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2-hour Oral Session

Prevalence and characterization of unusual *Staphylococcus aureus* strains with a mucoid phenotype recovered from the airways of cystic fibrosis patients

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Objectives: Cystic fibrosis (CF) patients suffer from chronic respiratory airway infections. *Staphylococcus aureus* is frequently isolated from the respiratory tract already in early childhood and often persists for extended periods. Mucoid bacterial colony morphology has been described for *Pseudomonas aeruginosa*, the most prominent pathogen in CF, but not for *S. aureus*. Recently, we identified mucoid isolates also for S. aureus. To determine the prevalence and the underlying mechanism of mucoid S. aureus isolates we used S. aureus isolates collected during two independent studies including 371 CF patients [a prospective multicenter study (n=195) and a longitudinal prospective study of two CF centers in Münster, Germany (n=176)].

Methods: To determine the underlying mechanism of mucoidy, the *ica* promoter region of all isolates and the entire *ica* operon of selected strains were sequenced. Biofilm formation was determined by a microtiter plate assay. Mucoid and normal isolates of one patient were characterized in terms of capsule expression and phagocytosis by neutrophils.

Results: From the respiratory specimens of 8 patients (prevalence of 2.2%) mucoid and mucoid-sticky *S. aureus* isolates were recovered. In contrast to normal *S. aureus,* mucoid and mucoid-sticky isolates were strong biofilm producers. Mucoidy was associated with a 5-bp deletion in the *ica* promoter region, resulting in overproduction of biofilm. Subsequent non-mucoid isolates, which also carried a 5-bp deletion, harbored a compensatory mutation in *icaC*. Transformation of a 5-bp deletion carrying normal strain with a vector expressing intact *icaC* restored mucoidy. In contrast to the normal phenotype, mucoid strains were protected against phagocytosis by neutrophils.

Conclusions: Although mucoid *S. aureus* strains are rarely identified during chronic airway infection in CF, biofilm hyper-production seems to be an effective strategy for protection against phagocytosis by neutrophils.