



Risk factors for *Pseudomonas aeruginosa* acquisition in intensive care units: a prospective multicentre study

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SUMMARY

Background: *Pseudomonas aeruginosa* is a major nosocomial pathogen in intensive care units (ICUs); however, endogenous versus exogenous origin of contamination remains unclear.

Aim: To identify individual and environmental ICU risk factors for *P. aeruginosa* acquisition.

Methods: A five-month prospective multicentric study was performed in ten French ICUs. Adult patients hospitalized in ICU for ≥ 24 h were included and screened for *P. aeruginosa* colonization on admission, weekly and before discharge. *P. aeruginosa* acquisition was

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defined by a subsequent colonization or infection if screening swabs on admission were negative. Water samples were obtained weekly on water taps of the ICUs. Data on patient characteristics, invasive devices exposure, antimicrobial therapy, *P. aeruginosa* water and patient colonization pressures, and ICU characteristics were collected. Hazard ratios (HRs) were estimated using multivariate Cox model.

Findings: Among the 1314 patients without *P. aeruginosa* on admission, 201 (15%) acquired *P. aeruginosa* during their ICU stay. Individual characteristics significantly associated with *P. aeruginosa* acquisition were history of previous *P. aeruginosa* infection or colonization, cumulative duration of mechanical ventilation and cumulative days of antibiotics not active against *P. aeruginosa*. Environmental risk factors for *P. aeruginosa* acquisition were cumulative daily ward 'nine equivalents of nursing manpower use score' (NEMS) [hazard ratio (HR): 1.47 for ≥ 30 points; 95% confidence interval (CI): 1.06–2.03] and contaminated tap water in patient's room (HR: 1.76; CI: 1.09–2.84).

Conclusion: Individual risk factors and environmental factors for which intervention is possible were identified for *P. aeruginosa* acquisition.

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Introduction

Pseudomonas aeruginosa is a ubiquitous micro-organism responsible for severe hospital-acquired infections.^{1–3} Length of stay, severity of underlying disease and exposure to invasive procedures but also bacterial adherence and virulence factors, and antimicrobial drug resistance are associated with *P. aeruginosa* acquisition in ICUs.^{4–6} Those risk factors were identified in single centre studies which most often focused on antimicrobial-resistant *P. aeruginosa* and rarely took into account the context of care delivery, i.e. colonization pressure or nurse staffing.⁷ However, patient case-mix, ecology, organization, and practices vary across ICUs. Endogenous origin was regarded as the most relevant route of *P. aeruginosa* infection, but, during the last ten years, a significant proportion of *P. aeruginosa* isolates have been shown to stem from the ICU environment (moist reservoirs) and from cross-transmission.^{8–11} Nseir *et al.* reported that the risk of multidrug-resistant *P. aeruginosa* acquisition was higher when the previous occupant of the room was colonized by such *P. aeruginosa*.¹² The relevance of exogenous reservoirs and the importance of cross-transmission were documented during outbreaks but their importance in non-epidemic situations remains controversial. A pilot study suggested that environmental factors might be associated with *P. aeruginosa* acquisition.⁶ Unlike patient and pathogen characteristics, environmental factors such as nursing workload or contamination of water taps could be modified. A prospective cohort study in ten French ICUs, DYNAPYO (Dynamics of Acquisition of *Pseudomonas aeruginosa*), was performed to evaluate the respective contributions of individual and ICU environmental risk factors for *P. aeruginosa* acquisition.

Methods

Study design

A prospective five-month observational survey was performed from February to October 2009 in ten ICUs from eight French healthcare facilities including four medical, two surgical and four mixed (medical and surgical) ICUs. University

hospitals of Besançon and Lyon included two ICUs each, university hospitals of Bordeaux, Garches, Montpellier and Paris, and general hospitals of Lens and Tourcoing included one ICU. These ICUs each had between nine and 20 beds and from 10 to 47 water taps. The average length of patient stay was eight to 16 days and ICUs admitted from 27 to 35 patients per month. Specific trained healthcare professionals were identified in each centre for the collection of data, which were entered on to a secured online electronic form. There was no policy to isolate patients with *P. aeruginosa* in each of the study ICUs, and changes such as to infection control initiative or antimicrobial stewardship were not implemented during the course of the study.

Exposure measurements

Every adult patient admitted for >24 h was included and had screening samples (oropharyngeal, rectal swab, and tracheobronchial aspirate) with 48 h of admission and thereafter once a week, and before discharge. The study was approved by the local ethics committees. Ceftrimide agar was inoculated and identification of *P. aeruginosa* was performed after 24 and 48 h of aerobic incubation at 42 °C.

Water samples were performed weekly on water taps of the ICUs. Samples were taken on the morning before use, without prior disinfection; taps were opened and the first 250 mL flush of water collected immediately into a sterile flask with 5 mg sodium thiosulphate (pre-flush sample), the aerator was not removed but swabbed and the swab broken into the water sample; samples were processed by membrane filtration: 100 mL were filtered through a 0.45 µm pore size membrane filter; membranes were cultured aerobically on to ceftrimide agar plates at 37 °C and examined for growth after 22 ± 4 and 44 ± 4 h; any colonies that grew on ceftrimide agar were identified using the API 20NE identification system (bioMérieux, Marcy l'Etoile, France).

Patients found to be positive within the first 48 h of admission were considered to have imported *P. aeruginosa*. Acquisition was defined by a subsequent positive swab culture or *P. aeruginosa* infection during ICU stay if, on admission, screening swabs were negative and the patient was not infected yet by *P. aeruginosa*.

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