

Supplementary Information for  
**Identification of an epidermal keratinocyte AMPA glutamate receptor  
regulated in dermatopathies associated with sensory abnormalities**

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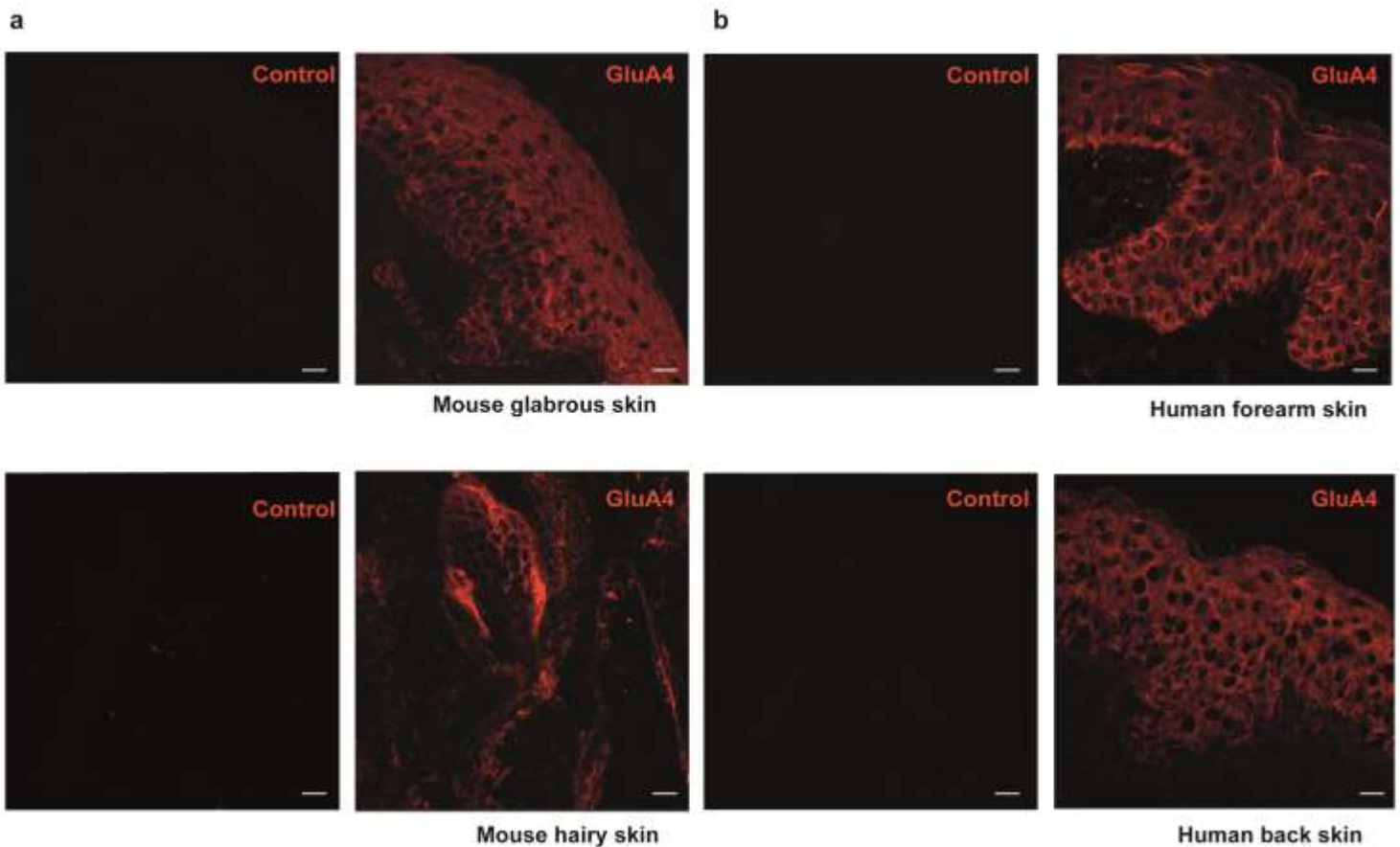
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**Supplementary Figure 1. Negative controls for the specificity of GluA4 antibody staining  
in mouse and human skin specimens.**

Supplementary Figure 2. Sequencing of mouse CD34+ keratinocyte *Gria4* RTPCR.

