DESCRIPTION FOR THE GENERAL PUBLIC (IN ENGLISH)

Arsenic (As) and antimony (Sb) are metalloids (half-metals) that are toxic for most organisms and show cancerogenic activity. On the other hand, arsenic and antimony compounds are used in the treatment of some types of leukemia and tropical diseases caused by protozoans. Unfortunately, arsenic and antimony therapies are often ineffective because of activation of specific defense mechanisms that allow cells to survive in the presence of toxic concentrations of metalloids. One of the most prevalent resistance mechanism is increased production of proteins that are capable of metalloid extrusion out of the cell. Budding yeast is an unicellular organism that serves as a model organism to study the mechanisms of antimony and arsenic resistance. We have previously shown that yeast expresses the Acr3 transporter that very efficiently extrudes arsenic out of the cell. Next, we have determined that this protein also mediates transport of toxic antimony. The aim of this project is to understand the mechanism of metalloid translocation via the Acr3 transporter, to identify amino acid residues in the Acr3 protein conferring substrate specificity and to determine the mechanism of Acr3 internalization and sorting to the vacuole for degradation. Our research study will include mutational analysis of selected amino acid residues in Acr3 and investigation of subcellular localization, stability and transport activity of resulting mutant proteins. The outcomes of this study will not only extend our basic knowledge about the mechanisms of arsenic and antimony detoxification but also in future may result in creation of transgenic plants that are capable of metalloid hyperaccumulation, that may be used for cleaning polluted soils, or new crop species that are free of toxic metalloids due to the presence of efficient detoxification systems.

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