

**Pseudomonas**  
[created by  
Paul Young  
02/10/07]

**general**

- *P. aeruginosa* is a Gram-negative aerobic rod
- It is a hardy organism, with minimal requirements for growth and tolerates a wide range of physical conditions. It is found in water, soil and plants including fruit and vegetables
- In hospitals, it has been isolated from food, cut flowers, sinks, toilets, floor mops, respiratory and dialysis equipment and other moist environments, including commonly used disinfectants and antiseptics

**colonisation**

- Detectable colonisation in healthy persons is unusual, with prevalences of up to 2% on skin, 7% in the throat and 24% in faeces.
- Rates of colonisation rise rapidly upon hospitalisation and within 7 days, 23% of patients may be colonised. This figure increases to as much as 60% within a fortnight.
- With a preference for moist body sites, *P. aeruginosa* can be recovered from throat, nasal mucosa, axillae and perineum

**infection**

- *P. aeruginosa* rarely causes disease in healthy persons. However, it is an extremely important nosocomial Gram-negative pathogen because of its frequency, ubiquity and intrinsic resistance to many antimicrobials.
- It has been repeatedly identified as one of the commonest causes of ventilator-associated pneumonia.
- Like other opportunistic organisms, *P. aeruginosa* can cause a wide variety of infections, including bacteraemia, urinary tract, surgical site, skin and eye infections. The mortality associated with serious *Pseudomonas* infections is thought to be greater than with other nosocomial pathogens
- Both endogenous and exogenous sources are significant in the acquisition of *Pseudomonas*. The gastrointestinal tract is the likely endogenous source for respiratory and skin colonisation, and is of particular importance in the pathogenesis of ventilator-associated pneumonia.
- Cross-contamination from environmental sources causing outbreaks has also been well described.

**resistance**

- *P. aeruginosa* is intrinsically resistant to many antibiotics because of membrane impermeability, multidrug efflux pumps and a chromosomal AmpC beta-lactamase.
- *Pseudomonas* can acquire both plasmid based and chromosomal resistance genes.
- Resistance to the antimicrobial used emerges during treatment in at least 10% of patients and appears to be the most likely with imipenem and the least likely with ceftazidime, ciprofloxacin or piperacillin. Imipenem is known to select for mutants with reduced membrane permeability due to loss of the porin OprD. These strains show resistance to imipenem and reduced susceptibility to meropenem.

**therapy**

- Agents with antipseudomonal activity include beta-lactam / beta-lactamase inhibitor combinations (piperacillin-tazobactam, ticarcillin-clavulanate), some cephalosporins (ceftazidime, cefepime, cefpirome), carbapenems (imipenem, meropenem), aminoglycosides, fluoroquinolones and aztreonam.
- Combination antibiotic therapy, typically with an antipseudomonal penicillin / beta-lactamase inhibitor combination and an aminoglycoside, is often recommended for suspected or proven *P. aeruginosa* infections.
- There is little clinical evidence of the superiority of combination therapy, although it has the theoretical advantage of antibacterial synergy.
- The addition of an aminoglycoside does not appear to reduce the emergence of resistance during therapy or prevent treatment failure. As a result, many reserve aminoglycosides for very severely ill patients and use an antipseudomonal penicillin / beta-lactamase combination alone. Alternatively, a carbapenem, antipseudomonal cephalosporin or fluoroquinolone may be used, either alone or in combination with an aminoglycoside.