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The peripheral nervous system transmits vital communications between the brain, spinal cord, and all other parts of the body. The disorder known as peripheral neuropathy interferes with this critical messaging system, resulting in sensory abnormalities that cause pain, numbness, or motor problems that lead to difficulties with movement or muscle control. While certain conditions may contribute to the development of peripheral neuropathy—diabetes, infections, alcoholism, heredity, and exposure to chemicals and certain medications—often a specific cause is unknown. There are so many different types of peripheral neuropathy that, despite its prevalence, relatively little is known about the disease process, prevention, and the most effective treatments. There is an enormous need to learn more about peripheral nerve biology at the basic level to better understand disease mechanisms and the potential for therapeutic intervention.

Research

The University of Chicago Center for Peripheral Neuropathy was established in May 2001 to increase knowledge of peripheral neuropathy through basic laboratory research. Since its establishment, Center investigators have made significant contributions to understanding the genetics of the disease, the role of the protective myelin sheath surrounding nerves, and the biological pathways that can incur damage and give rise to neurological disease. Center scientists share the ultimate hope that their discoveries will lead the way to more effective therapeutic approaches and preventive strategies for peripheral neuropathy and other neurological conditions.

Peripheral neuropathy is one of many nervous system disorders, and discoveries related to one disease often apply to other neurological conditions. Thus, the Center draws together researchers from other departments, fosters internal and external collaborations, and nurtures the exchange of scientific ideas to hasten discovery of peripheral neuropathy and several neurological disorders. The Center involves investigators from the Departments of Neurology, Pathology, Neurobiology, Pharmacology, Physiology, and Human Genetics, as well as scientists from other institutions.
**Clinical Referrals**

The Center serves as a referral resource for patients with peripheral neuropathy who seek specialized diagnostic and clinical services. Patients are regularly seen at the Duchossois Center for Advanced Medicine, including a twice monthly specialty clinic for neuropathy and neuromuscular disorders. Center-affiliated physician-scientists provide the special tests required to diagnose and distinguish specific types of peripheral neuropathy. Patients who receive care at the University of Chicago Medical Center (UCMC) have access to its vast, integrated network of clinical services and experts.

Patient experiences and patterns of disease progression inform work in the laboratory. Questions that arise in the clinical setting often lead to new hypotheses to be explored in the laboratory. The diagnostic and disease measurements used in the clinical setting are directly applicable to the laboratory studies involving animal models as well.

**Education and Training**

Research training is a natural extension of a vibrant basic scientific research program and an integral part of an academic medical center. As part of the University of Chicago Medical Center, the Center for Peripheral Neuropathy fully supports the educational mission of the Pritzker School of Medicine. Center faculty members participate in a full range of training programs, from teaching the fundamentals of neuroscience to mentoring post-doctoral research trainees. Undergraduate, graduate, and medical students, as well as residents and fellows have opportunities to learn critical laboratory skills and methodologies by participating in research investigations. A number of trainees in the Center for Peripheral Neuropathy are now highly successful, independent investigators.
Peripheral Neuropathy Research at Chicago

The Center’s researchers and physician-scientists are focused on basic, laboratory investigations to find answers in three key areas of peripheral neuropathy research:

**Myelination and Remyelination** – Peripheral neuropathy can result from a breakdown or loss of myelin, the sheet of membrane produced by glial cells that plays an important role in transmitting nerve impulses. Scientists are investigating processes involved in repairing the myelin and exploring how a variety of mechanisms, including the immune response, demyelination, and remyelination, play roles in disease pathology in both the central and peripheral nervous systems. Some recent advances in this area include:

- Discovering that conditions interfering with the endoplasmic reticulum in myelinating cells are involved in the pathogenesis of myelin disorders. This work has implications for both genetic and acquired disorders of the myelin sheath.

- Finding that a mutation in a specific gene blocks the myelination process at a very late stage—reminiscent of myelin disorders in which remyelination fails.

