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Correction: *Identification of a novel chemokine-dependent molecular mechanism underlying rheumatoid arthritis-associated autoantibody-mediated bone loss*

Krishnamurthy A, Joshua V, Haj Hensvold A, *et al*. Identification of a novel chemokine-dependent molecular mechanism underlying rheumatoid arthritis-associated autoantibody-mediated bone loss. *Ann of Rheum Dis* 2016;75:721–9. doi:10.1136/annrheumdis-2015-208093.

The specificity of the human monoclonal antibodies B02 and D10 used in functional experiments in this article, originally described as high affinity ACPAs has been re-evaluated. In accordance with data from others that were made available to us in the past, the two monoclonal antibodies used lack specific binding to citrullinated peptides in surface plasmon resonance (SPR) and other assays as described in the retraction note to Journal of Experimental Medicine (Amara et al Retraction J. Exp Med 2019; 216:245). As such the functional results reported for these monoclonal antibodies cannot be attributed to reactivity against citrullinated proteins and/or peptides, but are due to other yet unknown mechanisms. Thus, the pathogenetic implications derived from these experiments cannot be upheld as stated.

In light of the lack of specificity of these monoclonal antibodies, the functional results observed in Figures 1 B-E (effects of monoclonal antibodies on osteoclast formation and bone loss); Figure 2 A-B (Osteoclast stainings using monoclonal antibodies); and Figure 5 (effect of the monoclonal antibodies on bone density in mice) should not be attributed to reactivity against citrullinated proteins and/or peptides, but must have been due to other, hitherto unknown mechanisms.

Since the monoclonal antibodies were used to confirm and expand the data obtained with polyclonal antibody preparations, the remaining conclusions in the paper rely on the data from the polyclonal IgG antibodies purified by affinity chromatography on CCP2-linked Sepharose columns. Although all effects on osteoclast activation were seen for the CCP2-column eluate and not in the flow through fractions and these effects could be blocked by both PAD inhibitors and blockade of IL-8, also these results have to be interpreted with caution waiting for additional mechanistic studies. Thus, the pathogenetic implications provided in Figure 6 are still hypothetical and rely on data from the polyclonal preparations, in light of the lack of ACPA specificity of the monoclonal antibodies used.

The authors would like to correct the conclusion worded "We provide novel insights into the key role of citrullination and PAD enzymes during OC differentiation and ACPA-induced OC activation. Our findings suggest that IL8-dependent OC activation may constitute an early event in the initiation of the joint specific inflammation in ACPA-positive RA." To be corrected as follows: "While ACPA may induce OC activation, the conclusions concerning the specificity of these observations require additional experiments before detailed mechanisms can be elucidated. Further, it is also not yet clear if ACPA are pathogenetically involved in the initiation of the joint specific inflammation in ACPA-positive RA or not." As a note of clarification, the polyclonal antibody fractions did not contain LPS contaminations according to the limulous amoebocyte lysate (LAL) assay.

We specifically apologise for the delays from our side in communicating the information in this correction note to the readership of Annals of the Rheumatic Diseases.

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