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**MEDICAL UPDATE MEMO**  
**MARCH 19, 2004**

**MS SOCIETY-FUNDED RESEARCHERS IDENTIFY  
KEY COMPONENT IN MS-LIKE DISEASE**

**SUMMARY**

Researchers at McGill University Health Centre have identified a key enzyme that triggers MS-like disease in an animal model for multiple sclerosis. They also found that blocking the enzyme may play a critical role in preventing disease development and continued relapses. The Multiple Sclerosis Society of Canada funds the research, which was reported in the February 4, 2004 issue of *Neuron*.

**DETAILS**

Multiple sclerosis is a disease in which the body's own immune system attacks myelin, the insulating substance that surrounds nerve fibres. MS and an MS-like animal model called EAE are thought to result because of attacks by T-cells in the immune system on central nervous system tissue (the brain and spinal cord). How and why this process occurs is not fully understood.

The MS Society funded research carried out by McGill University researchers Dr. Samuel David and his Ph.D. student Athena Kalyvas suggests that a particular enzyme may be a key part of this process, which leads to patchy inflammation (lesions) within the brain and spinal cord. They found that in a mouse model of MS the amount of an enzyme called cytosolic phospholipase A2 (cPLA2) is increased in spinal cord lesions. They also found that when they treated the animals with a chemical inhibitor they were able to block the development of disease in some mice. In addition, those who developed MS-like disease had milder symptoms. In another experiment, they were able to prevent the occurrence of additional relapses in animals in which the disease had already been introduced. While this inhibitor cannot be used in people, it gives researchers valuable information to design drugs that might safely target and block the enzyme in humans.

Dr. David points out that while these findings are very encouraging, he cautions that not all treatments that work in animal models of disease work in humans. Another hurdle is

that in the mouse, the inhibitor worked only with the relapsing-remitting form of disease and not in animals with a progressive form.

The researchers are now working on identifying more specific inhibitors that can block the enzyme. Dr. David noted: “We cannot say how long it will take such treatments to go to clinical trial. What we have discovered is another piece of the puzzle of how the disease process can be triggered within cells in the spinal cord.”

The Multiple Sclerosis Society of Canada has funded Dr. David’s research since 2000.

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National Research Department  
National Communications & Social Action Department

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