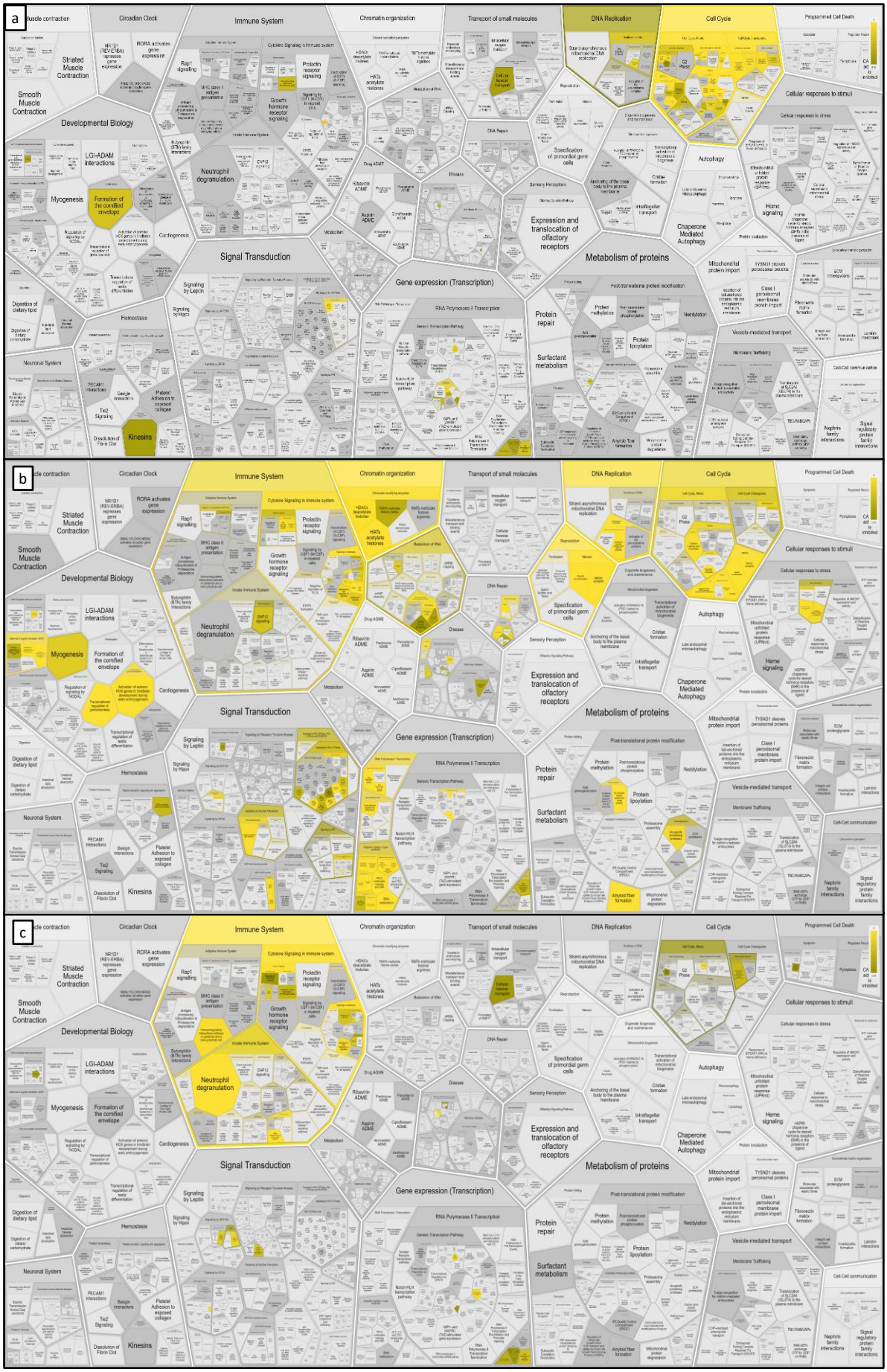
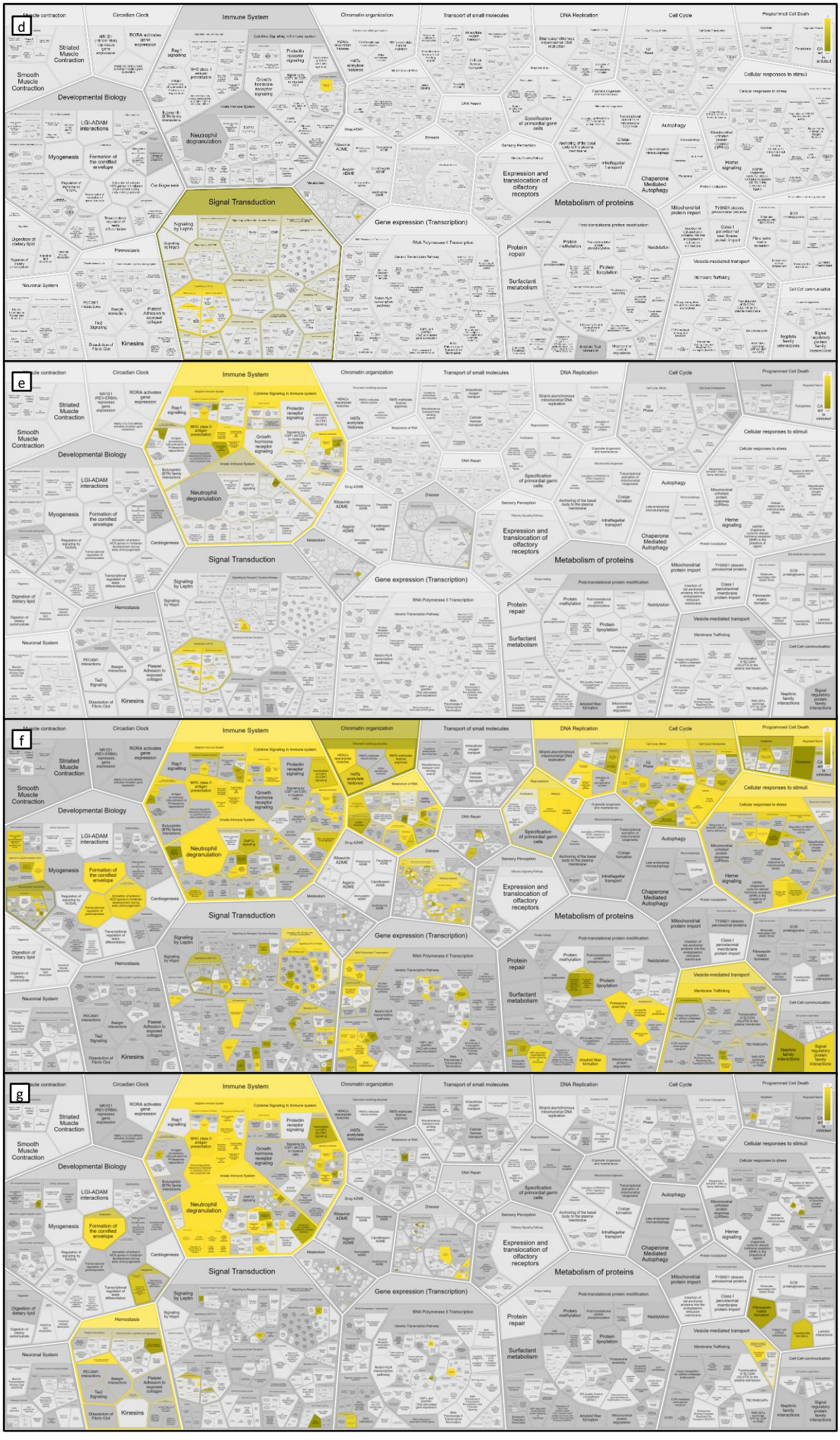


Fig. S1 Enriched pathways in concurrent skin diseases illustrating a reacfoam of the differentially upregulated genes common between **(a)** psoriasis and vitiligo, showing enhancement of pathways involved in cell cycle, DNA replication, and formation of the cornified envelope; **(b)** lichen planus and psoriasis, showing enhancement of pathways involved in the immune system, chromatin organization, DNA replication, cell cycle, reproduction, and metabolism of RNA; **(c)** psoriasis and alopecia areata, showing enhancement of pathways involved in the immune system including cytokine signaling (interferons, IL-2 family, IL-4, IL-6, IL-10, and IL-13) and neutrophil degranulation;



