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Editorial

Hydrogen Sulfide (H2S) Physiology with Crosstalk with GIT Sulfate-Reducing Bacteria (SRB) and Other Bacteria in Therapy of Obesity, Inflammatory Bowel Disease Along with Colorectal Cancer

Kulvinder Kochar Kaur*

Scientific Director Cum Owner Dr Kulvinder Kaur Centre for Human Reproduction, Jalandhar, Punjab, India

***Corresponding Author:** Kulvinder Kochar Kaur, Scientific Director Cum Owner Dr Kulvinder Kaur Centre for Human Reproduction, Jalandhar, Punjab, India.

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H2S delineate's a pivotal molecule for living organisms inclusive of humans. Enrichment of empirical corroborations are present which point a part of H2S in physiology as well as pathophysiology. The biological parts of H2S with plethora of concentrating on its part in the gastrointestinal tract (GIT) has been reviewed extensively. H2S is believed to be the third gasotransmitter, along with nitric oxide (NO) as well as carbon monoxide (CO), in addition to is implicated in i) inflammation, ii) gut motility, iii) oxidative stress (OS), iv) ulcer repair, v) vascular tone, vi) neuromodulation, vii) cryoprotection, viii) memory generation, ix) hormone liberation, x) apoptosis along with x) several other vital biologic working. Certain of such events are further targets for CO as well as NO despite the mechanistic modes of actions of such other gaseous signaling molecules might be variable. H2S in the GIT is generated apart from by the enzymes cystathionine-β-synthase (CBS), as well as cystathionine-γ-lyase (CSE) of the host however further by sulfate-reducing bacteria (SRB) which are resident microbes utilizing the fermentation by-product hydrogen in the form of the substrate.

H_2S in the intestine is further generated by certain members that reside in the form of gut bacteria.

Of the maximum pronouncedgenerators of H2S are sulfate-reducing bacteria (SRB). Dependence on the of the prevalence of methane excretors, sulfate reduction is the predominant

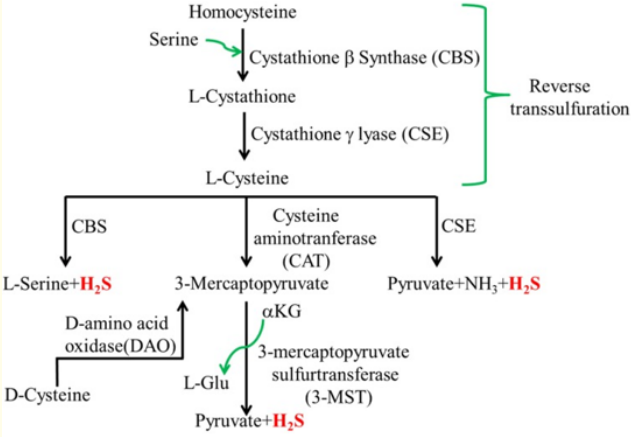

Figure 1: Courtesy ref no- 4 Biogeneration of H2S by mammalian cells.

hydrogenotrophic pathway in equivalent to 60% of humans. Enumeration of SRB by culture based methods documented a variety of 103–1011 bacteria per gm of human feces. SRB belong to the class δ-Proteobacteria in addition to generate H2S utilizing the enzyme complex dissimilatory sulfite reductases (DSR) (Figure 2).

The maximum pronounced genus in such category is *Desulfovibrio*, that use lactate as well as hydrogen in the form

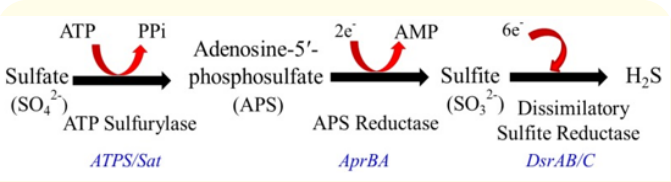

Figure 2: Courtesy ref no-4 Dissimilatory sulfate reduction by SRB. Sulfate is activated to adenosine-5-phosphosulfate (APS) by ATP sulfurylase (ATPS), further acknowledged in the form of sulfate adenylyltransferase (Sat). The second enzyme, APS reductase (Apr), transforms APS to sulfite, that is reduced to sulfide by dissimilatory sulfite reductase (Dsr).

of a substrate. Other genera being *Desulfobacter, Desulfomonas, Desulfobulbus*, in addition to *Desulfotomaculum*. The SRB utilize sulfate in the form of a terminal electron acceptor (TEA) for respiration, with the concurrent generation of H_2S. Other bacterial species affiliated to variable genera, for instance i) *Streptococcus,* ii) *Fusobacterium,* iii) *Salmonella,* iv) *Enterobacter,* along with v) *Helicobacter* further generate H_2S from L-cysteine, by the actions of cysteine desulfhydrase.

In an extra diet- dependent assessment, once obese subjects were fed diets to balance their gut microbes, weight reduction was associated with a reduction in H2S-generating families *Desulfovibrionaceae* as well as *Enterobacteriaceae* (i) *Escherichia*, ii) *Shigella*, iii) *Klebsiella,* along with iv) *Citrobacter*), in addition to a diminishing in proinflammatory i) Tumor necrosis factor (TNF-α) along with ii) Interleukin (IL-6) cytokines. There are conflicting reports inclusive of an extra study illustrating that *Desulfovibrio* quantities were substantially lesser in obese/overweight children in contrast to controls. Hence, the part of SRB in addition to SRB obtained H2S in the pathogenesis of a disease situation, for instance obesity, continue to be uncharted. Despite, mounting corroboration points that SRB is associated with inflammatory situations, it is not clear if such is a side derivative or SRB are the causative factors of the disease.

Apart from SRB, other H2S-generating gut bacteria, for instance *Fusobacterium*, have further been observed to be associated with IBD. The communication amongst H2S-generating gut bacteria in addition to inflammation was further embraced by the observations that *Bilophila wadsworthia*, a sulfite-reducing bacteria which forms H2S analogous to *Desulfovibrio* was higher in quantity in mice fed with high fat diet (HFD), along with this was correlated with proinflammatory reactions in genetically vulnerable mice.

Additional question is are SRB advantageous or inimical ? Does this knowledge possess the capacity of being used to generate innovative treatments to address inflammatory diseases dependent on targeting SRB?

Thus H2S can be utilized in Ischemia/Reperfusion damage, H2S in Intestinal Inflammation, H2S in Ulcer repair, and H2S in Intestinal Motility. Thereby utilized for therapy of IBD (inclusive of UC, CD) and CRC [1-4].

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