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Evidence for Spread of a Clonal Strain of *Pseudomonas aeruginosa* among Cystic Fibrosis Clinics

Recent advances in molecular typing techniques have led to the identification of a dominant clonal strain of *Pseudomonas aeruginosa* within several cystic fibrosis (CF) clinics (2–6, 8, 9). These strains have been described as "hypertransmissible," and "patient-to-patient" spread has been implied. We report the first isolation of a genetically identical clone of *P. aeruginosa* from five CF clinics along the Eastern Seaboard of Australia separated by a total distance of 1,800 km. In three of these clinics, the strain has been identified as the dominant strain.

Using a combination of pulsed-field gel electrophoresis (PFGE) and random amplified polymorphic DNA assays, we previously identified a dominant clone (pulsotype I) of *P. aeruginosa* affecting 55% of patients capable of expectorating a sputum sample in a large pediatric/adolescent CF clinic in Melbourne (2). This strain has been shown to be genetically distinct from the epidemic strains found in the United Kingdom in Liverpool (8) and Manchester (6).

Subsequent screening using only PFGE (1) has identified pulsotype I as being the dominant strain in a large adult CF clinic 800 km away in Sydney. Thus far, 107 *P. aeruginosa* isolates from 32 patients have been tested. Sixteen (50%) of these patients have been identified as carrying pulsotype I. In addition, three of eight (38%) patients thus far tested at a Sydney pediatric CF center have also been shown to carry pulsotype I.

Eight (five adults, three pediatric patients) of 100 patients tested a further 1,000 km away in Brisbane (i.e., 1,800 km north of the original site) have also been shown to carry pulsotype I, although this was not the dominant strain at these centers. Four of these patients are known to have relocated from Sydney. No association with patients from either Sydney or Melbourne could be identified for the remaining patients.

Exchange of representative samples between the laboratories at the three clinics has confirmed the presence of pulsotype I in each of the centers. In general, pulsotype I exhibits multiple antibiotic resistance and frequently has a mucoid phenotype. No definite association with increased virulence has been established.

Previously it had been thought that patients with CF were generally infected for prolonged periods by unique lineages of environmentally acquired *P. aeruginosa* (7). More recent studies have identified dominant or hypertransmissible strains within a single clinic setting (2–6, 8, 9).

This is the first report of the same strain of *P. aeruginosa* being identified in patients from five CF clinics in three geographically dispersed regions separated by large distances. In three of these clinics, pulsotype I has been found to be the dominant strain, while preliminary data from a fourth pediatric center suggest that it may also be the dominant strain at that site.

Despite extensive environmental testing both within and outside of the hospital environments, we have been unable to identify any environmental or nosocomial source of pulsotype I.

The isolation of the same pulsotype from several CF clinics separated by large distances, its dominance (approximately 50% of patients) in a number of these clinics, and the failure to isolate this pulsotype from either the hospital or external environment are strongly indicative of *P. aeruginosa* being transmissible from patient to patient.

This suggests that *P. aeruginosa* cross-infection may be more common than previously believed. We recommend more widespread use of molecular surveillance for *P. aeruginosa* in large CF clinics. If cross-infection is shown to be present, then cohort segregation of patients harboring *P. aeruginosa* and those that are not, as well as patients with different strains of *P. aeruginosa*, may be advisable.

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David Armstrong
Department of Respiratory Medicine
Monash Medical Center
Melbourne, Australia 3168

Scott Bell
Department of Thoracic Medicine
The Prince Charles Hospital
Brisbane, Australia 4035

Michael Robinson*
Peter Bye
Cystic Fibrosis Center
Royal Prince Alfred Hospital
Sydney, Australia 2050

Barbara Rose
Colin Harbour
Crystal Lee
Helen Service
Department of Infectious Diseases
University of Sydney
Sydney, Australia 2006

Michael Nissen
Melanie Syrmis
Claire Wainwright
Royal Children's Hospital
Brisbane, Australia 4029

*Phone: (61-2) 9515-6017
Fax: (61-2) 9515-8196
E-mail: mr@mail.med.usyd.edu.au