Mercury-Cell Surface Interactions on Mercury Methylation by *Geobacter sulfurreducens* PCA

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Microbial methylation of mercury (Hg) to methylmercury (MeHg) has received extensive interest due to trophic transfer and bioaccumulation of the neurotoxic MeHg in biota. MeHg is produced primarily by certain anaerobic bacteria, such as *G. sulfurreducens* PCA. Although the genetic basis of bacterial Hg methylation is recently discovered, the mechanisms of Hg uptake and biochemical pathways are not fully understood. Here, we examine the interactions between Hg and *G. sulfurreducens* PCA cells to gain insight into how microbes affect redox transformation of Hg and whether cell association of Hg may influence Hg uptake and methylation. We show that PCA cells are capable of not only reducing Hg(II) but also oxidizing dissolved elemental Hg(0), depending on the ratio of Hg to cell concentrations, the presence or absence of specific Hg-complexing ligands, and specific mutant strains. Under conditions of a low cell to Hg ratio, Hg(II) reduction was dominant, whereas at a high cell to Hg ratio, reoxidation of Hg(0) to Hg(II) occurred. The latter reaction was enhanced by certain thiol ligands, such as cysteine. The *c*-type cytochrome deficient mutant strain, *?omcBESTZ*, is impaired in Hg(II) reduction. The mutant showed a higher Hg-cell association and MeHg production than the wild type. However, the methylation deletion mutant, *?hgcAB*, showed the highest Hg(II) reduction. These results show interesting correlations on Hg(II) reduction, cell surface association, and its uptake and methylation: reduction of Hg(II) decreases the availability of Hg to microbial methylation, whereas increasing Hg(0) oxidation and Hg-cell association increase methylation. Our results also suggest that the reduction of Hg(II) to Hg(0) is likely an alternative detoxification mechanism when the methylation pathway is blocked.