DIRECTORY OF GRANT AWARDS
2008 B GRANT CYCLE

NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH

2008 B CYCLE

DIRECTORY OF GRANT AWARDS FOR SPINAL CORD INJURY AND DISEASE RESEARCH

DECEMBER 2007

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 2008 B 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.

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NEW JERSEY COMMISSION ON SPINAL CORD RESEARCH
GRANT AWARDS

INDIVIDUAL RESEARCH GRANT RECIPIENTS:

PRINCIPAL INVESTIGATOR – Li Cai, Ph.D.

Rutgers, The State University of NJ
Department of Biomedical Engineering
Basic Science Proposal
Grant Award - $400,000

Proposal Title: ***Control of Olig2 Expression in Spinal Cord Development***

Demyelination is a pathophysiologic component of compressive spinal cord injury (SCI). Oligodendrocytes (OL) are the myelin-forming cells in the brain and spinal cord; they are essential for axonal conductance and thus proper neuronal function. After a spinal cord injury, previously myelinated axons persist in the injury site, but do not become myelinated and, hence, remain non-functional or die. If these axons could be remyelinated some neuronal functions that are lost could be restored. Therefore, the study of the formation of OL progenitors and OL is of great clinical importance for SCI.

The Olig2 gene has been demonstrated to be essential for the differentiation of neural stem cells into myelinating OL as well as motor neurons (MN). In mutant mice, the lack of Olig2 expression leads to the complete loss of OL and MN. The dual roles of Olig2 implicate the existence of two types of genetic control elements, i.e., enhancers (non-coding sequences with the ability to determine tissue/cell type-specific expression of particular genes) that regulate OL- versus MN-specific Olig2 expression. Recently, enhancers that regulate MN-specific Olig2 expression (MN-enhancers) have been identified; however, enhancers that regulate OL-specific Olig2 expression (OL-enhancers) have remained yet to be identified. Based on the fact that many enhancers are evolutionarily conserved, and that enhancers can determine lineage-specific gene expression upon binding of specific trans-acting factors; we propose an integrative approach combining state-of-the-art computational prediction and experimental verification for the identification of OL-enhancer(s) of the Olig2 gene. First, evolutionarily conserved non-coding sequences surrounding the Olig2 gene will be computationally predicted as putative enhancers. The putative enhancers will be compared with the known MN-enhancers of the Olig2 gene to predict OL-enhancers based on the absence of MN-specific and the presence of OL-specific trans-acting factor binding sites. Then, the predicted OL-enhancers will be experimentally verified using molecular genetic methods.

The proposed research will define the role for OL-enhancers in regulating Olig2 expression during OL lineage development. A comprehensive knowledge on the regulatory mechanisms governing OL formation and regeneration after demyelination will be invaluable in directing stem cell differentiation into myelinating OL for future therapeutic transplantations in SCI patients.

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PRINCIPAL INVESTIGATOR – Dewey Royal, Ph.D.

Rutgers, The State University of NJ
Department of Molecular Biology & Biochemistry
Basic Science Proposal
Grant Award - $200,000

Proposal Title: ***Genes Contributing to Necrosis in Physiological Injury***

In spinal cord injury, some neurons are directly damaged, but many others die during a phase of secondary necrosis induced by exacerbated ion channel activity. It has been suggested that maintaining just 10% of spinal cord function is sufficient for basic mobility. Thus, blocking or delaying secondary neuronal necrosis would significantly limit incapacitation consequent to injury. A more detailed understanding of the molecular mechanisms of necrosis is required for design of novel and effective therapies.

A central goal of our work is the identification of genes critical for the progression through necrosis induced by ion channel-inflicted neuronal injury. We are exploiting uniquely applied genetic approaches in the C. elegans model system to identify necrosis suppressor mutations. Some key advantages of this system include the transparent body that allows us to directly observe dying neurons in the living animal and the ability to conduct exhaustive genetic hunts for mutations such as those that block neuronal necrosis. In general, experiments that are implausible or impractical in higher organisms can be conducted rapidly, cheaply, and with cleanly interpretable results in C. elegans. Since most basic biological processes, including cell death, are conserved from nematodes to humans, we can identify critical molecules and decipher the molecular basic rules of a given process in simple C. elegans and then use this information to address the function of related molecules (homologs) in humans. The underlying working hypothesis of our research is that molecular elaboration of necrosis mechanisms in C. elegans will identify key molecules needed for the progression through necrosis in humans-disruption of these human genes, or the inactivation of their protein products, is a highly plausible strategy to block the devastating consequences of necrosis that follows initial injury and to prevent the cascade leading to neuronal demise.

