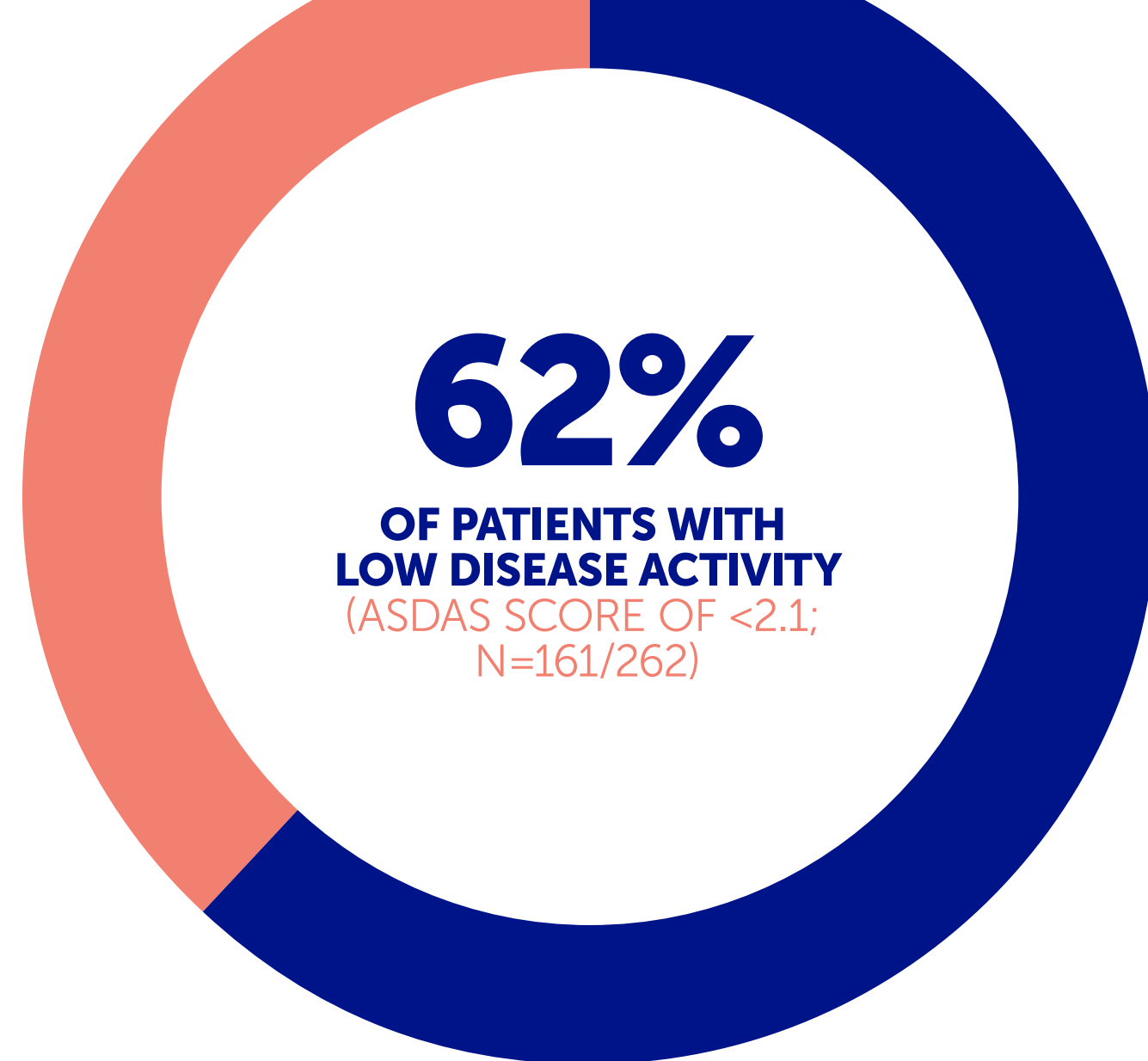
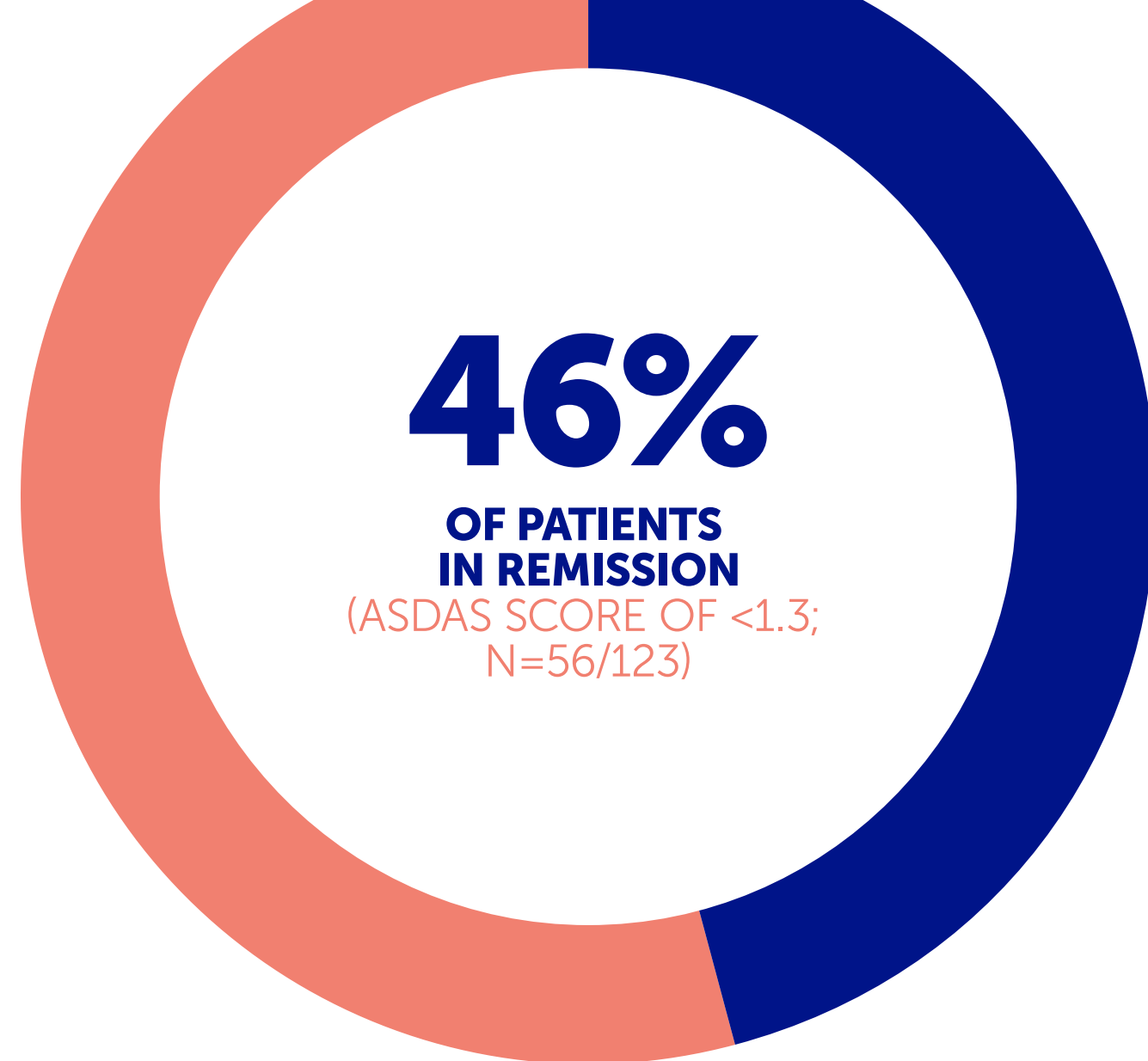


