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CORRECTION

Correction: Angiotensin-(1–9) prevents Angiotensin II-induced endothelial apoptosis through CNPY2/PERK pathway

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Correction: Apoptosis (2023) 28(3–4):379–396
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In the original article, the wrong Figs. 1H and 5B, C were published. Due to our negligence, we accidentally misplaced the wrong image for wound healing assays in Fig. 1H in the originally published version of this article. There was an error in Fig. 1H in the published article, and the image of wound healing in HUVECs at 0 h in Ang II + Ang-(1–9) group was wrong. In addition, we unintentionally used the same MitoSOX and DCFH-DA images in Fig. 5B, C. The corrected versions of Figs. 1 and 5 are shown below. This correction does not affect results and scientific conclusions of the research.

The corrected figures have been provided as the Revised Figs. 1 and 5.

Chun-ling Guo, Hui-min Liu have equally contributed to this work.

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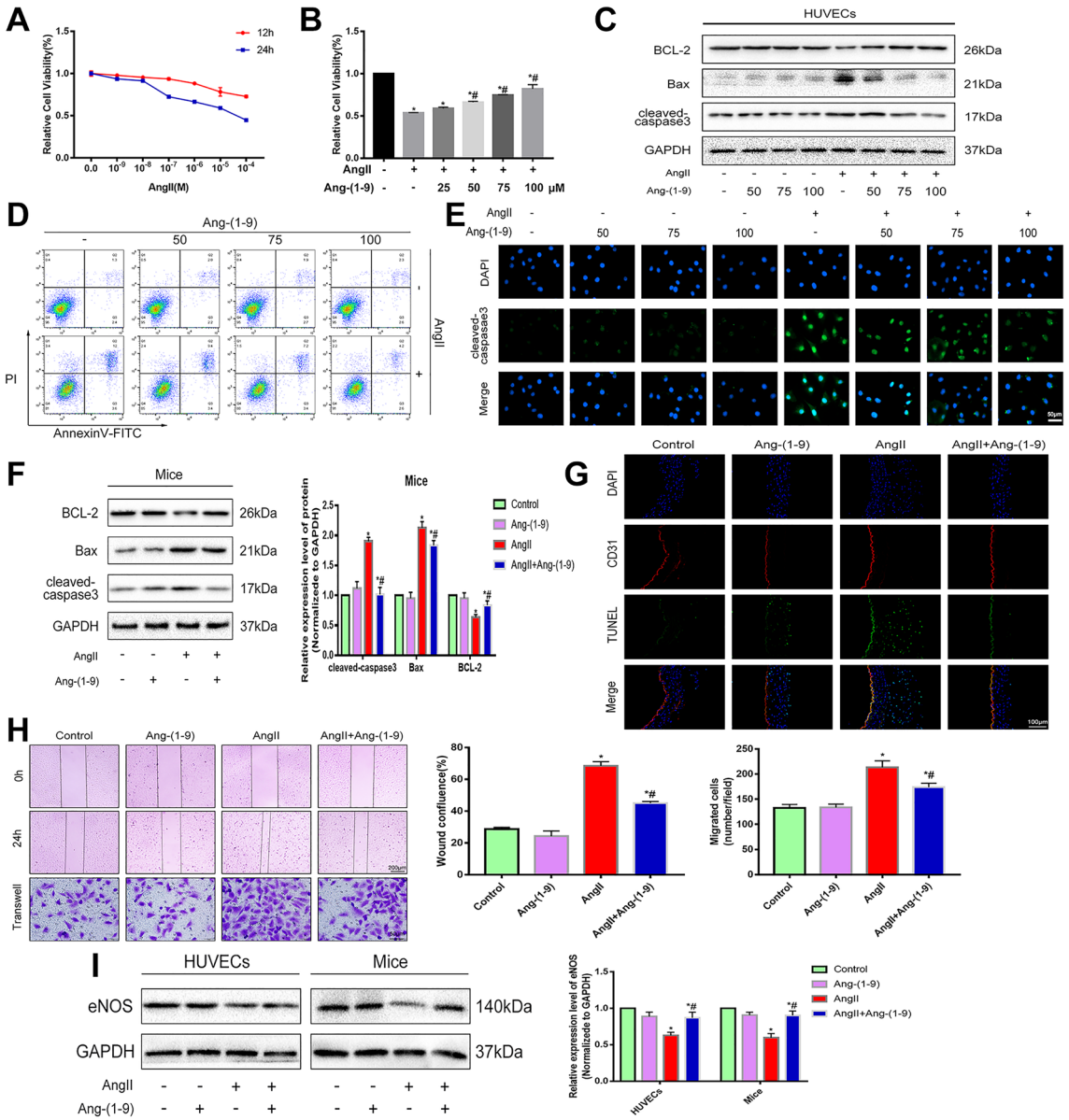

