Reprogramming fibroblasts towards cardiomyocyte-like cells

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Cellular reprogramming holds great promise as a novel therapy for heart failure, a common and morbid disease that is usually caused by irreversible loss of functional cardiomyocytes. Because the heart has very limited regenerative potential in response to injury, loss of cardiomyocytes results in impaired pump function and heart failure. Existing treatments are primarily pharmacological and device-based, and do not address the fundamental problem of myocyte loss. Indeed, the prevalence of chronic cardiomyopathy is steadily increasing worldwide, making the identification of novel and effective therapies for this morbid disease an urgent problem in biomedical research. Resident cardiac fibroblasts (CFs) comprise half of the total number of cells in the human heart. The ability to reprogram CFs into functional cardiomyocyte-like cells would theoretically permit regeneration of lost heart tissue. Recently, we showed that introduction of three transcription factors, Gata4, Mef2c and Tbx5 (GMT), directly reprogrammed fibroblasts into induced cardiomyocytes (iCMs) in vivo. In a murine acute myocardial infarction model, delivery of GMT converted CFs into functional iCMs that integrated electrically and mechanically with surrounding myocardium, resulting in a functional improvement and a reduction in scar size. These findings suggest that cellular reprogramming may be an efficient means of regenerating heart tissue in vivo for human patients with heart disease.

Biography

Li Qian has completed her Ph.D. from University of Michigan, Ann Arbor and postdoctoral studies from Gladstone Institute at University of California, San Francisco. She has published more than 20 papers in top peer-reviewed journals and was recently awarded the prestigious Louis N. and Arnold M. Katz Basic Science Research Prize for Young Investigator from American Heart Association.

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