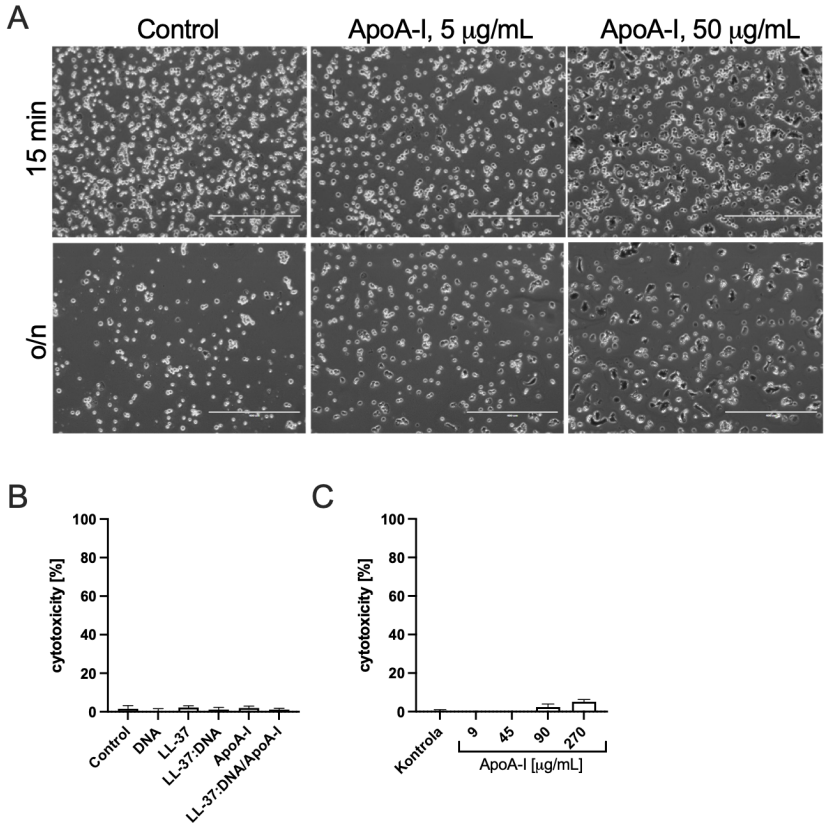
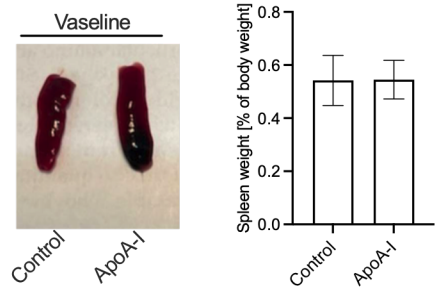
Supplementary Figure 1



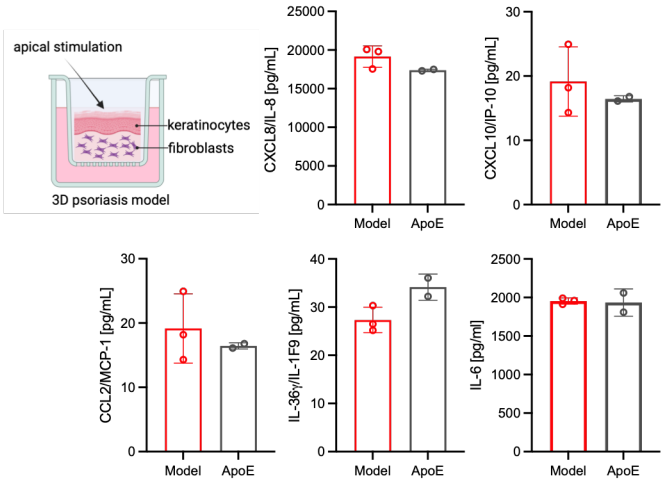
Supplementary Figure 1. Assessment of ApoA-I cytotoxicity toward pDCs and HEKn cells. (A) pDCs were exposed to ApoA-I at concentrations of 5 and 50 μg/mL for 15 min or o/n. Following incubation, ApoA-I was removed by washing, and pDCs were cytocentrifuged onto microscope slides, stained with trypan blue, and assessed for cell viability. (B) Additionaly, pDCs were treated with DNA, LL-37, LL-37:DNA, ApoA-Iand LL-37:DNA/ApoA-I for 16 h. After incubation, cell culture supernatants were collected and used for quantification of lactate dehydrogenase (LDH) release by colorimetric assay. (C) To evaluate the potential cytotoxic effects of ApoA-I on HEKn were treated with ApoA-I at concentrations of 9, 45, 90, and 270 μg/mL for 4 h. Following incubation, culture supernatants were collected and LDH levels were determined using a colorimetric assay.

