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Clinical value of simultaneous detection of anti-dsDNA and anti-chromatin antibodies using a multiplex immunoassay in systemic lupus erythematosus: a 5-year retrospective study

Thibault Sixt ,[1,2] Daniela Lakomy,[2,3] Adrien Guilloteau,[2,4] Maxime Samson,[1,2] Bernard Bonnotte,[1,2] Sylvain Audia[1,2]

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[1]Service de Médecine Interne et Immunologie Clinique, Centre de Référence des Maladies Auto-Immunes Rares de l'Adulte, CHU Dijon-Bourgogne, Dijon, France
[2]Université Bourgogne Europe, Dijon, France
[3]Laboratoire d'Immunologie, CHU Dijon-Bourgogne, Dijon, France
[4]UMR1231, Equipe Epi2THM, INSERM, Dijon, France

Correspondence to
Professor Sylvain Audia; sylvain.audia@chu-dijon.fr

ABSTRACT

Objective To assess the clinical relevance of concurrent measurement of anti-chromatin antibodies and anti-double-stranded DNA (anti-dsDNA) antibodies using a multiplex immunoassay in systemic lupus erythematosus (SLE), focusing on their association with organ-specific manifestations and disease activity.

Methods Adult patients diagnosed with SLE based on the 2012 Systemic Lupus International Collaborating Clinics or 2019 European Alliance of Associations for Rheumatology/American College of Rheumatology criteria and followed in our university hospital between 2015 and 2019 were retrospectively included if they had at least one positive detection of anti-dsDNA and/or anti-chromatin result by a multiplex immunoassay, with clinical data available within the 30 days before or after testing. Clinical manifestations, treatments, laboratory parameters and disease activity (SLE Disease Activity Index 2000 (SLEDAI-2K)) were compared using mixed models and correlation coefficients.

Results 98 patients (88% women; median age 42 (IQR: 35–50)) had 677 antibody evaluations. Levels of anti-dsDNA and anti-chromatin antibodies were correlated (r=0.39, 95% CI 0.21 to 0.55). Results were discordant in 59 patients (60%), representing 215 assessments (32%), with anti-chromatin antibodies being detected without anti-dsDNA in 111/677 samples (16%). Rise in anti-dsDNA antibody titre was associated with cutaneous (OR 2.29, 95% CI 1.43 to 3.68) and musculoskeletal involvement (OR 1.29, 95% CI 1.02 to 1.64), while anti-chromatin antibody increase was linked to neuropsychiatric manifestations (OR 1.54, 95% CI 1.03 to 2.29). The increase of either antibody titre was independently associated with proteinuria >0.5 g/24 hours (anti-dsDNA antibody: OR 1.17, 95% CI 1.11 to 2.74; anti-chromatin antibody: OR 1.71, 95% CI 1.02 to 2.87). Correlation with SLEDAI-2K was moderate for anti-dsDNA (r=0.55; 95% CI 0.39 to 0.67), and weaker for anti-chromatin (r=0.32; 95% CI 0.16 to 0.51]).

Conclusions Concomitant measurement of anti-dsDNA and anti-chromatin antibodies using a multiplex immunoassay system brings complementary information for diagnosis and monitoring of SLE. Both antibodies are associated with disease activity and renal involvement, while anti-chromatin antibodies are associated with neuropsychiatric flares. Anti-chromatin antibodies provide additional diagnostic value, particularly in the absence of anti-dsDNA positivity that was observed in 16% of our cohort.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Anti-double-stranded DNA (anti-dsDNA) antibodies are well-established markers of disease activity and organ involvement in systemic lupus erythematosus (SLE), although their detection varies by assay.
⇒ In contrast, the clinical relevance of anti-chromatin antibodies measured concomitantly using multiplex immunoassays remains underexplored.

WHAT THIS STUDY ADDS

⇒ This study provides assessment of 677 paired clinical and serological examinations and shows that increase in anti-dsDNA or anti-chromatin antibodies measured using a multiplex immunoassay is associated with different organ involvement and is complementary for disease activity evaluation.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Simultaneous detection of both antibodies through multiplex immunoassay may improve the management of patients with SLE.
⇒ These findings encourage broader integration of multiplex antibody profiling into clinical practice and biomarker-driven research.

