SUPORTING INFORMATION

Supporting figures

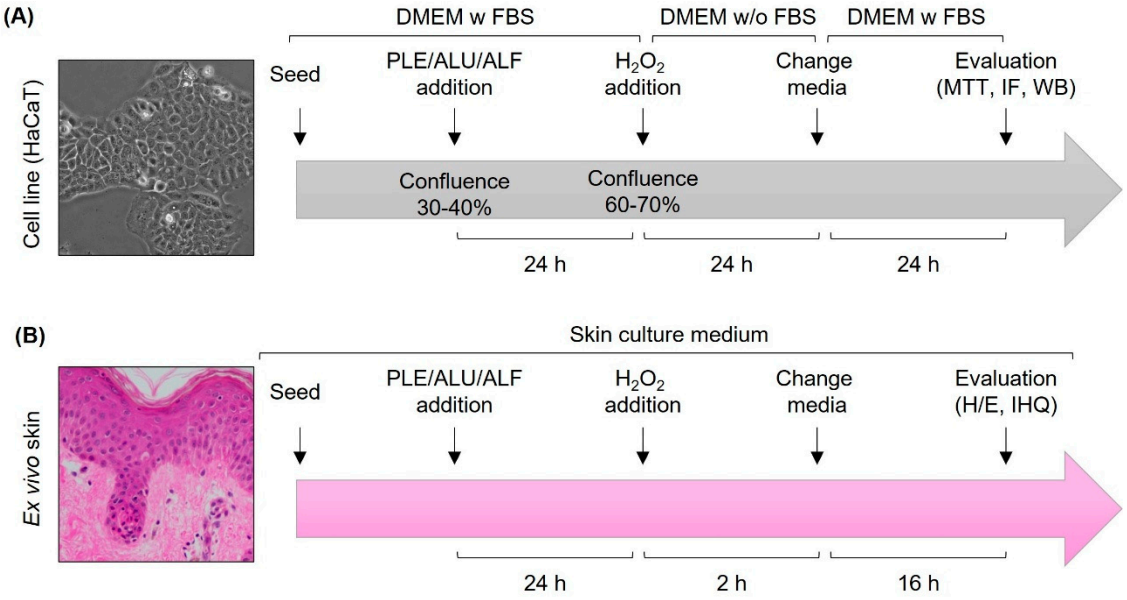


Figure S1. Schematic diagram of the time course treatment. (A) HaCaT cell line and **(B)** *ex vivo* skin.

