Introduction: Atrial arrhythmias are commonly observed in individuals with systemic lupus erythematosus (SLE), a chronic autoimmune disorder characterized by multi-systemic inflammation. The mechanisms linking these conditions remain to be elucidated. In this study, we conduct in silico experiments to explore the potential association between atrial arrhythmias in SLE patients and the combined impact of structural and electrical remodeling from chronic inflammation.

Methods: Structural remodeling was modeled as interstitial fibrosis, and electrical remodeling was attributed to chronic inflammation observed in SLE patients. Eikonal-based simulations were employed to simulate wavefront propagation in a biatrial tetrahedral mesh.

Results: Our results suggest that electrical remodeling related to SLE can modify the depolarization pattern and promote the emergence of reentry patterns, potentially initiating arrhythmias. We found that a combination of fibrosis (10% or 40%) and mild inflammation was insufficient to induce arrhythmias. In contrast, a combination of fibrosis (10% or 40%) and severe inflammation could produce non-sustained arrhythmias exhibiting repetitive patterns.

Conclusion: Our findings offer insights into the mechanisms responsible for developing atrial arrhythmias in SLE patients and propose that inflammation-driven structural and electrical remodeling may contribute to this condition. This study serves as a starting point for further examination of the complex relationship between SLE, chronic inflammation, and atrial arrhythmias. Moreover, thanks to in silico experiments, understanding inflammation’s role in arrhythmogenesis could help improve the management and outcomes for patients with SLE and atrial arrhythmias.