doi: 10.1038/nature07250

Figure S1

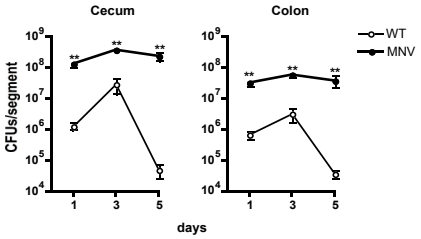

Figure S1: Antibiotic treatment increases VRE colonization in the cecum and colon. Wild type mice were treated with metronidazole (M; 1g/l), neomycin (N; 500mg/l) and vancomycin (V; 1g/l) (MNV) in drinking water starting 2 days before infection. All mice were orally infected with 10^9 VRE by gavage on day 0. Bacterial counts within the cecum and colon were determined 1, 3 and 5 days after VRE infection, n=5 each group and each timepoint.**p = 0.008, Mann-Whitney. Error bars denote S.E.M.

Figure S2

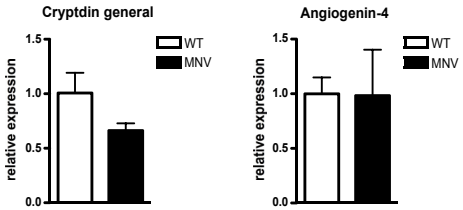

Figure S2 Expression of Cryptdin and Angiogenin-4 in distal small intestines of wild-type and MNV treated mice. mRNA was extracted from the terminal ileum of wild type mice and mice receiving antibiotics (MNV) for 7 days. Cryptdins and Angiogenin-4 expression were examined by quantitative real-time PCR. Expression levels were normalized to GAPDH and the results are expressed relative to wild type mice; n= 7-10 mice per group. Error bars denote S.E.M.

Figure S3

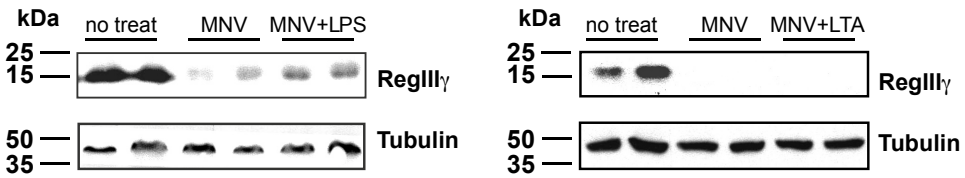

Figure S3 Prophylactic administration of oral LPS upregulates RegIIIγ protein levels. Protein extracts from the distal small intestine of wild type mice, mice receiving antibiotics (MNV) and mice receiving antibiotics (MNV) + LPS (2-4μg/μl) (right panel) or MNV + LTA (0.25 μg/μl) (left panel) for 7 days were analyzed by Western blotting with RegIIIγ-specific antiserum. Tubulin was used as a loading control.

Figure S4

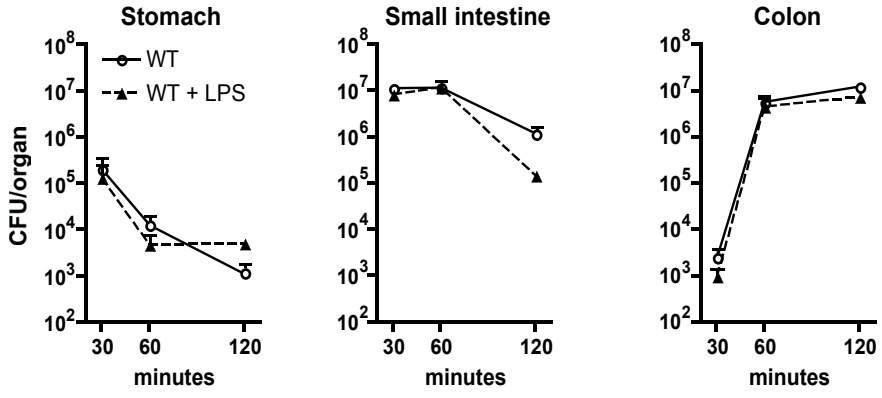

Figure S4 LPS does not directly kill VRE when orally administered. Mice were treated with LPS in drinking water for 6 hours prior oral infection with 10^9 VRE. Bacterial burden was determined in stomach, small intestine and colon after 30, 60 and 120 minutes. Error bars denote S.E.M.

