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**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 (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 pathophysiology.

**Pathophysiology of pulmonary vascular remodelling**

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

doi:10.1136/thoraxjnl-2012-202678.042

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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 p38MAPKα 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 p38MAPKα 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 MAPKα inhibitor from day 1 to day 4.