European Respiratory Society  
Annual Congress 2012

**Abstract Number:** 4750  
**Publication Number:** 3272

**Abstract Group:** 4.3. Pulmonary Circulation and Pulmonary Vascular Disease  
**Keyword 1:** Pulmonary hypertension  
**Keyword 2:** Animal models  
**Keyword 3:** Experimental approaches

**Title:** The anti-proliferative effects of apelin on pulmonary adventitial fibroblasts

**Body:**

Background: Pulmonary arterial hypertension (PAH) involves remodelling of the pulmonary artery resulting in right heart failure. Apelin is an endogenous peptide with physiological actions in the cardiovascular system. Pre-clinical models indicate that apelin deficiency may mediate or contribute to the pathogenesis of PAH which involves pulmonary artery fibroblasts (PAF). Aims: To determine if Apelin had an effect on the proliferation and migration of rat PAF. Methods: PAF were isolated from Sprague Dawley rats. The PAF were incubated in normoxic and hypoxic conditions (35mmHg for 24h) and proliferation in response to Apelin was assessed. Western Blotting was used to confirm the presence of the Apelin receptor (APJ). The PAF were also cultured to 100% confluency and the cell monolayer was scratched and cell migration was determined after incubation with Apelin. Results: APJ receptor was detected in PAF cells. Hypoxia alone increased the proliferation of PAF and this effect was abrogated with the addition of 200nM Apelin.

Hypoxia caused PAF migration both alone and to a higher degree with serum. The addition of Apelin prevented this migration. Conclusions: Apelin can reduce the increased proliferation of PAF to hypoxia and reduce the migratory capacity of these cells. Apelin may have anti-remodelling properties that require further investigation.