Two main lines of research have been established in my laboratory: acute neuroprotection and long-term stroke recovery. For acute neuroprotective strategies, my laboratory has examined several well known risk factors associated with higher incidence of cerebrovascular diseases and poor outcome. These include hypercholesterolemia, hypertension, diabetes, and obesity, and co-morbid conditions that have not been systematically examined in experimental animal models of stroke. In an effort to narrow the gap between animal models and clinical conditions, we proposed the inclusion of hyperlipidemia in our mouse model of stroke. In addition, close examination of stroke pathology revealed that multiple pro-death processes including inflammation, necrosis, apoptosis, oxidative stress, and vascular dysfunction are involved. Thus, targeting a specific pathway may not overcome the heterogeneous nature of stroke pathology. This led to the concept of a multi-modal approach: targeting a molecule that is involved in multiple pathogenesis, thereby simultaneously mitigating several pro-death pathways. To this end, we proposed that CD36, a class B scavenger receptor, is a potential target molecule for a multi-modal approach. We have been particularly focused on in vivo phenomena and the underlying events by which peripheral inflammatory status influence the acute outcome of stroke-induced injury and potential recovery in chronic stroke through a novel CD36 mechanism.

Recent relevant publications: