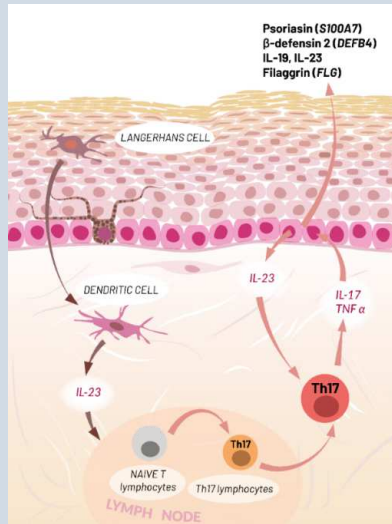


Psoriasis is a frequent multifactorial chronic inflammatory skin disease. IL-17, mostly secreted by Th-17 T-cells is the psoriasis main driver. It activates NFκB, C/EBP and STAT1 in keratinocytes and other skin cells. In response, activated keratinocytes synthesize antimicrobial peptides (e.g. psoriasis, βdefensin2) and cytokines (e.g. IL-23, IL-19).



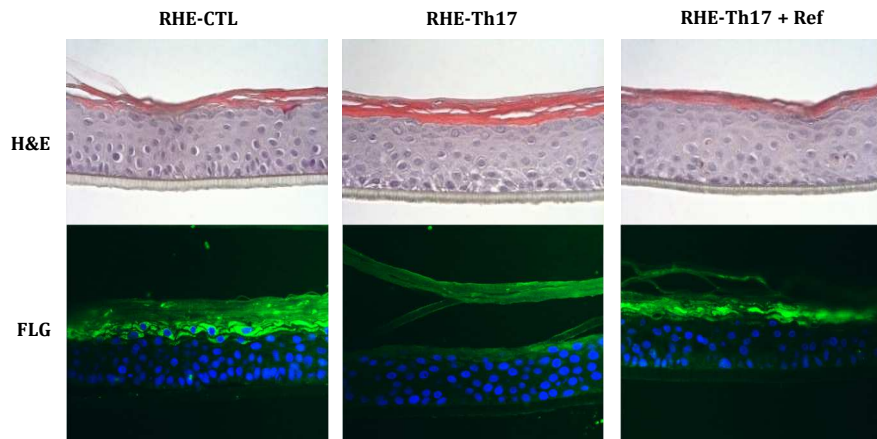
## SKIN MODELS:

- **RHE-Th17:** reconstructed human epidermis treated with Th17 interleukins
- **NHEKs-Th17:** primary human keratinocytes treated with Th17 interleukins
- *Positive reference (Ref) available for full objectivation*

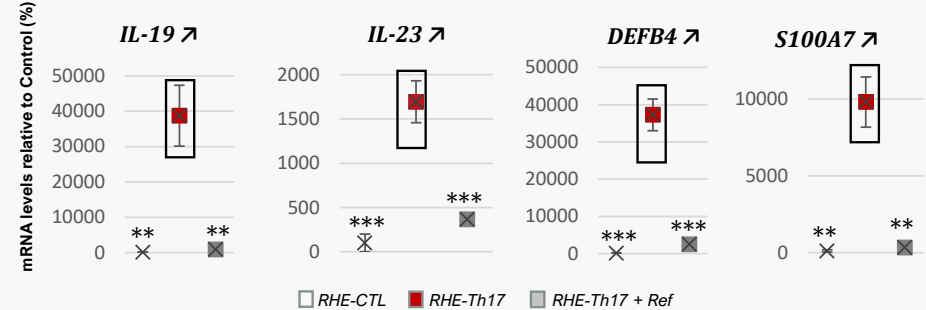
## ENDPOINTS:

- Morphological analysis by Hemalun/Eosin (H/E) staining
- Level of activation of **transcription factors** involved in Th-17 inflammation (NFκB, STAT3, etc.)
- Localization and quantification of protein markers by **western blotting** (IκBζ) or **immunostaining** (FLG, Psoriasis, βdefensin2, etc.)
- **Quantification of cytokines** released in culture supernatants by ELISA (IL-19, IL-23)
- **Expression of genes playing key roles in psoriasis, by RT-qPCR** : individual gene expression by TaqMan or 96 key genes expression by TaqMan Low-Density Array (contact StratiCELL for more details about the *Sensitive-TLDA* arrays)

H&E staining and Filaggrin (FLG) immunofluorescence of RHE untreated (CTL) or treated with Th17 cytokines (RHE-Th17) compared to positive reference (RHE-Th17 + Ref).



Gene expression levels evaluated by RT-qPCR (\*\*:  $p < 0,01$  - \*\*\*:  $p < 0,001$ ).



IL-19 and IL-23 release measured by ELISA in RHE supernatants (\*\*:  $p < 0,1$  - \*\*\*:  $p < 0,001$ ).

