



Lactobacillus reuteri-mediated dietary xylooligosaccharides enhance jejunal cell survival via suppression of oxygen-dependent apoptotic processes in a pig model

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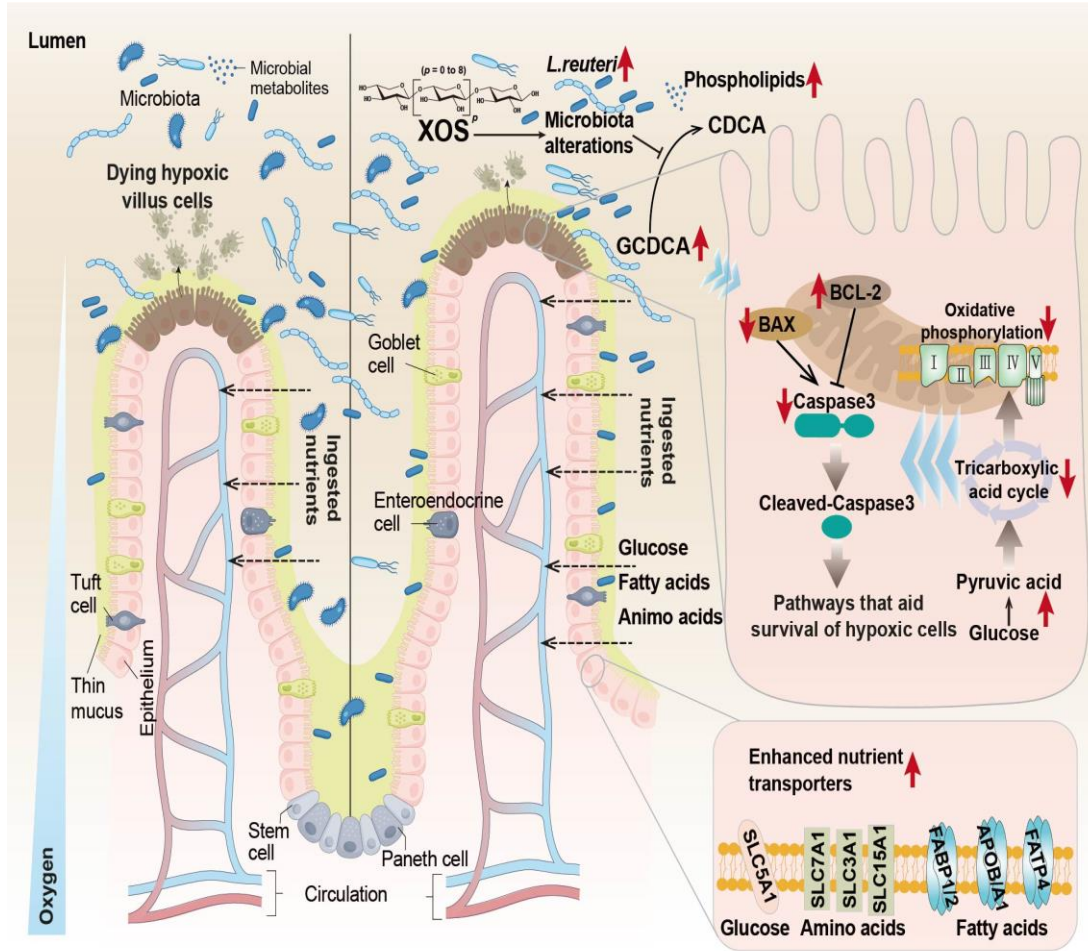
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Background

- ❑ The balance of the gut microbiota is essential for mammalian growth and development
- ❑ There is a contradiction between the traditional understanding of functional oligosaccharides and emerging discoveries
- ❑ The scope and mechanisms of xylooligosaccharides (XOS) on intestinal regulation need to be re-examined

