



PSEUDOMONAS SPP.

PATHOGEN SAFETY DATA SHEET - INFECTIOUS SUBSTANCES

SECTION I - INFECTIOUS AGENT

NAME: *Pseudomonas* spp.

SYNONYM OR CROSS REFERENCE: *P. aeruginosa*, *P. stutzeri*, *P. fluorescens*

CHARACTERISTICS: The genus *Pseudomonas*, of the *Pseudomonadaceae* family, are motile gram-negative aerobic bacteria, 2 – 4 µm long plump-shaped rods, with polar flagella which have an important role in pathogenicity [1](#)-[3](#). They are non-spore forming and can produce pigments, such as pyocyanine (green-blue) and pyorubrin (yellow-green) fluorescence [1](#), [4](#)-[7](#). *P. aeruginosa* can produce a large variety of extracellular toxins, including exotoxin A and enterotoxins [8](#). Other substances such as hydrocyanic acid, proteolytic enzymes, toxic surface slime, and haemolytic substances may also contribute to the pathogenicity of this species. Toxins combined with harmful substances are determinant factors in the high virulence of *P. aeruginosa* in a variety of different hosts [9](#).

SECTION II - HAZARD IDENTIFICATION

PATHOGENICITY/TOXICITY: As opportunistic pathogens, *Pseudomonas* spp. often invades the host tissue and cause infection and bacteremia in immunocompromised hosts (e.g., HIV/AIDS, cystic fibrosis, bronchiectasis, and severe chronic obstructive pulmonary disease, burns, malignancy, or diabetes mellitus) [10](#), [11](#). The common site of infection is the lower respiratory tract, and severity ranges from colonization without immunological response to severe necrotizing bronchopneumonia; such severe infection in patients with cystic fibrosis is almost impossible to eradicate once established in the airways [12](#). Pseudomonal pneumonia often develops from oro-pharyngeal contamination or secondary bacteremia, and is also a common cause of nosocomial ventilator-related pneumonia in intensive care settings. Infections also include endocarditis, osteomyelitis, urinary tract infections, gastrointestinal infections, meningitis, and, commonly, septicemia [13](#). *P. aeruginosa* is the most common agent associated with infection and inflammation during contact lens wear. The bacteria colonize on lenses and produce proteases to kill or invade corneal cells, an infection that can lead to scarring and vision loss [1](#). The species is also the most virulent with a mortality rate of 30%, which can be higher depending on predisposing conditions [4](#). *P. aeruginosa* can also readily colonize on open burn wounds, causing infections, abscesses, and sepsis, with edema and/or discoloration of unburned skin at wound margins and green pigment in subcutaneous fat [14](#), [15](#). *P. aeruginosa* is also associated with swimmer's ear (otitis externa). Other *Pseudomonas* species are also opportunistic; however, cases of infection are rare [3](#).

EPIDEMIOLOGY: Worldwide – often a problem in hospitals as it can be found on equipment, increasing the risk of nosocomial infections [12](#). 30 – 40% of those with cystic fibrosis will acquire chronic pseudomonal infection. *P. aeruginosa* infections account for 20% of pneumonia and 16% of urinary tract infections [16](#). Prevalence in the community is less than in the hospital, and cases of severe community-acquired infection are rare [17](#).

HOST RANGE: Humans, animals (wild, domestic, livestock), and plants (flora and fungi) [18](#).

INFECTIOUS DOSE: Unknown for humans. Studies with larvae models have found the infectious dose for the insects to be high [19](#).

MODE OF TRANSMISSION: *P. aeruginosa* have been found to survive within droplet nuclei and can remain in aerosols for long periods of time, thus there is evidence of potential airborne transmission [20](#). Contact with contaminated water is also a major route, but since the oral infectious dose is thought to be very high, routes that pose the greatest health risk are skin exposure (for example, in contaminated hot tub water) and lung exposure from inhaling aerosols discharged from infected respiratory tracts [13](#). The bacterial can often enter the body through injuries and wounds [3](#). The use of contaminated mechanical respiratory ventilators in hospital settings is also a common source of nosocomial infections [12](#).

INCUBATION PERIOD: Varies according to infection, eye infection can appear 24 – 72 hours after infection [21](#).

COMMUNICABILITY: Spread of infection from person-to-person is speculated to be highly possible during infection, especially amongst cystic fibrosis patients [22](#).

