Figure 1. Patient serum IgG titre specific for LPS isolated from B4 determined by ELISA.

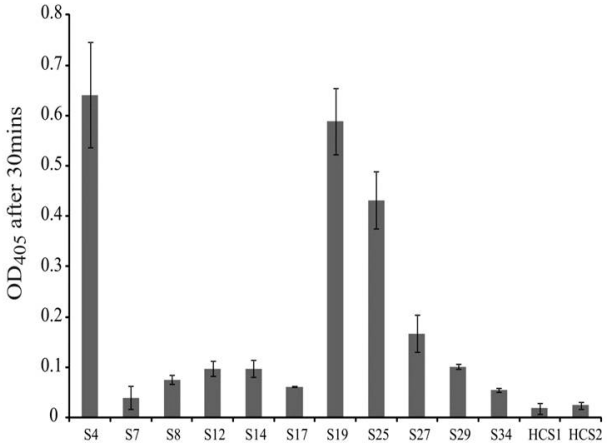

Figure 1: Only the 3 patients who had impaired serum killing (S4, S19 and S25) had high antibody titres to the LPS indicating that the LPS in strains isolated from these patients was responsible for high levels of IgG2 that impairs killing of the PA strain.

Abstract S105 Figure 1

measured by flow cytometry. Using RT-PCR, we observed an increase of PDL1 mRNA after X31 infection suggesting that the expression of this protein is transcriptionally regulated. In addition, we saw an increase in type I interferon expression by MDMs in response to X31 infection, but no expression of IFNγ. In contrast we observed a trend towards decreased expression of IL-10 mRNA. In further experiments, infection of alveolar macrophages with X31 also caused significant increases in HLA-DR and PDL1 cell surface expression.

Conclusions These data indicate that, in contrast to HIV infection of macrophages[2] influenza-induced expression of PDL1 may not be regulated by IL-10 in human macrophages.

1. Erickson et al (2012) J Clin Invest doi: 10.1172/JCI62860.
2. Rodriguez-Garcia, et al. (2011) J Leukoc Biol 89(4):507–15.

S107 EFFECTS OF EXPOSURE TO CIGARETTE SMOKE CONDENSATE ON PNEUMOCOCCAL GENE EXPRESSION IN RELATION TO BIOFILM FORMATION

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Although cigarette smoking is well-recognised as being the strongest independent risk factor for development of invasive pneumococcal disease, little is known about its direct effects on the expression of virulence factors by the pneumococcus. The primary objectives of the current study were to investigate the effects on gene expression in relation to biofilm formation following exposure of the pneumococcus to cigarette smoke condensate (CSC). Strain 172 (serotype 2–3F) of the pneumococcus was exposed to CSC (20–160 µg/ml) for 16 hours at 37°C in 6-well tissue culture plates to facilitate adherence and biofilm formation. Following incubation, biofilm associated with the adherent bacteria was stained with 0.1% crystal violet, extracted and assayed spectrophotometrically. In the case of gene expression, the bacteria (2 x 10^8 colony forming units/ml) were exposed to CSC (160 µg/ml) or solvent for 60 min at 37°C, after which RNA was extracted and converted to cDNA by reverse transcriptase-PCR (RT-PCR) and whole genome gene expression profiles determined using the *Streptococcus pneumoniae* TIGR4 DNA Microarray Chip. Six microarrays were performed (in triplicate for the control and CSC-treated systems). Exposure of the pneumococcus to CSC resulted in dose-related augmentation of biofilm formation which attained statistical significance ($P<0.05$) at concentrations of 80 and 160 µg/ml. CSC-mediated augmentation of biofilm formation was associated with selective and significant up-regulation of the genes encoding the two-component 11 system (TCS11), consisting of the genes *hk11* (histidine kinase) and its cognate response regulator, *rr11*, which has been implicated in biofilm formation by *S. mutans*. Relative to the non-exposed control system, the respective levels of up-regulation of each gene were 19.7- and 22.5-fold ($P<0.001$ and $P<0.0006$). Induction of biofilm formation, probably as a stress response resulting in activation of TCS11, may underpin cigarette smoke-mediated colonisation of the respiratory tract by the pneumococcus.

Evaluating impact in pulmonary rehabilitation

S108 THE EFFECT OF AN INTERDISCIPLINARY REHABILITATION PROGRAMME ON DAILY PHYSICAL ACTIVITY FOR PATIENTS WITH LESS ADVANCED COPD IN A PRIMARY CARE SETTING: A SYSTEMATIC REVIEW

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