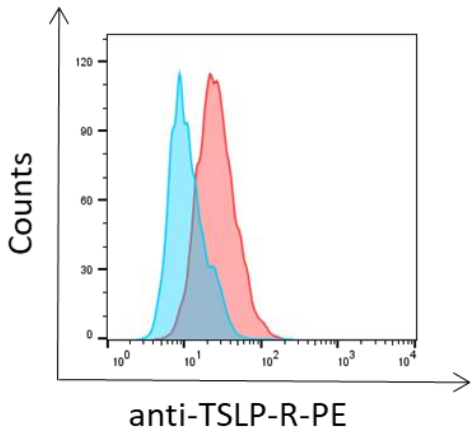

Figure S1. Representative flow cytometry histogram of TSLP-R surface expression on cultured skin MCs (representative histogram out of 3 independent stainings); blue: Isotype control; red: anti-TSLP-*R*.

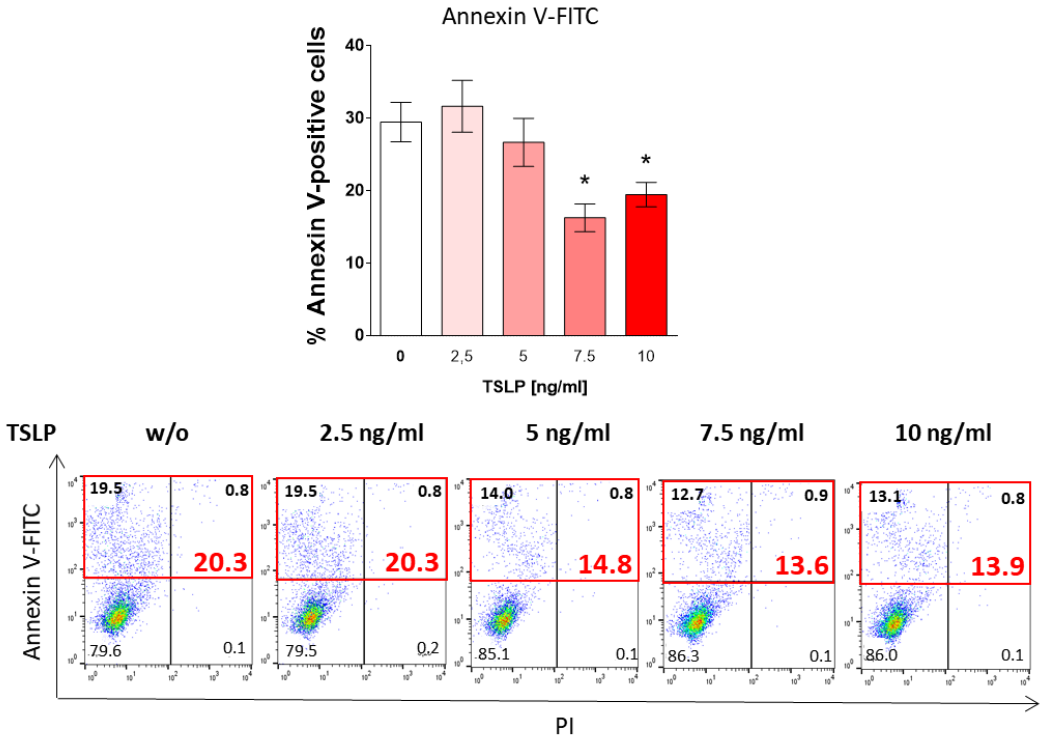

Figure S2. TSLP dose-dependently inhibits phosphatidylserine externalization in GF-deprived MCs. Impact of TSLP on the survival of skin MCs in serum/GF-free medium, as assessed by annexin V-FITC positivity (corresponding to the percentage of early and late apoptotic cells/necrotic combined) after 48h. Top: The results represent the mean ± SEM of 5 independent experiments; bottom: Representative flow cytometry dot plots (specified in red is the percentage of early and late apoptotic cells combined). The data were analyzed by One-way Anova with Turkey's post-test for multiple comparisons, comparing each treatment with the untreated control group, *$p < 0.05$.

