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Interactions Between Immune Cells and Fibroblasts during Myocardial Infarction

Myocardial Infarction (MI), commonly known as a heart attack, has many clinical implications particularly contributing to risk factors that encourage the development of heart failure. At the site of injury, heart muscle cells, cardiomyocytes, are not able to receive blood flow which activates cardiac fibroblasts. Fibroblasts are cells that are activated in response to injury and facilitate tissue repair. This activation stimulates the production of collagen resulting in thickening and scarring of the tissue in a process known as fibrosis. Additionally, myocardial fibrosis results in reduced functionality of the heart and impaired regeneration. Concurrently, immune cells infiltrate to the site of injury triggering an inflammatory response. To mitigate the effects of fibrosis and promote the process of regeneration, it is critical to understand the crosstalk between immune cells and myocardial fibroblasts. Immune cell infiltration and fibroblast activation was assessed in mouse MI tissue using immunofluorescence techniques. Positive immune cell and fibroblast staining was observed in the infarct region following MI during earlier time points (Day 3) with sustained fibroblast activation observed in later time points following the injury (Day 7). Here, we aim to characterize the immune cells involved in this process and their relationship with fibroblasts during times of tissue injury and healing and interrogate pathways involved in such processes. Targeting key cellular signaling pathways that contribute to the process of myocardial fibrosis may elucidate an effective treatment to prevent the progression of MI resulting in heart failure. This research may provide a therapeutic target in regulating MI thus improving patient outcomes.