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"HEAVILY ARMED" PSEUDOMONOAS AERUGINOSA: MECHANISMS AND GENETIC BACKGROUND OF DRUG RESISTANCE

Summary

The rapid spread of antibiotic resistance (AMR) in pathogenic bacteria is one of the greatest challenges of modern infectiology. In particular, the most threatening are nosocomial infections caused by multi-drug-resistant strains of several major species, such as *Pseudomonas aeruginosa*. This opportunistic pathogen exibits a broad-spectrum of natural resistance. Due to its high genome plasticity, comprising functional mutations and acquisition of foreign DNA, *P. aeruginosa* can easily adapt and persist in harsh environmental niches. The critical issue is its outstanding ability to acquire diverse AMR mechanisms, including those encoded by mobile genetic determinants. In addition to the intrinsic resistance, *P. aeruginosa* can be highly resistant to all of the currently available antipseudomonadal antimicrobials. *P. aeruginosa* is the etiological agent of a variety of infections, including acute pneumonia, bloodstream infections or skin and soft tissue infections (e. g. postoperative or burn wounds). It is responsible also for chronic infections, like those in cystic fibrosis (CF) patients. The major antimicrobials used in *P. aeruginosa* infections are newer-generation cephalosporins, carbapenems, fluoroquinolones or aminoglycosides. Owing to limitations of the effective therapeutic options against *P. aeruginosa*, new antimicrobials and novel indications and thus applications for older drugs are being developed.