The Merging of Technology and Biology to Enhance Function
This year promises to be one of the most exciting periods of discovery and translational progress for The Miami Project to Cure Paralysis. We are in the final stages of seeking permission from the FDA to initiate a Phase I safety trial in people with subacute spinal cord injury (SCI) using our human Schwann cell transplantation procedures. Therapeutic hypothermia studies continue to move forward and federal funding for a 17-center multicenter trial to test the efficacy of early cooling in a large number of individuals with acute spinal cord injury is being requested this year. Basic science discoveries continue to provide exciting information regarding mechanisms of axonal regeneration and combination approaches to promote more complete functional recovery in preclinical models of SCI. The potential use of cutting-edge bioengineering approaches to promote walking and hand function are becoming a reality within The Project as our scientists reach out to various biotechnological companies to build alliances to target paralysis. We greatly appreciate the support of our donors and scientific colleagues who are helping to provide critical resources as we move our investigations forward.

In this edition of our Research Review, several exciting areas of discovery are highlighted. Novel therapeutic interventions that target the acute injury setting to prevent secondary injury mechanisms provide hope for a better understanding of the injury process and repair strategies. In the area of acute protection, it is possible that cooling plus the addition of novel pharmacological interventions could provide more complete protection and functional recovery in combination rather than separately. Thus, continued investigations toward understanding mechanisms of neuronal cell death are important to the field and The Project’s ultimate goal of protecting against spinal cord injury.

In the area of repair for chronic injuries, the scientific field could not be more exciting. Novel molecules are being identified that can potentially target both intrinsic and extrinsic mechanisms for successful axonal regeneration and provide novel therapies for the chronically injured who are living with paralysis. For example, federal support is helping us to move...
a newly identified molecule that enhances regeneration forward in preparation for future clinical trials targeting axonal regeneration. Other novel therapies utilizing new gene therapies are targeting molecules that one day could be used in combination with other therapeutic strategies, including human Schwann cell transplantation. To further enhance long track regeneration, research collaborations using novel scaffolding materials to enhance axonal growth across gaps in the spinal cord are being evaluated. Because SCI is a heterogeneous condition, it is envisioned that different therapeutic interventions may be most appropriate depending on the severity of injury and degrees of structural damage. Thus, investigating multiple ways to protect and repair the nervous system is extremely important.

Neurorehabilitation work is also being initiated to be used in future trials as a means to enhance reparative strategies in individuals living with chronic spinal cord injury. A new study is being developed to establish baseline responses to protocols involving exercise conditioning and neurorehabilitation strategies in the different groups of chronic SCI (complete, incomplete, thoracic, cervical). That data will be used in the future when we are ready to amend our Investigational New Drug (IND application) targeting human Schwann cell transplantation in chronically injured individuals. For that clinical trial, which would be initiated after we establish safety in the subacute Schwann cell transplantation trial, we will combine Schwann cell transplantation with exercise conditioning and rehabilitation to maximize the potential amount of recovery. The potential use of recently developed robotic exoskeleton systems to enhance walking as well as novel brain-machine interface technology could be coupled with our established rehabilitation studies to further enhance function. Thus, the potential merging of biological and engineering approaches to target SCI is a very exciting area of investigation for Miami Project researchers.

We continue to concentrate on quality of life issues which affect people living with paralysis. Our pain group is testing novel therapies and continuing to understand the underlying mechanisms for abnormal sensation in our subjects. Fertility problems in men living with paralysis continue to be investigated with our goal of providing a clearer understanding of how to treat this condition. New concerns regarding the effects of aging with SCI are prompting our researchers to further target their clinical investigations in this area. Currently, approximately 17 active clinical trials are ongoing in the Lois Pope LIFE Center that are hypothesis-driven programs resulting in new information that is changing the way we think about consequences of spinal cord injury. In 2011, active trials enrolled over 500 individuals with chronic SCI, and we are grateful to all of our research participants for contributing to our knowledge regarding SCI and providing new information for future clinical studies. The Project therefore represents a unique scientific environment by which discovery, translational, and clinical research comes together with the ultimate goal of advancing new therapies to protect and promote recovery in our SCI population. These indeed are exciting times, and we again thank our friends and colleagues for their long-term support and commitment to our program. 2012 is going to be an outstanding year, and we thank everyone for their support of our research programs.
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A Publication of The Miami Project To Cure Paralysis & The Buoniconti Fund To Cure Paralysis

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The Miami Project scientific team is grateful for the dedication and hard work of the fundraising, administrative, and scientific support staff. This incredible group of people spend countless hours providing direct clerical and administrative support to the research staff, and raising the precious private funds to support Miami Project research endeavors.

**Fundraising, Administrative, and Scientific Support Staff**

**Management**

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Dr. Roberta Brambilla, a junior faculty member at The Miami Project, published results in 2011 that are key to unraveling the confusion surrounding an important immune signaling molecule involved in the disease progression of multiple sclerosis (MS). This is important for spinal cord injury (SCI) as well because many of the tissue damaging processes associated with MS are also associated with SCI, particularly the loss of insulation (demyelination) around individual nerve fibers (axons) and damage to axons themselves.

So what is this molecule? Well, its’ official name is Tumor Necrosis Factor, but let’s just call it TNF for short. TNF comes in two forms and they have opposing activities. The first form is attached to cells and it is called transmembrane TNF or tmTNF. This is the good form because it promotes cell survival, a reduction of inflammation, and the formation of myelin. The second form is not attached to cells; it moves around the body and it is called soluble TNF or sTNF. sTNF
They found that **inhibiting** just the soluble form of TNF with XPro led to a **significant improvement** in motor function, promoted axon survival, and **stimulated the reformation** of healthy myelin around demyelinated axons.

![Image of axons with myelin and different treatments](image)

Axons wrapped with myelin, under various experimental conditions, in the thoracic region of the spinal cord: **Naive** = from a healthy mouse; **Vehicle** = from a mouse model of Multiple Sclerosis (MS); **Etanercept** = MS mouse treated with the drug etanercept; **XPro1595** = MS mouse treated with the drug XPro1595. The black arrows point to completely collapsed, damaged axons in vehicle and etanercept, which are not seen as often with XPro. In the XPro picture, the red arrows point to remyelinating axons.

is bad since it causes cell death and chronic inflammation. Normally, these two forms of TNF work together to keep the system balanced. During diseases such as progressive MS, however, the balances appear to get shifted more towards the negative activities triggered by sTNF.

Dr. Brambilla, in collaboration with senior faculty member, Dr. John Bethea, demonstrated the effects of selectively inhibiting sTNF in an animal model of MS. They were clever in that they compared the effects of 2 drugs that inhibit TNF; XPro1595, which selectively inhibits only the sTNF form, and etanercept, more commonly known as Enbrel, which non-selectively inhibits both forms of TNF. This enabled them to tease out the different mechanisms by which each form contributes to the disease progression. They found that inhibiting just the soluble form of TNF with XPro led to a significant improvement in motor function, promoted axon survival, and stimulated the reformation of healthy myelin around demyelinated axons. Wow! So, instead of trying to knock out all TNF activity with a broad acting drug like Enbrel, its better to block the bad guy, sTNF, and leave the good guy, tmTNF, free to promote tissue repair. The research team took this a step further and evaluated post-mortem spinal cord tissue samples from three individuals that had chronic, progressive MS. They found the same characteristics indicative of sTNF and tmTNF activity in the human diseased tissue as they found in their animal model tissue. This is important because it demonstrates that the animal model is mimicking the human disease in a similar fashion and the results they observed with XPro in the animal model will likely translate to the human.

Her data was so compelling that the reviewers accepted her manuscript without requiring any modifications or additional data. That’s rare for any scientist! In fact, one reviewer went so far as to state that “the authors have addressed a very important area of research and have ‘demystified’ the issues around TNF that plagued the field for over a decade; congratulations to the authors for this work that truly advances our knowledge.” The manuscript was published in the highly regarded journal called *Brain* (2011, volume 134, pages 2736-2754). The results were also noticed by the Society for Neuroscience and were featured in their Hot Topics for the press at the annual meeting in November 2011 in Washington, DC.

The company that manufactures XPro1595, Xencor, recently obtained FDA approval to begin a clinical trial testing the drug in rheumatoid arthritis, another progressive inflammatory disease involving TNF (for more details see [www.xencor.com](http://www.xencor.com)). While the rheumatoid arthritis clinical trials are being planned, based on the promising data obtained with XPro1595 by various researchers in models of neurodegenerative diseases, including the studies by Drs. Brambilla and Bethea, Xencor is now seeking partnership with other pharmaceutical companies to license XPro1595 for use in neurological disease, MS in particular. Those other companies would then pursue clinical trials with XPro1595 unique to their interests. Thanks to the hard work of Dr. Brambilla, Dr. Bethea, and their research teams another mystery is unraveled in the disease arena involving inflammation! 🎉

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**Targeting TNF**

Axons wrapped with myelin, under various experimental conditions, in the thoracic region of the spinal cord: **Naive** = from a healthy mouse; **Vehicle** = from a mouse model of Multiple Sclerosis (MS); **Etanercept** = MS mouse treated with the drug etanercept; **XPro1595** = MS mouse treated with the drug XPro1595. The black arrows point to completely collapsed, damaged axons in vehicle and etanercept, which are not seen as often with XPro. In the XPro picture, the red arrows point to remyelinating axons.
The ability to induce regeneration of damaged nerve fibers (axons) in the central nervous system (CNS) has been a significant challenge to the neural regeneration field for decades. This neurotherapeutic target is important not only to spinal cord injury, but also to traumatic brain injury, optic nerve injury, and many other neurological diseases. For so long this has appeared as an insurmountable obstacle, but during the past few years Miami Project faculty members John Bixby, Ph.D. and Vance Lemmon, Ph.D. have been making significant headway.

