Insights into the regulation of inflammation

In a recent publication, scientists made a big advancement in their knowledge of how inflammation is controlled. They just found that an important immunological alarm protein that was previously thought to slow down the immune response really performs the reverse.

Numerous potential effects of their study exist, particularly regarding understanding and treating autoimmune diseases and inflammation.

Although our immune system plays a crucial role in keeping us safe from infection and harm, when immune responses are overly strong, they can cause harmful inflammation, which manifests in diseases like rheumatoid arthritis and psoriasis. Interleukins, which amplify our defenses against infection and damage by turning on various immune system components, cause inflammation.

Interleukin-37 was previously thought to act as an on and off switch for the immune system but it was discovered in this study that it really has an unanticipated role as an immune-activating molecule. Interleukin-37 was previously assumed to have immune-suppressive properties, but how exactly it turned off inflammation was highly contested. Scientists currently claim that Interleukin-37 has strong pro-inflammatory action when triggered properly.

As a result, it’s possible that Interleukin-37 and other

immunological alarm proteins developed into distinctive variants on the same theme that allow our systems to detect various forms of infection by getting triggered by enzymes specific to each infectious agent.

Journal article: Sullivan, G.P., et al., 2022. Myeloid cell–derived proteases produce a proinflammatory form of IL-37 that signals via IL-36 receptor engagement. *Science Immunology*.

Summary by Stefan Botha