Reply

We appreciate the interesting comments on our paper by Rozman and Kveder. As stated previously, our study was retrospective and based on hospital records. It was therefore not possible to focus on recent refinements and limitations of autoantibody characterization in systemic sclerosis (SSc). To the best of our knowledge, there have been no studies reported on autoantibodies to Ku, PM/ScI or RNA polymerases in South African patients. Nevertheless, this should not detract from the main thrust of our communication, that of highlighting inter-ethnic differences with respect to certain clinical features and autoantibodies. These observations are consistent with a previous report from Michigan by Laing et al. [1], which we alluded to in the paper. In the case of myositis, using similar criteria to those that we applied, they found the frequency of myositis to be significantly higher in Black Americans compared with White Americans (20.5 vs 12.1%, respectively, \( P = 0.02 \)). This supports a general trend towards ethnic differences with respect to muscle involvement.

The same applies to the occurrence of certain autoantibodies. Anti-RNP antibodies in the Michigan study were more prevalent in Blacks compared with Whites (22.4 vs 12.7%, respectively, \( P = 0.05 \)). It is noteworthy that similar inter-ethnic differences of anti-RNP antibody frequencies have been observed in systemic lupus erythematosus (SLE) [2, 3]. However, in contrast to observations in Whites, anti-ribonucleoprotein antibodies in our experience are rarely associated with overlap features [3]. Furthermore, Pudifin et al. [4], in a study focusing on antinuclear antibodies (ANA) in SSc patients from three ethnic groups in South Africa, found anti-centromere antibodies only in Whites and Asians but not in Blacks.

Rozman and Kveder point out problems of quality control in autoantibody tests, which have also been more recently addressed by the ANA subcommittee of the International Union of Immunological Societies’ Standardisation Committee [5]. More specifically, Rozman and Kveder express doubts regarding our findings of anti-Sm antibodies in two patients. Clearly it is not warranted to make any extrapolations from such small numbers. As we point out in the paper, there is need for larger prospective studies of SSc in South African patients, which should include more detailed autoantibody characterization. In this respect, we are in full agreement with Rozman and Kveder. Finally, studies of inter-ethnic differences could indeed provide better insight into the aetiopathogenesis of this complex disease.

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