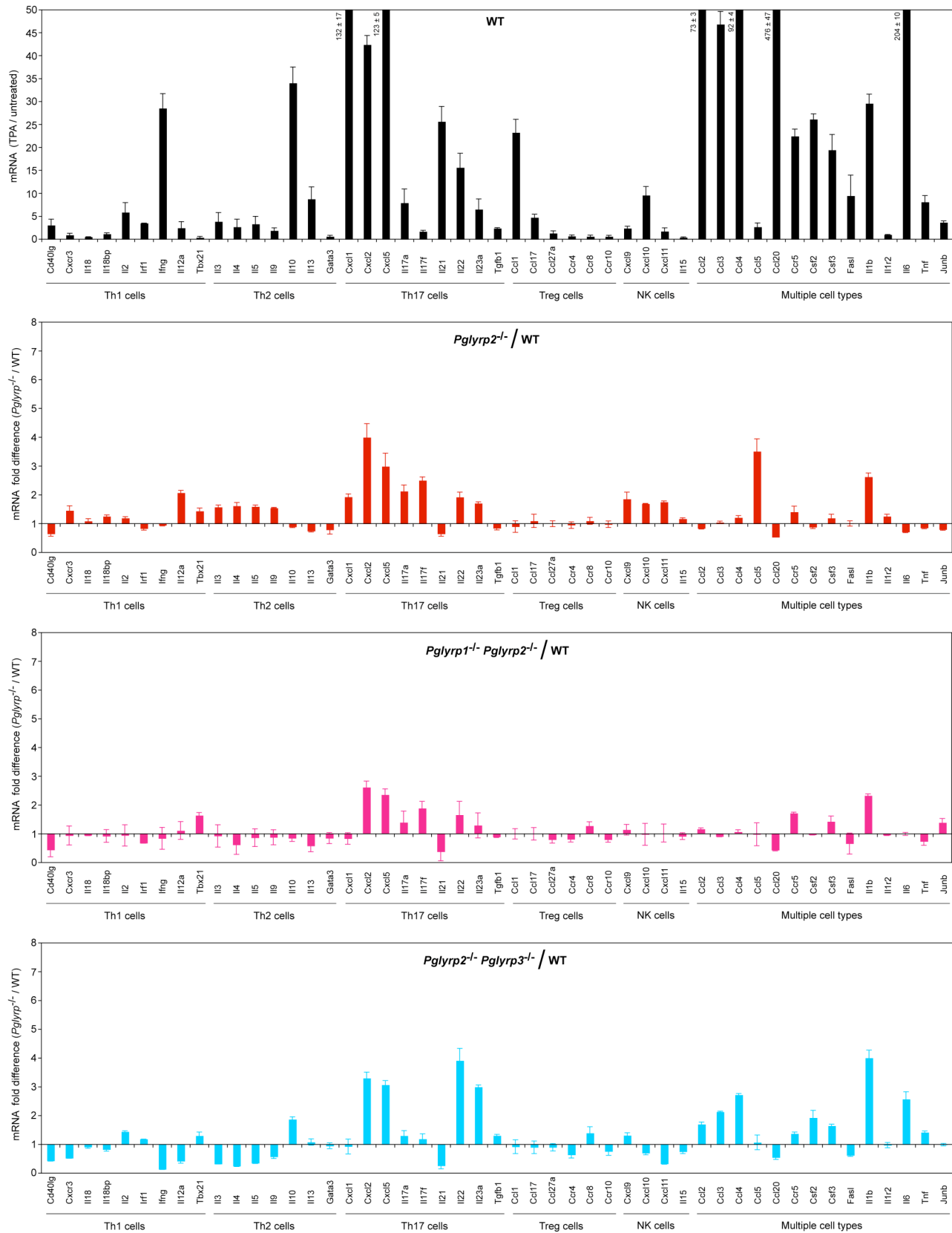
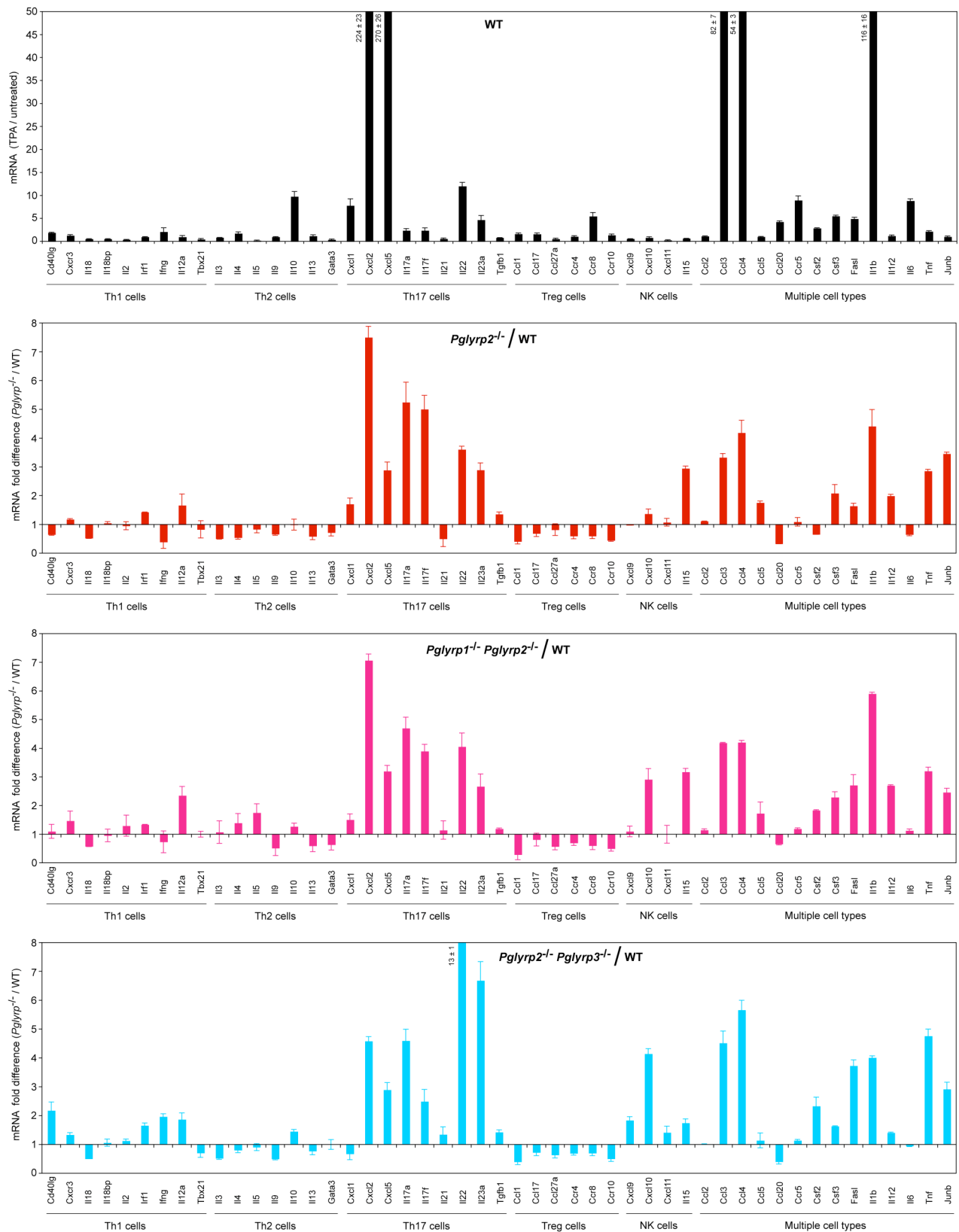


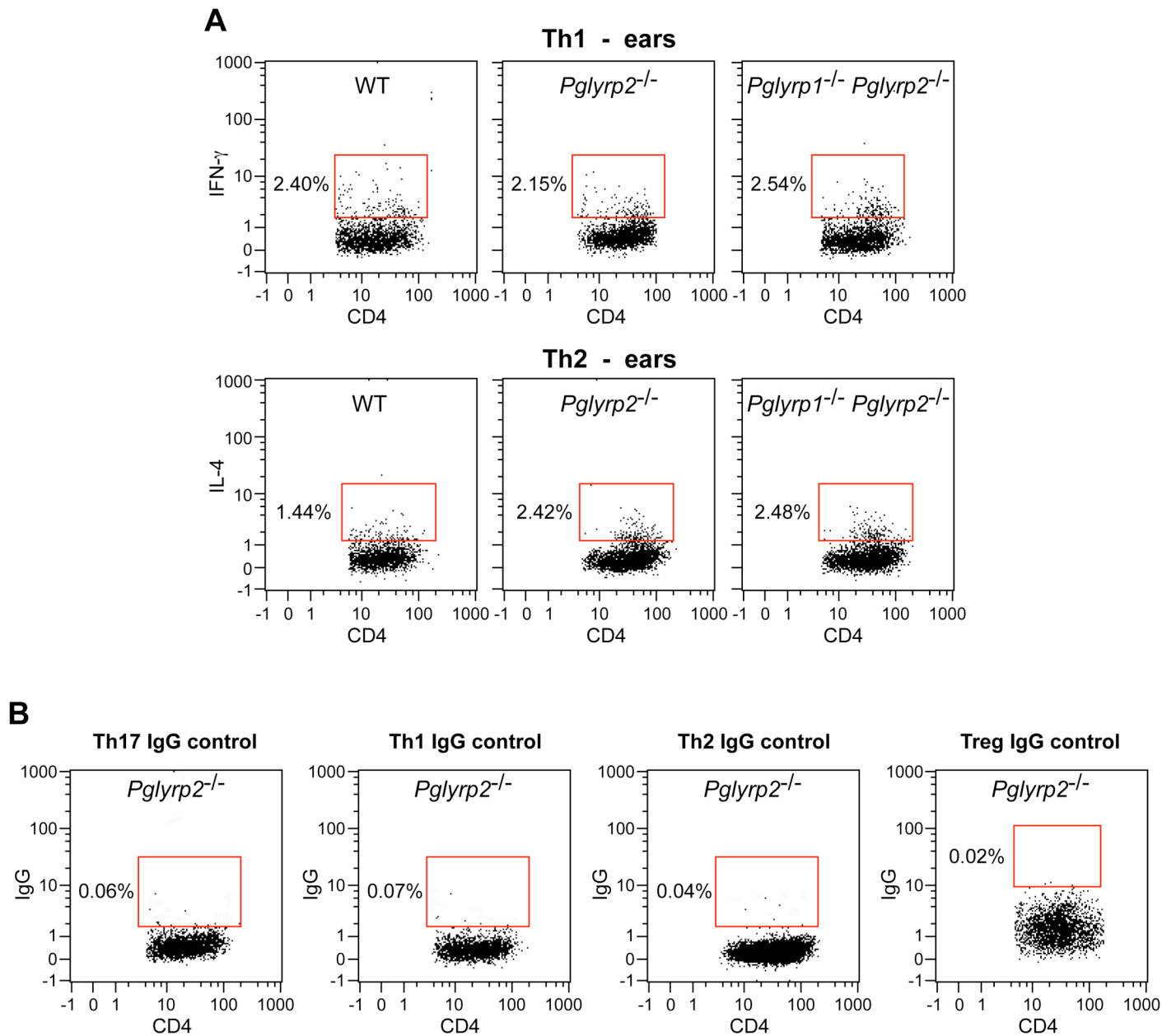
Supplemental Figure 1. TPA-treated *Pglyrp2*^{-/-} mice have increased PMNs, monocytes, B cells, and Th17 cells in the affected skin. Expression of a panel of marker genes characteristic of various inflammatory cell types in the ears of mice on day 9 after application of TPA to the ears on days 0, 2, 4, 6, and 8 measured by qRT-PCR is shown. For WT mice (top panel), the ratio of the amount of mRNA in TPA-treated to untreated mice for each gene (fold induction by TPA) is shown; for *Pglyrp2*^{-/-} mice, the results are the ratios of fold induction of each gene by TPA in *Pglyrp2*^{-/-} mice to fold induction of each gene by TPA in WT mice (which represents the fold difference in the response to TPA in *Pglyrp2*^{-/-} versus WT mice). The results are means ± SEM of 3 arrays from 4–5 mice/group and are shown as heat maps in Fig. 4A in the main article.



