Pseudomonas 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.

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.

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. 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.

- 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.

Agents with antipseudomonal activity include beta-lactam / beta-lactamase inhibitor combinations (piperacillin-tazobactam, ticarcillin-clavulanate), some cephalosporins (ceftazidime, cefepime, ceftiroxime), 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 cephalosporin or fluoroquinolone. Alternatively, a carbapenem, antipseudomonal cephalosporin or fluoroquinolone may be used, either alone or in combination with an aminoglycoside.