Results Cana reduced the lipid (such as TG, TC, and LDL-C) accumulation in serum, thus decreased atherosclerotic index of plasma and arteriosclerosis index values. More importantly, Cana decreased the thickness of the vascular basement membrane, improved cardiac mitochondrial homeostasis, and relieved oxidative stress (e.g. regulation of ROS, SOD, GSH, and MDA levels). And, Cana reduced the circulating markers of inflammation (such as TNF-α, MCP-1, and IL-6). Myocardial injury was alleviated after Cana treatment with decreasing levels of serous cTnI, MCP-1, and IL-6. Interestingly, Cana administration increased the ratio of Firmicutes/Bacteroidetes (from 230% to 98%) and the relative abundance of Olsenella, Alistipes, and Alloprevotella, while decreasing the abundance of Helicobacter and Mucispirillum in mice with diabetic CVD.

Conclusions Cana treatment improved CVD by decreasing the risk of atherosclerosis and reducing the thickness of the vascular basement membrane. Importantly, Cana treatment significantly elevated myocardial mitochondria homeostasis, thus ameliorated the oxidative stress and inflammatory states. Moreover, Cana subtly altered microbiota composition in T2DM mice with CVD, which contributed to the improvement of CVD. Collectively, the improvements of myocardial mitochondrial and gut microbiota homeostasis, may represent an important mechanism underlying the cardiovascular benefits of Cana treatment.

Background Bacillus amyloliquefaciens (BA) decomposes starch by producing alpha-amylase to produce short-chain fatty acids (SCFA). Resistant starch (RS) can be fermented by colonic microbiota and metabolized to SCFA containing acetic acid and butyric acid by which can ameliorate intestinal inflammation. In this study, dextran sulfate sodium (DSS)-induced colitis mouse model was administrated to assess the therapeutic effect of BA combined with RS in experimental colitis.

Methods 6-week-old male BALB/c mice were given 3% DSS drinking water for 1 week to induce acute colitis. Mice were fed with maintenance forage as control group. The content of RS was 465.692g/kg, and the abundance of BA by gavage administration in mice was 1*10⁷ CFU for 7 days. The mice were sacrificed on the eighth day. We collect specimens, measure colon length, evaluate histopathologic score and detect the level of IL-1β, IL-6, TNF-α, and occludin of intestinal tissue by qPCR.

Results Weight loss and DAI were more effectively improved in the live BA combined with RS than administered RS alone. Colonic inflammation was significantly alleviated in the live BA combined with RS. The colon length and histopathologic score of live BA combined with RS intervention were more improved than that of the control (p<0.001), including a smaller extent of intestinal epithelial damage and crypt structure disorder. The degree of inflammatory cell infiltration was lighter (p=0.029). The expression of IL-1β (p=0.008), IL-6 (p=0.02) and TNF-α (p<0.001) were reduced and the expression of Occludin (p =0.006) was increased than RS alone.

Conclusions The intervention of BA combined with RS may ameliorate DSS-induced acute colitis and promote colon barrier recovery.