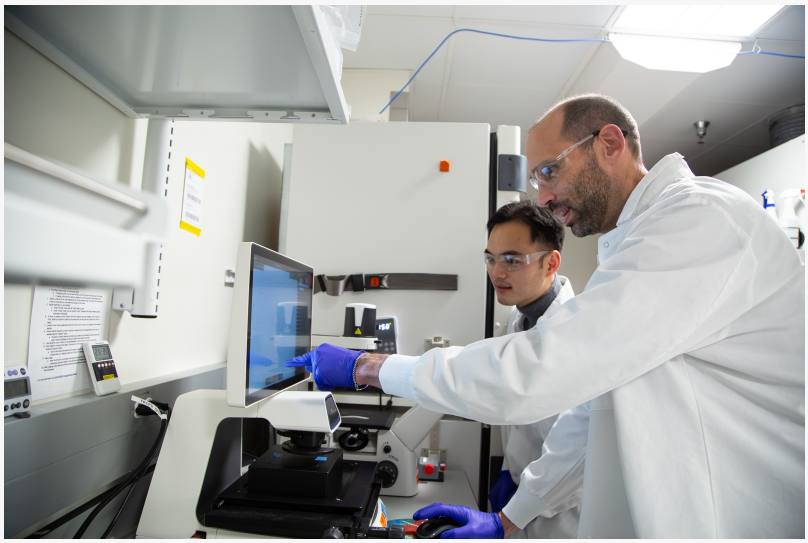
New study reveals rheumatoid arthritis begins long before symptoms, opening door to prevention

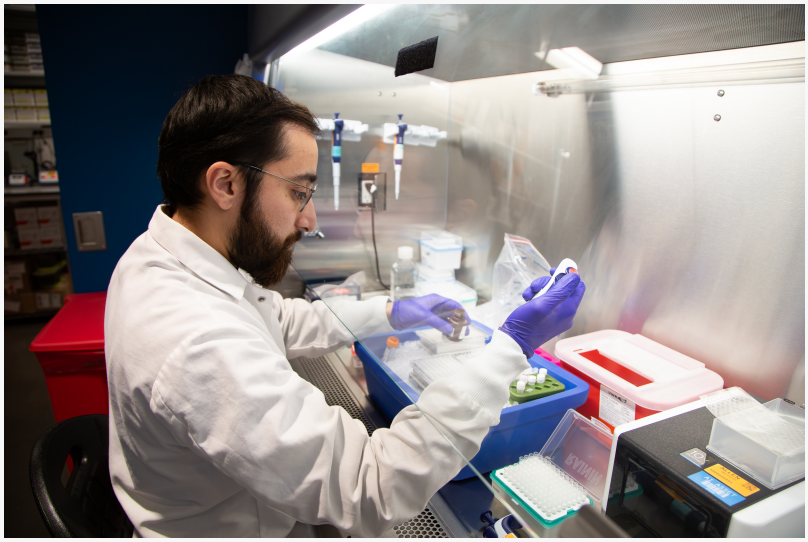
The research reveals early-warning signs that could help doctors catch the disease before it starts, potentially saving patients years of pain and disability.

SEATTLE, WA, UNITED STATES, September 24, 2025 / EINPresswire.com/ -- Scientists have discovered that rheumatoid arthritis (RA) doesn't start when the pain begins. It silently starts years earlier. RA is a debilitating autoimmune disease that causes painful joint inflammation and damage. The new research reveals that people at risk for RA experience dramatic immune system changes long before they feel symptoms. During this early phase, their bodies fight an autoimmune battle invisibly.

Researchers at the Allen Institute, in collaboration with the CU Anschutz, University of California San Diego, and Benaroya Research Insititute made the discovery. The study, published in the journal Science Translational Medicine delivers the most detailed look available into how rheumatoid arthritis develops, mapping immune changes in at-risk individuals long before symptoms appear and paving the way for earlier treatment and



