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SUPPLEMENTARY INFORMATION

In format as provided by the authors

Cutaneous lupus erythematosus: new insights into pathogenesis and therapeutic strategies

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Supplementary Figures

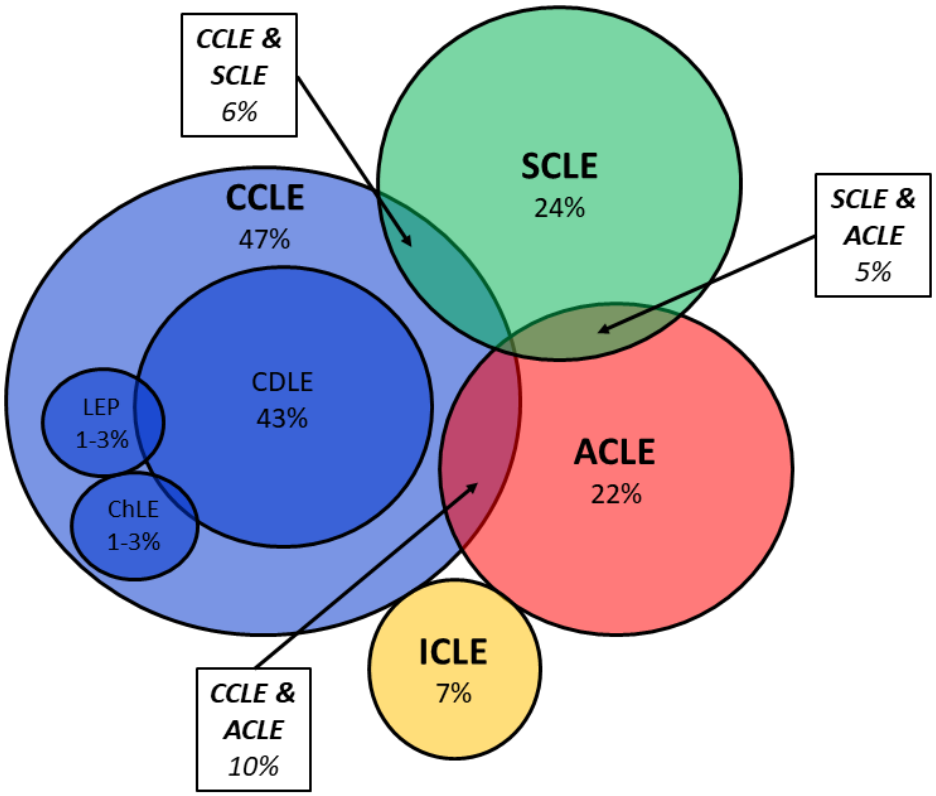

Figure S1: Distribution of CLE-Subsets

Distribution of specific subtypes within the spectrum of CLE with frequency and overlap between different subsets (ACLE = acute cutaneous LE, SCLE= subacute cutaneous LE, ICLE= intermittent cutaneous LE, CCLE= chronic cutaneous LE with its subtypes: chronic discoid LE (CDLE), LE profundus (LEP) and Chilblain LE (ChLE). This figure is based on data of 1002 patients within the EUSCLE register. Of those, 347 patients were suffering from more than one CLE subtype[1]. All percentages refer to the percentage of the 1002 patients with that subtype(s).

