

## Pigments of *Pseudomonas aeruginosa*

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### Editorial

*Pseudomonas aeruginosa* is an opportunistic pathogen that causes extensive morbidity and mortality in individuals who are immunocompromised or have underlying medical conditions such as, urinary tract, respiratory tract and skin infections and primarily causes of nosocomial infections [1]. It's a non sporulating, gram negative, oxidase positive motile bacterium with apolar flagellum [2], *P. aeruginosa* is a common nosocomial pathogen because it is capable of thriving in a wide variety of environmental niches [3]. It is a leading cause of hospital associated infections in the seriously ill, and the primary agent of chronic lung infections in cystic fibrosis patients [4]. They exist in very large numbers in the human environment and animal gut, they are capable of inhabiting/contaminating water, moist surface and sewage, hospital environment usually have resident *P. aeruginosa* [5].

Despite the apparent ubiquity of *P. aeruginosa* in the natural environment and the vast array of potential virulence factors, the incidence of community-acquired infections in healthy subjects is relatively low. However, in the hospital environment, particularly in immunosuppressed, debilitated and burns patients, the incidence of *P. aeruginosa* infection is high [6]. It produces many numbers of extracellular toxins, which include phytotoxic factor, pigments, hydrocyanic acid, proteolytic enzymes, phospholipase enterotoxin, exotoxin and slim [1].

*P. aeruginosa* grows well on media and most strains elaborate the blue phenazine pigment pyocyanin and fluorescein (yellow), which together impart the characteristic blue-green coloration to agar cultures [5]. Pyocyanin is a blue redox-active secondary metabolite [7], which induces rapid apoptosis of human neutrophils, with a 10 fold acceleration of constitutive neutrophil apoptosis in vitro but no apoptosis of epithelial cell or macrophages [8]. The redox active exotoxin pyocyanin is produced in the concentration up to 100 mol/l during the infection of CF patient and other bronchiectatic airways. The contributions of pyocyanin during infection of bronchiectatic airways are not appreciated [9]. Notably pyocyanin mediated

ROS inhibit catalase activity, deplete cellular antioxidant reduced glutathione and increased the oxidized reduced glutathione in the bronchiolar epithelial cell [10,11]. Excessive and continuous producing of ROS and inhibit of antioxidant mechanisms overwhelm the antioxidant capacity, leading to tissue damage, also pyocyanin inhibit ciliary beating of the airway epithelial cell [12]. Pyocyanin. Also increases apoptosis and inactivates 1-protease inhibitor. reducing agents such as GSH and NADPH can reduce pyocyanin to pyocyanin radical, which then mono-or divalently reduce O<sub>2</sub> to form superoxide anion O<sub>2</sub><sup>-</sup> or H<sub>2</sub>O<sub>2</sub> [13].

Pyoverdine per contra is the main siderophore in iron gathering capacity its function as a powerful iron chelator, solubilizing and transporting iron through the bacterial membrane via specific receptor proteins at the level of outer membranes. Pyoverdine is important because it has a high affinity for iron, with an affinity constant of 10<sup>32</sup> [14]. Moreover, has been shown to remove iron from transferrin in serum, probably assisting growth within, and ultimate colonization of the human host by *P. aeruginosa* [15]. Moreover experiments studying the burned models of *P. aeruginosa* infections have shown that ferric-pyoverdine is required infection and/or colonization, underlining the importance of ferric-pyoverdine to virulence of *P. aeruginosa* [14].

Pyomelanin, a dark brown/black pigment, is a potential target for anti-virulence compounds which is a negatively charged extracellular pigment of high molecular weight, derived from the tyrosine catabolism pathway [16]. Pyomelanin production has been reported in *P. aeruginosa* isolates from urinary tract infections and chronically infected Cystic Fibrosis (CF) patients [17]. Pyomelanin is one of the many forms of melanin that is produced by a wide variety of organisms. Production of pyomelanin is reported to provide a survival advantage, scavenge free radicals, bind various drugs, give resistance to light and reactive oxygen species, and is involved in iron reduction and acquisition, and extracellular electron transfer [18]. Non-pyomelanogenic strains of Burkholderiaceae are

more sensitive to externally generated oxidative stress and show reduced survival in phagocytic cells [19]. In *P. aeruginosa*, pyomelanin production results in increased persistence and virulence in mouse infection models.

*P. aeruginosa* it is highly resist to antibiotics this resistance can be conferred by the outer membrane which provides an effective intrinsic barrier in the cell wall (or) cytoplasmic membrane (or) within the cytoplasm and modifications in outer membrane permeability via alternations in porin protein channel represent a component of many resistance mechanisms. In addition in activating enzymes released from the inner membrane can function more efficiently within the confines of the periplasmic space, the mechanisms by which intracellular concentrations of drugs are limited include decreased permeability through the outer membrane and active efflux back out across the cytoplasmic membrane [20]. The production of B-lactamase is the most prevalent mechanisms of resistance to B-lactam antibiotics, the B-lactamase have been reported to hydrolyze all anti pseudomonal agents. Moreover, *P. aeruginosa* cell particularly in patients with chronic infections can develop a biofilm, In which bacterial cells are enmeshed into a mucoi dexo polysaccharide becoming more resistant to beta-lactams as well as decrease the outer membrane permeability that enable bacteria to gain resistance development [21].

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