Ankylosing Spondylitis (AS) vs Rheumatoid Arthritis (RA): The differences go deep

From cytokine signaling to clinical signs in AS and RA, research reveals key distinctions that are changing the way we see these conditions.[1,2]

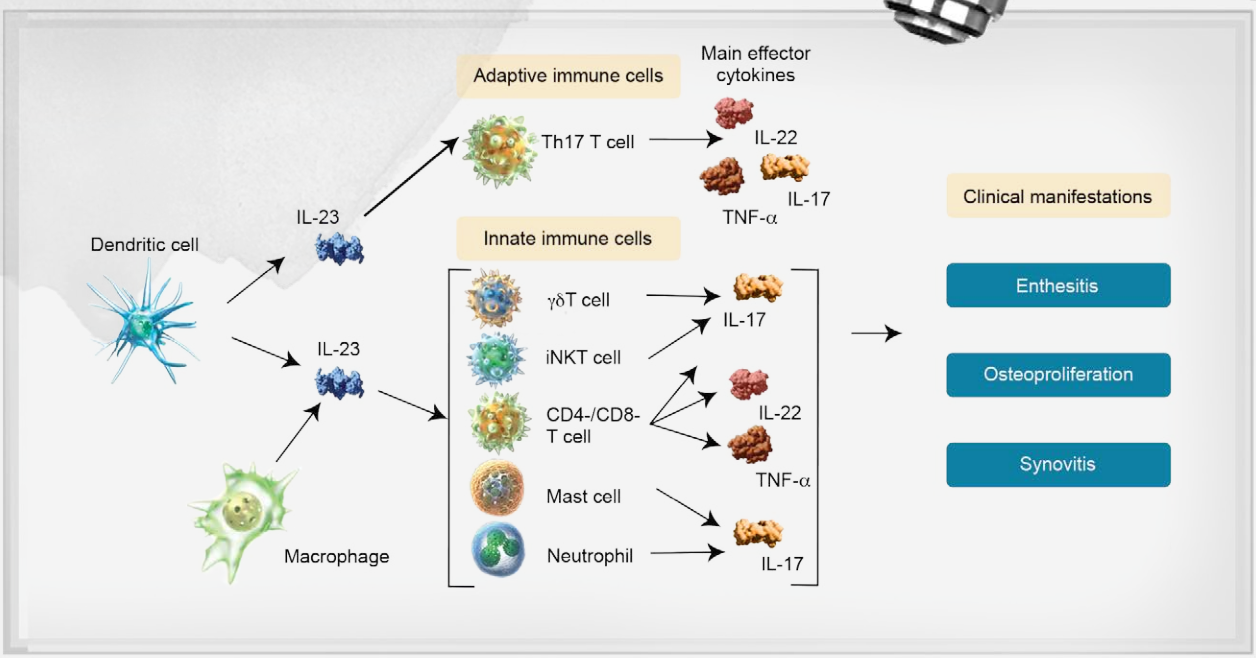
AS vs RA:
A deeply different pathogenesis

Recent investigations have revealed distinctions in the T cell subtypes associated with the pathogenesis of AS and RA:

- **B cells linked with rheumatoid factor (RF):** The presence of serum RF and spontaneous RF-secreting B cells is a common feature in most patients with RA[3,4]
- **T cells linked with HLA-B27:** In contrast, most patients with AS are RF-negative. Many AS patients in particular are positive for HLA-B27, which is thought to facilitate the role of T cells in the pathogenesis of all spondyloarthritis, including AS[5,6]
- **IL-17A–expressing T cell levels are higher in AS:** IL-17A–expressing T cell levels are elevated in the blood and facet joints of patients with AS[6-9]

Multiple cell types and cytokines are implicated in AS[10]

The pathophysiology of AS is complex and involves the interplay between multiple cell types and cytokines, including dendritic cells, T cells, TNF-α, IL-17, and IL-22.[10]



HLA=human leukocyte antigen; IL=interleukin; iNKT=invariant natural killer T; Th=T helper cells; TNF=tumor necrosis factor.
Adapted with permission from Smith JA, Colbert RA. *Arthritis Rheumatol*. 2014;66(2):231-241.

