

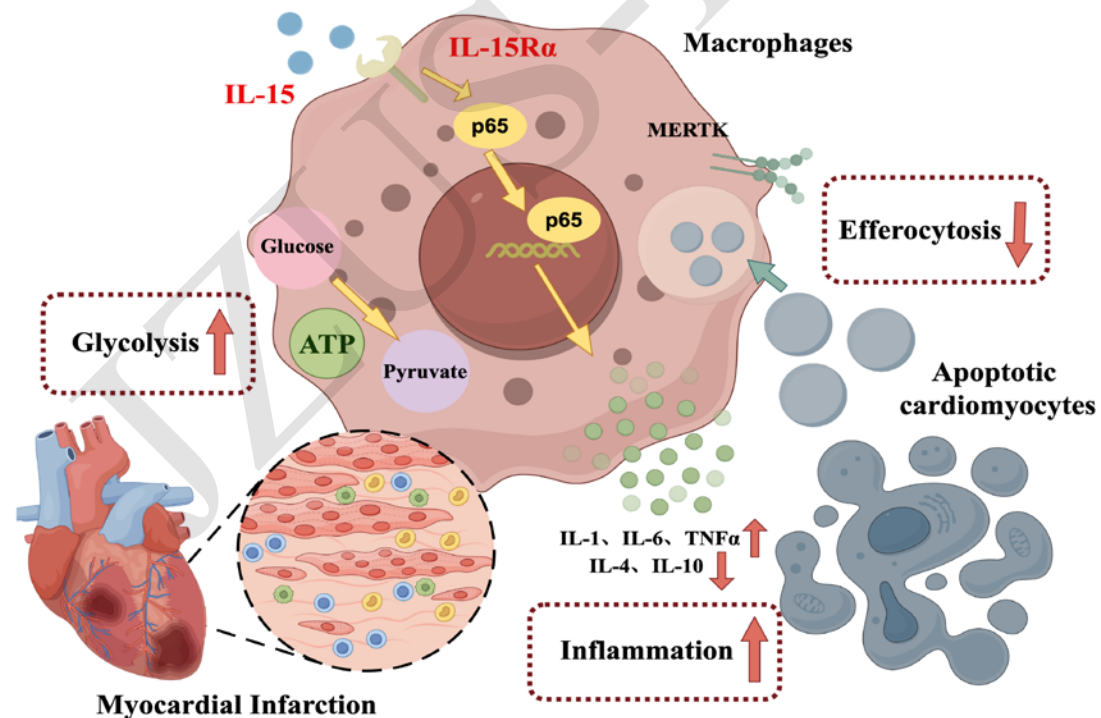
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IL-15 aggravates cardiac ischemic injury via impairing macrophage efferocytosis and driving inflammation

Key words: Myocardial infarction; Inflammatory phenotype; Macrophage function; Metabolic reprogramming; Interleukin-15 (IL-15)

Research Summary

This study delineates the functional role of IL-15 in the progression of AMI, with a particular focus on its influence on macrophage-driven inflammation, efferocytosis, and metabolic reprogramming.



Innovation points

- The genetic deletion of IL-15 or IL-15R α significantly ameliorated cardiac injury by reducing inflammation and apoptosis while preserving myocardial function
- IL-15 impaired macrophage efferocytosis via MERTK downregulation and promoted M1 polarization via NF- κ B pathway activation
- IL-15 reprogrammed macrophage metabolism by enhancing glycolytic activity.