

## IN BRIEF

**CRYSTAL ARTHRITIS****Apolipoprotein gene variants—causal role in gout?**

A previously detected association between gout and the APOA1–APOC3–APOA4 gene cluster has been confirmed in a larger study, suggesting a causal role. Data from 5,367 Atherosclerosis Risk in Communities and 2,984 Framingham Heart Study participants were compared with findings from a New Zealand cohort of European and Polynesian (Māori and Pacific) individuals, comprising 2,452 healthy controls and 2,690 patients with clinical gout. In Polynesians, the rs670 T allele of APOA1 increased the risk of gout in both sexes (OR 1.53,  $P = 4.9 \times 10^{-6}$ ), whereas the rs5128 G allele of APOC3 decreased the risk of gout in men only (OR 0.86,  $P = 0.026$ ). Similarly, in Europeans, the rs670 T allele tended to increase gout risk in both sexes, and the rs5128 G allele did decrease gout risk in men; however, the rs5128 effect was only seen after adjustment for triglyceride and HDL-cholesterol levels (OR 0.81,  $P = 0.039$ ).

**ORIGINAL ARTICLE** Rasheed, H. *et al.* Replication of association of the apolipoprotein A1-C3-A4 gene cluster with the risk of gout. *Rheumatology* <http://dx.doi.org/10.1093/rheumatology/kew057> (2016)

**SYSTEMIC LUPUS ERYTHEMATOSUS****Urinalysis reveals kidney-derived immune cells**

New research suggests that kidney-resident immune cells are present in the urine of most patients with systemic lupus erythematosus (SLE) and so can be characterized without the need for biopsy. Urinary flow cytometry revealed lymphocytes in >90% of 41 patients with lupus nephritis (LN) and in ~60% of 28 patients with non-LN SLE. Half of the patients with LN had urinary cell profiles resembling those of patients with non-LN SLE: T cells (predominantly CD8<sup>+</sup>) were always present, and B cells were either absent or a mixture of naive and memory B cells. However, the other half of patients with LN had a distinct urinary cell profile: B cells were exclusively antibody-secreting plasmablasts or plasma cells, and T cells were predominantly CD4<sup>+</sup>. The majority of patients in this group had proliferative nephritis and impaired kidney function (some had reached end-stage renal disease); plasmacytoid dendritic cells, IFN $\alpha$  and IFN $\beta$  were frequently also present.

**ORIGINAL ARTICLE** Scott, E. *et al.* Immune cells and type 1 IFN in urine of SLE patients correlate with immunopathology in the kidney. *Clin. Immunol.* <http://dx.doi.org/10.1016/j.clim.2016.04.005> (2016)

**RHEUMATOID ARTHRITIS****ESPOIR predictors of remission in early RA**

In 664 patients from the French ESPOIR early rheumatoid arthritis (RA) cohort, remission after 6 or 12 months could be predicted from baseline variables in 26.8–51.4% of patients. In addition to young age, six clinical variables included in core datasets predicted remission: tender joint count; swollen joint count; physician's global estimate; patient's global estimate; patient-reported physical function; and pain. Of the six remission criteria applied, four required a formal joint count (ACR–EULAR Boolean criteria; simplified disease activity index; clinical disease activity index; and 28-joint disease activity score) and two did not (routine assessment of patient index data 3 (RAPID3) and a RAPID3 score  $\leq 3$  plus 0–1 swollen joints). However, the absence of traditional indicators of poor prognosis in RA did not predict remission.

**ORIGINAL ARTICLE** Castrejón, I. *et al.* Prediction of remission in a French early arthritis cohort by RAPID3 and other core data set measures, but not by the absence of rheumatoid factor, anticitrullinated protein antibodies, or radiographic erosions. *J. Rheumatol.* <http://dx.doi.org/10.3899/jrheum.141586> (2016)