



**DIRECTORY OF GRANT AWARDS  
2015 GRANT CYCLE**

**NEW JERSEY COMMISSION ON  
SPINAL CORD RESEARCH**

**2015 GRANT CYCLE**

**DIRECTORY OF GRANT AWARDS  
FOR SPINAL CORD INJURY AND  
DISEASE RESEARCH**

**JUNE 2015**

## **NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH**

This data was compiled in compliance with the New Jersey Commission on Spinal Cord Research's statutory mandate, N.J.S.A. 52:9E-1, "...to compile a directory of spinal cord 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 2015 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 Spinal Cord Research.

Please feel free to contact the New Jersey Commission on Spinal Cord Research at P.O. Box 360, 369 S. Warren Street, Trenton, New Jersey, 08625. The Commission's office can be reached by telephone at 609-292-4055, by fax at 609-943-4213, or by e-mail at [NJCSCR@doh.state.nj.us](mailto:NJCSCR@doh.state.nj.us).

For information on the New Jersey Commission on Spinal Cord Research's grant award process, grant applications and deadlines, please see: [www.state.nj.us/health/spinalcord](http://www.state.nj.us/health/spinalcord).

### **2015 MEMBERSHIP INFORMATION**

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Cathleen Bennett  
Peter W. Carmel, M.D.  
John D. Del Colle  
James McCormack  
Michael J. Rhode  
Loran C. Vocaturo, Ed.D, ABPP

### **COMMISSION PERSONNEL**

Christine Traynor, Administrator  
Mary Ray, Fiscal Manager

**INDIVIDUAL RESEARCH GRANT RECIPIENTS:**

Jean Schwarzbauer, Ph.D.  
Princeton University  
Grant Award: \$548,914

Project Title: *A Cell-Assembled Matrix Template to Guide Nerve Regeneration in the Spinal Cord*

With nature as our guide, we will develop a biomaterial that is composed of a natural aligned extracellular matrix to direct axon growth across an implantable polymer scaffold for spinal cord repair. A spinal cord injury (SCI) is a trauma to the spine that damages nerve pathways and disrupts function leading to sensory and motor loss. SCIs occur at a rate of 30 per day in the United States with almost half resulting in complete loss of function below the level of injury. The initial injury is further compounded by wound healing responses in the spinal cord that involve bleeding, inflammation, and scarring at the site of injury. Regeneration of the nerve pathways is greatly restricted because of scarring and inhibitory factors, so it is highly uncommon for an injured person to gain complete restoration of function. Currently, the treatment of SCI is limited beyond stabilizing the injury and rehabilitation to maintain existing function. Clearly, new therapies for SCIs are greatly needed to promote full recoveries; these must overcome problems of the inhibitory microenvironment at the injury site and the failure of nerves to regenerate across it.

In order to bridge the injury site and promote nerve regeneration, we propose a new, double-pronged tissue-engineering strategy based on concepts of natural nerve development that integrates a spinal cord-mimicking hydrogel scaffold with native cell-assembled extracellular matrix (ECM) for neuron guidance. The two primary aims for this proposal are: (1) developing an optimal regenerative microenvironment combining ECM and growth factors for neurons; (2) constructing hydrogel scaffolds functionalized with these optimized ECM conditions to implant into SCI sites. The great novelty of this program lies in its uniting micropatterned hydrogel materials with a natural, cell-instructive ECM in order to develop successful therapies to restore function after SCIs.

In Aim 1, hydrogels will be micropatterned in order to create an aligned, cell-assembled ECM that can spatially guide nerve regeneration along the original nerve pathways. We will use this template to develop an optimized, regenerative ECM using nerve-specific cells and factors that facilitate enhanced nerve outgrowth. In Aim 2, to examine the efficacy of our engineered scaffolds and ECM, we will implant our scaffolds into a transected spinal cord model to assess restoration of function and nerve guidance in the longer term. Together, these studies will enable us to better understand how to create optimal regenerative microenvironments for nerve growth and how to use this information for the design of bioactive hydrogel scaffolds to facilitate recovery in SCI victims.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Li Cai, Ph.D.

