



**DIRECTORY OF GRANT AWARDS  
2016 GRANT CYCLE**

**NEW JERSEY COMMISSION ON  
SPINAL CORD RESEARCH**

**2016 GRANT CYCLE**

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

**JUNE 2016**

## **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 2016 grant cycle. The research projects are not categorized, or listed in any particular order.

This directory is not a complete listing of all scientific research being performed within the State of New Jersey due to the proprietary nature of the research being conducted at various institutions throughout the State. In addition, institutions are not obligated to share their research information with the New Jersey Commission on 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.nj.gov](mailto:NJCSCR@doh.nj.gov).

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).

### **2016 MEMBERSHIP INFORMATION**

Susan P. Howley, Chairperson  
Peter W. Carmel, M.D.  
John D. Del Colle  
James McCormack  
Michael J. Rhode  
Loran C. Vocaturo, Ed.D, ABPP  
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### **COMMISSION PERSONNEL**

Christine Traynor, Administrator  
Mary Ray, Fiscal Manager

**INDIVIDUAL RESEARCH GRANT RECIPIENTS:**

CSCR16IRG005  
Kamana Misra, Ph.D.  
Celvive, Inc.  
\$452,149

Project Title: *Mechanisms Underlying Bone Marrow Transplant Driven Activation of Endogenous Stem Cells for Spinal Cord Injury Repair*

Decipher mechanisms underlying the therapeutic effect of autologous, adult bone marrow derived stem cells on spinal cord injuries.

Spinal cord injury (SCI) results in drastic life style alterations for the patients and their family members. Therapeutic interventions require regeneration of multiple types of neurons to recreate lost neurons.

Research advances using cell therapies have ushered in an era of new hope and the field is witnessing a surge in clinical studies using cellular transplantation for treating SCI. Progression into main line therapies is however impeded largely due to ambiguity about A) variability in cell derivation protocols, B) fate of the transplanted cells, and C) mechanisms underlying the therapeutic benefit.

Our earlier work has demonstrated that patients own unexpanded, minimally manipulated adherent bone marrow cells (ABMCs) can effectively induce repair at spinal cord injury site. The scope of the current grant proposal is to address deficiencies in the field mentioned above by using transgenic mouse models and sophisticated in vivo imaging techniques to understand the interactions between the transplanted bone marrow stem cells and the endogenous neural stem cells that lie quiescent in the adult spinal cord.

We plan to study these interactions in real time. Such real time monitoring of in vivo transplanted cells and resulting mobilization of endogenous cell populations can help validate the full potential of cell-based therapy. Additionally, identification of potential impediments can help in designing improved cell transplantation strategies.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

CSCR16IRG007  
Jeffrey Zahn, Ph.D.  
Rutgers University  
Biomedical Engineering  
\$593,792

Project Title: *Multi-Layer Implantable Cortical Microelectrodes to Improve Recording Potential for Spinal Cord Injury Treatment*

We will create neural microprobes minimizing tissue damage while maximizing the number of recording electrodes using a multilayer fabrication approach for use in rehabilitative applications in SCI.

The purpose of this proposal is to improve neural recordings for use in brain computer interfaces (BCI). BCI is a method which records thoughts in the form of electrical activity from the brain and then processes and decodes these signals which can be used in different ways such to control a wheelchair or computer. There has been a rising interest in using BCI to assist people who suffer from spinal cord injury (SCI) by replacing the lost sensory and motor functions with external neural prosthetic devices so that patients can regain a level of independence and improve their quality of life.

One of the essential components of a functional BCI is the ability to record a strong lasting signal from the brain. One way of recording nerve signals is by using implanted microelectrodes, also called neuroprobes. Unfortunately, when these probes are implanted in the brain a process of tissue scarring, called gliosis, degrades the probe's recording potential within a few months. To address this issue we have created ultraminiaturized recording probes from flexible biocompatible plastic materials. Because these probes are so small and flexible once they are implanted in the brain this tissue scarring response is not seen. However, one challenge in creating these probes is that only a single recording electrode can be defined on such a small probe. Multielectrode recordings of distributed neurons improve recording fidelity due to activation of an area of the brain during task volition allowing better decoding of motor intent. So, in order to have the highest recording potential while limiting tissue scarring we will design and fabricate cortical recording neuroprobes which maximize the number of recording electrodes on a single miniaturized microprobe so that multiple neighboring neurons can be recorded simultaneously. To accomplish this task, we will use a novel multilayer fabrication process allowing vertical definition of recording sites. Each layer will consist of recording traces insulated by a thin polymer layer electrically isolating the traces from their neighbors. This will maximize the number of probe recording sites along the length of the probe while minimizing the overall probe dimensions. To our knowledge no other research group has investigated such a multilayered design. Once we have fabricated this device we will implant the probes in a mouse model to stimulate and record from nearby neurons to assess the probe function over a 24-week period. We will also use the animals as a model of brain plasticity relevant to SCI by training the animal in motor tasks and recording motor volition using our microprobes.

