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Implications of stem cells in cardiovascular infarction

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DESCRIPTION

Acute myocardial infarction is frequently the first symptom in a significant majority of these individuals, and it may proceed to heart failure. Additionally, the human heart has a poor potential for regeneration, which results in cardiomyocytes loss and persistent tissue scarring, both of which have severe pathologic consequences. There is a desperate need for new therapeutic methods. Induced pluripotent stem cells and embryonic stem cells are two types of stem cells that have the most potential for cell replacement therapy. They are also a useful tool for disease modeling and for testing new medications for potential cardiac side effects (Garikapati, et al. 2018). Even though they have a number of drawbacks, catheter-based or surgical procedures, such as coronary bypass surgery and the insertion of assist devices, continue to be the most often used clinical measures.

Despite significant advancements, the majority of surgical procedures are still merely preservatives, or efforts to keep the heart's functionally intact tissue alive as long as possible without structural support. But because CVDs are progressive, Heart Failure (HF) is typically unavoidable. SCs have raised the bar, with goals ranging from the simple enhancement of the cardiac milieu to partial regeneration and/or replacement of missing functional tissue and finally the full fabrication of a surrogate heart.

In addition, SC-based technologies have made it possible to investigate the etiology of CVD entities in great detail and have provided a platform for testing cutting-edge therapy strategies with less risk to patients and at significantly lower costs. These cells are undifferentiated, yet because of their capacity for self-renewal and cell type differentiation, they stand out on a spectrum of potency, such as Multipotent SCs. In organs and/or tissues with high turnover rates, adult SCs' regenerative potential stands out even more (Han, et al. 2017). However, more crucially, it manifests itself in response to tissue damage. Different adult SC populations are currently the subject of a plethora of information, and efforts are being undertaken to use these cells to treat CVDs.

The ability of SMs to regenerate heart tissue has been the focus of numerous preclinical studies in both small and large animal models of CVDs. This has been made possible by the fact that they are easily accessible from autologous muscle biopsies, expand quickly *in vitro*, are ischemic tolerable, and have a low risk of tumorigenicity. In fact, these research' findings have shown beneficial effects by minimizing infarct size and myocardial fibrosis, delaying ventricular remodeling, and enhancing overall heart function. Even though short-term follow-ups revealed favorable results, as seen by improved myocardial perfusion and Left Ventricular Ejection Fraction (LVEF), these trials were unable to demonstrate any long-term advantages (Menasché, et al. 2007). Most recently, findings from phase III clinical studies that were randomized, placebo-controlled, and double-blinded also demonstrated a consistent trend.

On the other hand, basic and translational research has focused more attention on MSCs (CD73-, CD105-, and CD90-positive). MSCs are exceptional in their capacity to elude the immune system, which is an additional benefit to their paracrine- and exosome-mediated immunosuppressive qualities. This is largely because they express HLA class I at moderate levels while not expressing HLA class II, B7, or CD40 ligand, which grant immunity privileges to their host and allow for allogenic transplantation without the requirement for concurrent immunosuppression (Goumans, et al. 2008). Nevertheless, two randomized pilot studies comparing autologous to allogenic MSC therapy were carried out in 2012 and 2017 in patients with ischemic cardiomyopathy (ICM) and no ischemic dilated cardiomyopathy (NIDCM), respectively. The findings of these studies, also known as POSEIDON, hinted at the effectiveness of MSC therapy in these patient cohorts, with allogenic transplantation being preferred.

In contrast to these results, more recent research by Li

and colleagues employing a new genetic-lineage tracking system rejected the myogenic potential of these cells in the adult. The same group has also demonstrated that no contribution to the production of new cardiomyocytes occurs beyond the early segregation of myocytes and no myocytes during embryonic development (E10.5 to E11.5), even during neonatal life (Messina, et al. 2004). Additionally, Elhelaly and colleagues' work from earlier this year claimed that c-kit-positive cells do not contribute to cardiomyogenesis, even at the neonatal stage. However, the general agreement in the field is that CPCs are relict SCs from developmental stages whose function, if any, in the adult heart is limited to preserving cardiac tissue homeostasis and that they have little cardio myogenic potential in the presence of injury.

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