Rutgers University – Biomedical Engineering

Grant Award: \$600,000

Project Title: *Role of Gsx1 in Activation of Neural Stem Cells and Neurogenesis after Spinal Cord Injury*

This project is to determine the functional role and molecular mechanism of Gsx1 in the activation of the neural stem/progenitor cells and neurogenesis after spinal cord injury.

The goal of this project is to elucidate the molecular mechanism underlying the activation (proliferation and differentiation) of neural stem/progenitor cells (NSPCs) after spinal cord injury (SCI). NSPCs persist in the adult mammalian central nervous system (CNS), and they function as a potential source of nerve cells for repair and regeneration after injury. Studies have shown that injury induces activation and proliferation of NSPCs in the adult mammalian CNS. However, the mechanism underlying injury-induced NSPC activation remains to be elucidated. In addition, the regenerative capacity of injury-induced neurogenesis is limited possibly due to the low efficiency. Thus, the potential uses of endogenous NSPCs in SCI to provide "self-repair" and regeneration cannot be fully realized.

Studies have established that the homeobox genes Gsx1 and Gsx2 play essential roles in the development of the mammalian spinal cord. Gsx2 is expressed in a subset of NSPCs and can be ectopically induced in injured brains, indicating a critical role of Gsx2 in injury-induced regenerative response. However, the role of Gsx1 in injury-induced neurogenesis is not well characterized. We have previously identified a NSPCs-specific cis-element, derived from the second intron of the Notch1 gene (Notch1CR2), can control reporter gene expressions in NSPCs. Using a Notch1CR2-GFP transgenic mouse line in which GFP expression correlates with endogenous Notch1 expression and is rarely visible in the adult CNS, we show that SCI induces a marked increase in the number of GFP+ NSPCs at the injury site. Sequence analysis shows that Notch1CR2 contains a Gsx1 binding site and we have showed that in the developing chick CNS, Gsx1 regulates Notch1CR2 function in NSPCs. Our preliminary studies further demonstrate that Gsx1 can control Notch1CR2 function in spinal cord NSPCs during embryonic development. It is known that Notch1 actively functions in the post-injury neural regeneration by regulating spontaneous cell proliferation, gliogenesis, synapse formation and axon remyelination. It is possible that Gsx1 can activate Notch signaling and/or other NSPCs-specific signaling pathways by interacting to its binding sites found in cis-elements. We hypothesize that the transcription factor Gsx1 is a key regulator of NSPC activation upon injury. We will determine the role of Gsx1 in injury-induced NSPC proliferation and differentiation; and the mechanism of Gsx1 in regulating NSPC-specific gene activation after injury.

Successful completion of this project will not only advance our understanding of the molecular mechanism of NSPC regulation during development and regeneration after injury, but also provide a basis for the future development of clinical interventions for millions of people who suffer from injury. Thus, our comprehensive studies will have a significant impact on both the basic stem cell biology and clinical translation.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Gabriele Di Luozzo, M.D.  
Hackensack University Medical Center  
Grant Award: \$599,482

Project Title: *Spinal Cord Protection: Understanding the Ischemic Insult to the Spinal Artery*

The development and testing of novel translational approaches to avoid ischemic spinal cord injury with endovascular approaches to thoracoabdominal aortic aneurysm repair.

The aorta is the largest blood vessel in the human body and is responsible for transporting oxygenated blood pumped from the left ventricle of the heart to all parts of the body through the systemic circulation. In some people, the wall of the aorta may become weak and enlarge, a disorder known as an aortic aneurysm. Though the risk for these localized bulges in the wall of the aorta are higher in some families due to genetic abnormalities in the connective tissues that make up the vessel wall, there are a wide range of factors that put people at an increased risk for acquiring this disorder, from uncontrolled high blood pressure to increasing age. The most common form of aortic aneurysm involves the part of the aorta that extends between the upper chest, or thorax, and the abdominal cavity. These types of aneurysms are called thoracoabdominal aortic aneurysms or TAAAs. Though many people have TAAAs without experiencing any symptoms, in some cases - particularly when the aorta dilates to greater than 1.5 times its normal size – individuals may experience back pain that is considered a sign that the vessel might burst. If TAAAs rupture, massive internal bleeding can occur and, unless treated quickly, can rapidly lead to shock and death.