Completing our goals, we will have demonstrated the ability to maximize recording potential using the novel flexible microprobes while minimizing the device dimensions. Neural recording from these probes implanted for long-periods of time holds great promise for development of rehabilitation strategies following SCI, and improving the effectiveness of microelectrodes currently limited by gliosis around the probe. Importantly, the new electrodes can be immediately integrated with the advanced approaches under development for the other three essential elements of a BCI, which currently utilize EEG- or ECoG-generated signals. Moreover, our approach for insertion can be used for microelectrodes developed in other laboratories.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

CSCR16IRG010

Nancy Chiaravalloti, Ph.D.

Kessler Foundation

\$585,303

Project Title: *A Longitudinal Examination of Aging with a Spinal Cord Injury: Cardiovascular, Cerebrovascular and Cognitive Consequences*

The goals of this proposal are to determine if cardiovascular and cerebrovascular dysfunction contribute to cognitive deficits in older and aging individuals with SCI.

The population is getting older; it is reported that the number of individuals in the United States older than 65 increased 10 times over the past 100 years. Likewise, individuals with spinal cord injury (SCI) are also aging and more than 80% are older than 50 years. However, as the general population is getting older, they are living longer; this is not the case in people with SCI. In fact, people with cervical SCI (neck injury) can expect to live 34 years less than an individual of the same age who is not injured. The reasons for reduce life expectancy in the SCI population are not clear, but cardiovascular and cerebrovascular diseases were the leading causes of death from 1952-2001.

Similar to the non-injured population, as people with SCI get older, they are faced with the increased likelihood of developing age-associated diseases like cardiovascular disease and stroke (cerebrovascular disease). In fact, people with SCI are 5-times more likely to have had a stroke than people without SCI, which may be due to the secondary complications of the SCI, such as the inability to control heart rate and blood pressure.

Furthermore, because of damage to the nervous system it is often more difficult to prevent and treat diseases and illness in the SCI population, which may worsen disease progression and reduce life expectancy. In addition to cardiovascular and cerebrovascular disease, people with SCI are reported to have impaired thinking (cognitive) abilities at a relatively young age, and as many as 60% of individuals with SCI have functional deficits in memory, information processing and executive function. In the general population there is an association between aging and the development of cognitive deficits that may be related to cardiovascular and cerebrovascular dysfunction. We do not know the effects of aging with SCI on cardiovascular, cerebrovascular or cognitive function, but we do know that the SCI population is getting older.

It is likely that the impact of worsening disease progression and the inability to adequately address these complications, coupled with advancing cognitive impairments would be expected to not only reduce life expectancy, but could significantly detract from independence, social interaction and quality of life. Improving our understanding of the cardiovascular, cerebrovascular and cognitive profiles in older individuals with SCI compared to older non-injured controls will help to define differences among these aging populations and inform clinical treatment algorithms for those with SCI. Further, getting a glimpse of how cardiovascular, cerebrovascular and cognitive function change over time in relatively young individuals with SCI compared to age-matched controls, will aid in the development of timely intervention strategies to prevent or ameliorate the pronounced functional deficits reported in the SCI population.

Therefore, the objectives of this project are to compare cardiovascular, cerebrovascular and cognitive function among older individuals with SCI (50-75 years) compared to older age-matched non-injured controls and to determine 3-5-year change in cardiovascular, cerebrovascular and cognitive function in relatively young individuals with SCI (28-45 years) compared to age-matched controls. The results will help guide interventional studies aimed at improving health, longevity and quality of life in the aging SCI population.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

CSCR16IRG013  
Tracy Tran, Ph.D.  
Rutgers University  
Chemistry & Chemical Biology  
\$599,716

Project Title: *Novel Cell Autonomous and Non-Cell Autonomous Mechanisms of Semaphorin-Neuropilin/Plexin Signaling Regulate Spinal Commissural Axon Pathfinding in the Mammalian CNS*

This project aims to understand how spinal cord projection axons use intermediate target structures and environmental cues to navigate long-distances to find their functional targets in the brain.

