Supplemental Data
Fig.S1

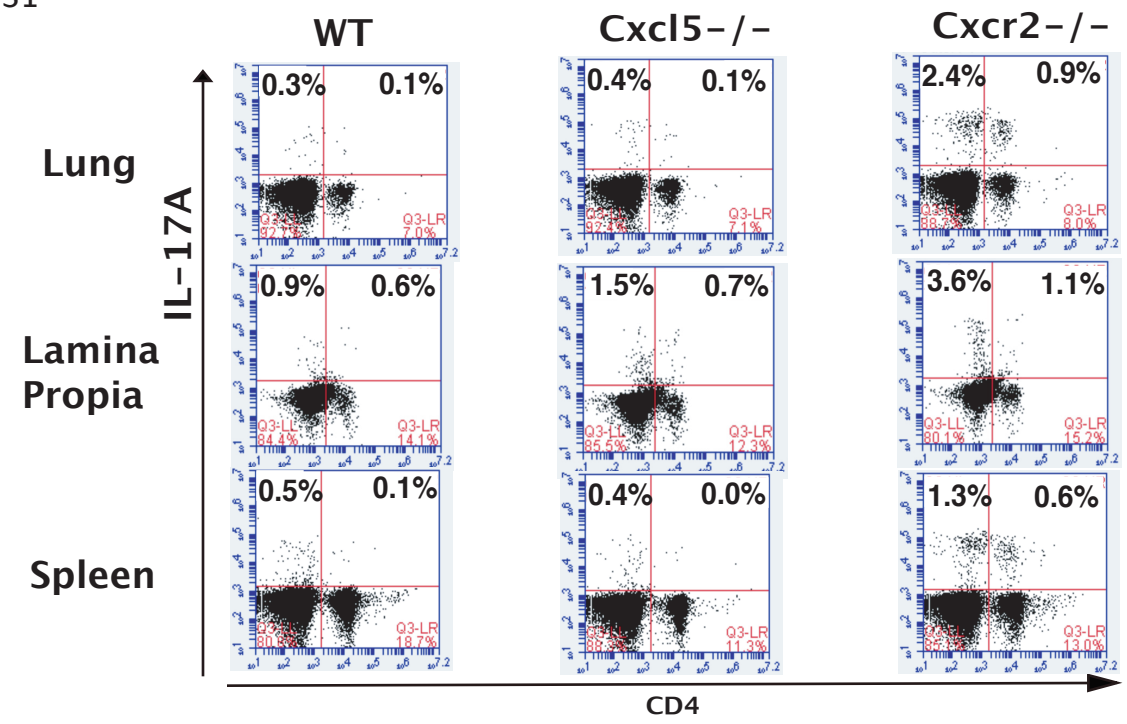

FIGURE S1. Characterization of IL-17A-producing cells in the lung, gut and spleen of WT, Cxcl5-/- and Cxcr2-/- mice. Representative flow cytometry histograms of isolated lung cells, lamina propria cells in terminal ileum and splenocytes from WT, Cxcl5-/- and Cxcr2-/- mice were shown. These cells were immunostained for intracellurar IL-17A and surface CD4 TCR□ and TCR□□ expression. The other representative histograms with immunostaining for surface CD3, TCR□, TCR□□, Gr-1, NK1.1 and intracellar IL-17A were shown in Fig.6 and supplemental Fig.S2-S3. These histograms are the representative of three separate experiments with similar results.

