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DELETION OF EITHER CD55 OR CD97 AMELIORATES ARTHRITIS

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CD55 (decay accelerating factor) is best known for its role in the negative regulation of the complement system. Indeed, lack of this molecule leads to disease aggravation in many inflammatory disease models. However, CD55 is abundantly present on fibroblast-like synoviocytes and is also a ligand of the adhesion class heptahelical receptor CD97, which is expressed by infiltrating macrophages. Treatment with antibodies to CD97 ameliorates the collagen-induced model of rheumatoid arthritis (RA) in DBA/one mice, but the net contribution of CD55 is unknown. We induced arthritis in C57BL/6J wild-type,

CD55, and CD97 mice using collagen-induced and K/BxN serum transfer models. Notably, lack of CD55, and to a lesser extent CD97, significantly mitigated collagen-induced arthritis (delayed onset and lower severity of disease). In the strongly complement-dependent K/BxN model, CD55 mice did not show aggravated disease, but instead showed some protection from disease development, although not as clear as seen in the CD97 animals. In conclusion, the lack of CD55 or CD97 in two different models of arthritis increased resistance to the disease. These findings provide insight into the complex role of CD55 in the pathogenesis of RA and are crucial for further development of CD55/CD97-directed therapeutic strategies.