Introduction & Purpose

Several studies have shown that the Achromobacter xylosoxidans (Ax) strain, which is highly resistant to a wide range of antibiotics, becomes increasingly prevalent in patients with cystic fibrosis (CF) [1,2]. Resistance mechanisms in Ax are active efflux through AxyABM and AxyXY-OprZ efflux pumps, which share homology with MexAB-OprM and MexXY-OprM in Pseudomonas aeruginosa, respectively [3,4]. Here, we explore the role of AxyABM and AxyXY-OprZ in the resistance of Ax to antibiotics previously described as substrates for MexAB-OprM (tacircillin and temocillin) and MexXY-OprM in Pseudomonas aeruginosa, respectively [3,4].

Materials & Methods

Strains: 3 Achromobacter xylosoxidans strains (Ax: clinical isolate, Ai:ΔB/ΔY mutants harbor pUC19 plasmid which encodes a carboxy-penicillinase. TZB was thus added to these MICs in broth (microdilution method) to consider innately resistant to Azithromycin MICs (mg/L) also showed significantly higher NPN accumulation, suggestive of lower efflux activity.

Statistical analysis: partition tree to determine the MIC value splitting distribution in 2 with the highest Gower's value (log, p-value), using JMP-Pro 14.

Main Messages

- While TIC, TMO, and AZI are substrates for MexAB-OprM in P. aeruginosa [5,6], they are not or only marginally affected by AxyABM efflux pump in Achromobacter.
- AMK and AZI are both substrates for AxyXY-OprZ efflux pump.
- The high MICs of beta-lactams in clinical isolates and the poor effect of TMB against these substrates suggest the presence of other resistance mechanisms in these strains beside production of TZB-inhibitable β-lactamases or efflux.
- Susceptibility to AMK in some clinical isolates was unexpected (Ax being considered innately resistant to aminoglycosides) and needs to be further explored.