Fig.S2

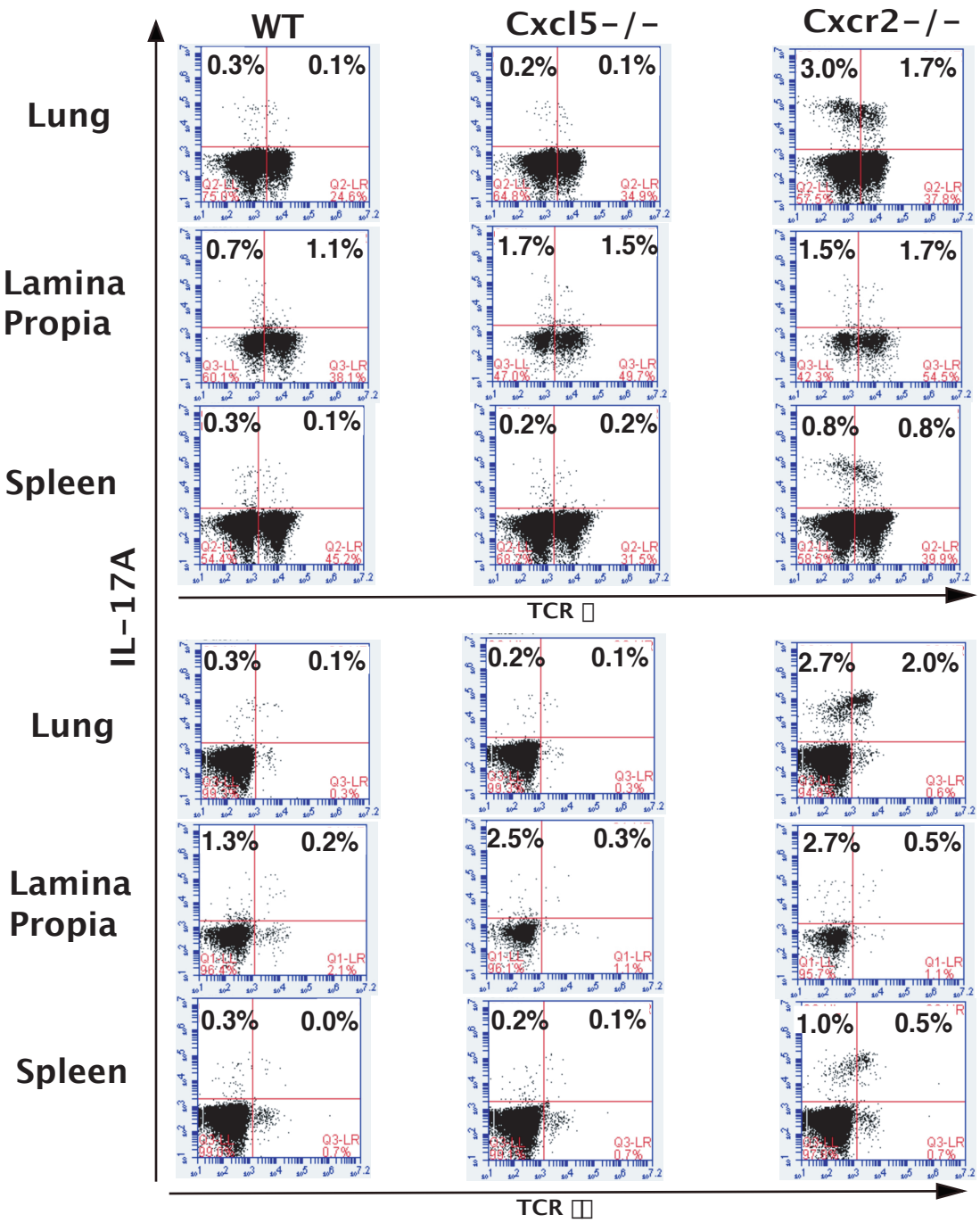

FIGURE S2. Characterization of IL-17A-producing cells in the lung, gut and spleen of WT, Cxcl5-/- and Cxcr2-/- mice. Representative flow cytometry histograms of isolated lung cells, lamina propria cells in terminal ileum and splenocytes from WT, Cxcl5-/- and Cxcr2-/- mice were shown. These cells were immunostained for intracellurar IL-17A and surface TCRγδ and TCRδδ expression. The other representative histograms with immunostaining for surface CD3, CD4, Gr-1, NK1.1 and intracellar IL-17A were shown in Fig.6 and supplemental Fig.S1, S3. These histograms are the representative of three separate experiments with similar results.

