Immunome Research
ISSN: 1745-7580
OPEN ACCESS Freely available online
Perspective

The Role of Immune Cell Subsets in Resolving Inflammation

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DESCRIPTION

Inflammation is a critical biological process aimed at eliminating harmful stimuli, such as pathogens, damaged cells, or irritants, and initiating tissue repair. While the onset and propagation of inflammation have been extensively studied, the equally vital phase of inflammation resolution has gained increasing attention in recent years. Resolution is an active, highly regulated process involving specialized immune cell subsets that coordinate to restore tissue homeostasis and prevent chronic inflammatory diseases.

Understanding the distinct roles of various immune cell subsets in orchestrating the resolution of inflammation holds promise for developing novel therapies to treat a wide range of inflammatory and autoimmune conditions. This perspective article explores key immune players in inflammation resolution, emphasizing their dynamic functions and interplay during this critical phase.

Innate immune cells: Orchestrators of early resolution

The innate immune system, traditionally associated with initiating inflammation, also plays a pivotal role in its resolution. Several innate immune subsets undergo functional transitions during the course of inflammation that promote clearance of debris, suppression of pro-inflammatory signals, and tissue repair.

Macrophages exemplify this plasticity. Initially activated into a pro-inflammatory (M1-like) state, macrophages produce cytokines such as TNF-α, IL-1β, and IL-6 to amplify the inflammatory response and recruit additional immune cells. However, as the infection or injury is controlled, macrophages shift toward an anti-inflammatory, reparative (M2-like) phenotype. These M2 macrophages secrete anti-inflammatory cytokines like IL-10 and TGF-β, promote efferocytosis (the clearance of apoptotic cells), and stimulate tissue remodeling and angiogenesis. This functional switch is tightly regulated by microenvironmental cues, including lipid mediators such as resolvins and protectins, and is essential for timely resolution.

Neutrophils, once thought to be short-lived foot soldiers solely involved in pathogen killing, also contribute to resolution. After fulfilling their antimicrobial roles, neutrophils undergo apoptosis and are cleared by macrophages, a process that actively dampens inflammation. Moreover, subsets of neutrophils can release anti-inflammatory mediators and contribute to the formation of Neutrophil Extracellular Traps (NETs) that trap and neutralize residual pathogens, facilitating cleanup.

Innate Lymphoid Cells (ILCs), particularly group 2 ILCs (ILC2s), have emerged as important modulators of inflammation resolution in mucosal tissues. ILC2s produce amphiregulin and IL-13, which aid in tissue repair and epithelial regeneration. Their ability to respond rapidly to tissue damage and promote healing places them as key innate contributors to resolution.

Together, these innate immune cells form the first wave of resolution agents, bridging the transition from inflammation to healing.

Adaptive immune cells fine-tuning and sustaining resolution

Adaptive immune cells, long associated with pathogen-specific responses, also play nuanced roles in resolving inflammation. Their contributions are critical for preventing chronic inflammation and autoimmune pathology.

Regulatory T cells (Tregs) are central to adaptive immune regulation. Tregs suppress excessive immune activation by producing anti-inflammatory cytokines such as IL-10 and TGF-β and by direct cell-cell contact inhibition. They modulate both innate and adaptive effectors, limiting tissue damage and facilitating repair. During resolution, Tregs accumulate in inflamed tissues and lymphoid organs, promoting an immunosuppressive milieu that favors homeostasis.

B cells can contribute to resolution through the production of anti-inflammatory cytokines like IL-10 (regulatory B cells, or Bregs). These subsets help restrain pro-inflammatory responses and support the generation of tissue-protective antibodies. Additionally, certain antibody isotypes may aid in opsonization

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Received: 25-Nov-2025, Manuscript No. IMR-25-39038; **Editor assigned:** 27-Nov-2025, PreQC No. IMR-25-39038 (PQ); **Reviewed:** 11-Dec-2025, QC No. IMR-25-39038; **Revised:** 18-Dec-2025, Manuscript No. IMR-25-39038 (R); **Published:** 25-Dec-2025, DOI: 10.35248/1745-7580.25.21.328

Citation: Jerry A (2025). The Role of Immune Cell Subsets in Resolving Inflammation. Immunome Res. 21:328

and clearance of apoptotic cells and debris, further facilitating resolution.

Effector T cells, including Th1 and Th17 subsets known for their pro-inflammatory roles, can also exhibit plasticity during the resolution phase. Under specific conditions, they may acquire regulatory features or undergo apoptosis, preventing chronic inflammation. The balance between effector and regulatory phenotypes is thus a determinant of inflammation outcome.

The adaptive immune system's ability to "fine-tune" inflammation ensures that pathogen clearance is followed by controlled suppression of immune activity, minimizing collateral tissue damage.

Harnessing the specialized functions of immune cell subsets involved in inflammation resolution offers exciting therapeutic avenues. Current anti-inflammatory drugs often suppress the initiation of inflammation but fail to promote active resolution, potentially leading to incomplete healing or chronic disease.

Emerging therapies aim to boost pro-resolving pathways. For instance, administration of synthetic lipid mediators such as resolvins and protectins mimics natural signals that drive macrophage reprogramming and neutrophil clearance. Therapies enhancing Treg function or expanding regulatory B cells are being explored for autoimmune diseases like rheumatoid arthritis and multiple sclerosis.

Moreover, understanding the temporal dynamics of immune cell subset activation and transitions will enable precision timing of interventions. Tailoring treatments to promote resolution-specific subsets without compromising host defense is a key challenge.

Advanced technologies like single-cell sequencing, multiparameter flow cytometry, and spatial transcriptomics are unraveling the heterogeneity and plasticity of immune cells in inflamed tissues, providing deeper insight into resolution mechanisms. Integration of these data with clinical outcomes will accelerate the development of resolution-targeted therapies.

CONCLUSION

The resolution of inflammation is an active, complex process governed by diverse immune cell subsets with specialized and evolving roles. Innate cells such as macrophages, neutrophils, and ILCs initiate the transition from inflammation to repair, while adaptive cells like Tregs and Bregs fine-tune and sustain this phase. Their coordinated actions ensure that inflammatory responses subside appropriately, preventing chronic pathology and enabling tissue regeneration.