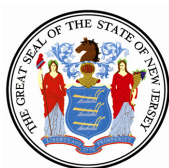




DIRECTORY OF GRANT AWARDS 2013 GRANT CYCLE



Chris Christie, Governor
Kim Guadagno, Lt. Governor



Mary E. O'Dowd, MPH
Commissioner

**NEW JERSEY COMMISSION ON
SPINAL CORD RESEARCH**

2013 GRANT CYCLE

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

JUNE 2013

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 2013 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.

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.

2013 MEMBERSHIP INFORMATION

Susan P. Howley, Chairperson
Cathleen Bennett
Peter W. Carmel, M.D.
John D. Del Colle
James McCormack

COMMISSION PERSONNEL

Christine Traynor, Administrator
Mary Ray, Fiscal Manager

INDIVIDUAL RESEARCH GRANT RECIPIENTS:

Gail Forrest, Ph.D.
Kessler Foundation

Grant Award: \$574,976

Project Title: *Non-Ambulatory SCI Walk Using a Robotic Exoskeleton: Effect on Bone and Muscle*

Functional gains after spinal cord injury (SCI) often plateau, leaving a person paralyzed and immobilized. There is a rapid wasting away of the muscle and bone in the lower limbs within the first six months of injury and osteoporosis often results. High fracture risk, especially about the knee, an energy imbalance leading to obesity and cardiovascular disease, urinary tract infection and skin issues such as pressure sores are often the result of muscle and bone deterioration and continued immobilization. These health issues are daily concerns, and affect the day to day quality of life of people who cannot walk following a spinal cord injury. The spinal cord injury is the primary injury, but the secondary consequences are often very challenging. Ultimately, treatment is expensive and often more expensive with short and long term medical consequences, and attendant costs.

Currently, the treatment of muscle and bone deterioration after SCI is very limited; therefore, there is a definite need to further understand the mechanisms of breakdown in the musculoskeletal system, and more importantly, find a clinical strategy to treat muscle and bone loss. Exoskeletons for assisted walking following spinal cord injury are novel robotic anthropomorphic mobile devices that are intended for rehabilitation, mobility and walking overground for those persons with SCI that are unable to walk. We are proposing that with continued walking overground with the device, muscle and bone will improve. We plan to test our hypothesis by allowing people to train or walk in the exoskeleton for 100 sessions, 5 times per week (1 hour/session). The device is easy to put on from a seated position and the skill of walking or stepping using the exoskeleton is relatively easy to learn. It is our intent to develop lines of research to show the benefits of walking overground in this device. These benefits include gains in muscle and bone, and an increase in general health and fitness for those persons who are 100% wheelchair reliant. This study identifies the progression of technology to enhance functional ambulation for persons with a spinal cord injury.

This project will be completed at two sites: Kessler Foundation Research Center will be the lead site that will be under the direction of Gail F. Forrest, Ph.D. (Principal Investigator), and Spinal Cord Damage Research Center, James J. Peters, VA Medical Center under the direction of Ann M. Spungen, Ed.D. (collaborator). Both sites have exoskeletal-assisted walking programs. We plan to measure many bone and muscle outcomes before and after the training protocol to answer our research question. And, more importantly, we are going to try and understand the mechanisms of why the bone and muscle is lost after the injury.

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NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Nancy Chiaravalloti, Ph.D.
Kessler Foundation

Grant Award: \$596,152

Project Title: *Impact of Age on Cardiovascular, Cerebrovascular and Cognitive Health in Spinal Cord Injury*

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, persons with tetraplegia 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. As people with SCI get older they are faced with increased likelihood of diseases, but they also are faced with the secondary complications of the SCI; such as the inability to control heart rate and blood pressure. As a result, it is more difficult to prevent and treat diseases and illness in the SCI population, which may worsen disease progression and reduce life expectancy. In addition, cardiovascular and cerebrovascular dysfunction may negatively affect thinking (cognitive) capacities and these individuals are reported to have increased likelihood of memory, information processing and executive function impairments.

In the general population there is evidence which supports a link between these cognitive impairments and cardiovascular and cerebrovascular dysfunction. Because the SCI population is aging, improving cognitive abilities while reducing cardiovascular and cerebrovascular dysfunction would be expected to improve health and quality of life, and contribute to increased life expectancy.