You may recall from previous magazine and newsletter articles that Drs. Lemmon and Bixby have developed a special laboratory that utilizes "high throughput" screening technology to discover novel genes and drugs that stimulate or inhibit regeneration in cell culture. Their laboratory can...this project was selected to be part of the National Institutes of Health (NIH) Blueprint Neurotherapeutics Network to develop drugs that can be used in clinical trials.
The Project

Lemmon - Bixby Lab

Jeffrey Goldberg, also at the University of Miami, and Prof. Young-Tae Chang at the National University of Singapore. They have identified four compounds that enhance axon growth of a variety of neurons in inhibitory environments. One of these has been found to enhance regeneration in an acute spinal cord injury model as well as an optic nerve injury model (see Figure 1). That compound is called F05.

So, how do we move this exciting compound forward to determine if it has potential as a neurotherapeutic drug to test in clinical trials? Can we just take this compound, F05, as is, design a clinical trial, and start giving it to humans?

Unfortunately, it’s not that simple. However, this project was selected to be part of the National Institutes of Health (NIH) Blueprint Neurotherapeutics Network to develop drugs that can be used in clinical trials. When a compound, like F05, is shown to be promising in an academic setting, such as ours, it must go through a rigorous drug development pipeline (see Figure 2) before it can be tested in humans. This pipeline is often called the valley of death because most basic science researchers cannot obtain the resources to perform all the “boring, but critical” tests necessary to develop the compound to a point where it becomes attractive to the pharmaceutical industry, and most pharmaceutical companies hesitate to invest in the neurotherapeutics drug development pipeline too early because of a long track record of failure, lack of clinically validated targets, and the fact that many CNS disorders affect relatively small populations in comparison to more widespread diseases such as cancer or heart disease. As a consequence, many potentially promising discoveries never make it through the pipeline.

One of their recent projects has been to screen a novel chemical compound library to identify compounds that can overcome the regeneration-inhibitory effects of the injured CNS. They have been working on this in collaboration with Dr.

Figure 1. F05 stimulates significant regeneration of damaged axons (green) in an optic nerve injury model when the cell bodies are kept alive with a growth factor (CNTF).

Figure 2. This diagram depicts the multiple processes that must occur from the time a target drug is discovered through completion of clinical trials. The “valley of death” constitutes all of the processes required to take a compound from proof of concept experiments to full-fledged clinical trials, and this is where the NIH Blueprint Neurotherapeutic Network has stepped in to assist academic centers with promising therapeutic compounds.
Fortunately, NIH recognized this problem and developed the Blueprint Neurotherapeutics Network to try to facilitate translation of basic discoveries to the clinic. After a highly competitive process, Drs. Lemmon, Bixby, and Goldberg were 1 of only 7 projects to be funded in the initial round. Their team will perform all of the biological activity and efficacy testing of F05 and new derivatives that are developed and they will also have access to millions of dollars worth of services that are normally available only to pharmaceutical and biotechnology companies.

Hopefully, this will *stimulate* pharmaceutical companies to *license* the most *promising* neurotherapeutic drugs and *invest* in the *clinical trials* needed to bring them to market.

Before F05 can move into clinical trials, its chemical structure must be redesigned to convert it into a safe and effective drug. This process is called chemical optimization and it involves the creation of hundreds of different chemical variations of F05, which must be tested and retested in cell-based systems and animal models to find one with the desired effects. This is what pharmaceutical companies, not academic centers, are designed to do on a large scale. Promising variants that are developed will undergo medicinal chemistry testing, pharmacology and toxicology testing, and Good Manufacturing Practices formulation. This will all be performed by contract research organizations that usually provide these services to the biotechnology and pharmaceutical industries.

Each project in the Neurotherapeutics Network, including the F05 project, is directed by a Lead Development Team (LDT) consisting of academicians, consultants from the pharmaceutical industry, and NIH staff (see Figure 3). Under the direction of the LDT, the F05 project is currently perfecting the biological assay, testing the drug-like properties of F05 and the other three “hits”, and designing and producing F05 derivatives for biological testing. The LDT has set milestones and deliverables, and confers biweekly to ensure that the project keeps on task.

A really important component of this relationship between our academic researchers and the industry service organizations is that NIH has ensured that our investigators will retain all intellectual property rights for any drugs they develop through the Neurotherapeutic Network. This is critical because it provides value to the end product. Drugs that successfully traverse the “valley of death” suddenly become very interesting to big pharmaceutical companies because they have potential to make it through the rigorous FDA approval process. Hopefully, this will stimulate pharmaceutical companies to license the most promising neurotherapeutic drugs and invest in the clinical trials needed to bring them to market.

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**Figure 3.** The operational model for each project within the Neurotherapeutic Network involves a Lead Development Team overseeing multiple drug development components carried out by research service providers.
Did you know that electrical stimulation of a particular area in the brain can trigger the release of chemicals way down in the spinal cord that facilitate walking? Well, Miami Project faculty member Dr. Brian Noga knows this and he has been focusing his research on how to apply this after spinal cord injury.

In the healthy central nervous system (brain and spinal cord) there is a group of nerve cells (neurons) within the middle of the brain that play an important role in signaling the body to start walking or locomotion. These neurons are known as the “midbrain” locomotor region, or MLR. Each of these neurons connect (via a nerve fiber or axon) to other neurons within the brainstem transmitting the “command” for initiating locomotion as well as other neurons which provide powerful neuromodulatory influences on the walking pattern. Together, these neurons form the “descending locomotor pathways”, sending axons down to the thoracolumbar region of the spinal cord that contains the neurons producing walking patterns. How do these pathways excite the spinal locomotor neurons? They do so by releasing specific chemicals, neurotransmitters, which are sensed by the locomotor neurons. Neurotransmitters are chemicals that relay, amplify, and modulate signals between nerve cells. Two of the neurotransmitters released by these pathways are the powerful neuromodulators called Serotonin and Norepinephrine.

After spinal cord injury, the MLR-to-brainstem pathway remains intact and functional, but the brainstem-to-spinal cord pathways get disrupted. However, in most human injuries there are spared nerve fibers that are still connected above and below the injury even though there may be partial or complete paralysis below the injury. Also important is the fact that spinal cord cells below the injury site remain alive. So as long as the spinal cord injury is above the thoracolumbar area, the locomotion circuitry will remain intact. These factors are important and Dr. Noga has figured out how to take advantage of them with an existing treatment called deep brain stimulation. Deep brain stimulation is approved for use in humans by the FDA for many purposes, including the treatment of movement disorders in persons with Parkinson’s disease where medications do not work to restore movement. This is good news for the spinal cord injury field because deep brain stimulation techniques already have a proven record of safety and efficacy. Dr. Noga has been investigating this method in spinal cord injury models by stimulating the MLR and measuring the release of neurotransmitters around the locomotion circuitry within the spinal cord. He has shown that cells activated by MLR stimulation include those important cells that release serotonin and norepinephrine in the spinal cord, which both help to stimulate locomotion. He has also demonstrated that many of the spinal locomotor-activated neurons have receptors which are sensitive to these neurotransmitters, thus providing a basis for their powerful neuromodulatory effects. The ability to enhance the activity of cells (and their release of neurotransmitters) that normally excite locomotor processes by the use of deep brain stimulation opens up the possibility of facilitating walking in persons with partial spinal cord injury. In accordance with this possibility, Dr. Noga has recently been awarded grants from the Christopher and Dana Reeve Foundation, the Craig Neilsen Foundation, and the National Institutes of Health to explore the therapeutic efficacy of deep brain stimulation over a variety of spinal cord injury types.
Dr. Juan Pablo de Rivero Vaccari led 2 studies evaluating different aspects of the innate immune system after experimental spinal cord injury. The first study was published in the journal *Glia* and discovered a novel mechanism involved in activating astrocytes after injury. Activated astrocytes go on to produce inflammatory mediators, which can contribute to causing secondary tissue damage. These findings are important because this mechanism is responsible for activating many different inflammatory events that are deleterious after injury. Thus, current research is focusing on understanding activation of this mechanism in neurons and in identifying inhibitors of this pathway that can be used as an anti-inflammatory treatment after spinal cord injury. The second study was published in the *Journal of Neuroscience* and expands his initial findings on neurons as cells of the innate immune response, which was initially published in the same journal back in 2008. This study identifies P2X4 as a therapeutic target to inhibit inflammation after injury. Therefore, future studies will test pharmacological blockers of P2X4 for their ability to improve function after spinal cord injury due to their anti-inflammatory properties.