Spinal cord injuries often lead to severing of nerve fiber tracts, made up of individual axons (thin processes extended by neurons) carrying important sensory and motor information, that leads to paralysis and other debilitating conditions. During development growing axons are capable of navigating over long distances and between a series of intermediate targets to find their proper functional targets via the instructional (attractive or repulsive) signals given by molecular cues present in their environments. Therefore, it is critical to identify the molecular mechanisms underlying axon guidance in order to establish a foundation to devise suitable treatments for promoting the regeneration of axons damaged by spinal cord injury.

Previous studies have shown that commissural neurons, which extend axons from one side of the spinal cord to the other, across the midline, and then project to the brain, represent components of important ascending axon tracts, which convey external sensory information (touch, pain and body position) to higher brain centers. However, the precise paths followed by different subsets of spinal commissural axons as they extend to the appropriate target cells in the brain are not known. Moreover, very little is known about the molecules that guide the specific subsets of axons to their appropriate targets.

Therefore, we propose to trace the trajectories of multiple classes of ascending spinal commissural axons in mouse embryos, and to identify the molecules and understand the guidance mechanisms that these axons use to navigate through their intermediate target, the floor plate, at the midline, and ultimately find their targets. In order to establish a “roadmap” for ascending commissural axons to find their targets, it is essential to first visualize the long-range trajectories of these spinal axons as they initially develop in the embryo.

The proposed studies will be carried out in whole mouse embryos using molecular genetic techniques to permanently label particular subsets of spinal commissural neurons and their axonal projections to their target sites. In addition, we will use mouse genetic techniques to specific delete genes encoding molecular cue receptors in the Semaphorin protein family that were previously shown to play important roles in guiding commissural axons to cross the midline and sort their trajectories on the contralateral side of the spinal cord. Curiously, members of the Semaphorin protein family and their receptors are also present in the adult nervous system.

Accumulating evidence indicates that the levels of these molecules are dramatically altered after spinal cord injury, which could underlie the inability of the spinal cord to regenerate following injury. Therefore, studying the mechanisms of how these guidance molecules function will not only give a better understanding of how they control axon navigation during development, but also will be critical for designing strategies to modulate their expression levels and function in the injured spinal cord. Thus, our proposed studies on how commissural axons are guided, by Semaphorins and their receptors, to their functional targets during normal development will significantly contribute to our understanding of the molecular mechanisms governing the formation of specific neuronal connections from the spinal cord to the brain. Collectively, our anticipated results will also provide the biological basis to spur new approaches for the design of therapeutic strategies aimed at regenerating the injured spinal cord.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

### **FELLOWSHIP GRANT RECIPIENT:**

CSCR16FEL008

Jyothi Shilpa Akella, Ph.D.

Rutgers University

Human Genetics Institute

\$150,000

Project Title: *Identifying Mechanisms that Regulate Stress-Induced Neuronal Restructuring using Caenorhabditis Elegans*

This proposal aims to identify the mechanisms that contribute to stress-induced neuronal restructuring and recovery using the powerful model system *Caenorhabditis elegans*.

Every year 12,500 Americans experience a Spinal Cord Injury (SCI). One of the immediate effects of SCI is lowering of extracellular pH (acidosis), an event that triggers processes affecting neuronal survival, pain and recovery. Acid Sensing Ion Channels (ASICs) are cation selective channels that get activated at low pH and play a role in acidosis-induced remodeling of dendritic spines- structures involved in learning, memory and pain. In rodent models of injury, inhibiting ASIC activity is neuroprotective and lowers pain. However, the role of ASICs in acidosis-induced neuronal remodeling in vivo and the contribution of neuronal remodeling to recovery of function remains a mystery. Since it is difficult in humans to study the connection between SCI, stress, acidosis, neuronal restructuring, and recovery of function, alternative experimental systems are necessary. The simple animal model *C. elegans* is excellent for studying the effects of stress and acidosis on neuron morphology, function and activity.

