POSTER 34. DISSECTING THE ROLE OF MICRO-RNA 200B IN EPICARDIAL DERIVED CELL DIVERSIFICATION AND MIGRATION.

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Cardiovascular disease continues to be a major cause of morbidity and mortality worldwide. Induction of cardiomyocyte regeneration is one proposed way to improve cardiac function, but it is clear that the non-cardiomyocyte populations in the heart also contribute to the repair process. Non-cardiomyocyte lineages (endothelial cells, vascular smooth muscle cells, and cardiac fibroblasts) are essential for blood vessel formation and matrix organization, and an understanding of the developmental signals that shape these cells may provide insights into disease pathogenesis and better heart injury therapies. Several fate mapping and cell lineage studies have demonstrated that coronary vascular smooth muscle cells (cVSMC) and cardiac fibroblasts develop from the epicardium-derived cells (EPDCs) in a multi-step process involving cell proliferation, epithelial-to-mesenchymal transition (EMT) and cell migration. However, the exact signaling cascades in EPDC migration and function still need to be elucidated. Here we show that *miR-200b* is expressed at E12.5 and E15.5 during heart development. LNA *in situ* hybridisation analysis in Wt1 Cre-eYFP, G2GATA4--YFP embryos as well as qRT-PCR of sorted cells showed that *miR-200b* is present in a cell subpopulation of the epicardial-derived cells at these stages of heart development. Moreover, gain and loss of functions experiments in epicardial-derived cell cultures revealed that miR-200b regulates cell motility. We are currently further characterizing the role of *miR-200b* onEMT *versus* EPDCsmigrationby using organ explant cultures of embryonic mouse ventricles. Collectively, our data suggest that this *miRNA* might be a key molecule regulating epicardial cell lineage diversification and migration during cardiac development.