Arsenic methylation across microbial phyla

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Arsenic (As) undergoes extensive microbial cycling in the environment, and, because of the toxicity of this metalloid, many bacteria and archaea harbor genes that confer resistance to it [1]. As methylation is a microbiallymediated process resulting in the addition of one or several methyl groups to inorganic arsenic. The reaction is catalysed by the enzyme arsenite methyltransferase (ArsM), generating volatile and non-volatile arsenicals. It has been proposed as a detoxification mechanism but also as an activation pathway or a precursor reaction for the synthesis of more complex arseno-organic molecules. As methylation has been demonstrated in several microbial species, including the Bacteroidete Arsenicibacter rosenii SM-1 [3] and the Firmicute Clostridium sp. BXM [4]. However, members of numerous other phyla harbor this gene and it is unclear whether all represent functional proteins and active methylating organisms. Thus, the goal of this study was to systematically probe the functionality and the in vivo activity of ArsM across phyla.

We assessed the capacity for As(III) methylation and volatilization across seven microbial strains encoding the *arsM* gene: two archaea (*Methanosarcina mazei* Gö1, *Methanosarcina acetivorans* C2A); two Firmicutes (*Anaeromusa acidaminophila* DSM 3853, *Clostridium pasteurianum* DSM 525); a Streptomycete (*Streptomyces vietnamensis* DSM 41927); a Deltaproteobacterium (*Geobacter metallireducens* GS-15); and a Bacteroidete (*Arsenicibacter rosenii* SM-1). Furthermore, all *arsM* genes were cloned into the arsenic sensitive *Escherichia coli* AW3110(DE3) and As methylation measured.

The results show that most of the strains were not able to methylate As despite harboring *arsM* genes that encode functional ArsM proteins. We hypothesize that more efficient As detoxification pathways might be prevalent, precluding methylation. We conclude that the presence of *arsM* does not equate As methylation activity and that more work is warranted to deconvolute *arsM* regulation.

[1]Andres et al. (2016) FEMS Microbiol. Rev 40, 299-322.
[2]Huang et al. (2016) Environ. Sci. Technol 50, 6389-6396.
[3]Wang et al. (2015) FEMS Microbiol Lett 362, 1-8.