Dr. Kevin Park, and colleagues, recently published 2 studies evaluating different aspects of central nervous system axonal regeneration. The first study was published in the preeminent journal *Nature* and demonstrated that deleting two specific genes from neurons at the same time promotes continued and significant long-tract regeneration following injury to the axons. This study also showed that double deletion of these genes works synergistically to regulate activation and expression of several growth-related genes that improve axon regeneration. These findings are important in unlocking the mysteries of long distance regeneration. The second study was published in the journal *Neuron* and focused on the problem of neuronal death triggered by central nervous system axonal injury. They discovered, in their optic nerve injury model, that axon damage stimulates distinct regulation patterns of molecules involved in the unfolded protein response, which contribute to death of the neurons. This is important because it could lead to a potential neuroprotective strategies to prevent axonal injury associated cell death.


If you are looking for information or have questions about spinal cord injury (SCI) and traumatic brain injury (TBI) research being conducted at The Miami Project and for updates describing continuing progress toward the goal of developing treatments for SCI and TBI, then the buck stops with the Education department. Headed up by Kim Anderson-Erisman, Ph.D., the little department that helps thousands of people each year is composed of Maria Chagoyen, Danielle Cilien, and Letitia Fisher. Aside from answering phone calls, and emails and giving numerous tours and lecturers about our research, the Education department also answers many broader requests for information regarding injury prevention, clinical care referral, resources for living with paralysis, and advice about unproven therapies around the world. The graph shows the total number of people reached each month during outreach activities.
On February 26, 2011 the Education department hosted the 1st Miami Project Community Open House. We opened up our doors to the public for an afternoon packed full of information. The Miami Project Scientific Director, Dr. Dietrich, gave a detailed overview of discoveries that were accomplished by faculty members throughout the years. This was followed by an open discussion session stimulated by the audience regarding clinical trials, risk versus benefit, and how to tell the difference between proven and unproven therapies. The final component of the day was behind-the-scenes tours of five laboratories, including a cell culture lab, cell transplantation lab, regeneration lab, human exercise lab, and human motor training lab. There were over 100 community members in attendance and the event was a great success! The 2nd Annual Community Open House was February 18, 2012 and was an even greater success! The focus this year was on chronic spinal cord injury and talks covered scar tissue, cell replacement, regeneration, muscle spasticity, neuropathic pain, and Brain-Machine Interface technology.

The education department continued its “Neurotrauma Lecture Series” curriculum for high school students in Virginia. As part of the curriculum, several faculty members gave lectures that were transmitted via telecommunication to students at two campuses in Virginia. The education department also participated in the 2nd Annual Brain Fair as well as Take Your Child to Work Day, hosted Disability Mentoring Day, had an educational booth at the National Neurotrauma Society annual meeting, and gave invited lectures to multiple SCI consumer groups. If you have questions, don’t hesitate to email us at mpinfo@med.miami.edu or call us at 305-243-7108.

Clockwise from top right: Dr. Dietrich speaks to guest at Open House 2011; Brain Fair 2011 activities; Open House 2012 audience listening to research update; Take your child to work day; Open House 2012 participants.
A major role of The Miami Project is to provide education and training for the next generation of neuroscientists. This aspect of The Miami Project’s mission is equally as important as conducting the research that will lead to a cure. Our long-term educational goal is to increase the number of scientists and laboratories working on paralysis research and central nervous system disorders around the world. Students and young scientists beginning their careers gain skills from The Miami Project’s state-of-the-art comprehensive research and academic environment. During 2011, the faculty members of The Miami Project were actively training almost 200 students of various levels (high school, undergraduate, graduate, medical) in their laboratories.

The Miami Project Summer Student Research Fellowship is offered to a handful of stellar students each summer eager to work in the laboratory of a Miami Project faculty member. In 2011, the following students were awarded this fellowship:

- Christina Baker – Sanchez lab
- Jesse Bryant – Nash lab
- David Douriez – Hentall lab
- Jessica Johnson – Widerström-Noga lab
- Jose Ramos – Lee lab
- Luis Montoya – Thomas lab
- Yoelkys Morales – Sagen lab
- Anthony Martin – Brambilla lab
The University of Miami Citizens Board also funded a summer research fellowship for one outstanding University of Miami undergraduate student working in a Miami Project laboratory. In 2011, that student was:

Cecilia Perez – Sagen lab

Drs. John Bixby, Dalton Dietrich, and Vance Lemmon received supplemental funding from the American Recovery and Reinvestment Act to fund 9 summer students during 2011, and those students were:

Marisa Adhikusuma - Tsoulfas lab collaboration w/ Bixby lab
Daniel Cabrera - Lemmon-Bixby lab
“JJ” Joaquin Jimenez - Lemmon-Bixby lab
Michelle LeBlanc - Pearse lab collaboration w/ Dietrich lab
Emily Packard - Bramlett lab collaboration w/ Dietrich lab
Lindsey Salay - Lemmon-Bixby lab
Christina Sedaghat - Atkins & Vaccari labs collaboration w/ Dietrich lab
Alex Torres - Lemmon-Bixby lab
Alexandria Wick - Dietrich lab

Two particularly motivated students volunteered full-time for the summer working in Miami Project laboratories, and they were:

Giselle Fontela – Thomas lab
Natalie Torrente – Park lab

All 20 of these exceptional students wrote an abstract about their specific research project and presented a poster at the 1st Miami Project Summer Student Research Session on August 5, 2011.

Summer Student Research Projects:


- Brain-Computer Interfaces, Functional Electrical Stimulation and Their Roles in Neurological Rehabilitation for Paralysis Patients. Christina Baker, Scott Roset, Zoe Englander, Edelle Field-Fote, Justin C. Sanchez.


- β-Amyloid Precursor Protein Expression Following Polytrauma. Emily Packard, Helen Bramlett.


- Impact of Prescription Pain Medication on Dorsal Column and Spinothalamic-Mediated Functions in Persons with...


Meningeal Fibroblast Invasion Following Penetrative Spinal Cord Injury. Jose Ramos, Cynthia Soderblom, Jae Lee.


Rab27b May Play a Role in the Anterograde Transport of Trk and p75NTR Receptors in Non-Neuronal and Neuronal Cells. Marisa Adhikusuma, Pantelis Tsoulfas.


Examination of Signaling Pathways Involved in KRAS Activation-Induced Optic Nerve Regeneration. Natalie Torrente, Kevin Park.

Innate Immune Response Proteins as Biomarkers of Presymptomatic Alzheimer’s Disease. Christina Sedaghat, Juan Pablo de Rivero Vaccari.

Developing the Natural Analgesic Conopeptide μ-SIIIA as a Candidate for Neuropathic Pain Gene Therapy to Repress Na_1.8 Mediated Neuropathic Pain. Yoelkys Morales, Krystyna Simon, Shyam Gajavelli, Stanislava Jergova, Blake H Priddy, Jacqueline Sagen.

Taking it to the next level

One Miami Project Summer Student Research Fellow, University of Miami senior Jesse Bryant, continued on with his research project into the Fall semester. He submitted an abstract on his research to the Biomedical Engineering Society and it was accepted for poster presentation. Congratulations! The title of his poster was “Sensitivity of wheelchair propulsion kinetics to increased physiological demands” and he presented it at the Biomedical Engineering Society 2011 Annual Meeting in Hartford, CT.

Bryant measured the response of several propulsion variables (force, torque, power, etc.) to the increased physical demands of increased speed, increased surface resistance, and increased wheelchair weight. The results showed that the widely studied propulsion variables (stroke angle, max force, and cadence) are not the most sensitive to changes in the physiological demands of wheelchair propulsion. The less commonly studied variables such as power, linear impulse, and torque actually showed the highest sensitivities to increases in speed, resistance, and mass, respectively, and thus, the variables chosen for future studies should be those best suited for the conditions being tested.

Jesse will be graduating from UM in May 2012 and is currently applying to graduate schools so he can begin pursuing a Ph.D. in biomedical engineering.
Each year, Miami Project scientists seek funding for their research by submitting proposals to the National Institutes of Health, the premier scientific body in the United States, as well as other funding agencies and foundations.

Their scientific peers rate the merits of these proposed experiments in a highly competitive process and only the best are funded. The agencies and organizations listed below supported the Principal Investigator(s) and the scientific project(s) indicated during 2011.