INTRODUCTION

Anti-double stranded DNA (anti-dsDNA) antibodies are highly specific markers for systemic lupus erythematosus (SLE) included in the 2019 European Alliance of Associations for Rheumatology/American

College of Rheumatology (EULAR/ACR)[1] and the 2012 Systemic Lupus International Collaborating Clinics (SLICC)[2] classification criteria. They are linked to disease activity[3] and lupus nephritis,[4] although results vary partly due to differences in detection methods.[5] Anti-chromatin (anti-nucleosome) antibodies target DNA-histone complexes, key in lupus pathogenesis. While some studies associate them with lupus nephritis,[6] this has been debated based on meta-analyses.[7] Their relationship with disease activity has been less explored.[8]

Multiplex bead-based immunoassays enable simultaneous detection of multiple autoantibodies, including anti-dsDNA and anti-chromatin. Despite its established role in SLE diagnosis,[7] the clinical relevance and relationship with disease activity of anti-chromatin antibodies measured by multiplex immunoassays remain poorly studied in SLE cohorts.[9]

Our aim was to assess the utility of concomitant testing of anti-chromatin and anti-dsDNA antibodies measured by such an assay in the monitoring of patients with SLE, focusing on their associations with disease activity and organ involvement.

METHODS

This retrospective, single-centre study was conducted in the Internal Medicine Department of Dijon University Hospital, France, and included patients managed between January 2015 and December 2019. Adult patients (≥18 years) were eligible if they had at least one positive anti-dsDNA and/or anti-chromatin antibody measurement obtained using the multiplex immunoassay system. A diagnosis of SLE fulfilling the 2012 SLICC or 2019 EULAR/ACR classification criteria[1 2] was required. The SLE diagnosis had to be established either before the study period or confirmed during the clinical follow-up associated with the antibody measurement. The patients included were either newly diagnosed patients or ones already followed regularly in our centre.

Each antibody measurement (performed between January 2015 and December 2019) required a clinical evaluation in the month preceding or following the date of sampling. Given this period of 30 days, clinical visits were conducted between December 2014 and January 2020.

Demographic, clinical (fever, cutaneous lupus, musculoskeletal symptoms, serositis, neuropsychiatric (psychosis,

