IS THERE LONG-TERM VALUE OF PATHOLOGY SCORING IN IGA NEPHROPATHY? A VALIGA UPDATE.

Rosanna Coppo1, G D'Arrigo2, G Tripepi2, ML Russo1, I.S.D Roberts3, S Bellur3, D Cattran4, TH Cook5, J Feehally6, V Tesar7, D Maixnerova7, S Lundberg8, AM Di Palma9, F Emma10, C Rollino11, M Praga12, L Biancone13, A Pani14, J Barratt6, L Del Vecchio15, F Locatelli15, A Pierucci16, Y Caliskan17, A Perkowska-Ptasinska18, Ballarin JC19

1Immunonephrology Working Group, ERA-EDTA,
1Fondazione Ricerca Molinette, Turin, ITALY, 2CNR-IFC, Epidemiology, Reggio Calabria, ITALY, 3OUH, Pathology, Oxford, UNITED KINGDOM, 4UHN, Toronto GH, Toronto, ON, CANADA, 5IC, Nephrology, London, UNITED KINGDOM, 6LGH, Nephrology, Leicester, UNITED KINGDOM, 7GUH, Nephrology, Prague, CZECH REPUBLIC, 8KI, Nephrology, Stockholm, SWEDEN, 9BFU, Nephrology, Bari, ITALY, 10OPBG, Nephrology, Rome, ITALY, 11OSGB, Nephrology, Turin, ITALY, 12H12Octubre, Nephrology, Madrid, SPAIN, 13CSST, Nephrology, Turin, ITALY, 14AOGB, Nephrology, Cagliari, ITALY, 15OAM, Nephrology, Lecco, ITALY, 16SapienzaU, Nephrology, Rome, ITALY, 17IstanbulU, Nephrology, Istanbul, TURKEY, 18WarsawU, Nephrology, Warsaw, POLAND, 19Puigvert, Nephrology, Barcelona, SPAIN.**

OBJECTIVES

The Validation the Oxford Classification for IgA Nephropathy (IgAN) collaborative study (VALIGA) - granted in 2009 by the ERA-EDTA and supported by the Immunonephrology Working Group - consisted of 1147 cases of IgAN from 13 European Countries (Figure1) (Kidney Int. 2014;86:828-36). It validated the prognostic value of MEST features of thThe Validation the Oxford Classification for IgA Nephropathy (IgAN) collaborative study (VALIGA) - granted in 2009 by the ERA-EDTA and supported by the Immunonephrology Working Group - consisted of 1147 cases of IgAN from 13 European Countries (Figure1) (Kidney Int. 2014;86:828-36). It validated the prognostic value of MEST features of the Oxford classification (mesangial hypercellularity M, endocapillary hypercellularity E, segmental glomerulosclerosis S, tubular atrophy/interstitial fibrosis T).e Oxford classification (mesangial hypercellularity M, endocapillary hypercellularity E, segmental glomerulosclerosis S, tubular atrophy/interstitial fibrosis T).

Aim of this study was to evaluate the long-term predictive value of the pathology features (MEST scores) detected at renal biopsy in patients with IgAN enrolled in the VALIGA study and investigated over a prolonged follow-up period.

METHODS

Updated records were gained for 780/1147 patients from 45/55 VALIGA Centers (Appendix 1). After the exclusion of patients with eGFR<15 ml/min/1.73m2 at renal biopsy, the follow-up analysis was performed in 1130 subjects. The median follow-up time resulted to be prolonged by 51% from 4.7 (2.4-7.9) years of the original VALIGA to 7.1 (4.1-10.8) years. The yearly results were limited to the analysis at 15 years of follow-up, where the cohort at risk still included 98 patients and had accumulated 262 events of combined outcomes (ESRD or 50% decline in eGFR).

RESULTS

Outcomes

Demographic and clinical data at renal biopsy and during the follow-up are reported in Table 1 and 2.
In the prolonged VALIGA follow-up ESRD developed from 12% of the first VALIGA study to 18.8% (212 patients) and the combined end-point of 50% decline in eGFR or ESRD increased from 16% to 24.7% (279 patients). The survival from the combined end-point at 10 years detected by the Kaplan-Meier curve was 73±1.7 % (70±1.8 % in adults and 91±3.1% in children) (Figure 2) versus 74% (73% in adults and 83% in children) in the first VALIGA study. The rate of renal function decline was superimposable to that calculated in VALIGA (1.8±7.2 ml/min/1.73 m2).