Dr. Stanislava Jergova (P.I. – Post-doctoral Fellowship)
- Recombinant Stem Cell Therapy for Spinal Cord Injury Pain

Dr. Robert Keane (P.I.)
- Inflammasome Regulation Following Spinal Cord Injury

Dr. Mark S. Nash (P.I.)
- Effects of Salsalate Monotherapy on Prandial-Induced Vascular Inflammation in Overweight Persons with SCI
- Effects of exercise on Prandial Lipemia and fat oxidation after Tetraplegia
- A Model Community/Home-based Exercise Program for SCI

Dr. Brian Noga (P.I.)
Dr. Ian Hentall (Co-I.)
- Acute Facilitation of Walking After SCI Using Deep Brain Stimulation

Dr. Jacqueline Sagen (P.I.)
- Utilizing Designer Genes to Alleviate Chronic SCI Pain

Dr. Eva Widerström-Noga (P.I.)
Dr. Alberto Martinez-Arizala (Co-I.)
- Utility of quantitative sensory testing in SCI-related pain

Department of Defense (DOD) Defense Advanced Research Projects Agency (DARPA)
Dr. Justin C. Sanchez (P.I.)
- Tissue, Electrical, and Material Responses in Electrode Failure
-Creating the Synthetic Brain Through Hybrid Computational and Biological Systems: Repairing and Replacing Neural Networks

**Department of Defense (DOD) Spinal Cord Injury Research Program (SCIRP) of the Office of the Congressionally Directed Medical Research Programs**

Dr. Mark S. Nash (P.I.)
-Obesity/Overweight in Persons with Early and Chronic SCI: A Randomized, Multicenter, Controlled Lifestyle Intervention

Dr. Damien Pearse (P.I.)
Dr. Mary Bartlett Bunge (Partner P.I.)
Dr. James Guest (Partner P.I.)
Dr. Dalton Dietrich (Co-I.)
-Schwann Cell (SC) Implantation for SCI Repair: Optimization of Dosing, Long-Term Cell Persistence, and the Evaluation of Toxicity and Tumorigenicity

Dr. Damien Pearse (P.I.)
Dr. Howard Levene (Partner P.I.)
-Translation of Novel PDE4 Inhibitors for the Treatment of Acute Spinal Cord Injury

Dr. Damien Pearse (P.I.)
-Site-directed Nanotherapeutics to Abrogate Relapsing/Remitting Multiple Sclerosis and Promote Remyelination Repair

**Department of Defense (DOD) Psychological Health and Traumatic Brain Injury Program of the Office of Congressionally Directed Medical Research Programs**

Dr. Helen Bramlett (P.I.)
-Pathophysiology of Polytrauma and Novel Treatment Strategies

Dr. M. Ross Bullock (P.I.)
-Laboratory Studies to Evaluate Perfluorocarbon in Models of Traumatic Brain Injury

Dr. Jed Hartings (P.I.)
Dr. M. Ross Bullock (Co-I.)
-Spreading Depressions as Secondary Insults after Traumatic Injury to the Human Brain

Dr. Frank Tortella (P.I.)
Dr. M. Ross Bullock (Co-I.)
-Treatment of Traumatic Brain Injury using the Neuroprotectant NNZ2566

**Department of Defense (DOD) United States Army Medical Research Acquisition Activity**

Dr. W. Dalton Dietrich (P.I.)
Dr. Helen Bramlett (Co-I.)
-Operation Brain Trauma Therapy

E. Matilda Ziegler Foundation for the Blind
Dr. Kevin K. Park (P.I.)
-Novel Combinatorial Approaches to Enhance Retinal Ganglion Cell Survival and Axon Regeneration after Optic Nerve Injury

Fa Bene Foundation
Dr. Damien Pearse (P.I.)
-Cellular Therapies for Chronic Spinal Cord Injury Repair

**Florida Department of Health Bureau of Emergency Medical Services**

Dr. Gillian Hotz (P.I.)
-WalkSafe Florida State Implementation

**Florida Department of Transportation**

Dr. Gillian Hotz (P.I.)
-Safe Route to School-WalkSafe County Program
-WalkSafe Miami-Dade
-WalkSafe Tool Kit
-BikeSafe Program

**International Spinal Research Trust**

Dr. James Guest (Center P.I.)
-Comparison of Schwann Cells and Skin-derived Precursor Cells for Repair of Demyelination in the Primate Corticospinal Tract

**Ironwood Pharmaceuticals, Inc.**

Dr. Jacqueline Sagen (P.I.)
-Testing Analgesic Effects of FAAH Inhibitors in a Rat Spinal Cord Injury Pain Model

**Medtronic Spinal and Biologics**

Dr. Allan Levi (P.I.)
Dr. Barth Green (Co-I.)
Dr. Steven Vanni (Co-I.)
-Pivotal IDE Study of the Bryan Cervical Disc Prosthesis in the Treatment of Degenerative Disc Disease

**Miami-Dade County Health Department via Center of Disease Control**

Dr. Gillian Hotz (P.I.)
-Community Putting Prevention to Work – Safe Routes to School

**National Center for Medical Rehabilitation Research in the National Institute of Child Health and Human Development and the National Institute of Neurological Disorders and Stroke**

Dr. Christine Thomas (P.I.)
-Verifying Automatic Analysis of Muscle Spasms in Large Datasets
**National Heart, Lung, and Blood Institute**
Dr. David Fuller (P.I.)
Dr. Justin C. Sanchez (Co-I.)
-Training Novel Host-Graft Circuits to Enhance Spinal Cord Repair
Dr. Stephan Schürer (P.I.)
Dr. Vance Lemmon (Co-I.)
-LINCS Information FramEwork (LIFE) to integrate and Analyze Diverse Data Sets

**National Human Genome Research Institute**
Dr. Vance Lemmon (P.I.)
-Bioassay Ontology and Software Tools to Integrate and Analyze Diverse Data Sets

**National Institute of Child Health & Human Development**
Dr. Edelle Field-Fote (P.I.)
Dr. Diana Cardenas (Co-I)
Dr. Mark S. Nash (Co-I.)
-Improving Hand and Arm Function in Individuals with SCI
Dr. Vance Lemmon (P.I.)
Dr. John Bixby (Co-I.)
- Novel Gene Targets for CNS Axonal Regeneration

**National Institute of Disability & Rehabilitation Research**
Dr. Diana Cardenas (P.I.)
Dr. Mark S. Nash (Co-I.)
-Spinal Cord Injury Model Systems
Dr. Mark S. Nash (P.I.)
-Exercise Interventions for Spinal Cord Injured Adults with Obesity-related Secondary Disorders
-Sleep Disordered Breathing in Persons with Chronic Tetraplegia: Characterization and Intervention
Dr. Suzanne Groah (P.I)
Dr. Mark S. Nash (Co-I.)
-Rehabilitation Research and Training Center on Secondary Conditions in SCI

**National Institute of Neurological Disorders & Stroke**
Dr. Coleen Atkins (P.I.)
-Rehabilitation Strategies for Memory Dysfunction after Traumatic Brain Injury
Dr. Mary Bartlett Bunge (P.I.)
Dr. Paula Monje (Co-I.)
Dr. Patrick Wood (Co-I.)
-Cytological Studies of Developing and Mature Neurons
Dr. John Bethea (P.I.)
Dr. Roberta Brambilla (Co-I.)
-The Role of Astroglial-NF-kB in SCI
-Astrocytes Play a Critical Role in the Pathology of EAE
Dr. John Bixby (P.I.)
Dr. Vance Lemmon (Co-I.)
-Novel Compounds that Overcome Glial Inhibition of Axonal Regeneration
Dr. John Bixby (P.I.)
Dr. Vance Lemmon (Co-P.I.)
Dr. Jeff Goldberg (Co-P.I.)
-Triazine-based Compounds to Promote Regeneration in Optic Neuropathies
Dr. John Bixby (P.I.)
-Predoctoral Training in the Neurosciences

Dr. Jesse Brodkin (P.I.)
Dr. Jacqueline Sagen (Sub-contractor for Small Business Innovation Research award)
-Using a Multi-touch Screen to Automate Pain Assessment
Dr. Akira Chiba (P.I.)
Dr. Vance Lemmon (Co-I.)
-In situ Protein-Protein Interaction Networks (PIN) of Neurons

Dr. Ramon Diaz-Arastia (P.I.)
Dr. M. Ross Bullock (Co-I.)
-Phase II, Randomized Controlled Trial of Brain Tissue Oxygen Monitoring
Dr. W. Dalton Dietrich (P.I.)
Dr. Helen Bramlett (Co-I.)
-Mechanisms of Recovery Following Traumatic Brain Injury
-Efficacy of Necrostatins on Post-traumatic Epilepsy (via American Recovery and Reinvestment Act)
Dr. W. Dalton Dietrich (P.I.)
Dr. Helen Bramlett (Co-I.)
-Cellular Transplantation Strategies Following Traumatic Brain Injury
-The Importance of Temperature on Inflammation after TBI

Dr. W. Dalton Dietrich (P.I.)
Dr. Helen Bramlett (Co-I.)
Dr. Coleen Atkins (Co-I.)
- Cyclic Nucleotide Regulation in Traumatic Brain Injury
Dr. John Harris (P.I.)
Dr. Justin C. Sanchez (Co-I.)
-An Ultra-Low Power Wireless Neural Recording Implant Based on a Novel Pulse Representation
Dr. Robert Keane (P.I.)
- Inflammusome Regulation After Spinal Cord Injury

