Isotopic consequences of metabolic inefficiency during microbial sulfate reduction

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The global biogeochemical sulfur cycle is set in motion and maintained by the microbiological reduction of marine sulfate to sulfide. The enzymatic cascade of redox reactions induces a distinct range of sulfur and oxygen isotope fractionations between the initial reactant sulfate and terminal product sulfide. What controls the magnitude of that fractionation, however, is the efficiency of that biochemical machinery in response to the organisms' environment. To better understand how variable fractionations are produced we utilize recent advances in microbial sulfate reducer (MSR) genetics and enzymology to determine the influence of the terminal step in MSR, the formation of hydrogen sulfide by the dissimilatory sulfite reductase C (DsrC). The DsrC enzyme couples the soluble cytoplasmic reduction of sulfite by DsrAB to the cytoplasmic membrane potential – in part determined by DsrMKJOP. This membrane potential ultimately allows the MSR cell to generate the chemical energy reserve, adenosine triphosphate (ATP). Here we employ three mutants in a background model sulfate reducing bacterial strain, *Desulfovibrio vulgaris* Hildenborough, in an effort to disrupt the efficacy of intracellular DsrC redox cycling, and ultimately sulfide production. We compare disrupted strains to the wild type in terms of growth and specific sulfate reduction rates, biomass yields, the magnitude of S isotope fractionation, and DsrC activity. Implications for the MSR metabolic network, both as the key metabolism in the reductive branch of the sedimentary S cycle and primary driver of S isotope fractionation in anoxic environments, are discussed.