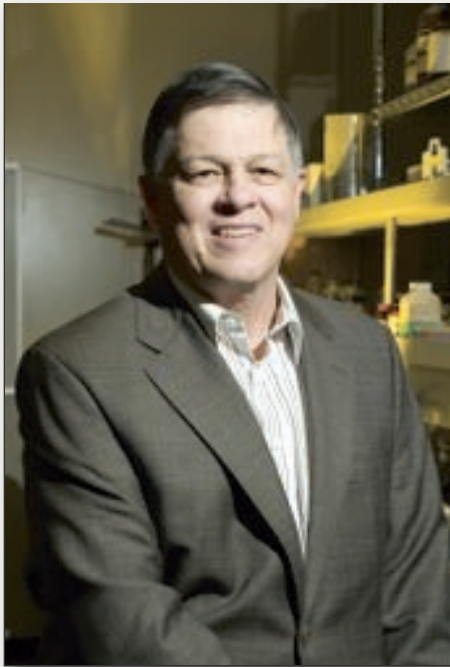


Scientists receive SIU Concept Development Awards



A plan for brain edema

Gregory Brewer, Ph.D., professor of medical microbiology and immunology received a Concept Development Award for his project, "Neuregen for Brain Edema."

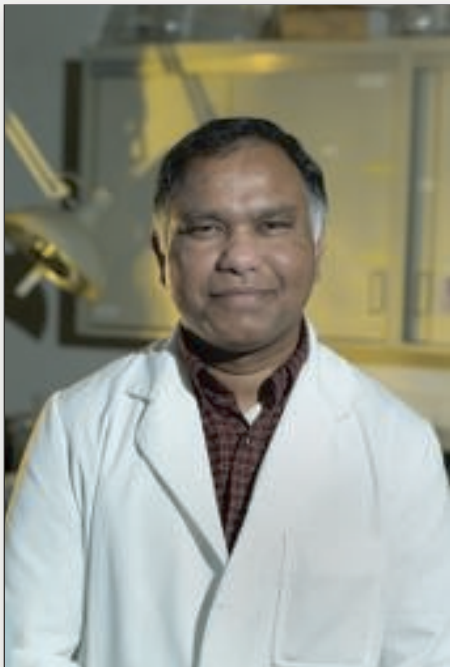
Each year more than 40,000 brain surgeries are performed for tumor resection or for head trauma. During brain surgeries, the incised brain is routinely irrigated with large volumes of normal saline. Any trauma to the brain can cause edema, or swelling, compress capillaries and ventricles, resulting in loss of blood supply, altered brain function and death if untreated.

The standard of care involves administration of glucocorticoids. But glucocorticoids only treat the symptom, rather than addressing the cause of the edema. Doi et al. (2006) determined that irrigation of experimental rat brain lesions with artificial CSF reduced postoperative edema and cellular damage by 50 percent. The aCSF was superior to

lactated ringers (buffered salts) or normal saline. Effects on vascular permeability, measured with Evans blue, showed similar limited improvement.

Dr. Brewer proposes similar studies to evaluate the efficacy of irrigation with Neuregen™, a liquid nutrient medium optimized for neuron growth and survival.

Dr. Brewer previously showed that brain scar tissue and neurodegeneration are virtually eliminated at the edge of an aspiration lesion of rat brain cortex after four weeks with implant of a surgical sponge soaked in Neuregen™. If the cavity and sponge are filled with saline, massive scarring and neurodegeneration occur at the edge of the lesion and extending deep into the brain (Brewer et al., 2003). If successful, Neuregen™ compared to saline will reduce scarring and edema.



Treating drug-induced ototoxicity

Vickram Ramkumar, Ph.D., associate professor in the Department of Pharmacology, received a Concept Development Award for his project, "Short Interfering (si)RNA in the Treatment of Drug-Induced Ototoxicity."

Certain drugs produce permanent hearing loss in humans, such as the platinum-based anti-cancer compounds and aminoglycoside antibiotics. Several reports have shown that these drugs increase free radicals, which damage hair cells in the inner ear, called the cochlea, leading to the hearing loss. Curbing free radical damage is a primary goal in the treatment of drug-induced hearing loss in Dr. Ramkumar's laboratory and those of others at SIU School of Medicine.

Dr. Ramkumar's lab has identified two proteins that contribute to free radical production and cell death in the cochlea: the enzyme NADPH oxidase and the transient receptor potential vanilloid 1 (TRPV1) channel. His studies indicate that free radicals

generated by NADPH oxidase activate TRPV1 channels and that this enhanced activity leads to the death of cochlear hair cells. Knockdown of one or both of these proteins should decrease free radical production, decrease damage to the cochlea, and thereby protect hearing.

A procedure called RNA interference (RNAi) can reduce the levels of these proteins by increasing RNA degradation. His studies have shown that using this procedure against TRPV1 decreases the level of this protein in the rat cochlea, reduces cisplatin-induced damage to the outer hair cells, and thereby reduces cisplatin-induced hearing loss in rats.

These results suggest that the RNAi method, targeting selective proteins for knockdown, may be useful for treating drug-induced hearing loss in humans.