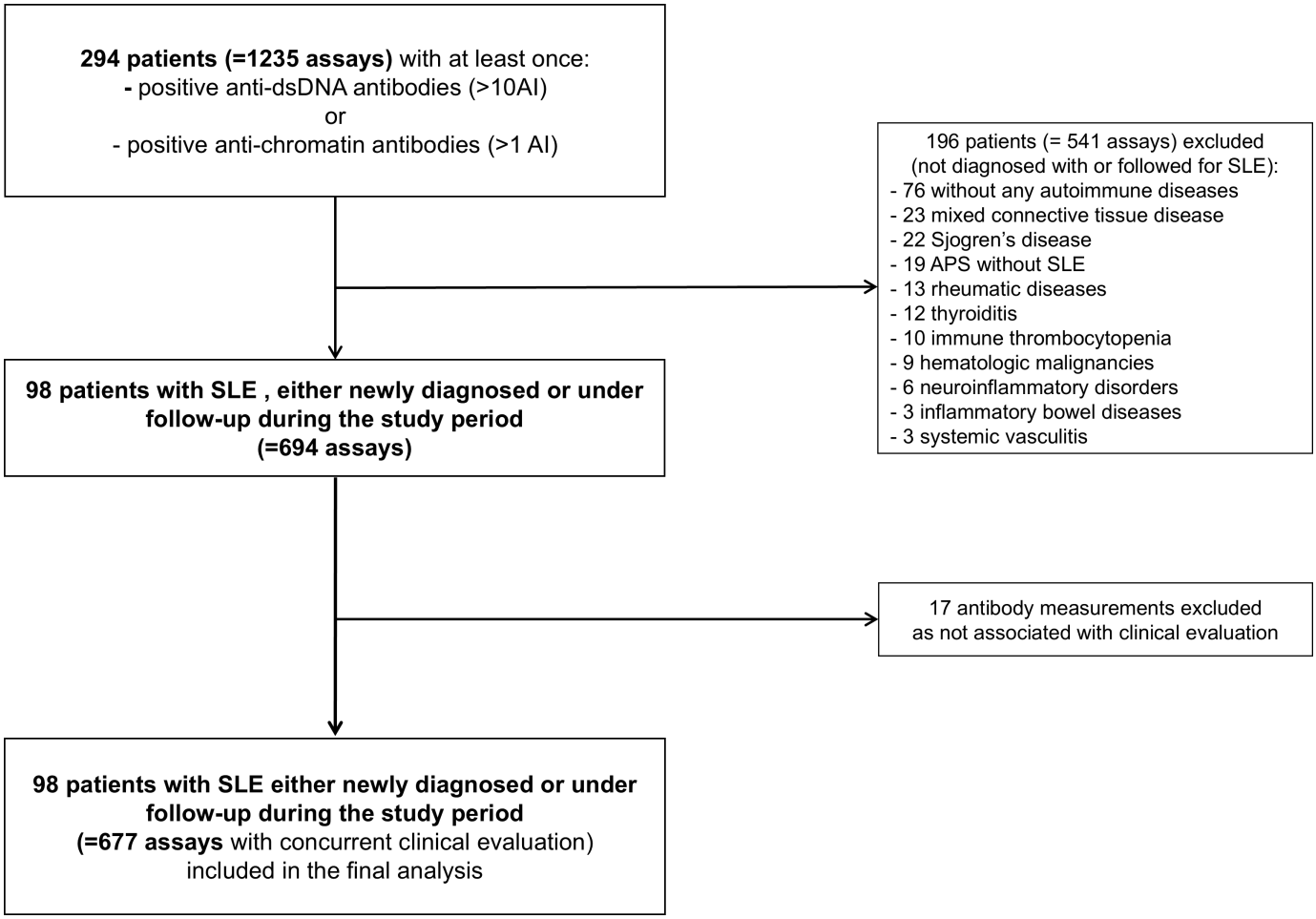

Figure 1 Flow chart of the study. AI, Antibody Index; anti-dsDNA, anti-double-stranded DNA; APS, antiphospholipid syndrome; SLE, systemic lupus erythematosus.

Table 1 Baseline clinical and biological characteristics of the SLE cohort

Follow-up characteristics, median (IQR)	**Overall patients n=98**
Age (years), median (IQR)	42 (35–50)
Number of assessments (clinical visit and blood sampling), median (IQR)	4 (3–9)
Minimal delay between two assessments (days), median (IQR)	88 (27–241)
Mean number of assessments per year, per patient, median (IQR)	2.3 (1.4–3.6)
Duration of follow-up (years), median (IQR)	3.5 (1.9–4.2)
Clinical manifestation during the follow-up period, n (%)*	**Overall patients n=98**
Musculoskeletal manifestations	68 (69.4)
Cutaneous manifestations	42 (42.9)
Neuropsychiatric manifestations	20 (20.4)
Antiphospholipid syndrome	15 (15.3)
Visual manifestations	12 (12.2)
Fever	11 (11.2)
Vasculitis	8 (8.2)
Serositis	6 (6.1)
Haematuria	65 (66.3)
Leucocyturia	38 (38.8)
Proteinuria >0.5 g/24 hours	20 (20.4)
Class III or IV lupus nephritis	17 (17.3)
Class II lupus nephritis	3 (3.1)
Class V lupus nephritis	4 (4.1)
Treatment exposure during the study period, n (%)†	**Overall patients n=98**
Hydroxychloroquine	80 (81.6)
Corticosteroids	43 (43.9)
Methotrexate	16 (16.3)
Mycophenolate mofetil	13 (13.3)
Azathioprine	12 (12.2)
Belimumab	9 (9.2)
Cyclophosphamide	5 (5.1)
Thalidomide	4 (4.1)
Mean SLEDAI-2K, per patient, median (IQR)	4.9 (2.5–8.0)
Mean anti-dsDNA antibodies (IU/mL), per patient, median (IQR)	9.5 (3.5–22.3)
Mean anti-chromatin antibodies (AI), per patient, median (IQR)	1.2 (0.3–2.8)
Mean haemoglobin (g/L), per patient, median (IQR)	131 (121–138)
Mean leucocyte count (×10^9/L), per patient, median (IQR)	6.0 (4.7–7.9)
Mean platelet count (×10^9/L), per patient, median (IQR)	233 (200–274)
Mean C3 (mg/L), per patient, median (IQR)	0.9 (0.8–1.1)
Mean C4 (mg/L), per patient, median (IQR)	0.2 (0.1–0.2)
Positive antibodies anti-chromatin antibodies, n (%)	Overall immunological testing n=667
Anti-chromatin antibodies (>1 AI)	339 (50)
Anti-dsDNA antibodies (>10 IU/mL)	343 (51)
Treatment at sampling, n (%)	Overall immunological testing n=667
Hydroxychloroquine	473 (70)
Corticosteroids	358 (53)
Methotrexate	104 (15)
Belimumab	103 (15)
Mycophenolate mofetil	90 (13)
Azathioprine	62 (9)
Cyclophosphamide	23 (3)
Thalidomide	24 (4)

