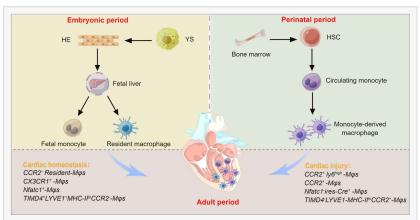


Cardiomyocyte Proliferation: A New Era for Myocardial Repair

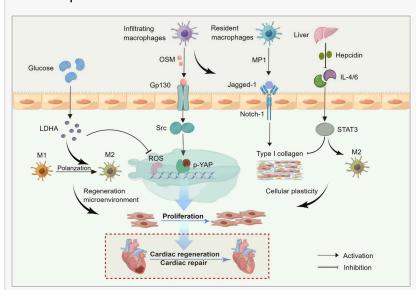
SHANNON, CLARE, IRELAND, March 4, 2025 /EINPresswire.com/ -- A new review published in Genes & Diseases highlights macrophage-targeted therapy as a promising frontier in cardiac regeneration and myocardial repair. The findings underscore the critical role of heterogeneous macrophages in promoting cardiomyocyte proliferation after myocardial infarction, offering a novel avenue for potential therapeutic interventions.

Heart disease remains the leading cause of mortality worldwide, with myocardial infarction often leading to irreversible cardiomyocyte loss and subsequent heart failure. Unlike other regenerative tissues, the adult human heart has limited capacity for self-repair, making cardiac regeneration a pivotal focus in modern medicine. This review emphasizes the significant impact of immune modulation, particularly through macrophage plasticity, in enhancing tissue repair mechanisms.

The article highlights the dual role of macrophages, with certain subtypes



Developmental origin and subtype function of cardiac macrophages. Embryonic development (E7.5-E12.5) revolves around the hematopoietic endothelium (HE) of the yolk sac (YS), which undergoes three waves of hematopoiesis.



Schematic representation of cardiac macrophages initiating cardiomyocyte proliferation and repair.

facilitating inflammation while others contribute to cardiac healing. Resident cardiac macrophages, originating from the embryonic endothelium, play an essential role in modulating the immune response, clearing apoptotic cells, and releasing key cytokines to promote

cardiomyocyte proliferation. These findings open new doors for macrophage-based therapies, which could enhance myocardial repair without relying on exogenous stem cell transplantation.

A point to note is the role of CX3CR1+ macrophages, which initiate cardiomyocyte proliferation via the Jagged-1/Notch1 signaling pathway. This pathway, largely active in neonatal hearts, holds promise for unlocking regenerative potential in adult patients. Additionally, metabolic factors such as lactate dehydrogenase A (LDHA) have been identified as crucial mediators in creating a pro-regenerative microenvironment, further enhancing myocardial repair mechanisms.

As cardiovascular research advances, the review suggests that targeting macrophage subsets could lead to tailored therapeutic strategies aimed at reducing fibrosis, promoting angiogenesis, and ultimately restoring cardiac function. The potential of immune cell reprogramming to stimulate cardiomyocyte proliferation marks a shift in how scientists and clinicians approach heart failure treatment.

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