Obesity and Psoriatic Arthritis Pathophysiology

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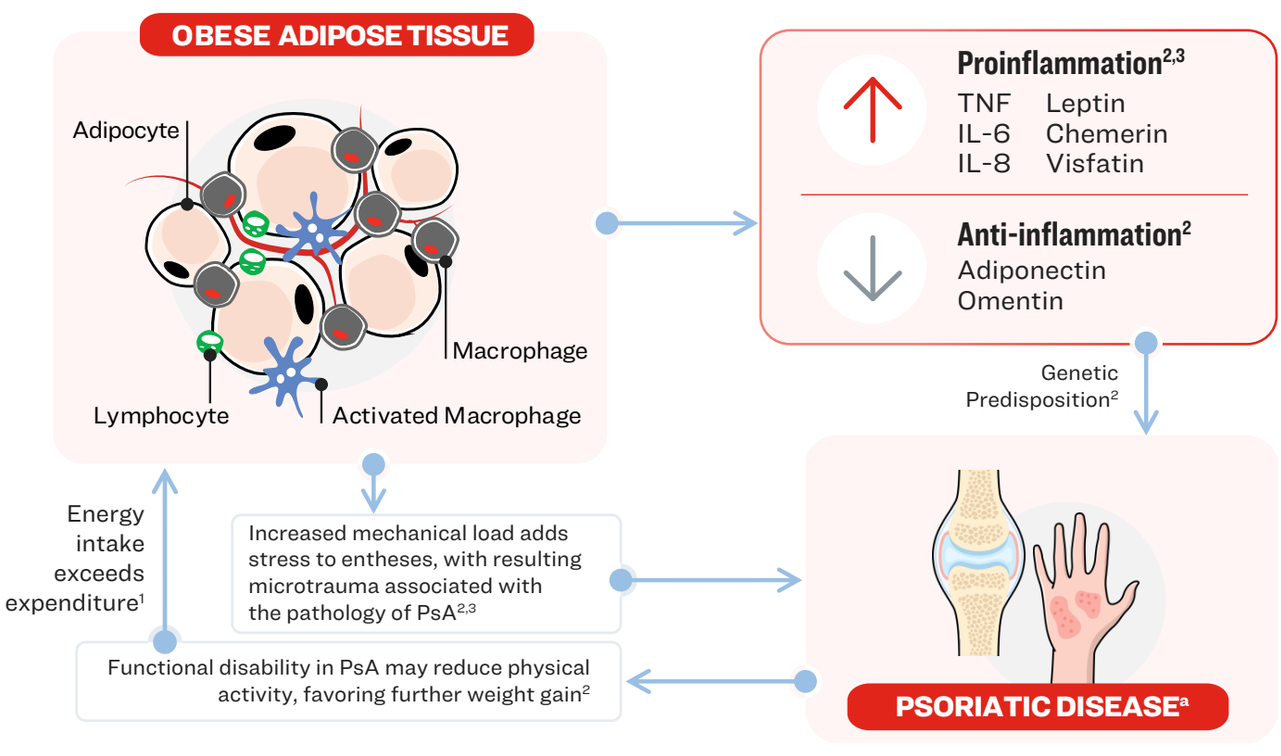
Obesity and PsA Have Mutually Reinforcing Mechanisms

Obesity results from an excessive expansion of white adipose tissue[1,2]

