A blueprint for the sulfur isotope phenotypes of sufate-respiring microbes

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Recent experiments have affirmed three critical features of S isotope fractionation associated with dissimilatory sulfate respiration. First, the magnitude of fractionation (quantified by $^{34}\varepsilon_{DSR}$, which is equal to the difference in molar ^{34}S-^{32}S ratios of sulfate and sulfide - the initial reactant and final waste product in the respiratory processing chain - relative to the molar ^{34}S-^{32}S ratio in sulfide) correlates inversely with intracellular cell-specific sulfate reduction rate (csSRR). Second, $^{34}\varepsilon_{DSR}$ correlates directly with extracellular sulfate concentrations ($[SO_4^{2-}]$). Third, the exponent that relates $^{33}\varepsilon_{DSR}$ to $^{34}\varepsilon_{DSR}$ ($^{33}\lambda_{DSR}$) increases regularly with $^{34}\varepsilon_{DSR}$, reaching values characteristic of thermodynamic equilibrium at the large $^{34}\varepsilon_{DSR}$ limit. However, despite the tendency to use these three relationships for distinct interpretations of the early evolution of microbial life on Earth, these relationships are not independent. They define an "isotopic phenotype" reflecting the modern physiology of dissimilatory sulfate reducing microbes. From this perspective, interpretations of S isotopes in the ancient rock record are based on a critical, unstated assumption: the isotopic phenotype of dissimilatory sulfate reduction has not changed in over 3.5 billion years.

In order to investigate this assumption, we constructed a biochemically based model of metabolic S isotope fractionation. Our new approach moves beyond a phenomenological description of isotope fractionation by taking into account, for example, the rich database of kinetic measurements for the enzymes in the dissimilatory sulfate reduction network. It predicts recent observations ($n > 100$) of the systematic variations of $^{34}\varepsilon_{DSR}$ with csSSR, $[SO_4^{2-}]$, and $^{33}\lambda_{DSR}$ with three free parameters: (1) S isotope fractionation associated with sulfate uptake into the cytoplasm; (2) the ratio of reduced to oxidized electron carriers in the respiration pathway; and (3) *in-vitro* to *in-vivo* levels of enzyme activity. These new results lead to three important general (and quantifiable) rules: (1) Environmental sulfate and sulfide levels control S isotope fractionation through the proximate influence of intracellular metabolites; (2) Low sulfate concentrations lead to less fractionation, but not when coupled to low respiration rates; and (3) No single reaction step controls fractionation, except at high respiration rates.