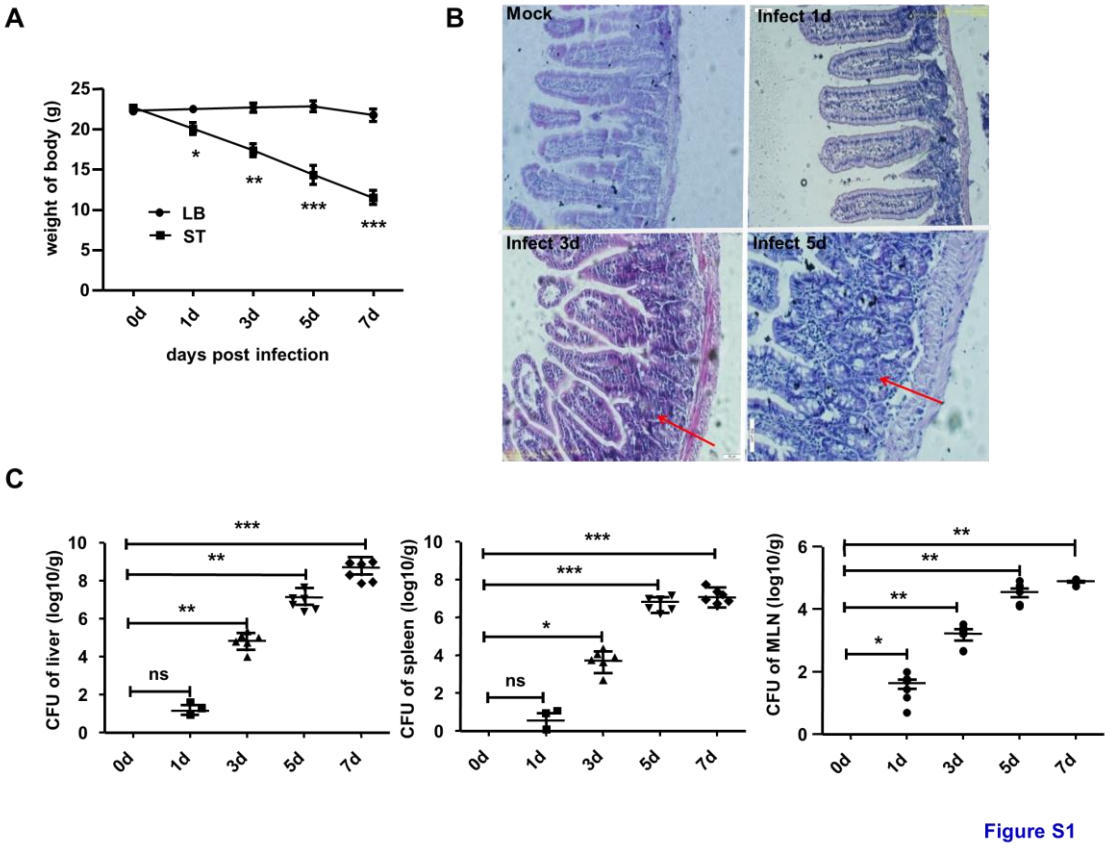
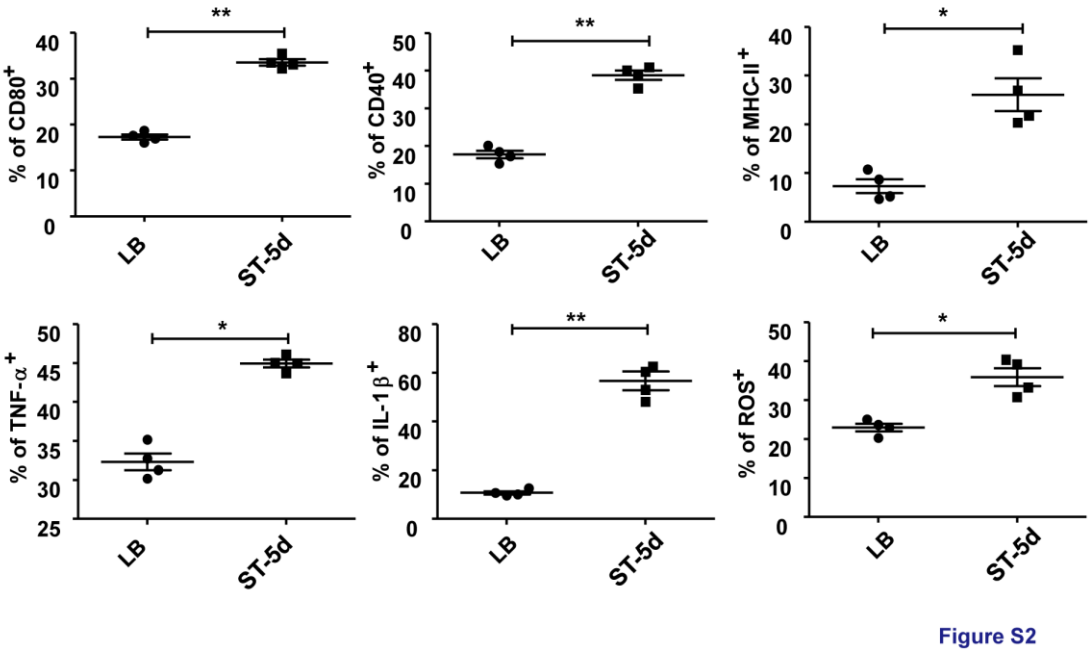
Supplementary Figure Legends



