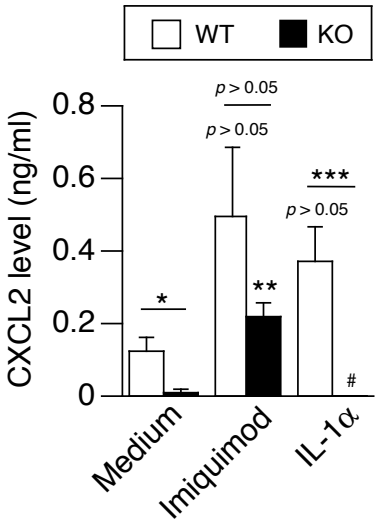
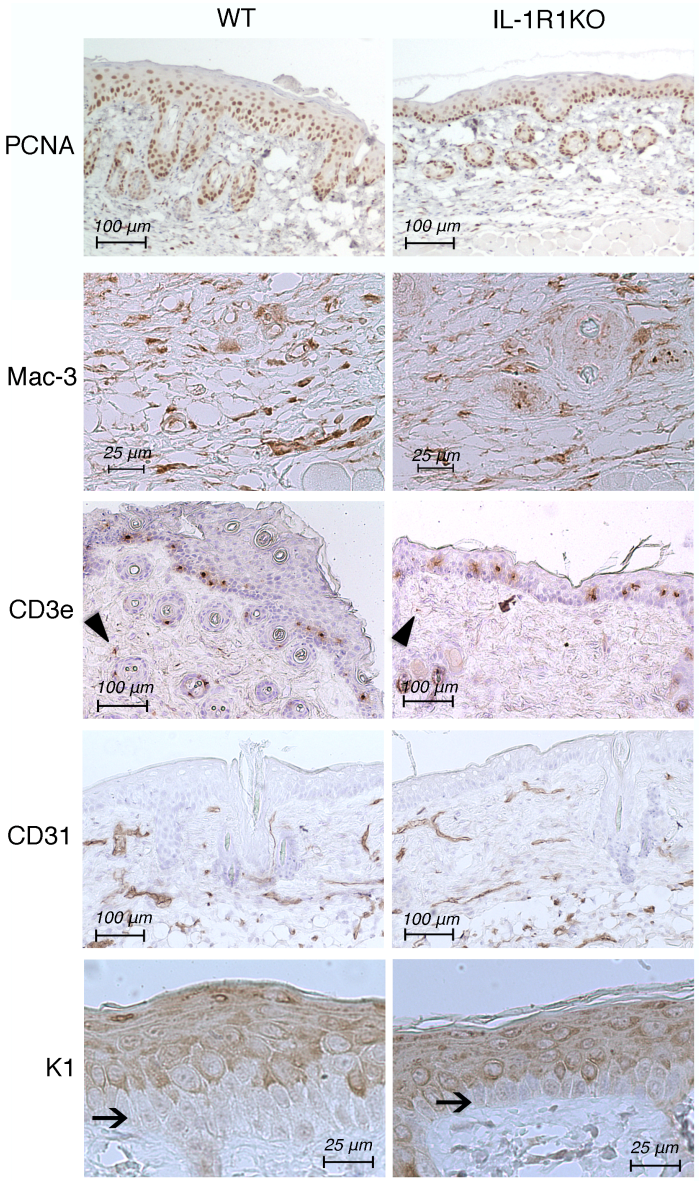
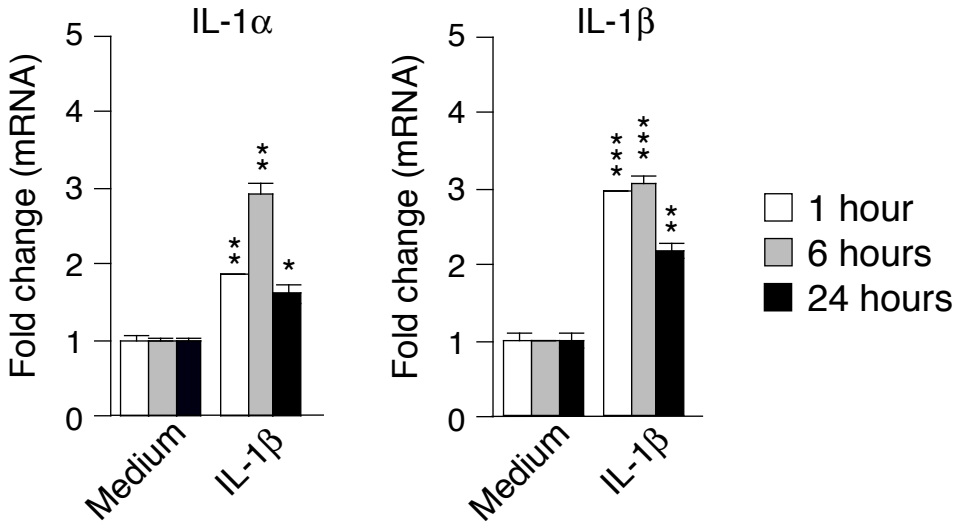


Supplementary Figure S1. IL-1R1 deficient cells constitutively express less chemokines *in vitro*.

Mouse primary keratinocytes isolated from wild type (WT, white bars) or IL-1R1 KO mice (KO, black bars) were treated with medium only, 50 μg/ml imiquimod or 10 ng/ml mouse IL-1α for 24 hours. Levels of CXCL2 were determined using ELISA (mean ± SD). *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$ (compared to medium only or as indicated). # Low levels of CXCL2 were detected.



Supplementary Figure S2. Epidermal, angiogenic and inflammatory phenotypes are differentially regulated by IL-1R1 *in vivo*. Wild type (WT) and IL-1R1 KO mice were treated with imiquimod as described in Figure 4c. Using immunohistochemistry skin sections were examined for presence of Proliferating Cell Nuclear Antigen (PCNA, Invitrogen), macrophages (Mac-3, monoclonal M3/84, BD Pharmingen), T cells (CD3e, monoclonal 500A2, BD Pharmingen), blood vessels (endothelial marker CD31, monoclonal MEC 13.3, BD Pharmingen) and keratin 1 (polyclonal AF 109, Covance). Large arrows and arrowheads indicate positions of proliferating keratinocytes that do not express keratin 1 and dermal CD3e positive cells, respectively.



Supplementary Figure S3. IL-1 stimulates expression of IL-1α and IL-1β mRNAs *in vitro*. Human primary keratinocytes were treated with medium only or IL-1β (10 ng/ml, PeproTech) for 1 (white bars), 6 (grey bars) or 24 hours (black bars). Levels of IL-1α and IL-1β mRNAs were determined using real-time RT-PCR. Levels of mRNA are represented as fold change (mean ± SD) compared to medium only treated samples at the same time-point. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$ (compared to medium only at the same time-point).