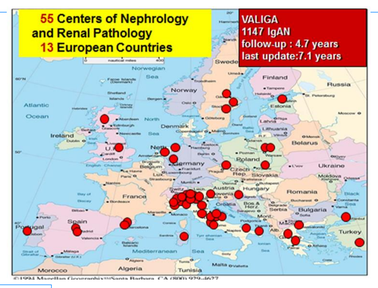


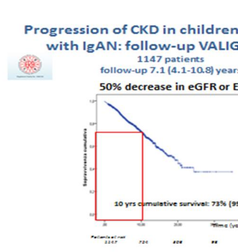
Table 1

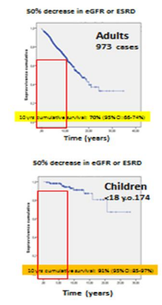
Baseline characteristics of 1130 patients with IgA nephropathy (IgAN) of the VALIGA cohort enrolled with eGFR > 15 ml/min/1.73 m2 and followed in the prolonged follow-up

Age (years)	35±16
Pediatrics subjects (% <18 years age)	15
Male (%)	73
Ethnicity (Caucasian/African/Asian/other)%	97.6/0.6/0.6/1.2
eGFR (ml/min per 1.73 m^2)	74±31
CKD stage (%)	
1 or 2	63.7
3	28.5
4	7.5
5	0.3
MAP (mm Hg)	98±13
SBP (mm Hg)	131±20
DBP (mm Hg)	81±12
Proteinuria (g/day)	1.2 (0.5-2.4)
Proteinuria g/day %	
<0.5	21
≥0.5-1	20
≥1-3	40
≥3	19
Prior tonsillectomy (%)	4
Prior RASB (%)	39

Table 2 – Follow-up data and clinical outcome

Duration of follow-up: median (IQ; years)	**7.1 (4.1-10.8)**
TA-MAP (mm Hg)	95±9
TA-SBP (mm Hg)	128±13
TA-DBP (mm Hg)	79±8
Median number of antihypertensive drugs	1.0 (1.0-2.0)
TA proteinuria (g/day)	0.8 (0.4-1.6)
TA Proteinuria g/day %	
<0.5	32
≥0.5-1	26
≥1-1.5	15
≥1.5-2	10
≥2	17
Treatments	
Corticosteroid/immunosuppression (%)	51%
RASB (%)	76
Fish oil (%)	9
Tonsillectomy (%)	2
Clinical outcome data	
Rate of decline in renal function	**-1.8±7.2**
50% decrease in eGFR n. (%)	**255 (23.5%)**
ESRD (<15 ml/min per 1.73m^2) n. (%)	**279 (24.7%)**
50% decrease in eGFR or ESRD n. (%)	**212 (18.8%)**





Correlations between renal pathology lesions and outcomes.

The rate of renal function decline was significantly associated at univariate analysis with MEST, proteinuria (UP) and mean arterial pressure (MAP) at renal biopsy (Table 3, column A). In patients not receiving steroids/ immunosuppressors (untreated) also the presence of crescents (C) was significantly associated with GFR decline (Table 3, column B). At multivariate linear regression analysis T only maintained a significant predictive value (Table 3, C), but the presence of C was a significant predictor when only untreated patients were considered (Table 3, D).
The country stratified survival from the combined end-point of renal failure or 50% drop in eGFR was significantly predicted at Cox univariate analysis by MST lesions, Art (arteriosclerosis) and baseline clinical data, including eGFR, UP, MAP, age, gender and BMI (Table 4, A). In untreated subjects C showed a significant predictive value (Table 4, B). Multivariate Cox regression analysis adjusted for MEST lesions, Art, C and for baseline clinical data confirmed an independent significant predictive value of M,S,T for the combined end-point (Table 4, C). M and T maintained a predictive value in untreated patients (Table 4,D)

