A Novel Therapy for the Treatment of Multiple Sclerosis
Technology ID# 628.7

Background
Researchers at the University of Calgary, led by Professor V. Wee Yong, have developed a novel therapeutic antibody for the treatment of multiple sclerosis (MS). The technology prevents the disease-specific entry of leukocytes into the central nervous system (CNS) and ameliorates both behavioral symptoms and neuroinflammation in the mouse experimental autoimmune encephalomyelitis (EAE) MS model. It also attenuates activated T-cell proliferation and neurotoxicity, monocyte adhesion and migration, and MMP-9 production by monocytes. The target protein is seen to be upregulated in the CNS of MS patients.

MS is a complex disease which has no cure. The primary pathology of MS is the loss of the myelin sheath which surrounds and insulates neurons. Evidence has shown that this demyelination is caused by an aberrant immune reaction within the CNS which specifically targets the oligodendrocytes – specialized cells which form the myelin sheath. This process ultimately results in an interruption of the flow of electrical impulses along nerve fibers which in turn produces the various functional deficits that are commonly seen in patients which can include loss of balance, impaired speech, extreme fatigue, double vision, and paralysis. In both MS and EAE, there is a breakdown of the basement membrane surrounding blood vessels which comprise the blood brain barrier which allows leukocytes (white blood cells) to traverse through this structure. Leukocyte trafficking into the brain and spinal cord promotes MS-related neuropathology.

Areas of Application
• A novel therapeutic for the treatment of MS.

Competitive Advantages
• Novel target protein and therapeutic strategy for the treatment of MS.

Stage of Development
• Extensive pre-clinical experiments demonstrate efficacy of clone 10, a therapeutic IgM antibody, in the mouse EAE model of MS.
• Development of efficacious IgG antibodies underway.

Intellectual Property Status
• PCT pending: WO 2009/141736A2
Publications