Fig. 1 Ang-(1–9) inhibited Ang II-induced apoptosis and improved vascular endothelial function. **A** HUVECs were treated with different concentration of Ang II for 12 h or 24 h, and the cell viability was detected by CCK-8 assay. **B** HUVECs were pretreated with different concentration of Ang-(1–9) for 1 h and followed by Ang II (10 μM) for 24 h, and the cell viability was detected by CCK-8 assay. **C** Cells were pretreated with different concentration of Ang-(1–9) (50, 75, 100 μM) 1 h prior to Ang II for 24 h. The expression of apoptotic relative proteins Bax, BCL-2 and cleaved caspase-3 were measured in HUVECs by western blotting. Quantitative analysis. **D** Cells were pretreated with different concentration of Ang-(1–9) (50, 75, 100 μM) 1 h prior to Ang II for 24 h. Annexin V-FITC/PI staining was performed in HUVECs apoptosis. **E** Cells were pretreated with different concentration of Ang-(1–9) (50, 75, 100 μM) 1 h prior to Ang II for 24 h. The representative immunofluorescence images of cleaved-caspase-3 (green), and the nuclei were stained with DAPI (blue). Scale bar = 50 μm, and the relative quantitative mean immunofluorescence intensity of nucleus caspase-3 was detected. **F** The expression of apoptotic-relative proteins Bax, BCL-2 and cleaved caspase-3 were measured in the aorta of mice by western blotting. **G** TUNEL (green) and CD31 (red) immunofluorescence staining was performed to determine the apoptosis of the aorta endothelial cell of mice. Fluorescence intensity of apoptosis endothelial cells was measured. Scale bar = 100 μm. **H** Monolayer confluent HUVECs were serum starved and scraped in the presence of Ang II (10 μM) with or without Ang-(1–9) (100 μM) to stimulate HUVECs migration toward the wound area (0 h and 24 h, first two lines). Scale bar = 200 μm. The third line shows the transwell assay of HUVECs. Scale bar = 50 μm. Representative images of the in vitro scratch-wound and transwell migration assay and quantification of migrated cells (number/field) are presented. **I** Protein expression of eNOS were examined by Western blotting. Quantitative analysis of protein eNOS. All data were expressed as mean ± SD, n = 3. $^{*}p < 0.05$ vs. control group, $^{\#}p < 0.05$ vs. Ang II group (Color figure online)

