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Supplemental Data

The Protective Effect of Heme Oxygenase-1 against Intestinal Barrier Dysfunction in Cholestatic Liver Injury Is Associated with NF-κB Inhibition

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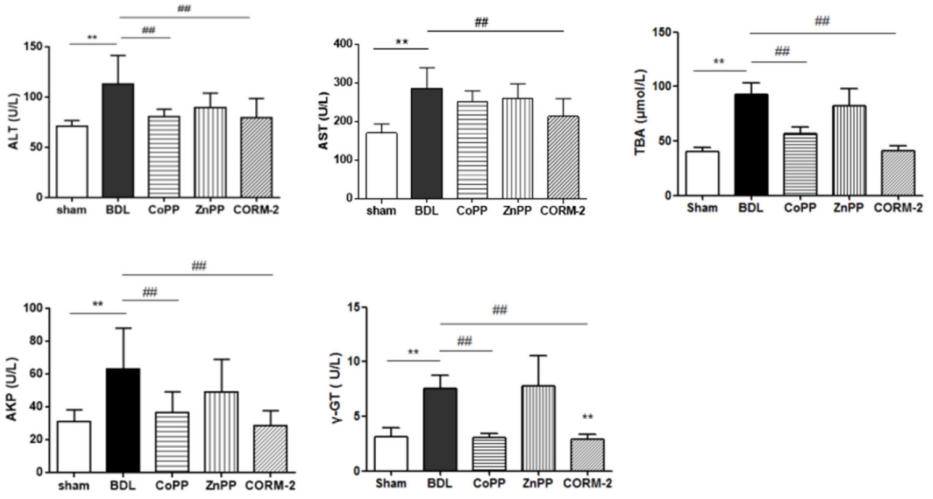
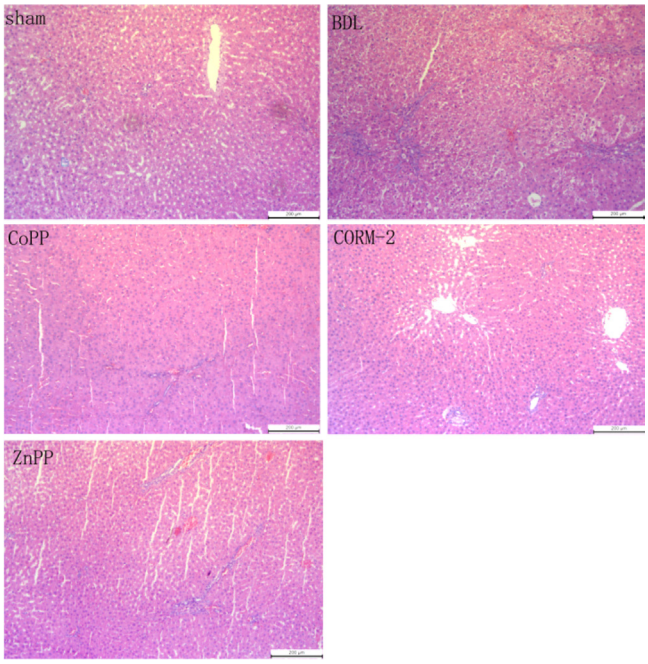
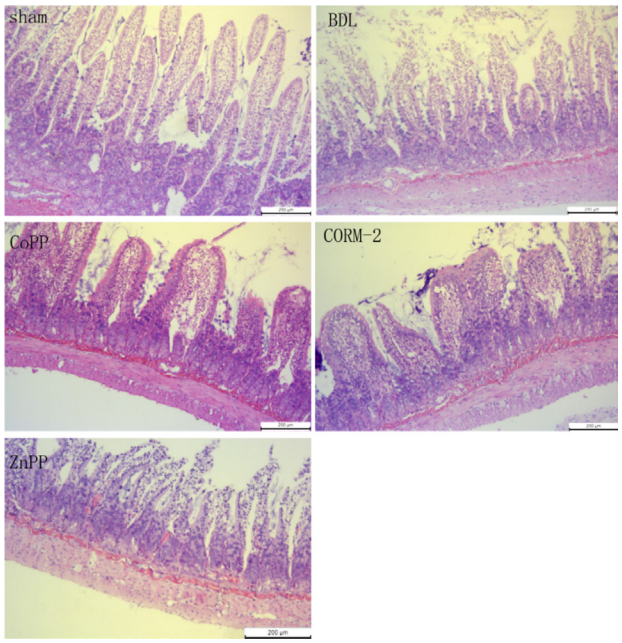