Supplementary Figure 2



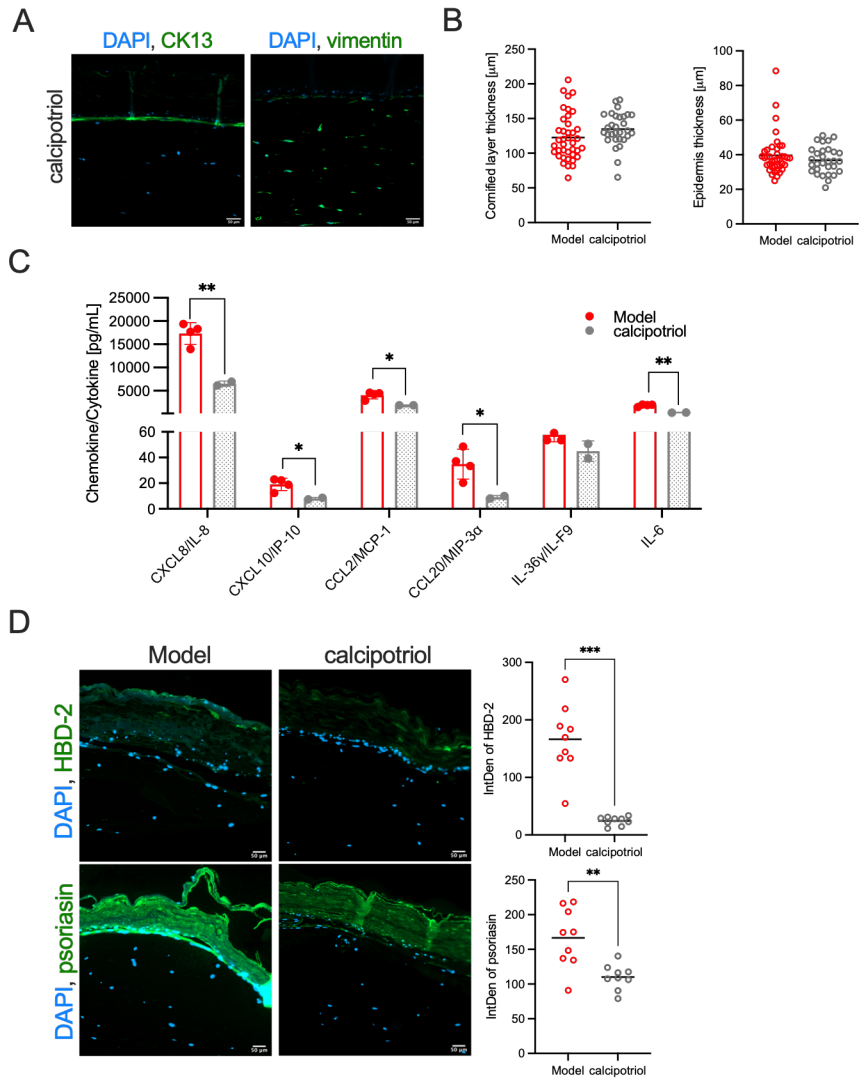
Supplementary Figure 2. Lack of splenomegaly following ApoA-I treatment. Representative images of spleens from control and IMQ-treated mice with or without ApoA-I administration (i.d. or i.p.). Quantification of spleen weight normalized to body weight is shown on the right. No significant increase in spleen size or weight was observed upon ApoA-I treatment, indicating the absence of splenomegaly. Data are presented as mean ± SEM. Statistical significance was evaluated by one-way ANOVA followed by Šídák's multiple comparisons test (**p < 0.01).