Highlights



- ❑ It was confirmed that XOS inhibits the apoptosis process of epithelial cells by changing the composition of jejunal microorganisms and reshaping the energy metabolism
- ❑ It was clarified that *L.reuteri* is the core microbiota mediating the regulation of XOS on jejunal health, and GCDCA is the key effector metabolite of *L.reuteri* in mediating anti-apoptosis
- ❑ It provides new insights into the molecular mechanisms of crosstalk among oligosaccharides, gut microbiota, and epithelial cells



XOS improve growth performance by enhancing small intestinal morphology in piglets

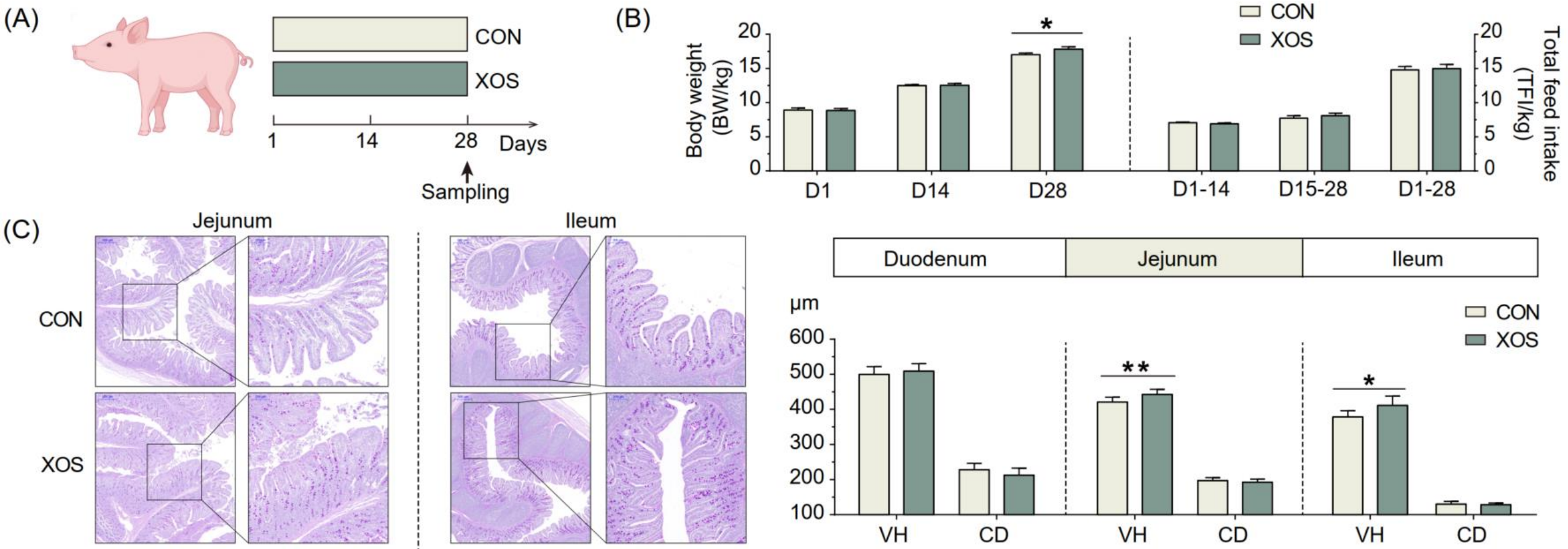
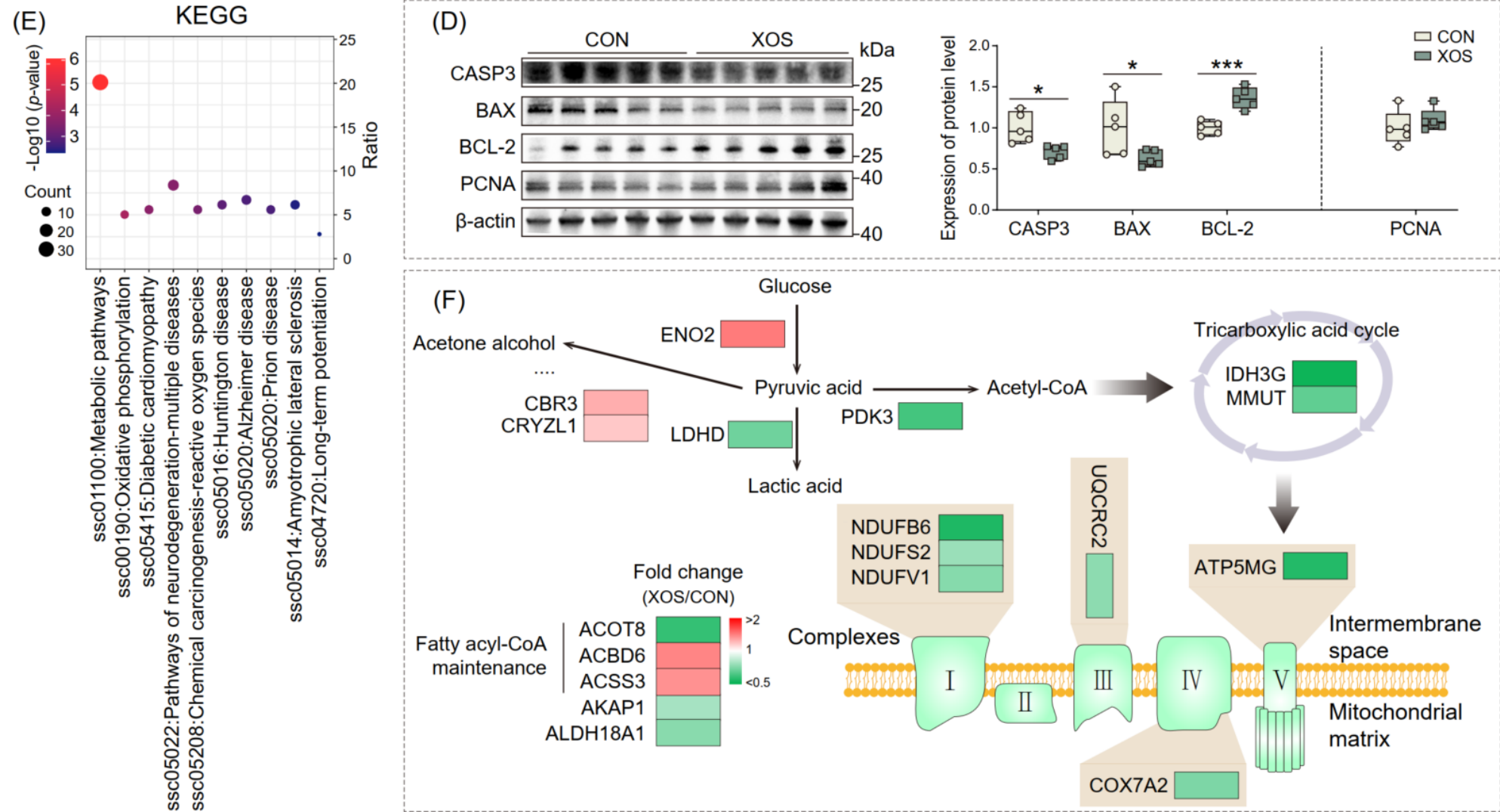


Figure 1. (A) Schematic diagram of the experimental design for continuous feeding of weaned piglets with Xylooligosaccharides (XOS) for 28 days. CON: basal diet; XOS: basal diet supplemented with 500 mg/kg XOS. (B) Growth performance of piglets after XOS supplementation. (C) Quantitative indicators and representative images of small intestinal morphology.

