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ABSTRACT
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ID214 | Exploring the fluoride resistome of two defluorinating bacteria

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With the increasing interest in bacterial defluorination as a bioremediation strategy to combat organofluorine pollution (including PFAS), fluoride toxicity arises as a bottleneck. Fluoride, an inevitable end product of these biocatalytic processes, is highly bacteriostatic even at micro- and millimolar levels, limiting the efficiency of enzymatic defluorination. Intracellular fluoride leads to rapid metabolic arrest by inhibiting essential enzymes in central metabolism and may also cause cytoplasmic acidification in low pH environments. While bacteria have evolved specialized membrane shuttles regulated by fluoride-specific riboswitches to export intracellular fluoride from the cell, their deployment during bacterial defluorination has shown to be insufficient in preventing fluoride-induced loss of metabolic viability. And yet, some defluorinating bacteria can accommodate the stoichiometric defluorination of fluorinated compounds that lead to the release of fluoride significantly beyond the known toxic thresholds of this halide for bacteria. In this light, this work aimed to study the transcriptome of two model defluorinating bacteria under fluoride stress, to characterize their homeostatic response and potentially highlight new functional traits linked to their tolerance to intracellular fluoride.

Delftia acidovorans MFA5 and *Labrys portucalensis* F11 were used as model strains for this study, given their capacity to defluorinate aliphatic and aromatic organofluorines. We conducted Minimum Inhibitory Concentration (MIC) assays with these strains to establish their susceptibility threshold towards fluoride. Then, we conducted an expression study by exposing these strains to fluoride at sub-MIC levels for 72 hours, taking samples for RNA-seq every 24 hours. Once we acquire the RNA-seq transcriptomic datasets, we plan to map them to each strain's genome and perform quantitative expression analyses to reveal differentially-regulated transcripts under fluoride exposure.

Results obtained so far showed high fluoride MICs for both MFA5 (500 mM) and F11 (250 mM), confirming their unusual tolerance to this halide. Resolving their transcriptomes under fluoride exposure at sub-MIC levels will allow us to establish a functional map of the homeostatic response of F11 and MFA5 towards fluoride, focused on looking for potentially novel traits of resistance that may explain their unusual tolerance to the halide.