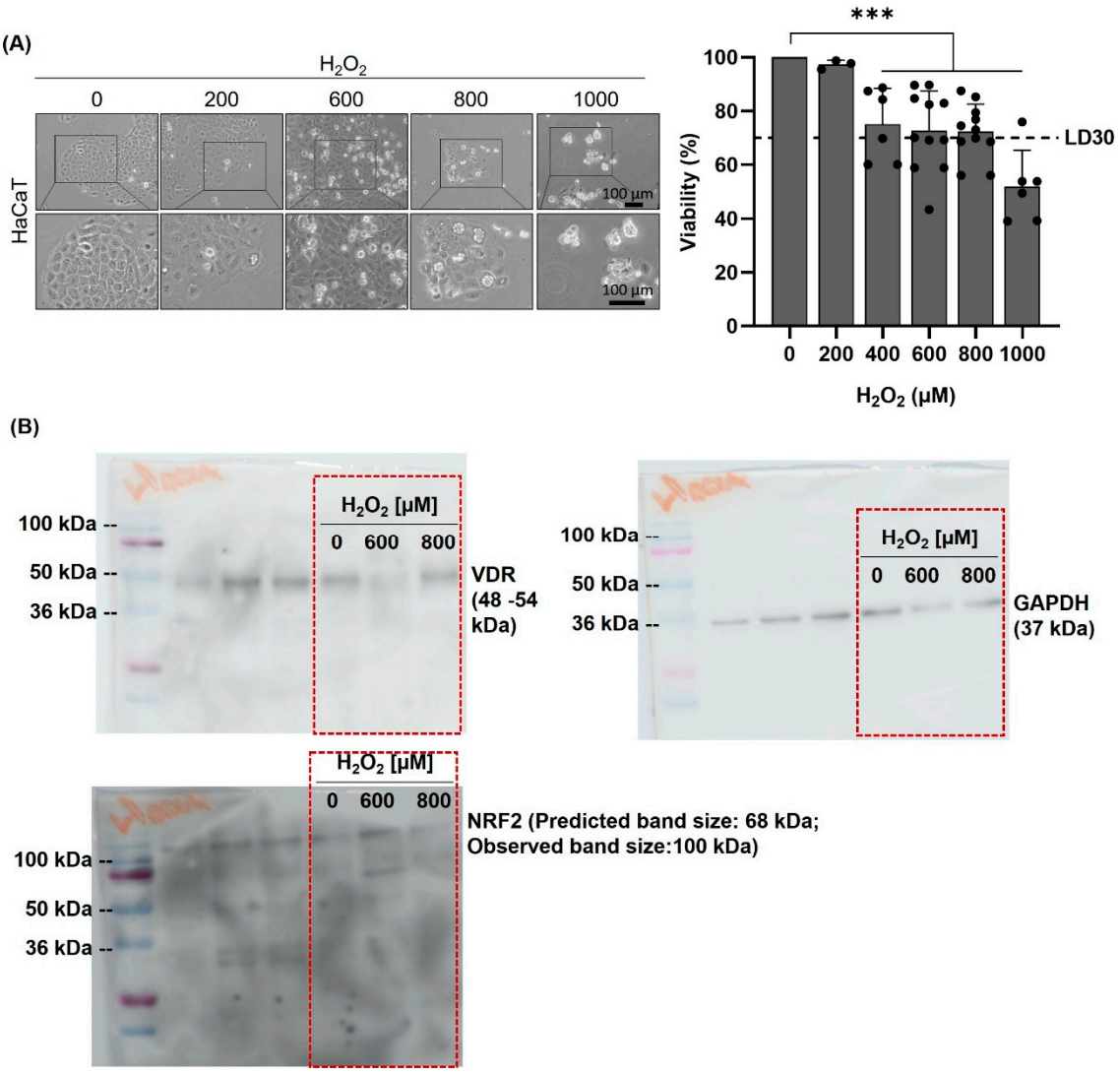


Figure S2. Effect of oxidative stress on HaCaT cells. A) Cell morphology and viability assay (relative to untreated cells) under OS induced by H_2O_2 (0-1000 µM). In the bar graph, sample values are represented by overlapping dots, and the height of the bars represents the mean of each group ± SD (***: $p < 0.001$; $n \geq 3$). **B)** VDR, NRF2, and GAPDH protein levels by western blot. Original membrane for the representation of Figure 1C.

