Objectives: The present study explored the possible protective effect of IgY on PA lung infection in vivo.

Methods: In vivo model of acute lung infection: Balb/c mice were anaesthetized with isoflurane and PA01 vaccine strain specific (S-IgY) or control (C-IgY) was inoculated intranasally. Mice were sacrificed after 2, 6 and 24h and lungs removed aseptically, weighted and suspended in PBS. A blinded observer engaged a clinical scoring system (0–5) of the mice. Lungs were homogenized, serially diluted and cultured on Conradii-Drigalski medium for estimation of bacterial load.

Results: Relative lung weight: Lung weights in the S-IgY treated group were significantly reduced 24h post-infection compared to PBS controls (p < 0.03). No significant difference between C-IgY and PBS groups were observed.

Clinical symptom score: The clinical score was significantly lower in the S-IgY group compared to controls after 6h (C-IgY: p < 0.05, PBS: p < 0.05). After 24h the clinical score in the S-IgY was reduced additionally compared to controls (PBS: p < 0.002, C-IgY: p < 0.04). No significant difference between C-IgY and PBS groups were observed.

Quantitative bacteriology: The bacterial load of S-IgY treated mice was significantly reduced 24h post-infection compared to PBS group (p < 0.02) and C-IgY (p < 0.03) and further reduced 6h post-infection compared to both control groups (PBS: p < 0.0001, C-IgY: p < 0.03). After 24h the lung bacteriology in S-IgY treated mice was reduced by 2 logs compared to PBS (p < 0.0001) and C-IgY (p < 0.0002). groups

Conclusion: The present results imply that anti-PA IgY antibodies protects against PA lung infection due to readily bacterial clearance in the airways.

Objectives: Anti-Pseudomonas aeruginosa IgY antibodies promote bacterial clearance in a murine pneumonia model.

Methods: To evaluate, as marker of early stages of P. aeruginosa infection, the anti-Pa immuneresponsein people with CF. The objective of this study was to investigate if pro-inflammatory changes in the lung affect gut inflammation.

Methods: Plasma levels of PGE2 in CF patients (n = 25) was detected. CF bronchial epithelial cells (AF508 homozygote, CFBE41o−) and human bronchial epithelial cells (16HBE132) were seeded at 2 × 104 cells/ml. Cells were stimulated with PGE2 and cell supernatant was harvested. HT29 colon epithelial cells were seeded at 2 × 106 cells/ml and treated with harvested supernatant. Cell proliferation of HT29 cells was determined and changes in cytokine levels of IL-6, IL-8 and TNFα were detected by ELISA.

Results: Suprantant from CF lung cells augmented IL-6, IL-8 and TNFα production by HT29 cells. Relative to HT29 cells cultured in normal bronchial 16HBE132 epithelial cells, secretion of IL-6, IL-8 and TNFα by HT29 cells incubated with CFBE41o− CF cell supernatant was significantly increased 2.7 fold, 1.8 fold and 1.4 fold respectively. Culture of the cells in PGE2-treated CF cell supernatant relative to PGE2-treated normal bronchial epithelial cells further increased IL-6, IL-8 and TNFα. There was no significant change in cell proliferation of HT29 intestinal epithelium cells.

Conclusion: This study shows that inflammatory changes in the CF lung can modulate cytokine production in the gut. Thus, independent of active CF lung infection this mechanism may regulate intestinal inflammation.

Objectives: How frequent is Clostridium in our CF patients?

Methods: Retrospective analysis over a ten years period was done, using the information from our CF center’s database. In all the patients, only presentation with diarrhea occurred during antibiotic therapy were taken into consideration. Diagnosis test for C. difficile infection was performed by enzyme immunoassay for detection of toxins A and B.

Results: Over a ten years period, 308 patients with cystic fibrosis were admitted in our clinic; only five of them (1.62%) were diagnosed with C. difficile infection. Patients were diagnosed in the last 4 years, by the detection e of toxin A or toxin A and B (in 2 patients) in the presence of diarrhea; they had a favorable outcome, with a good response to treatment (metronidazole in 3 cases, metronidazole and vancomycin in 2 cases). All patients had chronic Pseudomonas aeruginosa infection and received more than fourteen days of antibiotics.

Conclusion: Clostridium difficile infection should be considered for evaluation in cystic fibrosis patients with diarrhea who receive antibiotics. Special attention is necessary when antibiotic is given for a long time, as commonly recommended in cystic fibrosis patients.