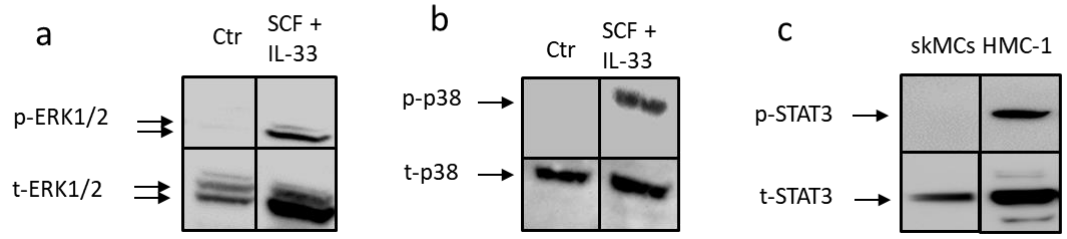

Figure S3. STAT3 is activated in HMC-1 cells, and in contrast to TSLP, the combination of SCF and IL-33 triggers phosphorylation of ERK1/2 and p38. To exclude that our inability to detect several phosphorylated signaling components was due to technical issues, Western blots were run on positive controls (shown are representative Western blots out of 3 independent experiments). Skin MCs stimulated with a combination of SCF [10 ng/ml] and IL-33 [20 ng/ml] served to detect pERK (strongly stimulated by SCF) and pp38 (strongly stimulated by IL-33) [31] , and cells of the HMC-1 cell line (exhibiting mutations in KIT, which confer activity in the absence of ligand) were employed to detect pSTAT3 [43]. skMCs – cultured skin mast cells (unstimulated).

