for IgG in humans, we included transgenic mice expressing FcyRIIA to our in vivo mechanistic investigations. Platelet activation and mitochondrial release were monitored upon intravenous injection of synthetic ICs in wild type (WT) and transgenic (Tg) FcyRIIA mice.

Results We found that on activation, platelets relocate their mitochondria toward the cell membrane and then release respiratory-competent mitochondria into the extracellular milieu, both as free organelles and encapsulated within vesicles called micro-particles (MP). Extracellular mitochondria were internalised by bystander leukocytes and, importantly, induced leukocytes rolling and adhesion to the blood vessel wall, suggesting that neutrophils and/or endothelial cells are activated by extracellular mitochondria. Activated platelets were more abundant in SLE patients than control subjects, and were associated with IgG. Extracellular mitochondria, both encapsulated in platelet MPs or naked, were also observed in blood circulation in SLE, and were frequently associated with IgG. Mechanistically, ICs present in blood induce profound cell activation, which is dependent on platelet FcyRIIA and its signalling cascade.

Conclusions Platelets represent an important source of mitochondria, which release in blood upon stimulation of FcyRIIA, might promote systemic inflammation in SLE. Whether the blockade of FcyRIIA might represent an attractive avenue in SLE research, and whether platelet activation markers and extracellular mitochondria might be utilised as potential biomarkers for the stratification of lupus patients needs to be further considered.

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**CSF-1 AND IL-34: DISTINCT POTENTIAL BIOMARKERS FOR LUPUS**

Julia Menke, Andreas Schwarting, Vicki Rubin Kelley. Department of Nephrology and Rheumatology, Johannes-Gutenberg University Mainz, Germany; Department of Medicine, Brigham and Women’s Hospital, Boston, MA

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**Background** A noninvasive means to predict the onset and recurrence of lupus is needed to optimise and individualise treatment. Macrophages (MØ) are prominent in inflamed tissues targeted for destruction in SLE. We hypothesised that the principal molecules required for MØ survival and proliferation are biomarkers for SLE. CSF-1 and IL-34 are promising candidates as both (i) bind to cFMS expressed by MØ, thereby promoting MØ survival and proliferation and (ii) promote destructive inflammation. However, IL-34 and CSF-1 have differing functions, which may be related to IL-34, not CSF-1, binding to a second receptor and distinct spatial temporal expressions.

**Materials and methods** We analysed serum and urine CSF-1 and IL-34 levels in SLE patients with nephritis (LN), arthritis (LA), cutaneous and serositis compared with healthy controls in two large cohorts (ELISA). While serum and urine CSF-1 expression is elevated in each manifestation, CSF-1 is notably higher in LN. In contrast, serum IL-34 expression is dramatically higher in LA, not LN. Thus, we probed for CSF-1 and IL-34 expression in LN (kidney) and LA (synovium). Moreover, we longitudinally tracked serum CSF-1 and IL-34 prior to LN (biopsy proven), with disease activity including flares and during LA in comparison to disease activity.