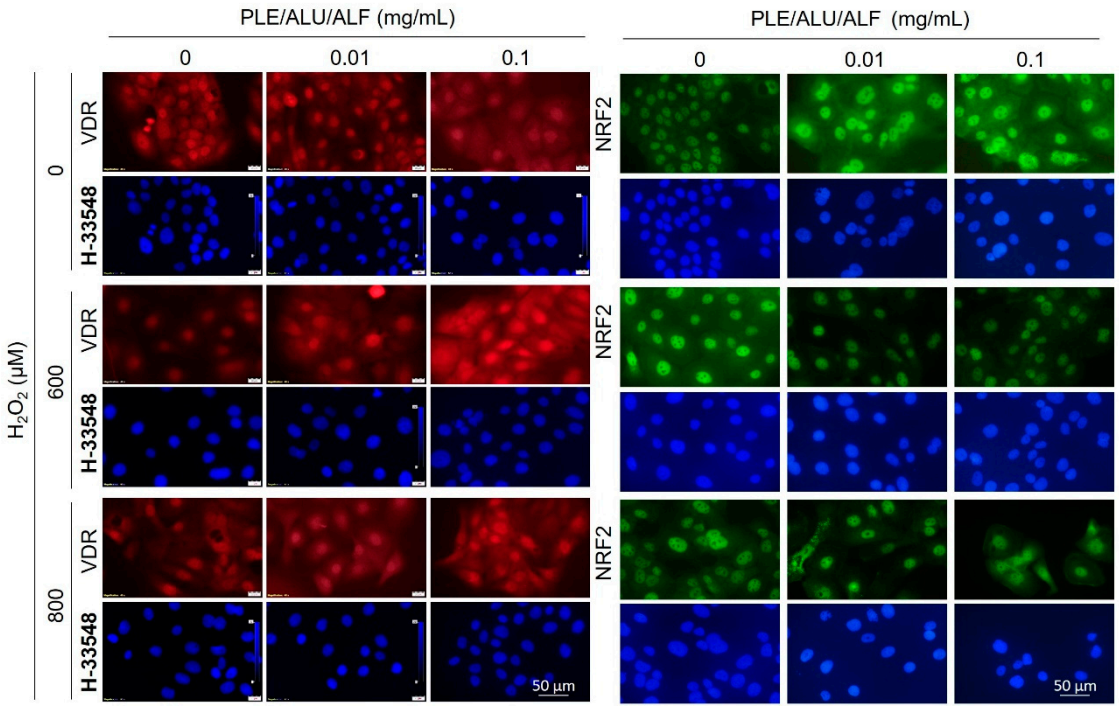


Figure S3. PLE/ALU/ALF protects from oxidative stress-induced VDR repression. Effect of PLE/ALU/ALF (0.01 and 0.1 mg/ml), H_2O_2 (600 and 800 μM), and their combination on VDR and NRF2 expression. Full immunofluorescence panel corresponding to Figure 3, including Höechst nuclear counterstaining.

