repair Following TBI*

Develop Nrf2 activating BDNF-enriched engineered extracellular vesicles (EVs) to promote neural stem cell survival and differentiation following traumatic brain injury.

The effective regeneration of neural tissue following traumatic brain injury (TBI) is a major challenge for TBI treatment due to the complex pathophysiological mechanisms that accompany the injury. The primary injury is a significant mechanical insult to the tissues, and the secondary injury is characterized by blood-brain-barrier (BBB) damage, neuroinflammation, and oxidative stress that leads to the death of mature neurons. Oxidative stress in particular can actually initiate and worsen processes such as neuroinflammation further damaging tissues. Moreover, the harsh environment induced by oxidative stress inhibits the proliferation and survival of endogenous neural stem cells (NSCs) crucial for regeneration and replacement of damaged tissue.

Nuclear factor erythroid 2-related factor 2 (Nrf2) is a transcription factor that regulates the antioxidant defense response during oxidative stress. Small molecules such as curcumin and its derivatives have been shown to activate Nrf2 and reduce oxidative stress following TBI. The attenuation of oxidative stress and promotion of NSC survival and differentiation is a crucial strategy for the regeneration of neural tissue following TBI. Stem cell derived extracellular vesicles (EVs) are ideal nanovesicles for TBI treatment as they carry a range of therapeutic cargoes such as neurotrophins for NSC survival and anti-inflammatory small molecules. Furthermore, EVs can be engineered to upregulate therapeutic biomolecules, deliver drugs, and cross biological barriers.

To further the development of engineered EVs for TBI treatment, the main goals of this proposal are (i) to investigate the therapeutic efficacy of curcumin derivatives for oxidative stress attenuation and (ii) engineer deferoxamine (DFO) conditioned neural stem cell (NSC) derived EVs for enhanced neurogenesis. The central hypothesis of this proposal is that EVs carry and transfer bioactive cargoes, and these cargoes combined with oxidative stress reducing drugs can encourage the differentiation and survival of endogenous NSCs for effective tissue regeneration and functional recovery following TBI.

The rationale includes: (i) curcumin derivatives can activate Nrf2/ARE pathway for oxidative stress attenuation; (ii) Deferoxamine (DFO) is an FDA approved iron chelator that has been demonstrated to upregulate therapeutic cargoes and neurotrophins such as brain derived neurotrophic factor (BDNF); and (iii) therapeutic molecules can be encapsulated inside EVs for BBB passage and effective TBI treatment. To accomplish this innovative design, we propose the following aims: (i) we will first investigate the therapeutic efficacy of commercially available curcumin derivatives on the activation of the Nrf2/ARE pathway and attenuation of oxidative stress; (ii) next, we will load the most effective curcumin derivative inside of EVs isolated from DFO-primed NSCs and further modify the EVs with a BBB targeting ligand; and (iii) lastly deliver this nanotherapeutic into an in vivo TBI model to enhance neurogenesis and motor function recovery. The proposed approach aims to develop dual functional DFO EVs for Nrf2 activation as well as BDNF delivery for NSC survival and differentiation following TBI. We expect to identify curcumin derivatives with superior oxidative stress attenuation compared to curcumin and reproducibly load these molecules into EVs for TBI treatment. The accomplishment of these aims can be extended to

other drugs for the treatment of other TBI related pathophysiological processes or other central nervous system pathologies such as neurodegenerative disease.

Contact Information

Li Ling Goldston
Rutgers, The State University of New Jersey
165 Davidson Road
Piscataway, NJ 08854
(570) 242-5405
leg125@scarletmail.rutgers.edu

FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR26FEL017
Alice Butera
Rutgers Biomedical and Health Sciences
$168,000

Project Title: *Efficacy of the SGLT2 inhibitor dapagliflozin to attenuate chronic neuroinflammation and related sequelae after blunt traumatic brain injury*

The project examines efficacy and mechanism of action of the SGLT2 inhibitor dapagliflozin to attenuate chronic neuroinflammation and related sequelae in a mouse model of blunt traumatic brain injury.