Fig. S3

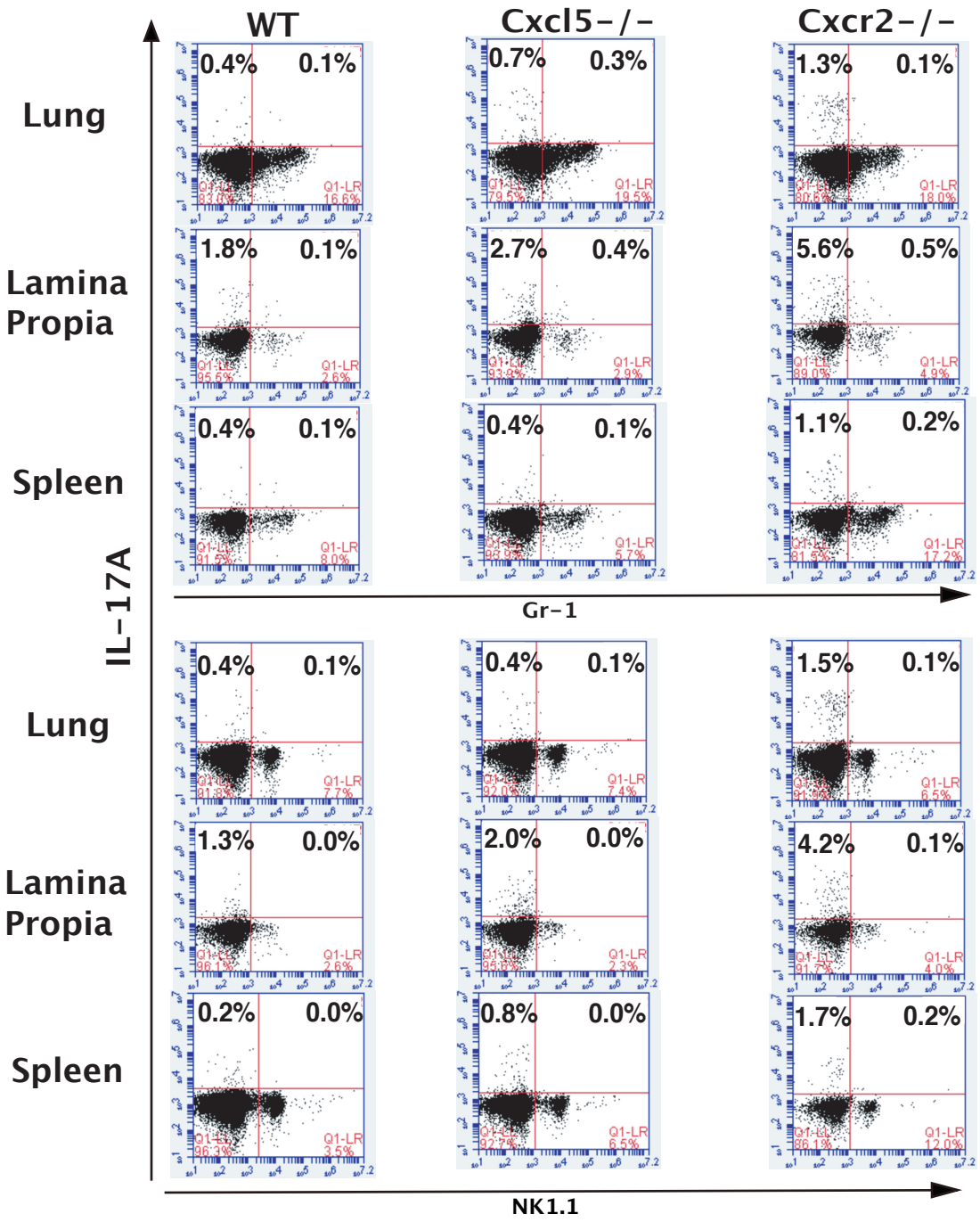

FIGURE S3. Characterization of IL-17A-producing cells in the lung, gut and spleen of WT, Cxcl5-/- and Cxcr2-/- mice. Representative flow cytometry histograms of isolated lung cells, lamina propria cells in terminal ileum and splenocytes from WT, Cxcl5-/- and Cxcr2-/- mice were shown. These cells were immunostained for intracellurar IL-17A and surface Gr-1 and NK1.1 expression. The other representative histograms with immunostaining for surface CD3, CD4, TCR□, TCR□□ and intracellar IL-17A were shown in Fig.6 and supplemental Fig.S1-S2. These histograms are the representative of three separate experiments with similar results.

