Poster presenter (name, credentials, title, institution)

Eric W. Robbins, MD

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Poster title:

Serial Quantitative Changes in Anti-Scl-70 Autoantibodies Are Associated with Lung Function Decline in Systemic Sclerosis

What is your poster about?

Our study investigates whether dynamic, quantitative monitoring of anti-Scl-70 autoantibody titers can predict lung function decline and disease progression in patients with systemic sclerosis (SSc). Using a research assay known as luciferase immunoprecipitation systems (LIPS), we evaluated 86 patients from the multicenter CONQUER registry. We found that baseline antibody levels alone did not correlate with clinical measures among antibody-positive patients. However, early increases in quantitative anti-Scl-70 levels over 6 months were significantly associated with subsequent forced vital capacity (FVC) decline. Specifically, a >30% early increase in anti-Scl-70 levels was associated with a nearly four-fold increased risk of pulmonary progression over two years.

Why did you decide to investigate this topic?

Our group had previously done work in myositis-associated interstitial lung disease (ILD), and we wanted to broaden our focus to other rheumatic disease resulting in pulmonary fibrosis. We knew that anti-Scl-70 autoantibodies are a well-established risk factor for systemic sclerosis–associated ILD, but we wanted to see if there was a more dynamic measure we could follow that might tell clinicians a patient's risk trajectory. Our hope was to find a measure clinicians could serially monitor to better predict a patient's risk of pulmonary worsening.

What are you working on next related to this research?

Broadly, we are working on a new project to investigate shared proteomic profiles of pulmonary fibrosis across a spectrum of autoimmune rheumatic diseases, such as myositis, systemic sclerosis, and Sjögren's syndrome.

Simultaneously, to directly extend the specific work in this abstract, we plan to implement high-breadth proteomic profiling in the CONQUER cohort. We anticipate this expanded analyte coverage will provide deeper biological insights and identify stronger molecular correlations with the risk of pulmonary progression.

We're currently working on a project looking at shared proteomic profiles of pulmonary fibrosis across a number of rheumatic diseases (myositis, systemic sclerosis, Sjögren's, etc.). To extend the work in this abstract, we think that incorporating high-breadth proteomic profiling. That approach will allow us to capture a more comprehensive biological narrative and hopefully uncover molecular signatures tightly correlated with clinical progression.

What excites you most about your work?

I'm excited about the possibility of having more tools to enable dynamic, proactive monitoring in scleroderma ILD care, rather than just a static risk assessment. Identifying an early biomarker trajectory that precedes clinical decline could give rheumatologists and pulmonlogits a crucial window of opportunity to tailor their monitoring or therapeutic strategies before irreversible lung damage occurs.

What are you most looking forward to at ACR Convergence 2026 in Orlando?

As a pulmonologist, I look forward to connecting with rheumatology colleagues, translational researchers, and other ILD specialists. There's some amazing work going on in the rheumatic biomarker field, and I'm keenly watching several ongoing trails testing new immunotherapies in refractory rheumatologic disease. I am eager to share these insights from the CONQUER cohort, see what other findings presented might be integrated into clinical workflows, and discover new advancements across the field of rheumatology.

- ACR 2026, abstract submission

Registration

Early Registration: August 26, 23:59 EST

Timing: November 6–11

Abstract Character Limit

- Title limit: 250 characters, excluding spaces
- Body character limit: 2,750 characters, which EXCLUDES the title, names of authors/co- authors, authors' affiliations, spacing, and disclosures.
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