Our first aim is to determine the molecular identities of two necrosis suppressor genes, to define the genetic mutations that cause necrosis suppression, to determine at which step of the necrosis process the new genes are likely to work, and to test if mammalian versions of these genes can exert the same function. This work is important because we will identify novel molecules that contribute in significant ways to the necrosis that accompanies injury. The impact of this work is that since these molecules are likely to be similar in nematodes and humans, the data we generate can allow intelligent design of much-needed effective intervention therapies.

Our second aim is to directly test the hypothesis that the ASIC1a channel contributes to neuronal loss in a mouse spinal cord injury model. Several lines of evidence suggest that ASIC1a channel (the mammalian counterpart of the C. elegans necrosis-inducing channel that we study) could contribute to secondary necrosis in mammalian SCI. For example, genetic elimination of this channel is hugely neuroprotective in brain ischemia. ASIC1a is present in mammalian spinal cord, but no one has yet tested whether it contributes to secondary necrosis in spinal cord injury. We will collaborate with investigators in the Keck Center for Collaborative Neuroscience to monitor outcome of spinal cord injury in ASIC1a knockout mice. Should we establish a contribution of ASICa to neuronal loss in SCI, the outcome will identify a new player (and possibly a very major player) in secondary necrosis and suggest a clear strategy for therapeutic intervention.

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PRINCIPAL INVESTIGATOR – Stella Elkabes, Ph.D.

UMDNJ – New Jersey Medical School
Department of Neurology and Neuroscience
Basic Science Proposal
Grant Award - $197,562

Proposal Title: ***Novel Targets for Neuroprotection in the Spinal Cord***

Traumatic injury of the spinal cord (sc) causes neuronal death. Although initially neuronal loss is the consequence of the mechanical impact, the induction of inflammation and the release of toxic materials by damaged cells further promote this process, exacerbating the outcome of sc injury. The mechanisms underlying the death of sc neurons are not well understood. Delineation of such mechanisms is critical for the identification of targets amenable to therapeutic interventions which will prevent neuronal death and promote neuroprotection.

Earlier studies in this laboratory indicated that plasma membrane calcium ATPase 2 (PMCA2), an ion pump which expels calcium from cells, is involved in sc neuronal damage. PMCA2-mediated calcium extrusion is an important mechanism that ascertains the appropriate calcium levels within nerve cells, as excess calcium can have detrimental effects. A decrease in the level or activity of PMCA2 may increase calcium concentrations causing neuronal dysfunction and death. Previous experiments indicated that the levels of PMCA2 are decreased, not only in the epicenter but also in the area surrounding the injury after sc contusion. Moreover, in sc neurons grown in the dish, prevention of pump activity caused accumulation of calcium which was followed by neuronal pathology and death. The molecular events that lead to neuronal loss after a decrease in PMCA2 are not yet defined. The present proposal will address this issue.

Additional studies have been initiated to determine the triggers that induce a change in PMCA2 activity or levels with particular emphasis on inflammatory agents or toxins that are found in the injured sc. Among those toxins is glutamate, a neurotransmitter which plays critical roles in neuronal communication in the healthy central nervous system. However, after sc trauma, high amounts of glutamate are released by injured neurons and some inflammatory cells. Elevated glutamate concentrations can cause damage, often due to abnormal increases in calcium levels within the neurons. Preliminary data indicate that glutamate suppresses and inflammatory agents have differential effects on PMCA2 levels in neurons. Therefore, it is postulated that management of the inflammatory environment after sc injury may inhibit the decrease in PMCA2 or restore its level and activity, preventing neuronal death. However, before manipulating the inflammatory milieu, it is necessary to define which agents modulate PMCA2 and how this affects calcium balance. The present proposal will also examine this question.

The long-term goal of these investigations is to determine whether changes in PMCA2 underlie neuronal damage and if restoration of calcium pump activity and expression confer neuroprotection in sc injury. Another aim is also to establish the beneficial or detrimental impact of the inflammatory milieu on PMCA-mediated neuronal damage.

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PRINCIPAL INVESTIGATOR – Melitta Schachner, Ph.D.