Therefore, the objectives of this project are to compare cardiovascular, cerebrovascular and cognitive function among individuals with SCI, age-matched non-SCI (NS) and older NS individuals. The results will help guide interventional studies aimed at improving health and longevity in the SCI population.

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NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Yi Ren, Ph.D.

Grant Award: \$592,568

Rutgers, The State University of NJ
W.M. Keck Center for Collaborative Neuroscience

Project Title: *Myelin Debris-derived Lipid Accumulation Modulates the Function of Macrophages in Spinal Cord Injury*

Macrophage activation and persistent inflammation are linked to the pathological process of spinal cord injury (SCI). There are mainly two types of macrophages: M1 (bad) and M2 (good) activation. M1 macrophages that produce pro-inflammatory cytokines act as an effector of cell killing. M2 macrophages are anti-inflammatory and contribute to wound healing and tissue-remodeling.

We demonstrated that infiltrated bone marrow derived macrophages migrated to the epicenter of injury, where they changed their M2 into M1 phenotype, which was induced by myelin debris generated in the injured spinal cord. There was striking defective lipid efflux in lesion site, which lead to the formation of foam cells and lipid plaques. These foam macrophages lost M2 phenotype and existed in the lesion site for a long period of time, which may contribute to the persistent inflammation because they showed a pro-inflammatory phenotype, enhanced neurotoxicity and impaired wound healing. Interestingly, there seems to be a negative correlation between lipid accumulation and the presence of M2 macrophages.

We therefore proposed a new concept that dysregulation of lipid efflux following macrophage phenotype switch may contribute to the pathological process of SCI. Approaches targeting reestablishment of the lipid homeostasis would be beneficial for the resolution of the inflammation.

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NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Michael Matise, Ph.D.

Grant Award: \$600,000

UMDNJ - Robert Wood Johnson Medical School

Department of Neuroscience & Cell Biology

Project Title: *The Role of the Shh in Mediating the Response of Adult Spinal Cord Stem Cells to Injury*

Spinal cord injury (SCI) in humans elicits a series of responses that serve two important roles: 1) to limit the extent of damage through reactive gliosis, and 2) to promote functional recovery through the generation of new cells to replace those lost as a result of trauma. Recovery from injury therefore requires that multiple, distinct cell types respond appropriately during both the acute response and prolonged recovery phases. Therapeutic strategies that are directed toward promoting recovery from SCI are based on one of two general approaches: cell replacement using transplantation of cultured "stem cells" that can produce specific derivatives, or encouragement of spinal cord resident stem cell populations to divide and differentiate. The latter approach has gained importance with the identification of adult CNS reservoirs containing quiescent, immature cells that can generate differentiated progeny following injury. In the spinal cord, cells with such properties have been identified in two locations: 1) scattered throughout the gray and white matter regions, and 2) within the ependymal zone (EZ) cell layer surrounding the central canal. Following injury, cells from both of these sources are induced to proliferate, and produce primarily glial derivatives (astrocytes and oligodendrocytes-OLs) that migrate toward the lesion site to play a role in the formation of the glial scar (reactive gliosis), restoration of the damaged blood-brain barrier, and re-myelination. However, the specific mechanisms involved in these important endogenous responses remain poorly understood and therefore cannot yet be fully exploited for therapeutic purposes in humans suffering from SCI.

We have recently obtained data indicating a novel role for the Sonic Hedgehog (Shh) pathway in establishing and/or maintaining glial stem cells in both the adult EZ and gray matter. This secreted signaling protein has been previously shown to play critical roles during embryonic development and also in maintaining adult neural stem cells in the brain; however its specific role in the spinal cord following injury has not been determined. Taken with experimental evidence which detected the production of Shh protein by spinal cord cells following contusion or de-myelinating lesions, as well as studies showing that the local injection of Shh following SCI in rodent injury models can lead to improved functional recovery, we hypothesize that the Shh pathway plays a crucial role in mediating the response of adult spinal cord stem cells to SCI. The goal of the current proposal is to test this hypothesis in mice, which will allow us to combine genetic manipulation with experimentally controlled SCI. We will accomplish the goals of this project in two aims. In Aim 1, we will determine the requirement for Shh signaling in adult EZ stem cells following SCI, while in Aim 2, we will ask whether Shh signaling is required in parenchymal stem cells following SCI. Both aims will combine sophisticated conditional genetic manipulations to block Shh signal reception in adult spinal cord glial progenitor cells following SCI. The fate of genetically-labeled stem cells will then be monitored at various times during the response and recovery periods to ascertain whether they are capable of generating the normal number of OLs and astrocytes in the absence of Shh signal reception. Together, these studies will help us better understand the important events that are involved in the response of spinal cord stem cells to injury and help lead to the development and implementation of therapeutic strategies directed at restoring function in humans suffering from SCI.