DOES ACHIEVING CLINICAL REMISSION ENSURE SYMPTOM RESOLUTION IN SpA?

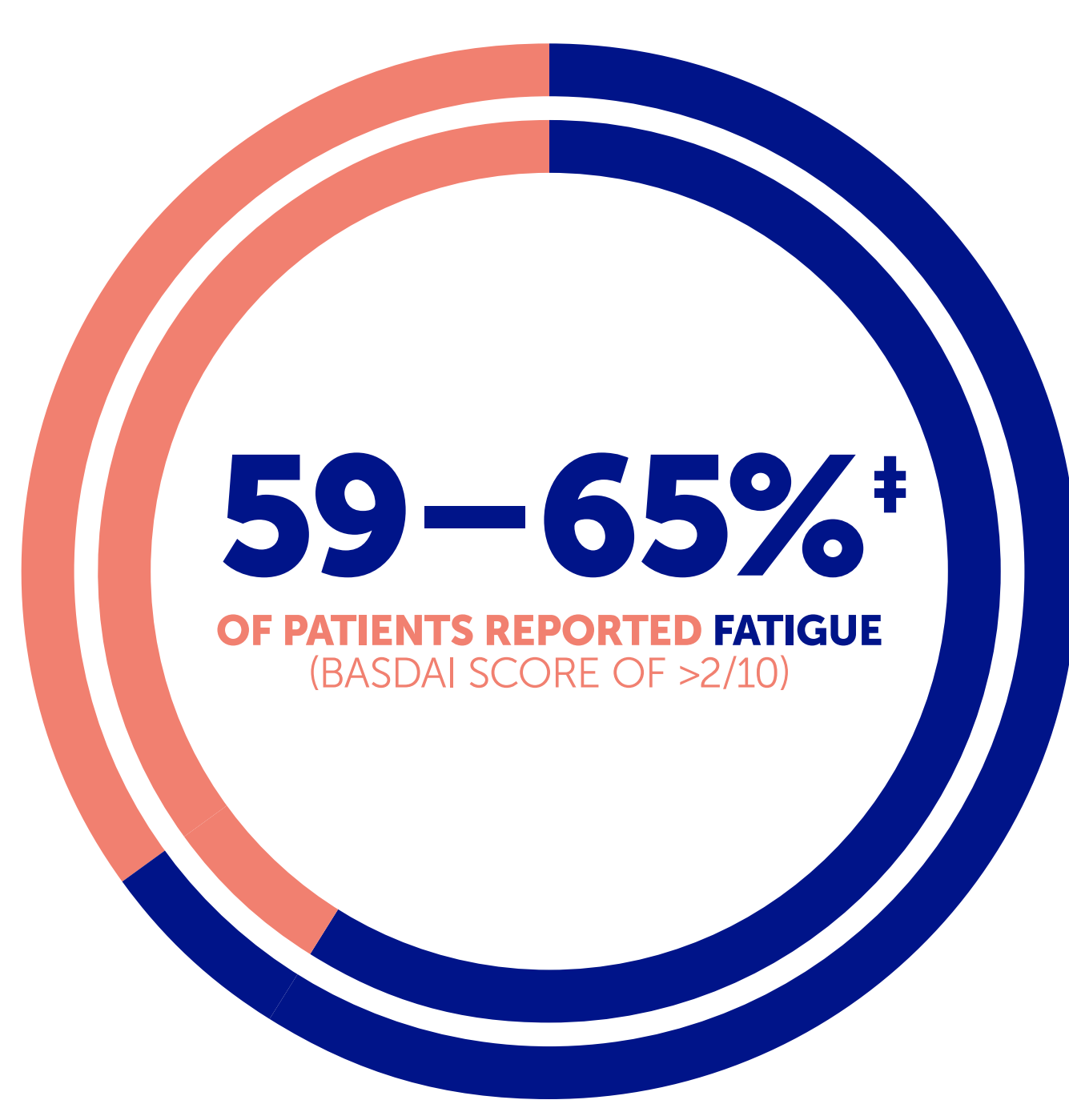
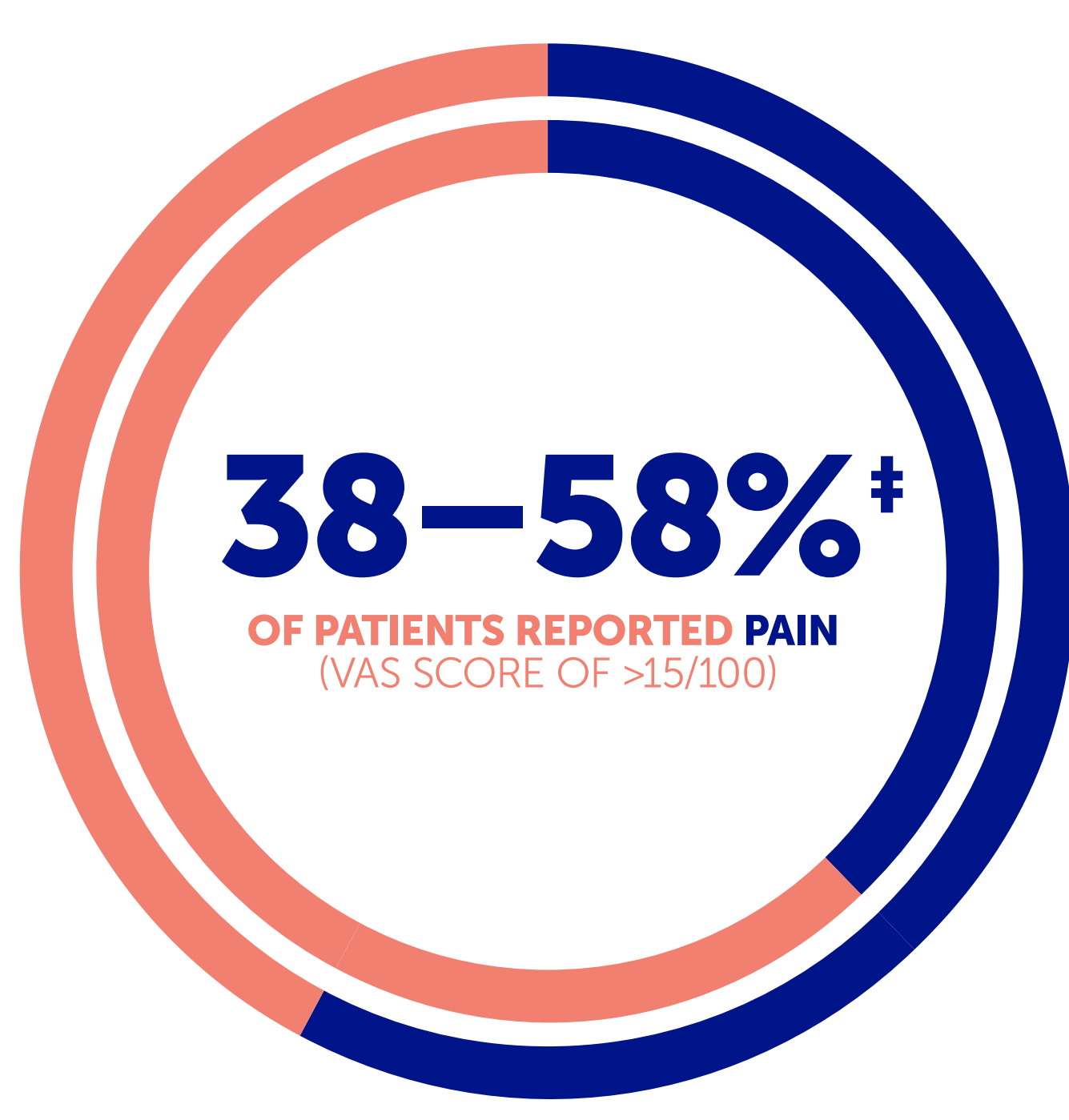
For patients with axSpA and PsA, achieving remission or low disease activity is a primary therapeutic goal.^{1–3} Yet even when these targets are met, residual inflammation and symptom burden may continue to impact patients.^{4–6}

