a higher prevalence of anti-Sm and a lower prevalence of anti-dsDNA, considerably shorter disease duration and more damage in Sudanese patients with SLE. Just as authors described in this paper, their findings 'will hopefully add to the general understanding of SLE in Sudan and demonstrate the importance of investigating this heterogeneous disease in larger cohorts and longitudinal designs from different parts of Africa'. We would like, however, to state that there are two confusions in this study that need to be explained more comprehensively.

Firstly, some laboratory data including proteinuria, anaemia, leucopenia, lymphopenia, thrombocytopenia, LE cell, anti-DNA, anti-Sm and ANA are required for diagnosis of SLE according to the 1982 revised ACR classification criteria [2]. It is obvious that the data for two cohorts of this study were obtained from different laboratories. How to ensure the comparability of the data between laboratories? If the comparability of laboratory data is uncertain, the comparability of SLE diagnosis may be questionable between the two cohorts, which can influence and even bias the results and conclusions to some extent. As a matter of fact, when evaluating the SLEDAI, to obtain the comparable data, authors modified SLEDAI by excluding DNA binding and complement components and urinary items. As such, the study results are more accurate and objective. Therefore, authors could know that it is also very important to ensure the comparability of data associated with SLE diagnosis between the two cohorts.

Secondly, what is the purpose of setting up 106 and 318 age- and sex-matched controls from Sudan and Sweden, respectively? Because the study results did not show data about the controls, and furthermore, there was no comparability between the two controls (healthy individuals for Sudanese controls and non-SLE individuals for Swedish controls), we feel that there may be no need to set up the controls.

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Zaixing Yang[1] and Yan Liang[2]

[1]*Department of Laboratory Medicine, Huangyan Hospital of Wenzhou Medical University, Taizhou First People's Hospital, Zhejiang and* [2]*Department of Laboratory Diagnostics, Changzheng Hospital, Second Military Medical University, Shanghai, China*

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Correspondence to: Zaixing Yang, Department of Laboratory Medicine, Taizhou First People's Hospital, 218 Hengjie Road, Huangyan District, Taizhou, Zhejiang, China.
E-mail: yangzaixingdiyi@163.com

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Comment on: Sudanese and Swedish patients with systemic lupus erythematosus, immunological and clinical comparisons: reply

Dear Editor, We have with interest read the response from Zaixing Yang and Yan Liang [1] to our recently published comparative paper on SLE in Sudan and Sweden [2]. The authors stress the fact that differences in how laboratory analyses are performed in different countries may result in a difference in which patients are actually included from the regions to be compared. We agree that this is a tentative problem with any study comparing SLE cohorts defined by classification criteria including laboratory measures, which is the case both for the 1982 criteria used in our study [3] and the recently published EULAR/ACR criteria [4]. This problem is especially prominent when using retrospective data. We also want to clarify that both the compared cohorts were incipient cohorts, and that when the Sudanese at an earlier time point were classified as SLE, the laboratory analyses were performed at the central laboratories serving the hospitals where the rheumatology clinics resided. Laboratory measures evaluated at the outpatient visit when the patients were included in the present study were, however, obtained from different hospital-based and private laboratories, making disease activity comparison based on such diverse data more difficult.

Drs Yang and Liang argue that we should have used the laboratory data obtained from each country instead of autoantibody analyses performed in Uppsala when comparing SLE patients from Sudan and Sweden, and that such an approach would yield information about the comparability of autoantibody laboratory data between the countries. We do not agree with this idea. Our main objective was to compare the patients living in Sudan and Sweden, and not to compare laboratory procedures. We think that a proper immunological comparison between patients living in different geographical areas relies on the use of identical laboratory analyses for both patient groups.

The Swedish population controls were chosen to individually match the Swedish SLE patients for age, sex and residential area, the only exclusion criterium being SLE. The authors are correct in that there is a difference between the Sudanese healthy controls and the Swedish population controls used to make national alignment of reference values. For that reason, we did not emphasize the difference between the Sudanese and Swedish control groups in our paper. Both this and our previous

papers on rheumatoid arthritis in Sudan [5, 6] and Malaysia [7] have shown that such national alignment of reference values and 'cutoffs' increases the value of autoantibody analyses.

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Sahwa Elbagir [1], Iva Gunnarsson[2] and Johan Rönnelid[1]

[1]*Department of Immunology, Genetics and Pathology, Uppsala University, Uppsala and* [2]*Division of Rheumatology, Department of Medicine Solna, Karolinska Institutet, Karolinska University Hospital, Stockholm, Sweden*
Accepted 7 September 2019
Correspondence to: Sahwa Elbagir, Department of Immunology, Genetics and Pathology, Uppsala University, Rudbeck Laboratory C5, 3rd floor, 751 85 Uppsala, Sweden.
E-mail: sahwa.elbagir@igp.uu.se

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Comment on: Lower urinary tract symptoms in systemic sclerosis: a detailed investigation

Dear Sir, We read with interest the article by Pacini *et al.* [1], which highlights a higher prevalence and severity of urinary incontinence (UI) in SSc patients than in healthy matched controls. We congratulate the authors for their findings, suggesting that SSc can represent an independent predictor for UI.

UI has a substantial negative impact on health-related quality of life and it is well known that urinary tract involvement is frequently associated with other unrelated diseases. Patients affected by pulmonary diseases are at risk for UI because of chronic cough [2, 3]. During cough, the intra-abdominal pressure increases and leads to involuntary urinary loss in two-thirds of women with chronic cough [4]. Even in SSc, cough is very common: in the Scleroderma Lung Study it was observed in 73% of patients [5].

UI predictors in SSc are not well defined. Sex and BMI seem to play a role in UI occurrence, but it is controversial which antibody profile is associated with UI. While Pacini *et al.* identified anti-Scl70, another multicentre study points to the anti-centromere [6]. This discrepancy may derive from a bias.

We undertook the same study as Pacini *et al.*, adding the quantification of cough using the Leicester Cough Questionnaire (LCQ) [7]. The Incontinence Questionnaire (ICIQ) has previously been used to assess UI presence and severity. Demographic, clinical and laboratory data were collected for consecutive SSc patients (according to ACR/EULAR classification criteria [8]), admitted to the Day Hospital of Rheumatology. Spearman rank test assessed the correlation between ICIQ and LCQ. The parameters associated with UI presence and severity were assessed respectively with logistic and multiple linear regression tests. $P < 0.05$ was considered statistically significant.

We enrolled 49 patients: female prevalence was 94% and median age was 68 years (95% CI 56, 71). The median disease duration was 7 years (95% CI 5, 14). ICIQ has a mild strength of correlation with LCQ (rho = −0.37; $P = 0.009$). Both logistic and multilinear regression tests showed a correlation between LCQ and ICIQ presence (odds ratio 0.95; 95% CI 0.92, 0.99; $P = 0.01$) and severity (β coefficient = 0.09; $R^2 = 25\%$ $P = 0.0009$). If LCQ is not considered in the multiple linear regression, BMI emerge as associated with ICIQ (b coefficient = 0, 29; $R^2 = 12, 7\%$; $P = 0.02$).

These results should be interpreted carefully as the study is observational and on a limited number of SSc patients. Moreover, the extremely high female prevalence could have excluded sex from UI predictors. However, cough appears to play, even in SSc, a relevant role in UI onset. As the authors aim to better understand this issue in future studies, we suggest taking cough into account.