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Programmed Death Ligand-1 was Effective in Preserving Heart Function in a Myocardial Ischemia Model

Patients who experience heart attacks often have immediate ischemia and cell death, which causes a decrease in cardiac function, contributing to higher mortality and morbidity. Currently, there is no minimally-invasive deliverable therapeutic that could be administered to stop or reverse the adverse effects of myocardial ischemia, much less the associated immune response that results in further deterioration of cardiac function and reverse remodeling of the ventricles.

The Woo and Lim labs at Stanford have invented a method that leverages inhibitory immune checkpoint expressions to reduce collateral damage caused by myocardial infarctions. The invention utilizes programmed death ligand-1 (PD-L1) to protect against autoimmunity from immune cells. When PD-L1 was delivered immediately intramyocardially following a heart attack in a rodent model, the left ventricular ejection fraction was significantly improved compared to control animals who did not receive PD-L1 treatment. PD-L1 when delivered systemically was found to have similar beneficial impact on heart function. Regardless of the dose and delivery route, the method significantly reduced left ventricular infarction size compared to controls without treatment. This invention has the potential to be a valuable tool to preserving cardiac function and significantly enhancing patient outcomes.

Applications

- Therapeutic, especially for cardiology
- Autoimmunity
- Cell signaling research tool

Advantages

- There is currently no effective therapeutics that can be delivered via a minimally invasive method to preserve cardiac function following myocardial infarctions
- Easily delivered to patients
- Effective at varying doses and delivery routes

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