**Genetics** – Genes play a role in the development of all diseases, including peripheral neuropathy. While certain forms of peripheral neuropathy are entirely genetic, genetic studies also help reveal the subtler roles genes play in disease development and the increased likelihood of developing the disorder. Specific recent studies have revealed:

- A molecule associated with the development of diabetes that when eliminated results in the development of peripheral neuropathy. This finding challenges previous thinking that peripheral neuropathy associated with diabetes was a secondary response to high blood sugar. It now seems possible that a metabolic defect could have a direct impact on the peripheral nerve.

- New methods of harnessing the power of forward genetics, a strategy that seeks to identify the gene or genes responsible for a phenotype or specific trait. The approach is important in probing the physiology, genetics, and chain of events that can lead to the development of disorders of the peripheral nervous system.
Discovering New Molecular Pathways of Function and Dysfunction – Genetic analysis can reveal new pathways critical to maintaining normal nerve function, (such that when abnormal conditions or mutations perturb these pathways, disease occurs). Better understanding of these pathways is critical to developing strategies to repair and prevent damage. Chicago investigators have recently found:

- A protein-modifying enzyme that is essential for normal peripheral nerve function. Interestingly, a target of this enzyme has been shown essential for susceptibility to infection by the bacterium that causes leprosy, which is one of the leading causes of peripheral neuropathy worldwide.

- Mutations in the gene for a protein called dynein, required for the proper functioning of sensory nerve cells, can reduce significantly the number of sensory nerve cells and cause locomotion problems in mice—a defect that is similar to some human neuropathies.
Principal Investigators in the Center for Peripheral Neuropathy

Leading the Center’s research efforts is Chicago scientist and Center Director Brian Popko, PhD, an internationally recognized expert in using genetic manipulation to enhance and protect the nervous system. He and his colleagues are looking intensively at neurological disorders that alter the interactions between myelinating glial cells and axons, the fiber-like extension of the nerve cell that makes contact with other cells. Axo-glial interactions play a critical role in forming and maintaining the nervous system, and interruptions in these actions can cause severe neurological dysfunction. For instance, demyelination of the central nervous system can lead to multiple sclerosis and alterations in the peripheral nerve myelin sheath, which underlies peripheral neuropathies such as Guillain-Barre syndrome and Charcot-Marie-Tooth disease.

Popko’s expertise in manipulating the mouse genome with increasing sophistication has resulted in the development of intricate models to support his team’s molecular genetic approach to better understand both normal function and dysfunction of the nervous system. These are also important to other investigators working in collaboration with the Center or independently, who share a commitment to advancing scientific understanding of neurological diseases. The models have enabled the identification of genes responsible for genetic disorders and phenotyping of specific traits. Popko’s ability to activate or inactivate predetermined genes has been critical to analyzing neurological disease processes. In addition to enabling insights into disease processes, the models will be invaluable to designing new therapies for these disorders.

The Importance of Animal Studies

Studies in mice are critical to learning more about disorders associated with the peripheral nervous system. The biological systems in mice have much in common with those in humans. The successful DNA sequencing of both human and mouse genomes has enabled the identification of abnormal and mutant genes associated with specific disorders. Chicago investigators have built rapidly on this knowledge and are leaders in manipulating the mouse genome to develop and duplicate highly specific research models of disease that would not be possible in humans. Similarities in the species increase the potential for discoveries in mice to be translated and applied to humans.
Popko collaborates regularly with other principal investigators affiliated with the Center, including:

- Kourosh Rezania, MD, a physician-scientist who provides care and treatment for patients with peripheral nervous system disorders, including peripheral neuropathy.

- Raymond Roos, MD, a physician-scientist and expert on neurodegenerative diseases. Some of Roos’s research studies focus on genes associated with the death and survival of neurons. In addition to laboratory research, he co-directs a specialized clinic for patients with neuromuscular problems related to motor-neuron disease and neuropathy, and that serves as a resource for clinical trials.

- Betty Soliven, MD, a physician-scientist and neurophysiologist. Soliven has special expertise in nerve conduction velocity and provides electrodiagnostic and clinical care for approximately 1,400 patients a year. Her expertise is invaluable to determining type of neuropathy, degree of degeneration, and disease progression in animal studies. She directs both the Electrodiagnostic Laboratory for Neuromuscular Diseases and the Clinical Neurophysiology Fellowship Program.