THE DISCONNECT BETWEEN MEASURED DISEASE ACTIVITY AND PATIENT-PERCEIVED CONTROL

In a post hoc analysis of patients with axSpA, residual symptoms were reported in:^{*7}



DESPITE ACHIEVING LOW DISEASE ACTIVITY, IN axSpA AND PsA:^{t6}



^tBased on four subgroups: patients with axSpA who achieved low disease activity defined by either ASDAS-ESR score of <2.1 or BASDAI score of <3/10, and patients with PsA who achieved low disease activity defined by either 5/7 cutoffs in MDA or a cDAPSA score of ≤13.⁶

IN A 36-ITEM SHORT FORM HEALTH SURVEY, PATIENTS WITH axSpA AND PsA WITH LOW DISEASE ACTIVITY ALSO REPORTED SUBSTANTIAL BURDEN IN:^{t6}



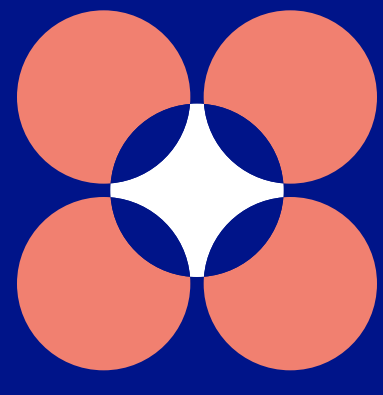
UNDERSTANDING THE DISCONNECT

Several factors contribute to the disconnect between patient-reported symptoms and objective markers of inflammation in axSpA. Persistent burden may arise from both inflammatory and non-inflammatory mechanisms, highlighting the need for a multidimensional approach to remission.⁵