Cont. Fig. S1 Enriched pathways in concurrent skin diseases illustrating a reacfoam of the differentially upregulated genes common between **(d)** vitiligo and alopecia areata, showing enhancement of pathways involved in signaling transduction and IL-10 signaling; **(e)** lichen planus and alopecia areata, showing enhancement of pathways involved in the immune system (TCR signaling and MHC class II presentation) and the cytokine signaling (interferon signaling, IL-10 signaling, ns IL-12 family signaling); **(f)** cutaneous lupus erythematosus and lichen planus, showing enhancement of pathways involved in the immune system, cell cycle, programmed cell death, DNA replication, cellular response to stimuli, and vesicle-related transport; and **(g)** acne and rosacea, showing enhancement of pathways involved in the immune system including cytokine signaling (IL-4 and IL-13 family, IL-10, and IL-20 family), the adaptive immune system (signaling by the B cell receptor, MHC class II antigen presentation, and TCR signaling), and the innate immune system (neutrophil degranulation, Fc gamma receptor dependent phagocytosis, and Fc epsilon signaling).



Fig. S2 Enriched pathways in concurrent skin diseases illustrating the top 20 enriched pathways common in **(a)** psoriasis and vitiligo, showing prominence of pathways involved in TNFR1-mediated processes such as ceramide production, proapoptotic signaling, and NF kappa B signaling (reactome analysis); **(b)** lichen planus and psoriasis, highlighting pathways involved in VEGFA-VEGFR2 signaling, TGF-β signaling, and Oncostatin M signaling (wikipathways analysis); (c) psoriasis and alopecia areata, showing prominence of pathways involved in complement activation (reactome analysis);



