New research reports new method to protect brain cells from diseases like Alzheimer's

August 20 2008

New research led by Chu Chen, PhD, Associate Professor of Neuroscience at LSU Health Sciences Center New Orleans, provides evidence that one of the only naturally occurring fatty acids in the brain that has the ability to interact with the receptors originally identified as the targets of THC (the psychoactive component of marijuana) can help to protect brain cells from neurodegenerative diseases like Alzheimer's and Parkinson's.

Published in the August 15, 2008 issue of the *Journal of Biological Chemistry*, the research focuses on the cellular and molecular mechanisms of inflammation, specifically the role these relatively recently discovered endogenous cannabinoids can play in the control of COX-2 and other cyclooxygenases.

COX-2 is a key player in neuroinflammation and has been implicated in the development of neurodegenerative diseases and worsening of damage from such insults as traumatic brain injury and stroke.

Chen and research associate Jian Zhang show that endocannabinoid 2-arachidonoylglycerol (2-AG) functions as an endogenous COX-2 inhibitor, turning off the production of COX-2 which normally goes into overdrive in response to pro-inflammatory and certain types of toxic stimuli, resulting in the injury or death of brain cells.

The researchers also revealed the specific signaling pathways that regulate the 2-AG suppression of COX-2. The paper, Endocannabinoid

2-Arachidonoylglycerol Protects Neurons by Limiting COX-2 Elevation, is available online at www.jbc.org .

"Our findings provide a basis for opening up new therapeutic approaches to protect neurons from inflammation and toxicity-induced neurodegeneration," notes Chen. "Selective COX-2 inhibitors were thought to be a promising medicine in treating neurodegenerative diseases, stroke, cancers and inflammation-related diseases like arthritis; however, the occurrence of a series of cardiovascular complications in patients receiving COX-2 inhibitors has led to their recent withdrawal from the market and limits on their usages. Our research has shown that the use of endogenous cannabinoid 2-AG may avoid such side effects. Therefore, elevation of endogenous 2-AG levels by facilitating its production, inhibiting its decomposition, or directly supplying 2-AG may result in treatment advances to prevent the devastation of disorders like stroke, Alzheimer's and traumatic brain injury."

Source: Louisiana State University