

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:

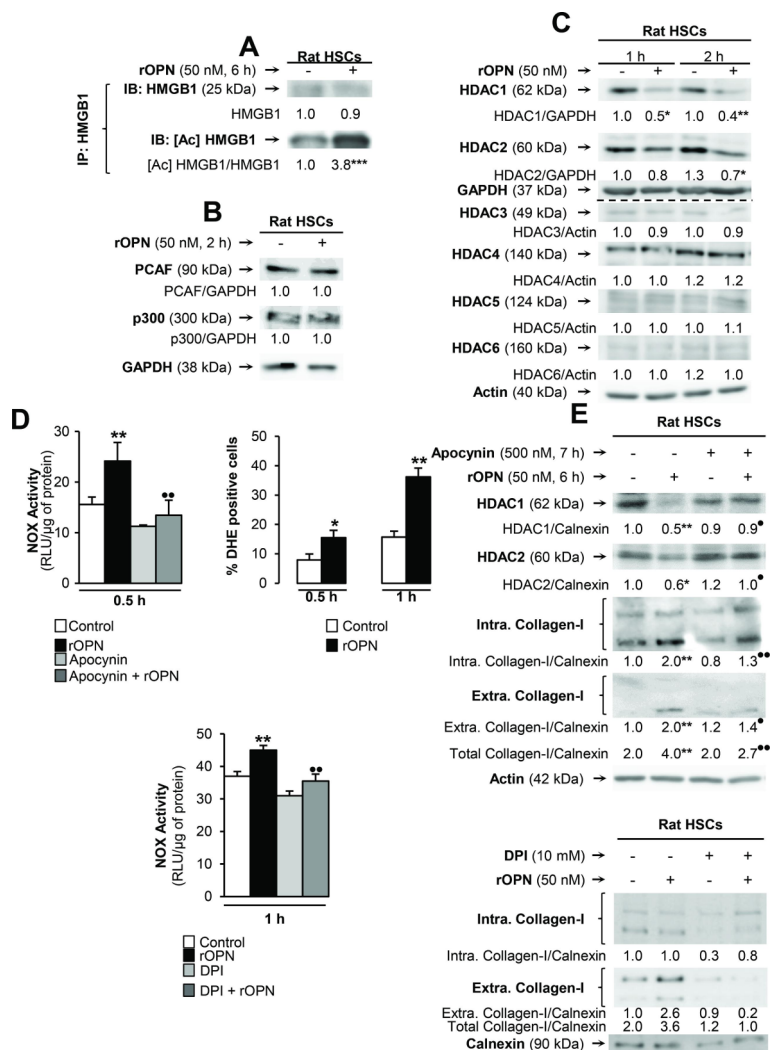


Figure 6 rOPN activates NOX and inhibits HDACs1/2 promoting HMGB1 acetylation and translocation along with collagen-I up-regulation in HSCs. Rat HSCs were treated with rOPN for 6 h. Immunoprecipitation of intracellular HMGB1 and immunoblotting for acetylated lysines (A). Rat HSCs were treated with rOPN 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^- 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 \pm 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.



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

