National Multiple Sclerosis Society

NEW RESEARCH

$14.6 Million for 34 New MS Research Projects

Despite a challenging economic environment, the National MS Society has just launched 34 new MS research projects, with cumulative multiyear commitments of $14.6 million. These new projects are part of our comprehensive research program and our commitment to move research forward.

The scope of this current launch is made possible by generous support of Society chapters and individual donors. When the National MS Society makes research commitments that span into future years, the money is not yet in hand to meet those needs. Contributions to the Society to help support these projects are essential to ensure that this important research proceeds in future years.

The new projects include studies focusing on discovering risk factors that lead to progressive disability, projects aimed at speeding diagnosis, research on protective mechanisms of vitamin D and estrogen, and tests determining whether a new device can improve walking ability.

Following are brief summaries of the new research projects, grouped according to avenues of MS investigation as they fit into the major goals of stopping MS, restoring function and ending MS forever.

STOPPING MS AND ENDING MS FOREVER

Epidemiology: Who Gets MS?

Epidemiologists evaluate disease patterns among people with a certain disease, taking into account variations in geography, demographics, socioeconomic status, genetics, and exposure to infectious and toxic agents. These types of studies, when carefully done, can help us to understand why this disease appears more frequently in certain populations, and why it progresses. Epidemiological studies ultimately seek to discover the cause of MS, and may also serve as the basis for developing future treatments. The National MS Society is currently funding 8 research projects in epidemiology, with multiyear commitments totaling $2.1 million.

Inside…

Alberto Ascherio, MD, DrPH
Harvard School of Public Health
Boston, MA
Area: Greater New England/Northeast
Term/Amount: 10/1/10-9/30/14; $121,728
"MS risk and progression: role of vitamin D, EBV, and smoking" Identifying modifiable factors that affect conversion and progression from a first neurological episode (CIS) to MS.

Research into risk factors that influence whether a person develops MS is key to finding the cause of and cure for this disease. Alberto Ascherio, MD, DrPH, and his team have previously found that higher vitamin D intake and high blood levels of vitamin D are associated with a significantly lower risk of developing MS, and that smoking, and elevated levels of antibodies to Epstein-Barr virus (EBV, a herpesvirus known to cause infectious mononucleosis), are associated with an increased risk of MS.

Now this expert group is attempting to determine whether these factors play a role in the progression from clinically isolated syndrome (CIS, a first demyelinating event indicating high risk for developing MS) to MS, and early progression of the disease. They are evaluating blood samples and data from more than 1600 individuals with CIS who were followed for progression to MS with clinical and MRI exams.

This study, which is being co-funded by the National Institutes of Health, presents a unique opportunity to examine links among vitamin D levels, EBV infection, and cigarette smoking and their possible impact on early MS progression.

Tanuja Chitnis, MD
Brigham and Women's Hospital
Boston, MA
Area: Greater New England/Northeast
Term/Amount: 10/1/10-9/30/13; $430,234
"Biomarkers and risk factors for disease progression in MS" Determining factors that may influence whether, and how rapidly, MS may develop and progress.

In most cases, MS initially presents as a relapsing-remitting disease, in which neurological symptoms come and go. However, as time goes by, many people with MS enter the secondary-progressive stage in which neurological symptoms do not go away, and disability becomes chronic. There is increasing evidence that the biological processes that cause disease progression are different than those that cause relapses. It is also becoming apparent that progressive disability may begin to accumulate early in the disease course.

Little is understood about why people with MS progress at different rates, and what are the biological processes that are important in progression. Also, there are no biological markers or indicators that would predict who may progress at a higher rate than others. Dr. Chitnis is tapping into a large group of people with MS for whom there is detailed clinical information, serial MRIs and longitudinal immunological blood studies, as well as stored serum samples, to begin addressing many of these unknowns.

The team hopes to identify clinical risk factors including those that are potentially modifiable, such as hormones and vitamin D levels, which may affect the rate of disease progression. In addition, the team will develop statistical models that can allow

Howard Weiner, MD
Brigham and Women's Hospital
Boston, MA
Area: Greater New England/Northeast
Term/Amount: 10/1/10-9/30/12; $627,400
Funded in full by the National MS Society's Greater Delaware Valley Chapter
"The Dr. John R. Richert Pilot Study on Risk Factors for Progression in MS" Identifying factors that influence the course and progression of MS.

The question of what factors influence the course and progression of MS is the focus of a new research initiative. The goal of this first step is for a consortium of investigators to test the feasibility of a longitudinal study to determine why some people with MS have mild courses while others experience serious worsening of symptoms over time.

As one of the two projects launched in response to a Request for Applications from the National MS Society, Dr. Weiner has established the "SUMMIT" core consortium. Four major academic MS Centers (representative of divergent geographical populations) that have established patient cohorts will, in a prospective pilot study that combines cohorts from each center, collect uniform data (clinical, MRI, blood, and genetics) on 1500 MS patients over a two-year period. In addition, five categories of epidemiological data will be collected: 1) Background and Family History; 2) Vaccination and Infectious Diseases History; 3) Tobacco Smoking History, 4) Diet and Sun Exposure, and 5) Gender Hormones and Pregnancy.

They will focus on factors or combination of factors that link to disease progression using data from this two year collection period, as well as data obtained in 5 years prior.

Researchers are seeking factors that influence whether a person will develop MS, and whether their MS will cause progressive disability

Bianca Weinstock-Guttman, MD
State University of New York at Buffalo
Buffalo, NY
Area: Upstate NY/Northeast
Term/Amount: 10/1/10-9/30/12; $646,012
Funded in part by the National MS Society's Greater Delaware Valley Chapter
"Clinical, MRI, neuropsychological and gene-environmental risk factors for progression in MS" Identifying factors that influence the course and progression of MS.

The question of what factors influence the course and progression of MS is the focus of a new research initiative. The goal of this first step is for a consortium of investigators to test the feasibility of a longitudinal study to determine why some people with MS have mild courses while others experience serious worsening of symptoms over time.

The New York State MS Consortium was created in 1996 to develop a unique demographic and clinical long-term follow-up database of MS patients to provide a durable resource for interdisciplinary research.

For this pilot study, one of two launched to test the feasibility of tracking risk factors that influence MS progression, Dr. Weinstock-Guttman and colleagues will define a distinct incidence cohort of 500 people with MS within the Consortium. They will look at genetic and environmental factors, such as vitamin D, smoking. They will include individuals with relapsing MS and primary-progressive MS, with a disease onset within 5 years. They will also include 20% African-Americans, who have been found to be at higher risk for more rapidly progressive disease.