SECTION III - DISSEMINATION

RESERVOIR: Infected humans, animals, contaminated water, soil [18](#). *Pseudomonas* spp. are ubiquitous in the environment.

ZOONOSIS: None.

VECTORS: None.

SECTION IV - STABILITY AND VIABILITY

DRUG SUSCEPTIBILITY: *Pseudomonas* spp. are resistant to many antibiotics. Susceptibility to extended-spectrum penicillins (such as ticarcillin, azlocillin, and piperacillin), aminoglycosides, cephalosporins, fluoroquinolones, polymyxins, and the monobactams [12](#).

DRUG RESISTANCE: Multi-drug resistant strains are emerging, such as against carbenicillin, cephalosporins, ceftazidime, and ciprofloxacin [12](#).

SUSCEPTIBILITY TO DISINFECTANTS: Susceptibility has been shown for 1% sodium hypochlorite, 70% ethanol, 2% glutaraldehyde, and formaldehyde [23](#); however, it has been found to be resistant to disinfectants that are used to treat drinking water such as chlorine, chloramines, ozone, and iodine [13](#). Certain adapted stains have been found to be able to grow in disinfectants; however, isopropyl alcohol 4% v/v or ethyl alcohol 6% v/v are effective disinfectants [24](#).

PHYSICAL INACTIVATION: Inactivation and sterilization using moist heat should be at 121°C for 15 minutes or longer, dry heat at 170-250 °C or higher for 30 minutes or more [25](#).

SURVIVAL OUTSIDE HOST: *Pseudomonas* can survive for months on dry surfaces and inanimate objects, and are one of the bacteria most frequently isolated from patients with nosocomial infections; humidity can improve persistence [26](#). Growth observed in distilled water can survive up to months with minimal nutrients [27](#).

SECTION V – FIRST AID / MEDICAL

SURVEILLANCE: Diagnosis is made by bacteriological culture on selective/non-selective culture media and laboratory identification [28](#).

FIRST AID/TREATMENT: Administer appropriate drug therapy. Aminoglycoside with β -lactam penicillin is usually the first line of treatment [12](#). Aggressive treatment can avoid development of chronic infection. Wounds should be cleaned with surgical detergent disinfectants and/or topical

antibacterial ointments, such as mupirocin [15](#).

IMMUNIZATION: None currently available, although studies have shown that live-attenuated *P. aeruginosa* vaccines in mice can protect against corneal infections [15](#).

PROPHYLAXIS: Antibiotics such as ciprofloxacin (a fluoroquinolone) can be used in patients with CF, but constant prophylactic therapy is not recommended as it can lead to drug resistance [12](#).

SECTION VI - LABORATORY HAZARDS

LABORATORY-ACQUIRED INFECTIONS: None reported to date.

SOURCES/SPECIMENS: Blood cultures, urine, skin, sputum, soft tissue samples, lower respiratory tract secretions, wound exudates, contaminated water samples, and mechanical ventilator equipment [4](#), [12](#), [14](#), [27](#), [28](#)

PRIMARY HAZARDS: Accidental parenteral inoculation, inhalation of infectious aerosols, accidental ingestion, or direct skin contact [4](#), [13](#), [20](#).

SPECIAL HAZARDS: None.

SECTION VII – EXPOSURE CONTROLS / PERSONAL PROTECTION

RISK GROUP CLASSIFICATION: Risk Group 2. This risk group applies to the genus as a whole, and may not apply to every species within the genus.

CONTAINMENT REQUIREMENTS: Containment Level 2 facilities, equipment, and operational practices for work involving infectious or potentially infectious materials, animals, or cultures. These containment requirements apply to the genus as a whole, and may not apply to each species within the genus.

PROTECTIVE CLOTHING: Lab coat. Gloves when direct skin contact with infected materials or animals is unavoidable. Eye protection must be used where there is a known or potential risk of exposure to splashes [29](#).

OTHER PRECAUTIONS: All procedures that may produce aerosols, involve high concentrations or large volumes should be conducted in a biological safety cabinet (BSC). The use of needles, syringes, and other sharp objects should be strictly limited. Additional precautions should be considered with work involving animals or large scale activities [29](#).

SECTION VIII – HANDLING AND STORAGE

SPILLS: Allow aerosols to settle and, wearing protective clothing, gently cover spill with paper towels and apply an appropriate disinfectant, starting at the perimeter and working towards the centre. Allow sufficient contact time before clean up [29](#).