Dr. Daniel Liebl (P.I.)
- Ephrins Regulate Stem Cell Proliferation following Traumatic Brain Injury
- Regulation of Synaptic Formation & Efficacy Following Traumatic Brain Injury

Dr. Allan Levi (P.I.)
- Transplantation of Autologous Schwann Cells for the Repair of Segmental Peripheral Nerve Defects

Dr. Andrew Maudsley (P.I.)
Dr. Eva Widerström-Noga (Co-I.)
- Volumetric MRSI Evaluation of Traumatic Brain Injury

Dr. Brian Noga (P.I.)
Dr. Ian Hentall (Co-I.)
- Control of Spinal Locomotor Activity by Monoamines

Dr. Michael Norenberg (P.I.)
Dr. John Bethea (Co-I.)
- NF-kappaB in Astrocyte Swelling/Brain Edema Associated with Acute Liver Failure

Dr. Damien Pearse (P.I.)
Dr. Mary Bartlett Bunge (Co-I.)
Dr. Brian Noga (Co-I.)
Dr. Patrick Wood (Co-I.)
- Axon Regeneration: Synergistic Actions of the MAPK and Cyclic AMP Pathways

Dr. Jacqueline Sagen (P.I.)
- Potent Analgesic Conopeptides for Treatment of Chronic Spinal Cord Injury Pain

Dr. Justin C. Sanchez (P.I.)
- Neural Correlates of Tourette Syndrome

Dr. Christine Thomas (P.I.)
- Muscle Function in Human Cervical Spinal Cord Injury
- Rescue of Denervated Muscle

Dr. Grace Zhai (P.I.)
Dr. Pantelis Tsoulfas (Co-I.)
- Mechanisms of Neuronal Maintenance and Protection

Paralyzed Veterans of America Education Foundation
Dr. Rachel Cowan (P.I. – Post-doctoral Fellowship)
Dr. Mark S. Nash (Sponsor)
- From Research into the Clinic: The Manual Wheelchair Propulsion Database

Ralph C. Wilson Jr. Medical Research Foundation
Dr. Jacqueline Sagen (P.I.)
- Cell and Gene Therapy Strategies for Delivery of Novel Marine Snail Peptides in Chronic Spinal Cord Injury Pain

State of Florida James and Esther King Biomedical Research Program
Dr. John Bixby (P.I.)
Dr. Murray Blackmore (Co-I.)
Dr. Vance Lemmon (Co-I.)
- Combination Therapy in SCI: Proof of Concept for New Compounds and Candidate Genes

The Karl Kirchgassner Foundation
Dr. Kevin K. Park (P.I.)
- Developing PTEN shRNA-mediated Combinatorial Approaches to Improve Optic Nerve Regeneration

University of Miami Interdisciplinary Research Development Initiative
Dr. Ian Hentall (P.I.)
- Whole Body Pharmacological Profiling and Imaging of FAST Probes

University of Miami Stanley J. Glaser Foundation
Dr. Coleen Atkins (P.I.)
- Rehabilitation Strategies for Cognitive Disabilities after Traumatic Brain Injury

Veterans Administration Biological Laboratory Research and Development
Dr. Helen Bramlett (P.I.)
- Novel Treatment Strategies for Targeting Post-traumatic Epilepsy

Veterans Administration Rehabilitation Research and Development
Dr. Robert Jackson (P.I.)
Dr. Diana Cardenas (Co-I.)
- Rehabilitation of IPF patients: Effects of Exercise and Oxidant Stress

Wings for Life Foundation
Dr. Damien Pearse (P.I.)
- Transcriptional Activation of Endogenous and Exogenous Neural Precursor cells for SCI Repair
Neuroprosthetics - The Merging of Technology and Biology to Enhance Function
When you mix neuroscience and biomedical engineering with the goal of developing devices targeting the restoration of function(s) lost as a result of disease or injury to the nervous system, you get the research field referred to as neuroprosthetics. This “young” field has exploded in the last decade and produced life-changing neural devices such as the cochlear implant to restore hearing, deep brain stimulators to reduce muscle tremors in Parkinson’s disease, and the spinal cord stimulator targeting neuropathic pain. What if they could create a device that was able to read your brain and trigger the appropriate movement? Science fiction you say? No, it’s the reality of the future – the very near future.

To use the **brain** to **restore** movement to **paralyzed limbs**.

You may have heard about monkeys being able to control a cursor on a computer screen with their thoughts. This has already been translated to humans with a system called BrainGate and BrainGate2. Already in clinical trials for people with high cervical injury, advanced Lou Gehrig’s disease, and locked-in syndrome, the BrainGate neuroprosthetics enable humans, who have no arm movement, to use their thoughts to control a computer cursor. This is an amazing feat and it is restoring the control of communication to individuals who are trapped inside their body due to severe neurologic disease. But wouldn’t it be great to take this technology one step further? To use the brain to restore movement to paralyzed limbs. The Miami Project is leaping into this field thanks to the excitement, intelligence, and drive of a biomedical engineer, Dr. Justin Sanchez.

Dr. Sanchez was recruited to the University of Miami department of Biomedical Engineering in 2010 and shortly thereafter was invited to become a faculty member of The Miami Project. Dr. Sanchez’s research specializes in the Brain Machine Interface (BMI) arena of neuroprosthetics and addresses 4 key issues:

1. Can we get signals out of the brain?
2. Can we decode those signals appropriately?
3. Can we build a fully implantable, i.e. invisible, BMI system?
4. Can we use that BMI system for rehabilitation and to restore functions that improve independence?

**Can we get signals out of the brain?**

The first step in any BMI system is to access signals from the brain. Changes in intent and behavior are produced by signals both on the surface of the brain as well as deep inside the brain. These signals could be related to motor, sensory, or goal information. The ability to sample activity from the brain depends heavily on electrodes.
since the nervous system communicates via electricity. An electrode is a medium for conducting an electrical current from the body to some kind of physiologic monitoring equipment. Electrodes come in many sizes and shapes that are tailored to accessing the information of interest. Microelectrodes (diameter of a human hair) can access the activity of single neurons. Typically, hundreds of these electrodes can access hundreds of neurons. These are the only electrodes that can access activity in the upper layers of the brain (cortex) and deep brain structures. In order to access the activity of thousands of neuronal assemblies, an electrocorticogram (ECoG) grid of electrodes is used, which can only sit on the surface of the brain. Finally, the electroencephalogram (EEG) has electrodes that sit on the scalp rather than on the surface of the brain and these can access very large scale activity in the brain related to behavior and cognition.

Can we decode those signals appropriately?
Once signals are out of the brain the next step is to decode them (i.e. interpret a user’s intent). The process of decoding in neuroprosthetics refers to the transformation of the signals recorded with electrodes into meaningful behavior for the user. If you’d like, you can think of decoding as the mysterious black box that magically takes thought and converts it to action. However, Dr. Sanchez makes it his business to remove the mystery from decoding. The process of decoding is very difficult because the activity of thousands or millions of neurons needs to be mapped to unique behaviors. These behaviors and the brain can also change over time or can be affected by experience and learning. Decoding brain signals falls into the domain of computational neuroscience and signal processing. Typically, engineering and signal processing do not contend well with scenarios that change over time (as is often encountered in biological systems) and much research is ongoing to develop innovative new approaches. Simple decoding approaches just match a change in a brain state to one behavior. More sophisticated decoders have the ability to adapt in synergy with their users and can learn through experience with their users. The great news is that advances in understanding the adaptation of
The components necessary for the ideal Brain-Machine Interface targeting functional recovery for SCI.

the brain and decoder is opening new perspectives in how the nervous system works and is influencing new medical device design.

Can we build a fully implantable, i.e. invisible, system?
So we can record thoughts and decode them into action, but who wants to have electrodes and wires sticking out of their head drawing more attention to the devices you use rather than who you are? Cosmesis, how it makes you look, is a significant issue for any therapy and plays a critical role when developing devices for persons living with spinal cord injury. Ultimately, the best neuroprosthesis will allow users to appear as any other individual without anyone being able to tell the difference. This is enabled with implantable electronics; information from the brain will be sampled, decoded, and control delivered to the body in a completely internalized manner. The design of fully implantable neuroprosthetics is difficult because of power, processing, and bandwidth issues. The activity of millions of neurons can create gigabytes of activity in very short periods of time. The ability to process all of that activity on the timescale of normal behavior requires fast processors. Ultimately, these implantable devices need to be powered for the lifetime of the users. The development of technology that addresses all of these requirements is on the cutting edge of what is currently possible with even the best electronics, but without a doubt all of those hurdles will be overcome in the near future. In fact, Dr. Sanchez and Dr. Jonathan Jagid (functional neurosurgery) are working to bring a leading edge technology that overcomes these issues to humans living with spinal cord injury. In a new effort led by Dr. Sanchez, a multidisciplinary team of Miami Project faculty members are seeking federal grant funding to perform feasibility testing of a brand new fully implantable BMI for individuals with motor complete, C5 or C6, chronic spinal cord injury. Expect to see more news about these developments later in 2012.

