the knee joint of 11 patients with RA were on average 3.7-fold higher ($p < 0.001$) than in those of 11 subjects with knee osteoarthritis and correlated with paired serum values ($r = 0.65$, $p = 0.039$). This suggests that circulating IIINys levels reflect in part local joint synovial tissue contribution.

Using a specific antibody, we showed that the IIINys sequence of type III collagen is highly nitrated in the synovium of patients with RA, in support of the growing body of evidence underscoring a central role for free radicals and NO in chronic inflammation.^{9–10} Markedly elevated serum IIINys values were detected in patients with active RA, suggesting that this biochemical marker may be useful for the clinical investigation of oxidative-mediated joint damage and for assessing effects of NO-targeted therapies. Further studies are required to determine whether serum IIINys reflects specifically synovial tissue inflammation and/or more general NO-mediated processes.

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Correction

There was an omission in an article published in the December 2008 Supplement (Furst DE, Keystone EC, Kirkham B, Fleischmann R, Mease P, Breedveld FC, *et al*. Updated consensus statement on biological agents for the treatment of rheumatic diseases, 2008. *Ann Rheum Dis* 2008;**67**(Suppl3):iii2–iii25). A Kavanaugh should have been added to the consensus statement as the fourth author. The statement should have read as follows: “AK conducts clinical research studies sponsored by Abbott, Amgen, BMS, Genentech, Biogen-Idec, UCB and Centocor.”

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