the VAP and non-VAP groups (Abstract S77 Figure 1). Soluble TREM-1 levels were not significantly different between the groups. There was no difference in peripheral blood white cell count, CRP, expression of CD11b, L-selectin and inflammatory cytokines between VAP and disease controls.

Conclusion The BALF/blood ratio of monocyte surface TREM-1 discriminates between the VAP and non-VAP groups. Measurement of surface TREM-1 using a two-compartment index may have utility in diagnosing VAP.

REFERENCES
mRNA levels showed no impairment to TNF in MyD88KD cells. implicating other members of the IL-1 family in the response of

A reduction in MyD88 signalling modulates specific epithelial cell responses to rhinovirus, and thus may be an important target to control acute inflammation induced by human viral pathogens.

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REFERENCE

PRIMARY TYPE II ALVEOLAR EPITHELIAL CELLS RESPOND DIFFERENTIALLY TO BACTERIAL VIRULENCE FACTORS

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1 O I Moncayo Nieto, 1 M Brittan, 2 S Wilkinson, 1 A Conway Morris, 1 K Dhaliwal, 1 W S Walker, 1 A J Simpson, 1 University of Edinburgh, Edinburgh, UK; 2 University of Swansea, Swansea, UK

Introduction and objectives The pathogens most commonly implicated in ventilator-associated pneumonia (VAP) are Pseudomonas aeruginosa and Staphylococcus aureus. Although a florid inflammatory response characteristically occurs in the alveolar space in VAP, the underlying mechanisms remain unclear, partly owing to a lack of adequate models of alveolar injury. We therefore sought to characterise the response of primary human type II alveolar epithelial (ATII) cells to virulence factors from these pathogens.

Methods Primary ATII cells were derived from seven patients undergoing surgical resection for lung cancer. Lung tissue was refrigerated overnight; flushed with saline; incubated with trypsin; diced; incubated with DNase I; and strained/filtered. Macrophages and fibroblasts were removed by adherence. The resulting cell population was centrifuged, washed, resuspended and plated onto tissue culture plates pre-coated with type I bovine collagen at $2 \times 10^6$ cells/ml. When cells achieved confluence medium was replenished and the following were added for 24 h: 100 ng/ml P. aeruginosa lipopolysaccharide (LPS); 10 ug/ml S. aureus lipoteichoic acid (LTA); 10 ug/ml S. aureus peptidoglycan (PGN); 10 ng/ml human recombinant tumour necrosis factor alpha (TNFα); or control medium. Supernatant was aspirated at 24 h and cytokines were measured by cytometric bead array.

Results Interleukin (IL)-1β, IL-6, IL-8 IL-10, IL-12p70 and TNFα were all detectable in control medium at 24 h. None of the measured cytokines were significantly altered by application of LPS or LTA. In contrast, PGN induced a significant rise in concentrations of IL-1β, IL-6, IL-8 IL-10 and TNFα. Addition of TNFs induced a significant increase in IL-6, IL-8 and IL-10. The only cytokine to be uniformly uninfluenced by stimulation was IL-12p70.

Conclusions In our hands primary ATII cells appeared to be unresponsive to P. aeruginosa LPS or to S aureus LTA. By contrast, S aureus PGN provoked a brisk and significant inflammatory response simultaneously affecting a range of cytokines. These data suggest that ATII cells have strikingly different responses to individual bacterial virulence factors. They further suggest that PGN (but not LTA) contributes, at least in part, to the florid inflammatory response seen in Staphylococcal pneumonia.

SURVIVAL OF HIV-INFECTED PATIENTS ADMITTED TO THE INTENSIVE CARE UNIT

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1 A G Adikha, 2 M Pavlou, 1 D Walker, 1 A J Copas, 1 S Batson, 1 G Edwards, 1 M Singer, 1 R F Miller, 1 University College Hospital, London, UK; 2University College London, London, UK

Background Several studies from USA and Europe have suggested the outcome for HIV-infected patients admitted to the intensive care unit (ICU) has improved, concurrent with both the introduction of highly active antiretroviral therapy (HAART) and
S80 The role of MyD88 in rhinovirus 1B infection

C A Stokes, E P Dick, J A Bennett, S Ismail, M R Edwards, I Sabroe and LC Parker

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