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Concept of Inflammation Resolution with Glucocorticoids Production

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DESCRIPTION

The concept of inflammation resolution is a revolutionary approach to the field of inflammation that has been developed during the previous two decades. Current therapies decrease active inflammatory processes by blocking cyclooxygenases, inhibiting the production of numerous cytokines with glucocorticoids, and targeting specific effectors or antigens with biologics such as anti-therapies. This, however, may just be half of the story. Inflammation begins with a tissue insult, which can be caused by infection, trauma, or injury. This triggers the first recruitment of neutrophils, which clean up any initial infection and summon macrophages. Once neutrophils and macrophages have cleared the inflammation, the neutrophils will die, the macrophage will alter its phenotype to one of proresolving and tissue repair, and the tissue will revert to its pre-inflammation state. This return to baseline, however, is not merely defined by the absence of the inflammatory insult, but also by a positive process involving its own arsenal of mediators that brings the tissue from an inflammatory state to its normal resting state.

There are now a slew of mediators engaged in the inflammation resolution process. Autacoids such as adenosine, locally produced hormones such as melanocortins and somatostatin, bioactive lipids such as lipoxins, resolvins, protectins, and maresins, and proteins such as heme oxygenase 1, annexin A1, galectins, and erythropoietin are just a few examples. We will only address a few examples of proresolving proteins and peptides in the melanocortin system due to space constraints. There are several inflammation-clearing processes that lead to the restoration of normalcy. The elimination of the primary insult, such as phagocytosis of invading bacteria, is critical because it prevents the production of proinflammatory mediators. The proinflammatory stimuli are subsequently broken down, as well as the creation of proinflammatory cytokines, chemokines, and other inflammatory mediators like proteolytic enzymes. The majority of current therapy focuses on this process.

The inflammatory cell infiltration must next be removed. Local cell death, usually via apoptosis, is followed by macrophage phagocytosis, and subsequently lymphatic outflow leaves the location. Apoptosis may cause some of these macrophages to perish and be removed by other resident cells. The key idea is that macrophages ingesting apoptotic neutrophils will prevent the formation of necrotic cells, which will eventually release their damaging contents, thereby perpetuating the inflammatory response. This procedure is also nonphlogistic, meaning it does not trigger an inflammatory reaction. Some cells may circulate throughout the body and leave the inflammatory location. In histological terminology, the resolution phase of an acute inflammatory process is described as the time period between the maximum neutrophilic count and the lowest neutrophilic count. The discovery of the proopiomelanocortin system, which includes a number of melanocortins and melanocortin receptors, has improved our understanding of the biological basis of these effects of is the prototype of the melanocortins, and its anti-inflammatory effects have been confirmed, forming the basis for its use in the clinical management of inflammatory arthritides, such as gout, where it is still used in the United States today. After two injections on alternate days, a placebo-controlled trial of synacthen, a synthetic version, in patients revealed an extra effect that lasted three months.

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CONFLICT OF INTEREST

The author has nothing to disclose and also state no conflict of interest in the submission of this manuscript.

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