with either segmental ('IV-S') or global ('IV-G') distribution of lesions as published data so far has failed to show a worse outcome between these groups. They also quote one publication in which the authors conclude that this subclassification 'may not be justified.'[3]

Poorer renal prognosis in pediatric LN may be due to different etiopathogenesis. We found a worse outcome in children with class IV-G LN in our published data of 39 cases (85% female) aged 3.3–18.0 (median 13.7) years at our single center over 10 years.[4] Patients underwent 49 percutaneous renal biopsies at 0.1–7.8 (median 1.0) years from diagnosis of systemic lupus erythematosus with renal and overall survival rates of 90 and 92% respectively at a follow-up of 1.3–15.4 (median 5.5) years. In our cohort, the three most severe cases of chronic kidney disease with estimated glomerular filtration rate $<25\,ml/min/1.73\,m^2$ were patients with diffuse global sclerosing (class IV-G(C)) LN. Therefore, we require larger studies of pediatric LN to evaluate progression of disease and guide physicians on treatment.

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SD Marks[1,2], NJ Sebire[3] and K Tullus[1,2]
[1]Nephro-Urology Unit, UCL Institute of Child Health, London, UK; [2]Department of Paediatric Nephrology, Great Ormond Street Hospital for Children NHS Trust, London, UK and [3]Department of Paediatric Pathology, Great Ormond Street Hospital for Children NHS Trust, London, UK
Correspondence: *SD Marks, Department of Paediatric Nephrology, Great Ormond Street Hospital for Children NHS Trust, Great Ormond Street, London WC1N 3JH, UK. E-mail: s.marks@ich.ucl.ac.uk*

The Gitelman syndrome mutation, IVS9 + 1G > T, is common across Europe

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To the Editor: In a paper published in *Kidney International*, Coto *et al.*[1,2] described a *SLC12A3* mutation, IVS9 + 1G > T, subsequently shown to result in aberrant splicing and exon skipping. The 21 homozygote patients known so far belong to unrelated Gypsy families from Spain, Portugal, France, and Germany, suggesting that IVS9 + 1G > T is a shared ancestral mutation.[1,2] To provide more comprehensive data on its molecular epidemiology in European Gypsies, we tested a panel of 599 unrelated control subjects of Roma/Gypsy ethnicity from Bulgaria (representing eight different sub-isolates), Romania, Hungary, the Czech Republic, and Spain. Amplification refractory mutation system-polymerase chain reaction was used for screening (conditions available upon request), with positive results verified by restriction-fragment length polymorphism.[1] We detected 12 heterozygotes, randomly spread between sub-isolates and geographic locations, pointing to a founder effect common to the entire European Gypsy population. The overall carrier rate is 2%, ranging between 0 and 7% in different sub-isolates. Given the variable and often nonspecific clinical manifestations of Gitelman syndrome,[3] this information should facilitate the diagnosis and management of affected Gypsy individuals. The allelic heterogeneity of Gitelman syndrome, with over 100 mutations in *SLC12A3* identified to date, has made genotype–phenotype correlations problematic.[3] The observed clinical heterogeneity in individuals homozygous for the same mutation points to the potential role of modifying genes.[1,4] The IVS9 + 1G > T founder mutation, with an expected incidence of affected homozygotes of 1:10 000 across Europe (up to 1:800 in some sub-isolates), should allow the collection of large numbers of genetically homogeneous patients, where phenotype diversity can be characterized further and the search for modifying factors can begin.

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ST Bouwer[1], E Coto[2], F Santos[3], D Angelicheva[1], D Chandler[4] and L Kalaydjieva[1]
[1]Western Australian Institute for Medical Research and Centre for Medical Research, The University of Western Australia, Perth, Western Australia, Australia; [2]Genética Molecular, Hospital Central de Asturias (Maternidad), Oviedo, Spain; [3]Pediatría, Hospital Central de Asturias (Maternidad), Oviedo, Spain and [4]Centre for Clinical Research in Neuropsychiatry, The University of Western Australia, Perth, Western Australia, Australia
Correspondence: *L Kalaydjieva, Laboratory of Molecular Genetics, Western Australian Institute for Medical Research, 'B' Block, QEII Medical Centre, Perth, Western Australia 6009, Australia. E-mail: luba@cyllene.uwa.edu.au.*