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Hydrogen Sulfide's Potential Case against Amyloid Formation, investigated by RUM Scientists [1]

Submitted by Kiara Sofia Vega-Bellido [2] on 10 June 2016 - 10:30am

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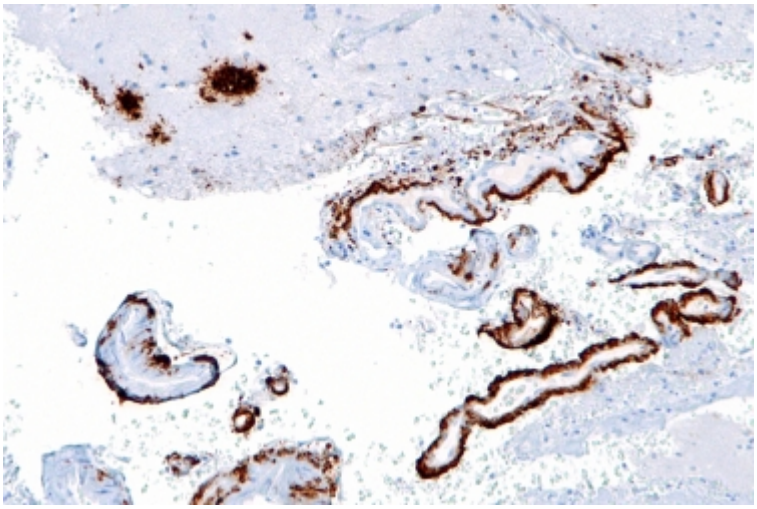

Cerebral amyloid angiopathy. Beta amyloid plaque formed by the aggregation of amyloid fibrils.

If examined from an exogenous viewpoint, hydrogen sulfide is a gas a reasonable, self-loving human wouldn't want to interact with. It's heavier than air, flammable, poisonous, corrosive, and explosive, with a smell suggestive of rotten eggs. However, H2S is in fact produced endogenously in the brain via the activity of an enzyme called cystathionine beta-synthase (i.e. CBS, catalyzes homocysteine into cystathionine, forming H2S in the process) and works as a chemical substance that alters synaptic transmission between neurons or, in other words, as a neuromodulator. It also functions as a smooth muscle relaxant and has several other interesting effects. But perhaps most remarkable is its potential role in the inhibition of amyloid formation, a role currently being investigated by the helpful folks at Dr. Juan López-Garriga's lab [3] in the Department of Chemistry at the UPRM.

Amyloid fibrils are endogenously produced and, when accumulated in considerable amounts, can become extremely harmful. They arise from the denaturalization of proteins in the body due to changes in pH or temperature, which leads to the loss of the proteins' tertiary structure, which in

turn leads to a loss of function. These dysfunctional proteins are too stable for the body to degrade and remain in the body, self-aggregating all over, in the brain, the pancreas, the liver, etcetera. This aggregation becomes extremely detrimental over time, contributing to the development of severely degenerative diseases such as Alzheimer's, Parkinson's, Huntington's, vascular dementia; it has even been linked to diabetes Type II. [4] Understanding how to inhibit amyloid fibril formation would indubitably serve the development of future treatments of these diseases.

Why H2S? Interestingly, H2S is able to interact with disulfide bridges and not all proteins are capable of forming amyloid fibrils, but it turns out that the ones that do usually have disulfide bridges in their tertiary structure. Moreover, another clue was discovered when other studies indicated a lower level of H2S in the brains of Alzheimer's patients in comparison to those of healthy individuals [5]. This knowledge coupled with previous findings on the activation of H2S in hemeproteins and the formation of a sulfheme protein complex (product of the interaction of H2S with the oxygenated form of human hemoglobin and myoglobin) [6] prompted an interest in the scientists at the Lopez-Garriga Lab: what will adding H2S do to amyloid fibril formation?

The title of their following publication, featured in the Journal of Physical Chemistry, speaks for itself: "Hydrogen Sulfide Inhibits Amyloid Formation". [7] Their experiment consisted in adding hydrogen sulfide to an in vitro amyloid formation process using the protein hen egg white lysozyme (HEWL), a protein that forms amyloid fibrils at certain conditions. This led to the complete repression of amyloid formation. Current efforts of the lab are focused on understanding the mechanism by which this inhibition occurs. Perhaps through the interaction between H2S and disulfide bridges? Stay tuned to the research being realized by the people at the Lopez-Garriga lab to find out.

Essay about the research being conducted by Dr. Juan Lopez-Garriga's lab from the Department of Chemistry at the UPRM on hydrogen sulfide's role in the inhibition of amyloid formation. Written by a member of the UPRM Science Communication Initiative in collaboration with the "Academia para Investigación para Facultad y Postdoctorales".

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