Can we use that system for rehabilitation and to restore functions that improve independence?
While the convergence of technology and neuroscience is providing the enablers for neuroprosthetics, the field will have limited impact if it does not effectively empower the user. The melding of rehabilitation with neuroprosthetics can provide the right combination of technology and function to enhance one’s ability to perform activities of daily living with more independence. Reconnecting the brain with the body is the key to unlocking the expression of intent and communication with the world. Rehabilitating those new connections and strengthening them through practice could offer new, more effective functions that increase independence and improve overall quality of life. We ultimately envision a battery of combined therapies (BMI with rehabilitation, cell therapy, regeneration drugs, assistive devices such as exoskeletons, etc.) to create customized treatments that are tailored to each individual’s needs.

These are exciting times indeed for people living with spinal cord injury! Technology is advancing as is our understanding of the nervous system after injury and how it responds to various inputs. The broad utilization of “neurotechnology” invokes the vision that neural interfaces, cell therapies, and neurorehabilitation can all be used together in a powerful way to repair and restore function to the damaged nervous system. By embracing technology we have found a new window through which we can learn how to enhance the reparative effects of neurotherapeutics and elicit the greatest amount of functional recovery for all the different variations of paralysis.
W. DALTON DIETRICH, PH.D.
Scientific Director
Kinetic Concepts Distinguished Chair in Neurosurgery
Senior Associate Dean for Discovery Science
Professor, Departments of Neurological Surgery, Neurology, and Cell Biology & Anatomy
Neuroprotection and Improved Recovery of Function following CNS Trauma

My research interest is the pathobiology and treatment of CNS injury in both the acute and chronic setting. Animal models of cerebral ischemia, and brain and spinal cord trauma are utilized to investigate the mechanisms of tissue injury. The ultimate goal is to target specific injury processes for pharmacological intervention, including the addition of growth factors, to promote circuit plasticity, regeneration and recovery of function.

BARTH A. GREEN, M.D., F.A.C.S.
Co-Founder
Professor and Chairman, Department of Neurological Surgery
Translational Interventions

Over the recent years my research efforts have mainly involved taking the cutting edge basic neuroscience work product and data created by our Miami Project team from the bench to our UM affiliated clinics and hospitals. A good example of such translational research efforts has included the use of modest hypothermia for neuroprotection both in cases of acute spinal cord injury and for use in the operating room for patients undergoing high risk spinal cord surgery. I am also privileged to be able to collaborate with The Miami Project cellular transplantation programs and have been working on projects involving adult mesenchymal stem cells as well as being part of the major effort transforming our successful Schwann cell laboratory model into human clinical trials. Another area of clinical interest and research includes the diagnosis and treatment of Chiari I malformation with and without syringomyelia. Each of these projects involve collaborations with Miami Project basic and clinical researchers as well as the faculty from the Department of Neurological Surgery and several other collaborating departments and Centers of Excellence at the University of Miami Miller School of Medicine.
MARY BARTLETT BUNGE, PH.D.
Christine E. Lynn Distinguished Professor in Neuroscience
Professor, Departments of Cell Biology, Neurological Surgery, and Neurology
Development of Combination Strategies to Repair the Injured Spinal Cord

The goal in my laboratory is to foster regeneration of axons across and beyond a spinal cord injury (SCI). To improve regeneration of axons, we are investigating reducing the accumulation of proteoglycans (molecules that inhibit axonal growth), improving survival of transplanted Schwann cells (SCs), and genetically engineering SCs before transplantation to improve their neurotrophic factor-secreting capability or neurons to enhance their ability to regenerate axons after injury. We pay particular attention to the interface between the SC implant and the host spinal cord.

JOHN R. BETHEA, PH.D.
Professor, Departments of Microbiology & Immunology and Neurological Surgery
Immunological Consequences of SCI and The Development of Neuroprotective Strategies

In my laboratory we are studying spinal cord injury (SCI) and diseases of the nervous system such as Multiple Sclerosis (MS) to try to understand the cellular and molecular mechanisms that contribute to astrogliosis and secondary neuronal cell death. To this end, my laboratory has two main research objectives. First, we are studying the neuro-inflammasory response that occurs following SCI and secondly, we are developing novel therapies for SCI and diseases of the central nervous system.

JOHN BIXBY, PH.D.
Professor, Departments of Molecular & Cellular Pharmacology and Neurological Surgery
Senior Associate Dean for Graduate & Postdoctoral Studies

VANCE LEMMON, PH.D.
Walter G. Ross Distinguished Chair in Developmental Neuroscience
Professor, Department of Neurological Surgery
High Content Screening and Functional Genomics of the Nervous System

Our laboratory has developed methods to test thousands of genes or chemicals in hundreds of thousands of neurons each week and obtain quantitative information about cell morphology and gene expression. This “high throughput” capability allows us to tackle questions about development and regeneration using Systems Biology approaches. The Lemmon-Bixby lab has several ongoing projects related to axon regeneration. One project is based on the observation that young CNS neurons have a greater regenerative capacity than old CNS neurons (collaboration with Dr. Jeff Goldberg). We have identified a family of developmentally regulated transcription factors (proteins that bind DNA and turn on or off scores of genes) that is very important in axon regeneration. Another project is to test the roles of known signaling proteins called kinases. In this screen we have tested hundreds of kinases by overexpression and have also tested a few hundred kinase inhibitors, many of which strongly promote neurite growth in vitro. The data from this screen are allowing us to begin to build models of neuronal signaling networks underlying axon regeneration. A third project is to screen a novel chemical compound library to identify compounds that can overcome the regeneration-inhibitory effects of the injured CNS (collaboration with Dr. Goldberg and Prof. Young-Tae Chang, National University of Singapore). In 2011, this project was selected to be part of the NIH Neuroscience Blueprint Initiative to develop drugs to promote axonal regeneration.
M. ROSS BULLOCK, M.D., PH.D.  
Professor, Department of Neurological Surgery  
Director, Clinical Neurotrauma

Preclinical Mechanistic and Neuroprotection Research in Traumatic Brain Injury and Clinical trials, and Neuromonitoring Techniques in the Injured Brain

We have recently obtained two year funding from the Department of Defense to evaluate the neuroprotective effect of Perfluorocarbons in four rodent models of traumatic brain injury (penetrating brain injury, closed traumatic brain injury with secondary hypoxia, tissue culture with stretch injury, and mechanistic and safety studies). These oxygen carriers have shown benefit in previous studies involving fluid percussion injury and subdural hematoma models. We are also evaluating hypothermia neuroprotection, in humans and animals, using novel biomarkers as surrogate indicators of possible benefit.

DIANA CARDENAS, M.D., M.H.A.

Professor and Chair, Department of Rehabilitation Medicine  
Chief of Service & Medical Director, Department of Rehabilitation Medicine

Pain Interventions and Prevention of Urinary Tract Infections

The goals of my research are to help find therapeutic interventions that improve recovery, reduce secondary conditions, and create a better life for persons with SCI and other conditions that impair physical or cognitive function. Currently, my research focus is in the areas of neuropathic pain and neurogenic bladder management.

EDELLE C. FIELD-FOTE, PH.D., P.T.

Professor, Departments of Physical Therapy and Neurological Surgery

Motor Restoration after Spinal Cord Injury

The studies in the Neuromotor Rehabilitation Research Laboratory cross the boundaries of basic neurophysiology of the brain and spinal cord and applied neurophysiology related to neuroplasticity and motor learning. We want to understand which interventions best promote recovery of function. Some of the rehabilitation studies focus on recovery of hand and arm function, while others are aimed at walking function.

ROBERT W. KEANE, PH.D.

Professor, Department of Physiology & Biophysics

Regulation of Innate Immunity after CNS Trauma

Innate immunity is the first line of defense against pathogens and host-derived signals of cellular stress. My research focuses on investigating mechanisms that direct normal innate immunity and its dysregulation in central nervous system injury and disease, including (1) agonists and activation mechanisms of inflammasomes, (2) regulatory mechanisms that potentiate or limit inflammasome activation after injury, and (3) emerging links between innate immunity and Alzheimer’s disease.
ALLAN D. LEVI, M.D., PH.D., F.A.C.S.
Professor, Departments of Neurological Surgery, Orthopedics, and Rehabilitation Medicine
Chief of Neurospine Service, Jackson Memorial Hospital/Chief of Neurosurgery, University of Miami Hospital
Cellular Transplantation Strategies after Spinal Cord Injury
My research interests have focused on developing cellular transplantation strategies to repair injuries within both the human central and peripheral nervous system. My current interests involve (1) further studies on the human Schwann cells extensively expanded in cell culture to determine whether the functional capacity of these cells with respect to myelination and regeneration are retained when compared to primary Schwann cell cultures and (2) the development and characterization of a model of a peripheral nerve injury with a lengthy gap in the rat so that we can further characterize the influence of transplanted Schwann cell grafts within a collagen based channel on peripheral nerve regeneration and pain.