Fig.S4

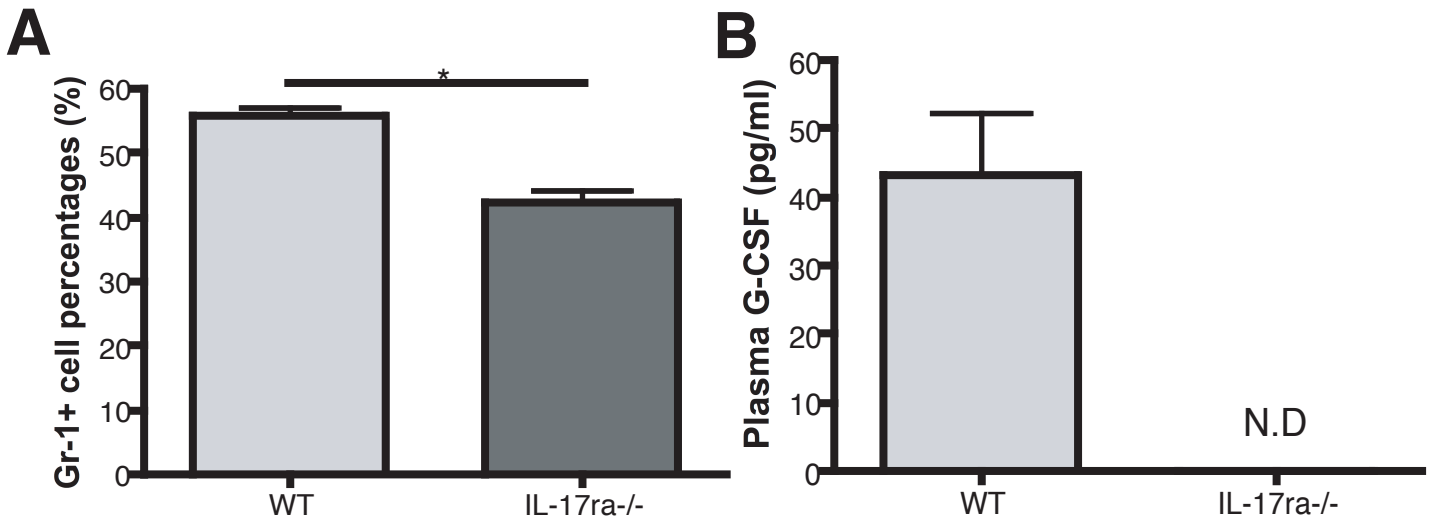

FIGURE S4. IL-17RA contribute to BM granulopoiesis and plasma G-CSF expression in normal WT mice. The BM Gr-1+ cell percentages (**A**) and plasma G-CSF (**B**) in WT and IL-17ra-/- mice (n=3 mice/group) were determined by flow cytometry and ELISA respectively.