In recent years, many advances have occurred in the surgical options available for the treatment and repair of TAAAs. Among these are minimally invasive options that take advantage of large vessels in the body that can be easily accessed through small cuts to the groin, also known as endovascular access. These approaches avoid the need to open the chest wall to gain access to the diseased portion of the aorta and allow for synthetic tubes (made of fabric-coated metal mesh), or stents, to be placed in the vessel to stabilize it and insure that it does not rupture.

Although endovascular techniques have been found to have a lower rate of mortality than open surgical methods, when properly placed, stents block a large number of the vessels that serve the tissues surrounding the aorta, including the spinal cord. This surgery, therefore, carries a high risk for spinal cord injury and paraplegia. Paraplegia is the severe or complete loss of the ability to move or feel the lower body. Symptoms of paraplegia may include an inability to walk or to control one's bladder and/or bowels.

The Cardiothoracic Research Group of Hackensack University Medical Center is trying to better understand the injury response of the vessels that service both the spinal cord, and the structures around it. They are looking at new ways to protect the spinal cord to reduce the large risk for paraplegia that comes with current methods to repair aneurysms. Though their ideas are gained through actual experiences with humans, new methods are tested using pigs since their anatomy is very similar to humans. This insures that any new findings can to be tested for their safety and effectiveness before they are offered to patients.

To this end, the research group is refining a new surgical strategy where endovascular procedures are done in two steps, the first of which is used to stimulate the growth of new and bigger vessels that will assure the spinal cord continues to get the blood it needs, even after a stent blocks those channels. After the two-stage procedure, samples collected from the pigs are used to look at mediators of inflammation and healing that are supported by this new method. Based on previous successes that have led to better outcomes in humans, the group is confident that their experiments will offer new insight that will help prevent the possible catastrophic consequences of this surgery for people in New Jersey and across the globe.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Francois Berthiaume, Ph.D.  
Rutgers University – Biomedical Engineering  
Grant Award: \$567,766

Project Title: *Nanoparticle-Based Treatment of Pressure Sores in Spinal Cord Injury Patients*

This project develops nanoparticles carrying bioactive peptides that speed up the healing of pressure sores.

There are approximately 400 new patients per year who suffer from spinal cord injury in New Jersey. A major secondary complication in spinal cord injury patients is the development of pressure ulcers, which are open wounds that occur as a result of injury to skin and muscle from prolonged sitting or lying. These wounds can take a long time to heal, which can prevent the patient from performing daily activities. Open wounds are also highly susceptible to infection, which can lead to amputation or severe septicemia.

Pressure sores can be difficult to treat, especially in patients who have co-morbidities, such as diabetes. In order to speed up the wound healing process, we propose to develop a nanoparticle technology that can release bioactive peptides when placed topically to the wound area and help accelerate wound healing. Although bioactive peptides, such as growth factors, have been proposed to improve wound healing, this strategy by itself does not work because the wound environment contains high levels of proteases that quickly degrade these factors.

The nanoparticle technology that we propose will protect the bioactive peptides from degradation and allow long-term release to the wound environment, so that they persist in the wound environment much longer. This nanoparticle technology also has several advantages from the standpoint of product manufacturing. First, the components can be made by genetic engineering bacteria to produce large amounts of the raw material. Second, the raw material spontaneously self-assembles into nanoparticles, which are very easily purified, unlike typical peptide growth factors that require slow and expensive purification methods. Third, the nanoparticles are very small, which makes them easy to incorporate into existing wound treatment strategies, such as bandages, skin substitutes, creams, and so on. Finally, the nanoparticle platform can be used with a variety of bioactive peptides that target different cellular components of the skin.

