WS18.1 Failure of eradication therapy of *Pseudomonas aeruginosa* in cystic fibrosis: watch the nose

M.C. Berkhout[1], L.H. el Bouazzaoui[1], E. Rijntjes[2], R.W. Brimicombe[3], H.G.M. Heijerman[1]. [1]*Haga Teaching Hospital, Pulmonary Diseases, The Hague, Netherlands;* [2]*Haga Teaching Hospital, Otorhinolaryngology, The Hague, Netherlands;* [3]*Haga Teaching Hospital, Microbiology, The Hague, Netherlands*

Eradication therapy of an early *Pseudomonas aeruginosa* lung infection in CF is currently standard of care. Treatment with tobramycin inhalation solution is proven to be effective. However, this therapy is not effective in all patients and a significant number of patients are reinfected within a year after eradication [1]. Risk factors for failure of eradication therapy or reinfection after eradication need to be identified in order to reach a higher success of eradication therapy.

Objective: Comparison of *P. aeruginosa* in nasal and sputum cultures in adult patients with CF.

Methods: In 91 adult patients with CF nasal cultures were taken in two different ways. Under endoscopic control cultures from the left and right middle meatus were taken with a swab. Secondly nasal lavage was performed by inserting a sterile isotonic saline solution in both nostrils. The subject was asked to expectorate sputum on the same day.

Results: Of the 91 sputum cultures 43 were negative for *P. aeruginosa*. However, despite previous 'successful' eradication of *P. aeruginosa*, in 3 patients nasal cultures still displayed *P. aeruginosa*.

Conclusions: Eradication of *P. aeruginosa* in CF patients can appear successful according to sputum cultures, but nasal cultures can still reveal *P. aeruginosa*. Therefore, nasal cultures may be important in the determination of *P. aeruginosa* eradication in CF. Moreover, eradication therapy may be extended with anti-pseudomonial treatment of the sinonasal area to reach total eradication and decrease the risk of reinfection.

Reference(s)

[1] Ratjen F, Munck A, Kho P et al. Treatment of early *Pseudomonas aeruginosa* infection in patients with cystic fibrosis: the ELITE trial. Thorax 2010; 65: 286–291.

WS18.2 Persistent elevation of antibodies against *Pseudomonas aeruginosa* following successful eradication?

M. Kappler[1], G. Heilig[1], T. Wesselak[1], C. Kröner[1], A.-C. Grimmelt[1], M. Feilcke[1], M.-T. Rieger[1], B. Woitsch[1], A. Irnstetter[1], J. Hildebrandt[1], J. Glöckner-Pagel[1], M. Griese[1]. [1]*Dr. von Hauner Children's Hospital, University of Munich, Cystic Fibrosis, Munich, Germany*

Objective: To define the 3 year course of antibodies against *P. aeruginosa* (alkaline protease, elastase, and exotoxin A) in CF patients following first microbiological detection in respiratory samples and the success of eradication therapy.

Methods: All CF patients with first-time *P. aeruginosa* detection between 01.01.2005 and 31.12.2008 were identified. Microbiological and serological results of the year before and the 3 following years were retrospectively collected. Antibody titres <500 U were defined as negative, giving a cumulative antibody score between 0 and 3. Success of eradication therapy was defined as complete absence of *P. aeruginosa* in the third year after first detection.

Patients: Of 382 CF, 59 patients with first-time *P. aeruginosa* detection were identified (mean age 8.4). In 40 of these serological and microbiological data were complete and they were analysed in detail.

Results: Eradication was successful in 25 patients (62.5%), 10 patients were intermittently infected, 5 patients were chronically infected. Positive antibody titres preceded microbiological detection in 8 patients (20%). Cumulative antibody titres in the successfully treated patients were rising insubstantially and declining after the first year (mean values in the first, second, third year: 0.23, 0.14, 0.13) but were significantly elevated in intermittently infected patients (0.63, 0.83, 0.75) and in chronically infected patients (1.4, 2.0, 2.1).

Conclusion: Persistent elevation of antibodies against *P. aeruginosa* indicated the failure of eradication therapy. The intensity of anti-pseudomonal antibiotic therapy should be driven not only by microbiological but also by antibody results.

