Results The analysis showed that there was an ICER (Incremental Cost-Effectiveness Ratio) of 32 000 GBP/QALY (Quality Adjusted Life Years) associated with high dose dry powder FP/S is not cost-effective. Extra control is achieved, treatment can be stepped down to the lowest option in the UK to maintain control of asthmatic patients stepped down from high dose FP/S 1000/100 μg vs extrafine BDP/F 400/24 μg. Additional analysis showed that there was an ICER of 85 200 GBP/QALY associated with high dose suspension formulation FP/S 1000/100 μg vs extrafine BDP/F 400/24 μg.

Conclusions BTS/SIGN guideline recommend that when asthma control is achieved, treatment can be stepped down to the lowest dose that maintains control. It was found that maintaining controlled patients on high dose FP/S is not cost-effective. Extrafine BDP/F 400/24 μg daily can be considered to be a cost-effective option in the UK to maintain control of asthmatic patients stepped down from high dose FP/S 1000/100 μg daily dry powder or suspension formulations and the magnitude of cost effectiveness is estimated to be highest when stepping down from the suspension formulation.

Cellular responses in the aetiology of COPD

PT15 CHRONIC DIESEL EXHAUST PARTICLE (DEP) EXPOSURE DIFFERENTIALLY ALTERS MONOCYTE DIFFERENTIATION AND FUNCTION IN HEALTHY CONTROLS COMPARED TO COPD

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Introduction and Objectives Alveolar macrophages are heavily implicated in the pathogenesis COPD. During chronic inflammation, macrophages mature continuously from infiltrating monocytes that are continually recruited to the airways. We have previously found DEP modulate life span and function of monocytes from healthy donors, but their effects on monocytes of people with COPD are unknown, and were therefore the subject of this study.

Methods Monocytes were purified from the blood of patients with GOLD II/III COPD and healthy age matched controls Monocyte-derived macrophages (MDMs) were generated in the presence or absence of DEP and their lifespan studied. Cytokine generation in response to TLR agonists and heat killed bacteria was assessed by ELISA and expression of CD14 was measured by FACS.

Results Chronic exposure of monocytes from patients with COPD to DEP concentrations above 10 μg/ml caused a significant reduction in cell survival. Lower concentration of chronic DEP exposure, as low as 1 μg/ml, caused impairment of cytokine responses to LPS and heat killed Escherichia Coli, and this phenotype was associated with a reduction in CD14 surface marker expression. However, COPD monocytes were generally more resistant to the effects of DEP compared to healthy control cells.

Conclusions In this study monocytes from healthy volunteers appeared to be more susceptible to the harmful effects of chronic DEP exposure compared to those from individuals with COPD. These findings reinforce the evidence that circulating leukocytes in COPD patients have altered phenotypes.

PT16 TESTING ANTIOXIDANT AND ANTI-INFLAMMATORY THERAPIES IN A COMPLEX LUNG TISSUE MODEL

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COPD is a disease of global importance and its primary cause airway inflammation as a consequence of cigarette smoking is well described. However, there remains a lack of effective therapies for this important condition. Animal models of disease are limited in their predictive utility and therefore creation of a complex, human disease model is an important step for testing new therapeutic interventions. We therefore established a tissue model of oxidative and inflammatory responses to relevant triggers—cigarette smoke and LPS and determined the impact of interventions in the optimised system.

Methods Human lung tissue explants from the resected lobes of six consented patients undergoing lobectomy were used. Uniform tissue explants were established on a novel culture system and then treated with CSE and LPS before the supernatants were collected and FeNO were higher than expected as was adherence to OCS and their predictive utility and therefore creation of a complex, human disease model is an important step for testing new therapeutic interventions. We therefore established a tissue model of oxidative and inflammatory responses to relevant triggers—cigarette smoke and LPS and determined the impact of interventions in the optimised system.