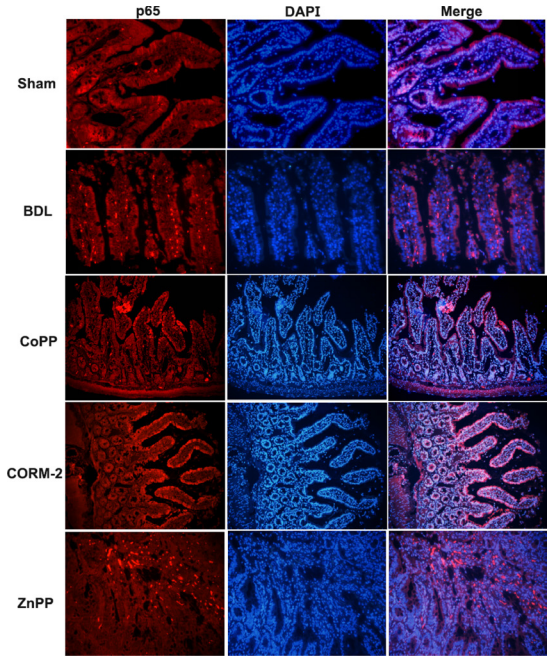
Supplementary Figure S1. Biochemical results. Serum levels of ALT, AST, TBA, AKP and γ-GT in Sham/BDL rats. ALT, AST, TBA, AKP and γ-GT were significantly higher in the BDL group than in the Sham group, ** $P < 0.01$. In the CoPP and CORM-2 treatment groups, ALT, AKP, γ-GT and TBA was obviously decreased compared with the BDL group, $^{\#\#}P < 0.01$.



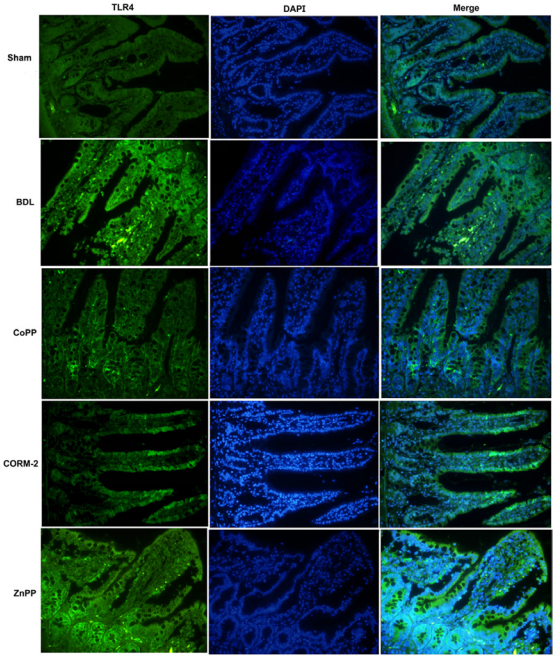
Supplementary Figure S2. Pathological images of the liver with hematoxylin and eosin staining in Sham/BDL rats. Normal lobular architecture in the Sham and BDL group exhibited different degrees of fibrosis. In the CoPP and CORM-2 groups, the liver injury was relieved.