- Robert Wollmann, MD, PhD, a neuropathologist with special expertise in peripheral nerve disorders. Wollmann collaborates on many neuropathy studies and directs the neuropathy-training program.

A Commitment to Scientific Collaboration—Within Chicago and Across the Nation

Scientists in the University of Chicago Center for Peripheral Neuropathy collaborate with investigators in other institutions to move research forward more rapidly. For over five years, Director Brian Popko has collaborated in a model program to advance myelin research sponsored by the Myelin Repair Foundation. Popko and three other principal investigators from Stanford University, Northwestern University, and Case Western Reserve University who have common research interests with complementary approaches are working together to accelerate myelenation studies. Members of this virtual dream team were selected for their knowledge, expertise, and contributions to understanding biological processes and interactions that control myelination, as well as for their shared commitment to developing treatments to repair myelin. The team communicates frequently and meets several times a year to share findings, new research techniques and approaches, and to develop new hypotheses.
Training the Next Generation of Investigators

As part of its robust research program, Center investigators train undergraduate, graduate, and medical students, as well as residents and postdoctoral fellows in basic laboratory research techniques and methodologies. Research discoveries are shared through peer-reviewed publications, and Center investigators commonly present findings to scientists, physicians, and young research trainees at national and international academic forums.

The Center promotes the exchange of information and scientific ideas by hosting visiting professorships and arranging visits between members of various research laboratories, both within the University of Chicago and beyond. Some of the visiting scholars include:

- John Bermingham, PhD, from the McLaughlin Research Institute
- Jeffrey Dupree, PhD, of Virginia Commonwealth University
- Michael Shy, MD, from Wayne State University School of Medicine
- Elior Peles, PhD, of the Weizmann Institute of Science
- Stephen Scherer, MD, PhD, of the Institute of Neurological Sciences at the University of Pennsylvania

The Center has hosted two major symposia devoted to peripheral neuropathy. Each of these two-day educational forums drew more than 150 physicians and scientists from across the nation.
Featured speakers for the 2006 symposium included:

- William Snider, PhD, from the University of North Carolina School of Medicine
- Elior Peles, PhD, of the Weizmann Institute of Science
- Clifford Woolf, MD, PhD, from Harvard University
- Jeffrey Milbrandt, MD, PhD, from Washington University School of Medicine
- Rhona Mirsky, PhD, of the University College of London
- Stephen Waxman, MD, PhD, from Yale University School of Medicine

Speakers for the 2004 symposium included:

- David J. Anderson, PhD, from California Institute of Technology
- Ardem Patapoutian, PhD, of the Scripps Research Institute
- Scott Brady, PhD, from the University of Illinois at Chicago
- Stephen Strittmatter, MD, PhD, of Yale University School of Medicine
- James Lupski, MD, PhD of the Baylor College of Medicine
- John Griffin, MD, of Johns Hopkins University School of Medicine

Faculty members also participate in community outreach events designed to educate and help patients with peripheral neuropathy. Three seminars have occurred since the Center’s opening—one at Rush University Medical Center in downtown Chicago and two in the Chicago suburbs—and were well attended by patients and families.

In addition to his scientific presentations at national and international meetings, Director Brian Popko participates in meetings of the Neuropathy Association to discuss the work and research advances made at the Center for Peripheral Neuropathy.

The University of Chicago is known for pioneering investigations that cut across disciplines to gain greater knowledge of disease processes to improve treatment, care, and the quality of life for patients. The Center for Peripheral Neuropathy and its investigators are committed to filling the void in understanding of peripheral neuropathy through its rigorous basic research program. New discoveries of the underlying scientific mechanisms and better understanding of the role of genetics in disease development are critical to developing innovative, more effective approaches to treating patients with peripheral neuropathy.
“Brian was my mentor”

Roumen Balabanov, MD, associate professor of neurology at Rush University Medical Center, treats about 2,000 patients with multiple sclerosis a year, but that is only part of his job. He is also a scientist who aims for his research investigations on nerve demyelination to produce new understanding and, ultimately, improved approaches to treating a range of devastating neurological disorders. He credits the training in the Center for Peripheral Neuropathy and the mentoring of Center Director Brian Popko for enabling him to gain the skills, knowledge, and support to launch his academic career and establish his own laboratory.

