RESIDENT MACROPHAGES CLOAK TISSUE MICROLESIONS TO PREVENT NEUTROPHIL-DRIVEN INFLAMMATORY DAMAGE

Neutrophils are attracted to and generate dense swarms at sites of cell damage in diverse tissues, often extending the local disruption of organ architecture produced by the initial insult. Whether the inflammatory damage resulting from such neutrophil accumulation is an inescapable consequence of parenchymal cell death has not been explored. Using a combination of dynamic intravital imaging and confocal multiplex microscopy, we report here that tissue-resident macrophages rapidly sense the death of individual cells and extend membrane processes that sequester the damage, a process that prevents the initiation of the feedforward chemoattractant signaling cascade that results in neutrophil swarms. Through this “cloaking” mechanism, the resident macrophages prevent neutrophil-mediated inflammatory damage, maintaining tissue homeostasis in the face of local cell injury that occurs on a regular basis in many organs due to mechanical and other stresses.

Keywords : macrophages, cloaking, anti-inflammation, neutrophils
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