WS18.3 A twenty-five year outbreak of *Pseudomonas aeruginosa*: identification of the Prairie Epidemic Strain (PES)

B.A. Glezerson[1], C.D. Sibley[1], K.A. Sibley[1], C.H. Mody[1], M.G. Surette[1], H.R. Rabin[1], M.D. Parkins[1]. [1]*The University of Calgary, Calgary, Canada*

Introduction: Transmissible strains (TS) of *P. aeruginosa* (PA) have been described in cystic fibrosis (CF) and can be associated with a worse prognosis. We sought to determine the prevalence and stability of PA strains within the Calgary Adult CF Clinic using our comprehensive strain databank.

Methods: Initial isolates (II) (from initial adult clinic visit) and final isolates (FI) (from most recent clinic visit, or transplantation/death) were examined by pulsed-field gel electrophoresis (PFGE) and compared against a collection of known TS and strains from patients with non-CF bronchiectasis (nCF-B).

Results: We generated 636 PFGE profiles from 111 CF patients. Of 108 patients with chronic PA infection, 66 patients had both II and FI spanning 674 patient-years of clinical/microbiologic follow-up available, whereas 19 patients had only II and 23 FI available for assessment. Only one patient was infected with a known TS, the Liverpool Epidemic Strain (LES). However, a novel, clonally related complex, we termed the Prairie Epidemic Strain (PES), was identified in 29% (31/108) of our cohort. This strain was present at enrollment in our clinic in 76% of patients. Five patients acquired this infection during 58 patient-years of follow-up. PES is distinct from previously described TS and was not observed in 17 patients with nCF-B.

Conclusion: PES has been present in our clinic population since at least 1987, is unique to CF, establishes chronic infection, can displace other PA strains, and has been found in patients at the time of transition from other CF clinics across the Prairies. Studies are currently underway to evaulate the clinical implications of PES infection.

WS18.4 Heterogeneity of shared *Pseudomonas aeruginosa* (*Pa*) strains between Australian states and centres in cystic fibrosis (CF)

T.J. Kidd[1], K.A. Ramsay[1], H. Hu[2,3], P.T. Bye[4], M.R. Elkins[4], G.B. Marks[5], C.E. Wainwright[1,6], P.J. Robinson[7], K. Grimwood[1], S.C. Bell[1,8], The ACPinCF Investigator Group. [1]*Queensland Children's Medical Research Institute, University of Queensland, Brisbane, Australia;* [2]*University of Sydney, Department of Infectious Diseases and Immunology, Sydney, Australia;* [3]*Macquarie University, Australian School of Advanced Medicine, Sydney, Australia;* [4]*Royal Prince Alfred Hospital, Department of Respiratory Medicine, Sydney, Australia;* [5]*The Woolcock Institute of Medical Research, Sydney, Australia;* [6]*Royal Children's Hospital, Department of Respiratory Medicine, Brisbane, Australia;* [7]*Royal Children's Hospital, Department of Respiratory Medicine, Melbourne, Australia;* [8]*The Prince Charles Hospital, Thoracic Medicine, Brisbane, Australia*

Background: A recent national survey of 18 Australian CF centres involving 983 patients with *Pa* found that >60% shared indistinguishable genotypes. Although most shared strains involved small clusters and few CF centres, 2 strains (AUST-01 and AUST-02) were widely dispersed and encountered frequently. It is uncertain if the high prevalence of these 2 strains was associated with geographic location and/or local infection control practices.

Objectives:

i. To examine shared *Pa* strain diversity across Australian CF centres and geographic regions;
ii. To determine the impact of infection control and molecular typing surveillance on AUST-01 and AUST-02 strain prevalence.

Methods: Geospatial relationships between *Pa* genotypes and residential postcodes were assessed using ArcGIS software. Infection control practices and molecular typing surveillance strategies for all centres were also reviewed.

Results: Overall, 73% (161/220) of AUST-01 detections were in South-Eastern Australia, whereas 87% (150/173) of patients with AUST-02 resided in Queensland and Western Australia. Despite these geographic trends, in some cities there were differences in detection rates between CF centres. For example, AUST-01 was found in 2% and 40% of children attending 2 Brisbane CF centres, QLD/01 and QLD/03, respectively. Lowest rates of AUST-01 and AUST-02 were recorded in paediatric centres with strict infection control practices and molecular typing surveillance.

Conclusions: The distribution of shared *Pa* strains in Australian CF patients cannot be explained fully by geographic location. Strict infection control is associated with a reduced prevalence of the most commonly encountered shared strains.