Balabanov completed his residency at University of Chicago Medical Center in 2001, followed by a four-year fellowship in neurology that included research in Brian Popko’s laboratory, where he learned critical methodologies and experimental techniques. “Brian was my mentor,” says Balabanov, adding that he was most instrumental in his work and career. Popko helped him gain a prestigious Mentored Scientist Development Award from the National Institute of Neurological Disorders and Stroke, which is designed to prepare outstanding young scientists for research and academic careers focused on neurological disorders. In addition to training and helping him gain his first major grant, Balabanov says Popko introduced him to key investigators in the field. Many of these physicians and scientists refer patients to Balabanov for treatment.

Balabanov joined Rush in 2006 as an assistant professor of neurology and is an independently funded investigator with his own laboratory and a team of four other researchers. In addition to undertaking basic laboratory research and providing clinical care, Balabanov says, “I do a lot of clinical trials.” He recently served as one of the principal investigators on a trial treatment of stem cell transplants in patients with multiple sclerosis.

Though established in his career, Balabanov remains in regular contact with his mentor and continues to learn from him. Last year, Balabanov looked to Popko to help him chair a symposium on myelinating diseases at the American Society for Neurochemistry, where some of the most important investigators in the field were speakers.
The Centers for Neurodegenerative Disease and Repair

In 2006, Christopher Gomez, MD, PhD was recruited to the University of Chicago as Chairman and Albina Y. Surbis Professor of the Department of Neurology. He was returning to his intellectual home, having received his BA, MD, and PhD at Chicago.

It is Dr. Gomez’s vision to mount a broad and powerful attack on neurodegenerative diseases by bringing together the Centers for Neurodegenerative Disease and Repair, among them the Center for Peripheral Neuropathy.

The Centers will leverage Chicago’s research strengths across disciplines to help translate advances in the laboratory to the clinic as quickly as possible. The Centers draw together faculty from multiple centers, all devoted to finding the molecular and cellular causes for diverse neurodegenerative disorders and to developing new interventions. Investigators across the Centers are advancing studies using five primary approaches: molecular pathways, protein folding and fate; genetics; glial biology; and plasticity. This broad and multifaceted approach increases the opportunities for innovative discovery; breakthroughs in one area can often lead to unexpected insights that can be applied to related diseases.

This combination of expertise, spanning from the laboratory to the clinic, sets the stage for a focused and aggressive effort to understand the mechanisms of neurodegenerative disease and to develop new interventions.
The Centers for Neurodegenerative Disease and Repair
Appendix A

Select Publications Generated Through Research at the The University of Chicago Center for Peripheral Neuropathy

Absence of oligodendroglial glucosylceramide synthesis does not result in CNS myelin abnormalities or alter the dysmyelinating phenotype of CGT-deficient mice
Saadat I., Dupree JL, Kilkus J, Han X, Traka M, Proia RL, Dawson G, Popko B.
Glia, March 2010

Myelin maintenance: axonal support required
Popko B.
Nature Neuroscience, March 2010

ZFP191 is required by oligodendrocytes for CNS myelination
Genes and Development, February 2010

Concurrent Lpin1 and Nrcam mouse mutations result in severe peripheral neuropathy with transitory hindlimb paralysis
Douglas DS, Moran JL, Bermingham JR Jr, Chen XJ, Brindley DN, Soliven B, Beier DR, Popko B.
Journal of Neuroscience, September 2009

Fingolimod and related compounds in a spontaneous autoimmune polyneuropathy
Kim HJ, Jung CG, Dukala D, Bae H, Kakazu R, Wollmann R, Soliven B.
Journal of Neuroimmunology, September 2009; Epub., August 2009

Concurrent Lpin1 and Nrcam mouse mutations result in severe peripheral neuropathy with transitory hindlimb paralysis
Douglas DS, Moran JL, Bermingham JR Jr, Chen XJ, Brindley DN, Soliven B, Beier DR, Popko B.
Journal of Neuroscience, September 2009

