025 Analysis of the related factors that leading to the resistance of topical treatment for bullous pemphigoid patients
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Psoriasis is an immune-mediated chronic inflammatory skin disease involving cell-to-cell interactions within the lesional skin. Although psoriatic skins contain a highly increased number of cells with great heterogeneity, population diversity and single-cell level transcription expression within psoriatic plaques have been incompletely understood. Here we profile the transcriptionomes of about 80,000 single cells from human psoriatic and normal skins to reconstruct a catalogue of lesional cell population. By aggregating whole individual cells, we identified 25 cellular clusters segregated by distinct patterns of gene expression. There were at least 4 different clusters of keratinocytes highly enriched in psoriatic skins expressing multiple keratin genes and IL36. Although psoriatic fibroblasts would be classified into two main subsets by DPP4 expression as in normal skins, the majority of fibroblasts in psoriatic skins would be classified into two main subsets by DPP4 expression as in normal skins, the majority of fibroblasts in psoriatic plaques expressed CCL2 which would recruit CR2+ inflammatory cells into the lesional dermis. Although both normal and psoriatic skins contained T cells with high level of TIGIT, psoriatic plaques harbored an additional population of CD69+ T cells expressing high level of multiple heat shock proteins, REL, JUN, and FOS, but negligible expression of checkpoint molecules. Population of CR2+ myeloid dendritic cells and CD14+CD68+ macrophages would significantly segregated between normal and psoriatic skin. To provide a comprehensive catalogue of whole cell populations in the inflamed human skin we expanded our knowledge of cellular diversity in psoriasis.