Traumatic brain injury (TBI) is a major cause of disability, and even mild injuries can lead to lasting problems with memory, mood, and daily functioning. There are very few treatments that can prevent the long-term complications of TBI. A key driver of these problems is brain inflammation, which can sometimes help repair tissue but often becomes persistent and harmful. Treatments that broadly suppress the immune system have not been effective, so new strategies are needed. Our research focuses on dapagliflozin (DAPA), which is FDA-approved for the treatment of diabetes and heart failure. In early studies, DAPA improved memory, reduced anxiety, and lowered brain inflammation in a rat model of blast TBI. Here we will test whether DAPA helps recovery after blunt head injury, the most common form in the civilian population, and whether it works by changing systemic metabolism, acting directly in the brain, or both. We will also study how DAPA affects microglia, the most abundant immune cells in the brain, using advanced single-cell technologies. This research will lay the foundation for future clinical trials to repurpose DAPA for the treatment of the psychological and cognitive problems that often follow TBI.

Contact Information:

Alice Butera
Rutgers Biomedical and Health Sciences
New Brunswick, New Jersey 08901
(332) 205-2049
ab2193@rutgers.edu

FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR26FEL019
Christopher Knapp
Rowan University
$307,080

Project Title: *Effects of repetitive mild traumatic brain injury on cognitive decline, markers of degenerative pathology, and catecholamine availability in a rodent model of Alzheimer's disease*

The proposed project titled, "Effects of repetitive mild traumatic brain injury on cognitive decline, markers of degenerative pathology, and catecholamine availability in a rodent model of Alzheimer's disease", employs a novel combined rodent model of repetitive mild traumatic brain injury and Alzheimer's disease (rmTBI-AD model) to examine how repeated mild head trauma accelerates cognitive decline and contributes to the degeneration of the prefrontal cortex and catecholamine-containing nuclei.

In the United States, most reported brain injuries are mild traumatic brain injuries, commonly known as concussions. These often result from falls, car accidents, sports injuries, or military-related incidents. While a single mTBI usually causes temporary symptoms, repeated injuries, especially in athletes and military personnel, can lead to more serious, long-term problems. Research shows that people with a history of repeated mild TBI are at greater risk for developing dementia, including Alzheimer's disease, particularly if they already carry certain genetic risk factors. Alzheimer's disease is a progressive brain disorder that affects memory, decision-making, and cognitive flexibility, which is a person's ability to shift strategies in response to changing environmental demands. These cognitive processes are mediated by the prefrontal cortex (PFC), a part of the brain often called the "executive center". The PFC relies on an optimal balance of catecholamines like dopamine and norepinephrine to function properly. Both repeated brain injuries and Alzheimer's disease can disrupt these important systems, possibly leading to earlier and more severe cognitive decline. Despite the clear link between repeated head injury and Alzheimer's risk, we still don't fully understand how repetitive TBIs contribute to disease progression. This project will use innovative rodent models of repetitive mild traumatic brain injury and Alzheimer's disease to explore how repetitive mild brain injuries influence cognitive decline and degeneration of important brain regions that mediate daily behaviors. The findings from this research represents a critical step toward understanding how brain injury contributes to Alzheimer's disease progression and holds strong potential to inform the development of treatment strategies for individuals at elevated risk due to prior head trauma and/or with genetic disposition.

Contact Information:

Christopher Knapp
Rowan University
2 Medical Center Drive
Stratford, New Jersey 08084-1500
(856) 669-9164
knappc0@rowan.edu

FELLOWSHIP RESEARCH GRANT RECIPIENT:

CBIR26FEL020
Ruizhe Tang
Rutgers Biomedical and Health Sciences
$150,000

Project Title: *Regulation of lysosome function after traumatic brain injury*

This project aims to elucidate how the neuronal polarity protein Par3 regulates Akt signaling and lysosomal acidification to maintain protein clearance and neuronal survival, thereby mitigating cognitive impairment after brain injury.

Traumatic brain injury (TBI) is one of the most common causes of lasting disability, affecting millions of people every year. Many survivors struggle with memory loss, poor attention, and other long-term cognitive problems, yet there are no effective treatments to prevent or reverse these outcomes. Research suggests that these lasting problems are not only due to the initial injury but also to ongoing disruptions in how brain cells clear waste and maintain energy balance. When these processes fail, harmful proteins build up, brain connections weaken, and cognitive decline worsens over time. Similar changes are also seen in stroke, cardiac arrest, and radiation-related brain injuries, pointing to a shared underlying mechanism.

Our work focuses on a protein called Par3, which helps brain cells stay healthy. We have found that Par3 supports two key processes: keeping survival signals active and ensuring that cellular "recycling centers" (lysosomes) remain acidic enough to break down waste. In models where Par3 is lost, brain cells cannot properly clear damaged proteins, leading to the buildup of toxic substances and worsening memory problems over time.