XOS improves jejunal morphology by inhibiting intestinal epithelial cell apoptosis and regulating energy metabolism in piglets



L.reuteri is enriched in the jejunal mucosa and lumen of XOS-fed piglets

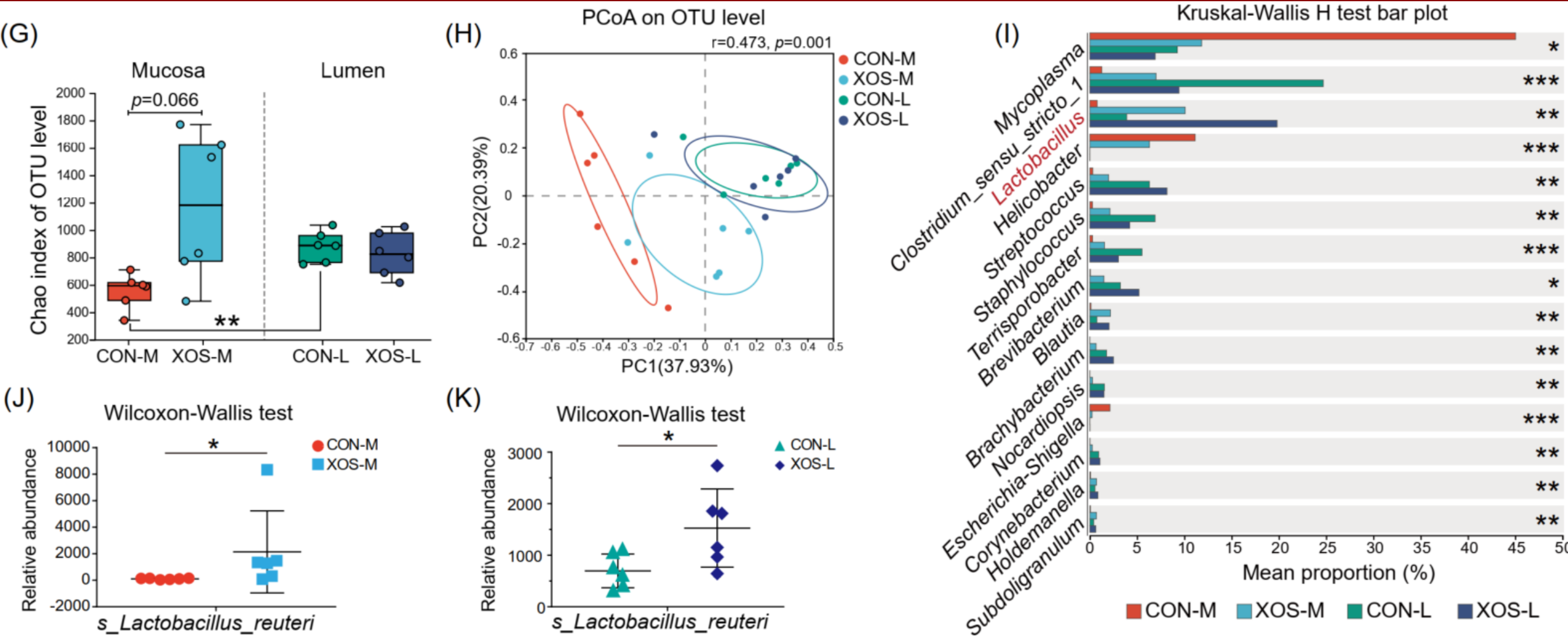


Figure 1. XOS optimize microbial composition. (G) α diversity (Chao index) and (H) β diversity (PCoA) of jejunal mucosa and lumen microbiota at the OTU level. (I) Microbial composition at the genus level. Abundance of *Lactobacillus reuteri* (*L. reuteri*) in jejunal (J) mucosa and (K) lumen.

