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Osteopontin: a bridge between bone and the immune system

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Erratum

Original citation: J. Clin. Invest.112:147–149 (2003). doi:10.1172/JCI19190. Citation for this erratum: J. Clin. Invest.112:627 (2003). doi:10.1172/JCI19190E1. During the preparation of this manuscript for publication, errors were introduced into the text. The corrected paragraph appears below. We regret these errors. Arthritis in the setting of OPN deficiency Based on this information, one might expect that arthritis would be significantly attenuated in mice deficient in OPN. In fact this has been demonstrated in a CAIA model of RA (13) similar to that used in the study presented in this issue of the JCI (4). OPN-deficient mice were found to have marked attenuation of joint swelling and articular cartilage destruction compared with arthritic wild-type mice and had no increase in urinary levels of deoxypyridinoline, a marker of bone destruction (13). These data support a role for OPN in both the inflammatory and the joint-destructive processes in arthritis. Interestingly, however, these results were called into question in a recent report in Science (14) in which OPN was deleted by homologous recombination of strain 129-derived cells, and backcrossed into a CIA- and CAIA-susceptible strain for 12 generations. These authors then induced CIA and CAIA in the OPN-deficient mice and in littermates and demonstrated no effect of OPN deficiency in either form of murine arthritis. They concluded that prior observations in OPN-deficient mice showing protection from […]

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