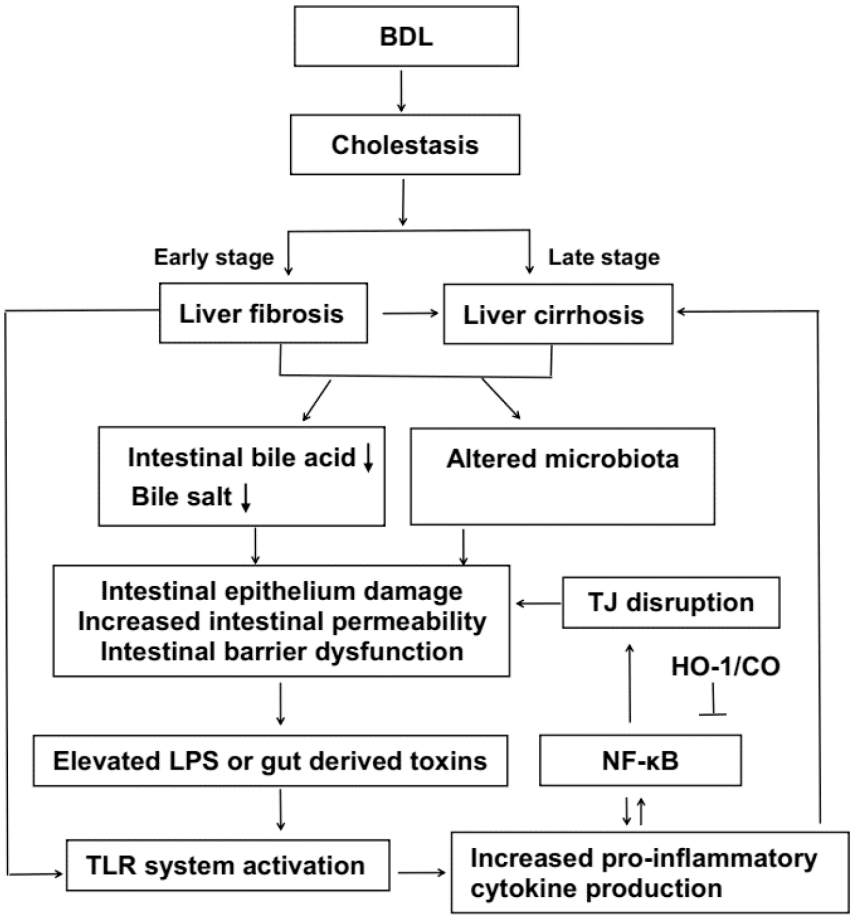
Supplementary Figure S3. Pathological images of the intestine in Sham/BDL rats with hematoxylin and eosin staining. In the BDL group, intestinal epithelial cell layer degeneration, necrosis and shedding were observed, and the intestinal villi were not well arranged. In the CoPP and CORM-2 groups, the injury was relieved and the intestinal mucosa showed obvious edema. There was no obvious difference between the BDL and ZnPP groups.



Supplementary Figure S4. Immunofluorescent staining results for NF-κB p65 in intestinal tissue from Sham/BDL rats. Red is p65; blue is DAPI (nuclei). p65 was mainly located in the cytoplasm of the intestinal epithelial cell in Sham group, while in the BDL rats, nuclear p65 expression was significantly increased. However, there was less nuclear translocation in CoPP/CORM-2-treated BDL rats than in BDL rats.



Supplementary Figure S5. Immunofluorescence staining results for TLR4 in Sham/BDL rats. Green is TLR4; blue is DAPI (nuclei). The immunofluorescence staining of TLR4 occurred in the cytoplasm. The rats subjected to BDL exhibited more intense TLR4 immunofluorescence, while less in CoPP and CORM-2 treatment group.



Supplementary Figure S6. HO-1/CO protects intestinal barrier in BDL rats through regulation of NF-κB. BDL exhibits cholestasis, liver fibrosis and ultimately lead to liver cirrhosis, which affect intestinal barrier function. Gut-derived products/toxins activate toll-like receptors with a subsequent production of proinflammatory mediators and systemic inflammation contributing to the development of liver disease through regulation of NF-κB. Abbreviations: BDL, bile duct ligation; LPS, lipopolysaccharides; TLR,Toll-like receptor; TJ, tight junction; NF-κB, nuclear factor kappa B; HO-1:heme oxygenase-1; CO: carbon monoxide.