Rutgers, The State University of NJ
Department of Cell Biology and Neuroscience
Basic Science Proposal
Grant Award - $275,000

Proposal Title: ***Glycan-Mediated Improvement of Spinal Cord Regeneration***

Glycans carried by neural cell adhesion molecules have recently come into focus as promoters of neuronal survival and neurite outgrowth, both of which are features that lend themselves to investigations on their effectiveness in spinal cord regeneration of mammals. We have shown that two prominent glycans shared by neural cell adhesion molecules enhance locomotor recovery after spinal cord lesion of adult mice. Glycan mediated recovery was effective already two weeks after infusion of the combination of the peptidomimetics for polysialic acid and sulfoglucuronyl carbohydrate structures into the lesioned spinal cord.

We now wish to analyze the molecular and cellular mechanisms underlying the improved recovery of locomotor function after spinal cord lesion. We will use immunocytochemical, biochemical and molecular biological methods as well as tracing the nervous system tracts known to be important for recovery of function to evaluate the basic mechanisms underlying regeneration and thus design tools to overcome the largely inhibitory environment of the spinal cord in a mouse model of spinal cord regeneration.

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PRINCIPAL INVESTIGATOR – Audrey Minden, Ph.D.

Rutgers, The State University of NJ
Department of Chemical Biology
Basic Science Proposal
Grant Award - $200,000

Proposal Title: ***The Role for Pak4 in Motor Neuron Development***

Spinal cord injury can result in severing of the nerves in the spinal cord, which leads to severe disability. The injury to spinal cord neurons leads to sensory and motor defects. The severing of motor neurons at the spinal cord often leads to complete or partial paralysis. There is considerable interest in using stem cell technology to regenerate new spinal cord neurons following injury. However, this requires an in depth understanding of how spinal cord neurons develop, and what molecular factors control their differentiation.

In our lab we work on a family of protein kinases called Pak4, Pak5 and Pak6. Pak5 and Pak6 are found mostly in the nervous system and also in several other tissues, while Pak4 is present in all tissues. We have generated knockout mouse models in which each of these proteins is completely eliminated. We have found that mouse knockouts which lack Pak5 and Pak6 are viable, but have a decrease in their motor activity. In contrast, Pak4 knockout mice die in utero, and examination of the knockout embryos reveals a severe defect in the development of motor neurons. The early death of these mice is most likely not caused by the poor development of motor neurons, but to an abnormality in the heart and extra embryonic tissue. Therefore, to study the role for Pak4 in motor neuron development in more depth, we have developed conditional knockout mice, in which Pak4 is eliminated only from the nervous system, but not from other tissues. The goal is to obtain live mice that lack Pak4 in the nervous system. These mice will be used to determine whether Pak4 is required for motor neuron development, and to determine at what stage in motor neuron development Pak4 plays an important role.

The mechanism by which Pak4 mediates motor neuron development will also be addressed. We will determine whether Pak4 plays a role in promoting survival of neurons, and we will also determine whether it has a role in regulating the activity of a transcription factor, Islet1, which has a key role in motor neuron development. In the future, a better understanding of how Pak4 regulates the development of motor neurons will aid in the development of regenerative therapies in which motor neurons can be restored or generated from stem cells. Such regenerative therapies will hold promise for benefiting spinal cord injury patients in New Jersey and throughout the world.

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FELLOWSHIP GRANT RECIPIENTS:

PRINCIPAL INVESTIGATOR –Ye He

UMDNJ-Robert Wood Johnson Medical School
Department of Neuroscience and Cell Biology
Graduate Student Fellowship - $60,000

Proposal Title: ***YY1 in Schwann Cell Proliferation and Myelination***

Spinal cord injury is a devastating neurological injury affecting half million people in United States including more than 6,000 New Jersey residents. SCI patients suffer from loss of sensation or motor ability, which is mainly due to disrupted axonal connection, demyelination and consequent neuronal death.

The current strategies to cure spinal cord injury include the protection of the neurons and the promotion of axonal growth and remyelination. Schwann cells are the myelin forming cells of the peripheral nervous system. Transplantation experiments have shown that implanting Schwann cells stimulates remyelination and thereby restores function. In addition, Schwann cells are also capable of secreting the nourishing factors that protect neurons and facilitate the re-growth of damaged axons. These properties make Schwann cells a promising candidate to be transplanted into the injured spinal cord. In addition, in clinical perspective, it is it is conceivable that limited amount of Schwann cells can be isolated from the biopsies of the sciatic nerve of the patients themselves. These cells need to be expanded in vitro and then transplant back into the injured spinal cord. However , how to expand the Schwann cells in culture and how to achieve efficient remyelination of the regenerated axons after engrafted into spinal cord remains challenging. Thus it is important to study the mechanism of Schwann cells proliferation and myelination.

