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Which came first, the bacterium or the epithelium?

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Abstract

The link between microorganisms and joint inflammation is nothing new. It has existed since possibly the times of Hippocrates. In 1880 Sir Benjamin Brodie described the triad of urethritis, arthritis and conjunctivitis. This and the association of dysentery with arthritis and conjunctivitis, and no viable organisms in the joints, resulted in the name: reactive arthritis. A lot of work performed in the 1980s and 1990s further identified antigenic material from microorganisms in the epithelia of the joints themselves, and the gut. Yet no viable organisms were found. Whilst the exact mechanisms were elusive and were thought to be a combination of both genetic susceptibility and microbial interaction, reactive arthritis was hypothesized as a paradigm for all autoimmune disease, especially those involving joints. Recently more sophisticated technologies have allowed more detailed analysis of organism communities in different niches in the human body (the micro biome), and this has resulted in analysis of the microorganisms in an even greater range of autoimmune diseases, for example rheumatoid arthritis. Here I briefly summarize the evolution of the notion that the microorganisms orchestrate damage to joints from afar, and how the link between modern day microbiology and rheumatology is stronger than ever.

Biography

Sahar Musaad graduated from the Faculty of Medicine, Khartoum University, Sudan and went on to obtain a PhD in Molecular Medicine, Auckland, NZ. She remained interested in host pathogen interaction in etiology of arthritis, even as a consultant microbiologist (FRCpath, UK), which she has been since 2006.

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