MARK S. NASH, PH.D., F.A.C.S.M.
Professor, Departments of Neurological Surgery, Rehabilitation Medicine, and Kinesiology & Sports Sciences
Physiological Assessment of Secondary Complications following SCI: Electrical Stimulation, Cardiometabolic and Vascular Physiology, Cardiovascular Pathology, and Exercise and Dietary Biochemistry
One of the enduring goals of The Miami Project has been to test and implement strategies that optimize health of persons with SCI. A significant target for this strategy has focused on physical activity to lessen secondary risks of SCI associated with physical deconditioning. We also examine complementary themes to validate exercise prescription after SCI, identify optimal dietary composition, and use of prescription and non-prescription agents that reduce hazards of fasting and postprandial lipid disorders, dysglycemia, and vascular inflammatory stress.

JACQUELINE SAGEN, PH.D., M.B.A.
Professor, Department of Neurological Surgery
Cellular Implants for the Alleviation of Chronic Pain and CNS Injury
Our laboratory continues to explore novel and more effective strategies in the therapeutic management of chronic debilitating pain. Our recent research is focused on (1) identification of more effective analgesic agents and combinations for alleviating pain using SCI models and (2) development of emerging therapeutic interventions, including cell transplantation and gene therapy, which have the potential to provide long-term alleviation in people with intractable pain, overcoming the need for repeated pharmacologic administration.

CHRISTINE K. THOMAS, PH.D.
Professor, Department of Neurological Surgery
Neuromuscular Weakness, Fatigue, Spasms, and Regeneration
Our laboratory is currently asking two main questions regarding SCI. First, in studies on people with SCI, we want to understand how well involuntary contractions of paralyzed muscles (spasms) are managed by exercise or by taking baclofen, a drug that is commonly used to control spasticity. Second, in our animal studies, we are exploring how to replace neurons that die because of SCI. Neuron death is common at the injury site and results in severe muscle weakness.
PATRICK M. WOOD, PH.D.
Research Professor, Department of Neurological Surgery
Changes in the Molecular and Biological Properties of Human Schwann Cells
Schwann cells have shown promise in animal studies in promoting recovery from SCI. We have developed protocols that allow the generation, from a small biopsy of human peripheral nerve, of large numbers of a person’s own Schwann cells that can be transplanted back into their injured spinal cord. Efficient growth of human Schwann cells in culture requires the addition of recombinant neuregulin and the cAMP enhancer forskolin. To better understand the effects of these reagents on Schwann cells, we are performing basic research to determine the mechanisms by which neuregulin and cAMP enhancers promote interaction between axons and Schwann cells, including axon-induced proliferation and the formation of myelin sheaths.

HELEN M. BRAMLETT, PH.D.
Associate Professor, Department of Neurological Surgery
The Pathophysiology and Treatment of CNS Injury
The focus of my neurotrauma laboratory is to investigate both acute and long-term consequences of brain and spinal cord trauma. My current research interests are on the pathophysiology of traumatic brain and spinal cord injury with an emphasis on the pathogenesis of progressive white matter damage as well as the benefits of therapeutic hypothermia. My laboratory is also investigating mechanistic events leading to the development of posttraumatic epilepsy. Additionally, my current work is also focusing on complex traumatic brain injury models that mimic polytrauma as this type of injury has become more prevalent in combat areas.

JAMES D. GUEST, M.D., PH.D., F.A.C.S., F.R.C.S.(C)
Associate Professor, Department of Neurological Surgery
Cellular and Molecular Strategies to Achieve Long Tract Regeneration in the Spinal Cord
The lab is engaged in translational research designed to develop useful models to test key questions of efficacy and safety in large animal models. The emphasis is on conducting testing of therapeutics to emulate human application as fully as possible; we use histological, behavioral, electrophysiologic, MRI, and ultrasound techniques. We design devices to deliver cells and therapeutics in a minimally injurious manner. Currently, we transplant Schwann cells and skin-derived precursor cells to repair tracts of the injured spinal cord. Other areas of research include studies of human post-mortem spinal cord tissue, intra-operative human spinal cord conduction studies, research design for human clinical trials, and active participation in human clinical trials.

DANIEL J. LIEBL, PH.D.
Associate Professor, Department of Neurological Surgery
Director, Neuroscience Graduate Program
Function of Growth and Guidance Molecules in the Developing and Regenerating Nervous Systems
The goal of my laboratory is to identify the mechanisms that lead to CNS pathophysiology and its regenerative potential. We focus on a family of molecules, called ephrins and Eph receptors, which play important roles in the developing, regenerating, and injured nervous systems. Specifically, we are currently interested in areas of adult neurogenesis, neuroprotection, synaptic plasticity, regeneration, and therapeutic strategies. Overall, our approach is to develop novel strategies to minimize CNS damage and maximize regeneration, which can be best achieved through a comprehensive mechanistic approach.
DAMIEN D. PEARSE, PH.D.
Associate Professor, Department of Neurological Surgery
Exploration and Translation of Therapeutic Strategies to Repair the Injured Spinal Cord and Brain
My laboratory focuses on several key aspects of CNS injury repair, including (1) the utility and clinical translation of exogenous and endogenously harnessed cell therapeutics (particularly when used in combinatorial approaches), (2) understanding the role of, and developing therapies for, altered cyclic AMP (adenylyl cyclase, phosphodiesterases, and PKA) and MAPK signaling in neurons and glia after CNS injury, (3) the use of nanotherapeutics for multifunctional and site-directed gene/drug targeting to the injured CNS, and (4) the application of methodologies for improved imaging of axonal regeneration and cell integration within the injured CNS such as 3D ultramicroscopy and diffusion tensor imaging.

JUSTIN C. SANCHEZ, PH.D.
Associate Professor, Department of Biomedical Engineering
Director, Neuroprosthetics Research Group
Neuroprosthetics and Neural Engineering
The goals of my research are to develop brain-machine interfaces (BMI) to restore communication and movement control to people with neurological impairments. The approach is to use technology to directly interact with the central and peripheral nervous system, interpret the internal coding of brain control for specific actions, and send commands to bionic devices to trigger movements. The laboratory uses electrophysiological and neural computational tools to seamlessly interface these devices with the nervous system.

PANTELIS TSOULFAS, M.D.
Associate Professor, Departments of Neurological Surgery and Cell Biology & Anatomy
Neurotrophins: Specificity of Action
My laboratory is interested in two areas of neurobiology that are significant for developing new strategies for spinal cord injury (SCI) repair. Over the past years, we have worked to modify neurotrophins that are better suited for use in SCI. We are also interested in understanding the processes involved in maintaining and differentiating neural stem cells.

MICHAEL Y. WANG, M.D., F.A.C.S.
Associate Professor, Departments of Neurological Surgery and Rehabilitation Medicine
Spinal Cord Injury Outcomes
My primary research has been in the investigation of spinal cord injury (SCI) Outcomes. I work with Miami Project researchers Drs. Allan Levi and Barth Green in studying the clinical effects of Hypothermia. Currently, a multi-center randomized, prospective study on the effects of hypothermia in SCI is planned. In addition, I am studying the clinical application of SCI biomarkers to predict the effects of both injuries as well as therapeutic interventions with Drs. Dalton Dietrich and Ross Bullock.
KIM ANDERSON-ERISMAN, PH.D.
Research Associate Professor, Department of Neurological Surgery
Translational Investigations for Chronic Spinal Cord Injury
My research focuses on translational investigations and bridging the gap between basic science, clinical science, and the public community living with spinal cord injury. I recently completed a multi-center clinical study evaluating the reliability and validity of the Spinal Cord Independence Measure in the US healthcare setting and am currently focusing on issues specific to chronic injury.

NANCY L. BRACKETT, PH.D., H.C.L.D.
Research Associate Professor, Departments of Urology and Neurological Surgery
Male Fertility following Spinal Cord Injury
Our research is focused on understanding and improving impairments to male fertility which occur following spinal cord injury (SCI). A major aim is to determine the cause of impaired semen quality in men with SCI. Our recent evidence indicates that the problem is related to the seminal plasma. Our current research is investigating inflammatory factors, including semen cytokine levels, as contributors to the problem. Our ultimate goal is to develop therapies to normalize semen quality in men with SCI, so that chances of biological fatherhood are increased.

IAN D. HENTALL, PH.D.
Research Associate Professor, Department of Neurological Surgery
Brainstem Influences on Neurotrauma
Our research is centered on the general idea that serotonin-containing brainstem neurons influence natural repair processes following brain or spinal cord injury. We study in rats (1) how these brainstem (raphé) neurons respond during injury; (2) how raphé activity influences restorative molecular mechanisms in damaged regions; and (3) how recovery from traumatic spinal cord or brain injury is improved by prolonged electrical stimulation of these nuclei, a procedure with potential for treating acute injury in man.

