ERRATUM

J Am Soc Nephrol 14: 823–824, 2003

In "P53 Mediates the Apoptotic Response to GTP Depletion after Renal Ischemia-Reperfusion: Protective Role of a p53 Inhibitor" by Kelly *et al.*, which appeared in the January 2003 issue of *JASN*, Figures 8 and 9 were erroneously printed in black and white. They are printed below in color.

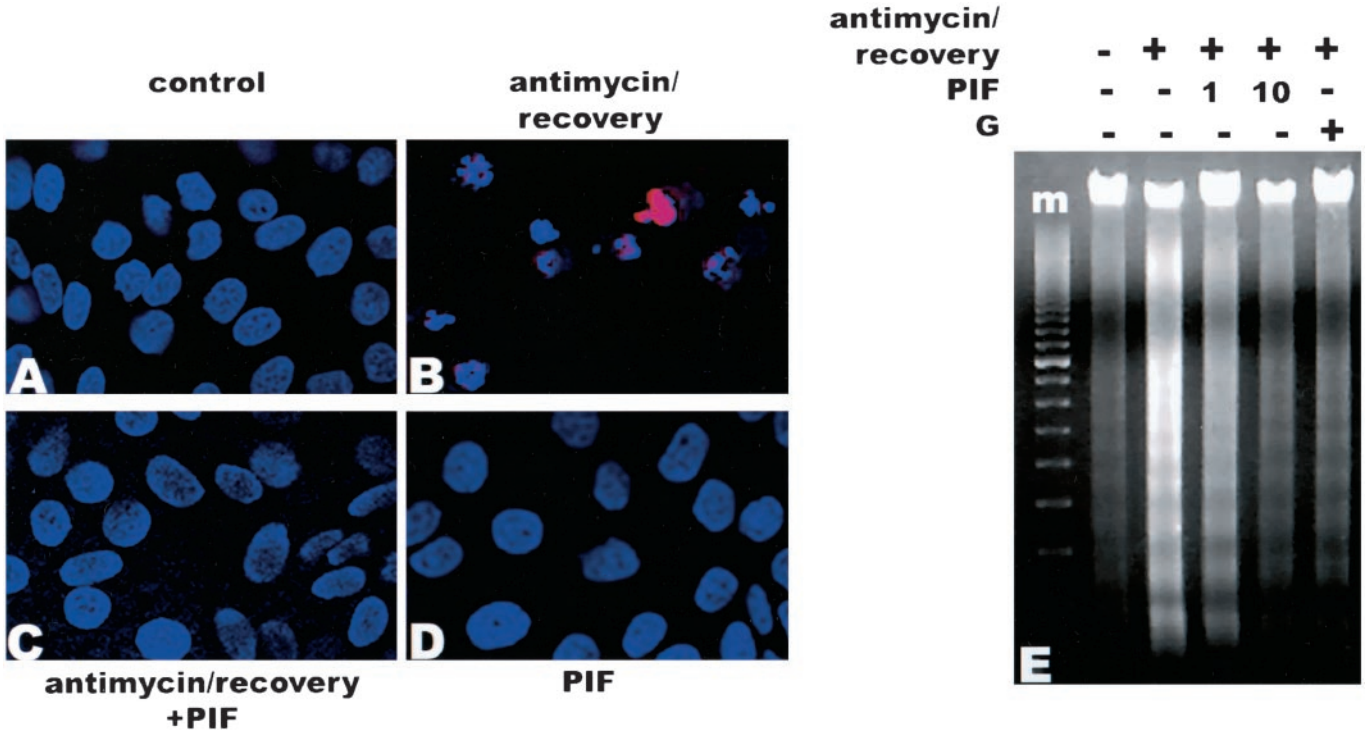

Figure 8. Effect of antimycin/recovery and treatment with the p53 inhibitor PIF or guanosine (G) on apoptosis in cultured renal tubular cells. A representative fluorescent micrograph of LLC-PK_1 cells demonstrating normal nuclear morphology is shown in panel A. Condensed, fragmented nuclei characteristic of apoptosis are seen after antimycin/recovery (0.1 μM antimycin A for 45 min and 24 h recovery; panel B). Apoptosis was not apparent in the cells treated with PIF (10 μM) before antimycin/recovery, as shown by the normal nuclear morphology in panel C. PIF alone did not alter nuclear morphology (panel D). Apoptosis after antimycin/recovery was also evident by characteristic laddering on DNA gel electrophoresis (panel E). Prevention of laddering was seen after antimycin/recovery in the presence of PIF (1 or 10 μM) or guanosine (G; 50 μM; panel E). m, DNA size markers.