Learn more about AS vs RA at RealityofSpA.com

A deeply different burden

Similar prevalence rates have been reported for **both AS and RA**[11]
- AS is largely undiagnosed; delays of up to 9 years have been reported

Age of onset is earlier in patients with AS than RA[11]
- AS: Mean age of **onset is at 20 to 30 years**
- RA: Younger **onset is at 42 years**, older **onset is at 68 years**

Radiographic changes may not be seen for up to 10 years after the onset of clinical symptoms in AS[11]

Main structural outcome in AS is abnormal new bone formation vs bone erosion in RA[2,12]

AS targets different sites than RA[2,12-15]

	RA	AS
Symmetrical	✕	
Entheseal		✕
New bone formation (syndesmophytes)		✕
Peripheral joints		
Metacarpophalangeal joints	✕	
Proximal interphalangeal joints	✕	
Lower limb joints		✕
Axial joints		
Spine		✕
Sacroiliac joints		✕

AS is different from RA

Investigators are taking a closer look at the distinctive pathogenic mechanisms of AS, including the roles of T cells and cytokines such as IL-17A and IL-22.[6,10]

Further elucidation of these AS disease mechanisms may point to an independent direction in the science behind AS.

See the differences at RealityofSpA.com

References: 1. Khan MA. Ankylosing spondylitis and related spondyloarthropathies: the dramatic advances in the past decade. *Rheumatology.* 2011;50(4):637-639. **2.** Lories RJ, Baeten DL. Differences in pathophysiology between rheumatoid arthritis and ankylosing spondylitis. *Clin Exp Rheumatol.* 2009;27(suppl 55):S10-S14. **3.** Hoffmann M, Lundberg K, Steiner G. Autoantibodies in rheumatoid arthritis. In: Hochberg MC, Silman AJ, Smolen JS, Weinblatt ME, Weisman MH, eds. *Rheumatology.* 6th ed. Philadelphia, PA: Elsevier; 2015:51-82. **4.** Nakken B, Munthe LA, Konttinen YT, et al. B-cells and their targeting in rheumatoid arthritis—current concepts and future perspectives. *Autoimmun Rev.* 2011;11(1):28-34. **5.** Rudwaleit M. Classification and epidemiology of spondyloarthritis. In: Hochberg MC, Silman AJ, Smolen JS, Weinblatt ME, Weisman MH, eds. *Rheumatology.* 6th ed. Philadelphia, PA: Elsevier; 2015:83-90. **6.** Baeten D. Etiology, pathogenesis, and pathophysiology of ankylosing spondylitis. In: Hochberg MC, Silman AJ, Smolen JS, Weinblatt ME, Weisman MH, eds. *Rheumatology.* 6th ed. Philadelphia, PA: Elsevier; 2015:102-113. **7.** Appel H, Kuhne M, Spiekermann S, et al. Immunohistologic analysis of zygapophyseal joints in patients with ankylosing spondylitis. *Arthritis Rheum.* 2006;54(9):2845-2851. **8.** Shen H, Goodall JC, Hill Gaston JS. Frequency and phenotype of peripheral blood Th17 cells in ankylosing spondylitis and rheumatoid arthritis. *Arthritis Rheum.* 2009;60(6):1647-1656. **9.** Appel H, Maier R, Wu P, et al. Analysis of IL-17$^+$ cells in facet joints of patients with spondyloarthritis suggests that the innate immune pathway might be of greater relevance than the Th17-mediated adaptive immune response. *Arthritis Res Ther.* 2011;13(3):R95. **10.** Smith JA, Colbert RA. Review: the interleukin-23/interleukin-17 axis in spondyloarthritis pathogenesis: Th17 and beyond. *Arthritis Rheumatol.* 2014;66(2):231-241. **11.** Cross MJ, Smith EU, Zochling J, March LM. Differences and similarities between ankylosing spondylitis and rheumatoid arthritis: epidemiology. *Clin Exp Rheumatol.* 2009;27(suppl 55):S36-S42. **12.** Schett G, Coates LC, Ash ZR, Finzel S, Conaghan PG. Structural damage in rheumatoid arthritis, psoriatic arthritis, and ankylosing spondylitis: traditional views, novel insights gained from TNF blockade, and concepts for the future. *Arthritis Res Ther.* 2011;13(suppl 1):S4. **13.** Aletaha D, Neogi T, Silman AJ, et al. 2010 rheumatoid arthritis classification criteria: an American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Arthritis Rheum.* 2010;62(9):2569-2581. **14.** Braun J, Sieper J. Ankylosing spondylitis. *Lancet.* 2007;369(9570):1379-1390. **15.** Lories RJ, Luyten FP, de Vlam K. Progress in spondylarthritis: mechanisms of new bone formation in spondyloarthritis. *Arthritis Res Ther.* 2009;11(2):221.

NOVARTIS

QR CODE FOR INTERNAL USE ONLY.

Novartis Pharmaceuticals Corporation
East Hanover, New Jersey 07936-1080

Printed in USA 5/17 XAT-1343506