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Parkinson's Disease Research Update for Veterans

February 17, 2011 posted by Veterans Today

Parkinson's Disease

Parkinson's Disease has been an issue of concern for Vietnam Veterans but the concern on pesticide (organophosphates) exposure and Gulf War Veterans should be tracked closely for rate of occurrence in the next generation of veterans. Developments in this research also highlights the need to examine mitrochrondial damage in the Gulf war veterans. Mitrochrondial damage mechanisms in neuromuscular disorders has to be continued and research in this area needs to continue.

Below are the summaries of two new research findings this week. In addition web resources for veterans with Parkinson's Disease are listed following the articles. Again these are areas that veterans might consider helping to distribute to other veterans, to healthcare providers, and others as appropriate.

We at Veterans Today welcome comments from the Gulf War Veterans of 1990-91 and are interested in following up with those veterans that are having neuromuscular disorders and their insights. Data is really needed in relationship to other diagnosed illnesses occurring within the Gulf War 1990-91 Veterans. It is through the pressure to get public data on veterans that we can get attention paid nationally and internationally on the illnesses of this next generation of veterans that needs the nation's attention in a more timely manner. Twenty years and data needs to be shared more readily with all veterans with the newer and faster communication systems that are now available.

An Early Step In Parkinson's Disease: Problems With Mitochondri

For the last several years, neurologists have been probing a connection between Parkinson's disease and problems with mitochondria, the miniature power plants of the cell.

Toxins that mimic Parkinson's effects act specifically to poison mitochondria, and mitochondria appear to be damaged in the brain cells that are endangered in the disease. But one unresolved question has been: are mitochondria simply the vulnerable "canaries in the coal mine" or is their deterioration a key step on the way to neurodegeneration?

Now researchers at Emory University School of Medicine have found that a protein called MEF2D, which helps brain cells withstand stress and toxins, also plays an unexpected role inside mitochondria. MEF2D's ability to keep mitochondria well tuned appears to be especially sensitive to impairment in Parkinson's disease, the research team found. The results will be published online in the *Journal of Clinical Investigation.*

"Our data suggest that problems with MEF2D in mitochondria could represent one of the earlier steps in the progress of the disease," says senior author Zixu Mao, PhD, associate professor of pharmacology and neurology at Emory University School of Medicine. Postdoctoral researcher Hua She, PhD, was the first author.

The Emory team showed that MEF2D binds one particular mitochondrial gene, ND6, which is necessary for assembly of complex I. Complex I begins the electron transport process that is necessary for mitochondria to function.

Mitochondria are thought to have evolved from bacteria that once lived independently, but were engulfed and harnessed by a primitive cell millions of years ago. Mao and his colleagues found an example of how this symbiosis has extended to having proteins like MEF2D turn on genes inside mitochondria.

"Our findings make a convincing and very intriguing case that dysregulation of mitochondrial DNA gene expression contributes to Parkinson's," Mao says.

Genes in the nucleus (that is, outside mitochondria) now encode most of the proteins that go into mitochondria. However, mitochondria still make a few of their own proteins, such as ND6.

In addition to showing how MEF2D functions in mitochondria, the team showed that toxins such as MPTP and the natural pesticide rotenone, which interfere with complex I and bring on Parkinson's in animals, also block MEF2D from working in mitochondria.

Mao's laboratory's previous research found that in Parkinson's, MEF2D levels are increased in the cell because of defects in a recycling process called autophagy. Now, they show that in the brains of Parkinson's patients, even when MEF2D levels are increased in the cell as a whole, they are reduced in mitochondria.

Because disruptions in mitochondria have been linked to other neurodegenerative diseases and heart disease as well, Mao says probing MEF2D's involvement in those disease processes may yield new insights.

NIH Study Finds Two Pesticides Associated With Parkinson's Disease

New research shows a link between use of two pesticides, rotenone and paraquat, and Parkinson's disease. People who used either pesticide developed Parkinson's disease approximately 2.5 times more often than non-users.

The study was a collaborative effort conducted by researchers at the National Institute of Environmental Health Sciences (NIEHS), which is part of the National Institutes of Health, and the Parkinson's Institute and Clinical Center in Sunnyvale, Calif.

"Rotenone directly inhibits the function of the mitochondria, the structure responsible for making energy in the cell," said Freya Kamel, Ph.D., a researcher in the intramural program at NIEHS and co-author of the paper appearing online in the journal Environmental Health Perspectives. "Paraquat increases production of certain oxygen derivatives that may harm cellular structures. People who used these pesticides or others with a similar mechanism of action were more likely to develop Parkinson's disease.

The authors studied 110 people with Parkinson's disease and 358 matched controls from the Farming and Movement Evaluation (FAME) Study to investigate the relationship between Parkinson's disease and exposure to pesticides or other agents that are toxic to nervous tissue.

FAME is a case-control study that is part of the larger Agricultural Health Study, a study of farming and health in approximately 90,000 licensed pesticide applicators and their spouses. The investigators diagnosed Parkinson's disease by agreement of movement disorder specialists and assessed the lifelong use of pesticides using detailed interviews.

There are no home garden or residential uses for either paraquat or rotenone currently registered. Paraquat use has long been restricted to certified applicators, largely due to concerns based on studies of animal models of Parkinson's disease. Use of rotenone as a pesticide to kill invasive fish species is currently the only allowable use of this pesticide.

These findings help us to understand the biologic changes underlying Parkinson's disease. This may have important implications for the treatment and ultimately the prevention of Parkinson's disease," said Caroline Tanner, M.D., Ph.D., clinical research director of the Parkinson's Institute and Clinical Center, and lead author of the article.

http://www.veteranstoday.com/2011/02/17/parkinsons-disease-research-update-for-veterans/