*Presence of at least one episode per patient recorded during the study period.
†Treatments with at least one exposure during the follow-up period
AI, Antibody Index; anti-dsDNA, anti-double-stranded DNA; APS, Antiphospholipid syndrome; SLEDAI-2K, SLE Disease Activity Index 2000.

defined as a severe disturbance of perception of reality including hallucinations, incoherence, impoverished thought content, illogical reasoning or bizarre, disorganised or catatonic behaviour or delirium; headaches; seizures; strokes), ocular, vasculitis signs and treatments) and laboratory data (full blood count, direct antiglobulin test, complement (C3, C4) and proteinuria) were retrospectively collected from medical records for each visit. The presence of antiphospholipid antibodies/syndrome (Sydney criteria) was also collected.

At each visit, disease activity was measured using the SLE Disease Activity Index 2000 (SLEDAI-2K; range 0–105). To prevent overestimation due to anti-dsDNA positivity, a modified SLEDAI excluding anti-dsDNA points was also calculated. Changes in anti-dsDNA and anti-chromatin antibody levels were analysed in relation to disease activity over time.

Anti-dsDNA and anti-chromatin antibodies were measured simultaneously using a multiplex automated immunoassay system (BioPlex 2200, Bio-Rad Laboratories, Hercules, California, USA), routinely implemented in our centre since 2013. This platform allows simultaneous quantification of multiple autoantibodies without increasing turnaround time, so both antibodies are automatically assessed together. The positivity thresholds were ≥10 IU/mL for anti-dsDNA and >1 Antibody Index for anti-chromatin, according to the manufacturer's recommendations.

Because outcomes were repeatedly measured, patient-level mean values were calculated and summarised as median (first to third quartiles) for continuous variables, and categorical variables as number (percentage). Due to the log-normal distribution, correlations between mean anti-dsDNA and anti-chromatin antibody concentrations and disease activity (mean SLEDAI-2K, with and without anti-dsDNA) were assessed on log-transformed values using Pearson and Spearman correlations. Since the measurements were repeated for the same patient, repeated-measure correlation coefficients were also calculated to account for multiple measurements per patient.[10] Associations between organ-specific manifestations and biological parameters were analysed using mixed linear models (continuous outcomes) or mixed logistic models (binary outcomes) with random intercepts. To allow meaningful analyses, some SLEDAI-2K items were grouped into broader categories (cutaneous, neurologic, serositis), while other rare manifestations, including leucopenia and thrombocytopenia, were not analysed due to their absence or very low frequency. Analyses were performed with R software using the rmcorr package with $p<0.05$ considered significant.

This study was conducted in accordance with French regulations governing retrospective research. Ethical approval was not required as the study involved the analysis of anonymised routine care data without patient re-identification.