This project will study how Par3 protects brain cells after injury and whether enhancing its activity can improve recovery. We will use mouse models of brain injury and innovative imaging tools to track changes in cellular waste clearance, neuronal survival, and cognitive function. The goal is to determine whether boosting Par3 can prevent long-term memory loss and other cognitive problems.

Contact Information:

Ruizhe Tang
Rutgers Biomedical and Health Sciences
675 Hoes Lane West
Piscataway, NJ 08854
(732) 983-7242
rt659@rwjms.rutgers.edu

PILOT RESEARCH GRANT RECIPIENT:

CBIR26PIL002
Li Cai, Ph.D.
Rutgers, The State University of New Jersey
$180,000

Project Title: Cas13d-mediated Sarm1 silencing as a transformative therapy for traumatic brain injury

This project is to a pilot test of Cas13d-mediated Sarm1 silencing as a transformative therapy for traumatic brain injury.

Traumatic brain injury (TBI) is a major public health issue that can lead to long-term disability or death. Unfortunately, there are currently no treatments that directly protect the brain or help it heal after injury, only supportive care is available. One of the key problems after TBI is that nerve fibers (called axons) continue to break down, which worsens brain function over time.
Our research focuses on a protein called Sarm1, which plays a central role in this damaging process. When Sarm1 is activated, it causes axons to fall apart, leading to further brain damage. We aim to block this process using a cutting-edge gene-silencing tool called Cas13d, which can temporarily turn off specific genes without permanently altering DNA. We will deliver this tool directly to brain cells using a virus that targets neurons.

We have three main goals:
Make sure the treatment reaches the right brain cells and works effectively.
We will test how well our approach targets neurons and blocks Sarm1 in a mouse model of brain injury.
Test whether this treatment protects the brain and improves recovery.
We will measure brain inflammation, nerve cell survival, and how well the mice recover in terms of movement and memory.
Understand how blocking Sarm1 helps the brain heal.
We will study changes in brain chemistry and cell function to uncover how this treatment works at a deeper level.

Why this matters:
If successful, this research could lead to a completely new way to treat traumatic brain injury—by protecting nerve fibers and helping the brain recover. This approach has the potential to move from the lab to the clinic and make a real difference in the lives of people affected by TBI.

Contact Information:

Li Cai, Ph.D.
Rutgers, The State University of New Jersey
599 Taylor Road
Piscataway, New Jersey 08854-0000
(848) 445-6559
lcai@rutgers.edu

PILOT RESEARCH GRANT RECIPIENT:

CBIR26PIL003
Wanhong Zuo, Ph.D.
Rutgers Biomedical and Health Sciences
$180,000

Project Title: *Oxytocin Modulation of Lateral Habenula Circuits in Sex-Dependent Pain After Traumatic Brain Injury*

This project explores how oxytocin reduces pain differently in men and women after brain injury to guide safer, targeted treatments.

Traumatic brain injury (TBI) is a major health concern that can cause lasting problems such as chronic pain. Blast-related TBIs, which are common among military personnel and accident survivors, are especially challenging because their long-term effects can be difficult to treat. Men and women often respond differently to TBI. Women are generally less likely to sustain blast TBIs and often show more resilience in recovery, yet they tend to report more severe and persistent pain compared to men. Our research explores the role of oxytocin—a natural brain chemical best known for reducing stress and promoting social bonding—in how the brain processes pain after TBI. We are focusing on a brain region called the lateral habenula, which regulates negative emotions and pain perception. Early evidence shows that oxytocin systems work differently in males and females, which may help explain their different experiences of pain after TBI. By uncovering these biological differences, our work aims to pave the way for more personalized and effective treatments to relieve pain and improve quality of life for all individuals living with brain injury.

Contact Information:

Wanhong Zuo, Ph.D.
Rutgers Biomedical and Health Sciences
65 Bergen Street
Newark, New Jersey 07107
(646) 549-3796
zuowa@njms.rutgers.edu

PILOT RESEARCH GRANT RECIPIENT:

CBIR26PIL006
Vikram Shenoy Handiru, Ph.D.
Kessler Foundation
$178,859

Project Title: *Multicomponent Candidate Neuroimaging Biomarkers of TBI Severity: Fractal Dimension and Graph-Theoretic Structural Connectome Analysis*

This project will analyze MRI data from nearly 500 individuals in the national TRACK-TBI study to identify multicomponent biomarkers of traumatic brain injury severity by integrating fractal dimension, diffusion tensor imaging, and graph theory-based structural connectome metrics, with the goal of improving diagnosis, prognosis, and guiding personalized rehabilitation strategies.
Traumatic brain injury (TBI) is a leading cause of long-term disability in the United States, with more than 5 million survivors living with persistent difficulties. In New Jersey alone, about 12,000 new TBIs occur each year. Many people experience lasting problems with memory, attention, movement, and daily functioning. A major challenge is that current tools for diagnosis and prognosis often fail to detect subtle but widespread brain damage, leaving patients and families without clear answers about recovery and limiting doctors' ability to provide targeted rehabilitation.

