The coupling between Mercury-cell surface interactions and mercury uptake and methylation

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Rare microbial genes bearing similarity to genes in the prokaryotic reductive acetyl–coenzyme A (CoA) pathway, assigned *hgcA* and *hgcB,* were recently found essential for mercury (Hg) methylation. This discovery facilitates studies into microbiological and geochemical factors that control the production of methylmercury (MeHg) in natural environments. Here we examine the Hg-cell surface interactions on Hg uptake and methylation in the methylation-deficient mutant strains (Δ*hgcAB*) of *D. desulfuricans* ND132 and *G. sulfurrenducens* PCA and compared their behavior to that of the respective wild-type (WT) strains. Hg reactions (e.g., adsorption, reduction and oxidation) on cell surface were found to occur simultaneously in laboratory culture studies under anaerobic conditions, resulting in dynamic changes in Hg speciation and thus Hg uptake and methylation. WT PCA cells were capable of not only reducing Hg(II) but also anaerobically oxidizing Hg(0) at increasing cell to Hg ratios, due to cell thiol (cell-SH) induced Hg complexation. By varying cell-SH to Hg molar ratios from 0.014 to 1.4, we found that mercury methylation was positively correlated to cell adsorption ($r = 0.96$) but negatively correlated to Hg reduction ($r = –0.87$). Deletion of the *hgcAB* gene cluster increased the reduction of Hg(II) but decreased the oxidation of Hg(0) in the mutant cells. The Δ*hgcAB* mutant also exhibited a lower abundance of cellular thiols, and decreased adsorption and intracellular uptake of Hg. These results demonstrate that deletion of the *hgcAB* gene cluster not only abolishes Hg methylation but also causes several unforeseen alterations in cell physiology, which are important to further understanding of the uptake mechanism and the biochemical pathways of microbial Hg methylation in the environment.