

Hydrogen Sulfide: Modern Roles of an Ancient Signaling Molecule in Neuronal Function

March 9

Tuesday, 12:30pm

Live Webinar
via Zoom Conference



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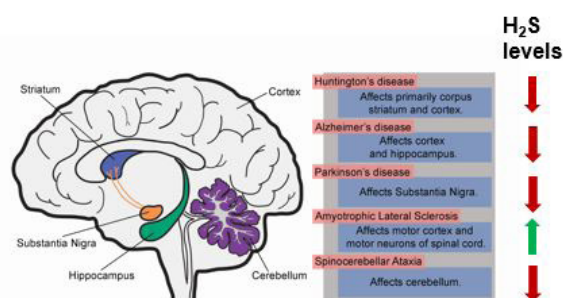
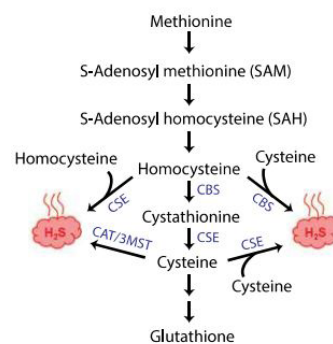
Host: Rajiv R. Ratan, M.D., Ph.D.

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Abstract

Life evolved in a hydrogen sulfide (H₂S)-rich environment, eons ago, and with it emerged signal transduction cascades modulated by sulfhydrylation. Sulfhydrylation/persulfidation is a newly discovered posttranslational modification, wherein the -SH groups of reactive cysteine residues are converted to -SSH or persulfide groups. Sulfhydrylation modulates diverse physiological processes, ranging from response to inflammation to neuroprotection. H₂S is generated via the reverse transsulfuration pathway in mammals from the amino acid cysteine and our studies reveal that this pathway is disrupted in aging and in neurodegenerative diseases such as Huntington's disease, Parkinson's disease and Alzheimer's disease. Accordingly, targeting the reverse transsulfuration, may afford therapeutic benefits in neurodegenerative disorders involving imbalanced cysteine and H₂S metabolism.



1. Paul BD, Sbdio JI, Xu R, Vandiver MS, Cha JY, Snowman AM and Snyder SH. Cystathionine γ -lyase deficiency mediates neurodegeneration in Huntington's disease. *Nature*. 2014; 509(7498):96-100.
2. Sbdio JI, Snyder SH* and Paul BD*. Transcriptional control of amino acid homeostasis is disrupted in Huntington's disease. *Proc Natl Acad Sci USA*. 2016; 113(31):8843-8848. (*Co-corresponding author).
3. Paul BD*, Sbdio JI and Snyder SH*. Cysteine metabolism in neuronal redox homeostasis. *Trends Pharmacol Sci*. 2018; 39(5):513-524. (*Co-corresponding author).
4. Sbdio JI, Snyder SH and Paul BD. Golgi stress response reprograms cysteine metabolism to confer cytoprotection in Huntington's disease. *Proc Natl Acad Sci USA*. 2018; 115(4):780-785. (*Co-corresponding author).
5. Giovinazzo D, Bursac B, Sbdio JI, Nalluru S, Snowman AM, Whiteman M, Filipovic MR, Snyder SH and Paul BD. Hydrogen sulfide mediates neuroprotection in Alzheimer's disease by sulfhydrylation of GSK3 β and inhibition of Tau hyper-phosphorylation. *Proc Natl Acad Sci USA*. 2020. In Press. (*Co-corresponding author).