In this particular proposal, we plan to generate nanoparticles that target three different aspects of skin wound healing. We hypothesize that these nanoparticles will significantly enhance the performance of existing skin substitutes and wound dressings.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Robert O'Hagan, Ph.D.  
Rutgers University - Genetics  
Grant Award: \$600,000\*

Project Title: *Enhancement of Cytoskeletal Dynamics and Motor Transport By Manipulation of Post-Translational Microtubule Glutamylation To Maximize Neuroregeneration*

We will manipulate post-translational glutamylation of microtubules to promote cytoskeletal dynamics and intracellular transport to maximize neuroregeneration after injury. Spinal cord injury (SCI) leads to devastating neurological defects and disability. Although neurons in our central nervous system (CNS) possess an intrinsic ability to regenerate, this ability is often insufficient to restore function after SCI. For functional regrowth, injured neurons must dramatically reorganize their microtubule (MT) networks. MTs act as both structural components of the cytoskeleton and “cellular highways” for intracellular transport. If MTs are the highways, molecular motors are the trucks that transport essential cargos to the far reaches of the neuron—especially the growing tips of axons where they are most needed after neuronal injury. Therapies that target the process of MT reorganization and motor trafficking might promote regrowth of injured neurons. But first, we need to understand how MTs and transport are regulated.

The dynamic cytoskeletal changes in injured neurons occur microscopically, so they cannot be visualized in human patients or in living vertebrate experimental model systems. Therefore, our lab studies these processes in *Caenorhabditis elegans*, a powerful experimental animal model. Major assets of *C. elegans* include a transparent body that allows us to directly observe neuronal remodeling after physical or genetic injury. Because most basic biological processes, including axon development and neuronal transport, are conserved between *C. elegans* and humans, we can apply the fundamental principles we discover in *C. elegans* to higher organisms. By using cutting edge technologies to visualize MT dynamics and transport in living *C. elegans* animals, we can identify molecules that enhance intrinsic regenerative capacity of a neuron.

Using these techniques, our lab has identified genes that regulate a MT modification, called polyglutamylation that acts as a signpost along the MT highways. Mutations that affect polyglutamylation cause fundamental defects in the MT highways, resulting in traffic jams of motors. Such traffic jams might mean that needed cargos cannot reach their destinations, and in some cases can result in neuronal degeneration. For example, MT glutamylation can act as “speed limit” sign to restrict the velocity of particular motors and cargos inside neurons. Additionally, we found that MT modifications act as “under construction” signs that specify the preservation, construction, or demolition of particular MT highways. Our discoveries suggest MT signposts are essential regulators of neuronal regrowth and survival, a notion that we will test in rodent models of SCI. The overall goal of this project is determine how MT signposts that regulate the MT highways in neurons can be used to improve regeneration after injury by controlling motor transport and MT dynamics. Outcomes of this research should have groundbreaking impact for understanding the most fundamental elements of neuronal degeneration and regeneration, and possibly provide new therapeutic avenues for SCI.

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\*Award Amount Not Finalized

**FELLOWSHIP GRANT RECIPIENT:**

Keerthana Deepti Karunakaran  
New Jersey Institute for Technology  
Biomedical Engineering  
Grant Award: \$60,000

Project Title: *Understanding Cortical Reorganization in Spinal Cord Injury Using Resting State fMRI*

A spinal cord injury (SCI), often a result of blunt or sharp force trauma, is a devastating event that results in paralysis to lower limbs and in more severe cases to both lower and upper limbs. A major goal of SCI is to reduce the neurological damage and reestablish functionality by utilizing the plastic property of the brain. Although it is known that rewiring occurs in the brain in order to compensate for the loss, secondary complications such as phantom sensations, neuropathic pains etc. are very common. Despite the occurrence of non-desirable outcomes first described a century ago, lack of non-invasive tools to study the dynamic properties of the brain has limited our understanding of the underlying mechanisms.

Functional magnetic resonance imaging (fMRI) is a noninvasive imaging technique that allows us to study the baseline activity of the brain at resting condition. This provides an advantage to the SCI community, as it does not require the subject to perform any tasks, but allows us to study the brain reliably.