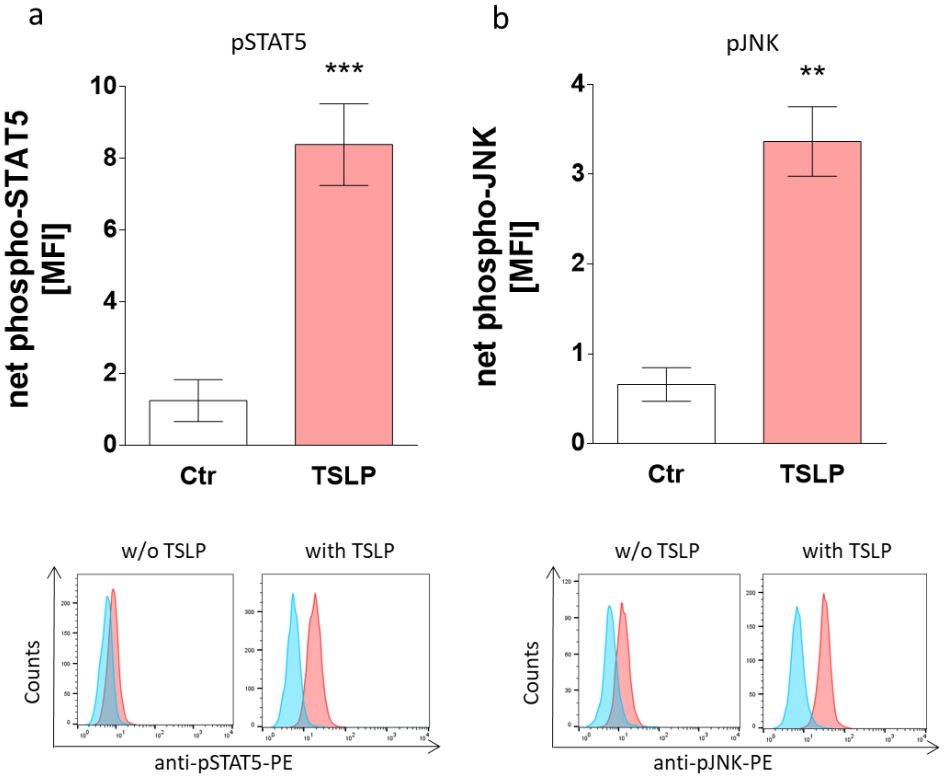

Figure S4. Phosphorylation of STAT5 and JNK by TSLP by flow cytometry. Impact of TSLP on proximal signaling intermediates after 30 min, evaluated by flow cytometry in permeabilized MCs, using primary antibodies against **a)** pSTAT5 and **b)** pJNK (and PE-labelled secondary antibodies). Top: The results represent the mean ± SEM of 5 independent experiments; shown is the net Mean Fluorescence Intensity (MFI). Bottom: Representative flow cytometry histograms; blue: Isotype control, red: anti-target antibody; w/o – without. The data were analyzed by paired t-test, $^{**}p < 0.01$, $^{***}p < 0.001$.

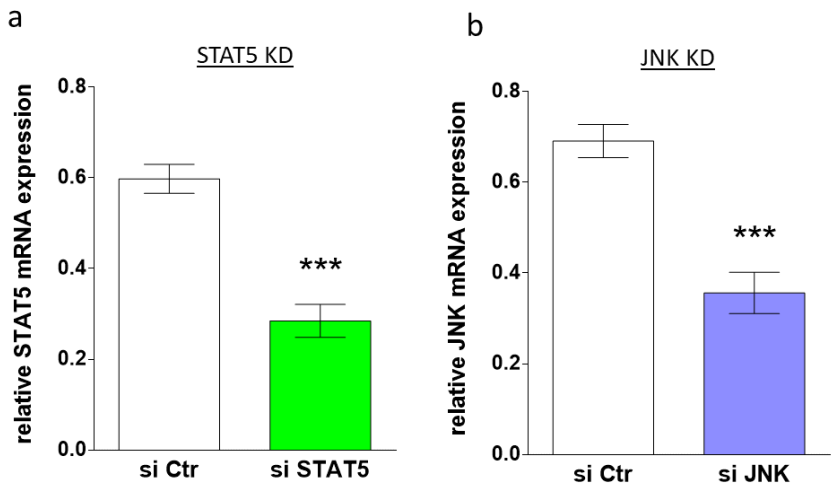

Figure S5. Knockdown efficiency of STAT5 and JNK. Confirmation of **a)** STAT5 and **b)** JNK knockdown by RT-qPCR analysis after 48h (normalized to Cyclophilin B). The results represent the mean ± SEM of 5 independent experiments. The data were analyzed by paired t-test, ***$p < 0.001$.

