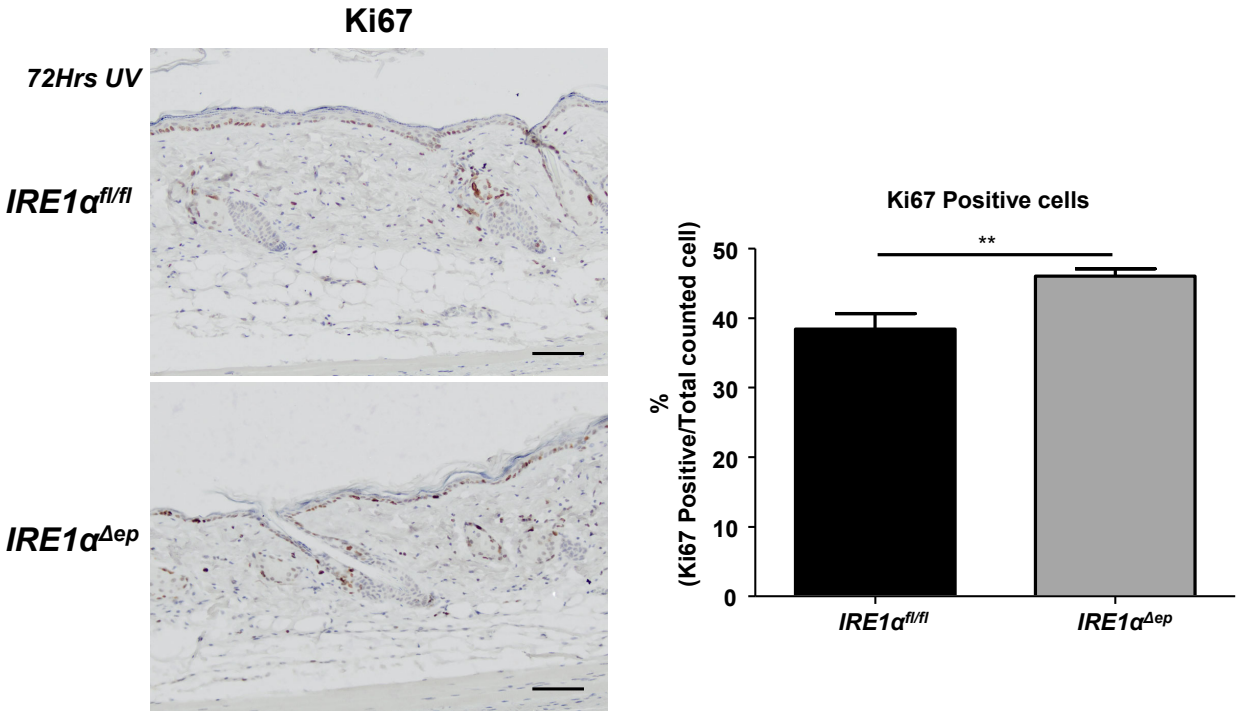
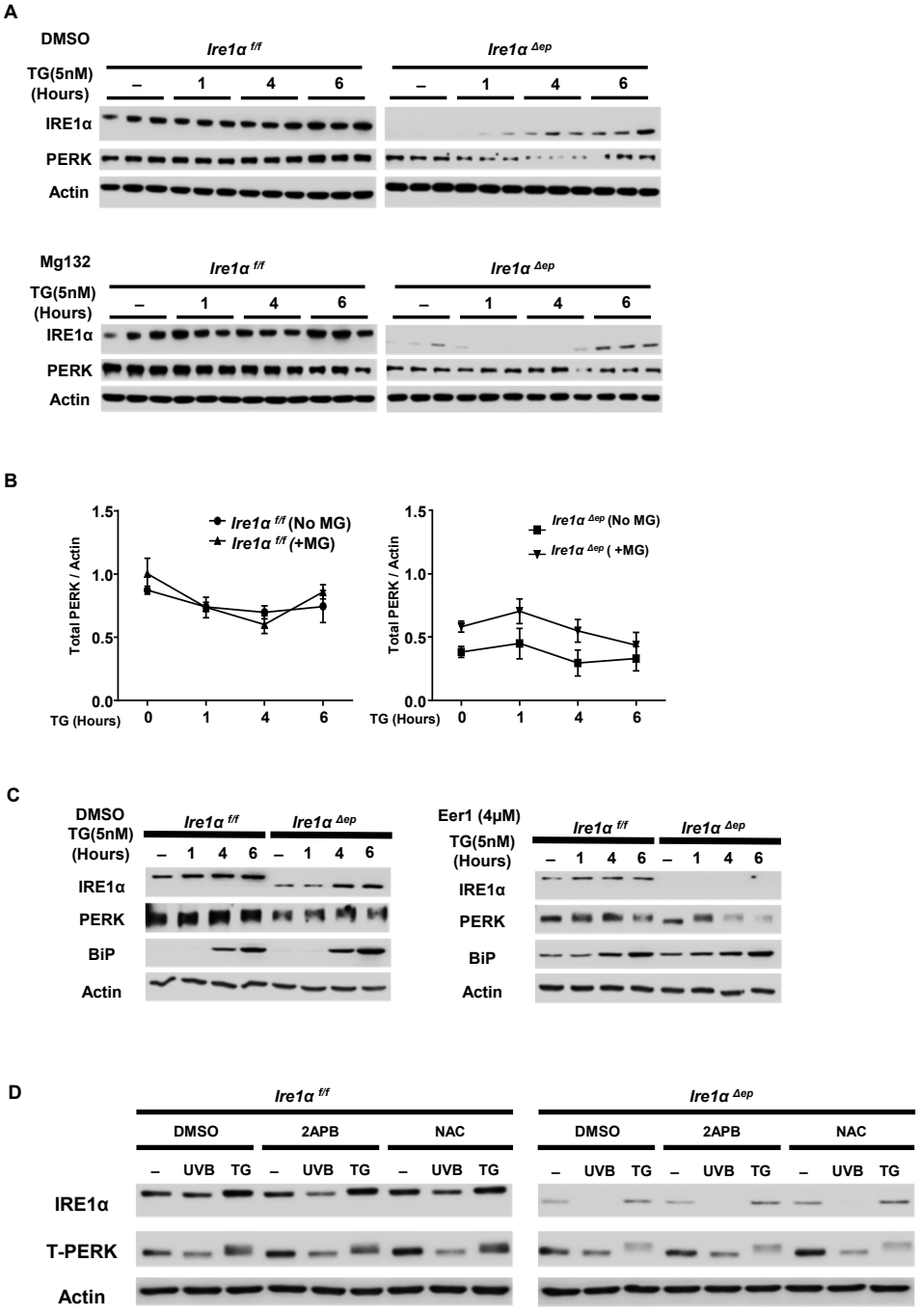