Supplementary Fig 1. Oral *S. typhimurium* infection induces mouse enteritis and system infection. C57BL/6 mice were infected orally with *S. typhimurium* (1×10^5) or mock-infected with Luria-Bertani (LB). Weight loss was evaluated at various time points post-infection (n = 5) (A). Hematoxylin and eosin (H&E)-stained sections of small intestines from infected mice (B). Bacterial loads of the liver, spleen, and MLN were determined by LB plate assay (n = 6) (C). *p < 0.05, **p < 0.01, ***p < 0.001, compared with the group on day 0.



Supplementary Fig 2. Phenotype and activation of macrophage in the Lamina propria of *S. typhimurium*-infected mice. The expression of CD80, CD40, MHC-II and ROS, as well as intracellular TNF-α and IL-1β on/in F4/80$^+$CD11b$^+$ macrophages was detected by FACS and statistically analyzed. Data are representative of three independent experiments (means ±SD). *p<0.05, **p<0.01, ***p<0.001, compared with the LB group.

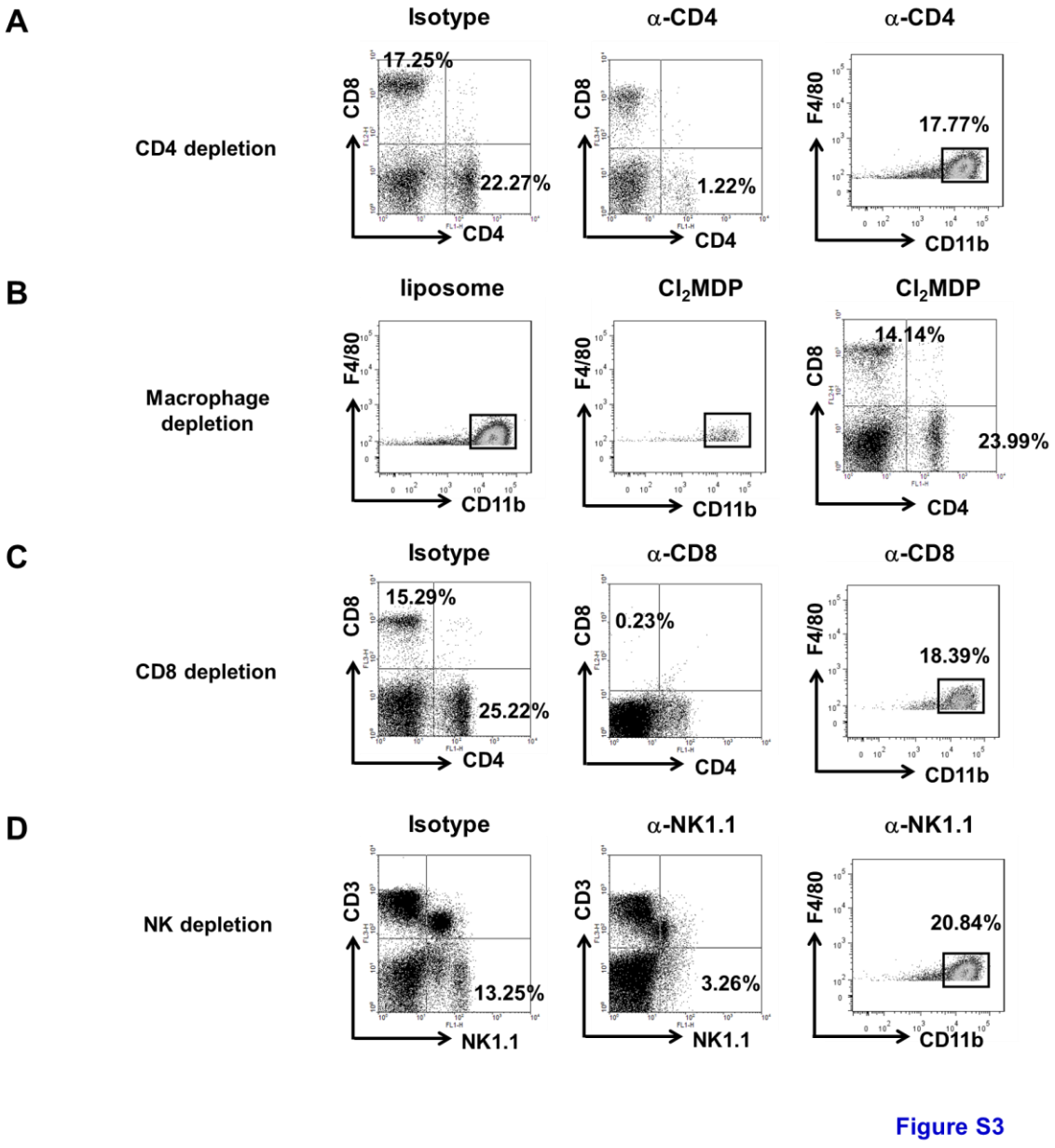


Figure S3

