Biophysical Society

Biophysical Society 61st Meeting, Feb. 11-15, 2017, New Orleans, Louisiana

Imbalance of Calcium in a Cell's Energy Factory May Drive Alzheimer's Disease

New research could lead to treatments for preventing or delaying neurodegenerative diseases.

EMBARGOED for release until 2:45 p.m. Eastern Time on Tuesday, February 14, 2017

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WASHINGTON, D.C., February 14, 2017 -- Calcium in the mitochondria -- the energy factory of cells -- may be one of the keys to understanding and treating Alzheimer's disease and dementia. Researchers at the Center for Translational Medicine at Temple University have now identified how an imbalance of calcium ions in the mitochondria may contribute to cell death and, specifically, neurodegeneration in brain cells during Alzheimer's and dementia. The findings could eventually point to new therapies for preventing or delaying these diseases. The team will present its work during the 61st Meeting of the Biophysical Society held Feb. 11-15, 2017 in New Orleans.

In the mitochondria of a neuron, calcium ions are thought to control the production of energy needed for the brain to function. But if there's too much calcium -- as has been suggested to occur in Alzheimer’s disease -- it can cause cells to die. Previous studies have suggested that an imbalance of calcium in neurons might play an important role in the onset of Alzheimer's disease. But how this was linked with mitochondrial dysfunction and neurodegeneration was unclear.

New research led by Pooja Jadiya, a postdoctoral fellow working in John Elrod's lab at Temple University, found that one possible mechanism of Alzheimer's disease involves the removal of calcium from mitochondria. Calcium ions exit a neuron's mitochondria with the help of a transporter protein called the mitochondrial sodium/calcium exchanger.

The researchers studied samples of human brains taken from Alzheimer's patients. In these diseased tissues, they found that the levels of this exchanger were so low, they were barely detectable. Such low levels caused calcium to build up in the diseased mitochondria. They hypothesized that this may trigger excessive production of reactive oxygen species, molecules known to wreak havoc in the cell and contribute to neurodegeneration. The researchers also found that the reduced activity of this exchanger was associated with impaired energy production and increased cell death, which may contribute to the neurodegeneration that causes Alzheimer's disease.

When the researchers studied mice that were genetically altered to develop Alzheimer's, they found that before the onset of the disease, the gene that encodes the exchanger protein was much less active -- suggesting that a drop in the gene's expression might contribute to disease progression.

To probe this mechanism further, the researchers studied this gene in an Alzheimer's cell culture model. Like the mice, the cells were genetically altered to exhibit the cellular symptoms of Alzheimer's. When the researchers genetically boosted the levels of the mitochondrial sodium-calcium exchanger, the diseased cells recovered and were nearly identical to the control healthy cells. Production of ATP increased, reactive oxygen species decreased, and fewer cells died.

"No one's ever looked at this before using these model systems," Elrod said. "It's possible that alterations in mitochondrial calcium exchange may be driving the disease process."

The researchers, including collaborators from the lab of Domenico Praticò, also at Temple University, are now trying to see if they can reverse the development of Alzheimer's in mutant mouse models by ramping up the gene that encodes the sodium-calcium exchanger. If they can, then this mechanism could eventually be the basis for new treatments aimed at boosting the function of the mitochondrial sodium-calcium exchanger, using approaches like new drugs or gene therapy.

"Our hope is that if we can change either the expression level or the activity of this exchanger, it could be a viable therapy to use early on to perhaps impede Alzheimer's disease development -- that's the home run," Elrod said. "We're not even close to that, but that would be the idea."

Session 184.29 - Posters: Molecular and Cellular Neuroscience 2186-Pos/B506 - "Genetic rescue of mitochondrial calcium efflux in alzheimer's disease preserves mitochondrial function and protects against neuronal cell death," is authored by Pooja Jadiya, Alyssa A. Lombardi, Jonathan P. Lambert, Timothy S. Luongo, Jin Chu, Domenico Praticò and John W. Elrod. It will be at 1:45-3:45 p.m. Central Time on Tuesday, Feb. 14, 2017 in Hall B-2 & C of the Ernest N. Morial Convention Center.

ABSTRACT: http://www.abstractsonline.com/pp8/#!/4279/presentation/1268

MORE MEETING INFORMATION

ABOUT THE MEETING

Each year, the Biophysical Society Annual Meeting brings together more than 6,000 researchers working in the multidisciplinary fields representing biophysics. With more than 3,600 poster presentations, over 200 exhibits, and more than 20 symposia, the BPS Annual Meeting is the largest meeting of biophysicists in the world. Despite its size, the meeting retains its small-meeting flavor through its subgroup symposia, platform sessions, social activities and committee programs. The 61st Annual Meeting will be held at Ernest N. Morial Convention Center in New Orleans, Louisiana.

PRESS REGISTRATION

The Biophysical Society invites professional journalists, freelance science writers and public information officers to attend its Annual Meeting free of charge. For press registration, contact Ellen Weiss at EWeiss@biophysics.org or the Media Line at the American Institute of Physics at media@aip.org or 301-209-3090.

NEWS RELEASES

Embargoed press releases describing in detail some of the breakthroughs to be discussed at the meeting are available on Newswise and Alpha Galileo or by contacting the Media Line at the American Institute of Physics at media@aip.org or 301-209-3090.

QUICK LINKS

Main Meeting Page: http://www.biophysics.org/2017meeting/Home/tabid/6672/Default.aspx
Symposia:
http://www.biophysics.org/2017meeting/Program/ScientificSessions/Symposia/tabid/6756/Default.aspx
Desktop planner: http://www.abstractsonline.com/pp8/#!/4279

ABOUT THE SOCIETY

The Biophysical Society, founded in 1958, is a professional, scientific Society established to encourage development and dissemination of knowledge in biophysics. The Society promotes growth in this expanding field through its annual meeting, monthly journal, and committee and outreach activities. Its 9,000 members are located throughout the U.S. and the world, where they teach and conduct research in colleges, universities, laboratories, government agencies, and industry. For more information on the Society, or the 2017 Annual Meeting, visit http://www.biophysics.org.

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