L.reuteri relieves jejunal morphological damage and inhibits epithelial apoptosis in piglets

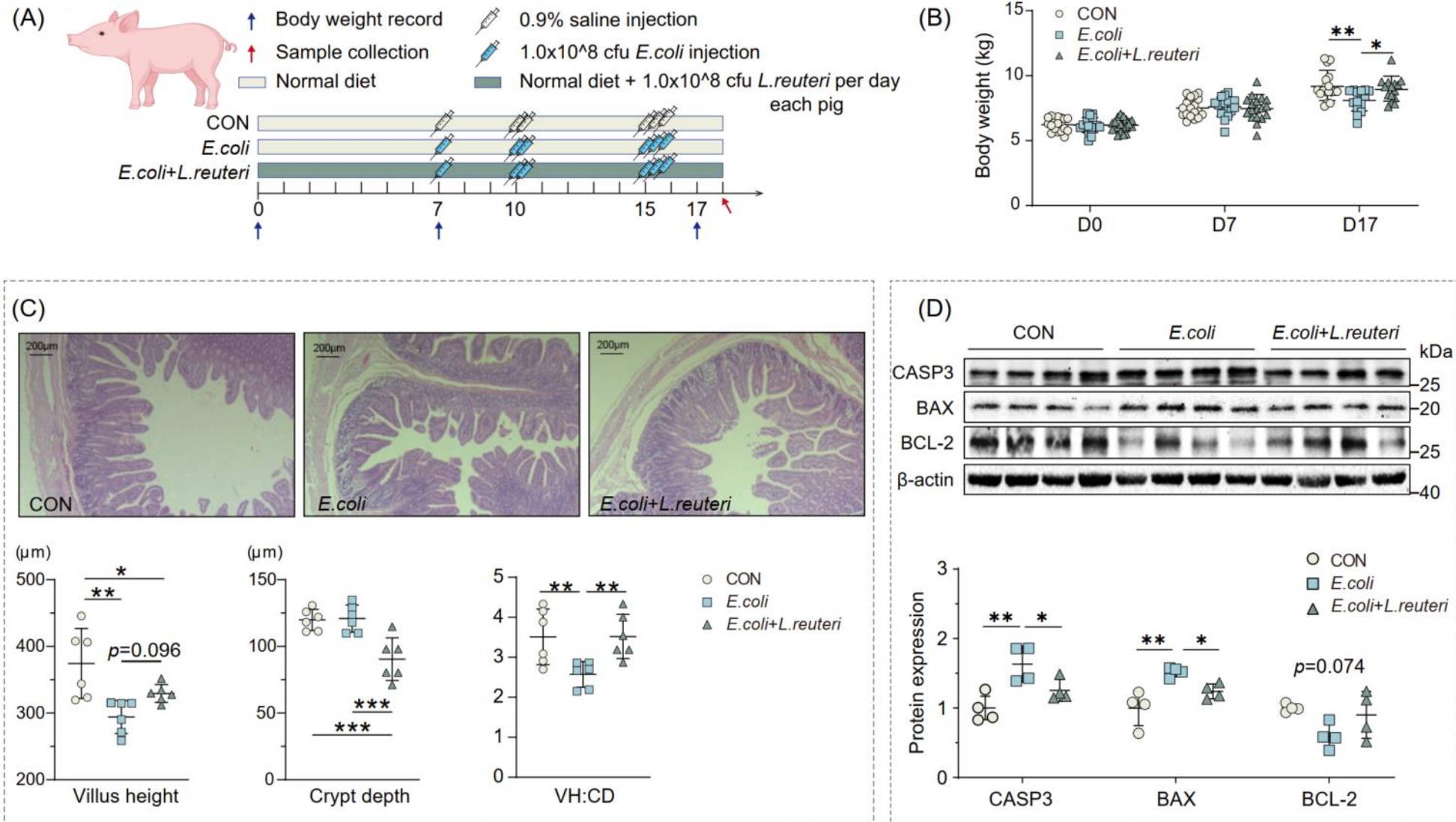


Figure 2. (A) Schematic diagram of the experimental design for *L. reuteri* supplementation in *Escherichia coli* (*E. coli*)-challenged piglets. (B) Body weight, (C) quantitative indicators and representative images of jejunal morphology, and (D) protein levels of apoptosis-related genes in the jejunal mucosa of piglets after *E. coli* injection (with or without *L. reuteri* supplementation).

