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P-080 - AMINOGLYCOSIDE RESISTANCE IN RALSTONIA PICKETTII

Ivone Vaz-Moreira[1,2]; Pompeyo Ferro[1]; Olga C. Nunes[2]; Célia M. Manaia[1]

1 - Universidade Católica Portuguesa, CBQF - Centro de Biotecnologia e Química Fina – Laboratório Associado, Escola Superior de Biotecnologia, Rua Arquiteto Lobão Vital, 172, 4200-374 Porto, Portugal; 2 - LEPABE, Laboratório de Engenharia de Processos, Ambiente, Biotecnologia e Energia, Faculdade de Engenharia, Universidade do Porto, Rua Dr. Roberto Frias, 4200-465 Porto, Portugal

Background

Ralstonia spp. are ubiquitous in water environments, including drinking water. The species *R. pickettii* is one of the most common in aquatic environments, including some environments classified as sterile or with extremely low microbial loads. This species has also been associated with nosocomial outbreaks. Aminoglycoside resistance is a variable phenotype in this species, suggesting that it can be acquired and not intrinsic as, for example, colistin resistance.

This study aims to: i) investigate the aminoglycoside resistance mechanisms in *R. pickettii* isolated from aquatic environments and assess a possible relationship with the type of water (mineral, tap, wastewater) from which they were isolated, and ii) searches for possible aminoglycoside resistance acquisition hints.

Method

Fifty five *R. pickettii* strains (including some evolved in the presence of gentamicin or UV radiation) were characterized for their antibiotics and metals tolerance. Based on the comparative genome sequencing analysis of two of the isolates, with distinct aminoglycoside resistance phenotype and isolated from the same habitat (hospital effluent), some genetic determinants were selected to analyse in all the strains under study. Those genetic elements were the ICEs (integrative conjugative elements), plasmids, genes encoding efflux pumps or associated with arsenic resistance.

Results & Conclusions

Besides to aminoglycosides, resistance to beta-lactams and colistin was frequent in *R. pickettii*, regardless the type of water. An association between aminoglycosides resistance and increased arsenite tolerance was observed. All the isolates resistant to aminoglycosides had minimal inhibitory concentrations (MIC) values of >256mg/mL to gentamicin and of 1.4mM to arsenite, while for the susceptible strains values of 6mg/mL and 0.05mM, respectively, were found. Accordingly, only the aminoglycoside resistant isolates had the genes *arsH* and *ACR3*, related to arsenite resistance. Also distinguishing the aminoglycoside resistant and susceptible strains was the presence of ICEs gene fragments, only in resistant strains, and the aminoacid sequence of the RND efflux pump. Although these findings suggested a common mechanism of resistance, such hypothesis was not supported by experimental evolution assays of aminoglycoside susceptible strains, in which aminoglycoside MIC values increased to resistance levels while arsenite tolerance did not vary. On the other hand, these data can suggest that besides a common aminoglycoside and arsenite resistance acquired eventually by horizontal gene transfer, mutation may lead to the acquisition of an alternative aminoglycoside resistance mechanism.

References & Acknowledgments

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