Supplementary Figure 3



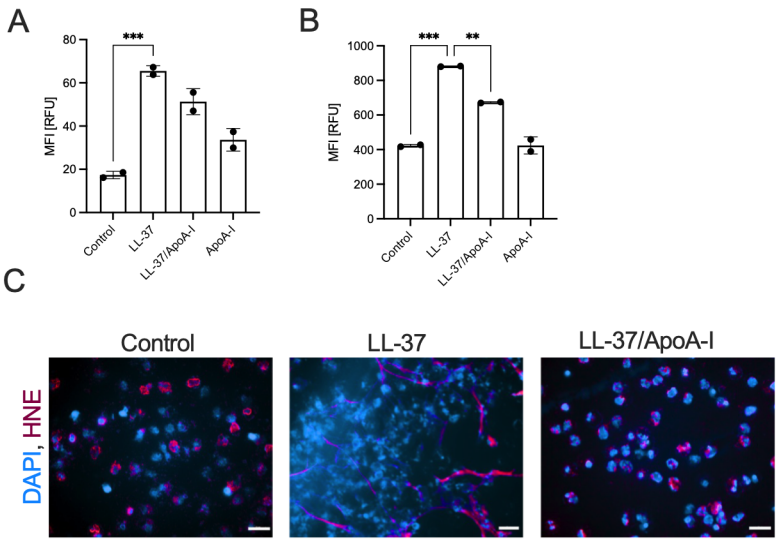
Supplementary Figure 3. ApoE does not modulate chemokine and cytokine production in the 3D psoriasis model. Inflammatory chemokines and cytokines were assayed with a cytokine bead array (CBA) kit. The conditioned media of 3D psoriasis tissue model were quantified after being treated with ApoE from the apical side of insert (model $n = 3$; ApoE $n = 2$; mean ± SD). Statistical significance was evaluated by t test.

Supplementary Figure 4



Supplementary Figure 4. Effects of calcipotriol in a human 3D psoriasis model. (A) Calcipotriol does not affect the expression of cytokeratin 13 and vimentin, as assessed by immunofluorescence. (B) Calcipotriol does not alter the thickness of the cornified layer or the total epidermis thickness. Thickness measurements were performed using ImageJ software and are presented as mean values. (C) Inflammatory chemokines and cytokines were quantified using a cytokine bead array (CBA) kit. Conditioned media of the 3D psoriasis tissue model were collected following apical treatment with calcipotriol (n = 4 or 2; mean ± SD). (D) Immunofluorescence detection of psoriasis-associated biomarkers, including human β-defensin-2 (HBD-2), psoriasin/S100A7 (green). Cell nuclei were counterstained with DAPI (blue). Scale bar: 50 µm. Green fluorescence was quantified using ImageJ software and is presented as mean ± SEM. Statistical significance was evaluated by one-way ANOVA followed by Šídák's multiple comparisons test (*p < 0.1; **p < 0.01; ***< 0.0001).

Supplementary Figure 5



Supplementary Figure 5. Effect of ApoA-I on NET formation. Quantification of extracellular DNA release from neutrophils stimulated with LL-37 (1 µg/mL) and/or ApoA-I (30 µg/mL) immediately after isolation (A) or after 16 h of culture (B). Cells were stimulated for 3 h. Data are presented as the mean ± SD from a single experiment performed in duplicates. Statistical significance was conducted using one-way ANOVA followed by Bonferroni's multiple comparisons test (ns, not significant; **p < 0.01; ***p < 0.001). (C) Representative images of neutrophil extracellular traps (NETs). Neutrophils were stained with antibodies against neutrophil elastase (HNE, red) and nuclei/chromatin were visualized using DAPI (blue). Scale bar, 20 µm.