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MOLECULAR REGULATION OF INFLAMMATORY SIGNALING IN CHRONIC RHEUMATIC DISEASES

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Systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA) are chronic autoimmune inflammatory diseases that predominantly effect women. RA primarily affects the synovial joints where infiltrating immune cells release inflammatory cytokines such as tumour necrosis factor (TNF) and interleukin-1 (IL-1). This leads to the destruction of cartilage and bone, whilst the synovial membrane becomes thickened due to cell infiltration and proliferation. The disease is both rapid and progressive, causing irreversible damage to the joints. SLE is a more systemic autoimmune disease with damage occurring to many tissues throughout the body. Symptoms can include arthritis, nephritis, neurological disorders and dermatological problems, with patients experiencing varying combinations of symptoms leading to a range of disease severity. Overall, SLE presents as one of the most clinically diverse autoimmune diseases, making diagnosis and treatment highly challenging. In both diseases, a family of innate immune receptors the toll-like receptors (TLRs) have been proposed to contribute to the pathogenesis. TLRs recognise both pathogens and damage associated molecular patterns resulting in inflammatory cytokine production. Using primary human tissue and blood samples from rheumatology patients our research has demonstrated potential roles for individual TLRs. Most recently, our work has focused on the molecular regulation of the downstream TLR signalling pathways in patient monocytes. This has led to the identification of several negative regulators of signalling that are altered in either RA or SLE compared to healthy volunteers. Changes to the function of these regulators have the potential to perpetuate inflammatory cytokine production in these patients. Understanding which receptors and signalling molecules are contributing to the production of excessive inflammation in these diseases is of great importance, as this knowledge will provide targets for the development of future therapies.

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