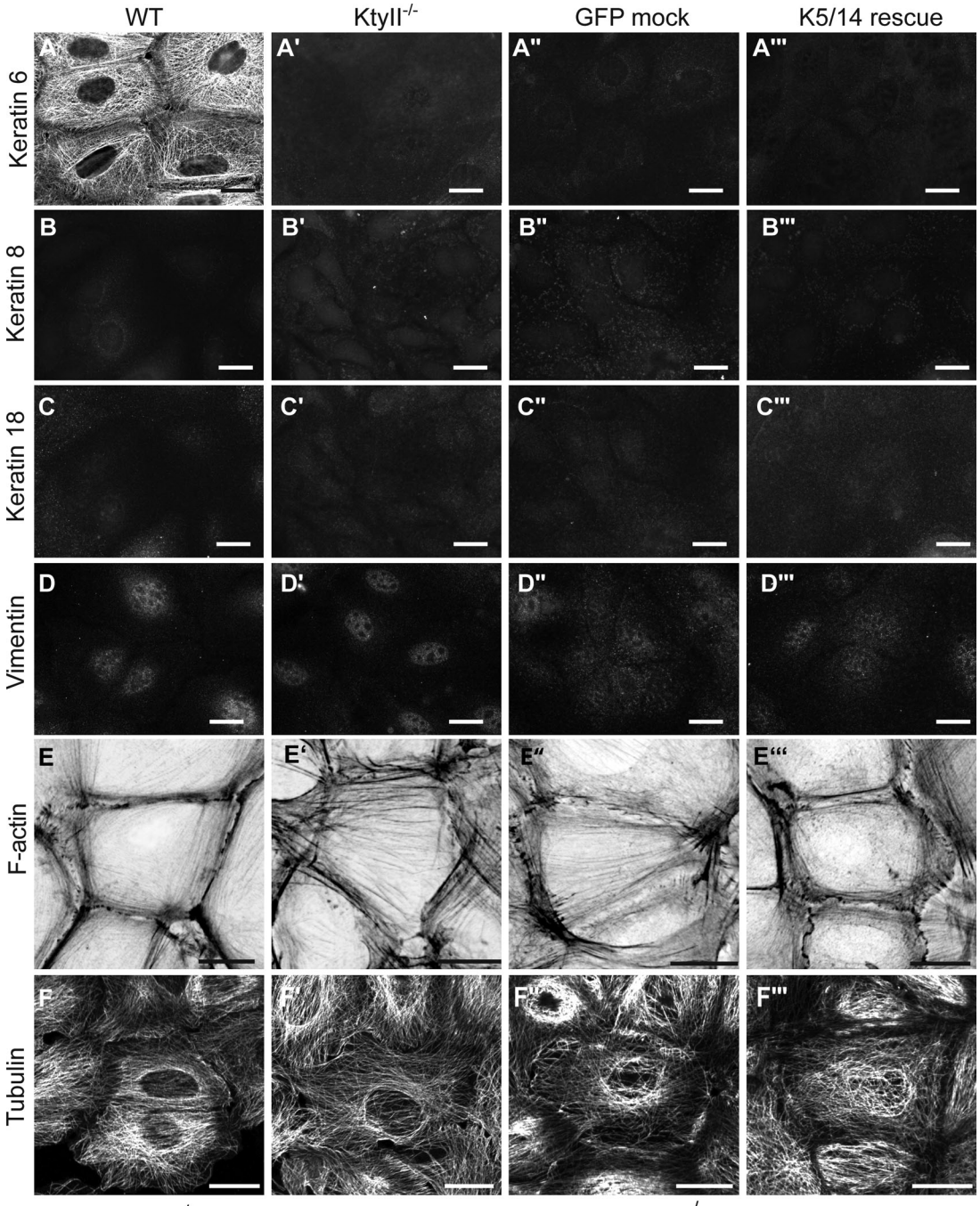
Kröger et al., http://www.jcb.org/cgi/content/full/jcb.201208162/DC1



Genes	FC	Genes	FC
K5	-78,22	K14	1,16
K1	-86,30	K10	-2,99
K6b	-2,70	K16	64,02

Figure S1. **Characterization of keratinocyte lines.** (A–D′′′) Immunofluorescence confirmed absence of keratin proteins in KtyII$^{-/-}$ and GFP mock cells (A′–A′′; B′–B′′). No other IF proteins compensated as revealed by K8 (B–B′′′), K18 (C–C′′′), and vimentin staining in both cell types (D–D′′′). (E–E′′) Strong increase in cytosolic and concomitant decrease in cortical actin stress fibers in KtyII$^{-/-}$ cells (E′) compared with controls (E and E′′). (F–F′′′) No major alterations of MT in the absence of keratins. Bars, 10 µm. (G) Confirmation of KtyII cluster deletion by genomic PCR for K5 and K8. Type I keratin K19 remained. (H) Transcriptome data from skin of KtyII$^{-/-}$ E.18.5 skin, confirming absence of type II keratin mRNAs and persistence at lower levels of K10 and K14 mRNA.