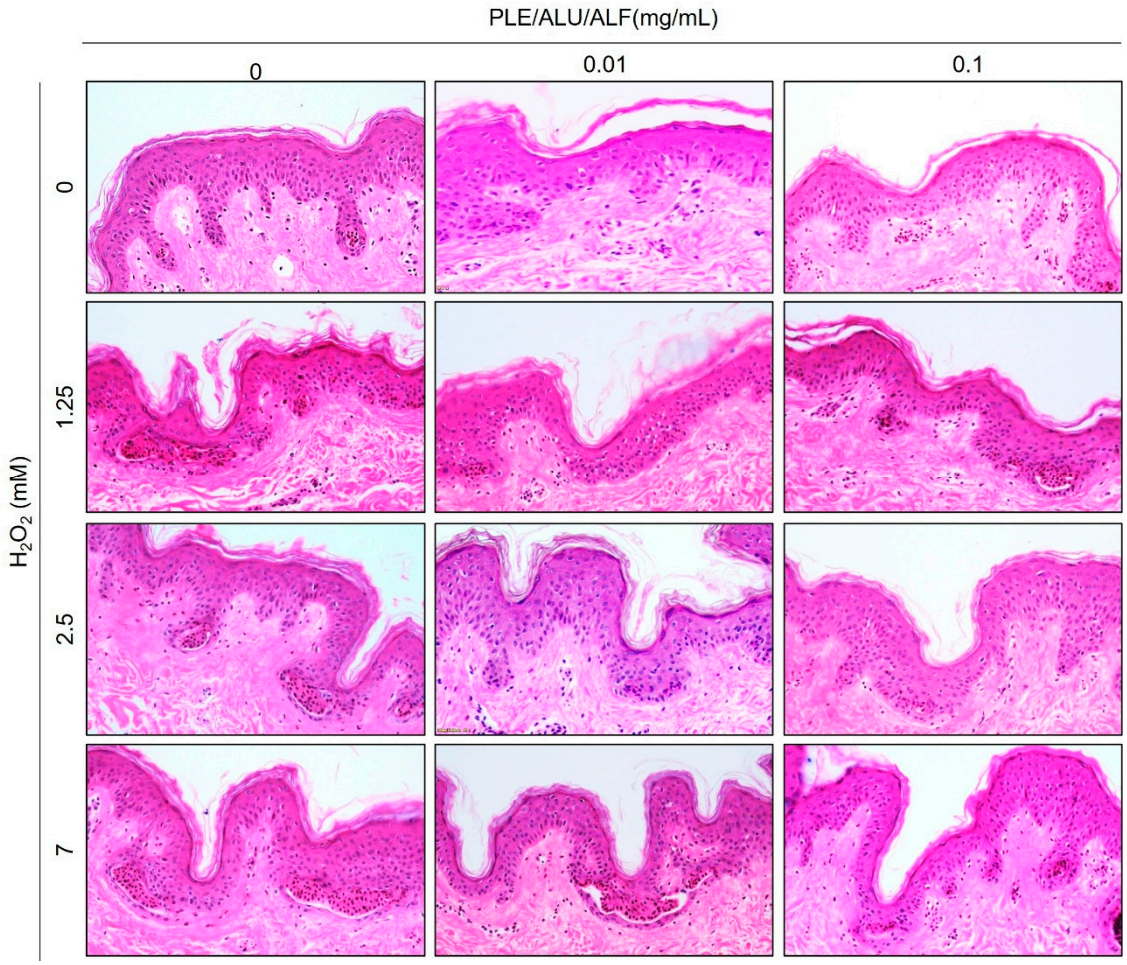


Figure S4. **Hematoxylin and eosin staining in *ex vivo* models**. Effect of PLE/ALU/ALF (0.01 and 0.1 mg/ml), H_2O_2 (1.25, 2.5, and 7 mM), and their combination on *ex vivo* skin samples.

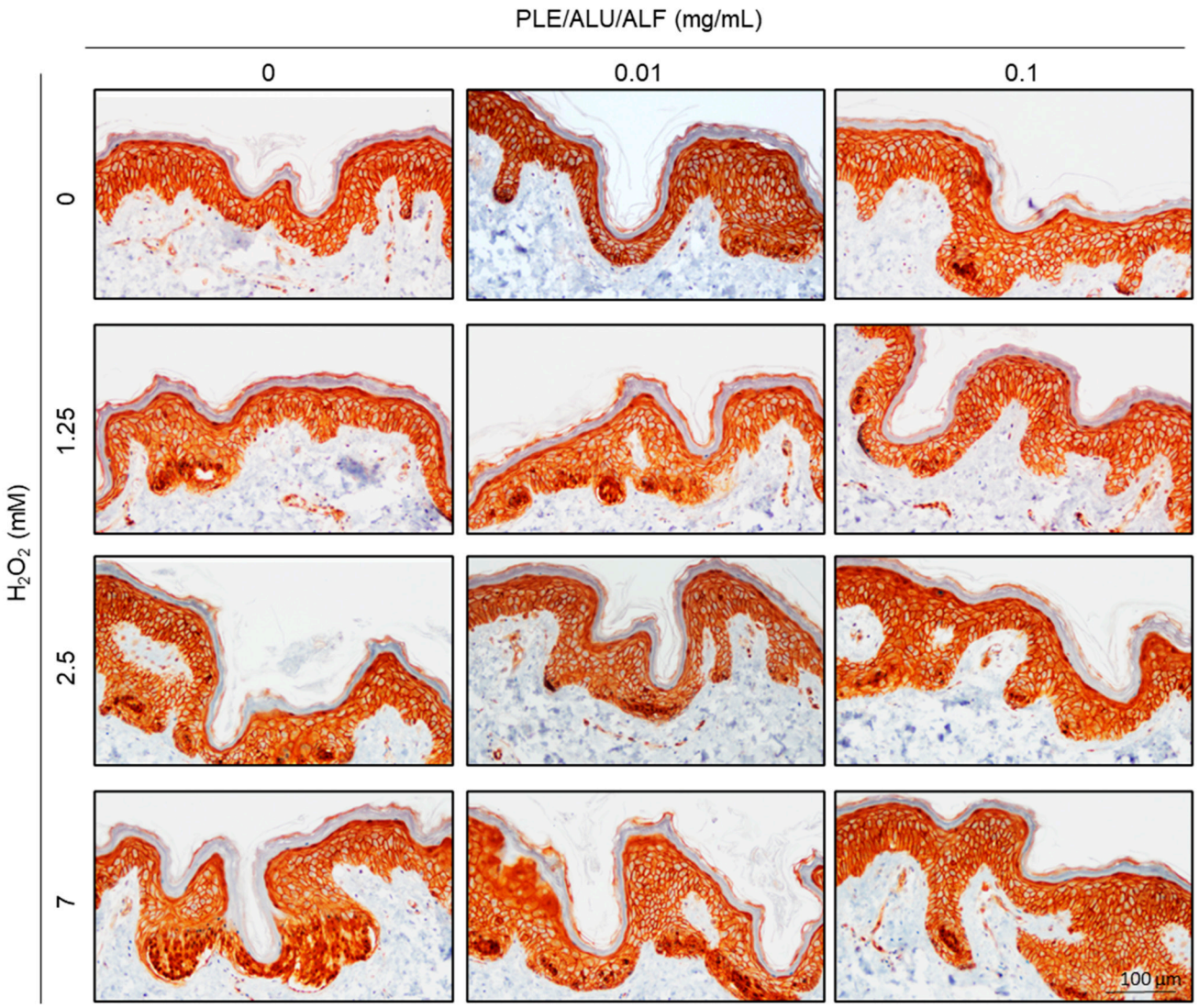


Figure S5. β-catenin immunohistochemistry in *ex vivo* models. Effect of PLE/ALU/ALF (0.01 and 0.1 mg/ml), H_2O_2 (1.25, 2.5, and 7 mM), and their combination on *ex vivo* skin samples.

