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# Satiated macrophages produce interferon-beta to orchestrate the resolution of bacterial infection

Acute inflammation is a localized, self-limited host defense mechanism against invading microorganism and tissue injury. Rapid recruitment of white blood cells called neutrophils (polymorphonuclear neutrophil granulocytes) from the blood to the site of infection or injury is critical for the elimination of invading pathogens and tissue repair. Once the pathogens are cleared, cessation of neutrophil recruitment and removal of neutrophils that have migrated into the inflamed site will assure timely resolution of inflammation and restoration of tissue integrity. Following killing of bacteria, neutrophils undergo programmed cell death (apoptosis) and express “find-me” and “eat-me” signals that allow their recognition and uptake by specialized immune cells called macrophages. Failure to remove neutrophils inflicts damage to the surrounding tissue and prolong inflammation, common mechanism underlying many chronic diseases. Hence, understanding the mechanisms that govern resolution is of outmost importance.

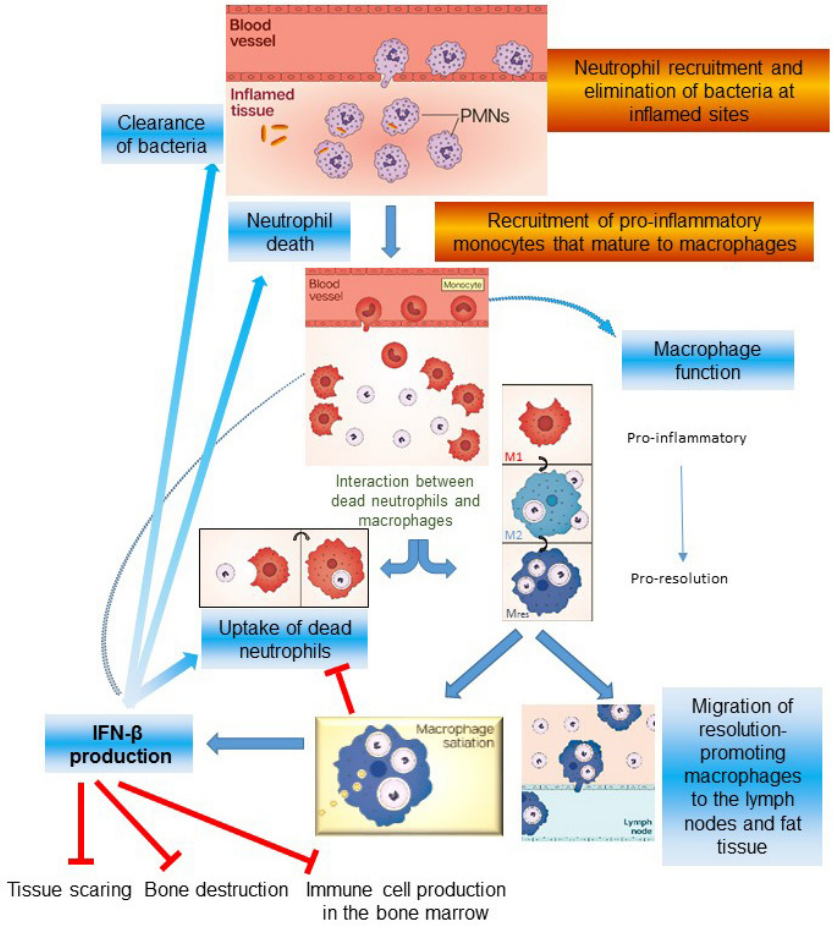


Fig. 1. Interferon-β (IFN-β) production by macrophages and resolution of inflammation.

Comparing gene expression profiles, we identified that resolution-phase macrophages produce the cytokine interferon-beta (IFN-β) following uptake of dead (apoptotic) neutrophils. IFN-β, in turn, facilitates removal of bacteria from infected tissues, accelerates death of inflammatory neutrophils and their uptake by macrophages, resulting in reprogramming of macrophages to resolution-promoting macrophages (termed Mres). These results identify an IFN-β-centered signaling mechanism that mediates cross-talk between neutrophils and macrophages to limit the extent of inflammatory reaction and facilitate the resolution of bacterial infections. These findings would also suggest the therapeutic potential of IFN-β in chronic inflammation, as well as fibrotic disorders and wound repair.

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## Publication

IFN-β is a macrophage-derived effector cytokine facilitating the resolution of bacterial inflammation
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