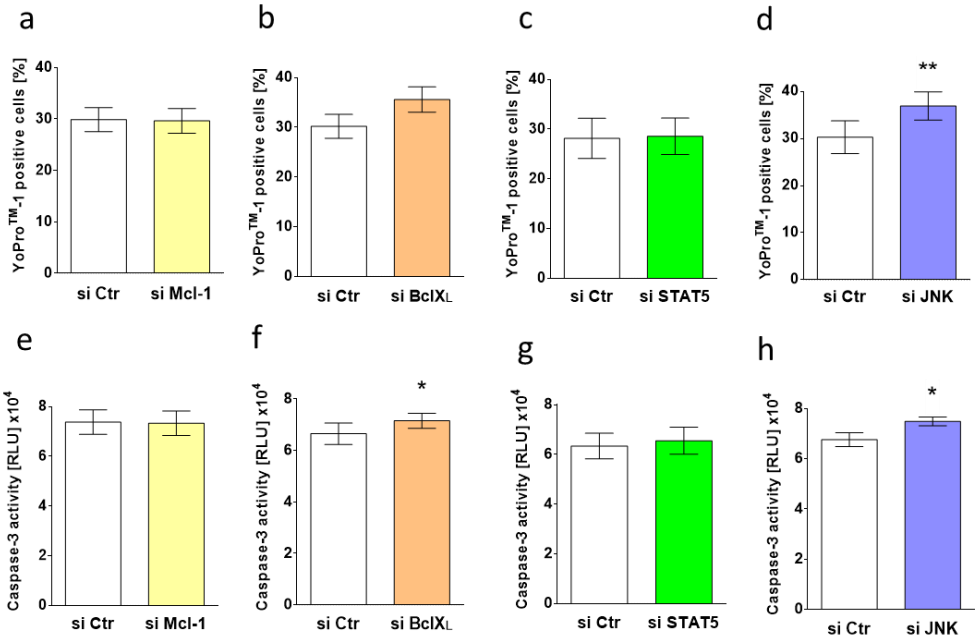

Figure S6. Influence of Mcl-1, Bcl-x_L, STAT5 and JNK on MC survival in the absence of TSLP. Effect of **a, e)** Mcl-1, **b, f)** Bcl-x_L, **c, g)** STAT5 and **d, h)** JNK knockdown on baseline survival in the absence of TSLP, evaluated by **a - d)** YoPro™-1 positivity and **e - g)** caspase-3 activity. The results represent the mean ± SEM of 5 independent experiments, and were analyzed by paired t-test, *$p < 0.05$, **$p < 0.01$.

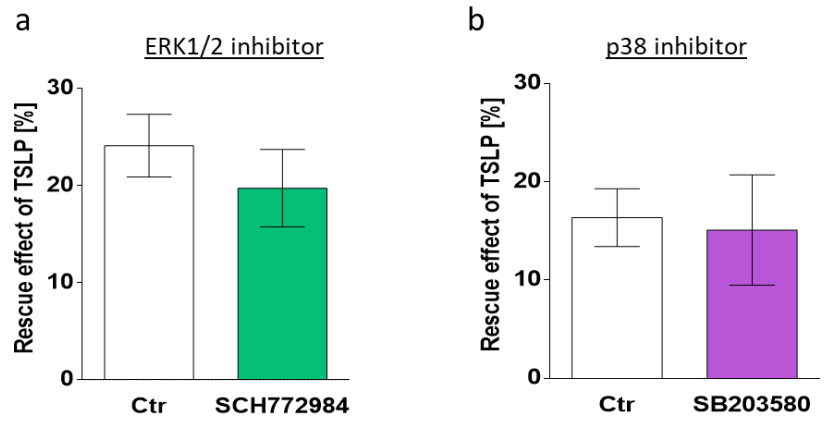

Figure S7. ERK1/2 and p38 are not implicated in TSLP-induced survival rescue. Impact of **a)** ERK1/2 and **b)** p38 inhibition on TSLP-promoted MC recovery, evaluated by the ratio of caspase-3 activity in TSLP-treated vs. untreated MCs. RLU = Relative Luminescence Units. The results represent the mean ± SEM of 6 independent experiments.

