Optimization of Myocardial Tissue Repair and Regeneration withIron-oxide Nanoparticles Formulated in Macrophage-Targeted Carriers (Ramot)

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The Technology

A novel approach for the treatment of AMI has been developed using IONPs. IONPs, when injected into the infarcted myocardium, lead to improved heart function after MI. IONPs activate anti-inflammatory macrophages and thus promote tissue healing and repair and prevent myocardial remodeling and dysfunction.

Background

Macrophages control the initiation, maintenance and resolution of inflammation. One of the earliest phases after MI involves acute inflammation leading to fibrosis and scar formation. Macrophages are essential for infarct healing and repair. Some macrophages exhibit a pro-inflammatory cytokine profile (M1 polarization), whereas others show an anti-inflammatory profile and tissue repair activity (M2 polarization). IONPs are used to label and track inflammatory and stem cells by MRI and are considered the most sensitive existing markers for cell labeling using MRI. They are nontoxic and biodegradable and do not affect proliferation and multi-lineage differentiation capacity in vitro.

The Need and Potential Application

IONPs can be used to treat AMI and other inflammatory conditions associated with pro-inflammatory activated macrophages and to promote tissue healing and repair.

Advantages

Nontoxic

FDA approved for use in MRI imaging in humans

Off –the-shelf

Drug repositioning pathway

Stage of Development

In vivo studies in mouse and rat models of MI and heart failure have demonstrated that IONPs switch infarct macrophages from pro-inflammatory (M1) to reparative (M2) phenotype and improve remodeling and function after MI in mouse

Patents

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