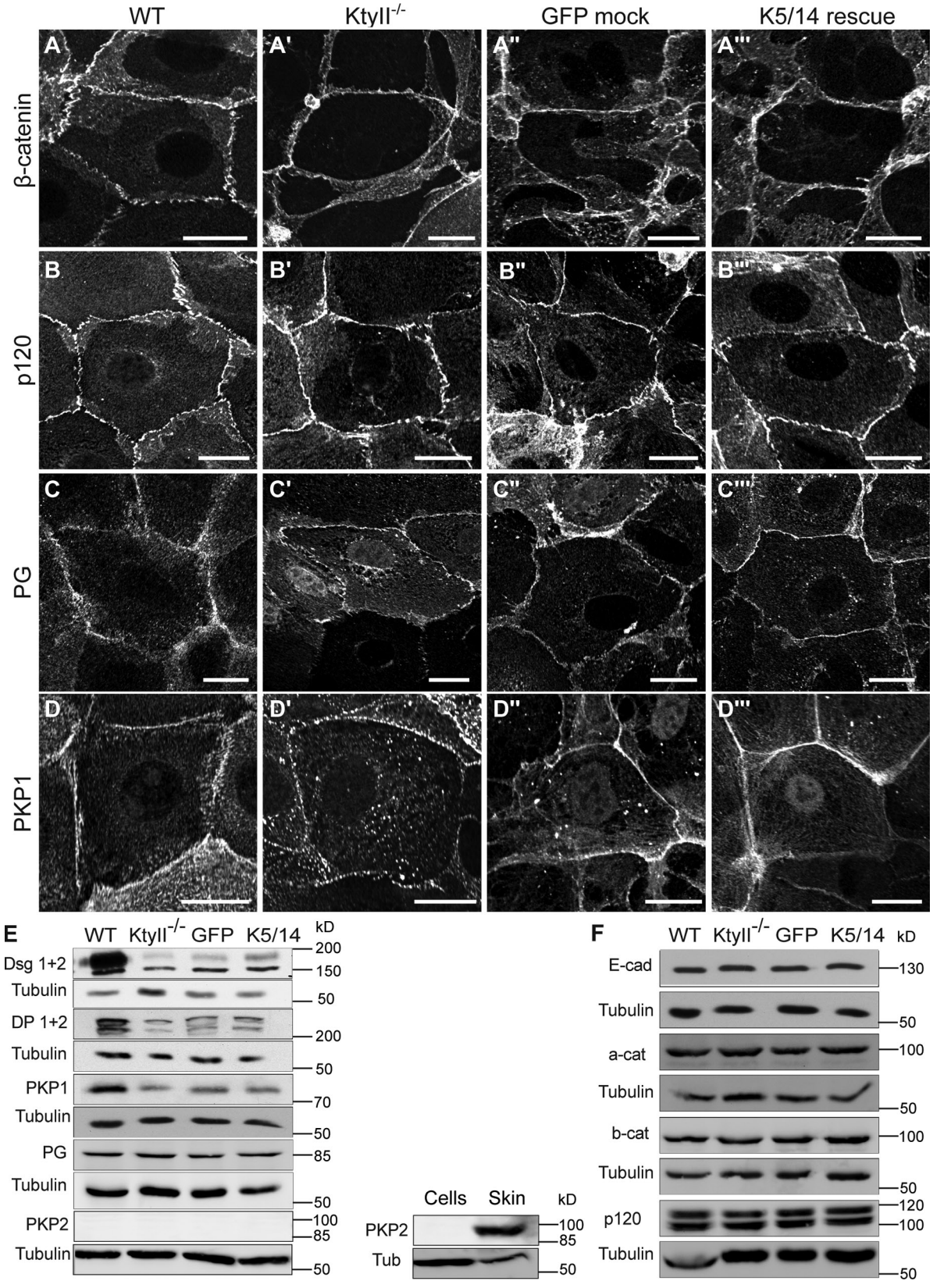


Figure S2. **Analysis of junction proteins.** (A–A‴ and B–B‴) AJ-associated proteins β-catenin (A–A‴) and p120 (B–B‴) seemed unaltered in WT (A, A‴; B, B‴) and KtyII$^{-/-}$ cells (A′, A″; B′, B″). PG displayed no difference in the four different cell types (C–C‴). PKP1 (D–D‴) staining showed same redistribution in KtyII$^{-/-}$ cells (D′, D″) as other desmosomal proteins. Bars: (A–D‴) 10 µm. (E-F) WB (n = 3) of desmosomal proteins showed strong reduction in KtyII$^{-/-}$ (E), and unaltered AJ proteins and plakoglobin in all four cell lines (E and F).

