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Perspective

Cardiac Repair and Regeneration of Myocardial Infarction

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DESCRIPTION

Heart failure is rampant in industrialized countries, killing more people than any other disease. It is often the result of a shortage of specialized heart muscle cells called cardiomyocytes, and powerful therapy to regenerate lost heart muscle can benefit millions of patients each year. Cardiac remodeling is well documented in amphibians and fish and in developing mammals. However, after birth, the regeneration of the human heart is limited to the replacement of heart muscle cells very slowly. Several experimental strategies for revascularization of injured hearts using adult and pluripotent stem cells, cell reprogramming, and tissue engineering are underway. Although challenging, these interventions could eventually lead to better approaches to treating or preventing heart failure.

Myocardial repair and regeneration is an important area of research motivated by the increasing occurrence and wide distribution of heart failure and heart disease. The desperate and unmet need for new interventional strategies for the treatment of cardiovascular disease has prompted the rapid deployment of clinical approaches to promote myocardial regeneration and repair, but the results so far have been modest. To promote an in-depth discussion of cardiovascular restorative drugs, an international collaborative research group has been formed called TACTICS. TACTICS recently issued an opening consensus statement for thematic manuscripts affirming the need for a better understanding of the mechanisms involved in myocardial repair and regeneration.

Several cellular changes occur in the myocardium after myocardial infarction. During the first 6-12 hours, coagulate necrosis occurs and the fibers at the periphery of the infarct lengthen and narrow with signs of vascular degeneration. Simultaneously, edema and neutrophils were observed in the intercellular space. This process takes 3-4 days. After this step, the necrotic cells are cleared by macrophages, which can be actively phagocytosis for 7 to 10 days. Finally, granulomatous tissue with loose collagen fibers and numerous capillaries initiates the healing and repair process in which necrotic cardiomyocytes are replaced by a collagen scar. The immune system is integral to the initial development of an organism as well as the constant replacement of differentiated cell types to maintain homeostasis. Unlike embryonic development, tissue remodeling is initiated by trauma, and mounting evidence suggests that an inflammatory response to this insult also guides regeneration. However, whether immune activation promotes tissue regeneration or wound healing is determined by many factors, including age, species, and accessibility of a group of stem or progenitor cells.

Our laboratory studies the interface of cardiac fibroblasts (scar-forming cells) and cardiac gonads to determine how these intercellular interventions regulate cardiac repair. We use a mouse model of cardiac injury and employ a variety of fate mapping and conditional knockout strategies to modify specific genes at specific time points after injury to investigate the our question. We study the went signaling pathway, a family of 19 closely related proteins that play important roles in organogenesis, wound healing, and cancer.

We recently demonstrated that Wnt1, a Wnt known to play an important role in central nervous system development, plays an important role in regulating the response to fibrous injury in heart. Using conditional inactivation and transgene strategies, we aimed to alter the heart's fibrous repair response to allow regeneration. Embryonic Stem Cells (ESCs) are undifferentiated cells obtained from the inner cell mass of blastocysts that exhibit unlimited self-renewal and pluripotency.

They have the potential to develop into derivatives of 3 germ layers: endoderm, mesoderm, and ectoderm. Autologous cardiac cell therapies have been shown to be safe and effective. The future of cardiac repair may rest on an understanding of the intrinsic mechanisms that regulate the endogenous mobilization and/or distribution of these cells. Arguably, these newest endogenous cardiogenic stem cells may be the best choice for heart repair. Ideally, these cells should be stimulated directly *in situ*, avoiding extraction, purification, culture, and recycling.

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