Stroke is still a major cause of disability and mortality in the United States and worldwide.

Ischemic stroke occurs when a vessel that delivers oxygen and nutrients to the brain is occluded, resulting in the rapid loss of neurons or brain cells.

Research in ischemic stroke have shown that the actual injury that happens after a stroke is not only due to the occlusion of the vessel, but also due to the response of the immune and inflammatory system in the brain to the actual injury.

This inflammatory response that happens early on after a stroke not only leads to the loss of neurons early, but can propagate for months after a stroke, contributing to ongoing degeneration of neurons.

Therefore, targeting the inflammatory response is currently the new frontier in the treatment of ischemic stroke.

The main challenge is that the inflammatory system cannot be simply inhibited or interrupted after ischemic stroke, and the reason being that this system is involved in the defense against infection as well as other homeostatic functions in the body and the brain.

To overcome this challenge, our lab has pioneered in the development of targeted inhibitors of this inflammatory system.

These inhibitors will only interrupt inflammation locally in the brain and for a transient period of time to prevent any systemic effects and allow the natural homeostatic mechanisms in the brain to occur.

Our research focuses specifically on the complement system, which is the recognition and activation component of the immune system in the body.

And it has a key role that occurs early after injury.

In this work we describe our latest strategy of delivering inhibitors of the complement system specifically to the brain after injury.

The concept behind this strategy is that stressed and dying cells tend to express markers on the surface that indicate these cells are stressed and need to be cleared by the immune system.

However, after stroke, these markers are expressed by cells that are actually stressed and could be potentially salvageable if blood has been restored to the area.

A major advantage of our new treatment strategy is that by a single dose administered early on after a stroke, we

can stop this inflammatory cascade from starting and also from propagating months after injury, preventing this continuous loss of neurons and providing a sustained productive effect in animals, and hopefully, in patients.