

Attenuation of Myocardial Fibrosis with Curcumin Is Mediated by Modulating Expression of Angiotensin II AT1/AT2 Receptors and ACE2 in Rats [Corrigendum]

Pang X, Zhang L, Bai F, et al. *Drug Des Devel Ther.* 2015;9:6043–6054.

The authors have advised there is an error in the Western blot bands shown in Figure 6 on page 6050, pSmad2 at 2 weeks.

Each band shown in Figure 6 represents one animal, and six or seven animals were used for each group. The statistical difference was calculated from the protein analysis using every animal tissue sample. Once the overall group statistical difference was found, three bands were chosen

to represent the difference more closely between the control (Con) and curcumin (Cur) groups. The authors acknowledge the error in using the same bands in lanes two and three to show pSmad 2 at 2 weeks in the Con group. The data have been newly and independently validated from additional samples, and the results have demonstrated symmetrical expression of pSmad2 in the Con group as shown in the correct Figure 6 below.

The authors apologize for this error.

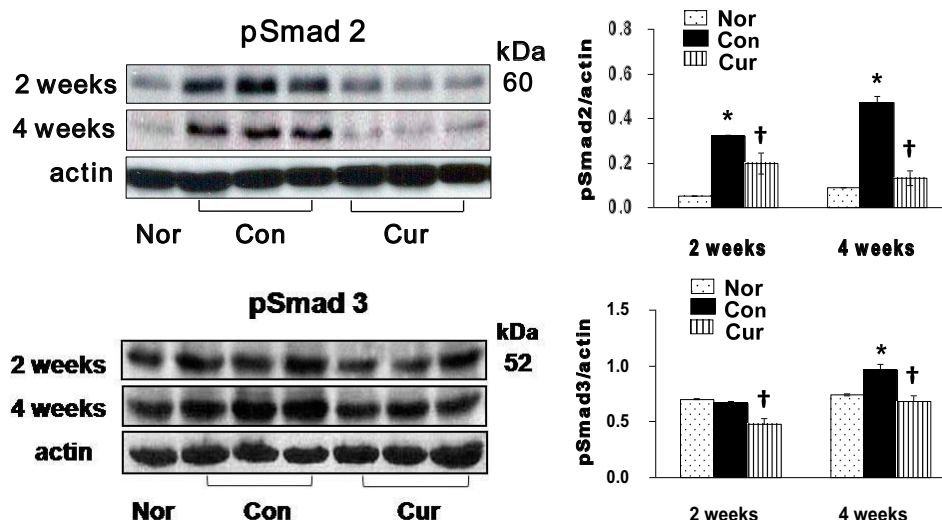


Figure 6 Phosphorylation of Smad2/3 during Ang II infusion. Ang II caused a significant increase in phospho-Smad2/3 levels at week 4, as normalized by actin for each band, which were significantly inhibited by curcumin.

Notes: Values are mean \pm SEM; n=6 for each group. * $P<0.05$ Ang II infusion (Con) vs sham (Nor); # $P<0.05$ curcumin (Cur) vs Con.

Abbreviations: pSmad, phosphorylated Smad; Ang II, angiotensin II; Con, control Ang II infusion treatment group; Nor, normal sham curcumin treatment group; Cur, curcumin plus Ang II treatment group; n, number of rats; SEM, standard error of the mean.

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