# Comment on editorial 'Pathogenic effector functions of ACPA: where do we stand?'

The recently published paper by Ge *et al* [1] has led to both retractions[2] and correction notes of papers[3 4] that have so far been a basis of the current understanding of anti-citrullinated protein antibodies (ACPAs). The recent and well-written editorial by Toes and Pisetsky[5] confirms the importance of this twist.

However, there is one statement in the editorial that needs to be clarified: 'Notably, the authors who described the monoclonal ACPA with osteoclastogenic and pain-inducing potential notified the community that, in reassessing the original data, the antibodies in question showed 'no measurable affinity for the tested citrullinated peptides' as detailed in a recent correction and retraction note; in other words, the monoclonal antibodies studied were not ACPA. This observation is in line with crystallographic studies of some of these monoclonal antibodies that could not confirm the binding of citrullinated peptides'. The crystal structures and the discovery that the monoclonal antibodies D10 and B02 did not have any citrulline specificity were already made in 2013, just after the now retracted Amara *et al*[2] paper was published, and this information was at this time communicated to the authors both through discussions and through written notice. To our disappointment, they decided not to retract the paper. Instead, they used these antibodies to prove functions of ACPAs in new papers.

This was also the reason that, several years later, we decided to include the old data of the B02 and D10 antibodies in our paper, showing for the first time the structural interactions between ACPA and citrullinated peptides.[1] It was only after this paper was made public, in August 2018, that the Amara *et al* paper was finally retracted.

It should also be noted that two papers based on these two previous 'ACPA' monoclonal B02 and D10 antibodies have been corrected, stating that it is not possible to conclude on the given data that ACPA specifically induces bone erosions. However, the authors continue to refer to these papers, iterating this statement as a proof. It is obviously critical for the future of rheumatology that published data are correct, and if this turns out not to be the case, it should be corrected as soon as proper information is available.

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