Long term follow-up prediction value of pathology features

Segmented Cox regression analysis (Country stratified and adjusted for age, gender, baseline eGFR UP and MAP) (n of events and patients at risk are reported in Table 5) showed an independent predictive value for the combined end-point of T lesions in the first 5 years of follow-up, with the addition of M at 10 years and S at 15 years. The Harrell C index (HC) including MEST in addition to baseline clinical data ranged from 0.845 at the first year after renal biopsy to 0.810 at 15 years with corresponding value of HC for baseline clinical data of 0.788 and 0.782 respectively (Table 5). The delta change in C statistics was 0.057-0.060% in the first 2 years, slowly decreasing to 0.23% at 5 years, remaining stable till the longest follow-up analysis of 15 years. The analysis of risk reclassification performed by the integrated discrimination improvement (IDI) test after 5 years of follow-up ranged from 3.7 to 4.5%, and it was always statistically significant over a 15 years long term follow-up (p<0.001). Crescents had an independent negative prognostic value after 15 years from renal biopsy in untreated patients (p=0.046)

Table 3 Linear regression analysis of the rate of renal function decline (eGFR slope)

Dependent variable: rate of renal function decline (beta and p)

	A: Univariate a. all patients	B: Univariate a. untreated patients &	C: Multivariate a. all patients*	D. Multivariate a. untreated patients *&
M	-0.08 (0.009)	-0.13 (0.002)	-0.03 (0.28)	-0.06 (0.18)
E	-0.07 (0.019)	-0.02 (0.64)	-0.06 (0.07)	0.08(0.07)
S	-0.09 (0.002)	-0.13 (0.02)	-0.05 (0.14)	-0.07(0.14)
T	-0.14 (<0.001)	-0.17 (<0.001)	-0.15 (<0.001)	-0.14 (0.003)
Crescents	-0.03 (0.26)	- 0.10 (0.015)	0.001 (0.97)	-0.11 (0.01)
Arteriosclerosis	-0.02 (0.47)	0.00 (0.99)	-0.002 (0.94)	0.03 (0.51)
age	-0.02 (0.53)	-0.03 (0.48)	-0.01 (0.73)	-0.00 (0.96)
GenderM1	0.03 (0.36)	0.02 (0.67)	0.02 (0.43)	-0.005 (0.90)
eGFR	-0.01 (0.77)	0.04 (0.34)	-0.18 (<0.001)	-0.12 (0.04)
proteinuria	-0.12 (<0.001)	-0.19 (<0.001)	-0.07 (0.02)	-0.12 (0.004)
MAP	-0.13 (<0.001)	-0.13 (0.002)	-0.12 (<0.001)	-0.08 (0.07)
BMI	-0.04 (0.21)	-0.8 (0.08)		

&:Untreated patients: patients without corticosteroids /immunosuppressors during the follow-up
*Multivariate linear regression analysis country stratified : M, E, S, T, crescents and arteriosclerosis adjusted for age, gender, baseline eGFR, MAP and proteinuria

Table 4 Cox regression analysis country stratified of the survival from ESRD or 50% drop in eGFR

Dependent variable: 50% decrease in eGFR or ESRD (Exp (B) is the hazard ratio HR, 95% CI and p)

	A: Univariate analysis all patients	B: Univariate a. untreated patients &	C: Multivariate a. all patients*	D. Multivariate a. untreated patients *&
M	2.06 (1.55-2.58), p<0.001	2.83 (1.97-4.07), p<0.001	1.34 (1.02-1.76), p= 0.03	1.84 (1.24-2.75) p<0.001
E	1.35 (0.96-1.90), p=0.09	1.29 (0.77-2.17), p=0.32	1.16 (0.79-1.72), p= 0.746	0.83 (0.45-1.53), p= 0.56
S	3.01 (2.11-2.28), p<0.001	3.57 (2.21-5.78), p<0.001	1.60 (1.09- 2.34), p= 0.01	1.47 (0.85-2.54), p= 0.15
T	5.27 (4.07-6.82), p<0.001	7.00 (4.74-10.34), p<0.001	2.49 (1.83-3.40), p< 0.001	2.70 (1.66-4.37), p<0.001
Crescents	1.36 (0.94-1.97), p=0.10	1.86 (1.05-3.30), p=0.03	0.84 (0.55-1.29), p=0.43	1.85 (0.92-3.72), p= 0.08
Arteriosclerosis	2.07 (1.60-2.67), p<0.001	2.37 (1.66-3.37), p< 0.001	1.19 (0.89-1.58), p=0.22	1.14 (0.75-1.73) p =0.51
age	1.02 (1.02-1.03), p<0.001	1.03 (1.02-1.05), p< 0.001	1.00 (0.99-1.01), p=0.44	1.00 (0.99-1.02) p= 0.51
GenderM1	0.71 (0.53-0.95), p=0.02	0.70 (0.47-1.07),p= 0.10	0.90 (0.66-1.22), p=0.51	0.96 (0.60-1.54) p= 0.88
eGFR	0.97 (0.97-0.98), p<0.001	0.96 (0.95-0.97), p< 0.001	0.89 (0.98-0.99), p< 0.001	0.97 (0.96-0.99), p <0.001
proteinuria	1.30 (1.23-1.37), p<0.001	1.32 (1.22-1.44), p< 0.001	1.19 (1.12-1.26), p<0.001	1.21 (1.11-1.32) p<0.0001
MAP	1.04 (1.03-1.05), p<0.001	1.04 (1.03-1.05), p< 0.001	1.01 (1.00-1.12), p<0.001	1.01 (1.00-1.03) p=0.26
BMI	1.06 (1.03-1.09), p<0.001	1.08 (1.04-1.12), p< 0.001		

