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Researcher studies protein's role in aging

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With time, the amino acid known as asparagine will eventually degrade. Long considered a type of protein "damage," the phenomenon has come to be accepted as yet another part of aging: our hair turns gray, our joints begin to ache, and our asparagine turns into isoaspartic acid.

The surprising thing about this change is that it forces the protein's backbone to follow a new track, just like a railroad switch sends a train on an entirely different journey. "This is exceptionally rare," said chemistry and chemical biology associate professor Sunny Zhou, who recently received a $1 million grant from the National Institutes of Health to study the etiologic role of isoaspartic acid, or isoAsp, in aging and disease. It's research that could dramatically change how doctors treat diseases such as Alzheimer's, which significantly elevates patients' isoAsp levels.

According to Zhou, the rate at which isoAsp forms depends on the sequence of amino acids in the protein. If asparagine sits next to the amino acid proline, it will take a long time to degrade. If it's next to glycine, on the other hand, it may take just half a day. Luckily, there's an antidote. The enzyme "protein isoaspartate methyltransferase," or PIMT, can rectify the damage.

The degradation process that leads to isoAsp happens in virtually all cells and PIMT is present in almost all animal systems except baker's yeast; how yeast cells regulate isoAsp remains a mystery. Additionally, animal studies have shown that eliminating PIMT from the body does reduce life expectancy—but not through aging. "IsoAsp levels in these animals

increase," said Zhou. "But only twofold, not tenfold." This suggests something else must be at play in the regulation process in other animals too, not just yeast.

IsoAsp has the same molecular weight as aspartic acid, making it extremely difficult to detect. At least it used to be. In previous research as a faculty fellow at Northeastern's Barnett Institute of Chemical and Biological Analysis, Zhou helped develop a method for easily tracking it down.

Degradation cannot be prevented, he said, because it happens spontaneously. But if researchers found a way to repair the damage, their work could have a significant effect on the ability to treat age-related disease such as Alzheimer's.

"If we can find the machinery that gets rid of isoaspartic faster, then we can somehow use a driver to boost that machinery," Zhou said, noting that the damaged cells in an Alzheimer's brain contain up 70 percent isoaspartic acid. "That's the hope."

Provided by Northeastern University

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