This project will use advanced brain imaging analyses to identify new "biomarkers" of TBI severity. Data from nearly 500 individuals in the national TRACK-TBI study will be examined using three complementary methods. First, fractal dimensions will be used to measure the structural complexity of brain tissue. Second, diffusion tensor imaging will capture the integrity of white matter fibers. Third, graph theory analysis of the structural connectome will assess how well different brain regions remain connected, using metrics such as efficiency and modularity that describe how information flows across brain networks. Together, these approaches can provide a detailed map of brain injury that goes far beyond what standard scans reveal.

The study will test whether these combined markers (1) can classify patients into mild, moderate, or severe symptomatic groups, (2) determine how brain network disruption relates to problems with memory and other symptoms, and (3) ensure that the findings are robust across different groups and recovery stages. The expected outcome is a reproducible set of imaging markers that can guide clinicians in tailoring rehabilitation to each patient's needs. For New Jersey residents, this work has the potential to improve diagnosis, provide clearer prognoses, and reduce the long-term impact of TBI, ultimately enhancing quality of life for survivors and their families.

Contact Information:

Vikram Shenoy Handiru, Ph.D.
Kessler Foundation
1199 Pleasant Valley Way
West Orange, New Jersey 07052
(973) 324-3578
vshenoy@kesslerfoundation.org

PILOT RESEARCH GRANT RECIPIENT:

CBIR26PIL007
Samuel S.-H. Wang, Ph.D.
The Trustees of Princeton University
$180,000

Project Title: *Novel Neurotrophin Mimetics as Therapeutic Agents for Accelerating Recovery in Pediatric Traumatic Brain Injury*

This research investigates whether psilocybin can restore cognitive function and synaptic plasticity after pediatric traumatic brain injury by enhancing neurotrophin signaling, using advanced imaging to track dendritic spine dynamics across developmental stages in mice and assessing recovery through behavioral assays.
Traumatic brain injury (TBI) is one of the most common medical emergencies affecting children, with emergency room visits for pediatric TBI increasing in recent years. When young children suffer brain injuries, they often face worse long-term outcomes than adults or older children, with more severe and lasting problems in thinking, learning, and memory. Despite this being such a critical issue for children, most research on brain injury uses adult animals, leaving us with limited understanding of how the developing brain responds to and recovers from injury.

The brain's outer layer, called the neocortex, makes up most of the human brain and has a remarkable ability to change and adapt its connections between brain cells. However, this ability to change—called plasticity—varies throughout a person's life, being highest during childhood and declining with age. After a child suffers a brain injury, rehabilitation therapies may be less effective as the child grows older because the brain gradually loses some of this natural plasticity. Understanding how to preserve or enhance this plasticity could dramatically improve recovery outcomes for young TBI patients.

Our research focuses on the microscopic connections between brain cells called synapses, specifically examining structures called dendritic spines where one neuron sends signals to another. These spines are constantly being formed, eliminated, and modified—a process that underlies learning and memory. Brain injury disrupts this delicate balance, both at the injury site and in distant brain regions that are connected to it, leading to cognitive problems.

We propose to study a novel therapeutic approach using psilocybin, a naturally occurring compound that has recently gained attention for its potential medical applications. Recent scientific discoveries have shown that psilocybin works by enhancing the action of brain-derived neurotrophic factor (BDNF), a protein that promotes the growth and survival of neurons. In our preliminary studies, we found that a single dose of psilocybin can restore sensory function in mice after a disruption that normally impairs brain development. We also found that psilocybin accelerates learning in adult mice through this neurotrophic pathway rather than through its effects on serotonin receptors.

In this project, we will create brain injuries in mice at different ages—equivalent to childhood, adolescence, and adulthood—and test whether psilocybin can improve recovery. We will use cutting-edge imaging technology that allows us to watch individual dendritic spines in living mice over several weeks, tracking how they change after injury and after treatment. We will also test whether treated mice perform better on tasks requiring memory and flexible thinking, abilities that are often impaired after TBI.

