Is Inflammation after Injury All Bad?

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Tissue healing after injury is a complex process that aims to replace damaged tissues and return them to a pre-injured state. Acute inflammatory reactions are characterized by rapidly resolving vascular permeability, edema, neutrophil and macrophage infiltration and T lymphocyte migration, and – ultimately – resolution into healthy tissue. In contrast, when an inflammatory process becomes chronic, we see a picture of chronic tissue destruction and fibrosis.[1]

The modern view of inflammation, therefore, is that acute inflammation (if not too robust) is beneficial, whereas chronic inflammation is detrimental.[2] This is in keeping with the concept of disease evolution as postulated by Reckeweg and seen in the Disease Evolution Table (six-phase table). We find acute inflammation in the 2nd phase of the table and degeneration in the 5th phase. Most 5th phase degenerative diseases have a common denominator, namely, chronic inflammation that leads to tissue destruction and fibrosis.[3] The result is organ damage and sometimes death.

Typically, inflammation is a Th1 response driven by pro-inflammatory cytokines such as IL-1, TNF-α, and IL-6. Although other mechanisms are also involved, fibrosis is primarily a Th2 response.[4] It is therefore important to restore the normal physiological balance between these two processes.

The aim of any therapy for injury should thus be to "subdue" inflammation to a level adequate to produce degeneration of damaged tissue yet permitting normal tissue remodelling. Especially in athletes, it is important to achieve normal repair of connective tissue, as fibrotic tissue is less elastic and is thus susceptible to re-injury and tends to impair performance.

Furthermore, chronic recurrent inflammation has been implicated in the development of overtraining syndrome in elite athletes, due to the neurological effects of pro-inflammatory cytokines.[5,6] Consequently, total suppression of inflammation after injury is not the best strategy.

Although not proven in clinical trials, NSAIDs have long been suspected of interfering with tissue healing if administered after injuries such as fractures, and many authors urge caution, especially in certain patient groups.[7] Corticosteroids, which are known to interfere with the remodelling process, should be used sparingly, if at all, in the acute phase of injury and should actually also be avoided in chronic inflammation.[4]

Immune regulation

How, then, can a balance between inflammation and repair be achieved in acute injury? As always in complex systems, interfering with just one aspect is unwise because it may negate normal feedback mechanisms and interactions, as is the case with nonsteroidal anti-inflammatory agents. Apart from increasing the risk of adverse events, full suppression of inflammation is not desirable because a certain level of inflammation (as we saw above) is needed to eliminate degraded tissue.

This delicate balance can be achieved through immune regulation. The hallmark of any biological regulation therapy is that it acts on multiple points in the process and supports the body's own mechanisms for achieving resolution. The combination product Traumeel is one such inflammation-regulating medication.

Traumeel has a long history of use, and a great deal of empirical evidence attests to its effectiveness and tolerability. Increasingly, however, research is discovering a mosaic of therapeutic effects for this product. Basic research has already indicated two or three possible mechanisms of action:

- Induction of T regulatory cells via the low concentration of plant materials in the product[8]
- Down-regulation of pro-inflammatory cytokines such as IL-1, TNF-α and IL-8[9]
- Perhaps also the action of helenalin (a sesquiterpene lactone glycoside contained in arnica), which has been shown to modulate NF-κB, a nuclear transcription factor in the inflammatory cascade

There is also an increasingly strong clinical evidence base for Traumeel, especially in sports injury and orthopedic surgery.[10-13] This product should be considered for its immune-regulating properties, which permit some degree of inflammation while simultaneously promoting repair.

Disease Evolution Table: Acute inflammation occurs in the 2nd phase of Dr. Reckeweg's concept of disease evolution; chronic inflammation belongs to the 5th phase (degeneration).

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