ACTA SCIENTIFIC MEDICAL SCIENCES (ISSN: 2582-0931)

Volume 3 Issue 8 August 2019

Mini Review

Inflammation: The Good or the Bad Guy?

Maria Benito

IPN Communications, Dublin, Ireland

***Corresponding Author:** Maria Benito, Clinical Medicine, IPN Communications, Dublin, Ireland.

Received: July 01, 2019; **Published:** July 11, 2019

DOI: 10.31080/ASMS.2019.03.0351

Abstract

Acute inflammation is a self-regulated mechanism that protect against insults or tissue damage. Dysregulation of this process can make the inflammation chronic and further damage normal tissue function.

Keywords: Inflammation; Resolution; Inflammatory Process; Dysregulation; Neutrophils; Macrophages

Abbreviations

LTB4: Leukotriene B4); LXA4: Lipoxin A4; PGD2: Prostaglandin D2; PGE2: Prostaglandin E2; PGJ2: Prostaglandin J2; RvE1: Resolvin E1; TGFβ: Transforming Growth Factor Beta; TNF-⍰: Tumour Necrosis Factor-Alpha.

Benefit that the body receives by inflammation

Inflammation is a process that protects tissues from many types of insults, from foreign pathogens attacks to autoimmune assault. To confront these harms, the body responds with acute inflammation that aims to get rid of the invader through the immune system (lymphocytes –T cells, B cells, and natural killer cells–, macrophages, dendritic cells and neutrophils).

Upon injury, neutrophils infiltrate the tissue and start secreting cytokines and mediators to recruit other immune cells that also secrete more inflammatory mediator perpetuating the inflammation. Prostaglandin E2 (PGE_2) and tumor necrosis factor α (TNFα) are some of the mediators involved in the initiation process [1,2].

Inflammation follows a sequence of induction, a peak of inflammation, and finally a phase of resolution to eliminate the injury. The inflammatory process has an internal cost in homeostasis and alteration of the normal tissue function.

Once the damage has been overcome, the resolution process starts with the cease of neutrophils recruitment to the inflamed tissue, switching the production from pro-inflammatory agents such as PGE_2 and Leukotriene B4 (LTB4) to pro-resolving lipid mediators such as PGD_2, PGJ2, lipoxin A4 (LXA4), resolvin E1 (RvE1), protectin D1, and maresin-1 [3]. Then, it is necessary the removal of neutrophils through apoptosis induced by death ligands produced by macrophages [4,5] or transforming growth factor beta (TGFβ) produced by regulatory T cells [6]. Macrophages need to change their role –from M1 to M2– acquiring anti-inflammatory and pro-resolving functions [7], and clear the apoptotic neutrophils through efferocytosis.

Damage caused by chronic inflammation

However, sometimes this process of inflammation is dysregulated and instead of resolving becomes chronic leading to other pathologies. Thus, although inflammation is a beneficial process designed to eradicate threats to the organism, a dysregulation of inflammation –either in magnitude or duration– contributes to multiple pathologies [8] such as rheumatoid arthritis, Crohn's disease, cancer, vascular diseases and asthma [9-12]. In extreme cases, the dysregulated inflammatory reaction can cause inflammatory tissue damage and sepsis [13,14].

Recently, mechanisms tissue and disease-specific that funnel the resolution of inflammation in arthritis, colitis, and asthma have been identified [12]. These pathways –as well as the discovery of new players that may play a role in inflammation– can be a potential target to reverse chronic inflammation in inflammatory diseases.

Conclusion

Despite the benefit of acute inflammation to defend against pathogens or insults, chronic inflammation can cause more damage and contribute to the development of additional pathologies.

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