This research is innovative because it combines advanced imaging, careful manipulation of specific molecular pathways in the brain, and behavioral testing across different developmental stages. By understanding how brain injuries affect young animals differently than older ones, and by testing whether enhancing neurotrophic signaling can promote recovery, we hope to open new avenues for treating children who have suffered brain injuries. If successful, this work could lead to new rehabilitation

strategies specifically tailored to a child's age and could establish whether compounds like psilocybin, which activate neurotrophic pathways, represent a practical therapeutic approach for pediatric TBI patients.

Contact Information:

Samuel S.-H. Wang, Ph.D.
The Trustees of Princeton University
Princeton Neuroscience Institute
40 Woodlands Way
Princeton, New Jersey 08544
(609) 258-0388
sswang@princeton.edu

PILOT RESEARCH GRANT RECIPIENT:

CBIR26PIL022
Zhifeng Kou, Ph.D.
New Jersey Institute of Technology
$180,000

Project Title: *Large Language Model Triage and Prediction Platform for TBI: From acute care to long term follow up*

This project develops an AI-powered smart assistant to help physicians triage, diagnose, and manage traumatic brain injury (TBI) patients, while guiding patients from early diagnosis through emergency care to long-term recovery to improve outcomes and treatment strategies.
Traumatic brain injury (TBI) is a major public health problem, affecting millions of people each year from accidents, falls, sports injuries, or violence. Many patients appear normal on standard brain scans like CT or MRI, yet still experience headaches, dizziness, memory loss, or mood changes that can last for months. For doctors, it is extremely difficult to determine which patients will recover quickly and which will face long-term problems. Families are often left uncertain about what to expect, and many patients are lost to follow-up after leaving the hospital.

Objective: This project proposes a new approach: an AI-powered smart assistant designed to help both physicians and patients from the moment of injury through long-term recovery. The system will continuously screen emergency department records to identify patients with possible TBI, guide physicians through triage and diagnostic steps, and provide evidence-based recommendations for acute management. It will also generate personalized outcome predictions to help doctors and families prepare for recovery. For patients, the system will serve as a trusted companion, explaining diagnoses in clear language, setting expectations for treatment, and staying in touch through scheduled follow-up survey with their smart phones at 3 days, 1 week, 1 month, 3 months, and 6 months after injury. By developing a trusted relationship and interacting with the patients at different time points for their recovery consultation, the system will resolve the issue of the loss of patient follow up in TBI research.

Novelty: Unlike traditional clinical studies, which often include only a narrow group of patients and struggle with poor follow-up, this platform is designed to be inclusive and adaptive. It will incorporate all patients seen in real-world clinical settings and continuously learn from both physician decisions and patient outcomes. Physicians' corrections will be used to refine the system's recommendations, while actual recovery data reported by patients will improve its predictions. This feedback loop ensures that the system becomes smarter, more accurate, and more representative of the full spectrum of TBI over time.

The project brings together a multidisciplinary team: experts in translational brain injury research, computer scientists specializing in large language models, and clinicians at New Jersey's busiest Level I trauma center, which treats nearly 1,000 TBI patients each year. The system will be trained on more than 50,000 historical trauma cases and enriched with national datasets from major TBI research programs. It will first be tested using past cases, then in silent mode alongside real clinical care, and finally in a pilot study with patients to validate safety and usefulness.

A central strength of this system is its patient-centered design. The communication agent will translate complex medical findings into accessible updates, provide structured surveys to track recovery, and suggest rehabilitation options when needed. This not only improves patient experience but also creates a valuable longitudinal dataset for research.

Impact: The potential impact of this project is significant. For physicians, it provides real-time support in diagnosing and managing TBI, reducing the risk of missed injuries. For patients and families, it offers

clear guidance and consistent follow-up, easing anxiety during recovery. For researchers, it generates high-quality, representative data that can drive new discoveries and better treatment strategies.

By merging cutting-edge artificial intelligence with frontline clinical care, this project aims to transform how TBI is diagnosed, managed, and studied. The proposed assistant has the potential to improve patient outcomes, enhance quality of life, and set a new standard for brain injury care in New Jersey and beyond.

Contact Information:

Zhifeng Kou, Ph.D.
New Jersey Institute of Technology
University Height
Newark, New Jersey 07102-1982
(973) 642-7421
zhifeng.kou@njit.edu