Introduction: *Pseudomonas aeruginosa*

NICHOLAS M MOORE, MARIBETH L FLAWS

ABBREVIATIONS: RNA = ribonucleic acid; rRNA = ribosomal ribonucleic acid

INDEX TERMS: *Pseudomonas aeruginosa*, molecular based methods, anti-microbial resistance.

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Nicholas M. Moore, MS, MLS(ASCP), Rush University, Department of Medical Laboratory Science, Chicago, IL

Maribeth L. Flaws, PhD, SM(ASCP)SI, Rush University, Department of Medical Laboratory Science, Chicago, IL

Address for Correspondence: *Nicholas M. Moore, MS, MLS (ASCP), Department of Medical Laboratory Science, 600 S. Paulina St., Suite 1014, Chicago, IL 60612, 312-942-2111, Fax: (312) 942-6464, Nicholas_Moore@rush.edu*

In 1882, Carle Gessard (1850-1925) first isolated *Pseudomonas aeruginosa*¹ from wound infections of soldiers whose bandages had a blue and green color. Walter Migula (1863-1938) first proposed the genus *Pseudomonas* in 1894 to encompass all gram-negative, rod-shaped aerobic bacilli that had polar flagella. Because this definition was so broad, unrelated organisms were added to the genus to the point that in 1984, there were over 100 species of *Pseudomonas* listed in *Bergey's Manual of Systematic Bacteriology*.^{2,3} When molecular-based methods were developed that involved sequencing and comparing 16S ribosomal RNA from all of these organisms, scientists were able to reassign many organisms originally classified as *Pseudomonas* into other genera. First, rRNA-DNA hybridization studies described by Palleroni⁴ divided the genus into five groups called rRNA Groups I-V. Over the last 30 years, members of rRNA Groups II-V have been all moved to different genera such as *Burkholderia*, *Ralstonia*,

Acidovorax, and *Brevundimonas*.³ The only remaining species of *Pseudomonas* are members of rRNA Group I and are divided into the following eight groups: *P. aeruginosa*, *P. chlororaphis*, *P. fluorescens*, *P. pertucinogena*, *P. putida*, *P. stutzeri*, *P. syringae* and *incertae sedis*, which is Latin for uncertain placement.

P. aeruginosa is a common organism in the soil and in water and it can also be found on plants and animals. It is an aerobic, saccharolytic, non-spore forming gram-negative bacillus measuring 0.5 to 0.8 µm by 1.5 to 3.0 µm.⁵ Most strains of *P. aeruginosa* possess a single polar flagellum that is used for motility. *Pseudomonas aeruginosa* commonly produces two soluble pigments: pyocyanin, which gives colonies a blue color and pyoverdinin, also known as the fluorescent pigment, which is a yellow-green or yellow-brown pigment. When a strain of *P. aeruginosa* produces both pyoverdinin and pyocyanin, the resulting colonies have a blue-green color. This organism may also produce other water-soluble pigments such as pyorubrin or pyomelanin which give colonies a red or brown color, respectively. On sheep blood agar plates, colonies of *P. aeruginosa* often display beta-hemolysis and a greenish metallic sheen due to their pigment production. No other species of gram-negative nonfermenters produce pyocyanin, making its presence helpful in identifying the organism. One of the most recognizable signs of an unknown colony being *P. aeruginosa* is the characteristic fruity, grape-like odor from the production of 2-aminoacetophenone by the organism.

P. aeruginosa is a glucose and lactose non-fermenting bacterium that is frequently isolated from hospitalized patients. The organism is an obligate aerobe, although it can use nitrate and arginine as a final electron acceptor when O₂ is not available allowing the organism to grow anaerobically.⁵ It tests positive with N,N,N',N'-tetramethyl-*p*-phenylenediamine dihydrochloride (oxidase). Because of its relatively simple nutritional

requirements for growth, *P. aeruginosa* has been isolated from many different environments, including even distilled water. It is very hardy and can grow in temperatures ranging from 37-42°C.

Isolates of *P. aeruginosa* can have multiple different colony morphologies. Most colonies are flat and spread out over the agar surface. Depending on the pigment(s) produced, isolates will vary in color from the most common bluish-green to red or brown. *P. aeruginosa* isolated from patients with cystic fibrosis in particular will have a mucoid appearance due to the production of alginate.⁵ Alginate is a polysaccharide layer that surrounds and causes many individual bacterial cells to clump together into a biofilm making them harder to kill with antimicrobial agents.⁶

P. aeruginosa has been shown to cause a variety of diseases and thus has been isolated from almost any type of specimen. Infections can be acquired from the community or during a stay in a healthcare facility. Typical community-acquired infections include skin and soft tissue infections (e.g. hot tub folliculitis), keratitis linked to the use of contact lens, and otitis externa. Nosocomial infections are more likely to be invasive and include infections of the bloodstream, urinary tract, surgical sites, and the respiratory tract. Patients who are particularly susceptible to infection with *P. aeruginosa* are those who have burns, cystic fibrosis or are on a mechanical ventilator. Nosocomial infections caused by *P. aeruginosa* are very common; 11-13.8% of all nosocomial infections are due to *P. aeruginosa*, with an even higher rate (13.2-22.6%) of nosocomial infections among patients in an intensive care unit (ICU).⁷

Infections caused by *P. aeruginosa* have always been difficult to treat, but like other bacteria *P. aeruginosa* is becoming increasingly resistant to antimicrobial agents.⁸ To make matters worse, multidrug resistant *P. aeruginosa* has been identified where the organism is resistant to three or more antimicrobial agents and in one study accounted for almost 30% of all isolates obtained from patients in nursing homes and the ICU.⁸ Accurate methods for determining the susceptibility of *P. aeruginosa* to antimicrobial agents are needed now more than ever as is the need for tracking and

documenting changes in the susceptibility pattern. Knowing the antibiogram of *P. aeruginosa* isolated in one facility is information that can be used to determine the most efficacious empirical therapy for patients suspected of having *P. aeruginosa* infections.

Because of the prevalence of infections caused by *P. aeruginosa* and the increasing incidence of drug resistance, this FOCUS series in *Clinical Laboratory Science* has been designed to provide an overview of the current body of knowledge of *P. aeruginosa*. It will cover three major topics: 1) epidemiology and pathogenesis; 2) resistance mechanisms; and 3) treatment options and strategies for patients with documented or suspected *P. aeruginosa* infections.

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