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Bioinorganic Aspects of the Bacterial Tolerance to Silver ions: Efflux versus Reduction

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Some gram-negative bacterial strains are able to tolerate high amounts of Ag^+ ions either via their efflux or via their reduction.

In the case of the Ag^+ efflux, bacterial strains such as Salmonella typhimurium and Escherichia coli can present a silver resistance as the result of either an outer membrane porin (OmpC and OmpF) deficiency associated to the expression of the Cus transporter, or a Sil transporter expression only. Although these two Ag^+ efflux systems are closely related and contain a similar set of proteins, the Sil tranporter is associated to another small protein, SilE, which has no counterpart in the Cus system, and which seems to avoid the need for porin deficiency. Mutational studies have shown that a suppression of this protein leads to a loss of silver resistance. However, the role of SilE in the Sil system is still debated, even if these discoveries suggest an important role of this protein in Ag^+ detoxification. The full understanding of these efflux machineries is of prime importance to develop new efficient drugs, such as efflux pump inhibitors (EPIs), able to counteract the bacterial mechanisms of silver efflux.

In the case of the Ag^+ reduction, bacterial strains such as Geobacter sulfurreducens can reduce Ag^+ ions into silver nanoparticles (AgNPs) via anaerobic respiration. Although experimentally observed, the mechanisms by which these bacteria can reduce silver ions through extracellular electron transfer (EET) are not fully understood.

The aims of this thesis were to identify the role of the SilE protein in the Sil efflux machinery by investigating its function through model peptide studies and to study the interaction of Ag^+ ions with Geobacter sulfurreducens and the subsequent AgNP formation.

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