&:Untreated patients: patients without corticosteroids /immunosuppressors during the follow-up
*Multivariate linear regression analysis country stratified : M, E, S, T, crescents and arteriosclerosis adjusted for age, gender, baseline eGFR, MAP and proteinuria

Table 5 Segmented Cox regression analysis country stratified :

Basic model: age, gender, baseline eGFR, proteinuria and MAP

Model 1: MEST adjusted for age, gender, baseline eGFR, proteinuria and MAP

HC : Harrell'C index

IDI: the integrated discrimination improvement

	1 year (n= 25 events) Patient at risk=1100	2 year (n=49 events) Patient at risk=1002	3 year (n=69 events) Patient at risk=925	4 year (n=99 events) Patient at risk=845	5 year (n=135 events) Patient at risk=748	10 years (n=219 events) Patient at risk=305	15 year (n= 262 events) Patient at risk=98
*Basic model	HC: 0.788	0.733	0.786	0.792	0.794	0.784	HC: 0.782
Model 1	HC:0.845 Delta C: 0.057 IDI: 2.3%, P=0.03	0.793 Delta C: 0.060 IDI 3.7%, P<0.001	0.826 Delta C: 0.040 IDI: 3.0%, P=0.001	0.823 Delta C: 0.031 IDI: 2.2%, P=0.006	0.817 Delta C: 0.023 IDI: 3.7%, P<0.001	0.808 Delta C: 0.023 IDI: 4.5%, P<0.001	HC:0.810 Delta C: 0.028 IDI: 3.5%, P<0.001
Basic model+							
M	HR:1.30, 95% CI: 0.54-3.14, P=0.56	HR: 1.61, 95% CI: 0.87-2.96, P=0.13	HR: 1.25, 95% CI: 0.74-2.11, P=0.40	HR: 1.35, 95% CI: 0.88-2.09, P=0.17	HR: 1.41, 95% CI: 0.97-2.06, P=0.07	HR: 1.42, 95% CI: 1.06-1.92, P=0.02	HR:1.36, 95% CI: 1.03-1.79, P=0.03
E	HR: 1.79, 95% CI: 0.66-4.86, P=0.25	HR: 1.15, 95% CI: 0.53-2.50, P=0.72	HR: 1.06, 95% CI: 0.54-2.08, P=0.87	HR: 1.02, 95% CI: 0.56-1.85, P=0.95	HR: 0.96, 95% CI: 0.56-1.63, P=0.88	HR: 0.96, 95% CI: 0.63-1.47, P=0.85	HR: 1.00, 95% CI: 0.68-1.48, P=0.99
S	HR: 1.85, 95% CI: 0.40-8.63, P=0.43	HR: 1.12, 95% CI: 0.41-3.06, P=0.82	HR: 1.16, 95% CI: 0.50-2.70, P=0.74	HR: 1.19, 95% CI: 0.60-2.34, P=0.62	HR: 1.01, 95% CI: 0.59-1.74, P=0.97	HR: 1.34, 95% CI: 0.86-2.08, P=0.20	HR: 1.50, 95% CI: 1.01-2.23, P=0.045
T	HR: 4.25, 95% CI: 1.48-12.2, P=0.007	HR: 4.52, 95% CI: 2.14-9.54, P<0.001	HR: 4.53, 95% CI: 2.41-8.50, P<0.001	HR: 3.23, 95% CI: 1.94-5.39, P<0.001	HR: 3.34, 95% CI: 2.16-5.16, P<0.001	HR: 2.84, 95% CI:2.02-3.99, P<0.001	HR: 2.55, 95% CI: 1.87-3.47, P<0.001

CONCLUSIONS

The long term follow-up of the VALIGA cohort indicates the value of MST pathology findings in patients with IgAN for outcome prognostication even decades after the renal biopsy.
The M and S lesions showed a predictive role in the long term follow-up which was not independently evident in the first years after renal biopsy, suggesting their predictive value for a slow progression to fibrotic irreversible lesions. The presence of crescents showed a negative impact on the long term prognostication in patients not treated with steroids/immunosuppressive drugs.