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FELLOWSHIP GRANT RECIPIENTS:

Stephanie Pyonteck, Ph.D.

Grant Award: \$150,000

Rutgers, The State University of NJ
Waksman Institute - Department of Genetics

Project Title: *The Regulation of Neuronal Mitochondrial Fusion by the Hypoxia Response Pathway Affects Functional Recovery and Survival Following Anoxic Stress*

Cells require oxygen and nutrients to make their own energy, which occurs in the subcellular powerhouse known as the mitochondria. The energy needs of neurons in the nervous system are dramatic, as high levels of cellular energy are required for the transmission of electrical signals in the brain and peripheral nervous system to allow for sensation and the control of movement. Following the initial trauma of spinal cord injury, swelling and decreased blood pressure impair blood flow and the delivery of oxygen and nutrients to the affected tissues, which is known as ischemia. Neurons are most acutely affected by ischemia following spinal cord injury because without oxygen and nutrients, neurons can no longer make more energy and they rapidly burn through their limited stored energy reserves. Thus, following energy depletion, the neurons rapidly begin to die. The massive amounts of neuronal cell death following spinal cord injury and the inability to effectively replace these cells often lead to permanent disability and paralysis with minimal chances for recovery.

Cells have many mitochondria to meet their energy needs, and mitochondria can exist either as small, individual units or fused together to function as a larger, more efficient network. Published research suggests that the interconnectivity of the mitochondrial network can affect how well cells are able to tolerate stress and if they are able to survive it. Using a well-established and transparent genetic model organism for studying neuron development and cell death, we are able to visualize the fusion and splitting of mitochondria in neurons in a more accessible model for what occurs in human neurons, and without the potential artifacts observed in a tissue culture system. We have found that oxygen deprivation triggers the breakdown of the mitochondrial network in neurons, and when oxygen is restored, mitochondria begin to fuse back together to restore the network. Similar findings have been reported in mouse and human neurons in response to ischemia, validating our model. We have also found that changes in an oxygen-sensing signaling pathway allow for neuronal mitochondria to “hyperfuse” and form a larger, more extensive network after oxygen deprivation. Importantly, this mitochondrial hyperfusion improves viability and speeds functional recovery of animals after long periods of oxygen deprivation, indicating that this hyperfusion can function as an inherent protective mechanism in neurons.

Our goal is to begin to understand (1) the molecular mechanisms of how neurons control mitochondrial splitting and fusion in response to oxygen levels, and (2) how and why hyperfused mitochondria can protect neurons following oxygen deprivation. This basic research will potentially allow for the identification of novel therapeutic targets to improve neuronal survival of ischemia following traumatic neural stress in the hope of improving patient outcome and reducing the clinical burden of spinal cord injury.

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NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Megan Damcott, Ph.D.

Grant Award: \$50,000

Kessler Foundation

Project Title: *Quantitative Measure of Force During Electrical Stimulation:
An Exploratory Study*

As indicated by the New Jersey Commission on Spinal Cord Research's Research Program Guidelines, priorities within spinal cord research encompass 'developing strategies which prevent or treat secondary complications' while 'translating basic and pre-clinical findings into clinical application'. Although basic science has provided critical insight regarding the treatment and prevention of musculoskeletal deterioration after spinal cord injury (SCI), the translation of this knowledge into clinical practice has yet to be accomplished. This project addresses the gap bridging the laboratory and clinic with the development of a quantitative measure which will allow for the translation of science into clinical practice.