Phase II trial of CoQ10 for ALS finds insufficient evidence to justify phase III
Annals of Neurology, August 2009
Endoplasmic reticulum stress in disorders of myelinating cells
Lin W, Popko B.
*Nature Neuroscience*, April 2009; Epub March 2009

Terbutaline in myasthenia gravis: a pilot study
Soliven B, Rezania K, Gundogdu B, Harding-Clay B, Oger J, Arnason BG.
*Journal of Neurological Science*, February 2009; Epub., October 2008

Mouse forward genetics in the study of the peripheral nervous system and human peripheral neuropathy
Douglas DS, Popko B.

Targeting of myelin protein zero in a spontaneous autoimmune polyneuropathy
Kim HJ, Jung CG, Jensen MA, Dukala D, Soliven B.
*Journal of Immunology*, December 2008

Nur7 is a nonsense mutation in the mouse aspartoacylase gene that causes spongy degeneration of the CNS
Traka M, Wollmann RL, Cerda SR, Dugas J, Barres BA, Popko B.
*Journal of Neuroscience*, November 2008

Spinal glioma: platelet-derived growth factor B-mediated oncogenesis in the spinal cord
Hitoshi Y, Harris BT, Liu H, Popko B, Israel MA.

Enhanced integrated stress response promotes myelinating oligodendrocyte survival in response to interferon-gamma
Lin W, Kunkler PE, Harding HP, Ron D, Kraig RP, Popko B.
*American Journal of Pathology*, November 2008; Epub., September 2008

Cyclical and dose-dependent responses of adult human mature oligodendrocytes to fingolimod
Miron VE, Hall JA, Kennedy TE, Soliven B, Antel JP.

Epigenetic control of myelin repair
Popko, B
*Nature Neuroscience*, September 2008

An elderly patient with Bickerstaff brainstem encephalitis and transient episodes of brainstem dysfunction
Roos RP, Soliven B, Goldenberg F, Badruddin A, Baron JM.
*Archives of Neurology*, June 2008
Cerebellar development and disease
Millen, K.J. and Gleeson, J.
*Current Opinion in Neurobiology*, 2008

Zic1 and Zic4 proteins regulate zebrafish hindbrain ventricle morphogenesis
Elsen, G.E., Choi, L., Millen, K.J., Grinblat, Y. and Prince, V.E.
*Developmental Biology*, 2008

A subgenomic segment of Theiler's murine encephalomyelitis virus RNA causes demyelination
*Journal of Virology*, June 2008; Epub., April 2008

FTY720 modulates human oligodendrocyte progenitor process extension and survival
Miron VE, Jung CG, Kim HJ, Kennedy TE, Soliven B, Antel JP.
*Annals of Neurology*, January 2008

Proprioceptive sensory neuropathy in mice with a mutation in the cytoplasmic Dynein heavy chain 1 gene
Chen XJ, Levedakou EN, Millen KJ, Wollmann RL, Soliven B, Popko B.
*Journal of Neuroscience*, December 2007

Functional consequences of S1P receptor modulation in rat oligodendrogial lineage cells
Jung CG, Kim HJ, Miron VE, Cook S, Kennedy TE, Foster CA, Antel JP, Soliven B.
*Glia*, December 2007

Reinduction of ErbB2 in astrocytes promotes radial glial progenitor identity in adult cerebral cortex
Ghashghaei HT, Weimer JM, Schmid RS, Yokota Y, McCarthy KD, Popko B, Anton ES.
*Genes and Development*, December 2007

Interferon-gamma-oligodendrocyte interactions in the regulation of experimental autoimmune encephalomyelitis
Balabanov R, Strand K, Goswami R, McMahon E, Begolka W, Miller SD, Popko B.
*Journal of Neuroscience*, February 2007

The integrated stress response prevents demyelination by protecting oligodendrocytes against immune-mediated damage
Lin W, Bailey SL, Ho H, Harding HP, Ron D, Miller SD, Popko B.
*Journal of Clinical Investigation*, February, 2007

