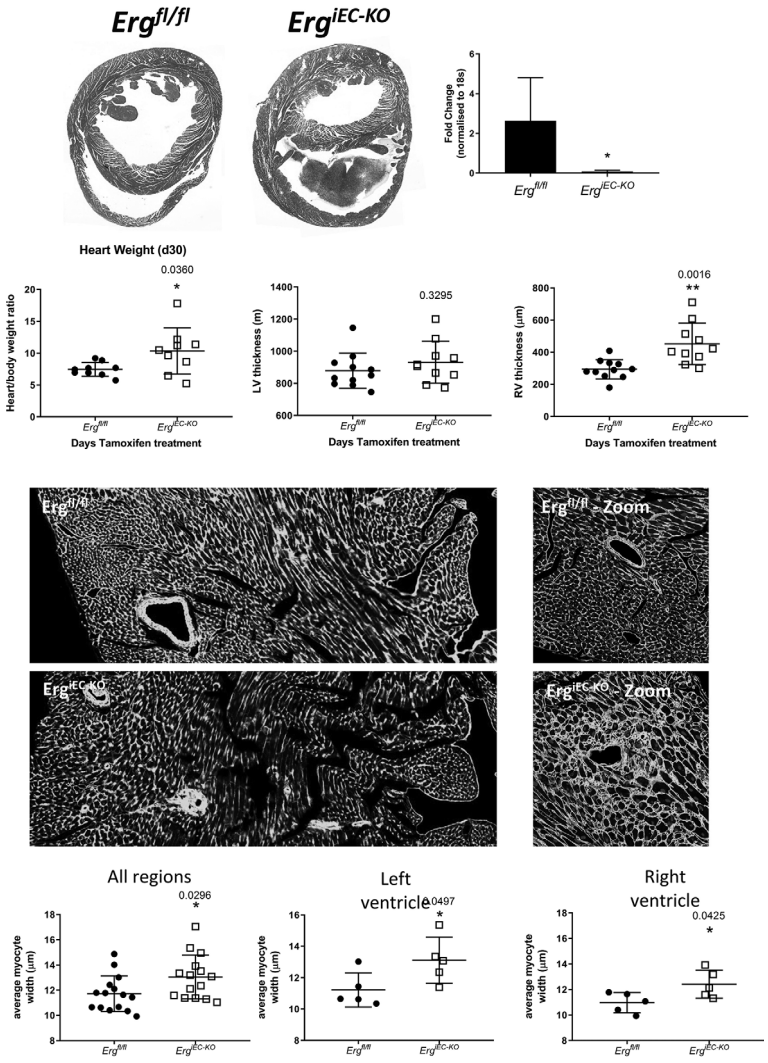

Abstract BS6 Figure 1

verse aortic constriction (TAC) to induce elevated cardiovascular load.

Results ECG measurements revealed that ErgiEC-KO had significantly reduced heart rate and lengthened cQT interval occurring from one week after ERG-deletion. Echo showed significantly reduced ejection fraction and fractional shortening in ErgiEC-KO. Tissue profiling at day 30 post-tamoxifen found a significant increase in ErgiEC-KO heart/body weight ratio. Immunohistochemistry revealed significant cardiomyocyte hypertrophy together with increased SMA and pSMAD3 in all regions of the heart. We observed significant thickening of the right ventricular wall, in line with signs of inflammation, fibrosis and haemorrhage in lung tissue from ErgiEC-KO. Interestingly, phenotypic changes in pulmonary microvascular EC were homogeneous while cardiac microvascular EC had geographical and subset-specific variations. TAC surgery resulted in all ErgiEC-KO reaching endpoint within 28 days (n=5) with significant heart dilation and reduced cardiac function. Notably all ErgiEC-KO exhibited severe inflammation and loss of lung structure. (Figure 1)

Conclusion Systemic deletion of EC-specific ERG significantly influences the microvascular profile, structural integrity and function of cardiopulmonary system. This study highlights that impaired EC functional can rapidly influence ventricular repolarisation and cardiac function. Furthermore, we uncover that chronic EC dysfunction in ErgiEC-KO causes systemic mechanisms and organotypic phenotypes affect the heart and lung emphasizing the role of the endothelium in regulating cardiovascular function.

Conflict of interest No

BS7 ANGIOTENSIN-(1–9) INHIBITS NEOINTIMA FORMATION IN A MURINE VEIN GRAFT MODEL AND MODULATES ERK1/2 PHOSPHORYLATION AND MICRORNA-132 PATHWAYS IN HUMAN VASCULAR SMOOTH MUSCLE CELLS

[1]Stacy Robertson*, [1]Stuart Nicklin, [1]Clare McKinney, [1]Lisa McArthur, [1]Christian Delles, [1]Graeme Milligan, [1]Simon Kennedy, [2]Andrew Baker. [1]*University of Glasgow*; [2]*University of Edinburgh*

10.1136/heartjnl-2019-BCS.171

Introduction Vascular smooth muscle cell (VSMC) migration is integral to vascular remodelling in acute vascular injury.