Transcription factor Yin Yang 1 (YY1) was identified in our laboratory as an important regulator of Schwann cell development. Preliminary study conducted in our lab has showed that lacking of YY1 in myelinating cells prevents the formation of myelin and results in the accumulation of immature cells. Here I propose to further explore the function of YY1 in the proliferation and myelination of Schwann cells and the possible underneath molecular mechanism using YY1 mutant mice generated in our laboratory. The proposed study will further our understanding of Schwann cells with the hope that information derived from basic science research will improve Schwann cells transplantation strategies for spinal cord injury in the future.

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PRINCIPAL INVESTIGATOR – Ian Guadet

Rutgers, The State University of NJ
Department of Biomedical Engineering
Graduate Fellowship Grant Award - $60,000

Proposal Title: ***Nerve Guidance via Photonically Derived Durotactic Gradients***

Spinal cord injury (SCI) is a huge economic burden, and the cost in terms of human suffering is even more staggering. SCI is characterized by damage to axons in the white matter of the spinal cord. The myriad inhibitory cues, along with the presence of the glial scar, present a barrier that prevents damaged axons from regenerating in and across the injury site.

Currently, many strategies for overcoming the inhibitory environment employ some type of artificial scaffold that can modify the local injury environment, deliver guidance cues to cells, and mitigate the inhibitory cues. One class of scaffold materials includes hydrogels, which are fluid polymers that can be crosslinked to become stable. Hydrogels have the advantage of potentially being injected into the injury site, then crosslinked to hold their shape. Several groups are exploring various ways to modify hydrogels to more efficiently guide neurite extension across the injury site, including incorporation of soluble cues for drug delivery, attachment of adhesion molecules, and variability in the mechanical properties.

Recently, we have discovered that neurons will respond to a gradient of mechanical stiffness in a 3D environment. Neurons can be guided by presenting an environment that becomes more compliant in one direction, and will preferentially extend neural processes such as axons towards an increasingly compliant substrate. Currently, we employ chemical crosslinking to create gradients of stiffness in collagen gels. Herein, we propose a method for creating gradients of stiffness using photonically reactive hydrogels in conjunction with variable incident light intensity. It has been previously shown that the material properties of hydrogels may be manipulated by incorporating photoinitiators, which can then be used to control the amount of crosslinking through exposure to light. Furthermore, cells can tolerate the photocrosslinking process, so that exogenously administered cells such as radial glia, astrocytes, or nerve cells that have shown promise in SCI regeneration might be compatible with this method. The most obvious advantage with this approach is that the liquid polymer can be applied to any shape of defect, then crosslinked in place to form a functional 3D scaffold.

The system outlined in this proposal is flexible, as the materials described can be grafted with adhesion peptides and guidance cues, modified to be degradable, and with the approach described here, crosslinked in a way that the material properties will guide the regrowth of neurites. Another important aspect of this method is that since light will be the tool used to form the gradient, almost any pattern can be created to guide neurons across complex geometrical constraints, which is not easily accomplished using chemically crosslinked scaffolds.

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PRINCIPAL INVESTIGATOR – Evangeline Tzatzalos

Rutgers, The State University of NJ
Department of Biomedical Engineering
Graduate Fellowship Grant Award - $60,000

Proposal Title: ***Notch Promotes Radial Glial Phenotype in Neural Progenitors***

Cell transplants are a potential therapy for spinal cord injury because they replace damaged cells, provide nutrients to the injured host tissue, and guide the regeneration of endogenous neurons. Transplants of mature brain or spinal cord cells into the spinal cord do not survive very well. However, transplantation of stem or progenitor cells are promising. Radial glial cells are neural stem/progenitor cells, which can give rise to neurons and glia. Glia are non-neuronal cells that provide support, nourishment, and waste-removal services for neurons. Radial glial also have the ability to migrate and release survival factors. It is hypothesized that radial glia can migrate over and around spinal cord injury sites, essentially creating bridges over lesion sites. It is also hypothesized that such bridges will act as guides for the damaged neurons to regenerate. A shortcoming of radial glial cells may be their differentiation into mature cells when exposed to the toxic environment of the injury site.