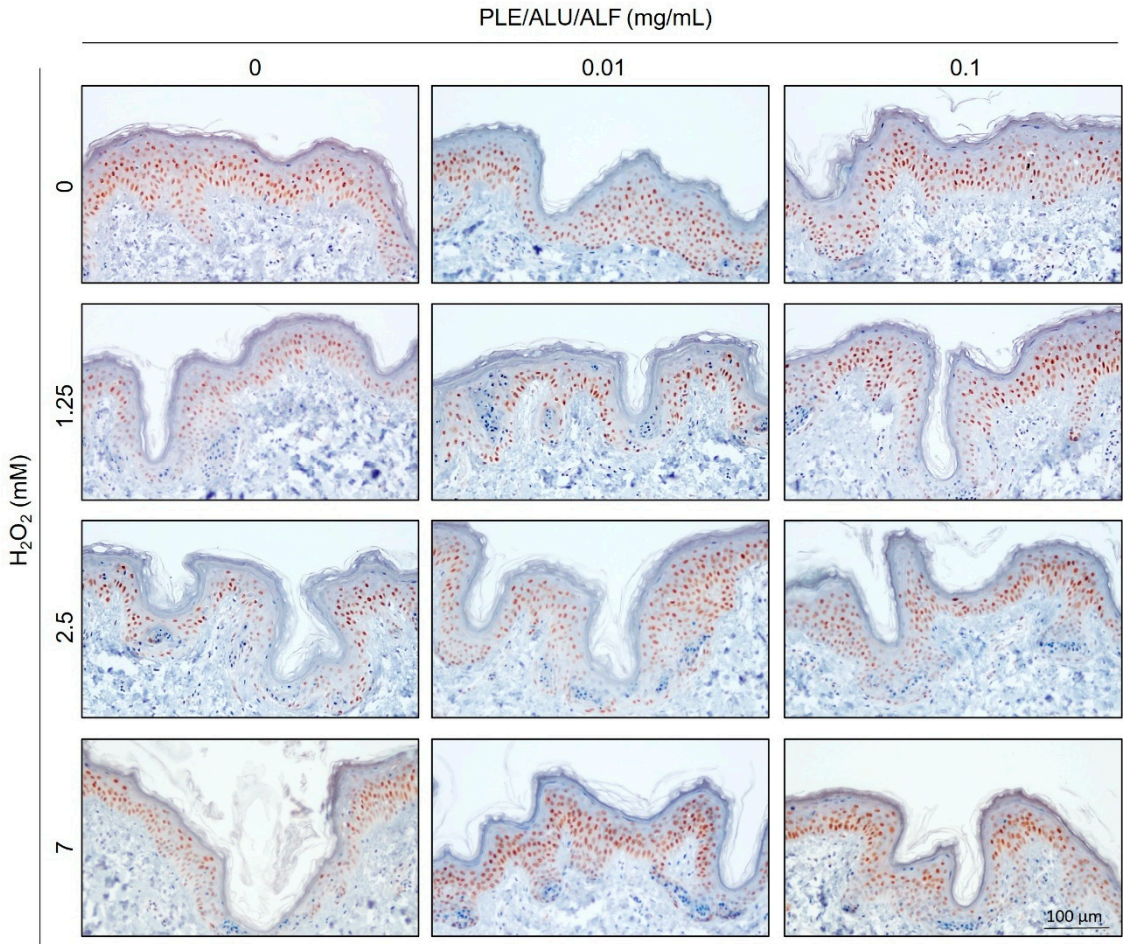


Figure S6. VDR immunohistochemistry in *ex vivo* models. Effect of PLE/ALU/ALF (0.01 and 0.1 mg/ml), H_2O_2 (1.25, 2.5, and 7 mM), and their combination on *ex vivo* skin samples.