Our goal is to study large-scale changes in brain function in SCI subjects during the first 3 months of SCI onset and later at 9 months of SCI onset, such that changes in brain activity during recovery can be analyzed. This research could provide a reliable marker that could be used to predict the likelihood of a patient responding positively to a treatment and monitor the efficiency of a particular rehabilitative approach.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

### **EXPLORATORY RESEARCH GRANT RECIPIENTS:**

Long-Jun Wu, Ph.D.  
Rutgers University - Cell Biology & Neuroscience  
Grant Award: \$200,000

Project Title: *The Role of Hv1 Proton Channel in Secondary Spinal Damage and Central Neuropathic Pain after Spinal Cord Injury*

We test the hypothesis that the Hv1 proton channel contributes to spinal cord inflammation, secondary spinal damage, and central neuropathic pain following experimental spinal cord compression injury.

Spinal cord injury refers to any injury to the spinal cord that is caused by trauma, typically associated with major trauma from motor vehicle accidents, falls, sports injuries, and violence. Depending on where the spinal cord is damaged, the symptoms can vary widely, from pain to paralysis to incontinence. According to the National SCI Statistical Center, the number of people in the United States who were alive in 2012, and who had spinal cord injury was estimated to be approximately 270,000 persons, with a range of 236,000 to 327,000 persons. Treatment of spinal cord injuries can vary widely depending on the location and extent of the injury, but usually starts with restraining the spine and controlling inflammation to prevent further damage. In general, we are interested in how inflammation causes the secondary damage and central neuropathic pain after spinal cord injury. Specifically, we are studying inflammatory cells, such as microglia, macrophage, and neutrophil, and their functions in spinal cord injury. Microglia/macrophages are immune cells that circulate in the brain to detect and control or kill invading bacteria. Their primary weapons are reactive oxygen species (ROS) and inflammatory mediators, and damage proteins and lipids in the bacteria, thus killing or hindering them. Unfortunately, ROS not only damage invading bacteria, but can also damage normal brain tissue and the vessels that supply blood to the brain under disease conditions, such as stroke and spinal cord injury.

Our laboratory recently discovered that the protein responsible for proton secretion is an ion channel we call Hv1. In order to test the function of Hv1, we have generated mice lacking Hv1. Using these mice, we have found that Hv1 is coupled with immune response including ROS and cytokine production. Under disease conditions such as ischemic stroke, Hv1 contributes to brain damage. Also, we have some preliminary results that show the Hv1 proton channel is important for neuropathic pain after peripheral nerve injury. In the current proposal, we will determine whether Hv1 is needed for the spinal cord inflammation, tissue damage, and central neuropathic pain after spinal cord contusion injury. If this is the case, drugs may be developed that inhibit Hv1 and can prevent the generation of harmful effects following spinal cord injury. Therefore, our study will provide a potential therapeutic target for treatment of spinal cord injury. In summary, our proposal will be the first attempt to investigate Hv1's function in secondary damage and central neuropathic pain in spinal cord injury, with the aim of evaluating Hv1 as a potential new therapeutic target for its treatment.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Zhiguo Jiang, Ph.D.  
Kessler Foundation  
Grant Award: \$197,794

Project Title: *Assessing Spinal Cord Structure Changes using Diffusion Tensor Imaging in Patients with Incomplete Traumatic Spinal Cord Injury*

This project proposes to use a special MRI technology to assess nerve fiber structural changes in spinal cord in acute incomplete spinal cord injury patients (some abilities to move their limbs).

Each year in the United States there are 12,000 new cases of spinal cord injury (SCI). Not only does SCI cause considerable physical damage and disability to the individuals, it imposes a significant economic and emotional burden on them and their families. Average lifetime costs can be in the millions for a given SCI patient.

There has been mounting evidence of movement function recovery and changes occurring in the spinal cord following SCI, especially after rehabilitation therapy. To evaluate the functional outcome of recovery, clinicians often rely on questionnaire-based neurological tests to score the motor and sensory functions. These tests are subject to shortcomings including inability to measure recovery below injury level and inherent variability across examiners.