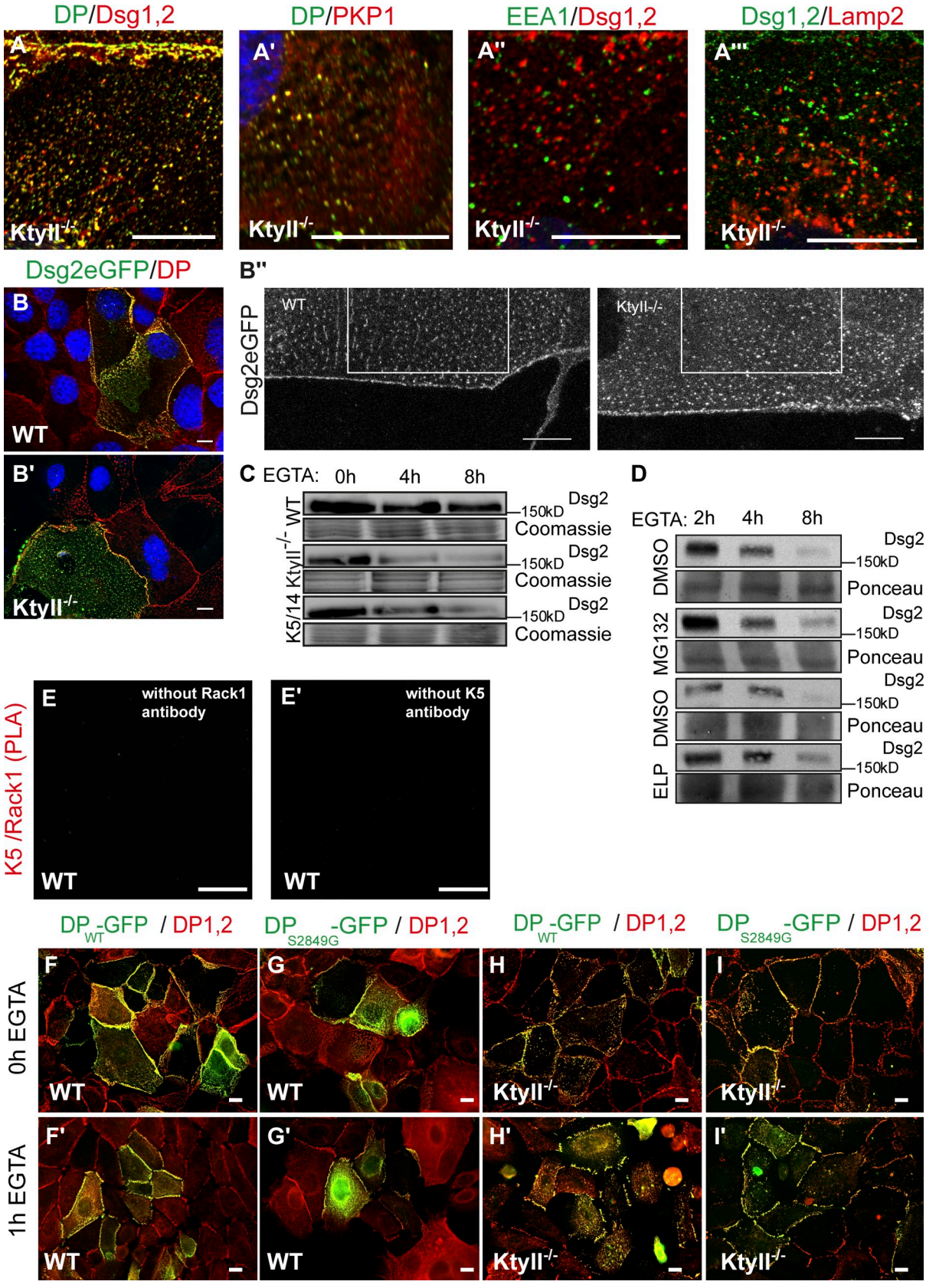


Figure S3. **Analysis of keratin–desmosome interactions.** (A–A‴) Colocalization of Dsg/DP (A) and DP/PKP1 (A′) in cyotosol of Ktyll$^{-/-}$ cells, no colocalization of Dsg2 with EEA1 (A″) or Lamp2 (A‴). (B–B′) Colocalization of Dsg2-eGFP with endogenous DP in WT and Ktyll$^{-/-}$ cells. (B″) Confocal images showing projections of 10 focal planes of WT and Ktyll$^{-/-}$ cells expressing Dsg2-eGFP depicting regions used for the tracking analysis. 120 frames were examined for image analysis in Fig. 2. (C) WB of total protein lysates after EGTA treatment demonstrated accelerated degradation of Dsg2 in Ktyll$^{-/-}$ cells reverted by reexpression of K5/14. (D) WB of total protein lysates after EGTA treatment demonstrated that MG132 and ELP inhibit Dsg2 degradation to distinct extents. (E–E′) Negative control for PLA. (F–I′) Colocalization of DP_{WT}GFP and DP_{S2849G}GFP with endogenous DP in WT and Ktyll$^{-/-}$ cells. Bars, 10 µm.

Table S1. **Antibodies**

Primary antibodies	**Host**	**Source**
Anti-KRT5	Rabbit	Magin laboratory
Anti-KRT5 head domain	Guinea pig	Betz et al., 2006
Anti-KRT6, monoclonal	Mouse	Progen
Anti-KRT8, monoclonal	Mouse	Progen
Anti-KRT14	Rabbit	Magin laboratory
Anti-KRT16	Rabbit	P.A. Coulombe, The Johns Hopkins School of Medicine, Baltimore, MD
Anti-KRT17	Guinea pig	L. Langbein, German Cancer Research Center, Heidelberg, Germany