Supplemental Figure 2. Multiple inflammatory and immune genes are induced early in TPA-treated skin. Expression of a panel of cytokines, chemokines, and other marker genes characteristic of Th1, Th2, Th17, Treg, NK, and other cell types in the ears of mice 6 hrs after a single application of TPA to the ears measured by qRT-PCR. For WT mice (top panel), the ratio of the amount of mRNA in TPA-treated to untreated mice for each gene (fold induction by TPA) is shown; for *Pglyrp2^{-/-}* mice the results are the ratios of fold induction of each gene by TPA in *Pglyrp2^{-/-}* mice to fold induction of each gene by TPA in WT mice (which represents the fold difference in the response to TPA in *Pglyrp^{-/-}* versus WT mice). The results are means ± SEM of 3 arrays from 4–5 mice/group and are shown as heat maps in Fig. 4B in the main article.



Supplemental Figure 3. Th17 gene expression profile is preferentially induced by TPA in the affected skin of *Pglyrp2^{-/-}* mice. Expression of a panel of cytokines, chemokines, and other marker genes characteristic of Th1, Th2, Th17, Treg, NK, and other cell types in the ears of mice on day 9 after application of TPA to the ears on days 0, 2, 4, 6, and 8 shows higher induction of several Th17 marker genes in *Pglyrp2^{-/-}* mice compared to WT mice (in addition to genes