Appendix
VALIGA Centers

The VALIGA centers' list of nephrologists includes the following (* marked the centers which sent the update by 2016).
V. Tesar, D. Maixnerova (Nephrology, First Faculty of Medicine and General University Hospital, Prague, Czech Republic)*; S. Lundberg (Nephrology, Karolinska University Hospital, Karolinska Institutet, Stockholm, Sweden)*; L. Gesualdo (Nephrology, Emergency and Organ Transplantation, University of Bari "Aldo Moro", Foggia-Bari, Italy)*; F. Emma, L. Fuiano (Nephrology, Pediatrico Bambino Gesù Hospital, Rome, Italy)*; G. Beltrame, C. Rollino (Nephrology, San Giovanni Bosco Hospital, Turin, Italy)*; R. Coppo, A. Amore, R. Camilla, L. Peruzzi (Nephrology, Regina Margherita Children's Hospital, Turin, Italy)*; M. Praga (Nephrology, Hospital 12 de Octubre, Madrid, Spain)*; S. Feriozzi, R. Polci, (Nephrology, Belcolle Hospital, Viterbo, Italy)*; G. Segoloni, L. Colla (Nephrology, S. Giovanni Battista University Hospital, Turin, Italy)*; A. Pani, A. Angioi, L. Piras (Nephrology, G. Brotzu Hospital, Cagliari, Italy)*; J. Feehally (John Walls Renal Unit, Leicester General Hospital, Leicester, United Kingdom)*; G. Cancarini, S. Ravera (Nephrology, Spedali Civili University Hospital, Brescia, Italy); M. Durlik (Transplantation Medicine and Nephrology, Warsaw Medical University, Warsaw, Poland)*; E. Moggia (Nephrology, Santa Croce Hospital, Cuneo, Italy)*; J. Ballarin (Nephrology, Fundacion Puigvert, Barcelona, Spain)*; S. Di Giulio (Nephrology, San Camillo Forlanini Hospital, Rome, Italy); F. Pugliese, I. Serriello (Nephrology, Policlinico Umberto I University Hospital, Rome, Italy)*; Y. Caliskan, M. Sever, I. Kilicaslan (Nephrology, Internal Medicine, Istanbul Faculty of Medicine, Istanbul, Turkey)*; F. Locatelli, L. Del Vecchio (Nephrology, A. Manzoni Hospital, Lecco, Italy)*; J.F.M. Wetzels, H. Peters (Nephrology and Pathology, Radboud University Nijmegen Medical Center, Nijmegen, The Netherlands)*; U. Berg (Pediatrics, Department of Clinical Science, Intervention and Technology, Huddinge, Sweden)*; F. Carvalho, A.C. da Costa Ferreira (Nephrology, Hospital de Curry Cabral, Lisbon, Portugal)*; M. Maggio (Nephrology, Hospital Maggiore di Lodi, Lodi, Italy)*; A. Wiecek (Nephrology, Endocrinology and Metabolic Diseases, Silesian University of Medicine, Katowice, Poland);M. Ots-Rosenberg (Nephrology, Tartu University Clinics, Tartu, Estonia)*; R. Magistroni (Nephrology, Policlinic of Modena and Reggio Emilia; Modena, Italy); R. Topaloglu, Y. Bilginer (Pediatric Nephrology and Rheumatology, Hacettepe University, Ankara, Turkey)*; M. D'Amico (Nephrology, S. Anna Hospital, Como, Italy)*; M. Stangou (Nephrology, Hippokration General Hospital, Aristotle University of Thessaloniki, Thessaloniki, Greece)*; F. Giacchino (Nephrology, Ivrea Hospital, Ivrea, Italy)*; D. Goumenos, P. Kalliakmani, M. Gerolymos (Nephrology, University Hospital of Patras, Patras, Greece)*; K. Galesic, L. Toric (Nephrology, University Hospital Dubrava, Zagreb, Croatia)*; C. Geddes (Renal Unit, Western