Decoding what goes wrong in arthritis

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Fresh insight into rheumatoid arthritis offers hope for transforming patient care

Scientists have discovered what they believe has the potential to prevent the onset of an aggressive and hard-to-treat form of rheumatoid arthritis - a condition that affects 700,000 adults in the UK.  
  
Published in the Journal of Experimental Medicine, a team of immunologists from Cardiff tread new ground in describing how an immune system protein - interleukin-27 - regulates the inflammatory process in lymphoid-rich rheumatoid arthritis, which causes the characteristic symptoms of swollen and painful joints.

This form of the disease, a long-term and disabling condition, accounts for up to 40% of diagnoses. Currently, an estimated two in every five patients with the disease do not respond to existing treatment and so the disease can often be problematic to treat.  
  
The Cardiff team have marked a first in being able to explain how this variant of the disease develops. To demonstrate this they used experimental models of arthritis involving mice, cells and tissue biopsies taken from patients with early symptoms of the condition, using a novel ultrasound technique.   
  
Understanding this process, they say, will enable doctors to divide patients into different sub-groups based on the often greatly varying patterns of disease, which is influenced by how much interleukin-27 is present in each patient’s joints.

Which disease sub-group a patient falls in to will inform the course of therapy they will receive, meaning a more tailored approach to treating their condition, offering them a much better chance of overcoming it.

The researchers also anticipate that their identification of interleukin-27 involvement in this specific disease context will kick-start the search for new drugs that manipulate the pathways controlled by this factor. Dr Gareth Jones, from the School of Medicine’s Institute of Infection & Immunity, said:   
  
“In all forms of rheumatoid arthritis, it is widely understood that early intervention offers the best chance for clinical remission. The sooner treatment begins, the more effective the therapeutic response is likely to be.   
  
“The key is identifying which drug is best suited for an individual patient. Making the correct treatment decisions, sufficiently early in the disease process will improve disease outcome, enhance a patients wellbeing and overall quality of life.   
  
“Our research is identifying crucial pathways and mechanisms that allow us to distinguish between different sub-types of rheumatoid arthritis, using experimental models that mirror human forms of the disease.  Agents that manipulate the activities of these pathways may also serve as potential therapies for future development.”

Professor Christopher Buckley, a researcher from the Rheumatology Research Group at the University of Birmingham, said:“The potential of interleukin-27 as a marker to stratify patients with RA into different groups is a very important discovery that will help transform our ability to use a more personalized approach in the management of patients with the most aggressive form of the disease.  
  
“Furthermore, identifying interleukin-27 as a bio-marker of the type of rheumatoid arthritis in which lymphoid tissue forms in the synovium, suggests that targeting this cytokine might be beneficial.”

Rheumatoid arthritis affects an estimated one per cent of the world’s population and there are 20,000 new diagnoses every year in the UK alone. Each year the NHS spends £560M on biological drug treatments to mitigate its effect.  
  
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