

UWM Psychology Researcher Supports a Graduate's Biotech Startup

MILWAUKEE - What are the odds that an alumnus of UWM's psychology department would return years after graduation looking for research support for his biotech business venture? And how likely would it be that he would find a near perfect match for his project?

Mark Underwood (BS '96) can't believe his good fortune. Underwood and his partner, Richfield businessman Mike Beaman, have recently launched QRG Bioscience, a new company that is producing a compound containing aequorin - a protein found in jellyfish - as a therapeutic drug to inhibit neurodegenerative diseases.

Underwood zeroed in on aequorin in college after reading an article that linked the stings of jellyfish with the symptoms of multiple sclerosis, a disease of the central nervous system that his mother has.

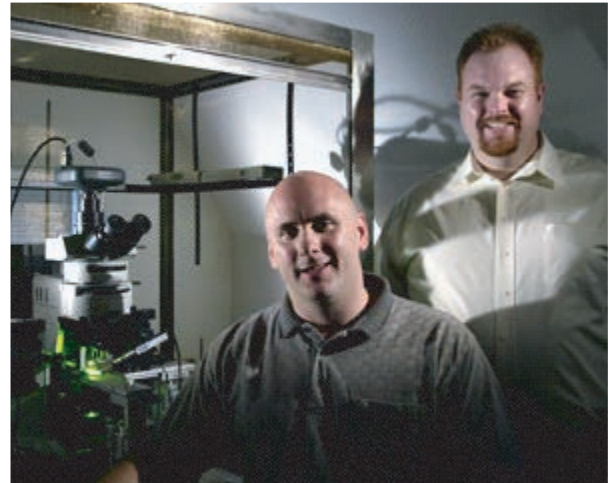
To help the businessmen test their compound, he enlisted Assistant Professor James Moyer, Jr., who joined the Psychology Department faculty in 2003. "Not only was he the closest to home, he was also the perfect choice," said Underwood.

Moyer, like Underwood, is interested in the "calcium hypothesis of aging and dementia," which is just one of many theories that attempts to explain what is going on in neuron degeneration.

Moyer studies how the brain changes as a result of aging. Specifically, he is interested in the part of the brain called the hippocampus, which is responsible for forming new memories. These capabilities not only deteriorate in neurodegenerative disorders such as Alzheimer's disease, but they also become impaired simply by aging.

Calcium is necessary for communication between neurons in the brain, and learning and memory are not possible without it. But too much calcium leads to neuron death, interfering with memory and contributing to neurodegenerative diseases.

Why this happens is the subject of Moyer's research.



Assistant Professor James Moyer Jr. (left) and psychology alumnus Mark Underwood examine the affect of aequorin on animal memory in Moyer's Garland Hall lab.

Photo by Alan Magayne-Roshak

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As we age, the number of ion channels, or "doors," that allow calcium ions to enter the cells increases, says Moyer. At the same time, the brain's ability to control or handle the amount of calcium "allowed in" declines with age.

"There are ways in which cells control the influx of calcium, such as sequestering it by binding it with certain proteins," says Moyer. "If it weren't for calcium-binding proteins, the high level of calcium would overwhelm the neuron and trigger a cascade of events ultimately leading to cell death. With advancing age there is a loss of some of these binding proteins, so we're going to attempt to replenish them in aged neurons with the aequorin."

Moyer says Underwood's idea makes sense. Now he will need to determine what the compound will do.

The researcher's role will be to evaluate the exact cognitive benefits and record any long-term effects. He will also answer questions like, "What is an effective dosage and how long will it last?"

Aequorin was discovered in the 1960s and has been used in research for a long time as an indicator of calcium. But the protein has never been tried as a treatment to control calcium levels.

Moyer, who came to UWM from a post-doctoral position at Yale University, performs Pavlovian trace conditioning experiments to evaluate aging-related learning and memory deficits. These tasks first teach rodents to associate one stimulus with another and then test their memory of the association. During training, the stimuli are separated by a brief period of time, which requires the animal to maintain a memory of the first stimulus. The "stimulus free" period makes the task more difficult, especially for older animals.

Moyer's work also has implications outside of disease. He is able to show that at middle age, when the animal's learning ability or memory is not yet impaired, it already shows a drop in the number of neurons that contain an important calcium-binding protein.

"That cellular changes precede memory deficits indicates there is a window of opportunity for intervention before it's too late," he says. "Once the cells are lost, there is little chance of regaining normal brain function."

Underwood believes he and his partner are at about the 10-year mark in the typical 15-year cycle for a new drug to be developed. Their compound is currently being genetically reproduced in bacteria at UW-Madison, where QRG Bioscience is headquartered.

"We'd like to keep Dr. Moyer's lab busy until he retires," says Underwood. "There's certainly enough work there."