Background: Among children with congenital heart diseases, right ventricular (RV) pressure overload is common. In contrast to the many studies on left ventricular hypertrophy, only few investigations have evaluated the mechanisms leading to right ventricular hypertrophy.

Aim: Identify cytokines that have altered gene expression in the right ventricle during pressure overload, and which induce hypertrophy.

Method and Results: RV hypertrophy was studied in a mouse pulmonary artery (PA) banding model with pressure overload of the RV. PA banding for 7 days resulted in significant increase in the RV/tibia length (TL) ratio (PA-banding vs. Sham, 2.19±0.03 vs. 1.17±0.05). Inclusion of mice for further studies were done by echocardiography, RV weight measurements and the expression of ANP, myosin heavy chain beta and alpha 1, skeletal muscle mRNA in RV measured by rt-PCR. PA-banded mice were included from the group with peak PA flow velocity of 1.8-2.3 m/s and RV/TL> 1.8 mg/mm which gave a 60% raise in RV/TL compared to Sham. RV tissues from 5 PA-banded mice and 4 sham-operated mice were analyzed by microarray technology (Affymetrix). A total of 16 cytokines were more than 2-fold up-or down regulated (fdr<0.05); 11 up regulated (SPP1 (Osteopontin), IL6, CXCL10, CXCL6, CCL8, CX3CL1, CCL5, CXCL16, CCL2, CCL3 and IL27). Currently we are stimulating neonatal rat cardiomyocytes with cytokines to identify the cytokines that stimulate cardiomyocyte growth.

Conclusion: Right ventricular pressure overload results in significant hypertrophy and regulation of several cytokines which might be importantly involved in remodelling of the RV.