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Researchers discover powerful defense against free radicals that cause aging, disease

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Free radicals cause cell damage and death, aging and disease, and scientists have sought new ways to repel them for years.

Now, a new University of Michigan study outlines the discovery of a protein that acts as a powerful protectant against free radicals. Ironically, the protein is activated by excessive free radicals. Human mutations of the gene for this protein are previously known to cause a rare, neurodegenerative disease.

Lysosomes, which comprise the cell's recycling center, are crucial for cleaning up injured and dying parts of the cells, said lead researcher Haoxing Xu, U-M associate professor of molecular, cellular and developmental biology.

When lysosomes "sense" an overload of free radicals, they activate a calcium channel on their membranes. This triggers the expression of many genes and the production of more and stronger lysosomes, which rev into overdrive to rid the damaged parts of the cells.

Free radicals are guilty in the aging process, Xu said.

"If we have chemical compounds that can directly activate this channel, we can lower the oxidative stress in aging and other diseases," he said. "The result will be that cell damage and free radical levels could be reduced, and one can possibly slow down aging."

How does the body tell itself that there are too many free radicals so that they can be reduced or removed? His study tells us how it's done, Xu said.

"Nature is really cool," said. "The janitor of the cell, the lysosome, has this radical-sensing ability."

The study, "MCOLN1 is a ROS Sensor in Lysosomes that Regulates Autophagy," is published online June 30 in *Nature Communications*. First authors are Xiaoli Zhang, Xiping Cheng and Yu Lu, all in the Xu lab.

Provided by University of Michigan