The team hopes to demonstrate the feasibility of developing and maintaining a prospective longitudinal cohort to investigate risk factor roles and interactions.

STOPPING MS

Diagnosing MS and Tracking its Course

To better understand the course of MS and factors that may influence that course, researchers are using advances in imaging and other techniques. This vital information may eventually be used to diagnose MS earlier, and to help track disease changes – either progression of disease, or improvements due to experimental treatments – before they are apparent clinically.

The National MS Society has current, multi-year commitments of about $7.5 million to support research projects focusing on improving diagnosis of MS and ways of tracking disease activity in MS.

Pallab Bhattacharyya, PhD
Cleveland Clinic Foundation
Cleveland, OH
Area: Ohio Buckeye/East
Term/Amount: 10/1/10-9/30/12; $333,609
"Role of inhibitory neurotransmitter GABA in motor performance in MS" Studying how a molecule used by nerves to communicate with each other may contribute to physical disability in MS.

"Motor performance," the strength and dexterity of limb and trunk movements, often declines in people with MS. Much of this deficit may be due to direct effects of slowed or blocked nerve signals caused by damage to myelin, the material that surrounds and protects nerve fibers in the brain and spinal cord. Nerve fibers themselves are also damaged. However, there may be other factors that contribute to movement difficulty in MS.

Pallab Bhattacharyya, PhD, is looking at the possible role of a substance called "gamma amino butyric acid (GABA). GABA is a "neurotransmitter," one of a number of signaling molecules that nerve cells use to communicate with each other. While some neurotransmitters are excitatory, increasing the activity of the cells they reach, GABA is an inhibitory molecule that reduces the activity of nerve cells. Using a sophisticated version of MRI called magnetic resonance spectroscopy (MRS), Dr. Bhattacharyya found indication of increased brain levels of GABA in a sampling of people with MS. Now he is attempting to correlate the amount of GABA with the severity of motor deficit in people with MS.

This research could establish a new way to measure MS activity and a very sensitive method to evaluate the effect of treatments in clinical trials for MS.

The search for MS "biomarkers" is heating up: These physical clues may soon help diagnose MS, predict its course, and help with treatment

Trevor Kilpatrick, MBBS, PhD
University of Melbourne
Melbourne, Australia
Term/Amount: 10/1/10-9/30/13; $225,245
"Testing paraclinical outcome measures in optic neuritis" Designing a method to measure nerve fiber protection for use in future clinical trials of new treatments for MS.

Demyelination – the destruction of the myelin sheath that surrounds and protects nerve fibers – in the central nervous system (CNS: brain, spinal cord and optic nerves) is a major cause of symptoms of MS. Moreover, the loss of demyelinated nerve fibers can lead to long-term disability. In order to test new therapies for their ability to protect nerve fibers from destruction, researchers need techniques to measure their effectiveness.

Trevor Kilpatrick, MBBS, PhD, is assessing the ability of several techniques to measure nerve fiber structure and function during and after incidents of optic neuritis – due to demyelination of optic nerve fibers – that occur in MS. Dr. Kilpatrick and colleagues want to see if measures of optic nerve structure as optic neuritis develops can predict the amount of damage to nerve fiber function that results from the attack.

The results could help establish a reliable method to assess new potential drug therapies designed to prevent loss of nerve fibers and function in MS.

Lauren Krupp, MD
State University of New York at Stony Brook
Stony Brook, NY
Area: Long Island/Northeast
Term/Amount: 10/1/10-9/30/11; $149,903
"Biomarkers in the early detection of MS"
Studying protein patterns in attempts to develop a new, rapid blood test to improve early diagnosis of MS.

Diagnosis of MS often takes a long time. Because the symptoms of MS are variable and similar to other diseases, many people in the early stages of MS experience the frustration of not knowing the cause of their symptoms. Slow diagnosis can mean delay in starting treatment for MS, so a rapid, accurate test would be an important advance.

Lauren Krupp, MD, is using high-tech "proteomics" to look at blood samples from three groups of children and adolescents – those with MS, those with other neurological diseases, and those with no disease. Proteomics enable researchers to identify a large number of different types of proteins. Dr. Krupp's team is looking for patterns of proteins that are unique to people with MS, and may relate to the severity of disease.

This project could establish a new way of diagnosing MS using a simple blood sample. The possibility that protein patterns could predict MS severity may lead to treatments tailored more closely to individuals with MS.

Sharon Lynch, MD
University of Kansas Medical Center
Kansas City, KS
Area: Mid America/Midwest
Term/Amount: 10/1/10-9/30/11; $144,195
"Glutathione as a measure of oxidative stress in multiple sclerosis" Analyzing levels of a brain chemical to understand the role of oxidative stress in disease severity and its potential as a therapeutic target.

Oxidative stress is being studied as an active problem causing damage to the central nervous system in people with multiple sclerosis. It may partially be responsible for chronic, ongoing damage in the progressive aspects of the disease. Glutathione (GSH) is a brain chemical that is employed in the brain's frontline defense against oxidative stress and is used up in the process of oxidation. A reduction in GSH in the brain indicates the presence of increased oxidative stress.

Using MR spectroscopy, Dr. Lynch's team has measured GSH levels in the brains of a small number of people with secondary-progressive MS and healthy subjects and found significant reductions of GSH levels in MS. The team is now embarking on a larger study comparing GSH levels in healthy individuals compared to different subtypes of MS, including 60 people with primary-progressive MS, 60 with relapsing-remitting MS and 60 with secondary-progressive MS. They aim to understand the role of oxidative stress during different phases of the disease and its relationship to disease severity.

They will also obtain information comparing GSH levels with the severity of physical and cognitive disability and abnormalities on standard MRI scans. This innovative study could lead to the development of medications that target oxidative stress, and indicators to help determine the timing of such therapy.

Steven Schutzer, MD
University of Medicine and Dentistry of New Jersey
Newark, NJ
Area: NJ Metro/Northeast
Term/Amount: 10/1/10-9/30/12; $296,487
"The Admiral Thor Hansen Biomarkers in Multiple Sclerosis Project" Analyzing proteins in spinal fluid to develop a new method of diagnosing early MS.

MS can be difficult to diagnose. Early symptoms of MS are variable and may mimic those of other diseases, and there is no single laboratory test to prove someone has MS. Because the available drugs for treating MS are most effective during early stages of the disease, a method of rapid, accurate diagnosis would allow treatment to begin more quickly.

