artery endothelial cells (iHCAECs) were then integrated within our parallel-plate flow apparatus, in which the response to varying flow conditions was observed.

**Results** From extensive published and unpublished data, the overexpression of BMI1 enables prolonged cell proliferation whilst inhibiting cell senescence. Utilising lentiviral overexpression, we were able to immortalise HCAEC with BMI1. Isolated iHCAEC clones were placed under varying hemodynamic conditions in order to screen their response. These iHCAEC clones lined up in conjunction with HCAEC counterparts demonstrating a continued mechanosensitivity. Current work investigating gene expression related to the differing hemodynamic environments is underway with full benchmarking against HCAEC controls in progress. TFAR gene constructs have been developed known to regulate atherosclerosis. The TFARs; NFkB, AP-1, IRF3, XBP1, KL2F and NRF2 have been developed along with lentiviral vectors with secretable luciferase reporters (VLuc and NLuc) allowing for incorporation into validated iHCAECs. Validating these reporter constructs in response to ox-LDL, TNFα and cigarette smoke extract via luciferase activity measuring these reporter constructs in response to ox-LDL, TNFα and cigarette smoke extract via luciferase activity measurement is currently underway, with selected constructs already cloned.

**Conclusion** Our initial results indicate the overexpression of BMI1 in HCAEC results in an extended lifespan and inhibition in cell senescence, with morphology unaffected. The resulting iHCAECs exhibit mechanosensitivity to a changing hemodynamic environment comparable to primary HCAECs. This highlights the advantages of this cell line for future CHD modelling, with its integration with our E-Sense system allowing for the development of a novel research tool and the potential to replace pre-existing methods.

**Conflict of interest** no