

ArsM: a bacterial As(III) S-adenosylmethionine methyltransferase

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In most organisms, resistance to arsenite results from removal from the cytosol, either by efflux or by vacuolar sequestration. Here we demonstrate for the first time that methylation of arsenite is an alternate mechanism to confer resistance to toxic metalloids. We have identified *arsM* genes encoding bacterial and archaeal homologues of the mammalian Cyt19 As(III) S-adenosylmethionine methyltransferase. In *Rhodopseudomonas palustris*, the *arsM* gene is regulated by arsenicals. Expression of *R. palustris arsM* in an arsenic-hypersensitive strain of *Escherichia coli* confers resistance to arsenite. The onset of resistance in cells of *E. coli* expressing *arsM* correlated with conversion of the arsenite to less toxic methylated species and volatilization of arsenic as trimethylarsine gas. Purified ArsM catalyzes methyl transfer from S-adenosylmethionine to As(III) to form di- and trimethylated arsenical species, including trimethylarsine gas. These results indicate that methylation of arsenic is a detoxifying process in bacteria and archaea. Supported by NIH grants AI45428 and ES10344.