Prolonged immobilization decreases the quality of life in individuals with SCI. The absence of mechanical loading within these individuals has been widely accepted as a significant factor in the prevalence of bone loss and osteoporosis: increasing the risk of fracture and mortality rates. To minimize and prevent musculoskeletal deterioration, mechanical loading interventions which allow for periods of upright posture and weight bearing have been investigated. While decades of research have been dedicated to developing therapeutic interventions for the improvement of bone health, determining the optimal parameters (mode, intensity, frequency, duration) for an efficacious loading intervention has been difficult due to the complexity of bone physiology. Basic science has provided critical insight: bones adapt to the loads in which they are placed. Specifically, if the forces produced on the bone are within a genetically predetermined threshold range, growth and mineralization to strengthen or maintain bone is generated. However, the inability to effectively quantify the forces placed on the bone during loading interventions has prevented the translation of this knowledge into clinical practice. Therefore, this project directly addresses this gap by exploring the development of instrumentation which can be utilized to quantify the forces applied to the bone across loading interventions and provide insight to allow determination of the optimal parameters to reduce bone loss, improve quality of life and maximize therapeutic benefits.

In individuals with SCI, mechanical loading has been investigated during stance and electrical stimulation (ES). However, the efficacy of ES in bone has yet to be reported. A limitation of early studies investigating the efficacy of ES has been the inability to measure the contractile force applied to the bone to determine if the magnitudes of loading were within the effective threshold range to modulate bone loss. Therefore, methods have recently been developed to quantify the magnitude of loading through the measure of external forces and moments (EKEFs and EKEMs, respectively) at the knee during ES induced isometric contractions of the quadriceps muscle. The measured EKEFs and EKEMs are then applied to mathematical models to estimate the internal forces applied to the femur. While these methods have provided quantification of loading during ES interventions, the accuracy of the measures and clinical applicability of the instrumentation and protocols is limited as the models are two-dimensional or use theoretical anatomical measures. Therefore, the primary aim of this project is to explore the limitations of the current measurement methods with the development of novel instrumentation, which will provide three-dimensional EKEFs and EKEMs during ES induced contraction of the quadriceps muscle while in stance. Three-dimensional quantification of the forces and moments holds the potential to increase the accuracy of the calculation of the internal forces applied to the bone and provide insight critical for maximizing the clinical efficacy of interventions.

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EXPLORATORY RESEARCH GRANT RECIPIENTS:

KiBum Lee, Ph.D.

Grant Award: \$200,000

Rutgers, The State University of NJ
Department of Chemistry & Chemical Biology

Project Title: *Enhanced Stem Cell Based Gene Therapy for Spinal Cord Injury
Using Novel Magnetic Coreshell Nanoparticles*

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. In addition, gliosis at the injury site is a source of a variety of inhibitory factors that prevent the damaged neurons from regenerating. 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, stem cell transplantation has shown great promise in treating SCI. In animal models, transplantation of neural stem/progenitor cells (NSPC) improved motor functional recovery in SCI via neuroprotection. However, this treatment has been found to be associated with increased hypersensitivity and neuropathic pain, which greatly affects the quality of life after SCI. Loss of inhibitory interneurons after SCI is thought to be a major mechanism underlying this altered sensory function. Therefore, replenishing inhibitory interneurons by NSPC transplantation may be beneficial. Yet, even though NSPCs show great potential to become inhibitory interneurons experimentally in the lab, it is not clear whether this formation will occur after SCI transplantation. In turn, an improved stem cell therapy would entail finding a way to force the NSPCs to become inhibitory neurons in the injury site, while also exploiting their innate neuroprotective ability described earlier.

In the proposed study, we have developed a nanotechnology-based approach to address these challenges. We propose to deliver specific genes into the NSPCs, which can then be activated after transplantation and direct the NSPCs to become inhibitory interneurons. This gene activation will be facilitated by the aid of a novel multifunctional magnetic core/shell nanoparticle (MCNP) system. The MCNP will provide three major advantages: 1) delivery of the gene vectors into the NSPCs, 2) spatial and temporal activation of the delivered genes, and 3) tracking of the NSPCs after transplantation. In this way, scientists and clinicians can harness the full potential of stem cells for an enhanced SCI treatment. Overall, our MCNP-based stem cell therapy is a hallmark of the next generation of SCI therapies that combine the benefits of nanotechnology and stem cell research.

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NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

Prabhas V. Moghe, Ph.D.