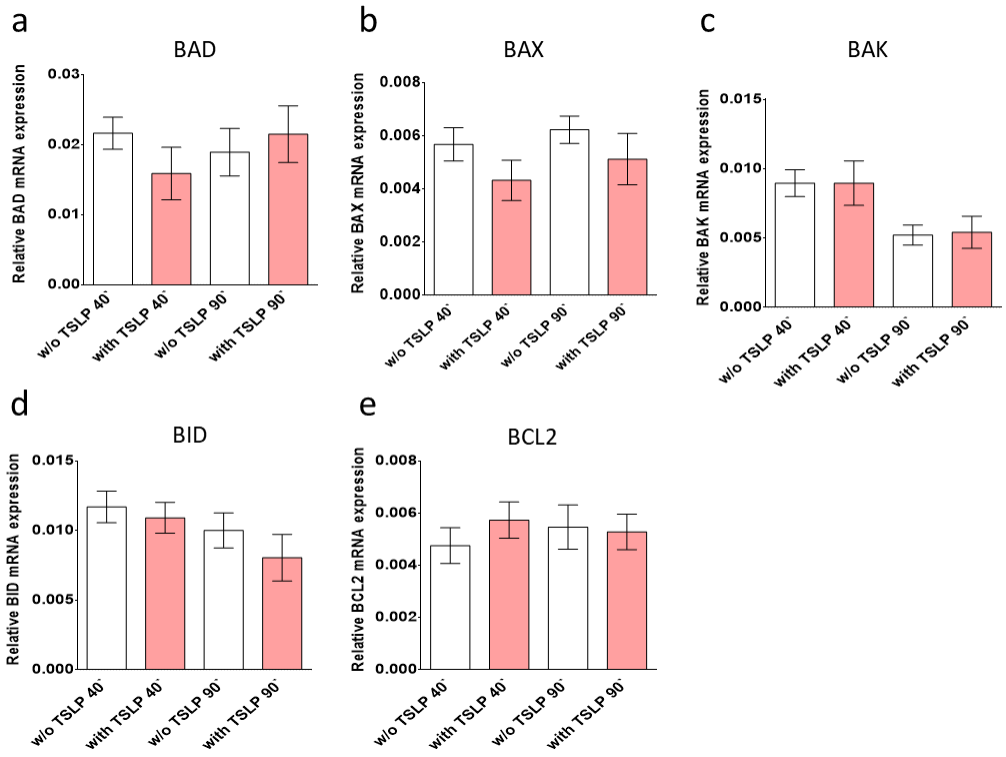

Figure S8. TSLP does not affect the expression of several Bcl-2 family members. Expression was studied by RT-qPCR analysis (exactly as in Figure 3a) of **a)** Bad, **b)** Bax **c)** Bak, **d)** Bid and **e)** Bcl-2 and was normalized to the expression of Cyclophilin B. The results represent the mean ± SEM of 9 independent experiments.