In order to take full advantage of the radial glial properties, we propose to genetically modify the transplant cells to delay their differentiation and maintain their radial glial state even when in the injured spinal cord. Notch is a gene that delays neural differentiation. It is hypothesized that Notch-expressing mouse neural stem cells will maintain their radial glial properties in the injury site, thereby allowing migration across the lesion site and creation of bridges for nerve regeneration and potential functional recovery. A potential limitation of Notch-expressing cells is the reduction in cell proliferation. To address this problem, we propose to engineer the combined expression of Notch and v-myc, which is a gene that can immortalize cells and recover proliferation. We will determine optimal Notch/v-myc levels for maintaining radial glial properties and proliferation with in vitro characterization of Notch/v-myc expressing cells. In vivo experiments will be performed to test whether Notch/v-myc expressing mouse cells will proliferate and maintain radial glial properties in the injured mouse spinal cord. We expect that cells that are generated will be to promote recovery following spinal cord injury.

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PRINCIPAL INVESTIGATOR – Damien Carrel, Ph.D.

Rutgers, The State University of NJ
Department of Cell Biology and Neuroscience
Postdoctoral Fellowship Grant Award - $100,000

Proposal Title: ***Neuroprotective Effects of Cypin after SCI***

In this proposal we will explore a way to protect neurons from dying during spinal cord injury, by blocking one of the most destructive pathways that occurs when there is an insult to the spinal cord.

During spinal cord injury, a neurotransmitter called glutamate is released in extremely high quantities. This glutamate can then act on proteins called receptors that transduce signals into the neurons. These signals include chemicals called reactive oxygen species or ROS that have detrimental effects on the neurons, and these effects often lead to neuronal death. Until now, very little is known about how we can block either the receptors that lead to the production of the ROS or how we can block or bind up the ROS so that they cannot do damage to the neurons.

We will focus on identifying methods to prevent the action of glutamate on its receptors. Indeed, we have been studying a protein called cypin and found that cypin decreases glutamate receptor and signaling proteins at the synapse, or signaling site of a neuron. By decreasing signaling, neuronal death should decrease. This new approach can lead to promising therapies that will decrease neuronal death during spinal cord injury, maintaining normal spinal cord function.

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PRINCIPAL INVESTIGATOR – Yee Shuan Lee

New Jersey Institute of Technology
Department of Biomedical Engineering
Graduate Student Fellowship Grant Award - $60,000

Proposal Title: ***Fibrous Scaffold for Functional Spinal Cord Injury Recovery***

Spinal cord injury (SCI) is the loss of movement and sensation from the areas of the spinal cord below the site of injury. In the US alone, there are approximately 250,000 people living with SCI and 11,000 new cases every year. Traditional interventions focus on prevention of further damage to the injury site.

Tissue engineering is an emerging technology that combines the aspects of engineering and biological science toward restoring, maintaining, or improving tissue function and may provide a possible solution for functional recovery. A biomaterial conduit (scaffold) is often used with specific cells to achieve tissue repair and regeneration made from synthetic or biological materials. The surface properties influence cellular behavior significantly and different surface patterns have the ability to guide neurite growth, attachment and proliferation. Local electric activities have been observed during neural development or neural injury in various species. The electric activities provided by the supporting tissue may provide guidance signal for outgrowth. Piezoelectric materials induce transient electric changes on the surface, which have been shown to enhance neurite extension. Thus, the use of an aligned fibrous piezoelectric scaffold in combination with stem cells is a novel approach to promote neuronal growth and enhance neurite extension to improve functional recovery after SCI. Piezoelectric compositions of PVDF and PVDF-trFe will be used to fabricate micro and nano size aligned and random fiber scaffolds. PLLA is a commonly used biodegradable polymer will serve as a control. Rat PC12 cells, is a cell line that has been well characterized for its neural morphology in the presence of nerve growth factor. Human neural stem cells (hNSCs) have the ability to self-renew and to differentiate into various functional cell types such as neurons, astrocytes, and oligodendrocytes when proper cues are presented.

This study will investigate the percentage of neuronal differentiation and neurite extension of PC12 cell and hNSCs on various fibrous scaffolds. This study hypothesizes aligned, fibrous piezoelectric scaffold will display physical cues to promote the expression of the neuronal phenotype and neurite extension. The scaffold shows maximum, uniaxial neurite extension and number of neurons will be selected for implantation in the animal study. Rats will be injured by contusion method to mimic spinal cord injury. The optimal scaffolds with or without hNSCs will be implanted to the site of injury after various time points to mimic the clinical scenario. The functional recovery of the rats will be evaluated by a scoring system for the range of motion that the animal can achieve at various time points after implantation. This study hypothesizes the scaffold with or without hNSCs will restore function after spinal cord injury. The results of this study will facilitate therapeutic interventions for spinal cord injury repair.

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