Correction: *Signally via the osteopontin and high mobility group box-1 axis drives the fibrogenic response to liver injury*

Arriazu E, Ge X, Leung T-M, *et al*. Signally via the osteopontin and high mobility group box-1 axis drives the fibrogenic response to liver injury. *Gut* 2017;66:1123–37. doi: 10.1136/gutjnl-2015-310752.

Panel B in figure 6 is incorrect and has been removed. The updated figure and legend should be:

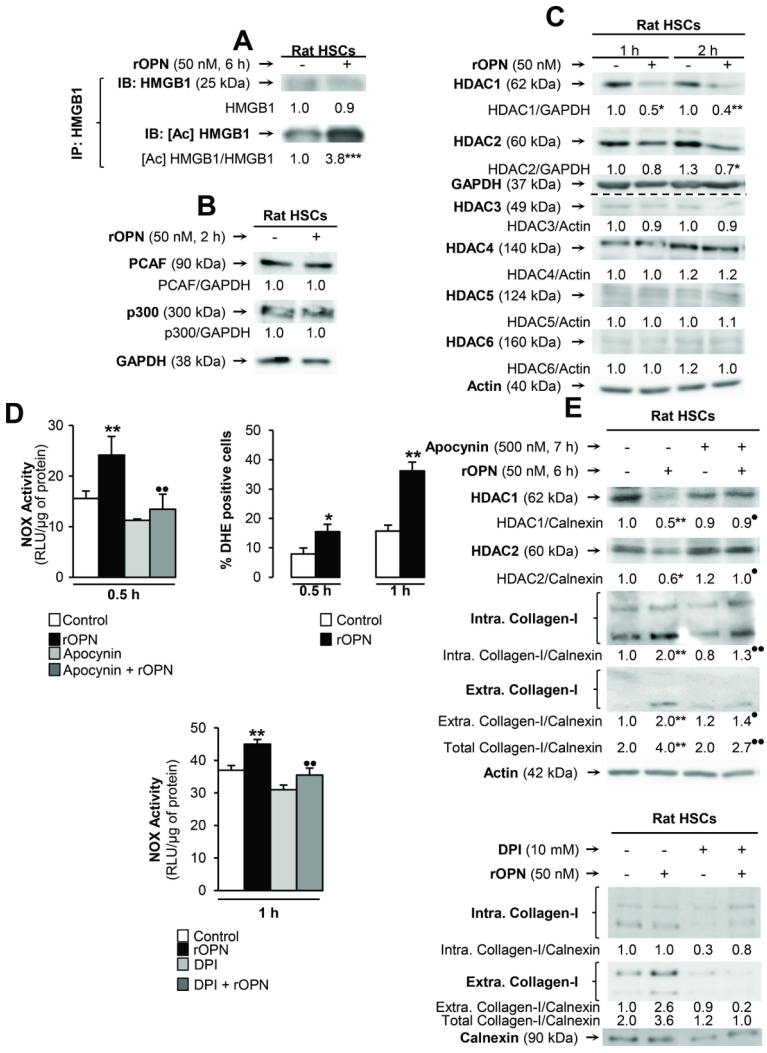

Figure 6 rOPN activates NOX and inhibits HDACs1/2 promoting HMGB1 acetylation and translocation along with collagen-I up-regulation in HSCs. Rat HSCs were treated with rOPN for 6 h. Immunoprecipitation of intracellular HMGB1 and immunoblotting for acetylated lysines (A). Rat HSCs were treated with rOPN for 2 h. Western blot analysis for PCAF and p300 (B). Rat HSCs were treated with rOPN for 1 and 2 h. Western blot analysis for HDACs1-6 (C). NOX activity in rat HSCs treated with rOPN for 6 h alone or pretreated for 0.5 h with apocynin or DPI, two NOX inhibitors. The percentage of DHE positive cells was measured by flow cytometry as an indirect measurement of $O_2^{\cdot-}$ production (D). Rat HSCs were treated with rOPN for 6 h in the presence or absence of apocynin or DPI. Western blot analysis of HDACs1/2 along with intra- and extracellular collagen-I (E). The results from the western blot analysis are corrected by the specific loading control and are expressed as fold-change of the controls, which are assigned a value of 1 and are mean values±SEM; n=3/group in experiments performed in triplicate four times. *P<0.05, **p<0.01 and ***p<0.001 for rOPN versus control; •p<0.05 and ••p<0.01 for co-treated versus rOPN.

OPEN ACCESS

Gut 2020;**69**:e2. doi:10.1136/gutjnl-2015-310752corr1