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.