Supplementary Fig 3. The *in vivo* depletion efficiency of $CD4^+$T, $CD8^+$T, macrophages, and NK cells. Depleting antibodies (α-CD4, α-CD8, α-NK1.1) and Cl_2MDP-liposome were injected *i.p*. into C57BL/6 mice. Three days later, the removal efficiency was detected by FACS.

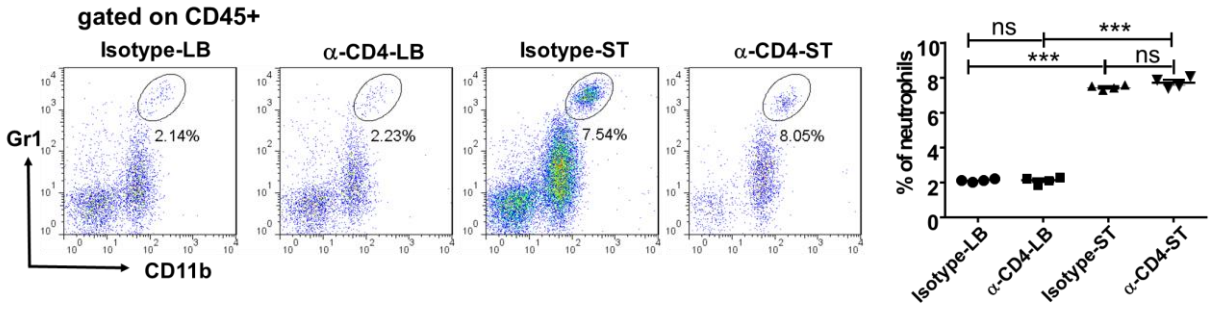


Fig. S4

Supplementary Fig 4. $CD4^+$T cells depletion has no effect on the proportion of intestinal lamina propria neutrophils during *S. typhimurium* infection. α-CD4 antibody were injected *i.p.* into C57BL/6 mice. Three days later, mice were orally infected with *S. typhimurium*. Five days later, the percentage of intestinal neutrophils were detected by FACS. The LP leukocytes were first gated (FSC-H vs. FSC-H) and further stained with 7-AAD to exclude dead cells, leukocytes were then selected by the expression of CD45. The percentage of $Gr1^+CD11b^+$neutrophils were determined by flow cytometry. Data are representative of three independent experiments (means ± SD). ***$p < 0.001$, compared with the control group.

