

Correction

MEDICAL SCIENCES

Correction for “LRP-6 is a coreceptor for multiple fibrogenic signaling pathways in pericytes and myofibroblasts that are inhibited by DKK-1,” by Shuyu Ren, Bryce G. Johnson, Yujiro Kida, Colin Ip, Kathryn C. Davidson, Shuei-Liong Lin, Akio Kobayashi, Richard A. Lang, Anna-Katerina Hadjantonakis, Randall T. Moon, and Jeremy S. Duffield, which appeared in issue 4, January 22, 2013, of *Proc Natl Acad Sci USA* (110:1440–1445; first published January 9, 2013; 10.1073/pnas.1211179110).

The authors wish to note the following: “Panel *L* in Fig. 3 incorrectly showed a low-power image of fibrosis extent in sham kidneys exposed to Ad-control instead of Ad-Dkk1. This problem has now been corrected by replacing the upper left image with a representative panel from sham kidney treated with Ad-Dkk1.” The corrected Fig. 3 and its legend appear below.

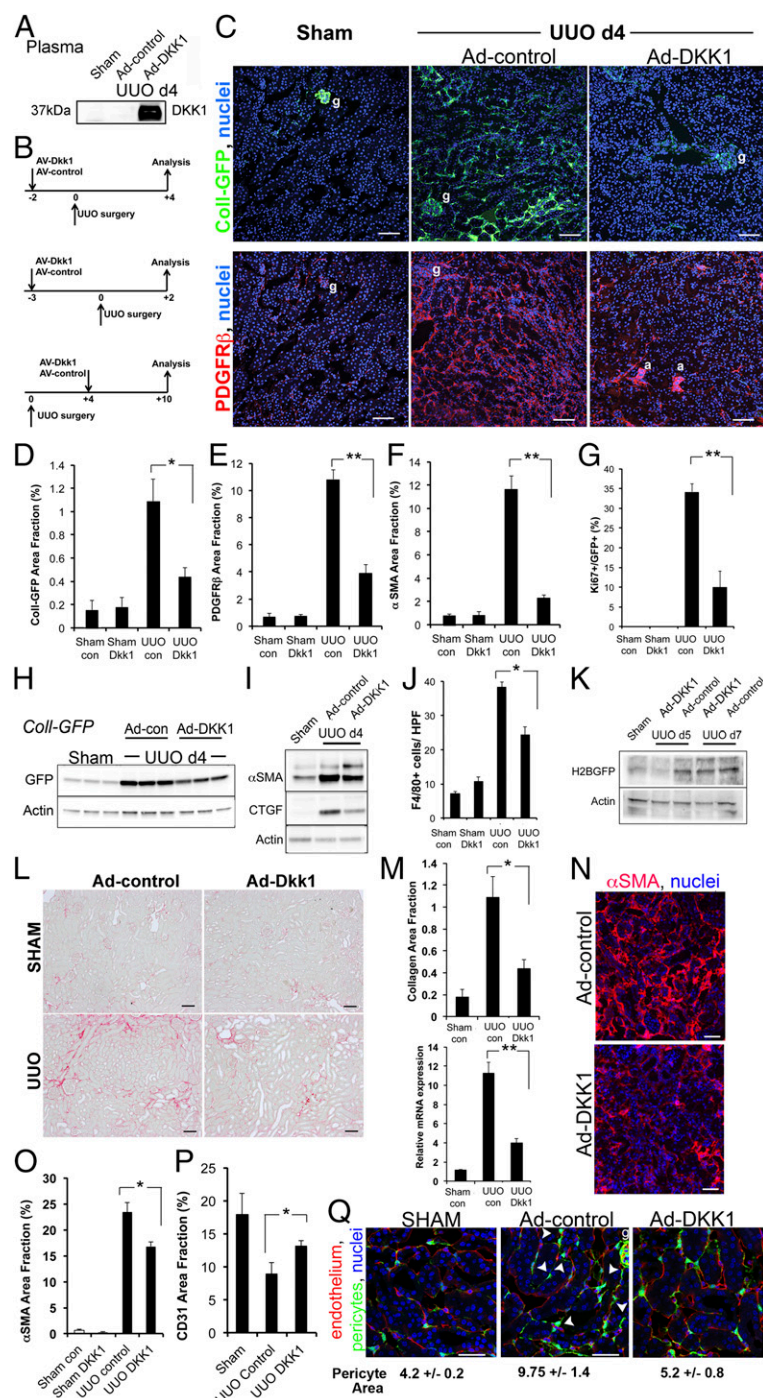


Fig. 3. DKK-1 blocks pericyte activation and transition to myofibroblasts and reverses myofibroblast activation in vivo, inhibiting fibrogenesis, capillary rarefaction, and inflammation. (A) Western blots of 5 μ L of plasma from mice 5 d after i.v. injection of Ad-control or Ad-DKK-1 and from mice subjected to sham surgery and injected with control. (B) Experimental schemata for adenoviral administration, kidney injury, and analysis in the UUO model. (C–M) Prevention studies. (C) Low-magnification confocal images of kidney cortex 4 d after sham operation or UUO in *Coll-GFP^{Tr}* mice that had received Ad-control or Ad-DKK-1 6 d previously, showing Coll-GFP cells or PDGFR β cells. g, glomerulus; a, arteriole. (D–F) Graphs showing quantification of Coll-GFP cells, PDGFR β cells, and α SMA cells in kidney 4 d after UUO. (G) Proportion of Coll-GFP cells that express the proliferation marker Ki67. (H and I) Western blot of GFP (H) or α SMA/CTGF (I) in whole *Coll-GFP* mouse kidney 4 d after UUO. (J) Quantification of macrophage numbers in kidney sections detected by F4/80 staining. (K) Western blot quantifying canonical WNT signaling by detecting the H2B-GFP fusion protein after Ad-DKK-1 vs. Ad-control treatment of *TCF/Lef:H2B-GFP^{Tr}* reporter mice during UUO kidney injury. (L) Sirius red-stained kidneys 10 d after UUO. (M) Morphometry of Sirius red-stained collagen (Upper) or qPCR for *Col1a1* transcripts (Lower) 10 d after UUO in mice treated with Ad-control vs. Ad-DKK-1. (N–P) Reversal studies. Confocal Images (N) and morphometric quantification (O) of α SMA staining 10 d after UUO in mice treated with Ad-control or Ad-DKK-1 from day 4. (P) Quantification of capillary density 10 d after UUO. Note that rarefaction occurs in response to kidney disease, but DKK-1 partially reverses rarefaction. (Q) Pericyte detachment. Images and quantification of pericyte area in *Coll-GFP* mice 2 d after UUO in the presence of circulating DKK-1 or control. Note that injury to the kidney stimulated pericyte spreading and detachment from endothelium (arrowheads). *P < 0.05, **P < 0.01. n = 4–6 per group. Error bars indicate SEM.

www.pnas.org/cgi/doi/10.1073/pnas.1616960113