promoted these effects through the membrane receptor GPER1 located in lipid rafts and that inhibition of lipid rafts and GPER1 suppressed SLE serum-induced skin inflammation and expression of inflammatory molecules.

Conclusions We conclude that oestrogen promotes the development of skin injury induced by SLE serum through the membrane receptor GPER1 and that lipid rafts play an important role in the regulatory effect of GPER1 in SLE skin inflammation.

Results We found that the severity of skin lesion in IL-1 receptor deficient mice and caspase-1 deficient mice was reduced compared with that in wild type mice. IL-1 receptor deficiency suppressed the expression of FcγRI (CD64) and MHC class II (CD74), and increased the level of FcγRII (CD32) induced by lupus serum. IL-1 receptor deficiency also suppressed the lipid raft clustering and IFN-γ in T cells, and reduced IgG internalisation and presentation in macrophage, and decreased expression of MCP-1 and TNFα in monocytes. In addition, TNFα could promote the proliferation of keratinocytes.

Conclusions Our findings indicate that IL-1 plays an important role in skin lesions of lupus erythematosus. This study suggests IL-1 is a therapeutic target in skin lesions of systemic lupus erythematosus.

Background and aims Skin injury is the second most common clinical manifestation in patients with systemic lupus erythematosus (SLE), but its pathogenesis has not been thoroughly elucidated.

Methods Based on skin deposition of IgG in SLE, we studied the features and mechanisms of intradermal IgG-induced skin inflammation

Results We found that skin inflammation appeared at 3 hour and peaked at 3 d after intradermal injection of lupus IgG. This phenomenon was related to the dose of injected IgG but not to systemic disease activity. The severity of skin inflammation induced by lupus IgG was significantly decreased in mice depleted of monocytes and in mice deficient in TNF-α but not in mice lacking mature lymphocytes. Furthermore, lupus IgG promoted the progression of monocyte differentiation to dendritic cells (DCs) and enhanced the expression of TNF-α. TNF-α was found to stimulate the IgG-induced maturation of DCs and played a major role in the proliferation and activation of keratinocytes.

Conclusions The results also indicate that the deposition of IgG in skin exerts an important role in the pathogenesis of skin injury in patients with SLE; therefore, blocking the IgG/FcR signalling pathway can be a therapeutic target in skin lesions of patients with SLE.