DISPOSAL: Decontaminate all wastes that contain or have come in contact with the infectious organism before disposing by autoclave, chemical disinfection, gamma irradiation, or incineration [29](#).

STORAGE: The infectious agent should be stored in leak-proof containers that are appropriately labelled [29](#).

SECTION IX - REGULATORY AND OTHER INFORMATION

REGULATORY INFORMATION: The import, transport, and use of pathogens in Canada is regulated under many regulatory bodies, including the Public Health Agency of Canada, Health Canada, Canadian Food Inspection Agency, Environment Canada, and Transport Canada. Users are responsible for ensuring they are compliant with all relevant acts, regulations, guidelines, and standards.

UPDATED: December 2011

PREPARED BY: Pathogen Regulation Directorate, Public Health Agency of Canada

Although the information, opinions and recommendations contained in this Pathogen Safety Data Sheet are compiled from sources believed to be reliable, we accept no responsibility for the accuracy, sufficiency, or reliability or for any loss or injury resulting from the use of the information. Newly discovered hazards are frequent and this information may not be completely up to date.

Copyright ©

Public Health Agency of Canada, 2011

Canada

REFERENCES:

- 1 Willcox, M. D. (2007). *Pseudomonas aeruginosa* infection and inflammation during contact lens wear: a review. *Optometry and Vision Science : Official Publication of the American Academy of Optometry*, 84(4), 273-278. doi:10.1097/OPX.0b013e3180439c3e
- 2 Dasgupta, N., Arora, S. K., & Ramphal, R. (2000). fleN, a gene that regulates flagellar number in *Pseudomonas aeruginosa*. *Journal of Bacteriology*, 182(2), 357-364.
- 3 Kayser, F. H., Bienz, K. A., Eckert, J., & Zingernagel, R. M. (Eds.). (2001). *Medical Microbiology* (10th ed.). Stuttgart, Germany: Georg Thieme Verlag.
- 4 Enoch, D. A., Simpson, A. J., & Kibbler, C. C. (2004). Predictive value of isolating *Pseudomonas aeruginosa* from aerobic and anaerobic blood culture bottles. *Journal of Medical Microbiology*, 53(Pt 11), 1151-1154.
- 5 Young, R. S., Deal, P. H., & Whitfield, O. (1968). The response of spore-forming vs. nonspore-forming bacteria to diurnal freezing and thawing. *Space Life Sciences*, 1(1), 113-117.
- 6 Shellito, J., Nelson, S., & Sorensen, R. U. (1992). Effect of pyocyanine, a pigment of *Pseudomonas aeruginosa*, on production of reactive nitrogen intermediates by murine alveolar macrophages. *Infection and Immunity*, 60(9), 3913-3915.
- 7 Palumbo, S. A. (1972). Role of iron and sulfur in pigment and slime formation by *Pseudomonas aeruginosa*. *Journal of Bacteriology*, 111(2), 430-436.
- 8 Liu, P. V. (1974). Extracellular toxins of *Pseudomonas aeruginosa*. *The Journal of Infectious Diseases*, 130 Suppl(0), S94-9.
- 9 Stover, G. B., Drake, D. R., & Montie, T. C. (1983). Virulence of different *Pseudomonas* species in a burned mouse model: tissue colonization by *Pseudomonas cepacia*. *Infection and Immunity*, 41(3), 1099-1104.
- 10 LIU, P. V., & MERCER, C. B. (1963). Growth, Toxigenicity and Virulence of *Pseudomonas Aeruginosa*. *The Journal of Hygiene*, 61, 485-491.
- 11 Feldman, M., Bryan, R., Rajan, S., Scheffler, L., Brunnert, S., Tang, H., & Prince, A. (1998). Role of flagella in pathogenesis of *Pseudomonas aeruginosa* pulmonary infection. *Infection and Immunity*, 66(1), 43-51.
- 12 Banerjee, D., & Stableforth, D. (2000). The treatment of respiratory pseudomonas infection in cystic fibrosis: what drug and which way? *Drugs*, 60(5), 1053-1064.
- 13 Mena, K. D., & Gerba, C. P. (2009). Risk assessment of *Pseudomonas aeruginosa* in water. *Reviews of Environmental Contamination and Toxicology*, 201, 71-115. doi:10.1007/978-1-4419-0032-6_3
- 14 Pruitt, B. A., Jr, McManus, A. T., Kim, S. H., & Goodwin, C. W. (1998). Burn wound infections: current status. *World Journal of Surgery*, 22(2), 135-145.
- 15 Zaidi, T. S., Priebe, G. P., & Pier, G. B. (2006). A live-attenuated *Pseudomonas aeruginosa*

vaccine elicits outer membrane protein-specific active and passive protection against corneal infection. *Infection and Immunity*, 74(2), 975-983. doi:10.1128/IAI.74.2.975-983.2006

