Fate of adsorbed arsenate in the presence of arsenic-, sulfate-, and iron-reducing bacteria

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Redox mobilizaiton of adsrobed arsenate, As(V), is generally regulated by microorganisms[1] [2]. The motivation of this study was to explore the molecular level interactions of arsenic with sulfate- and iron-reducing bacteria as well as *ars*C and *arr*A gene carriers.

Our experimental results demonstrated the release of adsorbed As(V) was substantially higher in biotic samples than that in abiotic controls. STXM results as shown in Figure 1 provided the direct evidence of appreciable reduction of adsorbed As(V) to As(III) [3].

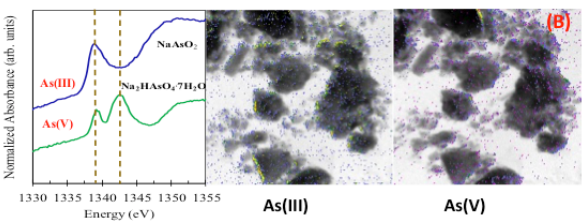

Figure 1: As L_{III} edge spectra and STXM images.

Our incubation and XANES results showed that the formation of the secondary iron mineral pyrite mediated by the iron-reducing bacteria contributed little to the uptake of the freed As [4]. The *ars*C gene carriers mainly control the As speciation in the aqueous phase in aerobic environments, whereas in anaerobic conditions the As speciation should be regulated by *arr*A gene carriers, and As mobility is greatly enhanced by iron reduction.

The arrA gene carriers are proposed to be able to directly reduce the adsorbed As(V). In our study, the addition of an arrA gene carrier during the 120 h incubation did not induce appreciable As release. This observation suggests that there should be no in situ reduction of adsorbed As(V), and desorption or release is the prerequisite for the subsequent microbial reduction. The As mobility in the presence of arrA gene carriers might have previously been overestimated.

[1] Dhar *et al*. (2011) *ES&T* **45**, 2648-2654. [2] Jiang *et al*. (2013) *ES&T* **47**, 8616-8623. [3] Luo *et al*. (2013) *ES&T* **47**, 10939-10946. [4] Tian *et al*. (2015) *ES&T* **49**, 2140-2146.