RESULTS

98 patients with SLE with at least one positive detection of anti-dsDNA or anti-chromatin antibody were included, representing 677 serological assessments with concurrent clinical evaluation (figure 1). The female/male ratio was 7.2 (with 86 women, 88%) and the median age was 42 (35–50) years (table 1). Common manifestations were musculoskeletal (n=68, 69.4%), cutaneous (n=42, 42.9%), neuropsychiatric (n=20, 20.4%, mostly headaches, 16/20) and renal involvement (proteinuria >0.5 g/day; n=20, 20.4%). At sampling, 69 patients (70%) were receiving hydroxychloroquine and 52 (53%) corticosteroids. The median of mean duration of follow-up per patient was 3.5 years (1.9–4.2), with a median number of mean annual assessments (clinical examination and blood tests) of 2.3 (1.4–3.6) per patient.

Anti-dsDNA and anti-chromatin antibody levels were moderately correlated (Pearson r=0.39 (0.21–0.55); repeated-measures r=0.51 (0.45–0.57)). Among the 677 assessments, 228 (44%) were concordant (both antibodies positive) and 226 (56%) discordant, involving 59 patients. Anti-chromatin without anti-dsDNA was seen in 111/677 (16%) assessments, and anti-dsDNA without anti-chromatin in 115/677 (15%). Among discordant results, 28 patients had at least one assessment positive only for anti-dsDNA during the study period (median of patient-level mean SLEDAI-2K without anti-dsDNA: 4.5 (3.5–5.4)), and 31 patients had at least one assessment positive only for anti-chromatin antibodies (median of patient-level mean SLEDAI-2K without anti-dsDNA: 4.1 (3.2–5.1)) (table 2).

As shown in table 3, a one log increase of anti-dsDNA levels was associated with increased cutaneous manifestations (OR: 2.29, 95% CI 1.43 to 3.68) and musculoskeletal symptoms (OR: 1.29, 95% CI 1.02 to 1.64). A one-log increase in anti-chromatin antibodies correlated with neuropsychiatric manifestations (OR: 1.54, 95% CI 1.03 to 2.29). Both antibodies were independently associated with proteinuria >0.5 g/day (OR: 1.17, 95% CI 1.11 to 2.74 for anti-dsDNA and OR: 1.71, 95% CI 1.02 to 2.87 for anti-chromatin antibodies).

Anti-dsDNA levels slightly correlated with disease activity (SLEDAI-2K): Pearson r=0.55 (0.39–0.67), repeated-measures r=0.25 (0.17–0.32). Anti-chromatin antibodies showed weaker correlations: Pearson r=0.32 (0.16–0.51), repeated-measures r=0.17 (0.09–0.25). Using a modified SLEDAI-2K (excluding anti-dsDNA points), both antibodies had similar correlations with disease activity (anti-dsDNA: Pearson r=0.30 (0.11–0.47), repeated-measures r=0.20 (0.12–0.27); anti-chromatin: Pearson r=0.30 (0.11–0.47), repeated-measures r=0.10 (0.02–0.18)). Correlations between antibody level changes and SLEDAI-2K changes were also weak (anti-dsDNA: r=0.19 (0.11–0.28); anti-chromatin: r=0.11 (0.02–0.20)).

DISCUSSION

This 5-year single-centre study of 98 patients with SLE, based on 677 paired serological and clinical assessments, showed frequent discordance between anti-dsDNA and anti-chromatin antibodies positivity, with detection of one antibody without the other in 30% of cases. This has

Table 2 Discordant results between anti-dsDNA and antichromatin antibodies

Anti-dsDNA	Anti-chromatin	Number of patients concerned during management	Number of immunological assessments corresponding, n (%)	Mean SLEDAI-2K, without anti-dsDNA, median (IQR)
+	+	43	228/677 (34)	9.1 (8.3–9.9)
+	–	28	115/677 (15)	4.5 (3.5–5.4)
–	+	31	111/677 (16)	4.1 (3.2–5.1)

anti-dsDNA, anti-double-stranded DNA; SLEDAI-2K, SLE Disease Activity Index 2000.

