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A short Review of the Macrophage's Cytokines

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Abstract

Antibiotics and anti-inflammatory drugs are increasingly used in conjunction to treat bacterial endotoxin-induced inflammation after illnesses or accidents. The level of macrophage-produced cytokines is crucial to the success of this regimen. Two common antibiotic and anti-inflammatory drug representations that have been reported to have satisfactory results are ciprofloxacin and indomethacin. They are both cost-effective. The goal of the current study is to find out how ciprofloxacin and indomethacin affect macrophages' in vitro production of inflammatory cytokines. Significant injury and infection can cause immunological dysfunction and overactive inflammatory responses, which can result in a variety of consequences, including multi-organ dysfunction syndrome, with a death rate as high as 70%. Antibiotics are frequently used against harmful germs, however they might increase inflammation in vivo and speed up the development of disease. Therefore, combining the use of antibiotics and anti-inflammatory drugs may offer a different approach to managing and/or preventing a variety of problems after wounds and infections.

Keywords: Antibiotics; Endotoxin-induced inflammation; Nanoparticles; Biocompatibility; Peritoneal macrophages

Introduction

According to these research, biomimetic nanoparticles with numerous cytokine receptors on their membranes may be created using inflammatory cell membranes to reduce the systematic cytokine storm in HLH. Recently, a promising therapeutic platform that inherits the characteristics of donor cells is emerging: cell membrane-coated nanoparticles. For instance, compared to polyethylene glycol (PEG)-based nanoparticles, red blood cell (RBC) membrane-coated nanoparticles demonstrated favourable biocompatibility and longer circulation time [1]. Additionally, pore-forming poisons, organophosphate poison, and pathogenic antibodies in autoimmune diseases can all be absorbed by RBC membrane-coated nanoparticles, protecting healthy RBCs. Additionally, cytokine absorption and joint damage reduction in inflammatory arthritis have been achieved using neutrophil membrane-coated nanoparticles. By attracting and expelling lipopolysaccharide (LPS), macrophage membrane-coated nanoparticles demonstrated as a detoxifying agent in a sepsis animal model [1,2].

Although many macrophage depletion techniques have shown that macrophages play a crucial role in bone repair, the underlying molecular pathways are not well known. We discovered that the cytokine oncostatin M [OSM or murine (m)OSM] was overexpressed during the early inflammatory phase and that the reduction of macrophages suppressed mOSM expression in this study using a mouse model of tibia injury. By using micro-computed tomography and histology on Osm/Osm mice, we found that there were fewer Osterix+ osteoblastic cells, fewer runt-related transcription factor 2 and alkaline phosphatase-expressing osteoblasts, and significantly less new intramedullar woven bone being formed at the injured site [2,3].

Method

Peritoneal macrophages from primary murine and RAW 264: Lipopolysaccharide (LPS) was given orally to cells for 24 hours. LPS-induced macrophage secretion was used to assess the appropriate dose and timing of ciprofloxacin or indomethacin in response to macrophage inflammatory response inflammation [4,5]. The effects of ciprofloxacin and indomethacin on the secretory capabilities and viability of various macrophages were then assessed using flow cytometry and enzyme-linked immunosorbent assay, particularly for the concentrations of interleukin (IL)-1, IL-6, IL-10, and tumour necrosis factor (TNF)-. By measuring the maximal inhibitory effect of the medicines on pro-inflammatory variables at each concentration or time point, the ideal ciprofloxacin dose and time course affecting macrophage inflammatory response were identified [3,6].

Result

Within a few hours of a bone injury, the innate immune system is triggered, and the first cells to appear in the inflamed fracture site are platelets, neutrophils, and macrophages. A wide range of inflammatory cytokines, growth factors, and chemokines are then produced in order to promote leukocyte recruitment and to initiate bone healing by activating mesenchymal progenitor cells[7,8]. With the use of the mouse model, we demonstrate that mOSM is a component of this early anabolic cytokine cocktail and plays a significant role in the first stage of bone apposition, or bone modelling, but not in the subsequent re-modelling stage. OSM is highly expressed in human shattered bones, according to preliminary findings (data not shown) [9-11].

Conclusion

In vitro, the combination of indomethacin reduced the amount of inflammatory cytokines that macrophages released. The regulating mechanism of medication combinations on innate immune cells that trigger inflammatory reactions is demonstrated in this work. It also offers a novel prospective antibacterial and anti-inflammatory therapeutic approach to avoid and treat future problems.

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Conflict of Interest

None

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