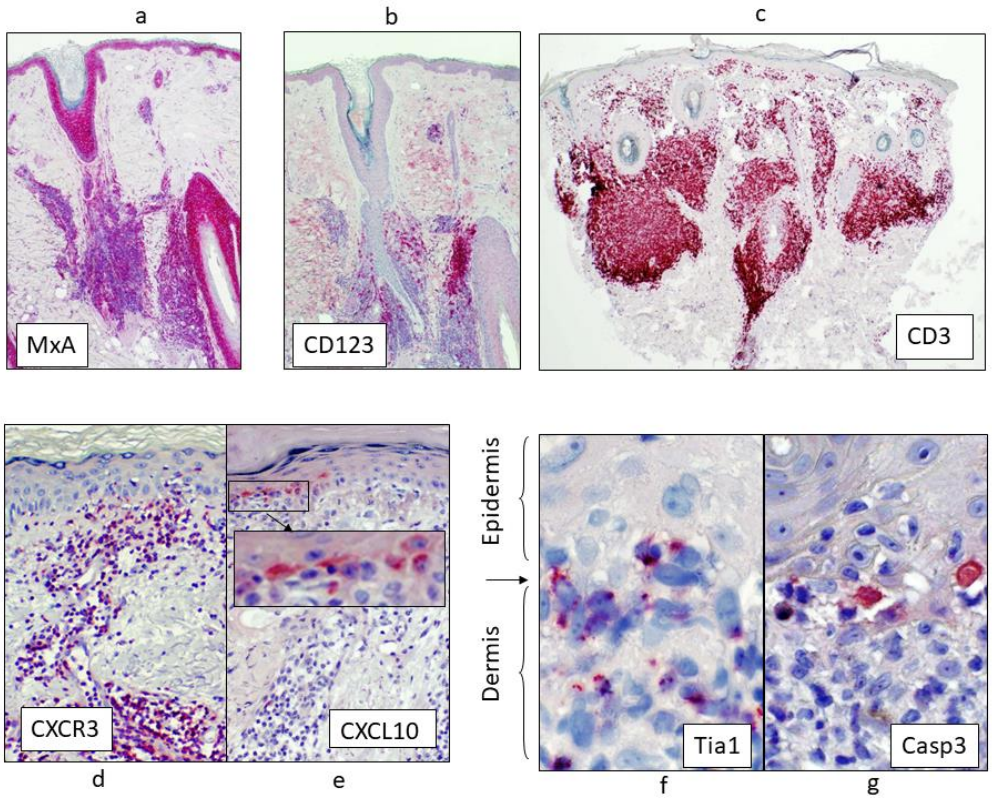

Figure S2: Main immunohistological features of LE-specific skin lesions

Main characteristics of CLE skin lesions in immunohistology. The type I/III-surrogate marker MxA identifies keratinocytes and infiltrating immune cells as main IFN producers (a) and CD123-staining reveals clusters of natural-type-I-IFN-producing pDCs within the infiltrate (b). In general, CD3+ T-cells form the largest immune cell population in LE (c). The chemokine CXCL10 is specifically expressed in skin areas with a pronounced interface dermatitis and recruits CXCR3+ effector cells (d, e). These effector cells bear several cytotoxic molecules, including Tia1, which induce keratinocytic cell death, visualized by the expression of caspase 3 (f, g).

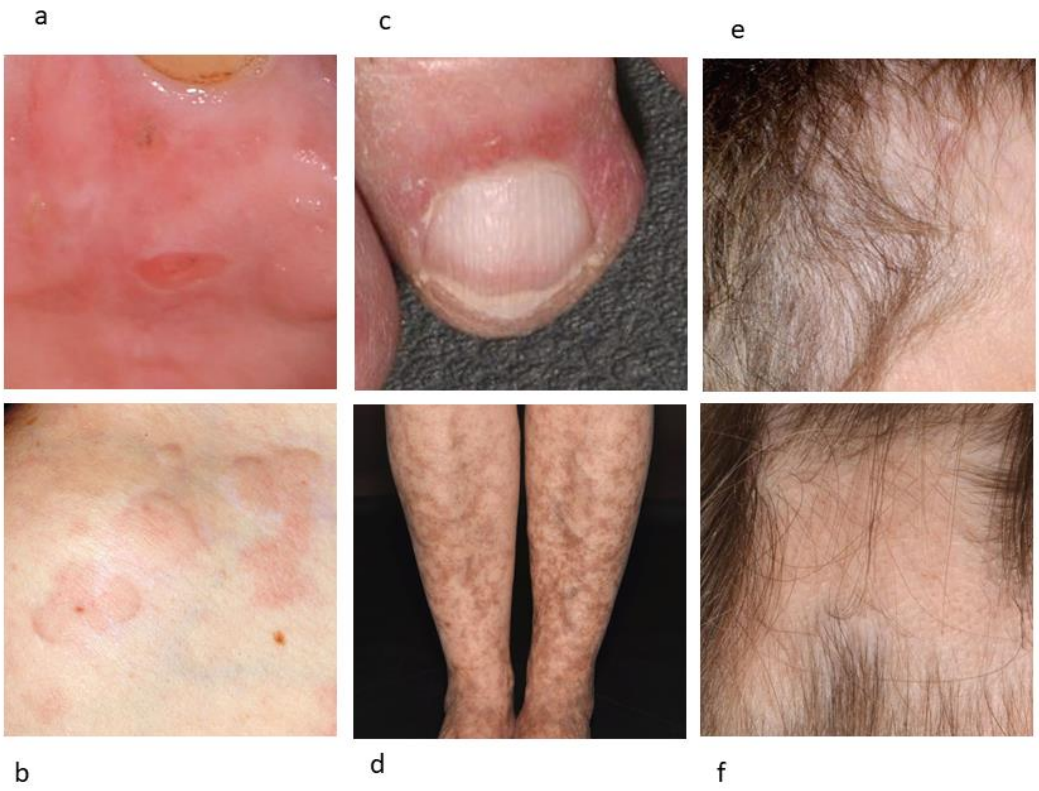

Figure S3: LE non-specific skin lesions

Common LE non-specific skin lesions: gingiva ulcer (a), urticarial vasculitis (b), periungual erythema (c), livedo racemosa (d), diffuse alopecia (e), and alopecia areata (f).

References:

1. Biazar, C. *et al.* Cutaneous lupus erythematosus: first multicenter database analysis of 1002 patients from the European Society of Cutaneous Lupus Erythematosus (EUSCLE). *Autoimmun Rev* **12,** 444–454 (2013).