Our lab identified six neurons of *C. elegans* that undergo reversible stress-induced neuronal restructuring. In non-stressful conditions, these IL2 neurons display a simple bipolar architecture with a single dendrite and single axon. When stressed, the dendrites and axons of the IL2 neurons extend branches. Upon removal of stress, neuronal branches are retracted and regain the simple bipolar neuronal features. These remodeling events are similar to the stress-induced changes in neuron structure observed in rodent models of SCI and upon acidosis injury. To identify genes that may play important roles in stress-induced neuronal remodeling and recovery, we defined the cell-specific transcriptome (parts list) of the IL2 neurons. We found two ASIC encoding genes *asic-2* and *egas-1* exclusively expressed in the IL2 neurons, but their functions remain unknown. We will determine the function of these ASICs in IL2 neuron restructuring (Aim 1). We will create mutations that will enhance or reduce the ASIC channel activity and then measure their ability to induce dendritic remodeling in living animals. This will establish *C. elegans* as an in vivo model to study the effects of acidosis-induced neuronal remodeling. Changes in dendrite morphology associated with alterations in neuronal activity contribute to pain. We will determine whether IL2 neuronal activity changes during stress-induced neuronal remodeling (Aim 2). Our work will reveal conserved molecules and mechanisms that modulate neuronal restructuring and activity and contribute to pain after SCI.

Extracellular vesicles (EVs) are submicron sized particles released by many cells in the nervous system and function in intercellular communication. EVs are found in body fluids including cerebrospinal fluid. The content and nature of the EVs is changes in health and neurodegeneration, and the factors that regulate the properties of EVs in disease or stress remain unknown. The *C. elegans* IL2 neurons in addition to dynamic remodeling ability, also release EVs. Since stress affects EV dynamics in vitro and since IL2 neurons also respond to stress by remodeling, we will use the IL2 neurons to examine the effects of stress on EVs and a role for EVs in stress-induced neuronal restructuring (Aim 3). Our studies will provide insight to mechanisms controlling stress-induced neuronal restructuring and EV dynamics. This knowledge will help design therapies that may promote formation of beneficial EVs that encourage recovery and neuroregeneration while blocking the formation of pathological EVs that promote neurodegeneration.

Our studies in a simple animal model for stress-induced neuronal restructuring and recovery may pave the way to therapies that promote neuron survival, recovery of function and pain reduction after SCI.

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**EXPLORATORY RESEARCH GRANT RECIPIENTS:**

CSCR16ERG019

KiBum Lee, Ph.D.

Rutgers, Chemistry & Chemical Biology

\$200,000

Project Title: *Promoting Axonal Regeneration in the CNS Using NanoScript (Nanoparticle-Based Transcription Factor)—Based Repression of PTEN*

A novel treatment method using nanoparticle based synthetic transcription factor, NanoScript, to genetically induce axon regeneration to effectively promote functional recovery in spinal cord injury.

Spinal cord injury (SCI) is one of the most common causes of disability in young adults, affecting approximately 12,000 people in the United States every year. SCI results in a number of cellular and molecular changes in and around the injury site, leading to a host of debilitating symptoms that result in increasing loss-of-function. Given the complex damage caused by SCI and the intrinsically limited regenerative potential of the mammalian CNS, there is a strong clinical need for effective strategies to: 1) alleviate the inhibitory environment, 2) regenerate the destroyed neural cells, and 3) re-establish the damaged neuronal circuitry in the injury site. To this end, gene therapy has shown great promise in treating SCI. Recent studies indicate that the phosphatase and tensin homolog (PTEN) gene regulates mTOR pathways, which stimulate axon growth and regeneration. Remarkably, it allows corticospinal tract regeneration in mice, despite the presence of growth inhibitory factors in the spinal cord and injury site. However, previous approaches required viral vectors to deliver siRNA to silence or Cre to knock out PTEN. Such viral approaches have significant safety obstacles to overcome, including the potential oncogenic effects of PTEN deletion. Addressing the aforementioned challenges in gene therapy, our group (KBLEE group) has previously developed a nanoparticle-based synthetic transcription factor platform that emulates the fundamental functions of transcription factors, thereby allowing for regulating transcriptional activity (e.g. activation or repression) and targeted gene expression (e.g. PTEN) in both an effective and selective manner. In addition to transiently regulating genes in both non-viral and efficient manner, we designed the NanoScript platform to be interchangeable, and hence applicable for almost any cellular application, especially stem cell differentiation and cellular reprogramming applications. Moreover, NanoScript, can be synergistically combined with epigenetic modulators (e.g. SAHA and CTB) and has been demonstrated to both effectively activate or deactivate specific endogenous genes in stem cells, which could lead to myogenesis, chondrogenesis, and neurogenesis. However, to facilitate the translation of our nanoparticle-based STF platform to the effective treatment of SCI, several critical parameters must be further investigated and improved, including: 1) the specificity of NanoScript-PTEN modulating the PTEN/mTOR pathways without off-target effects, 2) the functionality of our nanoparticle-based STF platform – by replacing the gold nanoparticle with a multifunctional nanoparticle we can bestow our platform with significantly more functionality, and 3) we need to determine its in vivo biocompatibility and efficacy.

