



## Microbes and ReA

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**Received:**  August 20, 2019

**Published:**  August 28, 2019

### Mini Review

Reactive arthritis (ReA) belongs to a group of diseases under spondyloarthritis (SpA). It is a systemic disease triggered by an infection at a distant site, other than joint. The infectious triggers are mostly genitourinary, gastrointestinal or respiratory tract. An association has been established between arthritis and microorganisms like Shigella, Yersinia, Salmonella, Campylobacter and Chlamydia [1]. The role of microbiomes in inflammatory arthritis and autoimmunity has been proposed decades ago. In genetically susceptible patients, infection activates Th17 mediated immune response which causes arthritis and systemic inflammation [2]. The interplay between HLA B27 gene and microorganism has also been studied for the pathogenesis of reactive arthritis. HLAB27 is a major histocompatibility complex, strongly associated with SpA. Around 45-90% of patients with ReA had HLA B27, which predicts the progression and severity of disease [1]. Several theories have been postulated for the pathogenesis of ReA. These include molecular mimicry, arthritogenic peptide, Toll like receptors (TLRs) [2]. The virulence factors such as invasion, protein tyrosinase, cytoxin, adhesion present in microbes facilitates their persistence in the gut.

The bacterial epitope presented by MHC class I is recognized by cytotoxic T lymphocytes. T cells then cross react with self-antigen derived peptides [3]. Through molecular mimicry between bacterial protein and HLAB27, HLA B27 presents its own peptides similar to bacterial antigens leading to autoimmunity [4]. Other genes like HLA-B39, HLA-B60, and HLA-DR1 are implicated in HLA B27 negative patients. Bacterial LPS helps in virulence of

bacteria and modulates immune system. The intraarticular LPS stimulates proinflammatory cytokines like TNF $\alpha$ , IL1 and IL6, enhances lymphocyte entry into synovium, activation of IL8 from chondrocytes leading to enhanced leukocytes recruitment in synovium and persistent phagocytosis. Chondrocytes produce collagenases, nitric oxide and proteases which lead to degradation of cartilage [3]. Though there are clear evidence of association between various pathogens and the occurrence of inflammatory arthritis, it would not be an overstatement to say that the exact pathogenetic mechanisms remain unclear. Clinical observations have suggested that the clinical features and natural course of the inflammatory arthritis varies to some extent depending on whether the trigger is genitourinary or gastrointestinal. Precision medicine in the subject would require further research on molecular mechanisms that will help to individualize the treatment of these patients.

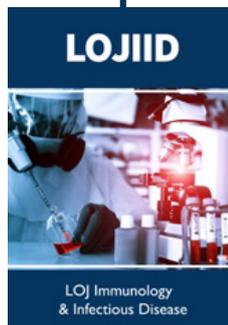
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