Infirmary Glasgow, Glasgow, United Kingdom)*; K. Siamopoulos, O. Balafa (Nephrology, Medical School University of Ioanina, Ioannina, Greece)*; M. Galliani (Nephrology, S.Pertini Hospital, Rome, Italy); P. Stratta, M. Quaglia (Nephrology, Maggiore della Carità Hospital, Piemonte Orientale University, Novara, Italy)*; R. Bergia, R. Cravero (Nephrology, Degli Infermi Hospital, Biella,Italy)*; M. Salvadori, L. Cirami (Nephrology, Careggi Hospital, Florence, Italy)*; B. Fellstrom, H. Kloster Smerud (Renal Department, University of Uppsala, Uppsala, Sweden)*; F. Ferrario, T. Stellato (Nephropathology, San Gerardo Hospital Monza, Italy); J. Egido, C. Martin (Nephrology, Fundacion Jimenez Diaz, Madrid, Spain)*; J. Floege, F. Eitner, T. Rauen (Nephrology and Immunology, Medizinische Klinik II, University of Aachen, Aachen, Germany)*; A. Lupo, P. Bernich (Nephrology, University of Verona, Verona, Italy); P. Menè (Nephrology,S. Andrea Hospital, Rome, Italy); M. Morosetti (Nephrology, Grassi Hospital, Ostia, Italy); C. van Kooten, T. Rabelink, M.E.J. Reinders (Nephrology, Leiden University Medical Centre, Leiden, The Netherlands)*; J.M. Boria Grinyo (Nephrology, Hospital Bellvitge, Barcelona, Spain); S. Cusinato, L. Benozzi (Nephrology, Borgomanero Hospital, Borgomanero, Italy)*; S. Savoldi, C. Licata (Nephrology, Civile Hospital, Ciriè, Italy)*; M. Mizerska-Wasiak , M. Roszkowska-Blaim (Pediatrics, Medical University of Warsaw, Warsaw, Poland); G. Martina, A. Messuerotti (Nephrology, Chivasso Hospital, Chivasso, Italy)*; A. Dal Canton, C. Esposito, C. Migotto (Nephrology Units, S. Matteo Hospital and Maugeri Foundation, Pavia, Italy);G. Triolo, F. Mariano (Nephrology CTO, Turin, Italy)*; C. Pozzi (Nephrology, Bassini Hospital, Cinisello Balsamo, Italy)*; R. Boero (Nephrology, Martini Hospital, Turin, Italy)*.
The VALIGA centers' list of pathologists includes the following:
G.Mazzucco (Turin, Italy); C. Giannakakis (Rome, Italy); E. Honsova (Prague, Czech Republic); B. Sundelin (Stockholm, Sweden); A.M. Di Palma (Foggia-Bari, Italy); F. Ferrario (Monza, Italy); E. Gutiérrez (Madrid, Spain); A.M. Asunis (Cagliari, Italy); J. Barratt (Leicester, United Kingdom); R. Tardanico (Brescia, Italy); A. Perkowska-Ptasinska (Warsaw, Poland); J. Arce Terroba (Barcelona, Spain); M. Fortunato (Cuneo, Italy); A. Pantzaki (Thessaloniki, Greece); Y. Ozluk (Istanbul, Turkey); E. Steenbergen (Nijmegen, The Netherlands); M. Soderberg (Huddinge, Sweden); Z. Riispere (Tartu,Estonia); L. Furci (Modena, Italy); D. Orhan (Ankara, Turkey); D. Kipgen (Glasgow, United Kingdom); D. Casartelli (Lecco, Italy); D. Galesic Ljubanovic (Zagreb, Croatia); H Gakiopoulou (Athens, Greece), E. Bertoni (Florence, Italy); P. Cannata Ortiz (Madrid, Spain); H. Karkoszka (Katowice, Poland), H.J. Groene (Heidelberg, Germany); A. Stoppacciaro (Rome, Italy); I. Bajema, J. Bruijn (Leiden, The Netherlands); X. Fulladosa Oliveras (Barcelona, Spain); J. Maldyk (Warsaw, Poland); and E. Ioachim (Ioannina, Greece).

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