Bacteria yield clues about why proteins go bad in ALS and Alzheimer's

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(Phys.org)—Scientists are unsure why proteins form improperly and cluster together in bunches, a hallmark of neurodegenerative diseases such as amyotrophic lateral sclerosis (ALS), Alzheimer's and Mad Cow Disease. In the Nov. 1 issue of the journal *Molecular Cell*, Yale scientists shed light on protein aggregate formation by studying the process in bacteria.

"The question we are all asking is what happens when protein synthesis goes wrong?" said Jesse Rinehart, assistant professor of cellular and molecular physiology at Yale's West Campus and co-senior author of the paper.

Proteins are created from instructions encoded in DNA and assembled in ribosomes within the cells. However, sometimes they are not assembled correctly, and these misfolded proteins tend to aggregate, a process typified by the plaques that form in the brains of Alzheimer's patients.

The Yale team—led by Rinehart and Dieter Söll, Sterling Professor of Molecular Biophysics and Biochemistry and professor of chemistry—showed that the antibiotic streptomycin can trigger protein aggregations in the bacterium E. coli. Using large-scale proteomics and genetic screens, they analyzed the aggregates and searched for bacterial proteins that make E. coli cells resistant to antibiotics and other threats. The researchers discovered how one of these proteins protecting the bacteria from hydrogen peroxide also suppressed the aggregation of proteins triggered by streptomycin.

"The properties of these protein aggregates are still mysterious, but here we have a glimpse of how they form and how cells escape from these aggregates in bacteria," Söll said.

The study not only provides insight into how these protein aggregates can form, but illustrates how bacteria defend themselves against toxic threats. Such information could help scientists develop more effective antibiotics, Rinehart said.

Provided by Yale University

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