characteristic of many cell types) measured by qRT-PCR. For WT mice (top panel), the ratio of the amount of mRNA in TPA-treated to untreated mice for each gene (fold induction by TPA) is shown; for *Pglyrp2^{-/-}* mice, the results are the ratios of fold induction of each gene by TPA in *Pglyrp2^{-/-}* mice to fold induction of each gene by TPA in WT mice (which represents the fold difference in the response to TPA in *Pglyrp2^{-/-}* versus WT mice). The results are means ± SEM of 3–4 arrays from 4–5 mice/group and are shown as heat maps in Fig. 4B in the main article.



Supplemental Figure 4. A. Representative dot plots of Th1 and T2 cells in the ears of WT, *Pglyrp2^{-/-}* and *Pglyrp1^{-/-}Pglyrp2^{-/-}* mice on day 9 after application of TPA to the ears on days 0, 2, 4, 6, and 8 measured by flow cytometry. The means \pm SEM of 5–9 mice/group are shown in Fig. 5A in the main article. **B.** Representative dot plots with ear CD4⁺ lymphocytes from *Pglyrp2^{-/-}* mice after 9 days of treatment with TPA as above in A, stained with isotype control IgG for IL-17 Ab (Th17 cells), IFN- γ Ab (Th1 cells), IL-4 Ab (Th2 cells), or Foxp3 Ab (Treg cells).