“We don’t know what causes Alzheimer’s Disease (AD), and we don’t know how to treat it,” says Greg Rose, Ph.D., professor of anatomy and physiology, and director of the Center for Integrated Research in Cognitive & Neural Sciences (CIR-CNS) at SIUC. “Current medications work pretty well for approximately 10 percent of the population but are only effective for about a year. We wouldn’t be doing our research if available AD medications worked better.”

According to the American Alzheimer’s Association, this disease is the sixth leading cause of death in the United States. As the population ages and lives longer, experts suggest cases of AD will increase.

Along with aging, diabetes is an important risk factor for developing AD. “Both aging and diabetes have been associated with reduced brain metabolism and cognitive decline, and reduced brain metabolism is a key characteristic of AD,” says Peter R. Patrylo, Ph.D., associate professor of physiology and anatomy at SIU in Carbondale, and a member of CIR-CNS.

It’s widely believed that amyloid, a protein that forms deposits (called senile plaques) in the brain of AD patients, causes the disease. Drs. Rose, Patrylo, and their CIR-CNS colleagues are approaching amyloid from a different perspective. “We don’t know how important amyloid actually is in AD. Many elderly people have amyloid deposits and are cognitively normal,” Dr. Rose explains. Thus, amyloid may be a symptom and not the cause. In support of this idea, the team has discovered that if metabolism in a brain region slows down (becomes hypometabolic), an enzyme that makes amyloid increases, and more plaques develop.

“We are working to understand why people develop such changes in their brains and how this leads to cognitive decline,” say the researchers. Numerous studies are under way at SIU to begin to understand how changes in peripheral and brain metabolism may contribute to cognitive impairment and dementia. Collaborators include CIR-CNS colleagues Gregory Brewer, Ph.D., April Strader, Ph.D., Robert Struble, Ph.D., and Jesse Trushenski, Ph.D. from the SIU Dept. of Fisheries and Xiaxin Yan, Ph.D., a former faculty member of the anatomy department.

The group aims to understand how the body regulates energy, including blood sugar and fatty acid levels, during aging. “We’ve observed changes in brain fat composition in our studies,” Dr. Rose explains. “A decline in the good types of fats can contribute to memory problems.” Some of their rodent models include transgenic “Alzheimer mice” that have genes taken from families that have a high incidence of AD.

An estimated 1-3 percent of the U.S. population develops AD. But many more Americans are at risk for Type 2 (adult onset) diabetes, a condition that prevents the body and brain from properly providing sugar to cells. Emerging data suggest that diabetics are at increased risk for developing AD or dementia. “The diabetic AD mouse model may give us insight into what’s making the disease advance,” Dr. Rose says. Diabetes is treated with insulin or with drugs that help insulin receptor function. In a promising development, Dr. Suzanne Craft and her colleagues at the University of Washington in Seattle have demonstrated that intranasal insulin delivery had helpful effects on memory and brain metabolism in AD patients.

“The brain’s function is compromised during conditions of reduced sugar,” Dr. Patrylo says. “Our ongoing studies suggest that diabetic-like characteristics can be detected months prior to amyloid deposition and severe cognitive decline.”

These SIU researchers continue their studies into the mechanisms of how sugar and fatty acids affect the aging brain. “We need to fundamentally understand what’s going on in order to develop superior treatments,” says Dr. Rose.