Poster sessions

Abstract P19 Figure 1  A549 cells plated on an iCelligence 8-well gold electrode coated plate were incubated with TNFR1 dAb™, a dummy dAb or Adalimumab™ for 1 h then exposed to exogenous TNF or vehicle control. Electrical impedance was measured continuously over 50 h. Trough normalised impedance was measured over 50 h post treatment (n = 3–5). Data are presented as mean ±SEM analysed by Kruskal-Wallis (Dunns). *p < 0.05, **p < 0.01

be due to disruption of epithelial junctional proteins; we speculate that this may alternatively be due to TNFR1 induced cell death.

P20 Delineating the contribution of formylated peptides and formyl peptide receptor 1 to the pathogenesis of acute lung injury

Introduction and objectives Neutrophils (PMNs) are a key component of the innate immune response to invading pathogens. They accumulate at sites of inflammation and infection, which are typically characterised by low oxygen tensions (e.g. in the acute respiratory distress syndrome (ARDS)). Human PMNs undergo constitutive apoptosis, their survival contingent upon pro-survival and pro-apoptotic signals derived from their microenvironment. Hypoxia profoundly delays PMN apoptosis, resulting in persistence of PMNs at inflammatory foci and this may perpetuate hypoxia-mediated lung injury. Given the importance of phosphoinositide 3-kinase (PI3K)-mediated signalling, we hypothesised that hypoxia-induced PMN survival may also involve PI3K-mediated signalling.

Methods Highly pure PMNs isolated from healthy volunteers were incubated for 20 h under normoxic (20 kPa) and physiologically relevant hypoxic (3 kPa) conditions with a pan-PI3-K inhibitor (LY294002 at 10 µM), a novel pan-Class I PI3-K inhibitor (ZSTK474 at 1 µM, 3 µM and 10 µM) or novel PI3-K Class I isoform-selective inhibitors (PI3-Kb at 1 µM; PI3-Kγ at 3 µM and 10 µM, or PI3-Kbγ at 3 µM). PMNs were also incubated in normoxia and hypoxia in the presence of GM-CSF (1 ng/ml) with the same panel of inhibitors, allowing comparison with GM-CSF mediated survival, which is largely PI3-K dependent. PMN apoptosis was assessed using two complementary techniques – morphology and flow cytometry following annexin V-FITC and propidium iodide staining.
Corrections

P20 - withdrawn


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