Steven Schutzer, MD, is attempting to uncover candidate proteins with diagnostic potential in the spinal fluid that bathes the brain and spinal cord. Dr. Schutzer has developed a database cataloging the thousands of proteins found in normal spinal fluid by a technique known as mass spectrometry. Now his team is looking at spinal fluid sampled from individuals after the first attack of MS, and attempting to identify those proteins that are unique to people with very early MS.

Results could lead to an important new method to identify early MS and thus the opportunity to start treatment at its most effective point.

National MS Society Tissue Banks: To Stop MS and End MS Forever

People living with MS may hold the key to curing this disease. The National MS Society supports three MS tissue banks, which are storage facilities that provide brain and spinal cord tissues to researchers studying the disease. These studies generally focus on the pathology of MS — its nature, cause, and effects on the brain.

Specimens from persons who had MS during their lifetimes are frozen or otherwise preserved very soon after death. The tissues are carefully catalogued with information about the donor's medical history. An important aspect of these banks is their ability to find new tissue donors and to meet the needs of hundreds of investigators who depend on them for their research. Visit our Website/Research/Researchers Need You for information on how to arrange a donation.

Rashed Nagra, PhD
Brentwood Biomedical Research Institute
Los Angeles, CA
Area: Southern California & Nevada/West
Term/Amount: 10/1/09-9/30/14; $1,268,602
"Human brain and spinal fluid resource center"

This is the longest standing of three tissue banks supported by the National MS Society. Recently, the bank has been working on a system to provide extracted RNA to investigators, which would greatly expand the tissues' utility for genetic and other studies. In light of its collaboration with the VA Healthcare System Research Service, this bank will benefit from a new VA-funded construction project to build new laboratory space specifically for Bank use, with state of the art labs and records storage.

John Corboy, MD
University of Colorado Health Science Center
Aurora, CO
Area: Colorado-Wyoming/West
Term/Amount: 10/1/09-9/30/12; $611,366
"Rocky Mountain MS Center Tissue Bank"

This bank is focusing on obtaining donations from the local population to facilitate processing of the brain and spinal cord within four hours of a person's death. This would greatly enhance the quality of the specimens and their usefulness in understanding the MS disease process with sophisticated lab techniques. This year the bank was successfully moved to the University's Biorepository Core Facility, which has trained staff and vastly greater freezer space and freezer emergency backup that is crucial to retaining these precious tissues.

STOPPING MS

Why the Immune System Goes Awry

The current therapies for MS emerged from our growing understanding of how the immune system works and how it can be manipulated to suppress or regulate immune attacks. We especially need to know more about the molecules that the immune system uses to attack the nervous system, because each of these serves as a potential therapeutic target for new therapies.

The National MS Society has current, multi-year commitments of about $32 million to support research projects focusing on stopping the immune system attack in MS.

Bonnie Dittel, PhD
Blood Center of Wisconsin
Milwaukee, WI
Area: Wisconsin/Midwest
Term/Amount: 10/1/10-9/30/13; $440,724
"Elucidating the role of cannabinoid receptor 2 in immune regulation during EAE"
Looking at how cannabis-related molecules interact with and affect the immune system and their potential for turning off immune attacks.

The class of drugs known as cannabinoids are perhaps best known for the effects of delta-9-tetrahydrocannabinol (THC), the active substance in marijuana. The effects of THC on the brain are mediated by molecules known as cannabinoid receptor 1 (CB1) on the surfaces of nerve cells, to which THC attaches. Immune system cells have a related cannabinoid receptor, CB2, which also binds THC along other cannabinoids.

In this research project, Bonnie Dittel, PhD, is looking at several cannabinoids that bind to CB2 and studying the effects they have on immune system cell activity. Dr. Dittel and colleagues have found that mice with the MS-like disease EAE, which were genetically modified to lack CB2 on some of their immune cells, developed very severe disease. Now Dr. Dittel's team is using natural and laboratory-manufactured cannabinoids to determine how they alter immune function and to find which ones suppress EAE most strongly, with an eye toward refining the use of cannabinoids to treat MS in people.

This research will produce better understanding of the role of CB2 in the immune system and could lead to a new class of treatments for MS.

Brian Evavold, PhD
Emory University
Atlanta, GA
Area: Georgia/Southeast
Term/Amount: 10/1/10-9/30/13; $442,898
"T cell affinity for myelin controls autoimmune disease severity and outcome" Using high-tech screening to study how the cells that control immune system activity respond to myelin, the nerve-protecting sheath targeted by MS immune attacks.

In MS, the immune system – the group of cells that normally protects the body from infectious agents such as viruses or bacteria – mistakenly attacks and damages myelin, the fatty substance that surrounds nerve fibers in the brain and spinal cord. Myelin and nerve fiber damage disrupts the signals in nerve fibers and leads to the varied symptoms of MS. Immune system cells called T cells coordinate immune system behavior: some increase the strength of an attack, while others limit or shut off attacks.

A vast body of evidence now supports a link between low levels of vitamin D and the development of MS

Brian Evavold, PhD, is looking at the affinity of T cells for myelin proteins – how tightly various T cells bind or stick to proteins from myelin – to see how this factor influences the course of disease. Using a newly developed high-tech method to measure T cell affinity, Dr. Evavold has found that in mice with EAE, a model of MS, T cells that respond to myelin proteins have a broad range of affinities for them. Now he is determining whether the different affinities influence the severity of disease. In addition, he is investigating whether human T cells have a similar range of affinities for myelin in MS.

This work could significantly increase our understanding of how the immune system attack on myelin is controlled and could lead to new avenues for developing treatments for MS.

Colleen Hayes, PhD
University of Wisconsin-Madison
Madison, WI
Area: Wisconsin/Midwest
Term/Amount: 10/1/10-9/30/13; $585,146
"Vitamin D and estrogen synergy in the control of EAE" Exploring how vitamin D and the sex hormone estrogen may interact to control MS-like immune attacks and its implications for MS.

In MS and EAE, an animal model of MS, myelin, the material that surrounds and protects nerve fibers, and the cells that produce myelin in the central nervous system are attacked by cells from the immune system. Colleen Hayes, PhD, and her colleagues suggested that the reason MS is more common in regions with less sunlight exposure is that the body makes vitamin D when the skin is exposed to ultraviolet (UV) light, and vitamin D is a natural inhibitor of MS. A vast body of evidence now supports this suggestion. Higher vitamin D levels have been strongly linked with less risk of developing MS and with fewer relapses and less disability in those with MS. Dr. Hayes and her colleagues have found vitamin D to be a potent inhibitor of EAE, and are investigating the details of this process to devise strategies for using vitamin D to inhibit MS.

