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UAMS Scientist Shows Brain Sugar Malfunction in Alzheimer's, Gains National Attention

LITTLE ROCK — Steven Barger, Ph.D. — a researcher at the University of Arkansas for Medical Sciences (UAMS) — has entered the national spotlight for showing how the mechanism that brings sugar to the brain malfunctions in people with Alzheimer's disease, highlighting a potential target for treatment.

The study also explains why Alzheimer's can mimic the symptoms of diabetes, and ultimately concludes there is not as strong a connection between the two diseases as previously believed. The work also deepens the understanding of how the brain uses sugar, or glucose, to fuel important mental functions and memory.

Barger is a professor with appointments in the Geriatrics, Neurobiology and Developmental Sciences, and Internal Medicine departments in the UAMS College of Medicine. His work was featured during a news conference hosted by the Society for Neuroscience at its annual meeting in Chicago. The findings were featured in *Forbes*, the American Association for the Advancement of Science EurekAlert!, and other outlets such as dLife.

Researchers have long tried to explain the apparent connection between Type 2 diabetes and Alzheimer's disease. A diabetes diagnosis doubles a person's risk of developing dementia. Memory loss and other symptoms of dementia are common among older adults with diabetes. However, Alzheimer's is just one form of dementia, and it includes other unique symptoms that diabetes does not. Notably, brain samples of diabetics do not show excessive beta-amyloid peptide, which is a hallmark of Alzheimer's.

These observations have led scientists to question whether diabetes leads to Alzheimer's or if the converse might be true. Barger's research began as an exploration of whether Alzheimer's might cause some cases of diabetes.

Barger looked at Alzheimer's brains and discovered a very specific process at work. He focused on a protein called glucose transporter 1 (GLUT1), which takes glucose from the blood vessels to the neurons in the brain, and found that beta-amyloid peptide can cause GLUT1 to be defective. The result is that the brain does not get enough glucose, and excess glucose backs up in the blood, mimicking diabetes.

“Our model indicated that, rather than diabetes, people with Alzheimer’s may have higher glucose in their blood simply because they are not transporting as much into the brain, and this may be responsible for the problems with the high blood sugar levels common to Alzheimer’s,” Barger said.

Barger suggests further research could focus on finding ways to ensure the brain continues to get glucose by targeting GLUT1.

“Multiple therapies that reduce brain levels of beta-amyloid have failed in Alzheimer’s treatment trials in recent years, perhaps because it is nearly impossible to remove the beta-amyloid fast enough, before it has kicked off its deadly chain reaction that causes the debilitating symptoms of Alzheimer’s,” Barger said. “Interfering with later events, further down the chain, may be the best hope for preventing dementia. If impaired glucose delivery is a key element of this chain reaction, it may be fruitful to reinforce the sugar bucket brigade.”

Barger’s work coincided with emerging studies by other scientists who have looked at diabetes brain samples and found that the cognitive symptoms of diabetes are related to what is called “vascular dementia,” which results from damage to blood vessels. High blood pressure and arterial plaque are common complications of Type 2 diabetes.

“The two diseases appear to be quite distinct,” Barger said.

Barger’s abstract was one of about 50 to be highlighted at the Society for Neuroscience news conference out of more than 14,000 submitted.

The project included UAMS colleagues Antiño Allen, Ph.D., in the College of Pharmacy; Gwen Childs, Ph.D., and Angela Odle, Ph.D., in Neurobiology and Developmental Sciences; Yang Ou, Ph.D., a research associate in Geriatrics; graduate student Jakeira Davis; and former graduate student Rachel Hendrix, Ph.D.

UAMS is the state’s only health sciences university, with colleges of Medicine, Nursing, Pharmacy, Health Professions and Public Health; a graduate school; hospital; a main campus in Little Rock; a Northwest Arkansas regional campus in Fayetteville; a statewide network of regional campuses; and seven institutes: the Winthrop P. Rockefeller Cancer Institute, Jackson T. Stephens Spine & Neurosciences Institute, Harvey & Bernice Jones Eye Institute, Psychiatric Research Institute, Donald W. Reynolds Institute on Aging, Translational Research Institute and Institute for Digital Health & Innovation. UAMS includes UAMS Health, a statewide health system that encompasses all of UAMS’ clinical enterprise including its hospital, regional clinics and clinics it operates or staffs in cooperation with other providers. UAMS is the only adult Level 1 trauma center in the state. *U.S. News & World Report* named UAMS Medical Center the state’s Best Hospital; ranked its ear, nose and throat program among the top 50 nationwide; and named six areas as high performing — cancer, colon cancer surgery, heart failure, hip replacement, knee replacement and lung cancer surgery. UAMS has 2,727 students, 870 medical residents and five dental residents. It is the state’s largest public employer with more than 10,000 employees, including 1,200 physicians who provide care to patients at UAMS, its regional campuses, Arkansas Children’s Hospital, the VA Medical Center and Baptist Health. Visit www.uams.edu or www.uamshealth.com. Find us on Facebook, Twitter, YouTube or Instagram.

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