Supporting Information

Astilbin attenuates hyperuricemia and ameliorates nephropathy in fructose-induced hyperuricemic rats

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Fig. 1S Astilbin attenuated the level of CTGF in fructose-induced hyperuricemic rats. A–F: Representative micrographs of anti-CTGF staining in kidney tissue of control (A), fructose-treated (B), allopurinol 4 mg/kg (C), astilbin 1.25 mg/kg (D), astilbin 2.5 mg/kg (E), and astilbin 5.0 mg/kg (F). Renal tubular epithelial cells express CTGF (arrow) (original magnification × 200).
**Fig. 2S** Astilbin attenuated the level of TGF-β1 in fructose-induced hyperuricemic rats.

A–F: Representative micrographs of anti-TGF-β1 staining in kidney tissue of control (A), fructose-treated (B), allopurinol 4 mg/kg (C), astilbin 1.25 mg/kg (D), astilbin 2.5 mg/kg (E), and astilbin 5.0 mg/kg (F). Renal tubular epithelial cells express TGF-β1 (arrow) (original magnification × 200).
**Fig. 3S** Effect of astilbin on pathological progress of renal morphology.

A-F: Representative micrographs of kidney tissue stained with HE from control (A), fructose-treated (B), allopurinol 4 mg/kg (C), astilbin 1.25 mg/kg (D), astilbin 2.5 mg/kg (E), and astilbin 5.0 mg/kg (F). Uric acid crystals (asterisks) were deposited within the renal tubules, renal inflammatory cells were infiltrated into interstitium, renal tubules (arrow) were atrophied, and brush border of epithelial cells remarkably disappeared (original magnification × 200).