

Rheumatoid Arthritis and Muscle Wasting: New Review Points to Overlooked Complications

Researchers explore how rheumatoid arthritis causes muscle loss, uncovering complex mechanisms that may guide future treatments

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EINPresswire.com/ -- Myopenia, a condition marked by abnormal muscle loss, affects people across all age groups. Though linked to various diseases, myopenia appears to have distinct origins and clinical features in rheumatoid arthritis (RA). Researchers have now examined its underlying mechanisms in unprecedented detail, revealing how RA drives muscle wasting. Their findings offer critical insights into age-specific presentations of myopenia and highlight emerging opportunities to improve care and functional outcomes for patients living with rheumatoid arthritis.

Rheumatoid arthritis (RA) is a chronic



Researchers have illustrated how inflammatory and systemic factors contribute to myopenia, sarcopenia, and cachexia in rheumatoid arthritis, driving musculoskeletal aging and increased morbidity.

autoimmune disease that affects individuals across all ages and genders. While its most visible impact is on the joints, RA also contributes to accelerated musculoskeletal ageing, often leading to progressive muscle degeneration and reduced muscle function. Emerging research has identified a specific form of muscle loss in RA—known as myopenia—which differs significantly from other disease-related muscle-wasting conditions such as cancer cachexia or heart failure. Unlike those conditions, myopenia is characterized by a decline in muscle mass without concurrent fat loss and can manifest across all age groups.

Despite growing recognition of myopenia's clinical impact, its precise biological mechanisms

remain poorly understood. To address these gaps, a team of researchers led by Professor Jiake Xu from School of Biomedical Sciences, The University of Western Australia, Australia, conducted a comprehensive review exploring the underlying pathways of myopenia in RA. Their findings were published in Bone Research on June 16, 2025. "The intersection of myopenia and accelerated musculoskeletal ageing in RA represents a multifaceted area of research, highlighting both the urgency and potential for optimizing patient outcomes through targeted care," explains Prof. Xu.

In their article, the authors investigate two overlapping conditions contributing to muscle loss in RA: myopenia and secondary sarcopenia. While primary sarcopenia refers to age-related muscle decline, secondary



sarcopenia results from underlying illness or its treatment. In contrast, myopenia involves clinically significant muscle loss that can affect individuals of any age, independent of normal ageing. In RA patients, myopenia leads to notable muscle mass loss without concurrent fat loss—worsening functional capacity, frailty, and mortality risk.

Importantly, the researchers describe how myopenia and secondary sarcopenia in RA follow a non-linear trajectory of muscle decline, differing from the steady, gradual loss seen in agerelated sarcopenia. "Patients with RA experience more severe reductions in muscle mass than their healthy age- and sex-matched peers, making myopenia an indicator of early, diseaserelated muscular ageing," notes Prof. Xu.

In discussing the role of myopenia in RA, researchers explore the known genetic factors contributing to RA and discuss the clinical characteristics of myopenia. Furthermore, the researchers differentiate between the distinct clinical characteristics of myopenia in elderly-onset rheumatoid arthritis (EORA) and young-onset rheumatoid arthritis (YORA), highlighting key differences in presentation.

The review also outlines the major factors contributing to RA-associated myopenia, including chronic inflammation, oxidative stress, pro-inflammatory cytokines, hormonal imbalances, and genetic influences. In addition, it discusses how the clinical features of myopenia vary depending

on age of RA onset. In elderly-onset RA (EORA), baseline muscle mass is already reduced due to ageing, and this is compounded by inflammatory pathways and neuromuscular deterioration. "EORA patients experience muscle mass reduction, further weakening their physical capacity," says Prof. Xu.

By contrast, young-onset RA (YORA) patients typically begin with higher muscle mass and less neuromuscular ageing, but prolonged inflammation over time can gradually impair function. Understanding these age-specific differences is crucial for designing appropriate interventions.

The review highlights the diagnostic and therapeutic relevance of myopenia in RA. Early detection of myopenia could serve as a clinical marker of RA onset, enabling earlier intervention. Evidence supports the use of physical activity—particularly aerobic and resistance training—as an effective strategy to preserve muscle mass. Additionally, nutritional support and pharmacological treatments may also play a role in prevention and management.

"Early detection of myopenia combined with a tailored management strategy—encompassing pharmacological treatments, personalized exercise regimens, psychological support, and dietary modifications—may alleviate muscle loss, reduce frailty, and mitigate risks associated with sarcopenic obesity," Prof. Xu concludes.

With further research, these insights may help enhance the quality of life and physical independence of individuals living with rheumatoid arthritis.

Reference Titles of original papers: Pathophysiology of Myopenia in rheumatoid arthritis Journal: Bone Research DOI: <u>10.1038/s41413-025-00438-9</u>

About Professor Jiake Xu from Shenzhen University of Advanced Technology, Shenzhen Institutes of Advanced Technology, and The University of Western Australia Prof. Jiake Xu is Distinguished Professor at Shenzhen University of Advanced Technology, and Shenzhen Institutes of Advanced Technology, Chinese Academy of Sciences, and adjunct Winthrop Professor and Head of the Molecular Laboratory and Division of Regenerative Biology at the University of Western Australia (UWA). A Fellow of the Royal College of Pathologists of Australasia, ASBMR, and the International Orthopaedic Research Society, he completed his PhD at UWA and postdoctoral training at Stanford University. His research focuses on osteoclast biology, RANKL signaling, osteoimmunology, and the molecular crosstalk between osteoclasts and osteoblasts. He also studies angiogenic and angiocrine regulation in the bone microenvironment. Prof. Xu has published over 250 SCI papers and is globally ranked among the top experts in skeletal biology.

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