Table 3 Organ-specific manifestations associated with a one-log increase of antibody titres

	Log anti-dsDNA antibodies		Log anti-chromatin antibodies	
Organ damages	**OR**	95% **CI**	**OR**	95% **CI**
Musculoskeletal manifestations	1.29	1.02 to 1.64	1.16	0.91 to 1.48
Cutaneous manifestations	2.29	1.43 to 3.68	0.82	0.57 to 1.19
Neuropsychiatric manifestations	1.32	0.9 to 1.94	1.54	1.03 to 2.29
Proteinuria >0.5 g/24 hours	1.17	1.11 to 2.74	1.71	1.02 to 2.87
Antiphospholipid syndrome	0.72	0.09 to 5.8	0.79	0.09 to 7.08
Fever	1.5	0.71 to 3.19	1.02	0.49 to 2.13
Serositis	5.28	0.77 to 36.32	1.37	0.34 to 5.55
Haematuria	1.15	0.90 to 1.46	1.14	0.89 to 1.47
Low C3 (<0.8 g/L)	0.96	0.43 to 2.13	2.13	0.89 to 1.47

anti-dsDNA, anti-double-stranded DNA.

already been documented using ELISA[11] and the discordance observed with multiplex assays is consistent with previous studies reported in the literature.[9 11–13]

Increases in antibody titres were associated with the presence of organ-specific manifestations. Anti-dsDNA antibody increase correlated with skin and musculoskeletal manifestations, consistent with prior research.[14] Conversely, the rise of anti-chromatin antibodies was linked to neuropsychiatric manifestations. Contrasting with prior studies, we do not observe an association between anti-chromatin antibodies and haematological,[15] musculoskeletal, serositis[4] or skin manifestations.[16] These findings underscore the complementary value of simultaneously measuring both antibodies to better capture organ-specific involvement.

Both antibodies were independently associated with renal disease, particularly proteinuria >0.5 g/day, which reinforces their role in lupus nephritis. Similar associations with anti-dsDNA and anti-chromatin have also been reported using multiplex assays, supporting their robustness as non-invasive biomarkers of renal activity.[17]

Regarding disease activity, anti-dsDNA antibodies demonstrated a slight to moderate correlation with the SLEDAI-2K, whereas anti-chromatin antibodies showed a weaker association. However, when the anti-dsDNA component was excluded from the SLEDAI score, anti-chromatin antibody levels correlated with disease activity to a similar extent as anti-dsDNA antibodies. This finding is consistent with previous studies reporting similar associations.[9 11 15]

Importantly, a high rate of discordance (32% of biological assessment) between antibody results was observed, in accordance with prior studies,[9 11–13] underlining the importance of dual testing for comprehensive disease monitoring.

Also mentioned in a previous study,[8] fluctuations in antibody levels poorly correlated with changes in disease activity, suggesting that absolute titres may be more relevant than their variation over time.

Our study has several limitations, including its single-centre and retrospective nature, which may have influenced the reported frequencies of the different clinical manifestations, in the absence of standardised data recording. The limited number of patients has also precluded the analysis of certain clinical associations, such as ophthalmologic manifestations that could not have been analysed due to low events. However, a large number of immunological assessments were available, each coupled with clinical evaluations.

In conclusion, multiplex immunoassay technology offers a valuable tool for SLE monitoring through simultaneous detection of anti-dsDNA and anti-chromatin antibodies. While anti-dsDNA increase is linked to cutaneous, musculoskeletal and renal involvement, rise in anti-chromatin antibody titres is associated with neuropsychiatric manifestations. Their combined assessment enhances the disease activity and organ damage evaluation, especially in cases of discordant serology. This dual-marker approach provides a more comprehensive picture of SLE, supporting earlier detection of flares and more individualised patient management.

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Contributors TS and SA designed the study. TS collected data. TS and AG performed data checking. AG carried out the statistical analyses. TS and SA wrote the original draft. All authors participated in the proofreading and editing. SA and TS had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. GuarantorProf AUDIA Sylvian is the guarantor of the study and takes responsibility for the overall content of the manuscript.

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ORCID iD
Thibault Sixt https://orcid.org/0000-0002-2966-6346

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