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Heart-resident macrophages call in neutrophils following ischemic injury

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Tissue injury, such as occurs in response to a lack of oxygen, promotes an influx of immune cells to the site of damage. After an ischemic injury to the heart, such as occurs after a heart attack or heart transplant, these responses are often maladaptive, resulting in decreased contractility and possible failure. Innate immune cells called neutrophils infiltrate the heart and are linked to pathogenic responses following an ischemic event; however, it is not clear how these cells are recruited to the site of damage.

In this issue of *JCI Insight*, a team led by Daniel Kreisel of Washington University of Medicine determined that a macrophage population that resides in the heart promotes neutrophil recruitment in response to ischemic injury. Using a mouse heart transplant model of ischemic damage and live animal imaging, the authors determined that a subset of heart-resident macrophages that express a molecule known as CCR2 mediate neutrophil infiltration.

Specifically, these macrophages produced molecules that attached neutrophils from the blood to the site of injury.

Loss of CCR2 macrophages or blocking the production of neutrophil-attracting molecules reduced the numbers of neutrophils the migrated to the transplanted heart.

Together, the results of this study reveal that resident macrophages provide signals that call neutrophils to the site of injury.

More information: Wenjun Li et al, Heart-resident CCR2+ macrophages promote neutrophil extravasation through TLR9/MyD88/CXCL5 signaling, *JCI Insight* (2016). DOI: 10.1172/jci.insight.87315