Now Dr. Hayes and Dr. Halina Offner, M.D., are studying how vitamin D and estrogen, a female sex hormone, work together to decrease EAE in female mice. Their preliminary work has shown that estrogen is required for vitamin D to have its maximal protective effect on EAE. Now, using genetically modified mice, they are looking at the details of how vitamin D, estrogen, and their respective receptors interact, as a prelude to understanding these interactions in women

with MS.

This research could lead to a new understanding of the rapidly increasing female sex bias in MS, and to new ways to treat and possibly prevent MS in women.

Robyn Klein, MD, PhD
Washington University
Saint Louis, MO
Area: Gateway Area/Midwest
Term/Amount: 10/1/10-9/30/13; $460,638
"Regulation of blood-brain barrier immune privilege during CNS autoimmunity" How to prevent breakdown of the barrier separating the brain and spinal cord from the bloodstream, for clues to stopping MS.

MS involves activity of immune system cells that damages the brain and spinal cord. The small blood vessels in the brain have a layer called the "blood-brain barrier" (BBB) that ordinarily limits the movement of immune system cells from the blood into the brain. In MS, and in an animal model of MS known as EAE, portions of the BBB break down, allowing destructive immune system cells to enter brain tissue and launch attacks.

For this project Robyn Klein, MD, PhD, is using EAE and brain tissue from people with and without MS to study the activity of several molecules involved in the movement of immune system cells through the BBB. Dr. Klein and colleagues hope to identify a substance that can be used to restore the BBB to normal function and block the entry of immune system cells into brain tissue.

This research will lead to new understanding of how the BBB fails in MS and could provide clues for treatments to restore function to the BBB and block immune system attacks in MS.

Vijay Kuchroo, PhD, DVM
Harvard Medical School
Boston, MA
Area: Greater New England/Northeast
Term/Amount: 10/1/10-9/30/13; $430,650
"Pathogenic and regulatory mechanisms in EAE" Finding ways to control the immune system attack on myelin, for clues to stopping nerve tissue damage in MS.

In MS, the immune system, which normally defends the body against foreign invaders, attacks and destroys myelin, and nerve fibers are destroyed as well. Myelin is the material that surrounds and protects nerve fibers in the brain and spinal cord. Immune system cells that are primarily responsible for coordinating and controlling the attack on myelin belong to a class known as "T cells". There are a number of different types of T cells, each with a specific role in activating or suppressing the immune system attack.

Vijay Kuchroo, DVM, PhD, and his colleagues are studying the behavior of a group of T cells known as "Th17" cells in mice with the MS-like disease EAE. They have found that Th17 cells are particularly aggressive in stimulating myelin damage. They have also found that other immune system cells can turn off the aggression of Th17 cells with signaling molecules known as cytokines. They are now looking at how Th17 cells and several other types of immune system cells interact to regulate EAE, and by implication, MS.

The results of this work could greatly increase our understanding of how the immune system damage to myelin can be controlled and may lead to new clues for stopping nervous tissue damage in MS.

National MS Society Research

The National MS Society is committed to freeing the world of MS. Our global support of MS research and treatment focuses on three key areas: stopping the progression of the disease, restoring function that's been lost, and ultimately ending the disease forever.

We do this by:

- Funding the most promising avenues
- Engaging the best and brightest minds
- Acting as a vital connector for people, resources and ideas
- Developing more and effective treatments faster
- Identifying and filling gaps in MS research

Research Objectives Outlined in Our Strategic Response 2011-2015

- We better understand the scientific mechanisms that lead to disease progression and we accelerate the development of new therapies.
- We pursue new avenues to discover how nerve cells are damaged and potentially repaired.
- We pursue new rehabilitation techniques and symptomatic treatments to restore neurological function and enhance quality of life.
- We identify risk and triggering factors that cause MS, and understand the biological interactions that lead to its development so that MS can be prevented.
- We expand and strengthen the quantity and quality of MS research worldwide to accelerate new discoveries and treatments for people with MS.

Society Research Spending:
$36 million in 2009 for 375 projects
Cumulative Investment:
$686 million (by end FY '09) since first 3 grants in 1947
Major Types of Society Research Support:
Grants: multiyear investigations by university-based scientists for basic and clinical research
High risk/high potential Pilot grants: one-year awards to test innovative, cutting-edge ideas
Industry Partnerships: milestone-driven drug development funding for private companies
Fellowships: to attract and train promising young investigators and doctors to focus on MS
Rehabilitation Research Fellowships: to meet the unmet need for specialists trained to conduct quality rehabilitation research
Health Care Delivery and Policy Contracts: to inform advocacy efforts and enhance quality of life for people with MS

Yasmina Laouar, PhD
University of Michigan
Ann Arbor, MI
Area: Michigan/Midwest
Term/Amount: 10/1/10-9/30/13; $435,049
"Role of TGFbeta in the control of autoimmune encephalomyelitis" Studying a signaling molecule that can inhibit immune attacks, and its implications for stopping MS.

In MS, myelin, the material that surrounds and protects nerve fibers, is attacked and destroyed in the brain and spinal cord by the immune system. The nerve fibers themselves are also damaged. The immune system consists of different types of cells that communicate and coordinate activity through a number of signaling molecules. Some of the molecules increase immune system attacks, while others tend to reduce or halt them.

Yasmina Laouar, PhD, is studying the role of the immune system signaling molecule known as TGF-beta, which often acts to suppress or limit immune system attacks. Dr. Laouar is using "transgenic" mice specifically engineered to lack docking sites to which TGF-beta attaches (called TGF-beta receptors). Dr. Laouar and colleagues found that these mice develop severe MS-like disease (EAE). Now they are trying to understand how the lack of TGF-beta receptors leads to spontaneous EAE, and how TGF-beta normally helps suppress EAE.

This work will provide new understanding of the role of TGF-beta in immune system control and may provide new avenues for research to treat MS.

Virtually every new molecule uncovered in the immune attack in MS has potential as a target for new therapies to stop the disease process

Jason Lees, PhD
University of Maryland at Baltimore
Baltimore, MD
Area: Maryland/East
Term/Amount: 10/1/10-9/30/13; $268,236
"Molecular mechanisms of secondary T-cell recruitment to established CNS lesions" Determining how the population of immune system attack cells changes from early to later stages of MS-like disease for clues to new treatment approaches.