Cont. Fig. S2 Enriched pathways in concurrent skin diseases illustrating the top 20 enriched pathways common in **(d)** vitiligo and alopecia areata, showing prominence of pathways involved in chemokine receptors bind chemokines (reactome analysis); **(e)** lichen planus and alopecia areata, highlighting pathways involved in the immune system (reactome analysis); **(f)** cutaneous lupus erythematosus and lichen planus, showing prominence of pathways involved in the immune system and cellular responses to stress and stimuli (reactome analysis); and **(g)** acne and rosacea, highlighting pathways involved in fatty acids metabolism.

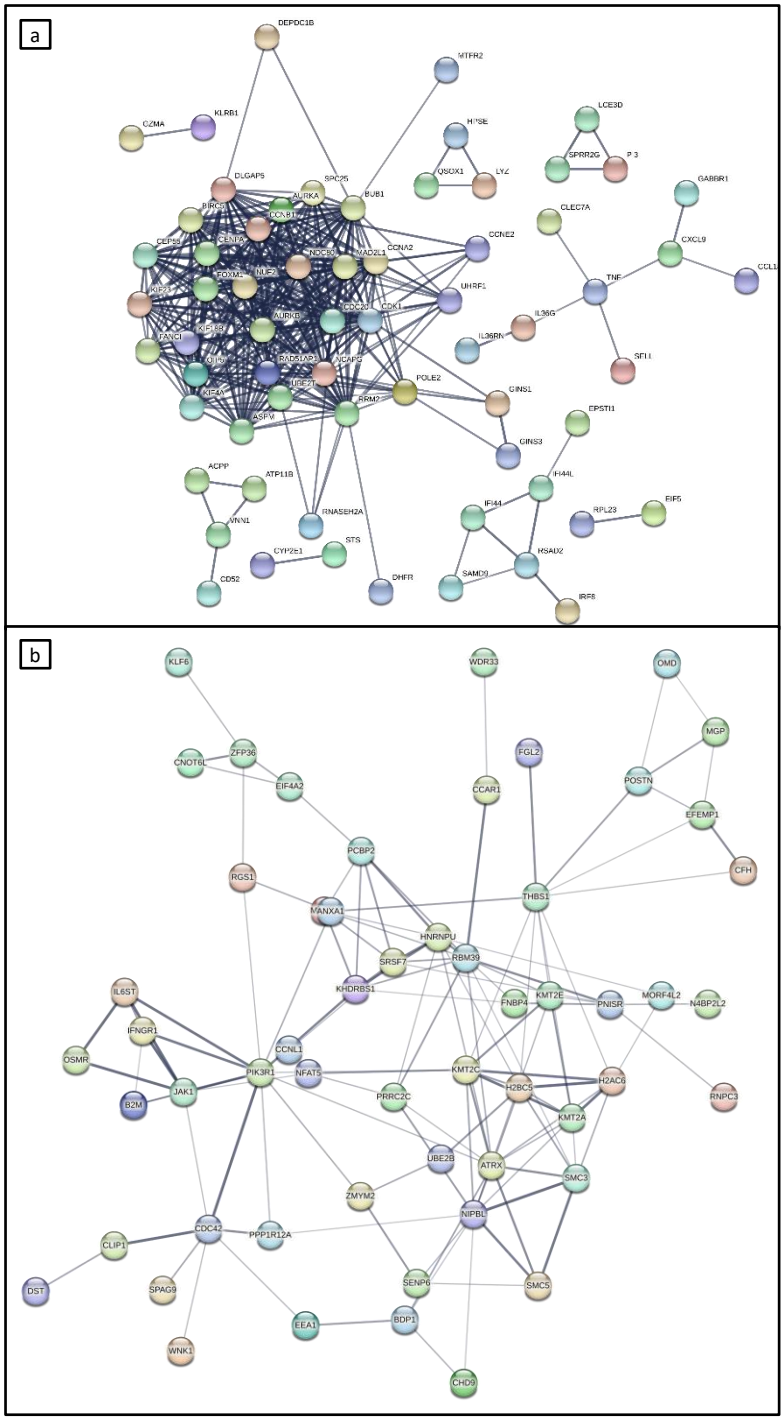
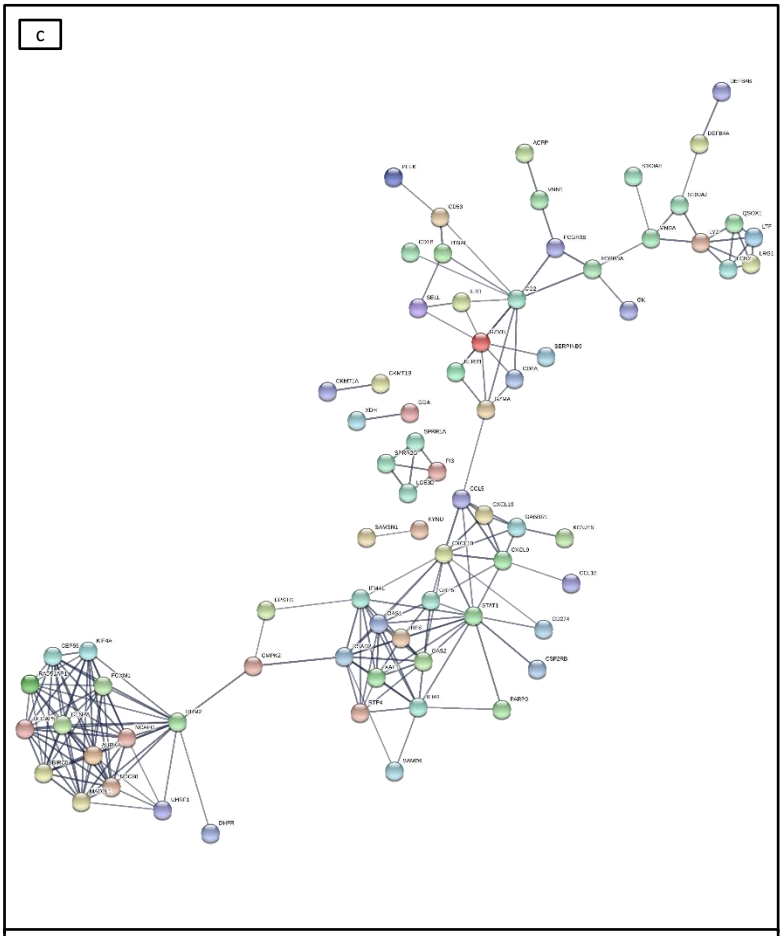
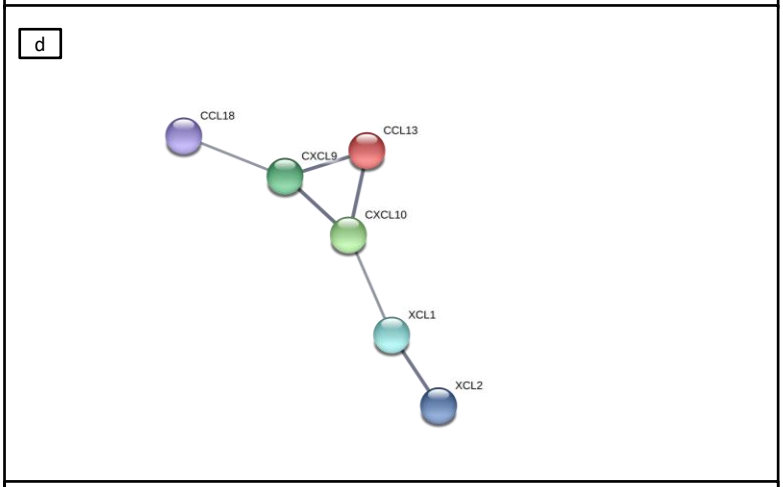
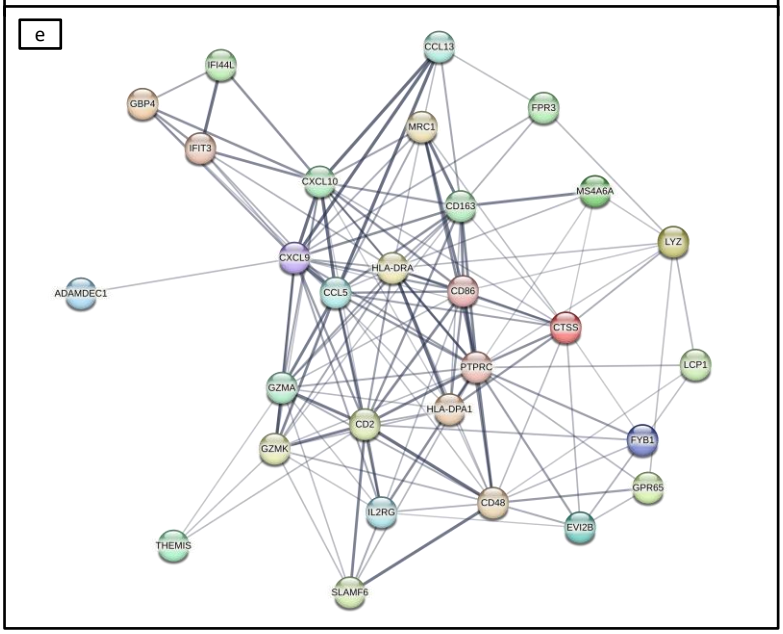


Fig. S3 Protein-protein interaction of the upregulated genes common between concurrent skin diseases, such as **(a)** psoriasis and vitiligo, showing the interaction between upregulated genes in psoriasis (PI3 and LCE3D) and those involved in the immune system (CCL18, CXCL9, TNF, IL36G, and IL36RN) in the upper right corner; **(b)** lichen planus and psoriasis, showing a predominance of those involved in the immune system and cell cycle;







Cont. Fig. S3 Protein-protein interaction of the upregulated genes common between concurrent skin diseases, such as **(c)** psoriasis and alopecia areata, showing the close interaction between the top upregulated genes in psoriasis (DEFB4 and S100A7A) and alopecia areata (*ACPP and* VNN1) in the upper right corner; **(d)** vitiligo and alopecia areata; and **(e)** lichen planus and alopecia.