Figure S5

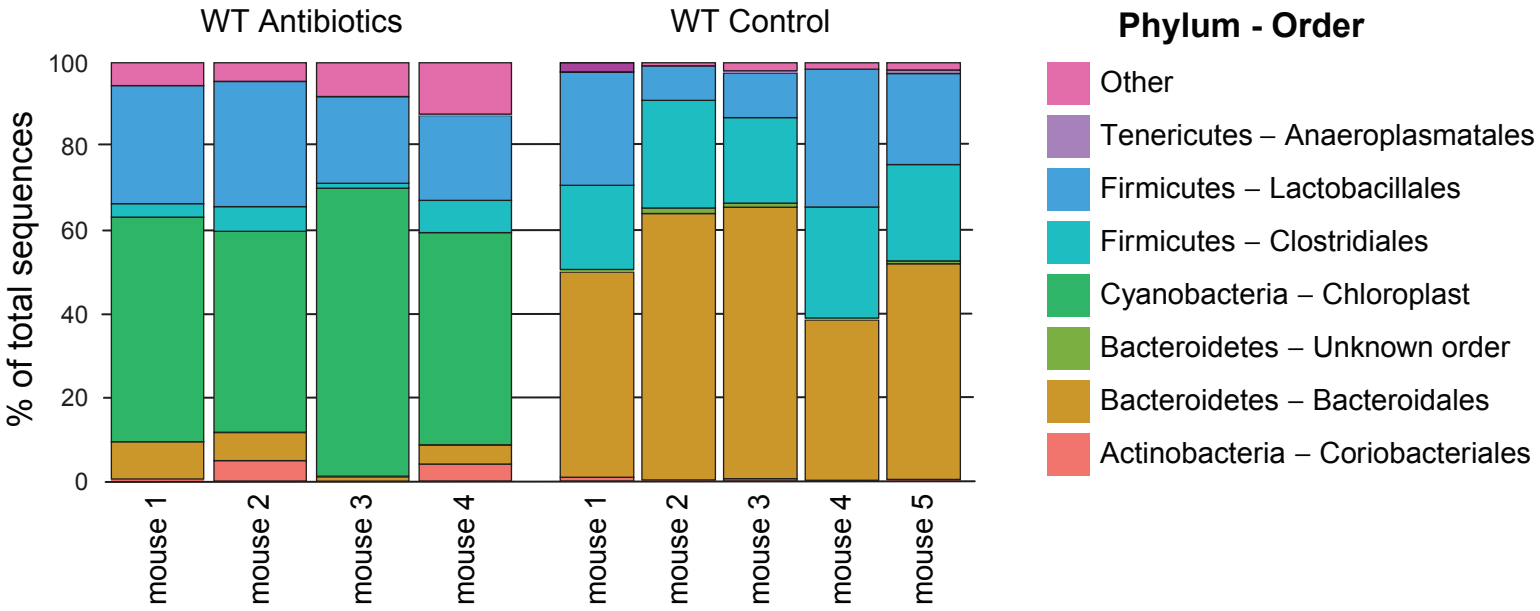

FIGURE S5. Antibiotic treatment alters colonic bacterial community structure in WT mice. The Phylum-order frequencies of cecal bacterial communities in C57BL/6 mice treated with and without antibiotic treatment was determined by pyrosequencing of 16s rDNA. The gut microbial communities from WT mice were significantly different between control mice and antibiotic treated mice, regarding their composition as well as relative abundance as measured by unweighted and weighted Unifrac, respectively (unweighted Unifrac: $p<=0.002$; weighted Unifrac: WT $p<=0.0001$).