The symptoms of MS result from an immune system attack against myelin, the material that surrounds and protects nerve fibers, in the central nervous system (CNS: brain, spinal cord and optic nerves). The nerve fibers are also damaged. Immune system cells known as T cells are important in MS and in an experimental model of MS known as EAE. In MS and EAE, T cells enter the tissue of the CNS from the blood in small numbers at the start of the disease and in larger numbers, a behavior known as secondary recruitment, as the symptoms develop.

In this research project, Jason Lees, PhD, is studying T cells throughout the course of EAE, looking for evidence that the mechanisms that allow T cells to be recruited to already established areas of myelin damage differ from the mechanisms that allow early T cells to initiate the damage. Dr. Lees and his colleagues are also attempting to discover how the T cells that are recruited to an existing area of myelin damage may then go on to cause damage in a new region of the CNS.

This research will provide a better understanding of how the population of T cells change during the progress of EAE, and could yield new approaches for therapies effective in specific stages of MS.

Michael Racke, MD
Ohio State University
Columbus, OH
Area: Ohio Buckeye/East
Term/Amount: 10/1/10-9/30/13; $468,954
"Regulation of oxidative stress in autoimmune demyelination" Investigating a molecule in clinical trials to understand its influence on immune factors and how it may provide protection to the nervous system in MS.

While there are now several treatments for MS, all of those approved by the FDA are given by injection or intravenous infusion. An experimental drug that is given orally that recently showed promising results in a clinical trial in MS patients is called BG00012 (Biogen Idec) or dimethyl fumarate.

Dr. Racke's team is trying to find out how BG00012 works in MS. They believe that it may alter signaling molecules or cytokines made by immune cells in such a way that they no longer have the same disease-causing potential. The team is investigating its influence on immune factors, and also looking at changes in the nervous system that may make it more likely to withstand the immune attacks that occur in MS, a process called neuroprotection.

These studies should give us added insight into how BG00012 works in MS and may also provide new ideas about how the brain gets damaged by the immune system in the first place.

John Russell, PhD
Washington University
Saint Louis, MO
Area: Gateway Area/Midwest
Term/Amount: 10/1/10-9/30/13; $489,697
"Cytokine and chemokine regulation of regional CNS inflammation and pathogenesis" Analyzing interactions between immune messengers in directing the location of immune attacks for clues to causes of MS severity and symptoms.

Cells of the immune system normally protect the body from infectious agents including viruses and bacteria. In MS, however, the immune system attacks myelin, the substance that surrounds and protects nerve fibers in the central nervous system (CNS: brain, spinal cord and optic nerves). In their normal function, and in MS, immune system cells use a number of "signaling molecules" known as cytokines and chemokines to organize and coordinate their activity. MS is known for its variability among different individuals and at different times in the same individual. Differences in the location and the type of active signaling molecules may explain much of the variability of MS.

John Russell, PhD, and colleagues are studying how the distribution of cytokines and chemokines affects the movement of immune system cells in different regions of

the CNS in mice with EAE, a model of MS. They are also looking at how specific gene alterations that cause the loss of particular signaling molecules affect the course of the disease.

This research could provide new insights about why MS is so variable, and lead to new ideas for treating specific stages of MS.

Katharine Whartenby, PhD
The Johns Hopkins University
Baltimore, MD
Area: Maryland/East
Term/Amount: 10/1/10-9/30/13; $523,971
Paid by the National MS Society South Central Region

"Inhibition of FLT3 signal trasduction in APCs as an approach to MS therapy."
Evaluating a way to stop MS attacks by blocking specialized cells that make nervous system tissues a target.

In the central nervous system (CNS) of people with MS, the material that surrounds and protects nerve fibers, myelin, is destroyed by immune system cells. Without their protective myelin, nerve fibers do not conduct signals correctly, and they become vulnerable to destruction, resulting in the symptoms of MS. The immune system cells known as T cells are main players in the attack that destroys myelin, but T cells get clues about what to attack from cells known as antigen presenting cells (APCs).

Katharine Whartenby, PhD, is studying drugs that interfere with a process that allows APCs to give clues about what to attack. She is focusing on mice with EAE, an MS-like disease. Dr. Whartenby and colleagues have found that some of these drugs can reduce the number of APCs in the CNS, and that this makes EAE less severe. Now they are looking at details of how the drugs alter the interaction between APCs and T cells, and determining which ones enter the CNS easily. Potential advantages of these drugs are that they are given orally and are already in trials for other diseases, such as myeloid leukemia.

This research will shed light on whether a new treatment approach may hold potential for MS.

To convince insurers that rehabilitation really does help MS, there needs to be evidence that can only come from carefully designed and conducted studies

RESTORING FUNCTION

Rehabilitation

Rehabilitation regimens that can help people with MS achieve maximal physical, psychological, social and vocational potential have gained increasing acceptance in recent years. But to convince doctors and insurers that rehabilitation really does help, there needs to be scientific evidence that can only come from carefully designed and conducted studies.

The National MS Society has current, multi-year commitments of about $6 million to support investigations focusing on rehabilitation in MS.

Francois Bethoux, MD
Cleveland Clinic Foundation
Cleveland, OH
Area: Ohio Buckeye/East
Term/Amount: 10/1/10-9/30/12; $274,868
"Impact of a hip flexion assist orthosis on gait performance in multiple sclerosis patients" Evaluating a new device designed to aid walking in people with MS.

People with MS often have difficulty with walking and balance because of weakness and sometimes spasticity (stiffness) of their leg muscles. Weakness of the lower leg muscles that move the foot can be helped by a passive brace or "ankle foot orthosis." But the muscles that lift the leg and swing it forward (hip flexors) may also be weak and cause people to stumble or fall because the foot drags on the ground.

Francois Bethoux, MD, and colleagues have already completed a pilot study of a light weight and low cost device called a Hip Flexion Assist Device (HFAD) that uses a combination of straps and elastic bands to supplement the activity of weak hip flexors. The results of the pilot study suggested that the HFAD improves walking and leg strength, and is safe to use. In this new research project, Dr. Bethoux's group is evaluating the effectiveness of the HFAD in 88 people with MS. Half of the participants will wear the device for eight weeks, and the other half will not wear the brace (control group). Those in the control group will be given the brace and will be trained to use it at the end of the study. A series of tests will evaluate muscle strength, spasticity, and walking ability with and without the brace in both groups.

The data will be analyzed to determine whether use of the HFAD improves walking performance. They will also gauge usage and satisfaction with the brace, and they will record potential side effects. This study will help us understand how the HFAD can be used to improve mobility in people with MS, and will help refine how such devices can be evaluated in a clinical trial. Results from this study will also be used to generate ideas for new active mobility

RESTORING FUNCTION

Health Care Delivery/Policy Research to Improve Care Standards

What if the cure were found today but insurers refused to pay for it? Access to high quality health care is one of many issues tackled by the Society's Health Care Delivery and Policy Research Program, providing data that can serve as the basis for influencing public policy and improving the quality of MS health care and the quality of life of people with MS and their families.