A Developmental Classification of Malformations of the Brain Stem
Barkovich, J.A., Millen, K.J. and Dobyns, W.B.
*Annals of Neurology*, 2007
Cilia proteins control cerebellar morphogenesis by promoting granule progenitor pool expansion
Chizhikov, V., Davenport, J., Zhang, Q., Shih, E.K., Cabello, O.A., Fuchs, J., Yoder, B.K., and Millen, K.
Journal of Neuroscience, 2007

Tarlov cysts masquerading as peripheral neuropathy
Baek WS, Rezania K.
Archives of Neurology, 2006

Nmf11 is a novel ENU-induced mutation in the mouse glycine receptor alpha 1 subunit
Traka M, Seburn KL, Popko B.
Mammalian Genome, September 2006; Epub, September 2006

Rewiring enervated: thinking LARGEr than myodystrophy
Levedakou EN, Popko B.
Journal of Neuroscience Research, August 2006

Suppressor of cytokine signaling 1 expression protects oligodendrocytes from the deleterious effects of interferon-gamma
Balabanov R, Strand K, Kemper A, Lee JY, Popko B.
Journal of Neuroscience, May 2006

Interferon-gamma inhibits central nervous system remyelination through a process modulated by endoplasmic reticulum stress
Lin W, Kemper A, Dupree JL, Harding HP, Ron D, Popko B.
Brain, May 2006 May; Epub 2006

Patterns and significance of concomitant central and peripheral inflammatory demyelination
Rezania K, Arnason BG, Soliven B.
Neurological Research, April 2006

Trak1 mutation disrupts GABA(A) receptor homeostasis in hypertonic mice

Roof plate–dependent patterning of the vertebrate dorsal central nervous system
Chizhikov, V. and Millen, K.J
Developmental Biology, 2005

Glial cell line-derived neurotrophic factor-induced signaling in Schwann cells
Iwase T, Jung CG, Bae H, Zhang M, Soliven B.
Journal of Neurochemistry, September 2005; Epub August 2005
Endoplasmic reticulum stress modulates the response of myelinating oligodendrocytes to the immune cytokine interferon-gamma
Lin W, Harding HP, Ron D, Popko B.
*Journal of Cell Biology*, May 2005

Disruption of the mouse *Large* gene in the *enr* and *myd* mutants results in nerve, muscle, and neuromuscular junction defects
Levedakou EN, Chen XJ, Soliven B, Popko B.
*Molecular and Cellular Neuroscience*, April 2005

Myelin repair: developmental myelination redux
Balabanov R, Popko B
*Nature Neuroscience*, March 2005

Inducible production of interferon-gamma in the developing brain causes cerebellar dysplasia with activation of the Sonic hedgehog pathway
Wang J, Lin W, Popko B, Campbell IL.
*Molecular and Cellular Neuroscience*, December 2004

Interferon-gamma induced medulloblastoma in the developing cerebellum
Lin W, Kemper A, McCarthy KD, Pytel P, Wang JP, Campbell IL, Utset MF, Popko B.
*Journal of Neuroscience*, November 2004

Astrocyte-specific overexpression of insulin-like growth factor-I promotes brain overgrowth and glial fibrillary acidic protein expression
Ye P, Popken GJ, Kemper A, McCarthy K, Popko B, D’Ercole AJ.
*Journal of Neuroscience Research*, November 2004

K+ channel blockade impairs remyelination in the cuprizone model
Bacia A, Wollmann R, Soliven B.
*Glia*, November 2004

Oligodendrocytes assist in the maintenance of sodium channel clusters independent of the myelin sheath
Dupree JL, Mason JL, Marcus JR, Stull M, Levinson R, Matsushima GK, Popko B.
*Neuron Glia Biology*, August 2004

Pathogenesis of chronic inflammatory demyelinating polyradiculoneuropathy
Rezania K, Gundogdu B, Soliven B.
*Frontiers in Bioscience*, January 2004

Heterozygous deletion of the linked genes *ZIC1* and *ZIC4* is involved in Dandy-Walker malformation
Grinberg, I., Northrup, H., Ardinger, H., Prasad, C., Dobyns, W.B. and Millen, K.J
*Nature Genetics*, 2004
**Mechanisms of roof plate formation in the vertebrate CNS**
Chizhikov, V. and Millen, K.J.
*Nature Reviews Neuroscience*, 2004

