Methods 100 adults with CF provided sputum, nose- and throat-swabs every 2 months between December 2010 and June 2011 within a prospective observational study. Samples were repeated if new respiratory symptoms developed between regular visits. Specimens were analysed using PCR assays for adenovirus, influenza, metapneumovirus, parainfluenza, respiratory syncytial virus and rhinovirus. Positive viral identification rates for each specimen type were compared. FEV1, inflammatory markers and symptoms scores for upper respiratory tract infection (URTIs) and pulmonary exacerbation (PEx) were recorded at each visit.

Results 210 sets of samples were collected. A respiratory virus was identified at 52 (24.8%) visits of which rhinovirus accounted for 62%, influenza A 10.5%, metapneumovirus 9% and influenza B 7%. Among virus-positive cases, sputum was positive in 34 (65%), nose swab in 25 (48%) and throat swab in 21 (40%). A single specimen type was positive in 52 (62%) cases; two specimens in 12 (23%) and all three specimens in only 8 (15%). Sputum alone was positive in 17 (33%) cases, nose-swab alone in 8 (15%) and throat-swab alone in 7 (13%). An increasing number of positive specimens was associated with higher mean (SD) URTI scores (4.9 (5.6) vs 0.7 (6.6) vs 10.5 (5.5) for 0, 1 and ≥2 positive specimens respectively; p = 0.046 for = 2 vs 1 specimens) and higher PEx scores (2.2 (2.8) vs 3.2 (2.2) vs 5.1 (1.5); p = 0.002). FEV1, CRP and WCC were similar between these groups. There were no significant differences in lung function, symptoms or inflammatory markers when viruses were detected in sputum compared with the upper airways.

Conclusions Sputum is superior to nose- and throat-swabs for the diagnosis of respiratory viruses in adults with CF but all three are required for optimal identification rates. Viral positivity in =2 specimens is associated with higher upper and lower respiratory symptom scores.

Results PA readily induced apoptosis and cell death in human DCs, with cytotoxicity seen within 3 h of infection. Induction of apoptosis by PA was an active process requiring live organisms, but was not dependent on a functional type III secretion system. A significant decrease in viable DCs was seen in response to infection with clinical PA strains at 3 h and 20 h compared with laboratory PA103 strains (p < 0.05 and p < 0.001, respectively). Due to increased cytotoxicity of clinical PA isolates, post-infection DCs demonstrated no increase in co-stimulatory molecule expression compared with uninfected DCs (p > 0.05).

Conclusions These data demonstrate that human dendritic cells are susceptible to apoptosis induced by P aeruginosa, with clinical isolates of PA demonstrating high levels of cytotoxicity, and a subsequent reduction in DC antigen-presenting capacity. Elimination of these important antigen-presenting cells could lead to impairment of immune responses and thus a factor in the establishment of chronic PA colonisation in the CF lung.