Figure: Lung function data according to the GLI-2012 ethnic-specific reference equations. Data from South Asian children were expressed according to the 'Black' Equation. The "other" group comprise children of "mixed ethnic backgrounds" and those not covered by the other 3 groups.

Abstract S33 Figure 1

Abstract S34 Table 1 Comparison of Lung Function: Children with SCD vs. healthy Black controls.

	Baseline Mean (95% CI) Diff (SCD – Health)	Bronchodilator Response Mean (95% CI) Diff (SCD–Health)
Resistance at 10Hz Z Score ^[2]	–0.2 (–0.4; 0.1)	0.3 (–0.2; 0.7)
sReff Z Score ^[3]	–0.3 (–0.6; 0.1)	0.5 (0.1; 0.8)
TLC Z Score ^[4]	–0.8 (–1.1; –0.5)*	
FVC Z Score ^[5]	–1.0 (–1.2; –0.7)*	0.2 (–0.1; 0.5)
FEV1 Z Score ^[6]	–1.2 (–1.5; –0.9)*	0.2 (–0.1; 0.5)

*p<0.001.

S35 DIFFERENT EARLY LIFE FACTORS ARE IMPORTANT IN THE DEVELOPMENT OF ATOPIC AND NON-ATOPIC ASTHMA

doi:10.1136/thoraxjnl-2012-202678.041

¹E Petley, ²KC Pike, ³HM Inskip, ⁴KM Godfrey, ²JSA Lucas, ⁴G Roberts, ³Southampton Women's Survey Study Group. ¹Clinical and Experimental Sciences, University of

Southampton Faculty of Medicine, Southampton, UK; ²NIHR Southampton Respiratory Biomedical Research Unit, Southampton University Hospitals Trust, Southampton, UK; ³Southampton Medical Research Council Lifecourse Epidemiology Unit, University of Southampton, Southampton, UK; ⁴Human DevelopmentHealth, University of Southampton Faculty of Medicine, Southampton, UK

Background Many factors have been related to the development of childhood asthma but there is inconsistency between studies.

Objective To understand how early life factors are linked to the development of the various asthma phenotypes at age 6 years in the Southampton Women's Survey (SWS) children's cohort.

Methods Data was collected from 940 children and their parents, primarily through questionnaires during pregnancy and at 6m, 1, 3 and 6 years. Prevalent asthma was defined by a doctor's diagnosis and a wheezing episode in the last year. Data was analysed using STATA v9. A relative risk analysis using a univariate approach was undertaken, followed by a multivariate analysis.

Results Both maternal (RR=1.61, p=0.041) and paternal (RR=2.05, p=0.002) atopic disease increased the risk of asthma at age 6 years. The risk increased with atopy, defined as a positive skin prick test, at 3 years (RR=3.05, p<0.001) and with wheeze in the first 3 years (RR=8.79, p<0.001). Episodes of bronchiolitis and chest infections were associated, in a dose-dependent manner, with the risk of asthma (RR=1.50, p=0.022). Predictors in the multivariate model were wheeze in the first 3 years (RR=10.74, p<0.001), atopy (RR=2.87, p<0.001) and maternal atopy (RR=2.22, p=0.011). When asthma at age 6 years was split into atopic and non-atopic asthma, the predictors were very different. Atopic asthma was associated with paternal atopy (RR=4.13, p=0.002), male sex (RR=2.56, p=0.030), atopy at 3 years (RR=10.31, p<0.001) and wheeze in the first 3 years (RR=5.91, p=0.004). In the multivariate analysis, the following were predictive: wheeze in the first 3 years (RR=13.55, p=0.012), atopy at 3 years (RR=10.13, p<0.001), paternal atopy (RR=2.97, p=0.017) and a 12 month infant dietary pattern that follows current guidelines (RR=1.79, p=0.016). For non-atopic asthma, bronchiolitis or chest infections (RR=1.76, p=0.047), wheeze in the first 3 years (RR=20.69, p=0.003) and tobacco smoke exposure at 6 years (RR=2.16, p=0.035) increased the risk. Only wheeze in the first 3 years remained in the multivariate model.

Conclusions Different hereditary and early life factors modify the risk of atopic and non-atopic asthma at 6 years of age. This suggests that these two asthma phenotypes have different pathophysiologies.

Pathophysiology of pulmonary vascular remodelling

S36

P38 MAPK: AN IMPORTANT PATHWAY IN THE PATHOBIOLOGY OF PULMONARY HYPERTENSION AND PULMONARY VASCULAR REMODELLING

doi:10.1136/thoraxjnl-2012-202678.042

¹AC Church, ²R Wadsworth, ³G Bryson, ³DJ Welsh, ¹AJ Peacock. ¹Scottish Pulmonary vascular Unit, Glasgow, UK; ²University of Strathclyde, Glasgow, UK; ³University of Glasgow, Glasgow, UK

The p38 MAPK pathway is increasingly recognised as important in inflammation leading to systemic vascular disease but its role in pulmonary vascular disease is unclear. Our group has previously identified the p38MAPK α isoform to be critical in hypoxic-induced proliferation of pulmonary artery fibroblasts, a key step in the pathogenesis of pulmonary vascular remodelling. This study sought to investigate the role of p38MAPK in animal models of pulmonary hypertension and in human disease.

Methods Sprague Dawley rats were exposed to chronic hypoxia for 14 days and received a selective p38 MAPK α inhibitor from day 1