PATIENTS WITH NO SYMPTOMS BUT OBJECTIVE SIGNS OF INFLAMMATION

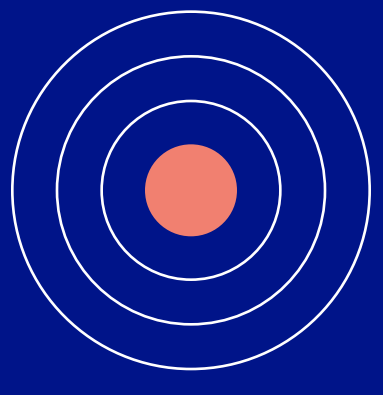
Silent inflammation

Even in the absence of symptoms, inflammation may persist and drive long-term structural damage and disease progression.⁵



Biomarker limitations

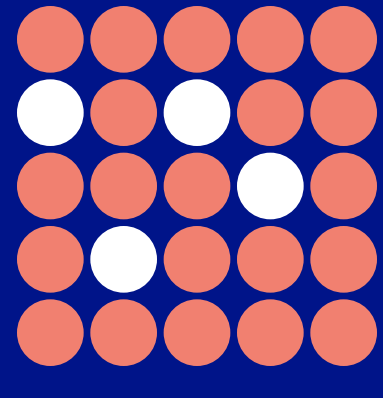
CRP lacks specificity and doesn't always reflect the true inflammatory activity attributable to axSpA. For example, obesity, smoking, infections and subclinical comorbidities can independently elevate CRP.⁵



PATIENTS WITH SYMPTOMS WITHOUT OBJECTIVE SIGNS OF INFLAMMATION

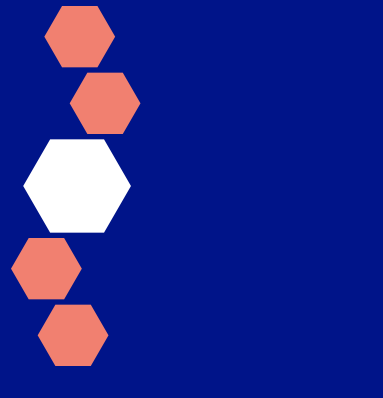
Residual pain post-inflammation

Previous inflammatory episodes can lead to structural changes, peripheral nerve sensitisation, or maladaptive neuroplastic changes.⁵



Mechanical pain

Co-existing degenerative spine disease or irreversible structural damage can alter biomechanics and reduce mobility.⁵



Central sensitisation and fibromyalgia

Pathological enhancement of nociceptive processing can lead to disproportionate pain perception.⁵



EVALUATING THE FULL PATIENT BURDEN

PRO measures are essential for assessing remission, enabling a more comprehensive evaluation of the disease's impact on patients.^{5,8}

