Market Need: Multiple sclerosis (MS) is an autoimmune disease affecting the protective myelination of the central nervous system (CNS). The age of onset is between the ages 20 and 40. The disease results in debilitating impairments in motor control, speech and thought. Canada has one of the highest rate of MS in the world, followed by the United States while European countries. In the United States, patient costs ranged from $8,528-$54,244 and in 2006, the worldwide market for MS therapeutics was $5.85 billion. Current therapies rely on anti-inflammatory and immunosuppressant agents such as interferon-β, glatiramer acetate, and natalizumab. These medications reduce the rate of relapse during the initial disease stages, and do not prevent disease progression. As these medications are immunosuppressants they induce side effects including flu-like symptoms, dermal reactions, and susceptibility to CNS infections. Furthermore, they do not reduce long-term neurodegeneration. To date, there is no cure for MS and despite treatment progression; the disease remains a significant therapeutic challenge.

Technology Description: Our scientists have identified a protein-protein coupling between the glutamate R2 (GluR2) - α-amino-3-hydroxy-5-methylisoxazole-4-propionic acid (AMPA) receptor and the extracellular protein glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as a novel target for the treatment of MS. By characterizing the protein-protein (GluR2-GAPDH complex) interaction we have developed a peptide that interferes with this coupling, thereby leading to the development of a breakthrough therapy capable of an enhanced affinity, efficacy, and a superior side effect profile.

Stage of Development:
- Our researchers have observed an enhanced GluR2-GAPDH interaction in the autoimmune encephalomyelitis (EAE) preclinical model, a widely accepted preclinical model for studying the clinical and pathological features of MS.
- Systemic delivery of interfering peptide TAT-G-G pep to EAE preclinical models significantly improves neurological outcome.
  - Mitigates neuronal death and increases oligodendrocyte (cells that produce protective myelin) survival in the spinal cord.
  - Reduces axonal damage in the spinal cord of EAE preclinical models.
- We are starting the identification of small molecules mimicking the functional effect of our peptides.
  - Investigators have purchased a high throughput robotics system for automating the screening protocols.
  - We are also currently performing the in silico prediction of novel molecules for biological targets in collaboration with a software development company.

Advantages:
- Novel and highly specific mechanism of action.
- In vitro data - our interference peptide can selectively inhibit the interaction of two functionally distinct neurotransmitter receptors.
- Positive invivo data in the EAE model of MS.
- The interference peptide selectively inhibits the aberrant interaction between GluR2 and GAPDH.
- Does not interfere with normal physiological functions associated with the GluR2 receptor.
- Agents that selectively inhibit highly specific interactions are likely to be safer than receptor antagonists.


Intellectual Property: Patents filed in Canada, United States and Europe.

Business Opportunity: CAMH is seeking a partner to complete pre-clinical development and to launch clinical trials that would employ either peptides or small molecule approaches. We are also interested in testing propriety compounds provided by pharmaceutical companies.