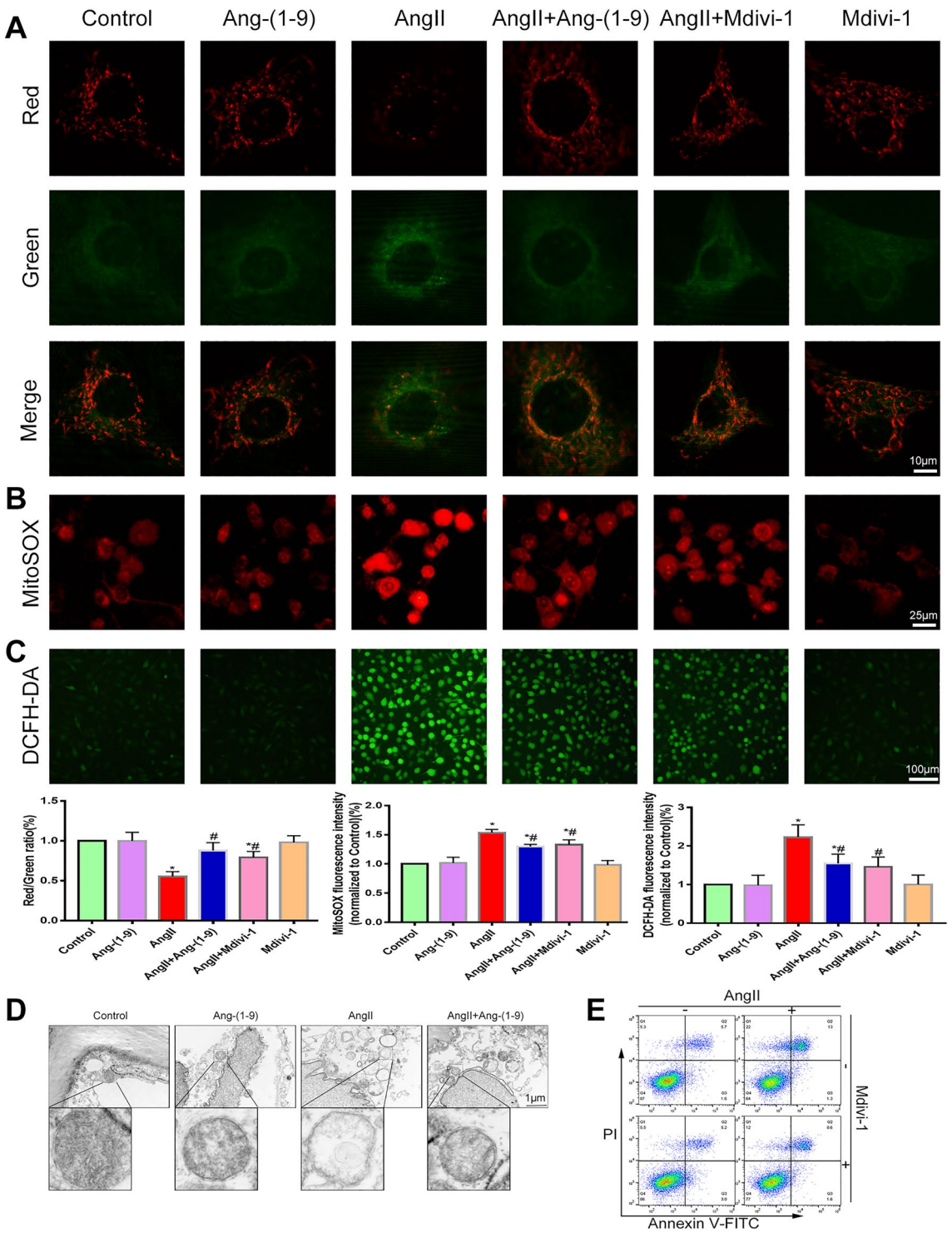

Fig. 5 The Ang-(1–9) suppressed Ang II-induced excessive mitochondrial fission. **A** JC-1 staining of the mitochondrial membrane potential, a hallmark of mitochondrial apoptosis (n = 5). Scale bar = 10 μm. **B** Cells were pretreated with Mdivi-1 (10 nM) or Ang-(1–9) (100 μM) for 1 h before Ang II treatment for 24 h. Cells were stained with MitoSOX (n = 5). Scale bar = 25 μm. **C** Cells were pretreated with Mdivi-1 (10 nM) or Ang-(1–9) (100 μM) for 1 h before Ang II treatment for 24 h. Cells were stained with DCFH-DA (n = 5). Scale bar = 100 μm. **D** The TEM of mitochondrial morphology in HUVECs. Scale bar = 1 μm. **E** Cell apoptosis was assessed by Annexin V-FITC/PI staining. Results were presented as mean ± SD. n = 3. All immunofluorescence and TEM images shown are representative. $^{*}p < 0.05$ vs. control group, $^{\#}p < 0.05$ vs. Ang II group