Figure S5

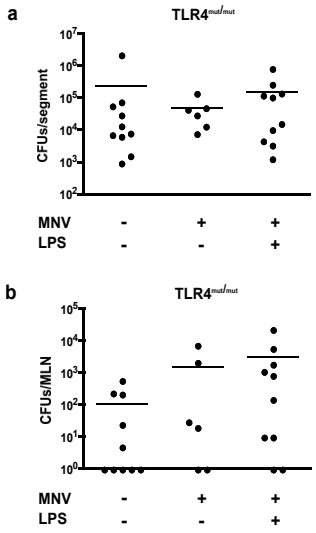

Figure S5 (a) TLR4$^{mut/mut}$ mice (C3HeJ), either left untreated or treated for 7 days with MNV or MNV + LPS, were orally infected with 10^{10} VRE. 24 hours later, bacterial counts within the distal small intestine were determined, n=6-10. (b) TLR4$^{mut/mut}$ mice (C3HeJ), either left untreated or treated for 7 days with MNV or MNV + LPS, were orally infected with 10^{10} VRE. 24 hours later, bacterial counts within MLNs were determined, 0 colonies in MLNs were plotted as 1, n=6-10.

Figure S6

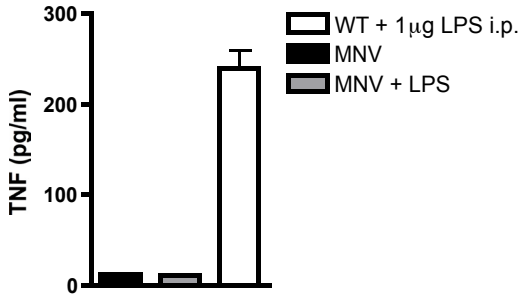

Figure S6 Oral LPS treatment does not lead to systemic immune activation. TNF levels in serum were determined in mice receiving antibiotics, antibiotics + LPS (4 μg/μl) for 7 days or in wild type mice injected with 1μg LPS i.p. n=5 for each, MNV and MNV + LPS, n=2 for mice receiving i.p. LPS. Error bars denote S.E.M.

Figure S7

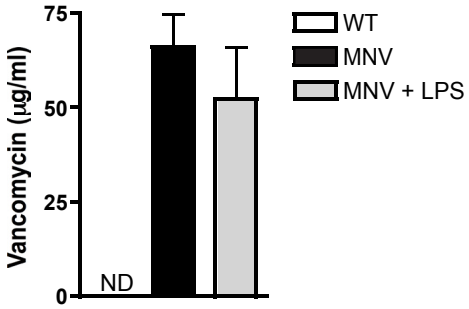

Figure S 7 Similar vancomycin levels in the colon of wild-type, antibiotic and antibiotic + LPS treated mice.
Vancomycin levels in the colon of wild-type, antibiotic and antibiotic + LPS (4μg/μl) treated mice were determined 7 days after treatment started; n=7. Error bars denote S.E.M.

Figure S8

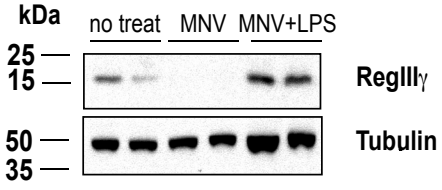

Figure S8. Oral treatment of mice with LPS upregulates RegIIIγ protein levels. Protein extracts from the distal small intestine of wild type mice, mice receiving antibiotics (MNV) and mice treated first with antibiotics for 4 days and then receiving a combination of antibiotics (MNV) + LPS (1-4μg/μl) for 7 days were analyzed by Western blotting with RegIIIγ-specific antiserum. Tubulin was used as a loading control.