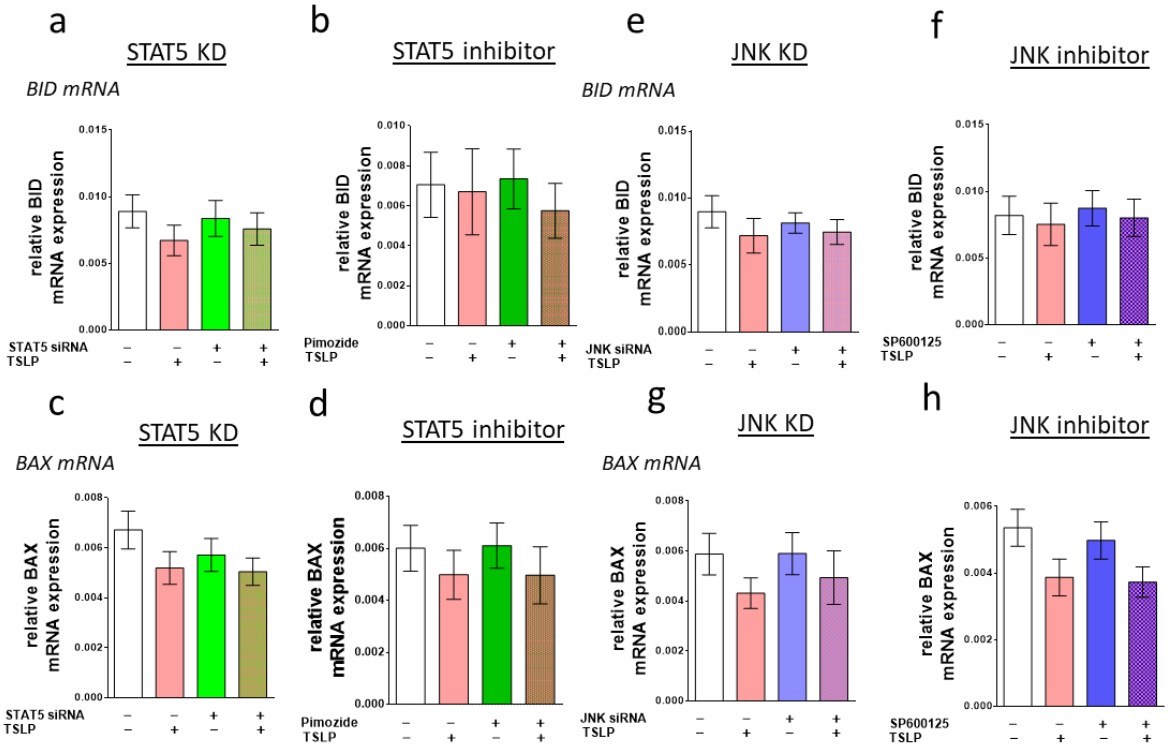

Figure S9. Perturbation of STAT5 and JNK activity leaves expression of Bid and Bax unaffected. Impact of **a - d)** STAT5 and **e - g)** JNK perturbation on TSLP-triggered Bcl-2 family member expression. **a, b, e, f)** Bid and **c, d, g, h)** Bax levels after 40 min, using **a, c, e, g)** knockdown by RNAi (48h prior to the experiment), and **b, d, f, h)** pre-incubation with specific inhibitors (STAT5 inhibitor: pimozide used at 5 μM, JNK inhibitor: SP600125 used at 5 μM), evaluated by RT-qPCR analysis and normalized to Cyclophilin B. The results represent the mean ± SEM of 5 (RNAi) or 6 (inhibitors) independent experiments.

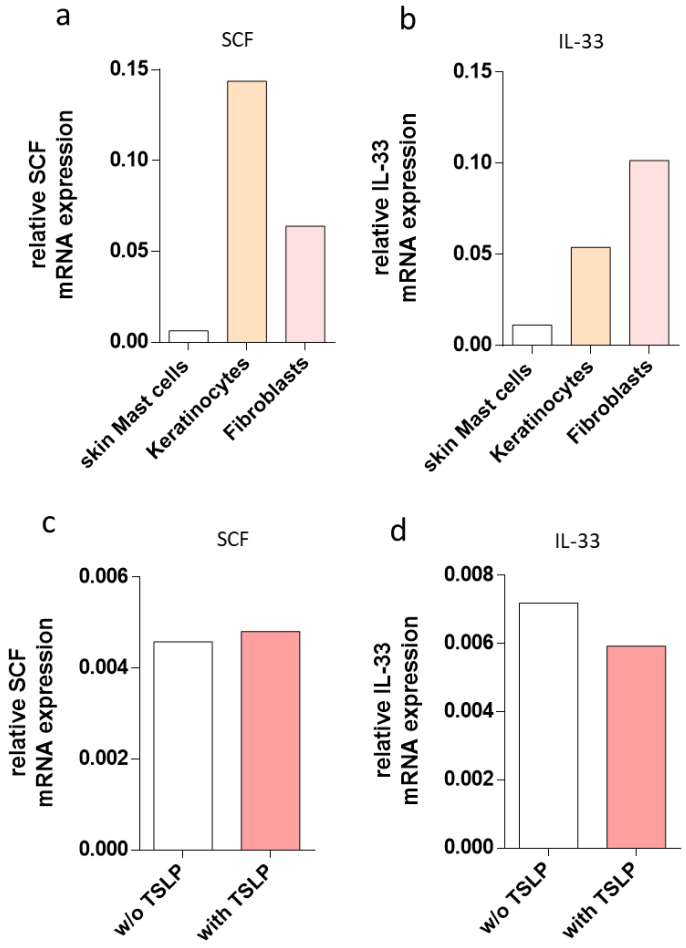

Figure S10. Basal and TSLP-treated human skin MCs are poor producers of SCF and IL-33 in comparison to keratinocytes and fibroblasts – no regulation by TSLP. SCF and IL-33 mRNA levels in **a, b)** untreated skin MCs vs. keratinocytes vs. fibroblasts, **c, d)** untreated vs. TSLP-treated skin MCs, as evaluated by RT-qPCR after 2h. The data were normalized to the housekeeping gene Cyclophilin B. One experiment of n=2 is shown, both with similar outcomes. w/o – without