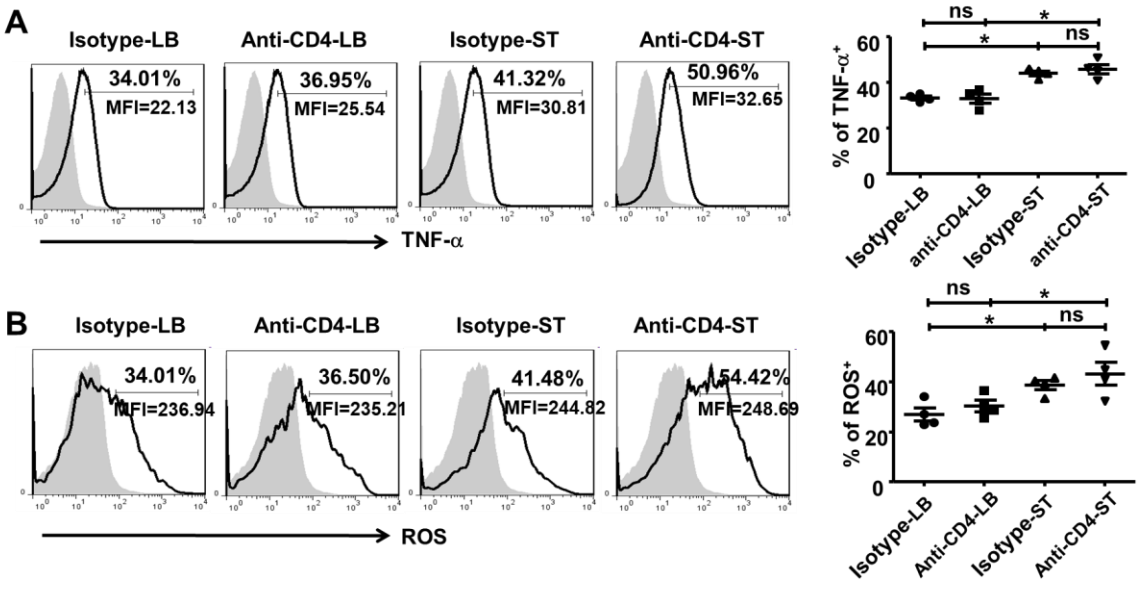


Figure S5

Supplementary Fig 5. Depletion of CD4$^+$T cells did not impair the production of TNF-α and ROS by intestinal macrophages during *S. typhimurium* infection. Mice were injected *i.p.* with anti-CD4 antibodies. Three days later, the treated mice were orally infected with *S. typhimurium*. Five days after infection, the small intestinal LP lymphocytes were isolated and the production of TNF-α (A) and ROS (B) by F4/80+CD11c+ macrophages was detected by flow cytometry. Data are expressed as the means±SD of at least three independent experiments. *$p < 0.05$.

