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Multidrug resistance in *Acinetobacter, Pseudomonas* and *Stenotrophomonas*

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The increasing trend of antibiotic resistance in *Acinetobacter baumannii* and *Pseudomonas aeruginosa* worldwide is worrisome since it limits drastically the therapeutic alternatives. In particular, resistance to ß-lactams and especially to carbapenems is increasing and panresistant strains are now often isolated, sometimes at the origin of outbreaks. Metallo-ß-lactamase (MBL) and extended-spectrum ß-lactamase (ESBL) production are enzymatic mechanisms responsible for high-level resistance to carbapenems and expanded-spectrum cephalosporins, respectively. They have been found in *Pseudomonas*, and more rarely in *Acinetobacter*, although *Stenotrophomonas maltophilia* produces both types naturally. There are also carbapenem-hydrolysing class D ß-lactamases (CHDLs) that are mostly specific for *A. baumannii*. They belong to three unrelated groups of clavulanic-acid resistant ß-lactamases represented by OXA-23, OXA-24 and OXA-58 that can be either plasmid- or chromosome-encoded. In addition to these acquired mechanisms, *A. baumannii* and *P. aeruginosa* possess naturally a carbapenem-hydrolysing oxacillinase (OXA-51 and OXA-50, respectively) which expression may vary and likely play a role in carbapenem resistance. Along with ß-lactamases, carbapenem resistance in *A. baumannii* and *P. aeruginosa* may be also the result of porin modifications or loss. Several porins including the so-called 33-kDa CarO protein that was found to constitute a pore channel for influx of carbapenems might be involved in carbapenem resistance in *A. baumannii* and the key protein for that resistance in *P. aeruginosa* is OprD (or D2 porin).

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Selected References for Further Reading

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