Figure S1

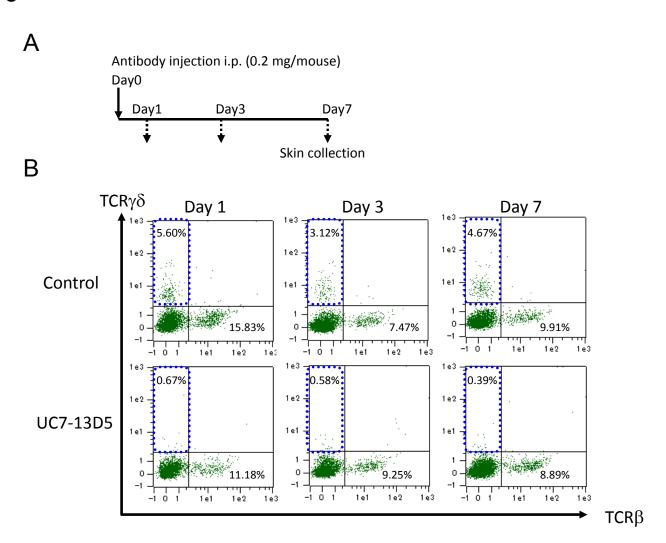


Figure S1. In vivo depletion of $\gamma\delta$ T cells by anti-TCR $\gamma\delta$ (clone UC7-13D5 polyclonal antibody. (A) Schematic representation of the experimental plan. Solid allows indicate timepoint (days) at which the intraperitoneal injection of the antibody were performed. (B) The efficacy of $\gamma\delta$ T-cell depletion was confirmed through an FCM analysis of the skin samples. The administration of anti-TCR $\gamma\delta$ antibody was successful in maintaining a significant decrease in the TCR $\gamma\delta$ -positive $\gamma\delta$ T cells but not TCR β -positive $\alpha\beta$ T cells for 7 days after the antibody administration.

Figure S2

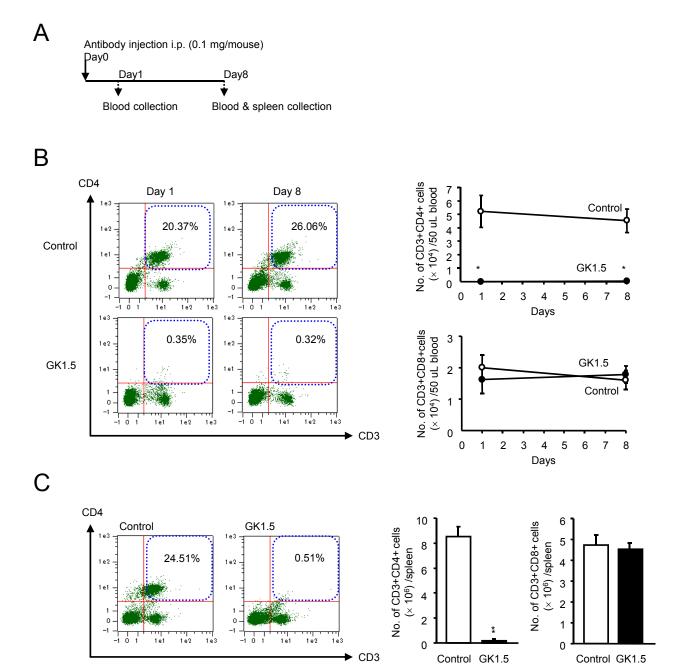


Figure S2. In vivo depletion of CD4-positive T cells by anti-CD4 (clone GK1.5) monoclonal antibody. (A) Schematic representation of the experimental plan (A). Solid allows indicate time-point (days) at which intraperitoneal injection of the antibody were performed. The efficacy of CD4-positive T cell depletion was confirmed by FCM analysis of T cell population in the peripheral blood (B) and spleen (C). FCM analysis confirmed that anti-CD4 antibody treatment effectively reduced the number of CD3+CD4+ T cells but not CD3+CD8+ T cells in the peripheral blood and spleen of the treated mice compared to the control mice. * $P < 0.05 \ vs.$ control; $t = 1.5 \ vs.$ con

Figure S3

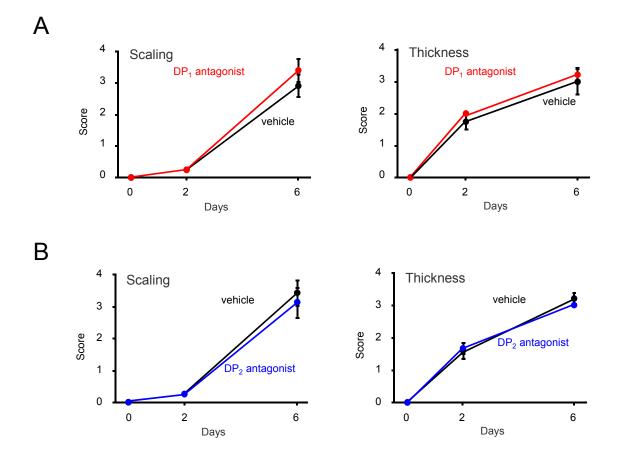


Figure S3. Effect of pharmacological DP receptor inhibition on facilitated psoriasis under the condition of mPGES-1 deficiency. Given that PGD₂ production was increased in the skin of mPGES-1^{-/-} mice after the induction of psoriasis, we investigated the effects of PGD₂ in the IMQ-induced psoriasis pathology under the condition of mPGES-1 deficiency using antagonists specific for each DP subtype DP₁ and DP₂. (A) DP₁ antagonist BWA868C (Cayman) was administered intraperitoneally to the mPGES-1-/- mice at a dose of 1 mg/kg daily for 6 days immediately before the IMQ treatment, and the psoriasis pathology was evaluated. There was no significant difference in the skin scaling and thickening scores between mPGES-1^{-/-} mice treated with a DP₁ antagonist and mPGES-1^{-/-} mice treated with a vehicle (n=6-9). (B) The DP₂ antagonist CAY10471 (Cayman) was administered intraperitoneally to the mPGES-1-/-mice at a dose of 2 mg/kg daily for 6 days immediately before the IMQ treatment. Similarly, there was no significant difference in both scores between the DP₂ antagonist and vehicle control in mPGES-1^{-/-} mice (n=8–9). These results suggest that PGD₂, which is increased in the skin of psoriasis-induced mPGES-1^{-/-} mice, is unlikely to exacerbate or alleviate the major symptoms of IMQ-induced psoriasis. *P < 0.05; ANOVA followed by Tukey's multiple comparison test.