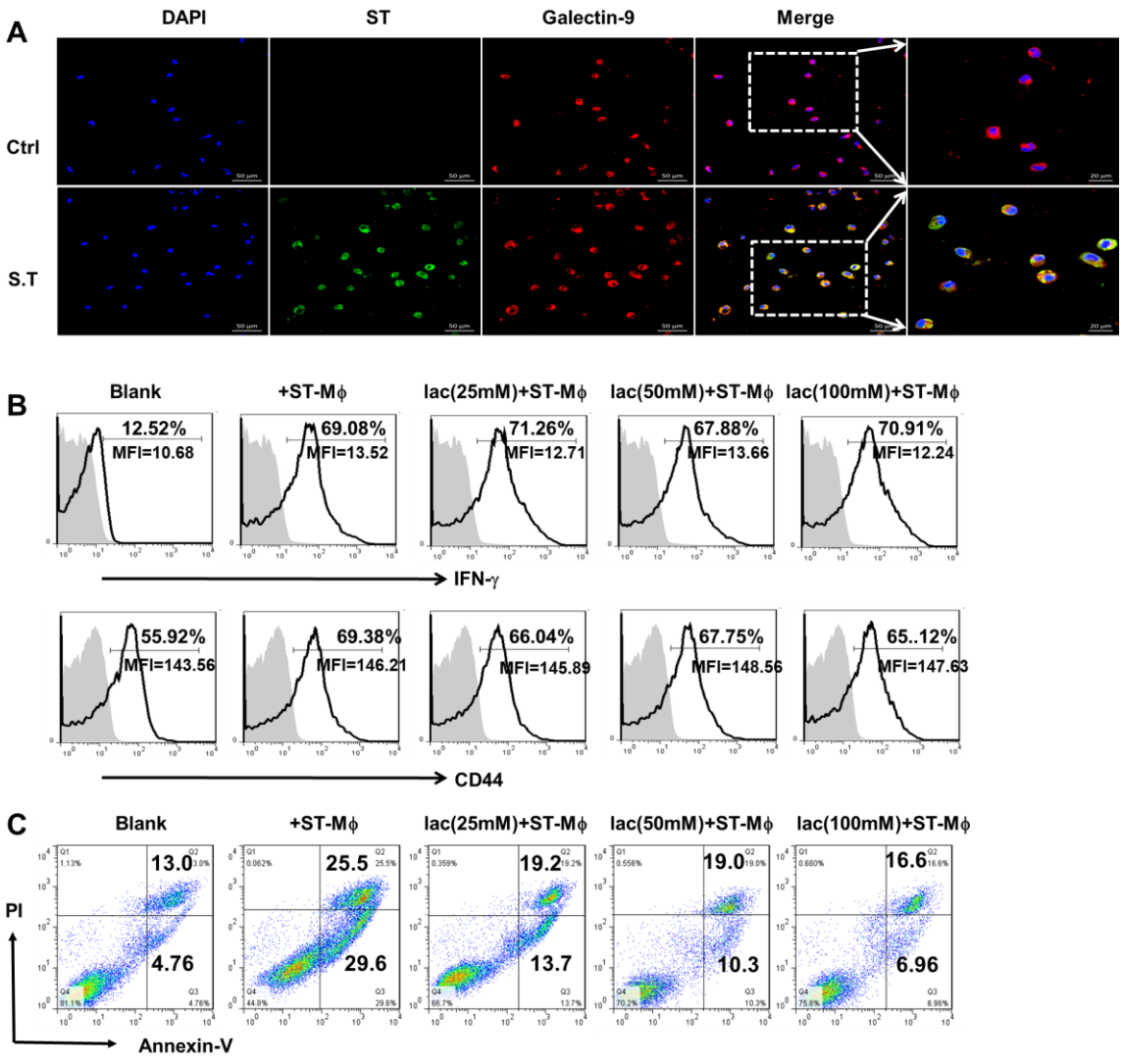


Figure S6

Supplementary Fig 6. Tim-3−galectin-9 pathway is involved in the induction of $CD4^+$T cell apoptosis. LP macrophages were infected with *S. typhimurium* (MOI=10:1). One hour later, the expression of galectin-9 on macrophages was determined by immunofluorescence staining. Green and red colors represent *S. typhimurium* and galectin-9, respectively; blue represents nuclei. (A). $CD4^+$T cells isolated from LP lymphocytes by MACS were co-cultured with *S. typhimurium*-infected macrophages. The production of IFN-γ and expression of CD44 by $CD4^+$T cells were measured by FACS (B). The apoptosis of $CD4^+$T cells were measured by Annexin-V/PI double staining methods (C). Data are representative of three independent experiments.

Supplementary Table 1. Antibodies used for flow cytometry.

Antibody	Company	Clone
NK1.1	BD Biosciences	PK136
CD4	eBioscience	GK1.5
CD8	Biolegend	53-5.8
CD11c	Biolegend	N418
CD40	eBioscience	HM40.3
CD80	eBioscience	16-10A1
CD69	eBioscience	H1.2F3
CD44	eBioscience	IM7
IFN-γ	eBioscience	XMG1.2
IL-1β	R&D Systems	166931
CD11b	Biolegend	M1/70
F4/80	eBioscience	BM8
Tim-3	eBioscience	RMT3-23
MHC II	Biolegend	M5/114.15.2
CD3e	Biolegend	145-2c11
galectin-9	eBioscience	RG9-35
Ly-6C	ebioscience	HK1.4
CD45	BD Biosciences	30-F11
Siglec-F	BD Biosciences	E50-2440
Ly-6G	Biolegend	1A8
Gr1	Biolegend	RB6-8C5