Fig.S6

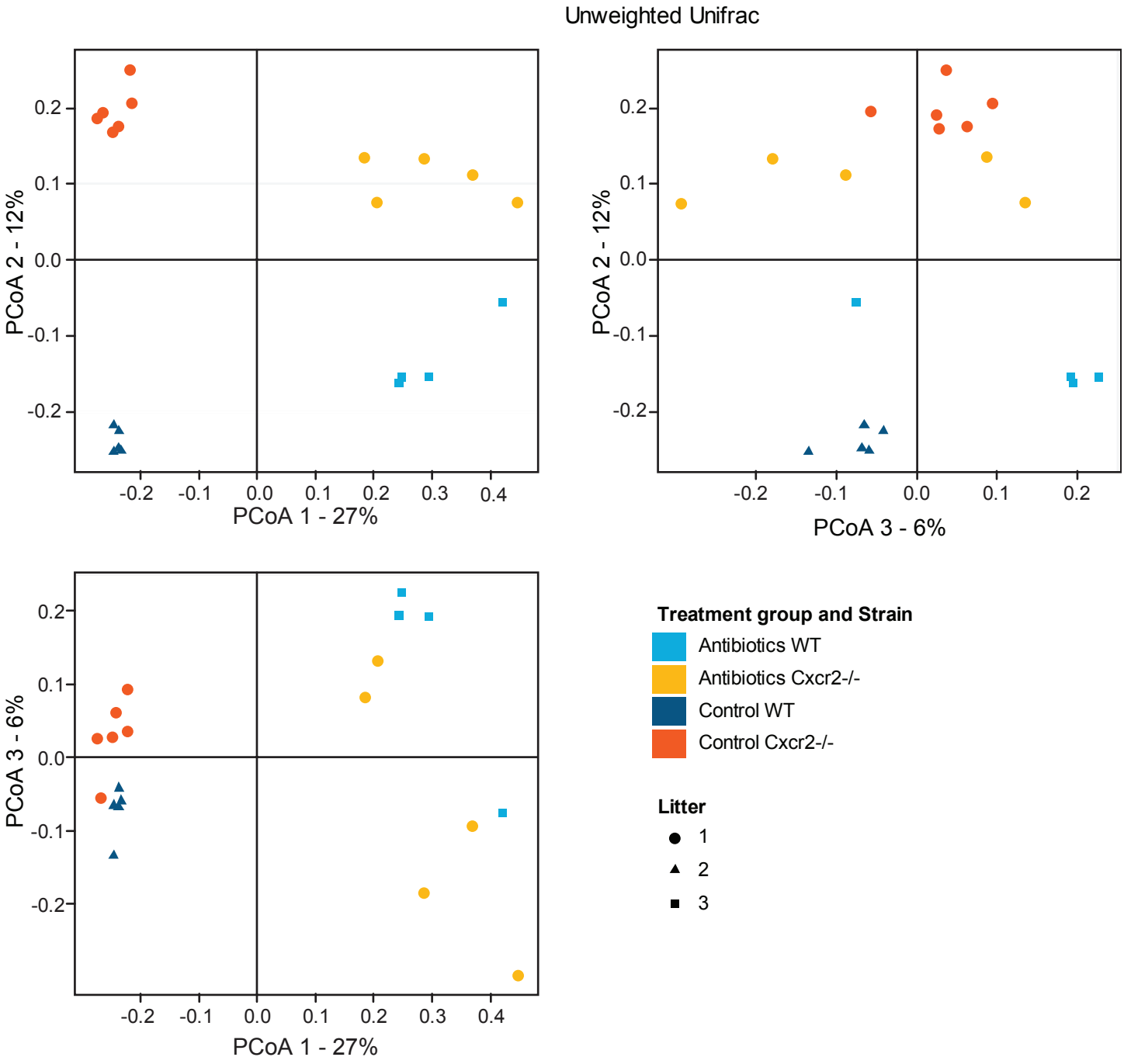

FIGURE S6. Antibiotic treatment alters colonic bacterial community structure in WT and Cxcr2-/- mice. Based on the 16s rDNA sequence data from cecal bacterial communities, unweighted UniFrac analysis of genotypes and treatment were performed. The gut microbial communities from both genotypes and different litters were significantly different between control mice and antibiotic treated mice, regarding their composition. For the effects of antibiotic treatment on both genotype communities: on WT mice: p<=0.002; on Cxcr2-/- mice: p<=0.004 (Unweighted UniFrac analysis). For the effects of different litters on control-treated mice: p-value <= 0.0003 (Unweighted UniFrac analysis).

