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CXCR4 and CXCR7 play distinct roles in cardiac lineage specification and pharmacologic β-adrenergic response

An adult heart has an intrinsically limited capability to regenerate damaged myocardium, regardless of the underlying etiology. Embryonic and induced pluripotent stem cell (ESC/iPSC)- based therapies offer a unique strategy for developing cell replacement therapies for numerous, varied disorders including cardiac diseases. iPSCs hold great promise in the field of regenerative medicine because of their ability to grow indefinitely and give rise to all cells of the body. Recently, investigators shown that pluripotent stem cells produce tissue-specific lineages through the programmed acquisition of sequential gene expression patterns that function as a road map for organ formation, therefore, identifying a procardiogenic network that promotes iPSCs differentiation to favor a cardiac lineage is of great interest. Since adult human hearts have very little ability to regenerate postnatally, stem-cell-based cardiac regeneration has also been considered as a therapeutic approach to treat ischemic heart disease. Since these cells have been shown to migrate to sites of injury and inflammation in response to soluble mediators including the chemokine stromal cell derived factor-1 (SDF-1 also known as CXCL12). Here, we studied the role of SDF-1 and its receptors; CXCR4 and CXCR7 in transformation of pluripotent stem cells into IPSC-derived cardiomyocytes. This study demonstrates that CXCR4 and CXCR7 induce differential effects during cardiac lineage differentiation and β-adrenergic response in human induced pluripotent stem cell-derived cardiomyocytes (hiPSC-CMs). In engineered cardiac tissues, depletion of CXCR4 or CXCR7 had opposing effects on developed force and chronotropic response to β-agonists demonstrating distinct roles for the SDF-1/CXCR4 or CXCR7 network in hiPSC-derived ventricular cardiomyocyte specification, maturation and function.

Biography

Sima T Tarzami has received her BSc and MSc degrees from Hofstra University, New York, and her PhD from Albert Einstein School of Medicine, New York in 2002. She was a Faculty in Mount Sinai School of Medicine from 2007 to 2015. She is currently an Associated Professor of Medicine at Howard University. Her laboratory studies the role of chemokines on cardiac myocyte biology. She focuses on cardiac physiology in both *in vitro* and *in vivo* models of heart failure. She is an author of 20 peer-reviewed papers and over 15 published abstracts.

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