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PYRIDINOLINE EXCRETION IS RELATED TO BONE LOSS AND CARTILAGE DAMAGE IN EARLY ACTIVE RHEUMATOID ARTHRITIS

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Pyridinoline cross-links are released during degradation of mature collagen and can be measured in the urine. Pyridinoline (PYD) results from degradation of several types of collagen in bone and cartilage, while deoxypyridinoline (DPD) is only released from breakdown of collagen type I from bone. Active rheumatoid arthritis is characterised by increased periarticular bone loss and joint damage, but the relative contribution of bone and cartilage collagen is not documented. PYD ands DPD were therefore measured in 155 patients with early active RA who participated in the COBRA study, a randomised comparison of active treatments (Boers M *et al*, Lancet 1997; 350: 309–18). Changes were related with disease activity, bone density and radiographic damage of the joints. At baseline, PYD and DPD were significantly correlated with ESR ($r = 0.485$ and $r = 0.422$, respectively), and bone density in the hip ($r = -0.140$ and $r = -0.209$) but not with bone density in the spine, HAQ or radiographic joint damage. During treatment the maximum decrease in PYD was −41%, and −37% for DPD. Changes in PYD and DPD were correlated with changes in bone density in the femoral neck ($r = -0.325$ and $r = -0.363$, respectively), with radiological progression ($r = 0.406$ and $r = 0.370$) and with changes in ESR ($r = -0.317$ and $r = -0.180$) but not with changes in HAQ. In a multiple regression model, radiographic progression and changes in bone density and ESR explained 43% of the variance of changes in PYD ($r = 0.657$) and 36% of the variance of changes in DPD ($r = 0.597$); addition of HAQ did not improve the model fit.

Conclusions: in early active RA, changes in PYD and DPD reflect both the combination of progression of joint damage and changes in bone resorption associated with inflammation.