Fig.S7

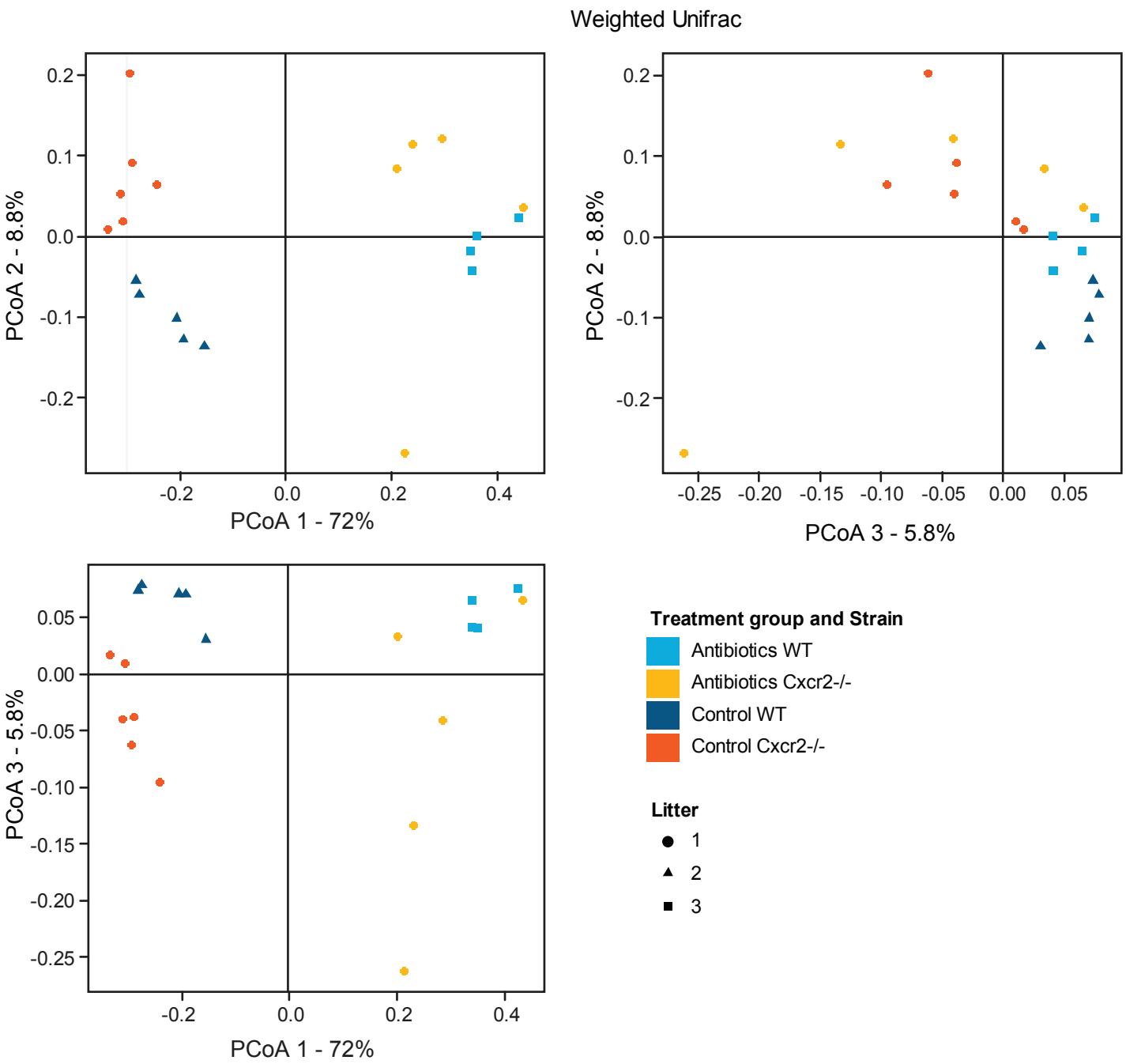

FIGURE S7. Antibiotic treatment alters colonic bacterial community structure in WT and Cxcr2-/- mice. Based on the 16s rDNA sequence data from cecal bacterial communities, weighted UniFrac analysis of genotypes and treatment were performed. The gut microbial communities from both genotypes and different litters were significantly different between control mice and antibiotic treated mice, regarding the abundance of bacterial communities. For the effects of antibiotic treatment on both genotype communities: on WT mice: p<=0.001; on Cxcr2-/- mice: p<=0.026 (weighted UniFrac analysis). For the effects of different litter on control-treated mice: p-value <= 0.06 (weighted UniFrac analysis).