Mechanism of H_2S oxidation by dissimilatory (per)chlorate-reducing microorganism *Azospira suillum* PS

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All dissimilatory (per)chlorate-reducing microorganisms (DPRM) tested to date innately oxidize H_2S, producing elemental sulfur as the primary end-product. Although this is a thermodynamically favourable metabolism ($\Delta G^{0'}$=-206 kJ.mol^{-1} H_2S) the underlying biochemical mechanism(s) are unknown. While many DPRM can alternatively utilize nitrate, sulfur oxidation is a perchlorate specific metabolism, suggesting that it requires some unique components of the perchlorate respiratory pathway. Interestingly, there is no growth benefit although H_2S is preferentially utilized over physiological organic electron donors such as lactate or acetate. The perchlorate specific oxidation of H_2S was hypothesized to be due to a combination of biotic/abiotic interactions with the (per)chlorate reductases as well as the reactive chloride species (RCS) or molecular oxygen that are generated as intermediates of perchlorate respiration. These abiotic reactions may have integrated with enzymatic steps as a part of a metabolic strategy, or may be solely an inadvertent consequence of generating highly oxidized intermediates in an anaerobic environment. Using various high throughput-sequencing approaches such as Bar-Seq, RNA-Seq and proteomics, along with targeted mutagenesis and biochemical characterization, we identified all facets of perchlorate-dependent H_2S oxidation in the model DPRB, *Azospira suillum* PS. In support of our proposed model, deletion of known H_2S oxidation genes (eg. Sox, SQR) had no phenotypic effect on H_2S oxidation and purified perchlorate reductase alone was able to mediate H_2S oxidation to perchlorate reduction in the absence of any other redox active electron transfer proteins. Our studies highlighted upregulation of a variety of stress response proteins such as metal efflux pumps and divalent heavy-metal transporter proteins under H_2S conditions. The results of these studies indicate that H_2S oxidation is an innate ability of DPRM that is mediated by short circuiting of the electron transport chain and direct oxidation of H_2S by the perchlorate reductase.