

Resolving the molecular mechanisms essential to expression of *hgcA* by mercury methylators

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The *hgcAB* gene pair encodes mercury methylation capability in anaerobic microorganisms spanning a diverse array of metabolic clades. Yet it is unclear what environmental conditions have resulted in the conservation of these genes in microbial genomes. It has been postulated that an alternative physiological function of the mercury methylation proteins (HgcAB) may be connected to the gene pair's conservation in microbial genomes. Possible functions include one-carbon metabolism for acetyl-CoA and methionine biosynthesis, metal resistance, or metalloid methylation. Clues to the native biochemical function of HgcAB may lie in determining the environmental conditions that control expression and translation of *hgcA*. Here we explored the transcriptional regulation of *hgcA* under different growth parameters using *Desulfovibrio desulfuricans* ND132 as a model organism. We utilized both molecular (RT-qPCR) and meta-omic (RNA-seq) methods to test whether changes in *hgcA* expression occurred when cells were grown in conditions that require the postulated biochemical functions of HgcAB (e.g. +/- formate, methionine, arsenate, mercury). Indeed, our results indicate that *hgcA* expression is significantly regulated across the growth stages of *D. desulfuricans* ND132 under some but not all conditions tested. We also explored whether deletion of *hgcAB* in *D. desulfuricans* ND132 hindered growth or produced any major phenotype in environmental test conditions. We measured differences in the proteome, metabolome, and metal speciation in growth media between wild-type and $\Delta hgcAB$ cultures. Significant differences in substrate consumption, acetate and biomass production, and expression of C1 metabolism proteins were observed between the strains under fermentative and sulfate-reducing conditions. The presence and abundance of Hg-methylators are often poor indicators for environmental MeHg concentrations. Therefore, understanding the molecular mechanisms essential to expression and translation of *hgcA* by mercury methylators is needed to better predict environmental conditions that drive microbial production of the neurotoxin.