The National MS Society has current, multi-year commitments of about $7.2 million to support 11 research projects focusing on health care delivery for people with MS.

Malachy Bishop, PhD
University of Kentucky
Lexington, KY
Area: Kentucky/SE Indiana/Southeast
Term/Amount: 10/1/10-9/30/12; $275,718
"Specialized housing needs in multiple sclerosis: a comprehensive analysis" Focusing on specific housing needs of people with MS to help direct resources to better meet those needs.

Many people with MS experience a range of symptoms and may experience progressive disability. Some require relatively simple home modifications, such as ramps or grab

Health care delivery and policy research gathers data that can be used to influence public policy and improve quality of life for people with MS

bars in bathrooms, while others may require accessible housing, assisted or supported living, nursing care, and a variety of other community living options. There is little research to quantify and help understand the scope of their housing needs and alternatives available for people with MS.

Dr. Bishop is conducting a comprehensive analysis of the need for, and availability of, specialized housing for adults with MS in the U.S. His team is surveying more than 4,000 people with MS across the country about their current and future needs for specialized housing, the resources available to meet these needs, and the barriers to accessible and affordable housing. People with MS will be recruited through the membership of the National MS Society and the North American Research Committee on Multiple Sclerosis (NARCOMS) patient registry. The team also is surveying MS health care and advocacy professionals related to housing needs and alternatives to nursing home care.

This information will be used to educate health care professionals, advocate for people with MS, and produce helpful information for people with MS and their families in meeting their housing needs and improving quality of life.

Michael Halpern, MD, PhD
RTI International
Washington, DC
Area: National Capital/East
Term/Amount: 10/1/10-9/30/13; $376,772
"Analysis of MS Physician Workforce" Understanding issues that discourage doctors from becoming MS neurologists and identifying possible solutions.

There is a growing shortage of physicians specializing in the care of individuals with MS (MS subspecialists). Dr. Halpern is evaluating the root causes of MS physician shortages across the U.S. and in specific regions, in particular urban versus rural settings, with an eye toward developing strategies to reduce current and future shortages. The team is working with an advisory panel of MS subspecialists, general neurologists, and other health care professionals who provide "real-world" clinical experience.

The team is surveying practicing physicians, doctors in training, and others to gather data to assess the current and future adequacy of the MS physician workforce and to better understand barriers and incentives/disincentives to entering the subspecialty of MS. They are also analyzing data from the Sonya Slifka Longitudinal MS Study (a survey of MS patients funded by the National MS Society) to assess the geographic distribution, treatment patterns, and types of physicians and non-physician health care providers treating MS patients

by disease severity.

This study and the resulting recommendations will provide critical information for the Society and other stakeholders to address MS physician shortages and help ensure optimal care for all people with MS.

RESTORING FUNCTION

Myelin's Growth, Injury and Repair

Myelin insulates the wire-like extensions of nerve cells, speeding nerve conduction and protecting the nerve from harm. Because myelin is thought to be the main target of the immune attack that underlies MS, it's vital that we understand its development, function and repair.

The National MS Society has current, multi-year commitments of about $14 million to support research projects focusing on myelin biology in MS.

Roumen Balabanov, MD
Rush University Medical Center
Chicago, IL
Area: Greater Illinois/Midwest
Term/Amount: 10/1/10-9/30/13; $557,137
Paid by special funds by the Illinois Lottery
"Role of IRF-1 in oligodendrocytes" Looking for ways to protect the cells that make nerve-protecting myelin from damage in MS.

MS involves immune system damage to myelin, the fatty material that surrounds and protects nerve fibers (axons) in the central nervous system (CNS: the brain, spinal cord and optic nerves). Oligodendrocytes, the cells that manufacture and maintain myelin in the CNS, are also damaged. Some immune system cells release a chemical known as interferon gamma (IFN-gamma), which may contribute to myelin damage by preventing oligodendrocyte growth and repair of myelin.

In this research project, Dr. Balabanov is investigating how a molecule called interferon regulatory factor 1 (IFR-1), found inside oligodendrocytes, may play a role in the damage caused by IFN-gamma. Preliminary data indicate that suppressing the action of IFR-1 may protect oligodendrocytes from IFN-gamma, and Dr. Balabanov will test this idea using mice that have had their genetic make-up altered so that their oligodendrocytes do not produce IFR-1. The research will compare the severity of the MS-like disease EAE in IFR-1 deficient mice with normal mice to better discern the role of IFR-1 in tissue damage.

This research could lead to new understanding of how IFN-gamma damages oligodendrocytes, and provide a clue for developing new ways to prevent myelin damage in MS.

Ben Barres, MD, PhD
Stanford University Medical Center
Palo Alto, CA
Area: Northern California/West
Term/Amount: 10/1/10-9/30/13; $496,237
"Signaling mechanisms that control the blood-brain barrier" Determining how the protective barrier between the bloodstream and the brain and spinal cord breaks down in MS.

The capillaries, or tiny blood vessels, of the brain have a layer around them called the blood-brain barrier (BBB). By controlling what can move from the blood into the tissue of the brain, the BBB protects the

Up Close: Research Fellow Angela Hahn, PhD

Making sure we attract the best and brightest minds to conduct MS research is a major goal of the National MS Society's research programs. This summer the Society made it possible for dozens of energetic young doctors and scientists to get the training they need to make MS the focus of their careers. Meet one of them.

Angela Hahn, PhD (University of California at San Francisco) is going after myelin – a major target of the immune attack in MS – and it's personal. While in graduate school, Dr. Hahn was diagnosed with multiple sclerosis.

"At the time, my knowledge of neurobiology was limited to what I had learned in a few courses," she says. "What was a topic of interest became a topic of utmost importance." Dr. Hahn is now completing her training through a postdoctoral research fellowship from the National MS Society. Her project focuses on finding a way to rebuild myelin at sites of damage by stimulating oligodendrocytes (myelin-making cells).

Dr. Hahn's emotions about having MS fuel her studies. "As a patient, MS frustrates me," she says. "After decades of research there is no cure, just a handful of treatment options; no drug to repair the damage already inflicted; and no way of knowing what the progression of my illness will be. "As a scientist, MS intrigues me because I can logically separate myself from the "no's" that frustrate me to see the fascinating biological problems behind them."

