Response to: 'Detection of myositis-specific antibodies' by Vulsteke *et al*

It is with great interest we read the letter titled 'Detection of myositis-specific antibodies' by Dr Vulsteke et al¹ published in the Annals of the Rheumatic Diseases. The authors analysed the presence of myositis-specific autoantibodies in patients with idiopathic inflammatory myopathies (IIM) and in controls consisting of patients with other inflammatory conditions and blood donors. These analyses were performed using three different assays. In addition, the authors studied the association of autoantibody positivity from the different assays with specific clinical phenotypes in patients with IIM. In conclusion differences in specificities between assay manufacturers and between individual antibodies were found. The authors point to the fact that autoantibody data, namely anti-Jo-1 autoantibody positivity, were included in the 2017 European League Against Rheumatism/American College of Rheumatology classification criteria for adult and juvenile IIM and their major subgroups,² and that users should be aware of the characteristics of the autoantibody assays used in clinical settings. The authors also emphasise the need for initiatives to harmonise assays across manufacturers, and we welcome their contribution in this field.

In the original publication² we acknowledged the limitation of the low frequency of autoantibody data recorded in the data set, due to not yet identified antibodies and lack of available antibody assays at the start of the study. Our study required that antibody tests were performed in serum using standardised and validated tests. There were no requirements on the format of the assays, but in a survey performed among the study participants we found that ELISA was the most often used assay for anti-Jo-1 antibody detection (44%), followed by line blot (26%) and immunoprecipitation (15%). One important consideration for the classification criteria work was to include all medical disciplines involved in myositis research and care, as well as to obtain a broad geographical coverage of participating clinics. A stricter requirement of a specific antibody assay would not have been applicable to clinical practice and would have limited the available data even further. Although classification criteria should have high specificity, they also need high sensitivity, as well as be clinically applicable and practical.

We encourage a future revision of the new classification criteria with inclusion of more autoantibody data. Our commitment to this is reflected by the large international interdisciplinary collaboration, the Global Myositis Network MyoNet, which we have initiated. The network includes clinicians and researchers with interest in myositis, and one important topic on the research agenda is the standardisation and harmonisation of sample collection and analyses, including autoantibody assays. A global longitudinal registry for data on patients with myositis, the EuroMyositis database, is accompanying the network including more than 20 centres worldwide. At present,

more than 4500 patients are registered. Large emphasis has been placed on systematic collection of autoantibody data, using validated and standardised procedures. We believe that these data will be of great value for a future revision of the classification criteria.

We thank Vulsteke and colleagues for their important contribution to the research of myositis-specific antibodies and for increasing the knowledge and awareness of the importance of appropriate assays and interpretation of results. The results from their cohort underline the possibility of different performance between different assays, which emphasise the need for further validation studies of commercially available assays using large cohorts including patients with a broad spectrum of clinical phenotypes and including the 'golden standard' assay immunoprecipitation for comparison. Such a study is ongoing within the MyoNet and EuroMyositis collaboration, and will be important for future updates of classification criteria for IIM.

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REFERENCES

- 1 Vulsteke J-B, De Langhe E, Claeys KG, et al. Detection of myositis-specific antibodies. Eletter. Ann Rheum Dis 2019;78:e7.
- 2 Lundberg IE, Tjärnlund A, Bottai M, et al. 2017 European League Against Rheumatism/ American College of Rheumatology classification criteria for adult and juvenile idiopathic inflammatory myopathies and their major subgroups. Ann Rheum Dis 2017;76:1955–64.