**Control of roof plate development and signaling by Lmx1b in the caudal vertebrate CNS**
Chizhikov, V. and Millen, K.J.
*Journal of Neuroscience*, 2004

**Control of roof plate formation by Lmx1a in the developing spinal cord**
Chizhikov, V. and Millen, K.J. (2004)
*Development*, 2004

**Notch signaling: a rheostat regulating oligodendrocyte differentiation?**
Popko B.
*Developmental Cell*, November 2003

**Nogo-A at CNS paranodes is a ligand of Caspr: possible regulation of K(+) channel localization**
*EMBO Journal*, November 2003

**A rare Cu/Zn superoxide dismutase mutation causing familial amyotrophic lateral sclerosis with variable age of onset, incomplete penetrance and a sensory neuropathy**
Rezania K, Yan J, Dellefave L, Deng HX, Siddique N, Pascuzzi RT, Siddique T, Roos RP.
*Amyotrophic Lateral Sclerosis and Other Motor Neuron Disorders*, September 2003

**Association of TAG-1 with Caspr2 is essential for the molecular organization of juxtaparanodal regions of myelinated fibers**
*Journal of Cell Biology*, September 2003

**Membrane lymphotxin is required for resistance to Theiler’s virus infection**
Lin X, Ma X, Rodriguez M, Feng X, Zoecklein L, Fu YX, Roos RP.
*International Immunology*, August 2003

**An integrated stress response regulates amino acid metabolism and resistance to oxidative stress**
*Molecular Cell*, March 2003
Myelin: not just a conduit for conduction
Popko B.
*Nature Genetics*, March 2003

Nodal sodium channel domain integrity depends on the conformation of the paranodal junction, not on the presence of transverse bands
Rosenbluth J, Dupree JL, Popko B.
*Glia*, February 2003

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) with hypertrophic spinal radiculopathy mimicking neurofibromatosis
Pytel P, Rezania K, Soliven B, Frank J, Wollmann R.
*Acta Neuropathology*, February 2003

PDGF upregulates delayed rectifier via Src family kinases and sphingosine kinase in oligodendroglial progenitors
Soliven B, Ma L, Bae H, Attali B, Sobko A, Iwase T.

Inducible site-specific recombination in myelinating cells
Doerflinger NH, Macklin WB, Popko B.
*Genesis*, January 2003

Galactolipids are molecular determinants of myelin development and axo-glial organization
Marcus J, Popko B.
*Biochemistry Biophysics Acta.*, December 2002

A myelin galactolipid, sulfatide, is essential for maintenance of ion channels on myelinated axon but not essential for initial cluster formation
*Journal of Neuroscience*, August 2002

The neuronal adhesion protein TAG-1 is expressed by Schwann cells and oligodendrocytes and is localized to the juxtaparanodal region of myelinated fibers
Traka M, Dupree JL, Popko B, Karagogeos D.
*Journal of Neuroscience*, April 2002

Molecular markers that identify human astrocytomas and oligodendrogliomas
Popko B, Pearl DK, Walker DM, Comas TC, Baerwald KD, Burger PC, Scheithauer BW, Yates AJ.
*Journal of Neuropathology and Experimental Neurology*, April 2002
Paranodal junction formation and spermatogenesis require sulfoglycolipids
Proceedings of the National Academy of Science, April 2002; Epub March 2002

Myelin-associated glycoprotein and myelin galactolipids stabilize developing axo-glial interactions
Marcus J, Dupree JL, Popko B.
Journal of Cell Biology, February 2002; Epub February 2002

Development of spontaneous autoimmune peripheral polyneuropathy in B7-2-deficient NOD mice
Journal of Experimental Medicine, September 2001; Erratum in: Journal of Experimental Medicine, November 2001

Cytokine-induced cell death in immortalized Schwann cells: roles of nitric oxide and cyclic AMP
Nagano S, Takeda M, Ma L, Soliven B.
Journal of Neurochemistry, June 2001