

# Stowers researcher answers fundamental question of cell death

January 10 2005

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Chunying Du, Ph.D., Assistant Investigator at the Stowers Institute, has published findings that reveal a previously unknown pathway of Bruce, the gene encoding a protein that inhibits apoptosis, or programmed cell death.

Bruce has long been recognized as an inhibitor of apoptosis, but until now, its method of inhibition was not clear. Dr. Du analyzed Bruce mutant mice and found that Bruce regulates p53, a tumor suppressor gene, and the mitochondrial pathway of apoptosis.

Bruce's primary function resides upstream of mitochondria. Loss of function of Bruce increases the level of p53, making cells more sensitive to apoptosis. The transcriptional activity of p53 is responsible for the activation of genes including Pidd, Bax, and Bak. These in turn activate mitochondria, leading to apoptosis.

"The identification of Bruce as a regulator of p53 raises the possibility that therapeutic inactivation of Bruce activity could keep p53 levels high to combat certain tumors," said Dr. Du. "On the other hand, over expression of Bruce may help maintain cell survival in neurodegenerative diseases such as Alzheimer's disease." "Dr. Du's findings answer a fundamental question of apoptosis and have implications for a wide variety of diseases," says Robb Krumlauf, Ph.D., Scientific Director of the Stowers Institute. "These findings are an example of the broad impact of basic research conducted at the Stowers Institute."

Dr. Du joined the Stowers Institute in 2001. She holds B.S. and M.S. degrees from Beijing Normal University, and a Ph.D. from Iowa State University. From 1998 to 2001, she was a Howard Hughes Medical Institute Postdoctoral Fellow with Dr. Xiaodong Wang at the University of Texas Southwestern Medical Center. She currently holds a secondary appointment as an Assistant Professor of Biochemistry and Molecular Biology at the University of Kansas School of Medicine.

The findings are available online at [www.pnas.org/cgi/reprint/0408744102v1](http://www.pnas.org/cgi/reprint/0408744102v1) and will be published in the Jan. 18 issue of Proceedings of the National Academy of Sciences (PNAS).

Source: Stowers Institute for Medical Research

Citation: Stowers researcher answers fundamental question of cell death (2005, January 10)  
retrieved 24 April 2024 from  
<https://phys.org/news/2005-01-stowers-fundamental-cell-death.html>

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