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Correction to “Inhibiting heme oxygenase-1 attenuates rat liver fibrosis by removing iron accumulation”

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Abstract

AIM: To investigate the effects of the heme oxygenase (HO)-1/carbon monoxide system on iron deposition and portal pressure in rats with hepatic fibrosis induced by bile duct ligation (BDL) .

TO THE EDITOR

We found a mistake in Figure 6. Panels A (Sham group) and F (DFX group) (180 degrees rotated) is same images. We have replaced the incorrect images (Panels F) with the correct Figure. We only revised the incorrect figure, and the Manuscript NO: 75978 don't need to revise. **1**
METHODS: Male Sprague-Dawley rats were divided randomly into a Sham group, BDL group, Fe group, deferoxamine (DFX) group, zinc protoporphyrin (ZnPP) group and cobalt protoporphyrin (CoPP) group. The levels of HO-1 were detected using different methods. The **1** serum carboxyhemoglobin (COHb), iron, and portal vein pressure (PVP) were also quantified. The plasma and mRNA levels of hepcidin were measured. **1** Hepatic fibrosis and its main pathway were assessed using Van Gieson's stain, hydroxyproline, transforming growth

factor- β 1 (TGF- β 1), nuclear factor-E2-related factor 2 (Nrf2), matrix metalloproteinase-2 (MMP-2) and tissue inhibitor of metalloproteinase-1 (TIMP-1). RESULTS: Serum COHb and protein and mRNA expression levels of HO-1 and Nrf2 were increased in the BDL group compared with the Sham group and were much higher in the CoPP group. The ZnPP group showed lower expression of HO-1 and Nrf2 and lower COHb. The levels of iron and PVP were enhanced in the BDL group but were lower in the ZnPP and DFX groups and were higher in the CoPP and Fe groups. Hepcidin levels were higher, whereas superoxide dismutase levels were increased and malonaldehyde levels were decreased in the ZnPP and DFX groups. The ZnPP group also showed inhibited TGF- β 1 expression and regulated TIMP-1/MMP-2 expression, as well as obviously attenuated liver fibrosis. CONCLUSION: Reducing hepatic iron deposition and CO levels by inhibiting HO-1 activity through the Nrf2/Keap pathway could be helpful in improving hepatic fibrosis and regulating PVP.

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