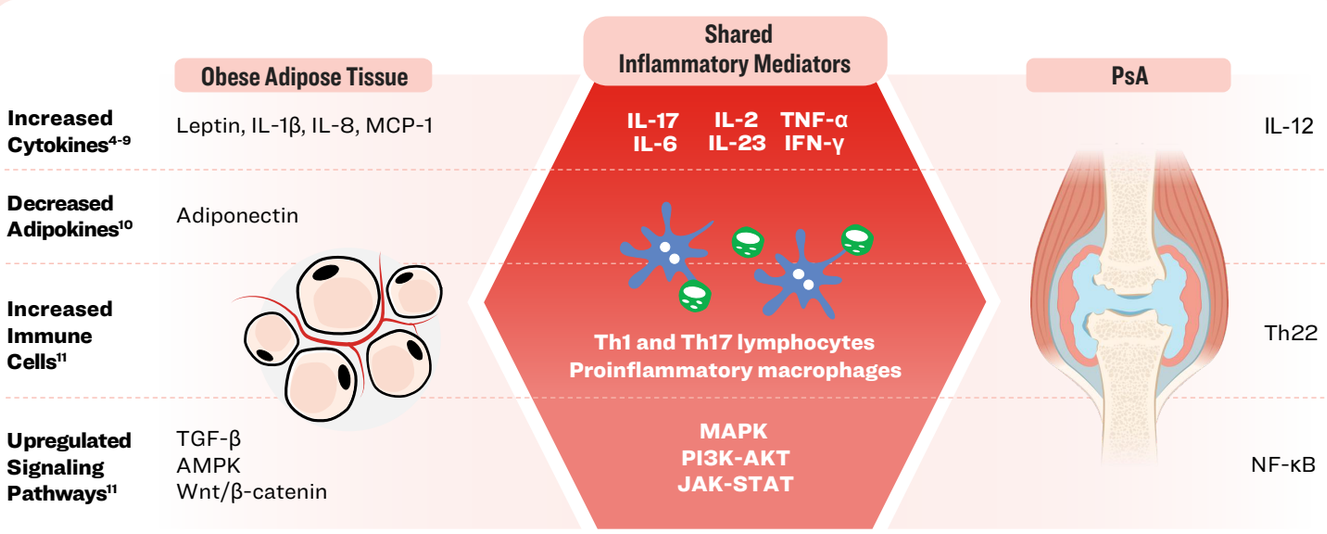
- Hypertrophy of existing adipocytes
- Increased infiltration of proinflammatory cells
- Decrease in the anti-inflammatory cell population

The dysfunctional adipose tissue acts as an endocrine organ[2,3]

- Increasing the secretion of adipokines and proinflammatory mediators



Obesity Activates Immune Pathways Relevant to PsA Pathogenesis



[a]Proposed mechanisms are based on current evidence and remain hypothetical; further research is needed to fully elucidate causal relationships.

AKT=protein kinase B; AMPK=AMP-activated protein kinase; IFN-γ=interferon gamma; IL=interleukin; JAK-STAT=janus kinase-signal transducer and activator of transcription; MAPK=mitogen-activated protein kinase; MCP-1=monocyte chemoattractant protein-1; NF-κB=nuclear factor kappa B; PI3K=phosphatidylinositol 3-kinase; PsA=psoriatic arthritis; TGF-β=transforming growth factor-β; Th1=T helper 1; Th17=T helper 17; TNF-α=tumor necrosis factor alpha; TNF=tumor necrosis factor.

1. Guo Z, et al. *JID Innov.* 2022;2(1):100064. 2. Porta S, et al. *Front Immunol.* 2021;11:590749. 3. Kumthekar A, Ogdie A. *Rheumatol Ther.* 2020;7(3):447-456. 4. Inouye KE, et al. *Diabetes.* 2007;56(9):2242-2250. 5. Bruun JM, et al. *J Clin Endocrinol Metab.* 2001;86(3):1267-1273. 6. Straczkowski M, et al. *J Clin Endocrinol Metab.* 2022; 87(10):4602-4606. 7. Thrum S, et al. *Int J Obes (Lond).* 2022;46(10):1883-1891. 8. Picó C, et al. *Rev Endocr Metab Disord.* 2022;23(1):13-30. 9. Considine RV, et al. *N Engl J Med.* 1996;334(5)292-295. 10. Goropashnaya AV, et al. *Metabolism.* 2009;58(1):22-29. 11. Williams JC, et al. *Ther Adv Musculoskelet Dis.* 2024;16:1759720X241271886.