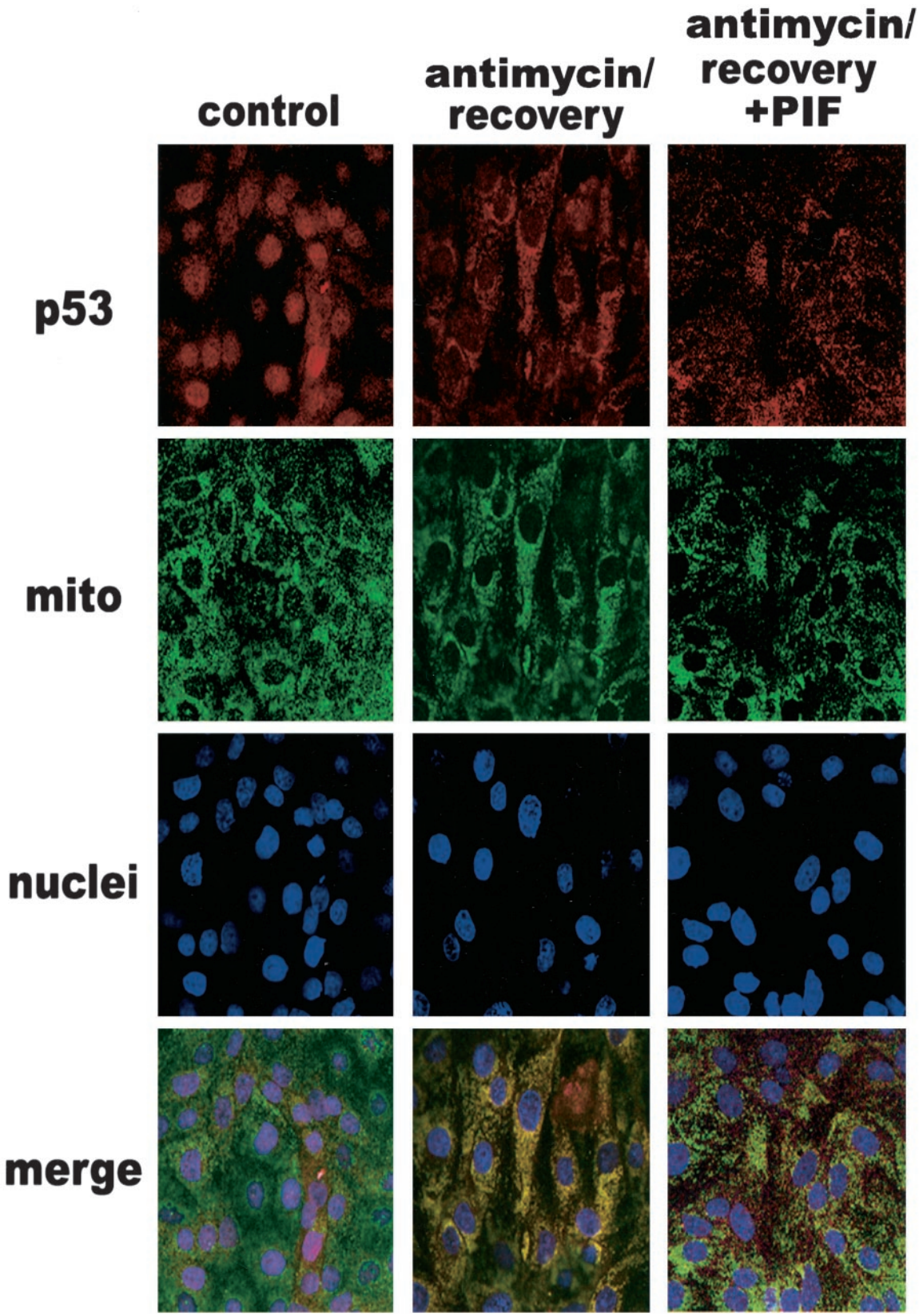

Figure 9. Effects of antimycin/recovery and PIF on the cellular localization of p53 in cultured renal tubular cells. Cultured LLC-PK_1 renal tubular epithelial cells were harvested 24 h after exposure to antimycin A (0.1 μM for 45 min) with or without pifithrin-α (PIF; 10 μM) as detailed in Materials and Methods. Cells were fixed and immunostained with antibodies to p53 (Texas red-label, red) and the mitochondrial marker cytochrome c oxidase (FITC-label, green). Nuclei were stained with To-Pro-3 iodide (blue). Control cells were maintained in standard media.