Background: Cytokines are upregulated in heart failure, but little is known of various etiologies’ possible differences in inflammatory responses.

Hypothesis and aim: We hypothesised that the diverse etiologies of heart failure lead to distinct cytokine patterns in the circulation. The aim was to study patterns of circulating cytokines in three animal models for heart failure.

Methods: Myocardial hypertrophy of the left and right ventricles was induced in 7-8 weeks old C57BL/6 male mice by banding of the ascending aorta (AB) and pulmonary artery (PB), respectively. The AB mice were divided into a failure and non-failure group. Mice with conditional knockout of SERCA2 were used as a model for cardiomyopathy. 25 circulating cytokines and chemokines were quantified by Luminex technology in serum samples obtained one week after AB or PB, and seven weeks after induction of knockout in the SERCA2 KO mice.

Results: Our main findings were: (I) No upregulation of cytokines in neither the AB failure nor non-failure group, while seven cytokines were downregulated in the AB failure group. (II) Five cytokines were upregulated in both the PB group and the SERCA2 KO group. (III) Interleukin (IL)-12p40 and CXCL9 were regulated differently in the different etiologies, but differently.

Conclusion: Heart failure due to left ventricular pressure overload did not increase significantly any of the 25 cytokines measured in this study. However, right ventricular pressure overload and SERCA2 KO increased a specific subset of cytokines. IL-12p40 and CXCL9 were regulated differently in the different etiologies, and may be potential etiology-specific biomarkers.