GCDCA is a key effector metabolite of *L.reuteri*-mediated anti-apoptosis

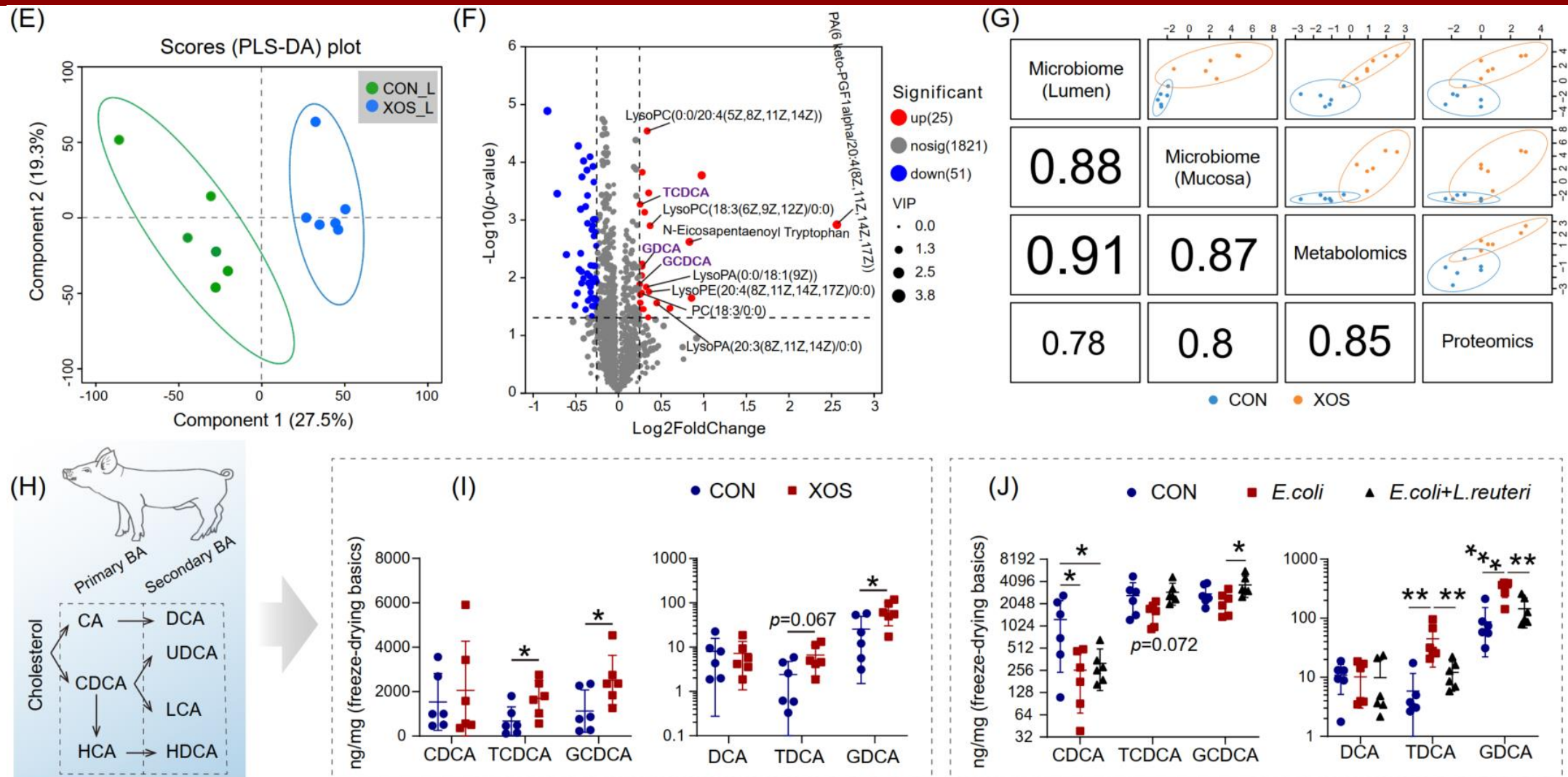


Figure 2. Metabolomic analysis of jejunal mucosa from XOS-fed pigs. (E) PLS-DA score plot and (F) volcano plot of differentially abundant metabolites. (G) Global correlation structure among proteome, microbiome (jejunal lumen and mucosa), and metabolome (jejunal digesta) revealed by DIABLO. (H) Bile acid composition in the pig model. Targeted metabolomics validated the content of bile acids in jejunal digesta from (I) XOS-fed and (J) *E. coli* + *L. reuteri* groups.

Cell experiments confirmed that GCDCA inhibits the hypoxia-induced apoptosis of jejunal epithelial cells