Supplementary Figure 1. Elevated epidermal keratinocyte proliferation in IRE1αΔep mice after acute UVB irradiation.

Mice were harvested at the indicated timepoint after UVB irradiation (360mJ/cm^2), and FFPB skin section were used for Ki67 IHC staining. The sections were counter stained with hematoxylin. The quantitation of Ki67 positive cells was done using Qupath software-H-DAB positive cell analysis (*Ire1α$^{f/f}$* n=4, *Ire1α^{Δep}* n=6). Bar = 20μm. All calculated values were graphed with GraphPad Prism 5.0 software. The statistical analysis was done by Student's t-test Values = mean ± SD. * indicates significant difference (**: P ≤ 0.005).



Supplementary Figure 2. IRE1α-dependent PERK stability is not limited to UVB-mediated stress.

(A) Immunoblot analysis of IRE1α and PERK expression in control and *Ire1α*^{Δep} primary keratinocytes after TG treatment. Control and *Ire1α*^{Δep} primary keratinocytes were treated with thapsigargin (TG) 4 days after initiating cell culture. Cells were pre-treated with DMSO as a vehicle (0.1%, v/v) or MG132 (30μM) 2 hours prior TG treatment. Samples were harvested at the designated timepoint. Beta-actin was used as loading control. (B) Quantitation of PERK expression from A. After densitometry PERK expression values were normalized with beta-Actin expression values. The relative PERK levels values were normalized with DMSO treated control cell values to convert to fold change with 3 biological repeats (n=3). (C) Control and *Ire1α*^{Δep} primary keratinocytes were pre-treated with DMSO as a vehicle (0.1%, v/v) or eeyarestatin I (Eer1) (4μM) 2 hours prior to TG (5nM) treatment. (D) Control and *Ire1α*^{Δep} primary keratinocytes were treated with 2APB (10μM) or NAC (2.5mM) 24 hours prior to UVB (30mJ/cm^2) or TG (5nM) treatment. Samples were harvested 6 hours after UVB and TG treatment. Actin was used as a loading control.