Salmonella spp. remain major public health problems for the whole world. A better understanding of pathogenesis of these food-borne pathogens is a prerequisite for the design of improved intervention strategies that could reduce the use of antimicrobial agents and drug-resistant Salmonellosis. Increasing studies suggested 1,25-dihydroxyvitamin D3 (1,25D3), the active form of vitamin D, was effective in ameliorating colitis via the lumen of the intestinal tract. Stimulation of NO2 expression by 1,25D3-stimulated antimicrobial peptides production enhancing autophagy imply that vitamin D would boost autophagy. Therefore, we aims to investigate the effect of active vitamin D3 on the severity of Salmonella colitis.

Salmonella colitis model was conducted with 6–8 wk-old male C57BL/6 mice: Streptomycin -pretreated C57BL/6 mice were mock infected with sterile PBS or infected orally with S. Typhimurium wild-type strain SL1344 for48 h. Mice were randomly assigned to control, model and 1,25(OH)2D3 treated group. At the end of the experiment, mice were sacrificed; tissue samples from the intestinal tracts, spleens, and livers were collected. The bacteria colonicisation (CFU/mg tissue) in liver (1.02 ± 0.20´ 10 2 vs. 17.24 vs. 129.93 ± 18.05, p < 0.0001), but enhanced the autophagy expression in Western blot, comparing to SL1344 infection only. In conclusion, active vitamin D3 could reduce Salmonella colitis by reducing inflammation and bacterial colonisation via autophagy induction.