



β -Catenin stabilization promotes proliferation and increase in cardiomyocyte number in chick embryonic epicardial explant culture

Anisha Polley¹ · Puja Sen¹ · Arunima Sengupta² · Santanu Chakraborty¹

Received: 2 February 2017 / Accepted: 24 July 2017 / Editor: Tetsuji Okamoto
© The Society for In Vitro Biology 2017

Abstract Cardiomyocyte (CM) differentiation from proepicardial organ- (PEO) and embryonic epicardium (eEpi)-derived cells or EPDCs in a developing heart emerges as a wide interest in purview of cardiac repair and regenerative medicine. eEpi originates from the precursor PEO and EPDCs, which contribute to several cardiac cell types including smooth muscle cells, fibroblasts, endothelial cells, and CMs during cardiogenesis. Here in this report, we have analyzed several cardiac lineage-specific marker gene expressions between PEO and eEpi cells. We have found that PEO-derived cells show increased level of CM lineage-specific marker gene expression compared to eEpi cells. Moreover, Wnt signaling activation results in increased level of CM-specific marker gene expression in both PEO and eEpi cells in culture. Interestingly, Wnt signaling activation also increases the number of proliferating and sarcomeric myosin (M20)-positive cells in eEpi explant culture. Together, this data suggests that eEpi cells as a source for CM differentiation and Wnt signaling mediator, β -catenin, might play an important role in CM differentiation from eEpi cells in culture.

Keywords Proepicardial organ (PEO) · Epicardium · Wnt/ β -catenin · Cardiac lineage markers · Regenerative therapy

Electronic supplementary material The online version of this article (doi:10.1007/s11626-017-0191-9) contains supplementary material, which is available to authorized users.

✉ Santanu Chakraborty
santanu.chs@presiuniv.ac.in

¹ Department of Life Sciences, Presidency University, 86/1, College Street, Baker building, 2nd floor, Kolkata 700073, India

² The Department of Life sciences and Biotechnology, Jadavpur University, Kolkata 700032, India

Introduction

During cardiogenesis, different cell types are formed from precardiac mesoderm in vertebrates (Schluter and Brand 2013). Initially, two cell types, a contractile myocardium and an inner endocardium, are formed. Thereafter, a third layer, epicardium, appears outside the developing heart (Ishii et al. 2010; Braitsch and Yutzy 2013). A large number of studies have recently demonstrated a complex interaction and cross talk between developing myocardium and epicardium during heart organogenesis (Kruithof et al. 2006; Kennedy-Lydon and Rosenthal 2016). Epicardium is the outer protective layer derived from the extracardiac proepicardial organ or PEO (Kruithof et al. 2006; Maya-Ramos et al. 2013; Asli et al. 2014). In vertebrates, PEO is a cluster of mesothelial cells located at the venous pole of the developing heart. Next, the PEO-derived cells migrate over the whole myocardium forming the outer epicardial layer or epicardium, thus encapsulating the whole embryonic heart (Perez-pomares et al. 2001; Combs et al. 2011; Braitsch and Yutzy 2013). Developmental defects in the epicardium result in embryonic lethality and a number of cardiac abnormalities including many, but not limited to, thin myocardium and disorganized ventricular trabeculae during adulthood (Jenkins et al. 2005; Ishii et al. 2010; Braitsch and Yutzy 2013). Later, a subset of eEpi cells delaminates from the epicardium and undergo epithelial to mesenchymal transformation (EMT) and contribute to various cardiac lineage-specific cells including smooth muscles, fibroblasts, endothelial cells, and CMs, collectively known as EPDCs (Kruithof et al. 2006; Austin et al. 2008; Zhou et al. 2008).

Due to the limited proliferative capacity of adult CMs, repair and regenerative response after adult cardiac injuries remain restricted. Therefore, it results in severe morbidity and mortality in mammals, including humans. Multiple signaling pathways, including Wnt/ β -catenin, have been