To overcome these shortcomings, we propose to use advanced and non-invasive diffusion tensor imaging (DTI), a special MRI technology that measures distribution of water elements in nerve fibers, to detect nerve fiber structural changes above and below the injury level in SCI. A group of patients with incomplete SCI (iSCI) will be enrolled into the study and treated by standard rehabilitative therapies. Before, 1 month, 2 month, 4 month and 6 month after the treatment program, the DTI measurements will be taken and statistically analyzed. It is expected that the DTI measurement of nerve fiber structure in SCI will be worse in patients than that of healthy people, and the integrity or quality of the fiber structure in patients will improve as a result of combined repair effect from rehabilitation and spontaneous recovery, indicating SCI recovery. It is also expected that the improvement of spinal cord nerve fiber quality will be associated with gains in movement ability of the patients. This association will potentially allow us to use the DTI measurement to predict SCI patients' motor recovery and vice versa. Thus, the MRI DTI measurement may potentially serve as an objective tool to aid clinicians for more accurate SCI diagnosis.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Lisa Boulanger, Ph.D.  
Princeton University  
Grant Award: \$200,000

Project Title: *SCI-Induced Denervation of the Diaphragm: Regulation by MHCI Immune Proteins*

We will attempt to enhance the body's own neuroprotective mechanisms as a new therapeutic approach to preserve or restore normal breathing after paralyzing spinal cord injury.

After spinal cord injury (SCI), one of the most common and devastating complications is breathing paralysis. More than half of all SCI cases affect the cervical spine, which contains the neurons that control the diaphragm, an essential breathing muscle. Injury to the cervical spine is common in car accidents and in sports and recreational injuries, which affect thousands of New Jersey residents every year. The resulting breathing problems require intensive management, and increase the risk of complications, including pneumonia. Thus identifying ways to improve respiratory function is a high priority for treating patients with SCI. The goal of this proposal is to explore a new therapeutic approach to preserve or restore normal breathing after a paralyzing spinal cord injury.

Remarkably, not all damage to breathing circuitry with SCI occurs at the time of the accident. After the initial physical trauma, damaged cells release chemicals that cause prolonged, insidious secondary damage. In particular, the neurotransmitter glutamate is released in excessive amounts after SCI, over-activating a class of glutamate receptors called AMPARs, and causing excitotoxic damage. This secondary excitotoxic damage significantly expands the tissue damage and loss of muscle control after SCI. Because secondary excitotoxicity is still occurring weeks after spinal cord injury, there may be an opportunity to stop this damage before it happens, and help protect breathing in patients with SCI. Drugs that block excitotoxicity often have serious side-effects, because they block both the harmful and essential functions of glutamate. A different approach is needed to reduce excitotoxicity while leaving essential AMPAR function intact.

The hypothesis guiding the current proposal is that enhancing the body's own neuroprotective mechanisms can reduce secondary damage after SCI, while minimizing harmful side-effects. The proposed experiments build on recent, ground-breaking findings that specific immune proteins, members of the major histocompatibility complex class I (MHCI), inhibit the AMPARs that mediate excitotoxicity. These results suggest that MHCI might have the ability to reduce AMPAR-mediated excitotoxicity after cervical SCI. Promisingly, mice that have been genetically engineered to make more MHCI show significantly improved leg movements after injury to the thoracic spinal cord. These results show that in animal models, MHCI can promote recovery from damage to parts of the spinal cord that control the legs. However, it is unknown if MHCI can help preserve breathing after damage to the cervical spine.

This proposal will test if enhancing MHCI function can help preserve diaphragm innervation and breathing following cervical SCI. The proposed studies will build a new collaboration between scientists at Princeton and Thomas Jefferson Universities. The combined expertise of the two groups is uniquely suited to answer these questions: The Boulanger Lab first identified MHCI's

ability to inhibit glutamate receptors, and the Lepore Lab has developed and tested an animal model of cervical SCI that reproduces denervation of the diaphragm.

The proposed studies will evaluate the effects of MHCI on histological and functional outcomes associated with most human cervical SCI, including phrenic MN loss, diaphragm denervation, and respiratory dysfunction. The cervical SCI model developed in the Lepore Lab is ideal to study the role of MHCI in secondary damage and recovery after SCI. In these studies, cervical SCI will be induced in mice in which MHCI levels have been genetically reduced or enhanced, and the effects on diaphragm innervation and breathing determined. The ways in which MHCI levels change after SCI will also be determined. Together, the proposed research could help identify an unexpected approach to prevent respiratory compromise and reduce morbidity and mortality following cervical SCI.

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