Microbial uncoupling and energy storage: A modeling study of nitrite toxicity-induced stress response

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Under low oxygen conditions, denitrification and dissimilatory nitrate reduction to ammonium (DNRA) compete to reduce nitrite (NO_2^-), a reactive intermediate produced by nitrate (NO_3^-) reduction. Isotopically labeled nitrogen studies have recently highlighted that DRNA can outcompete denitrification under certain conditions. The relative rates of production (by NO_3^- reduction) and consumption (by DNRA or denitrification) of NO_2^- determine whether or not nitrite accumulates in the environment. Above certain concentrations, nitrite is recognized as a toxin for a number of nitrate-reducing microbes, despite its role as an electron acceptor. Nevertheless, the role of NO_2^- in modulating nitrogen-cycling as a combined function of its electron accepting power and toxicity remains largely unaddressed. We present biogeochemical data from two experiments: (1) a well-mixed batch reactor, and (2) a flow-through reactor, which monitored the growth of *Shewanella oneidensis* during DNRA coupled to lactate oxidation. In both cases, biomass growth was suppressed during the build-up of nitrite, at concentrations well below 1 mM, the previously reported toxicity threshold. A biomass-explicit reactive transport model was fitted to the experimental results. The integration of nitrite toxicity as a key modulator of the energy metabolism of *S oneidensis* cells successfully predicted microbial growth inhibition. In addition, incorporating a formulation for toxicity-induced metabolic uncoupling predicted the observed delay in biomass growth. Our results suggest that *S oneidensis* cells can draw energy from endogenous reserves accumulated during periods of toxicity-induced stress to build biomass, even after the complete consumption of electron acceptor. The model presented herein has implications for the inclusion of microbial physiological adaptations in reactive transport models to better predict the role of toxicity in regulating microbial metabolic processes, and reaction rates.