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-dihydroxyvitaminD3 (1,25D3), the active form of vitamin D, was effective in ameliorating colitis via the lumen of the intestinal tract. Stimulation of NOD2 expression by 1,25D3-stimulated antimicrobial peptides production enhancing autophagy imply that vitamin D would boost autophagy. Therefore, we aimed 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 for 48 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 removed for analysis of bacterial colonisation, Western blot for proteins expression, and RTPCR for mRNA expression.

We observed 1,25D3 reduced the severity of Salmonella colitis in C57BL/6 mice by reducing cecal mL-ibeta (79.36 ± 24.60 vs. 271.40 ± 60.88, p < 0.01), mL-6 (206.32 ± 52.18 vs. 491.74 ± 39.44, p < 0.005) and mTNF-alpha (44.18 ± 17.24 vs. 129.93 ± 18.05, p < 0.005) mRNA expression, bacterial colonisation (CFU/mg tissue) in liver (1.02 ± 0.20 × 102 vs. 4.97 ± 0.66 × 102, p < 0.001) and spleen (1.50 ± 0.42 × 102 vs. 4.54 ± 3.56 × 102, 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.