PROs offer insights into:^{8,9}

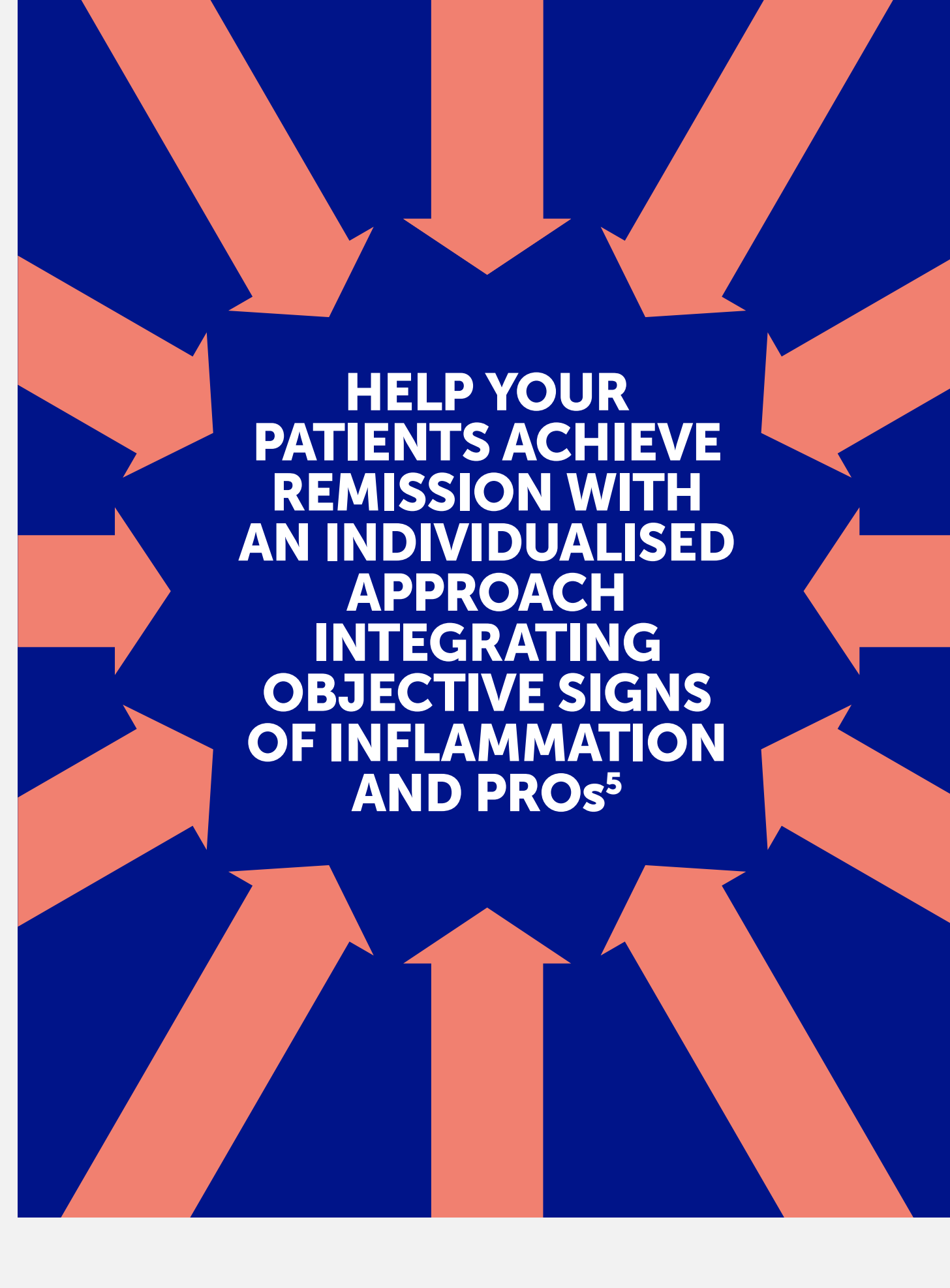
FUNCTIONAL STATUS AND DAILY ACTIVITIES

PAIN SEVERITY

FATIGUE

PHYSIOLOGICAL WELL-BEING

HEALTH-RELATED QUALITY OF LIFE



^{*}Post hoc analysis of the QUASAR study, which included 480 people with axSpA. Remission or inactive disease was defined as having an ASDAS score of <1.3. Low disease activity was defined as having an ASDAS score of <2.1.⁷

^tCross-sectional baseline data of the PREcision medicine in Spondyloarthritis for Better Outcomes aND Disease Remission (PRESPOND) registry from a tertiary rheumatology centre in Singapore General Hospital from 2012 to 2018. Data from a total of 262 patients with axSpA and 142 patients with PsA were analysed in the study. Low disease activity for axSpA was defined as having an ASDAS-ESR score of <2.1 or a BASDAI score of <3/10. Low disease activity for PsA was defined as 5/7 cutoffs in MDA or a cDAPSA score of ≤13.⁶

Abbreviations

ASDAS: Ankylosing Spondylitis Disease Activity Score; ASDAS-ESR: Ankylosing Spondylitis Disease Activity Score based on erythrocyte sedimentation rate; axSpA: axial spondyloarthritis; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; cDAPSA: Clinical Disease Activity in Psoriatic Arthritis; CRP: C-reactive protein; MDA: minimal disease activity; PRO: patient-reported outcome; PsA: psoriatic arthritis; SpA: spondyloarthritis; VAS: visual analogue scale.

References

1. Coates LC, et al. Nat Rev Rheumatol. 2022;18(8):465–479.
2. Gossec L, et al. Ann Rheum Dis. 2024;83(6):706–719.
3. Ramiro S, et al. Ann Rheum Dis. 2023;82(1):19–34.
4. Macchioni P, et al. Front Med (Lausanne). 2022;9:1096547.
5. Protopopov M, Proft F. Joint Bone Spine. 2026;93(2):105946.
6. Liu V, et al. J Rheumatol. 2021;48(5):677–684.
7. D'Angelo S, et al. J Clin Med. 2022;11(12):3553.
8. Orbai AM, Ogdie A. Rheum Dis Clin North Am. 2016;42(2):265–283.
9. Garrido-Cumbrera M, et al. Rheumatol Ther. 2017;4(2):219–231.