Our unique NanoScript system is designed to induce axon regeneration to effectively promote functional recovery. In this proposal, we combine our expertise in nanomedicine (Lee lab) and spinal cord injury repair (Young lab) to develop a new nanomaterial-based treatment strategy to SCI. Given the complexities caused by the injury, we believe our novel NanoScript platform can serve as a multifunctional tool for developing future therapies for CNS-related diseases and injuries.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

CSCR16ERG014

Treena Livingston Arinzeh, Ph.D.  
New Jersey Institute of Technology  
\$200,000

Project Title: *Schwann Cell GAG Mimetic Combination Strategy for Spinal Cord Repair*

This study will evaluate the use of glycosaminoglycan (GAG) mimetics in combination with Schwann Cells for spinal cord repair

In the United States alone, there are approximately 300,000 persons with damaged spinal cords with 12,000 new cases added annually. Spinal cord injury (SCI) is a devastating condition for which there is no effective treatment at the present time.

Neural tissue engineering strategies using scaffolds that more closely mimic the native extracellular matrix (ECM) during neural development may be a promising strategy to promote axonal growth. During neural development, glycosaminoglycans (GAGs) have been known to play an important role in axonal guidance and growth. Depending upon the degree and pattern of sulfation, GAGs can provide a permissive environment for cellular and axonal growth. We have developed GAG mimetics derived from cellulose that have chemical structures similar to native GAGs but can be tailored to have different chemistries. The GAG mimetic, sodium cellulose sulfate (NaCelS), can be synthesized to vary in the degree and pattern of sulfation, which makes it attractive over native GAGs because it can be tailored.

In preliminary studies, neurite extension was greatest on scaffolds containing NaCelS in comparison to naturally-occurring GAGs and controls. In the proposed studies, we will combine NaCelS containing scaffolds with Schwann cells (SCs) to promote axonal growth.

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## NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

CSCR16ERG018  
Kathy Chiou, Ph.D.  
Kessler Foundation  
\$190,953

Project Title: *Investigation of Structural White Matter Substrates Underlying Cognitive Impairment After SCI*

This study utilizes diffusion tensor imaging/neuropsychological testing to examine and document the relationship between cerebral white matter integrity and cognitive performance in adults with SCI. Advances in neuroimaging techniques have allowed scientists to expand their knowledge of the neural mechanisms underlying traumatic spinal cord injury (SCI). Diffusion tensor imaging (DTI), is a non-invasive neuroimaging technique that yields information about the movement of water molecules in the intercellular architecture of the central nervous system. Measurements of the magnitude and direction of water diffusion (or movement) allows for inferences to be made about the integrity of white matter tissue in the central nervous system. Through DTI, researchers have discovered that traumatic SCI not only affects the white matter tracts of the spinal cord, but also negatively impacts white matter tissue in the brain. Specifically, persons with SCI have been found to have reduced white matter integrity in the cerebral cortex compared to healthy peers. Importantly, disruption of cerebral white matter integrity has been shown to be associated with poor cognitive function in other populations. While impairments in cognitive functioning have, in fact, been well-documented after SCI, the influence of cerebral white matter integrity on such cognitive deficits remains unknown.

This study utilizes DTI and neuropsychological evaluation to document the association between cerebral white matter integrity and cognitive functioning in adults with SCI. Measures of the magnitude (mean diffusivity) and direction (fractional anisotropy) of water diffusion in the brain will serve as measures of cerebral white matter integrity. These measures will be correlated with scores from neuropsychological testing to document the relationship between cerebral white matter integrity and cognitive functioning.

This project will expand the field of SCI research by simultaneously investigating 2 important, but understudied areas in spinal cord research: cerebral integrity and cognitive functioning. Importantly, the documentation of the relationship between cerebral white matter integrity and cognitive functioning in SCI will provide a clearer understanding of the neural mechanisms underlying cognitive functioning after injury. Through this knowledge, predictive biomarkers of cognitive functioning can be identified. These biomarkers may have significant clinical applicability as they may facilitate easier and faster detection of cognitive deficits after SCI. Improving the detection of these deficits would assist individuals with SCI in gaining access to services that could ultimately improve their functional outcome.

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