Grant Award: \$200,000

Rutgers, The State University of NJ
Department of Biomedical Engineering

Project Title: *Engineering Reprogrammed Neurons on Transplantable Scaffolds for Management of Spinal Cord Injury*

A major roadblock for the treatment of neurotraumatic disorders like spinal cord injuries is the lack of available neural tissue source that can be implanted at the site of injuries and lead to successful integration and restoration of sensory and motor functions of the spinal cord. Human neurons typically die when detached, which limits the possibility of harvesting healthy human neurons prior to transplantation to a patient. Human neural stem cells can be derived from a variety of cell sources, but neural stem cells do not efficiently differentiate to specific types of neurons and undifferentiated cells run the risk of becoming tumorigenic after transplantation in vivo. For all of these reasons, we propose to apply a novel approach to generate a human source of neurons and enrich the neuronal population using transplantable devices for transplantation.

The exploratory program takes on the challenge of reprogramming the behaviors of human somatic cell-derived stem cells (induced pluripotent stem cells) by transfecting these with neuronal transcriptional factor genes. The novelty of this program would be to improve the efficiency of neuronal reprogramming to yield sub-type specific motor neurons using minimal transfection using single factors and by harnessing combination of three-dimensional scaffold microenvironments (to select for neuronally maturing lineages) and presentation of biological signals (to retain the neuronal phenotypes).

To examine the efficacy of these neuronal constructs for healing spinal cord lesion, we will derive human iPSC-reprogrammed neurons using the 3-D constructs and then test the maturation and function of the neurons longer term.

As an exploratory program, this proposal sets the stage for design of transplantable devices containing rapidly reprogrammed neurons which would show high levels of survival and motor capacity upon implantation. In the long term, these insights could also help to develop appropriate human neuron-transplantation approaches in patients afflicted with spinal cord injuries.

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SPINAL CORD TECHNIQUES TRAINING GRANT RECIPIENT:

Stephen Kelly, Ph.D.

Grant Award: \$950

NJ Neuroscience Institute at
JFK Medical Center

Research in my laboratory is aimed at exploring the pathophysiology of and developing therapies for brain injury and related neurological disorders. My primary research goal is to develop a meaningful, widely applicable treatment for ischemic brain injury. I use a variety of approaches in this pursuit, including molecular, cellular, gene therapy, regenerative medicine (such as, stem cells, cell transplantation, enhancement of endogenous repair mechanisms), pharmacological, biomaterials and engineering techniques. I have a long-standing interest in spinal cord injury research (SCI) and strongly believe that my expertise and interests could be meaningfully translated to study this condition. Like stroke and traumatic brain injury, SCI is unpredictable and has a relatively rapid and inherently complex pathology that can, in time, result in a barren, acellular lesion surrounded by scar tissue. Moreover, many of the pathological features encountered during this transition and the barriers to meaningful treatment, repair and regeneration are also common to or similar to those faced in stroke.

There are two lines of work in my lab that I believe hold significant potential for SCI. The first of these explores the role of microRNAs (miRNAs) upon gene regulation in the ischemic and inflamed brain. We have identified a number of miRNAs that we believe can be targeted to reduce the degree of injury induced by these insults, and to enhance endogenous repair mechanisms. We have found that one of these miRNAs, miR-132, is significantly reduced by severe ischemia in vivo, but increased by neuroprotective bouts of ischemic preconditioning. Moreover, overexpression of miR-132 was found to protect cultured hippocampal neurons from ischemic and excitotoxic injury. As well as its potential neuroprotective role, miR-132 is induced by neuronal activity, contributes to dendritic plasticity, and plays a key role in synaptic integration and survival of newborn neurons in the brain. We hypothesize that miR-132 could be beneficial in SCI and will use our inducible miR-132 mutant mice to investigate this.

The second line of investigation is exploring the utility of combining novel, microstructured biomaterials with neurons and stem cells to develop tailored 3D cellular networks that can be deposited into the ischemic cortex following stroke. We are generating tailored poly lactide nerve growth conduits using a combination of computer-aided design and 3D printing. These structures have been shown to support neural and stem cell growth and can be modified to release factors of interest (such as growth factors or anti-inflammatory molecules) to enhance cell survival and modify the host environment. While we continue to work toward using this approach to treat cortical stroke, we strongly believe that it may be far better suited to SCI.

A New Jersey Commission SCI Techniques and Training grant would provide me with expert training, an opportunity to interact with potential collaborators in this field, and would kick-start this SCI work in my laboratory/department.

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