

Relaxin inhibits cardiac fibrosis and endothelial–mesenchymal transition via the Notch pathway [Corrigendum]

Zhou X, Chen X, Cai JJ, et al. *Drug Des Devel Ther*. 2015;9: 4599–4611.

On page 4600, the product type and source of the relaxin used in these experiments was missed from the ‘Materials and methods’ section.

Under the subheading ‘Rat model of cardiac fibrosis’ the second paragraph should read:

Rats were randomly divided into five groups (ten per group) for treatment: control; myocardial fibrosis (isoproterenol

[Iso]); and low-, middle-, and high-dose RLX (0.2, 2, and 20 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$, respectively). RLX was obtained from Peprtech, Rocky Hill, NJ, USA (product number 130-15). For the Iso-model, on days 1–6, Iso (5 $\text{mg}\cdot\text{kg}^{-1}\cdot\text{d}^{-1}$; Sigma-Aldrich Co., St Louis, MO, USA) was injected subcutaneously in the rats. In the therapeutic groups, Iso administration was the same as in the Iso-model group, and RLX at different concentrations (0.2, 2.0, and 20 $\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$) were injected at the same time as Iso injection and lasted for 6 days, then RLX injection was continued for another 8 days. The same volume of saline was injected for controls.

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