- 16** Nadeem, S. G., Qasmi, S. A., Afaque, F., Saleem, M., & Hakim, S. T. (2009). Comparison of the in vitro susceptibility of Clinical isolates of *Pseudomonas aeruginosa* in a local hospital setting in Karachi, Pakistan. *British Journal of Medical Practitioners*, 2(4), 35-39.
- 17** Ishihara, S., Takino, M., Okada, Y., & Mimura, K. (1995). Septic shock due to *Pseudomonas aeruginosa* in a previously healthy woman. *Intensive Care Medicine*, 21(3), 226-228.
- 18** Pimay, J. P., Bilocq, F., Pot, B., Cornelis, P., Zizi, M., Van Eldere, J., Deschaght, P., Vaneechoutte, M., Jennes, S., Pitt, T., & De Vos, D. (2009). *Pseudomonas aeruginosa* population structure revisited. *PloS One*, 4(11), e7740. doi:10.1371/journal.pone.0007740
- 19** Banerjee, A., & Dangar, T. K. (1995). *Pseudomonas aeruginosa*, a facultative pathogen of red palm weevil, *Rhynchophorus ferrugineus*. *World Journal of Microbiology and Biotechnology*, 11, 618-620.
- 20** Clifton, I. J., & Peckham, D. G. (2010). Defining routes of airborne transmission of *Pseudomonas aeruginosa* in people with cystic fibrosis. *Expert Review of Respiratory Medicine*, 4(4), 519-529. doi:10.1586/ers.10.42
- 21** Ziady, L. E., & Small, N. (Eds.). (2004). *Prevent and Control Infection*. Cape Town, South Africa: Juda and Co Ltd.
- 22** Speert, D. P., Campbell, M. E., Henry, D. A., Milner, R., Taha, F., Gravelle, A., Davidson, A. G., Wong, L. T., & Mahenthiralingam, E. (2002). Epidemiology of *Pseudomonas aeruginosa* in cystic fibrosis in British Columbia, Canada. *American Journal of Respiratory and Critical Care Medicine*, 166(7), 988-993.
- 23** *Laboratory Safety Manual* (1993). (2nd ed.). Geneva: World Health Organization.
- 24** Burdon, D. W., & Whitby, J. L. (1967). Contamination of hospital disinfectants with *Pseudomonas* species. *British Medical Journal*, 2(5545), 153-155.
- 25** Nail, S. L., & Akers, M. J. (Eds.). (2002). *Development and Manufacture of Protein Pharmaceuticals*. New York, NY, USA: Kluwer Academic / Plenum Publishers.
- 26** Kramer, A., Schwebke, I., & Kampf, G. (2006). How long do nosocomial pathogens persist on inanimate surfaces? A systematic review. *BMC Infectious Diseases*, 6, 130. doi:10.1186/1471-2334-6-130
- 27** Leung, D. K. C., Mok, W. F. M., Yu, D. M. W., & Au, T. C. (2001). Use of distilled white vinegar dressing supplemental to oral antibiotics in the management of *Pseudomonas aeruginosa* exit site infection in continuous ambulatory peritoneal dialysis patients. *Hong Kong Journal of Nephrology*, 3(1), 38-40.
- 28** Xu, J., Moore, J. E., Murphy, P. G., Millar, B. C., & Elborn, J. S. (2004). Early detection of *Pseudomonas aeruginosa*--comparison of conventional versus molecular (PCR) detection directly from adult patients with cystic fibrosis (CF). *Annals of Clinical Microbiology and Antimicrobials*, 3, 21. doi:10.1186/1476-0711-3-21
- 29** Public Health Agency of Canada. (2004). In Best M., Graham M. L., Leitner R., Ouellette M. and Ugwu K. (Eds.), *Laboratory Biosafety Guidelines* (3rd ed.). Canada: Public Health Agency of Canada.