

Unveiling pH-Dependent metabolic flexibility and homeostasis mechanisms in *Oleidesulfovibrio alaskensis* G20

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Abstract

Sulfate Reducing Bacteria (SRB) demonstrate remarkable metabolic adaptability spanning various environments, including marine sediments, wastewater systems, and oil reservoirs, highlighting their substantial metabolic flexibility influenced by the variability in pH conditions. Nevertheless, the precise molecular mechanisms underlying their adaptation strategy remain elusive. Investigating the adaptation strategies of *Oleidesulfovibrio alaskensis* G20 to varying pH conditions, we found notable pH-dependent variations in growth rates, with pH 7 yielding the highest specific growth rate (μ_{max} , 0.030 hr⁻¹), followed by pH 6 (0.032 hr⁻¹), while pH 8 exhibited a lower μ_{max} of 0.024 hr⁻¹. Additionally, pH 8 converged to pH 7 by day 2, while pH 6 reached pH 7 on day 3. Lactate consumption rates were highest at pH 7 (0.35 mM lactate.hr⁻¹) and lowest at pH 8 (0.09 mM lactate.hr⁻¹). Noteworthy hydrogen production was observed under acidic and alkaline conditions, while neutral pH showed no hydrogen production. Differential gene expression was confirmed via RTPCR analysis at mid-exponential (day 2) and stationary (day 4) phases, encompassing genes related to hydrogenase, F₀-F₁ ATPase, sulfate reduction, lactate dehydrogenase, amino acid synthesis, and carbon/energy metabolism. The study revealed significant upregulation of genes related to hydrogenases, lactate dehydrogenase, and sulfur metabolism during the mid-exponential phase, while genes associated with carbon and energy metabolism were downregulated in both acidic and alkaline conditions, indicating pH homeostasis regulation. This mechanism involves ATPases as proton pumps, hydrogenases facilitating reversible conversion of protons to hydrogen, and sulfate and energy metabolism confined to electron donors (lactate) and acceptors (sulfate), alongside amino acid synthesis for generating basic and acidic amino acids to counter pH and maintain homeostasis.