GILLIAN A. HOTZ, PH.D.
Research Associate Professor, Department of Neurological Surgery
Director, KiDZ Neuroscience Center
Director, Concussion, WalkSafe™ & BikeSafe™ Programs
As a behavioral neuroscientist my clinical interests have always been investigating the neurocognitive deficits of those individuals that have sustained a traumatic and acquired brain injury. I have co-authored two neurocognitive tests, The Brief Test of Head Injury for Adults and the Pediatric Test of Brain Injury for Children. My research has also focused on injury prevention, preventing brain and spinal cord injuries in children, and I have developed the WalkSafe program, which has been shown to decrease the number of elementary school age children that get hit by cars, and now the BikeSafe program to educate middle school age children on bicycle safety skills. As the Director of the Concussion Program we have a comprehensive program including neurologic evaluation, neuroimaging, neuropharmacological management, and neuropsychological testing using ImPACT, a computerized neurocognitive screening measure.
ALBERTO MARTINEZ-ARIZALA, M.D.  
Clinical Associate Professor, Departments of Neurology, Neurological Surgery, and Orthopaedics & Rehabilitation  
Pathophysiology and Treatment of Secondary Complications in Spinal Cord Injury  
My research interests focus on common complications that are seen following spinal cord injury: pain, spasticity, syringomyelia, and tethered cord syndrome. My interests include investigating the basis for the development of the different spasticity and pain profiles in the spinal cord injured population and to study potential novel treatments for those conditions.

BRIAN R. NOGA, PH.D.  
Research Associate Professor, Department of Neurological Surgery  
Brain and Spinal Mechanisms Controlling Walking  
Our long-term goal is to develop and optimize treatments for spinal cord injury based on transmitter enhancement strategies that include deep brain stimulation, delivery of neurotransmitters or similarly acting drugs, or transplantation of cells secreting these substances. Of the many possible neurotransmitter candidates that could be used for this purpose, monoamines hold particular promise. We have concentrated our recent research effort on understanding the role monoamines play in the control of walking in the normal and injured spinal cord.

EVA WIDERSTRÖM-NOGA, D.D.S., PH.D.  
Research Associate Professor, Departments of Neurological Surgery, Rehabilitation Medicine, Neuroscience Program, and Health Scientist Veterans Affairs  
SCI-related Neuropathic Pain Phenotypes and Biomarkers  
My research program is focused on the identification of clinical correlates of underlying mechanisms of neuropathic pain associated with neurological trauma in order to facilitate the translation of basic research finding treatments tailored to specific mechanisms. My research program is highly collaborative and includes extensive interdisciplinary protocols for a multimodal evaluation of pain symptoms and its psychosocial impact, quantitative assessment of neurological function, and biomarkers including non-invasive brain imaging and genetic polymorphism.

COLEEN ATKINS, PH.D.  
Assistant Professor, Department of Neurological Surgery  
Developing Novel Therapies for Traumatic Brain Injury and Spinal Cord Injury  
The research in my laboratory focuses on developing novel therapeutic interventions for traumatic brain injury (TBI) and spinal cord injury (SCI). The research goal of my laboratory is to enhance rehabilitation and recovery by manipulating synaptic plasticity at specific levels of the neuroaxis following TBI and SCI. We have found that specific synaptic plasticity signaling pathways are altered after TBI and we are currently using pharmacotherapies to target those pathways to improve behavioral recovery after TBI.
JAE K. LEE, PH.D.
Assistant Professor, Department of Neurological Surgery
Neutralizing inhibitors of axon regeneration; stimulating plasticity
The long term research goal in my laboratory is to elucidate the mechanisms of cellular interactions in the injured adult spinal cord that creates an environment inhibitory to axonal growth. Currently, we know which cells can produce what types of inhibitory molecules, but we need a better understanding of how these cells interact and the relative significance of the different inhibitory molecules they produce in order to identify an effective therapeutic target to treat spinal cord injury and related neurological disorders.

HOWARD B. LEVENE, M.D., PH.D.
Assistant Professor, Department of Neurological Surgery
Schwann Cell Transplantation after SCI
One proposed therapy for spinal cord injury is to introduce cells to the injury site to help repair, restore, or support existing neurons. My research focuses on a large animal model to study the effect and behavior of transplanted autologous Schwann cells. I have been involved in the refinement of this animal model. This approach allows for the scientific study of the behavior of implanted cells and generates the groundwork for clinical trials. Research utilizing this model is done in collaboration with clinicians and scientists at the Miami Project such as Drs. Guest, Solano, Pearse, Wood, Bunge, and many more.

KEVIN K. PARK, PH.D.
Assistant Professor, Department of Neurological Surgery
Intrinsic mechanisms of axon regeneration
My lab is interested in understanding the neuron’s intrinsic mechanisms that account for failure of axon regeneration in the central nervous system (CNS). Previously, I and others have identified several key proteins that block axon regeneration, which are present in mature CNS neurons. In my current research, I will further extend my findings in order to better understand the mechanisms governing robust axon regeneration and also to explore the potential of developing therapeutic strategies for spinal cord injury and other neurodegenerative conditions.

ROBERTA BRAMBILLA, PH.D.
Research Assistant Professor, Department of Neurological Surgery
Modulation of the immune response in neurologic disease
The main focus of my research has been to understand the role of neuroinflammation in the pathophysiology of neurodegenerative disorders (e.g., spinal cord injury and multiple sclerosis), with a specific interest in the contribution of the astrocytes, a type of glial cell that represents the most abundant cell population in the nervous system. Currently, my laboratory is developing two specific lines of research in the area of neuroimmunology, which focus on (1) investigating the role of tumor necrosis factor in the processes of demyelination and remyelination and (2) investigating the occurrence of neuropathic pain associated with multiple sclerosis.
**JUAN PABLO DE RIVERO VACCARI, PH.D.**  
Research Assistant Professor, Department of Neurological Surgery  
Underlying mechanisms of the innate immune response and contributions to various CNS diseases  
My research focuses on understanding early inflammatory events in central nervous system (CNS) trauma and neurodegenerative diseases. Currently, my laboratory is focusing on the effects of pattern recognition receptor (PRR)-activation after spinal cord injury (SCI) and neurodegenerative diseases such as Alzheimer's disease.

**PAULA V. MONJE, PH.D.**  
Research Assistant Professor, Department of Neurological Surgery  
Molecular signals controlling Schwann cell proliferation and differentiation  
In vitro methods to enhance the Schwann cell’s potency for CNS repair  
My laboratory studies basic aspects of Schwann cell biology and their use in transplantation for central nervous system regeneration. Specifically, we are investigating the role of cAMP and growth factors in the reciprocal interactions between Schwann cells and neurons underlying the regulation of Schwann cell proliferation and myelination, as well as the initiation of Schwann cell dedifferentiation after injury. Our lab works intensively towards refining the use and developing new cell culture methods for the growth and assessment of function of both human and rodent Schwann cells. One important goal is to improve the quality of cultured adult Schwann cells for an intended use in clinical trials.
The Miami Project has brought many renowned neuroscientists from around the world to our campus as part of The Gail F. Beach Memorial Visiting Lectureship Series. The lectureship series is dedicated to Gail F. Beach, a schoolteacher and person with SCI, whose generosity and foresight continues to provide outstanding educational opportunities for The Miami Project researchers and our neuroscience colleagues at the University of Miami.

September 7, 2011
**Qiufu Ma, Ph.D.**
Harvard Clinical and Translational Science Center, Harvard University Medical School
“Generation of Sensory Neuron Diversity and Populating Coding of Pain Versus Itch”

November 2, 2011
**Bente K. Pedersen, M.D., DMSc**
Centre of Inflammation and Metabolism, University of Copenhagen
“Skeletal Muscle as an Endocrine Organ with Focus on IL-6”

December 7, 2011
**Christine E. Schmidt, Ph.D.**
Department of Biomedical Engineering, University of Texas
“Engineering Novel Biomaterials for Axonal Regeneration”

January 4, 2012
**Eva L. Feldman, M.D., Ph.D., FAAN**
A. Alfred Taubman Medical Research Institute, University of Michigan
“Intraspinal Stem Cell Therapy for ALS: Challenges and Opportunities”

February 3, 2012
**David Hovda, Ph.D.**
UCLA Brain Injury Research Center, David Geffen School of Medicine at UCLA
“Cerebral Effort In Terms of Traumatic Brain Injury and Recovery of Function”

March 7, 2012
**Andrew A. Pieper, M.D., Ph.D.**
University of Texas Southwestern Medical School
“In Vivo Discovery of Pharmacologic Agents for Neuropsychiatric Disease”

April 4, 2012
**Patrizia Casaccia, MD, PhD**
Icahn Medical Institute, Mount Sinai School of Medicine
“Protein Acetylation in Multiple Sclerosis”

June 6, 2012
**Alvaro Pascual-Leone, MD, PhD**
Beth Israel Hospital, Harvard University Medical School
“TBA”
Published studies that have passed the test of peer review are the benchmark of scientific progress. Listed here are the 2011 research publications by Miami Project scientists and colleagues.


Entropy and Mutual Information Compression. IEE Trans Neural Syst Rehabil Eng. 19:35-44.


The Miami Project is doing everything possible to make paralysis a thing of the past. Please help us.