neuromyelitis optica, optic neuritis, and acute disseminated encephalomyelitis. This study may give us a foothold in understanding all of these disorders; how they may be related to each other, and how they may be distinguished from each other. The benefits from our findings will not only be to those who are paralyzed by TM, but to those who have disabilities due to a variety of autoimmune disorders. We are actively using these findings to aid in developing future diagnostic, prognostic and therapeutic advancements.

Acknowledgments
This research was supported by The Transverse Myelitis Association, the Noel P. Rahn Fellowship, the Dana Foundation, the Miriam and Peter Haas Foundation, the Katie Sandler Fund for Research at Johns Hopkins University, Bruce Downey, and Barr Laboratories and the National Institutes of Health.

Dr. Adam Kaplin receives NIH award for research on depression and cognitive impairment in TM

This year for the first time in its history the National Institute of Health (NIH) funded a research project specifically studying Transverse Myelitis. The title of the study is “Depression and Cognitive Impairment in Transverse Myelitis.” The NIH has allocated $885,354 to this investigation over five years. The principle investigator overseeing this work is Adam Kaplin, MD, PhD, the chief psychiatric consultant at the Johns Hopkins Transverse Myelitis Center (JHTMC) and member of the TMA Medical Advisory Board. He is working in close collaboration with the founder and director of the JHTMC, Douglas Kerr, MD, PhD, who is serving as the principle co-sponsor on this grant. The project seeks to understand the biological basis of depression and memory impairment that are commonly caused by TM. There was great enthusiasm for this work by the NIH grant application review committee, and they noted that the findings from this investigation could significantly expand our ability to diagnose, predict and treat mood and memory difficulties that occur in TM and related autoimmune conditions. Furthermore, the NIH noted that results of this study could help illuminate the underlying cause of all types of clinical depression, not just those found associated with autoimmune diseases such as TM that could result in new and more effective treatments. In addition to the research project, the award will provide resources for the career development of Adam Kaplin. With the recent dwindling of federal allocation of funds to the NIH, this was one of only two such NIH research and career development grants in the Department of Psychiatry awarded this year to the Johns Hopkins School of Medicine.

The Presence of Anti-Ro (SSA) Autoantibodies in Recurrent Transverse Myelitis
Chitra Krishnan, M.H.S.
Research Associate
Johns Hopkins Transverse Myelitis Center

This article was originally published in NEUROLOGY 2004; 62:147–149. Copyright © 2004 by AAN Enterprises, Inc. 147

Hopkins researchers report an association between recurrent Transverse Myelitis (TM) and anti-Ro autoantibodies. The association of this unique clinical phenotype (visible characteristics that result from a combination of genetic and environmental factors) and a specific autoantibody provides circumstantial evidence that an autoimmune process has pathologic (possible explanation of the cause) importance in recurrent TM.

Transverse myelitis (TM) is a rare inflammatory disorder of the spinal cord that can be idiopathic or associated with a specific disease such as systemic lupus erythematosus (SLE), Sjögren syndrome, and antiphospholipid antibody syndrome. Typically TM is monophasic; however, some patients develop recurrent TM without any identifiable associated disease. The study reported an association between anti-Ro antibodies and recurrent TM, which suggests that the mechanism of spinal cord injury may be autoimmune in nature. In this retrospective case-control study, antibodies to 52-kd Ro were demonstrated in 77% of recurrent cases (10/13) compared with only 33% of control subjects (4/12).

In this study, recurrence was defined as more than one episode of TM separated in time with intervening improvement both clinically and radiologically. Patients were excluded if they had evidence of multiple sclerosis (defined as demyelinating lesions on MRI of the brain at presentation or in follow-up). All control cases were diagnosed as either idiopathic monophasic TM (five), idiopathic monophasic myelopathy (four), recurrent transverse myelopathy (one), or disease-associated TM (two). They were also evaluated during intercritical periods at the center.

Anti-Ro (SSA) is the name of an autoantibody in the blood. An autoantibody is a protein that binds to your own tissue/cells. Normal people do not have autoantibodies. The B lymphocytes that make antibodies make them only to foreign substances, like viruses or bacteria, in order to eradicate the infection. In people with an autoimmune disorder, the B lymphocytes make antibodies to self tissue/cells. NMO-IgG is an example of an autoantibody (associated with Neuromyelitis Optica or Devic’s disease).
There are several possible clinical and pathologic implications of the association of anti-Ro antibody and recurrent TM. First, the presence of these antibodies in patients who present with their first episode of TM may be predictive of recurrence. Second, patients with idiopathic TM may have an incomplete expression of a connective tissue disorder. Last, patients with autoantibodies may respond to immunosuppressive therapy, including maintenance therapy to prevent recurrences of TM.

The strengths of this retrospective analysis include a case-control design with a large number of patients with this rare disease. A prospective study of a larger population with recurrent TM is now underway at our center to better define the associated serologic and clinical features of these patients.

**Original Research Paper:**

L.K. Hummers, MD; C. Krishnan, MHS; L. Casciola–Rosen, PhD; A. Rosen, MBChB; S. Morris, MS; J.A. Mahoney, PhD; D.A. Kerr, MD, PhD; and F.M. Wigley, MD. 2004. Recurrent transverse myelitis associates with anti-Ro (SSA) autoantibodies. Neurology; 62: 147-149.

My goal in this article is to provide you with a basic primer on the function and purpose of the brain and the spinal cord and how these organs work together. Once you have an understanding of how the spinal cord works, you can better understand transverse myelitis and how the damage to the spinal cord causes the many different symptoms of this disease.

**The Basic Concepts**

The brain is the most important organ of the body, because it functions to control all parts of the body. The brain plays an important role in every aspect of our activities of daily living. The brain is connected with every structure in the body and generates a lot of information about the body, and at the same time, it receives a tremendous amount of information that is processed by millions and millions of cells that are called neurons. The brain and spinal cord are comprised of neurons and other cells that maintain the function of what we call, collectively, the central nervous system. Neurons are the main center of central nervous system function.

The spinal cord is part of the nervous system and facilitates the interactions between the brain and the rest of the body. The major control system is at the top (the brain) and the spinal cord acts as a bridge, communicating constantly with the brain, receiving and sending information from and to every part of the body. For example, we are able to communicate because our brain is able to generate words, and at the same time, we understand what people are saying, because our brain is processing that information. We are able