Jonah Chan, PhD – Dr. Hahn's mentor – says that her emotion will serve her well in these experiments. "Angela possesses great dreams for the future," he says. "She has a vision for the 'big picture' concerning MS research and – more importantly –her life. While most researchers and scientists focus on the details of the experiments, Angela has the unique ability to bring a touch of humanity into scientific research." Dr. Chan is a former fellow himself, whose independent research career was launched with funding from a National MS Society Harry Weaver Neuroscience Award.

Dr. Hahn will spend the majority of her fellowship in the laboratory conducting and designing experiments, learning new techniques of studying brain cells and new microscope technologies. In the short-term, she is learning the ropes of neurobiology, but her long-term goal is to better the lives of people with MS – like herself. "By understanding the mechanisms involved in how oligodendrocytes make myelin, I want to help discover a treatment to repair the damage, and also the physical and emotional stress caused by MS."

brain from many toxic substances. In MS, the BBB breaks down, allowing molecules and the immune system cells that cause tissue damage to move into the brain.

In an attempt to understand what factors contribute to the health and proper functioning of the BBB, a team led by Ben Barres, MD, PhD, has been studying its development in growing animals. Dr. Barres found that pericytes – cells that wrap around small blood vessels – act in concert with the cells of the capillaries and with other brain cells known as astrocytes to form the BBB. In this research project, the team is studying how the signaling mechanisms, or communication pathways, among these cell types are altered to make the BBB "leaky" in EAE, an animal model of MS.

This research project will lead to better understanding of how the BBB breaks down in MS, and may open new avenues for preventing that breakdown to halt MS.

Maria Givogri, PhD
University of Illinois at Chicago
Chicago, IL
Area: Greater Illinois/Midwest
Term/Amount: 10/1/10-9/30/13; $375,507
Paid by special funds by the Illinois Lottery
"Regulation of neurogliogenesis in health and multiple sclerosis by sulfatides"
Studying molecules that limit myelin repair and looking for ways to improve myelin repair in MS.

In MS, the immune system, which normally protects the body from infective agents such as viruses and bacteria, attacks and damages myelin. Myelin is the material that surrounds and protects nerve fibers, and the oligodendrocytes that make and repair myelin can also be lost. Cells that are capable of developing into oligodendrocytes – neural stem cells (NSCs) and oligodendrocyte precursor cells (OPCs) – exist in brain but they fail to produce new oligodendrocytes rapidly enough to keep up with their destruction in MS.

Maria Givogri, PhD, is investigating a group of molecules known as sulfatides. Sulfatides are released from damaged myelin, and Dr. Givogri and colleagues have found that these molecules prevent neural stem cells from making OPCs, and thus reduce the number of oligodendrocytes available to repair myelin. Now Dr. Givogri is looking at how sulfatides act on NSCs in laboratory dishes, and will also determine how prevalent they are in the brains of people with MS.

This research could provide new clues about why myelin repair is deficient in MS and how to improve myelin repair to restore function in people with MS.

Carlos Parras, PhD
INSERM U711
Paris, France
Term/Amount: 10/1/10-9/30/13; $274,423
Paid by the National MS Society South Central Region
"Promoting remyelination from endogenous oligodendrocyte precursors" Studying genes inside the body's spare cells that are capable of maturing into myelin-forming cells to find ways to stimulate nervous system repair in MS.

Myelin, the material that surrounds and protects nerve fibers enabling them to carry nerve signals rapidly, is damaged and destroyed in the central nervous system (CNS: brain, spinal cord and optic nerves) in MS. Myelin is made and maintained in the CNS by cells known as oligodendrocytes. In developing animals, oligodendrocytes are gen-

Because myelin is thought to be the main target of the immune attack in MS, understanding its development, function and repair is vital to reversing the damage

erated by oligodendrocyte precursor cells (OPCs). Some OPCs remain in the adult brain and are able to replace damaged oligodendrocytes, but this repair process fails to keep up with the damage in MS.

Dr. Parras is modifying genes that are active in OPCs in mice to determine how the genes influence the ability of the OPCs to form oligodendrocytes. In addition, he is applying the modified genes to demyelinated mice, to see how they influence the repair of myelin (remyelination).

The results of this research could provide a basis for developing ways to foster myelin repair that would restore functions in people with MS.

David Pleasure, MD
University of California, Davis
Davis, CA
Area: Northern California/West
Term/Amount: 10/1/10-9/30/13; $433,947
"Corticospinal tract degeneration in EAE: role of endolysosomal TLRs" Exploring the role of specific immune reactions in nerve fiber damage and testing ways to block them to protect the nervous system in MS.

While many of the early symptoms of MS are caused by the failure of nerve fibers to carry signals properly after their protective myelin sheath is damaged, long-term progressive disability is caused by the death of nerve cells. The destruction of nerve cells that supply the "corticospinal tract" – essentially a group of nerve fibers, similar to a bundle of wires – running from the brain into the spinal cord, leads to weakness of muscles in the arms and legs.

David Pleasure, MD, is investigating the role of a group of molecules known as "endolysosomal TLRs" found inside nerve cells. These TLRs become active when nerve cells are damaged, and seem to be involved in self-destruction by the damaged cells. Looking at mice with the MS-like model EAE, which has corticospinal tract degeneration similar to that seen in MS, Dr. Pleasure and colleagues are investigating how the TLRs cause the loss of nerve cells, and studying whether drugs that block their action can prevent the loss of nerve cells in MS.

This work has great potential for developing new therapies to prevent the progressive loss of function experienced by many people with MS.

Betty Soliven, MD
University of Chicago
Chicago, IL
Area: Greater Illinois/Midwest
Term/Amount: 10/1/10-9/30/12; $319,778
Paid by special funds by the Illinois Lottery
"Lysosphingolipid receptors and growth factors in oligodendroglial regeneration"
Studying how FTY720, a potential oral treatment for MS, protects against injury and /or promotes repair of myelin.

The first oral disease-modifying therapy for MS, fingolimod (Gilenya) was recently approved by the U.S. FDA for the treatment of relapsing forms of MS. Aside from the effect of fingolimod on inhibiting immune attacks in MS, its actions on glial cells and neurons in the central nervous system (CNS) have also been an area of exciting research. Dr. Soliven and other investigators have found that fingolimod and related agents that act on "sphingosine-1-phosphate (S1P) receptors" regulate the survival, proliferation, and differentiation of cells that make myelin in the CNS. These cells are called oligodendrocytes.