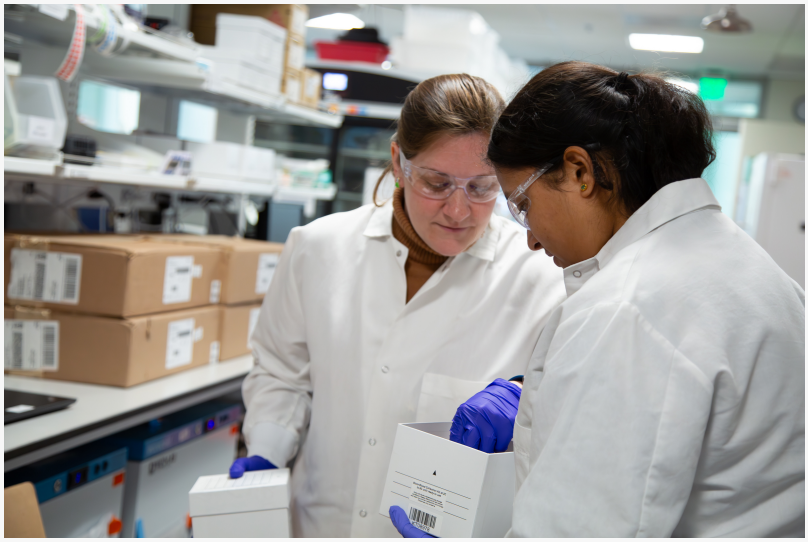
Ziyuan He, Ph.D., (left) and Mark Gillespie, Ph.D., working in the lab at the Allen Institute. Photo by Jenny Burns.



Neel Kaul, a researcher at Allen Institute, works with immunology research samples under the hood in the laboratory. Photo by Jenny Burns.

prevention.

“Overall, we hope this study raises awareness that rheumatoid arthritis begins much earlier than previously thought and that it enables researchers to make data-driven decisions on strategies to disrupt disease development,” said Mark Gillespie, Ph.D., assistant investigator at the Allen Institute and co-senior author with Kevin Deane (CU Anschutz), M.D./Ph.D.; Adam Savage (Allen Institute), Ph.D.; Troy Torgerson (Allen Institute), M.D./Ph.D.; and Gary S. Firestein (UC San Diego), M.D. During the seven-year study, researchers tracked people carrying ACPA antibodies, which are known biomarkers for individuals at-risk for developing RA, and identified previously unknown factors associated with disease development, including widespread inflammation, immune cell dysfunction, and cellular reprogramming.



Christy Bennett, M.S., M.B.A., (left), and Pravina Venkatesan, (right), preparing research samples in the lab at Allen Institute. Photo by Jenny Burns.

Pravina Venkatesan, (left), Christy Bennett, M.S., M.B.A., (middle) and Ziyuan He, Ph.D., (right) observing data collection at Allen Institute. Photo by Jenny Burns.

“We expect that going forward the findings from this study will support additional studies to identify ways to better predict who will get RA, identify potential biologic targets for preventing RA as well as identify ways to improve treatments for those with existing RA.,” said Kevin Deane, M.D./Ph.D.

Key Findings:
— Widespread inflammation: Researchers discovered that systemic inflammation was already present throughout the body in at-risk individuals. This wasn't localized joint inflammation, but rather a body-wide inflammatory state that resembles what's seen in people with active RA.
— Immune cell dysfunction: Several types of immune cells showed significant abnormalities: B cells, which normally produce protective antibodies, had shifted toward a pro-inflammatory state. T helper cells, particularly a subset resembling Tfh17 cells, were dramatically expanded

beyond normal levels. These cells play crucial roles in directing immune responses, including autoantibody (antibodies that attack the body's own tissue) production and their overactivity helps explain why the immune system begins attacking healthy tissue.

— Cellular reprogramming: Perhaps most remarkably, the study found that even "naive" T cells—immune cells that haven't encountered threats before—showed epigenetic changes. This means the cells' DNA wasn't mutated, but the way genes were turned on and off had been altered, essentially reprogramming these cells before they even encountered their first threat.

Pravina Venkatesan observes immunological data collection at Allen Institute. Photo by Jenny Burns.

— Joint-like inflammation in blood: The researchers identified monocytes (a type of white blood cell) in the bloodstream that were producing high levels of inflammatory molecules. Significantly, these blood cells closely resembled the macrophages found in the inflamed joint tissue of RA patients, suggesting the disease process was already preparing to target joints.

The study reveals new early-warning signs (biomarkers and immune signatures) that could help doctors identify who among at-risk individuals is most likely to develop RA, enabling more targeted monitoring and earlier intervention. If caught early, RA could be stopped before it starts—saving patients years of pain and disability. This research may enable a major shift away from reactive treatments that rely on the appearance of joint damage and towards proactive prevention.

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