How to communicate in science

We have read the editorial from Toes and Pisetsky1 and the letter from Holmdahl2 concerning the role of ACPA in the pathogenesis of RA.

With regards to the editorial, we appreciate the comments on how methodological hurdles may impact the interpretation of functional data. We like to add to this discussion that we have recently published data using new extensively characterised monoclonal ACPAs3-4 and new batches of polyclonal ACPAs5 that support a role of ACPAs in osteoclast activation and pain generation as was suggested from experiments with polyclonal ACPA in the two 2016 ARD papers.6,7 We have recently also published an updated protocol on the generation and testing of monoclonal ACPAs.8 We want to emphasise, however, that much work from many groups will be needed before we get a full understanding of the role of ACPAs and other antibodies from RA patients in bone erosion, pain and synovitis.

Concerning the letter from Holmdahl,2 we need to acknowledge that a personal conflict has made the communication with Holmdahl deficient and inconsistent. We have different opinions about what has been communicated, and allegations that we should have consciously concealed data from the scientific community are unwarranted. Our re-evaluation of our monoclonal antibodies was a gradual process which occurred after the publication of the two discussed ARD papers,6,7 notably with Holmdahl as a co-author of one of them.7

In order to conclude this discussion, scientific progress is achieved by the generation of new data but also by a re-evaluation of existing data. Whereas the most common way of reporting such re-interpretations is by dissemination in new original articles, we acknowledge that increasing use of other routes, such as correction notes9,10 and open archives, are needed to avoid delays in communication as discussed in the editorial by Toes and Pisetsky.1

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