Stat3 regulates desmoglein 3 transcription in epithelial keratinocytes

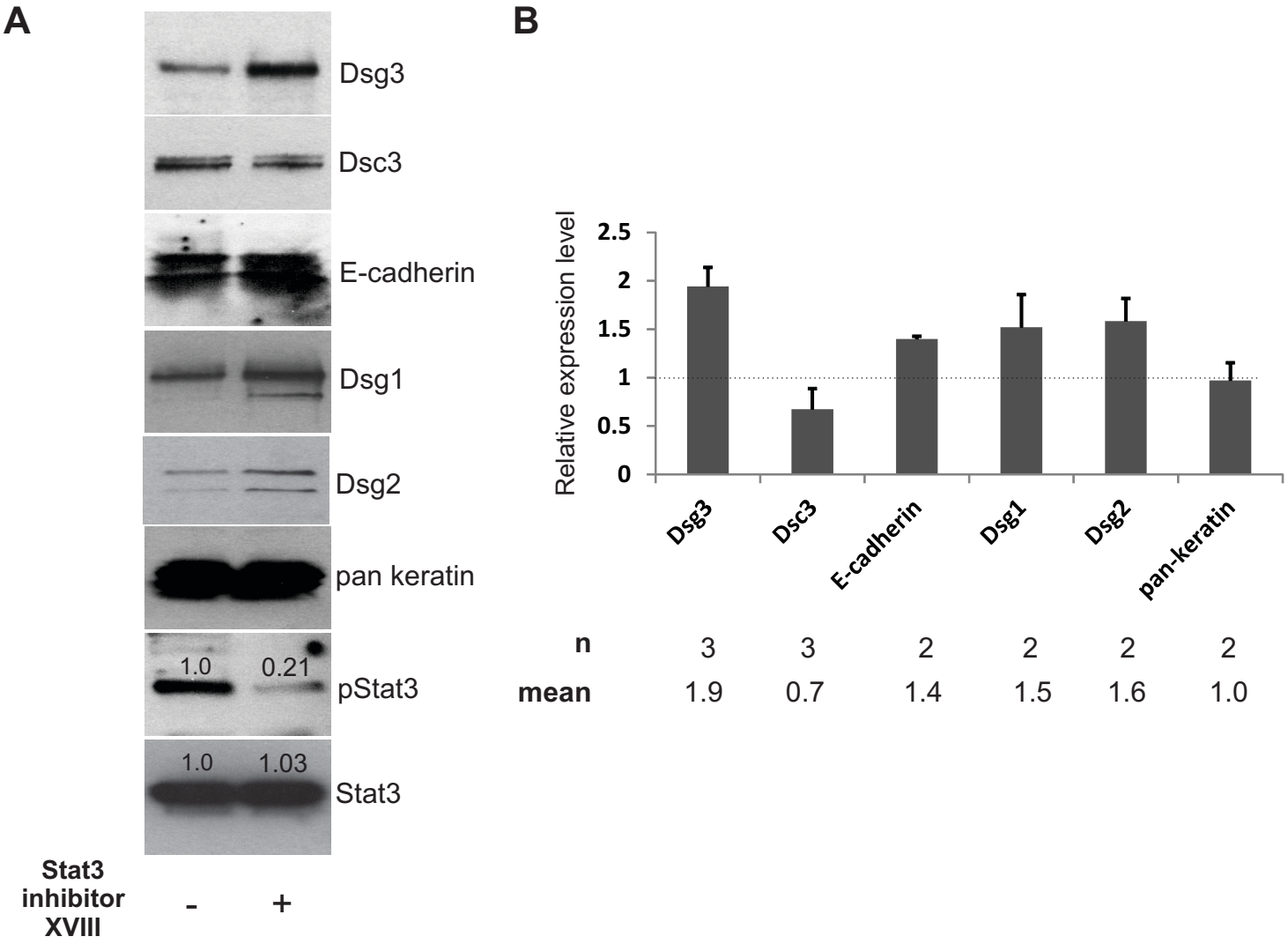
Xuming Mao, Michael Jeffrey T. Cho, Christoph T. Ellebrecht, Eric M. Mukherjee, Aimee S. Payne

Department of Dermatology, University of Pennsylvania, Philadelphia, PA 19104

Supplemental Figure Legends

Supplemental Figure 1. Stat3 inhibition increases keratinocyte expression of desmoglein 3 (DSG3) as well as other adhesion molecules. A. Treatment of primary human keratinocytes with 25 μM Stat3 inhibitor XVIII causes increased protein levels of desmoglein 3 (DSG3), as well as DSG1, DSG2, and E-cadherin, whereas levels of pan-keratin and desmocollin 3 (DSC3) are unchanged to decreased. Levels of phospho-S727 Stat3 (pStat3) and total Stat3 levels are shown for the represented experiment as a control. B. Quantitation of representative experiments in part as shown in panel A. The number (n) of independent experiments for each molecule is shown.

