cells ($P=0.031$) but also C4 and the amount of *Unclassified Ruminococcaceae* ($P=0.032$).

Conclusion: Abnormality of T cell subsets, especially the level of Naïve CD4⁺T, $\gamma\delta$ T, Tfh, Treg, and Th17 cells contributes to the occurrence and progression of SLE, which may be related to the disturbance of gut microbiota. Therefore it is necessary to attach importance to the alteration of gut microbiota to prevent the outbreak of inflammation and maybe they can be identified as biomarkers predicting disease activity.

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What can we learn from the tissue

OP0308

SENESCENT PROGENITOR CELLS IN THE SKIN OF LUPUS PATIENTS

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Background: Systemic lupus erythematosus (SLE) is an autoimmune disease, which is characterized by skin lesions, amongst other symptoms. These lesions (chronic discoid lupus erythematosus (CDLE) and subacute cutaneous lupus erythematosus (SCLE)) feature lymphocytic infiltration close to basal layer of the epidermis (i.e. the location of the epidermal progenitor cells), namely the epidermal dermal junction (EDJ) area. Epidermal progenitor cells maintain the homeostasis of the skin through their proliferation and differentiation into keratinocytes. In fast turnover tissues, like the skin, a population of 'transient amplifying cells' (TA cells), additionally facilitates generation of enough daughter cells to maintain skin homeostasis. These cells are located in an upper layer (suprabasal layer) of the epidermis, next to the basal layer. Senescence is an irreversible and locally spreading phenomenon that induces permanent cell cycle arrest.

Objectives: To evaluate expression of senescence markers p16 and p21 in the epidermal progenitor and TA niches in patients with SCLE and CDLE. This was compared to a panel of other dermatological conditions with and without infiltration close to EDJ, as disease controls, and to control skin tissue.

Methods: Age-matched skin lesions from patients with SCLE (n=12), CDLE (n=8), other conditions with EDJ infiltration (e.g. lichen planus, n=22), and dermatoses without EDJ infiltration (e.g. eczema, n=27), and non-lesion control biopsies (n=3) from SLE patients were employed. p16 and p21 expression in the progenitor niche (basal layer) and TA cell niche (suprabasal layer), and the whole epidermis of skin lesions biopsies were examined by immunohistochemistry.

Results: In healthy skin biopsies, 0 ± 0 SEM p16⁺ cells/mm² in the progenitor niche and 5 ± 4 SEM p21⁺ cells/mm² in TA niche were observed. In skin lesions from patients with CDLE and SCLE, significantly more p16⁺ cells in the progenitor cell niche (45 ± 14 SEM/mm²) and p21⁺ cells (182 ± 38 SEM/mm²) in TA niches were detected, compared to control biopsies ($p < 0.05$), and compared to those dermatoses without EDJ infiltration (p16⁺ 11 ± 3 SEM/mm², p21⁺ 86 ± 28 SEM/mm², $p < 0.05$, Figure 1). p16 and p21 expression in CDLE and SCLE lesions did not significantly differ from other dermatoses with EDJ infiltration ($p > 0.05$). Across all dermatoses analyzed, the number of p16⁺ cells was significantly correlated with the number of p21⁺ cells, both in the progenitor niche ($r=0.45$, $p < 0.0001$) and TA niche ($r=0.47$, $p < 0.0001$). p16⁺ cells however were more frequently found in the progenitor cell niche, and p21⁺ cells conversely in the TA cell niche ($p < 0.0001$).

Figure 1. Increased p16⁺ and p21⁺ cells in the progenitor and TA niches in dermatoses with EDJ infiltration. A. Graphic illustration for the progenitor cell niche (the basal layer), the transient amplifying (TA, the supra-basal layer) cell niche, and the further differentiated area. B-D. Representative staining of p16 (blue) and epidermal growth factor receptor (EGFR, brown, an epithelial cell marker) in dermatoses with (CDLE) and without (PMLE) epidermal-dermal junction (EDJ) infiltration groups. Black arrowheads point to p16⁺ progenitor epidermal cells. E-G. Representative staining of p21 (blue) and EGFR (brown) in dermatoses with (CDLE) and without (PMLE) EDJ infiltration groups. Hollow arrowheads point to p21⁺ TA epidermal cells. The dashed line indicates the EDJ. CDLE: chronic discoid lupus erythematosus, PMLE: polymorphous light eruption.

Conclusion: In CDLE and SCLE, cutaneous manifestations of SLE, more progenitor and TA cells expressing markers of senescence were detected. This was in common with other dermatological conditions where lymphocytic infiltration is in close proximity to the progenitor and TA cell niche, namely in the EDJ. Increased senescence might infer the collapse of homeostasis in target (local) epidermis, which may influence tissue repair in the lesion. Elimination of senescent cells may therefore represent a viable therapeutic option to encourage timely and complete wound healing, in skin lesions of CDLE and SCLE patients.

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EULAR Projects in musculoskeletal imaging

OP0309

THE ROLE OF IMAGING TO GUIDE INTERVENTIONAL PROCEDURES IN RHEUMATIC AND MUSCULOSKELETAL DISEASES: A SYSTEMATIC LITERATURE REVIEW TO INFORM EULAR RECOMMENDATIONS

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Background: Imaging guidance gives the opportunity to visualize a needle or other instrument when performing interventions on musculoskeletal sites in patients with rheumatic and musculoskeletal diseases (RMDs). Studies assessing the role of imaging guided procedures have been performed, however, no consensus has been made yet about when and which imaging techniques to use for these interventions.