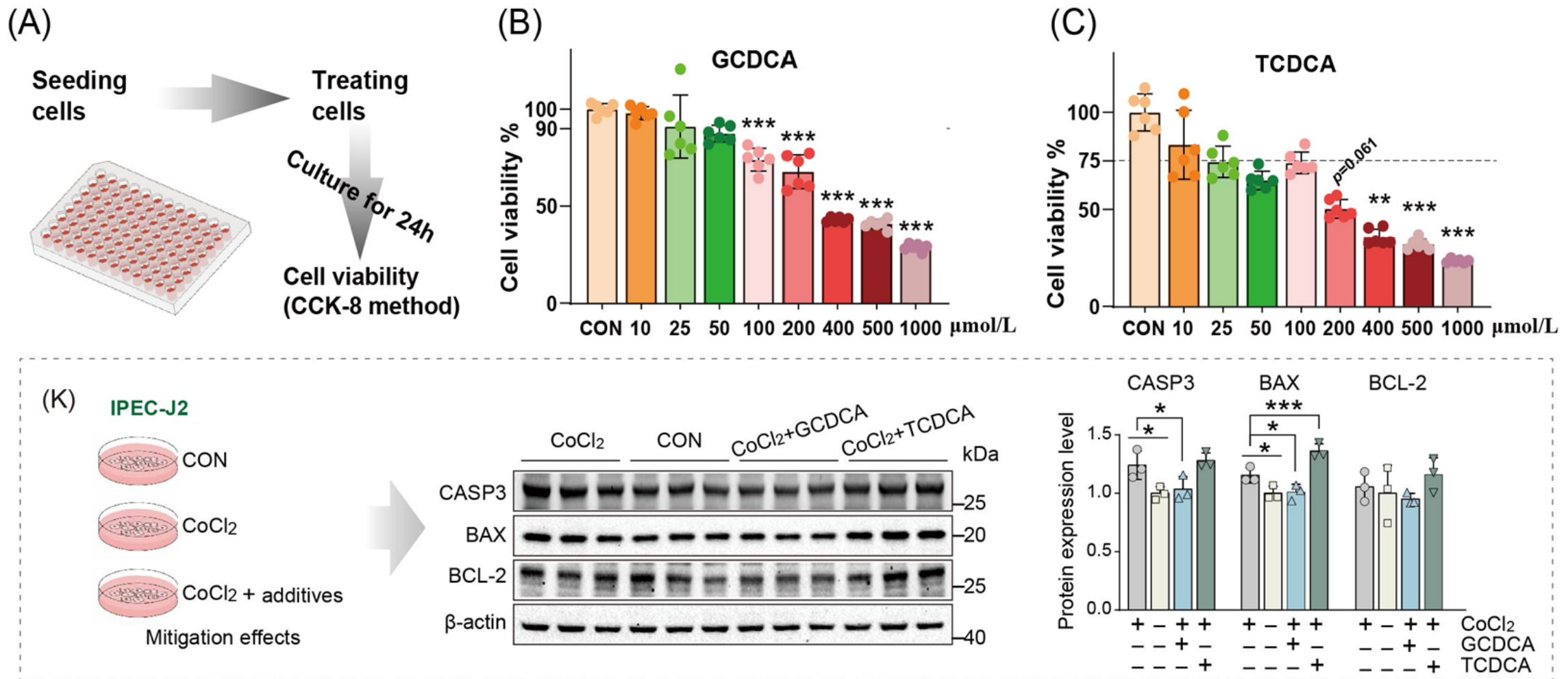


Figure S14. (A) Process of cytotoxicity experiment of additives. Cytotoxicity of exogenous (B) GCDCA and (C) TCDCA measured by the CCK-8 assay. Figure 2. (K) Relative expression of apoptosis-related proteins in IPEC-J2 cells under CoCl₂-induced hypoxia (300 μM) with or without GCDCA/TCDCA supplementation (0.1 μM).



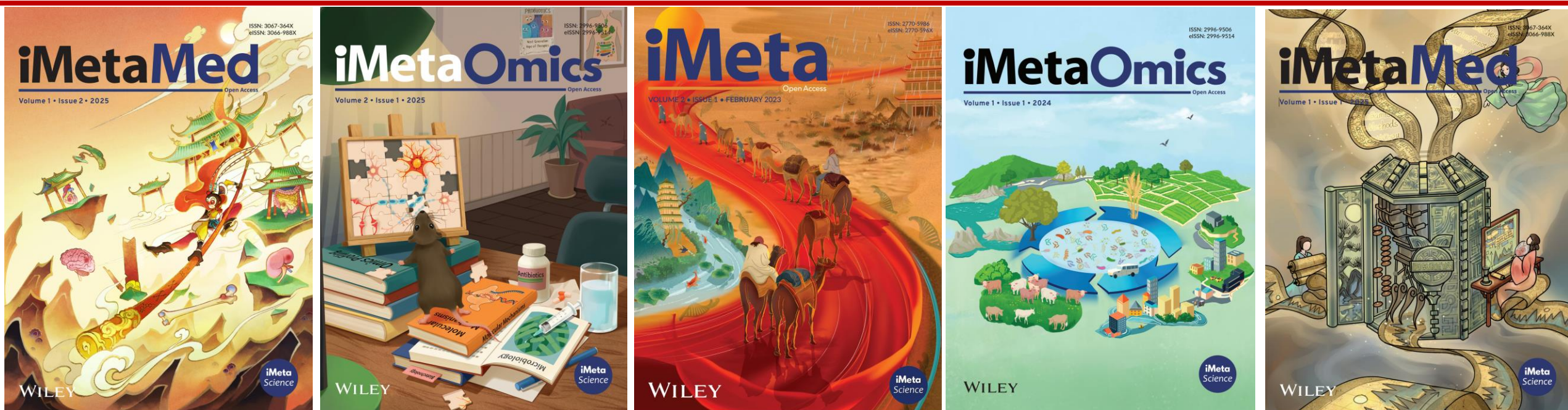
Summary

- ❑ Dietary XOS lengthens jejunal villi and promotes nutrient absorption, thereby improving growth performance in pigs
- ❑ The lengthening of jejunal villi results from improved cell survival, achieved through the remodeling of energy metabolism, reduced oxygen demand, and the subsequent inhibition of epithelial cell apoptosis
- ❑ The dietary XOS-induced increase in *Lactobacillus reuteri* improves jejunal cell survival by inhibiting oxygen-dependent apoptotic processes
- ❑ Mechanistically, the increased survival of intestinal epithelial cells under hypoxic conditions is related to the anti-apoptotic efficacy of GCDCA

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