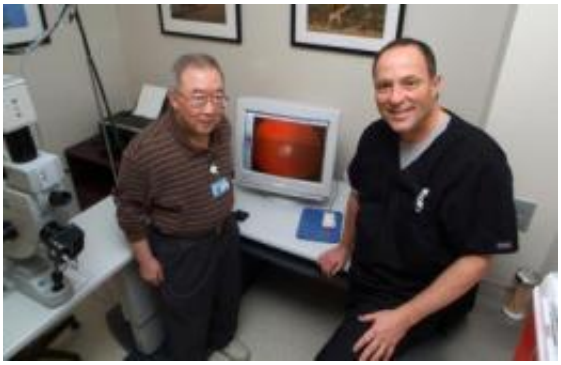
Better treatment sought for blinding traumatic optic nerve damage

April 6 2011



Drs. Gregory Liou and Julian Nussbaum of Georgia Health Sciences University want to find a better way to treat traumatic optic nerve injuries that can occur on roadways or battlefields. Credit: Phil Jones/GHSU photographer

Scientists want to protect the optic nerve when the eye takes a blow on the battlefield or in a car wreck.

It's called traumatic optic nerve damage when the fragile, spaghetti-sized nerve tethered to the back of the eye gets rattled, resulting in swelling and inflammation that can destroy its major component – the retinal ganglion nerves – causing vision loss and blindness.

"The optic nerve is not completely straight so it can tolerate movement of the head or eyes," said Dr. Julian Nussbaum, Chairman of the Department of Ophthalmology at Georgia Health Sciences University.

This extension of the brain also has an insulating myelin sheath and, like the spinal cord, fluid to help protect it.

But as with traumatic brain injury, inflammation and swelling following a concussive injury can still prove deadly.

As with TBIs, few treatment options are available for traumatic optic nerve damage other than high-dose steroids to try to minimize the destructive response. However the nerve's response to these injuries may hold the key to better treatment, said Dr. Gregory Liou, GHSU molecular biologist. He recently received a three-year grant from the U.S. Department of Defense to explore the possibilities.

When an injured nerve cries out for help, the immune system sends pro-inflammatory factors, such as cytokines, to kill off injured neurons and clean up the damage, Liou said. That works pretty well in the peripheral nervous system, the source of nerves for the arms and legs and where nerves can regenerate. But inside the central nervous system, there is little regeneration, Liou said.

To help preserve the balance of Yin and Yang, a nerve injury also produces the energy source adenosine to help combat some of the inflammation. Ironically, inflammation triggers degradation of an enzyme that synthesizes adenosine, the GHSU researchers have found.

"Adenosine is weaker at a time it needs to be stronger," Liou said, noting he wants to strengthen it by giving an agonist that binds to the adenosine receptor so it makes more of the anti-inflammatory substance. "All we are doing is trying to boost the self-defense system that is already there."

He's using a mouse whose ganglion nerves are genetically engineered to glow with exposure to ultraviolet light; they are brightest when healthy, dim when injured and dark when dead. "As the ganglion nerves go, so

goes the optic nerve. If you destroy the ganglion cell layer, the optic nerve dies," Nussbaum said. They will inject the A2AAR-selective agonist into the eye and see how it impacts recovery of traumatic optic nerve injury. They believe it will prevent permanent vision damage and give the nerve time to rest and recover.

Nussbaum hopes the treatment will also block inflammation triggered by acute glaucoma, where sky-high pressures inside the eye compress the optic nerve.

A2AAR-selective agonists are showing promise in inflammation-driven diseases such as diabetic retinopathy and nephropathy as well.

Provided by Georgia Health Sciences University