Supplemental Figure 2. Topical Stat3 inhibition protects from pemphigus vulgaris (PV) mAb-induced skin blistering. A. Pathogenic anti-desmoglein (DSG) PV mAb Px43, but not nonpathogenic anti-DSG PV mAb Px44, causes spontaneous suprabasal skin blistering that is inhibited by topical Stat3 inhibition at the gross and histologic levels (**B**, left panels). Direct immunofluorescence (DIF) analysis confirms equal binding of PV mAbs to the keratinocyte cell surface (**B**, right panels). Scale bar=100 μm. Results are representative of 2 different experiments.



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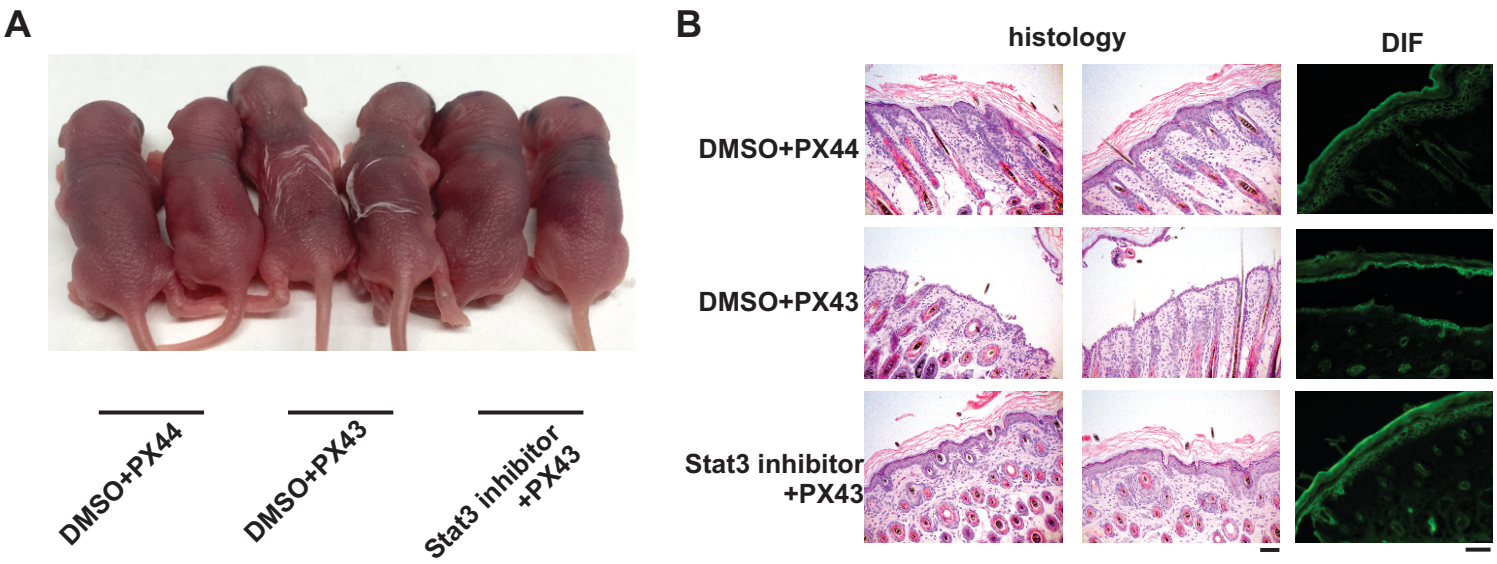

Supplemental Figure 2. Topical Stat3 inhibition protects from PV mAb-induced skin blistering. **A.** Pathogenic anti-Dsg3/1 PV mAb Px43, but not nonpathogenic anti-Dsg3/1 PV mAb Px44, causes spontaneous suprabasal skin blistering that is inhibited by topical Stat3 inhibition at the gross and histologic levels (**B**, left panels). Direct immunofluorescence (DIF) analysis confirms equal binding of PV mAbs to the keratinocyte cell surface (**B**, right panels). Scale bar =100 μm. Results are representative of 2 different experiments.