Supplementary Figure 3. Comparisons of GluA4-IL in skin biopsies from 5 normal  
subjects and 5 age-matched postherpetic neuralgia (PHN) subjects.

## Supplementary Figure 1



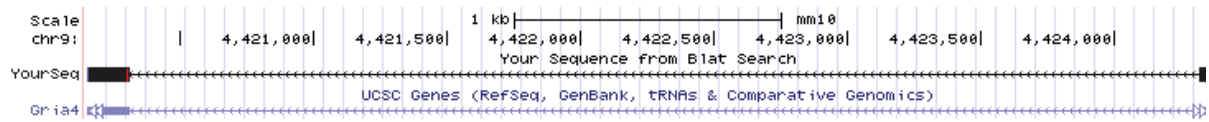
**Supplementary Figure 1. Negative controls for the specificity of the GluA4 antibody staining in mouse and human skin specimens.**

**S1a.** Representative confocal images in the absence (control) or presence (GluA4) of the c-terminus GluA4 antibody (anti-GluA4C) in specimens of mouse glabrous and hairy skin.

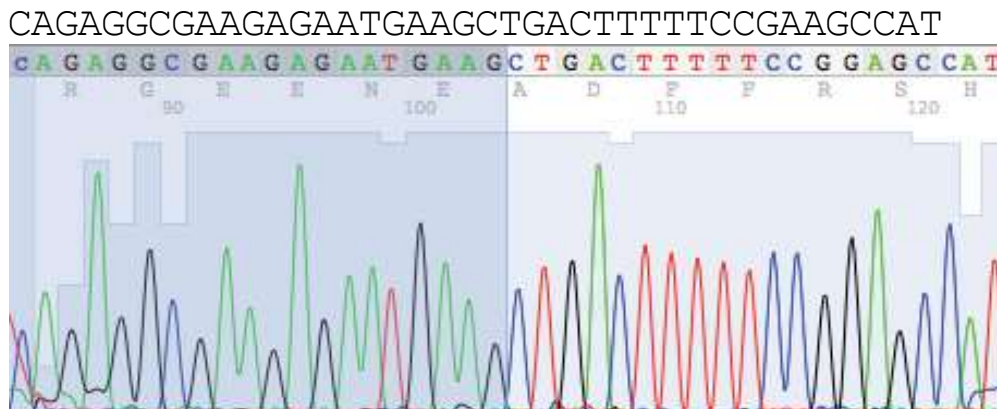
**S1b.** Representative confocal images in the absence (control) or presence (GluA4) of the c-terminus GluA4 antibody (anti-GluA4C) in specimens of human forearm and back skin.