The goal of this research project is to investigate further the CNS effects of fingolimod and other S1P receptor modulators. Her team will study the mechanisms underlying the protective and stimulating action of these compounds on oligodendrocytes and immature oligodendrocytes. They will also examine whether fingolimod exerts any effect on nerve fibers, which would lead to a change in neurological symptoms or in disease progression.

These studies will provide useful information on the potential long-term CNS effects of drugs that act on S1P receptors, which is highly relevant to future therapeutic use of fingolimod in MS.

RESTORING FUNCTION
Nervous System Repair

Decades of research into nerve physiology and the biology of myelin and glial cells that support nerve cells have been laying the groundwork for finding ways to restore normal function in individuals with MS.

The National MS Society has current, multi-year commitments of about $28 million to support research projects focusing on finding ways to repair the nervous system and restore lost function in people with MS.

Cheryl Dreyfus, PhD
University of Medicine and Dentistry of NJ
Piscataway, NJ
Area: NJ Metro/Northeast
Term/Amount: 10/1/10-9/30/13; $448,215
Paid by the National MS Society South Central Region
"The role of glial cell-derived factors in a cuprizone model of MS" Investigating factors that enhance the repair activity of myelin-making cells, for clues to restoring function in people with MS.

In MS, the oligodendrocyte (a cell that produces a supportive and insulating myelin sheath around axons) may become a target of the immune attack and die. Many groups are searching for molecules that reverse this destruction. Recent results indicate that one molecule that may play such a role is a specific stimulator (called ACPD) of a docking site or receptor for a neurotransmitter. This stimulator is being shown to play protective roles in a number of models of brain disease. Importantly, receptors for ACPD are increased in active chronic MS lesions, suggesting that the role of the stimulator may impact the disease and be enhanced follow-

ing injury.

To test the possibility that ACPD may enhance myelin regrowth after injury, Dr. Dreyfus's team treated mice that were subjected to myelin damage with ACPD. They found that ACPD enhanced expression of traits associated with mature oligodendrocytes, and also increased a growth factor known to increase proliferation and differentiation of oligodendrocytes. The team is now following up these preliminary results to explore whether and how ACPD protects against myelin damage and stimulates myelin repair.

They believe that understanding the actions of this small molecule will provide insights to optimize maintenance and repair of oligodendrocytes that deteriorate in MS.

Gareth John, VetMB, PhD
Mount Sinai School of Medicine
New York, NY
Area: New York City-Southern NY/Northeast
Term/Amount: 10/1/10-9/30/13; $448,405
"Reactive astrogliosis regulates blood-brain barrier permeability" Tracing events leading to the breakdown of the barrier that restricts immune cells from entering the brain, to find ways to minimize lesion formation and relapses in MS.

A structure called the blood-brain barrier (BBB) separates the central nervous system – the brain and spinal cord – from the blood and maintains the environment to facilitate nerve impulse transmission. Breakdown of the BBB is an early and prominent event in an MS relapse, allowing entry of immune cells and proteins that exacerbate nervous system function and restrict repair. However, the mechanisms underlying BBB breakdown in people with MS relapses are not fully understood.

Dr. John's team has identified a novel mechanism for BBB breakdown in MS. Cells within the BBB use structures called tight junctions to restrict permeability, and the properties of these junctions are determined by proteins called claudins and occludin. Dr. John has found that a specific molecule called VEGF-A disrupts the activity of these proteins. Now they are testing this possible mechanism in BBB cells isolated in the laboratory and in models of MS-like disease.

The goal of this work is to minimize the breakdown of BBB permeability and prevent relapses in people with MS.

Samia Khoury, MD
Brigham and Women's Hospital
Boston, MA
Area: Greater New England/Northeast
Term/Amount: 10/1/10-9/30/13; $497,521
"Regulation of EAE through the PDL1/PDL2 pathway" Studying signals in immune system cells that control the attack on nerve-insulating myelin, seeking ways to turn off the attack in MS.

MS and one of its animal models, EAE, result from the destruction of myelin – the material that surrounds and protects nerve fibers – in the central nervous system (CNS: brain, spinal cord and optic nerves), and damage to the nerve fibers themselves. In both MS and EAE, the nervous system damage is caused by the immune system. Immune system behavior is complex, because of the large number of cell types and because of the large number of molecules the cells use to communicate with each other and coordinate their activity.

Although many of the communication molecules the immune system uses are released into the blood or tissue fluid, Samia

Khoury, MD, and her colleagues are studying molecules that stay on the surfaces of cells. When these signaling molecules on adjacent cells touch, they activate signals in the "PDL1/PDL2 pathway" inside the cells that "turn off" an immune response.

The results of this research will lead to greater understanding of how immune system activity is controlled, and could provide new ways to turn off the immune system attack in MS.

ENDING MS FOREVER

Seeking Infectious Triggering Factors

Because MS is thought to occur in people whose genes make them susceptible, researchers have been exploring the possibility that viruses or bacteria could act as disease triggers for MS.

The National MS Society has current, multi-year commitments of about $3 million for research projects focusing on specifically on identifying possible infectious triggers. Many immunology projects are closely related to this effort as well.

Byung Kim, PhD
Northwestern University
Chicago, IL
Area: Greater Illinois/Midwest
Term/Amount: 10/1/10-9/30/13; $485,904
"Pathogenic mechanisms of virus-induced demyelinating disease" Studying how viruses can trigger immune attacks against nerve tissue to understand how a virus might trigger MS.

MS involves a mistaken attack by the immune system, which normally protects the body from infections, against myelin, the material that surrounds and protects nerve fibers, in the central nervous system (CNS: brain, spinal cord and optic nerves). Other tissues including nerve fibers and myelin-making cells are also destroyed. Destruction of myelin (demyelination) by the immune attack interferes with the ability of nerve fibers to carry signals properly, leading to the symptoms of MS. It is not known what causes the immune system to attack myelin, but one possibility is that a viral infection may help trigger the attack.

Byung Kim, PhD, is studying mice infected with a virus that can result in demyelination by the immune system, producing symptoms similar to MS. Dr. Kim and colleagues are looking at how viral infection of some cells in the CNS stimulates the development of immune system cells that attack myelin, and are attempting to find ways to reduce or eliminate the cells that cause demyelination.

This research will improve our understanding of how virus infections may trigger MS, and could lead to new avenues to prevent a virus from triggering the disease.

Index of New Research Projects by National MS Society Chapter

Index by National MS Society Region

Lists of all Society-supported research projects are available on our Website at the following link: http://www.nationalmssociety.org/research/research-we-fund/index.aspx

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