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TRANSCRIPT

Transcript Details
This is a transcript of an educational program accessible on the ReachMD network. Details about the program and additional media formats for the program are accessible by visiting: https://reachmd.com/programs/rheumatoid-arthritis-addressing-unmet-needs/il-6-molecular-origins-ra-inflmmation/9804/

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IL-6 and the Molecular Origins of RA Inflammation

Announcer:

This is ReachMD. Welcome to this special series, *Rheumatoid Arthritis: Addressing Unmet Needs,* sponsored by Lilly.

On this episode, titled *IL-6 and the Molecular Origins of RA Inflammation*, we will hear from Dr. Alan Epstein, a Clinical Professor of Medicine at the University of Pennsylvania School of Medicine in Philadelphia.

As we know, rheumatoid arthritis is a chronic progressive inflammatory autoimmune disease characterized by joint damage, joint inflammation, systemic manifestations and important comorbidities. It is driven by a complex network of cytokines including TNF, interleukin 1, interleukin 12, and interleukin 17. One of the most important cytokines, if not the most important cytokine, is interleukin 6. I say this because interleukin 6 drives the inflammatory aspects of the disease, the destructive aspects of the disease and the systemic aspects of the disease. Interleukin 6 is a cytokine that mediates inflammation, immune responses and hematopoiesis. Over production of interleukin 6 has been implicated in the pathogenesis of rheumatoid arthritis.

Normally, interleukin 6 is present in a serum at very low levels. Patients with rheumatoid arthritis have

sustained elevations of interleukin 6 in the serum and even higher levels in their synovial fluid. Interleukin 6 levels have been shown to correlate with both disease activity and radiographic progression in patients with rheumatoid arthritis. You will remember that the normal synovium is 1 to 3 cells thick. The rheumatoid pannus, however, is perhaps 10 to 20 cells thick. The most important cell in the rheumatoid pannus is the fibroblast-like synoviocyte. Interleukin 6 stimulates these cells which have inherent invasive properties to produce matrix metalloproteinases, which degrade cartilage. They also produce RANK ligand which stimulates osteoclasts to erode bone. The fibroblast-like synoviocyte also produces interleukin 6, thus creating a positive feedback loop.

From this discussion it should be apparent that interleukin 6 is a critical cytokine in driving the inflammatory aspects of rheumatoid arthritis, the destructive aspects of rheumatoid arthritis and the systemic aspects of what might be called rheumatoid disease. It only makes sense that inhibition of this cytokine might be expected to have an ameliorative effect on this disease.

Announcer:

The proceeding program was sponsored by Lilly. To revisit any part of this discussion and to access other episodes in this series, visit **ReachMD.com/addressingRA**. Thank you for listening.

This is ReachMD.

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