Anti-KRT18, monoclonal	Rabbit	Epitomics
Anti-vimentin	Rabbit	Magin laboratory
Anti–α-tubulin, monoclonal	Mouse	Sigma-Aldrich
Anti–β-actin, monoclonal	Mouse	Sigma-Aldrich
Anti–E-cadherin, monoclonal	Rat	Sigma-Aldrich
Anti–α-catenin	Rabbit	Sigma-Aldrich
Anti–β-catenin, monoclonal	Mouse	Transduction Laboratories
Anti–p120-catenin, monoclonal	Mouse	Santa Cruz Biotechnology, Inc.
Anti-DP (II-5F)	Mouse	D. Garrod, University of Manchester, Manchester, UK
Anti-DP	Guinea pig	Progen
Anti-Dsg3.10, monoclonal	Mouse	Progen
Anti-PKP1, monoclonal	Mouse	Epitomics
Anti-PKP3, monoclonal	Mouse	Santa Cruz Biotechnology, Inc.
Anti-plakoglobin, monoclonal	Mouse	Progen
Anti-p0071, monoclonal	Mouse	Progen
Anti-phosphoserine	Rabbit	Abcam
Anti–PKC-α, monoclonal	Rabbit	Epitomics
Anti-Rack1, monoclonal	Mouse	Santa Cruz Biotechnology, Inc.
Secondary antibodies		
Anti–mouse-DL488, -DL549, -DL649, -HRP	Donkey	Dianova
Anti–rabbit-DL488, -DL549, -DL649, -HRP	Donkey	Dianova
Anti–rat-DL488, -DL549, -DL649, -HRP	Donkey	Dianova
Anti–guinea pig-DL488, -DL549, -DL649	Donkey	Dianova

Reference

Betz, R.C., L. Planko, S. Eigelshoven, S. Hanneken, S.M. Pasternack, H. Bussow, K. Van Den Bogaert, J. Wenzel, M. Braun-Falco, A. Rutten, et al. 2006. Loss-of-function mutations in the keratin 5 gene lead to Dowling-Degos disease. *Am. J. Hum. Genet.* 78:510–519. http://dx.doi.org/10.1086/500850