incidence of arthritis, more severe clinical symptoms, and more pronounced joint inflammation and bone damage. NKp46 deficiency had no significant influence on the incidence and severity of arthritis in collagen-induced arthritis mice.

**Conclusion:** This study examined the proportion of NKp46+ ILC3-like cells in the peripheral blood, spleen, lymph nodes, and paws in CIA mice and their correlation with disease severity. We confirmed that infiltration of NKp46+ ILC3-like cells in CIA joints positively correlates with arthritis progression, inflammation, cartilage erosion, and bone destruction. Most importantly, we revealed the pathogenic role of NKp46+ ILC3-like cells in rheumatoid arthritis through adoptive cell transfer, which prominently exacerbates CIA arthritis. NKp46 may not be the primary actor in the pathogenic function of NKp46+ ILC3-like cells in CIA. Overall, our current work suggests that NKp46+ ILC3-like cells infiltrate in inflamed joints and participate in the pathogenesis of autoimmune arthritis.

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