Scale bar = 20 $\mu$ m

## Supplementary Figure 2a



## Supplementary Figure 2b

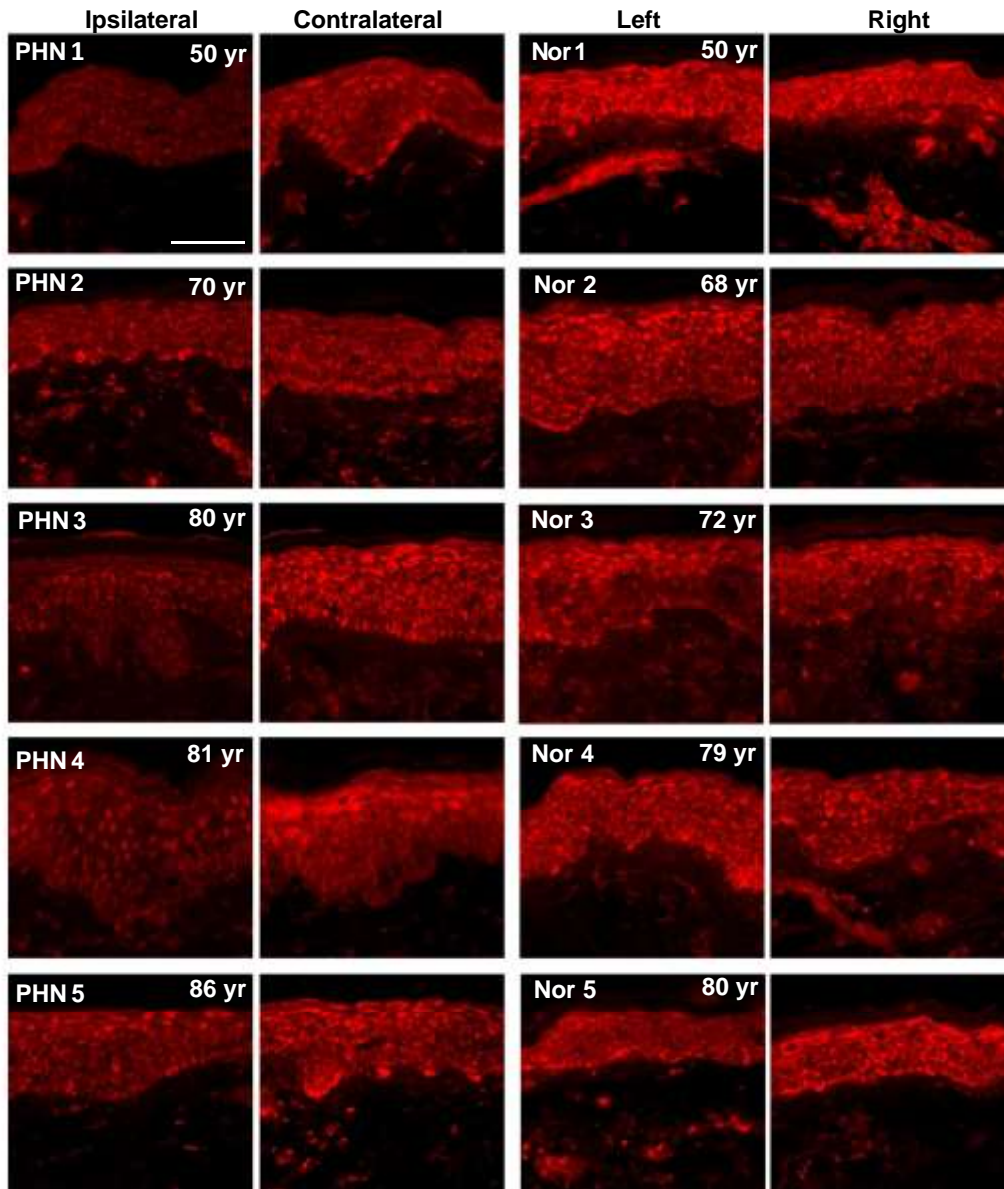


### Supplementary Figure 2. Mouse CD34+ keratinocyte *Gria4* RTPCR sequencing.

**S2a.** The TOPO cloned *Gria4* ex18-ex19 RTPCR product insert sequence was BLAT aligned against the mouse genome, using the UCSC genome browser. Note that the *Gria4* locus is on the antisense strand, with exon 18 represented on the right, and exon 19 on the left.

**S2b.** The sequence of the RTPCR product at the splice junction between exon 18 and exon 19 is identical to other RefSeq entries of *Gria4* splice variants employing these exons. The sequence in black above the chromatogram is the sequence of the aligned region from the UCSC genome.

### Supplementary Figure 3



**Supplementary Figure 3.** Comparisons of GluA4-IL in skin biopsies from 5 normal subjects and 5 age-matched postherpetic neuralgia (PHN) subjects. All biopsies are labeled against GluA4N, except PHN4 which is against GluA4C. The intensity of GluA4-IL is comparable in mirror image right and left side biopsies from each of the normal subjects. The intensity of GluA4-IL is consistently lower in the PHN biopsies than in the mirror image unaffected biopsies.