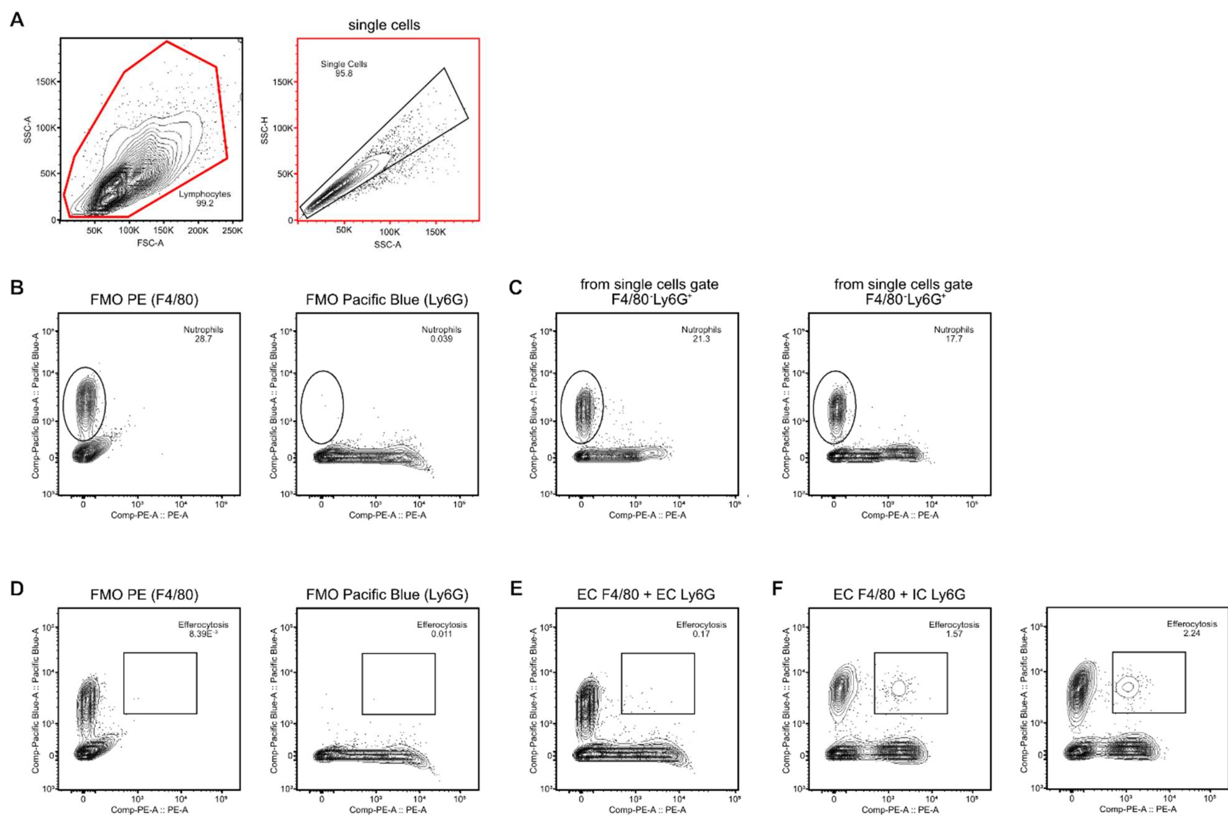
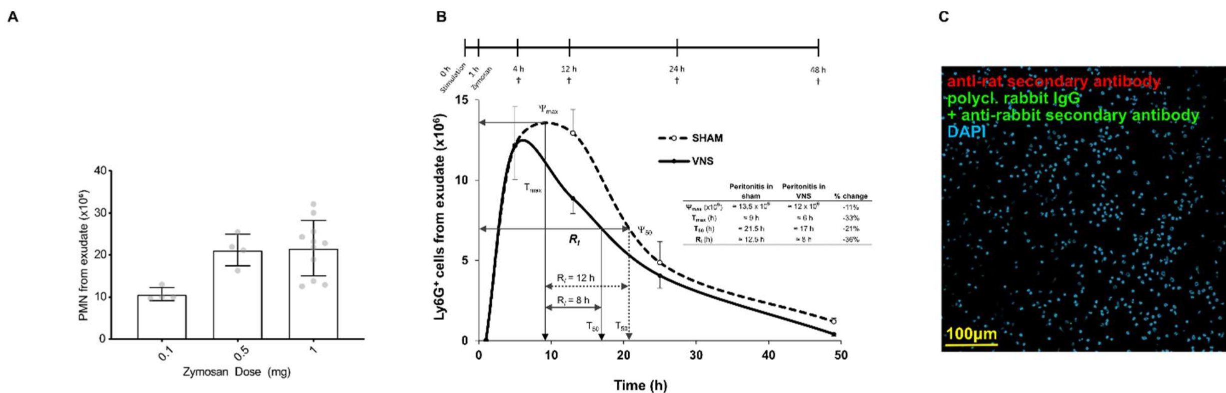
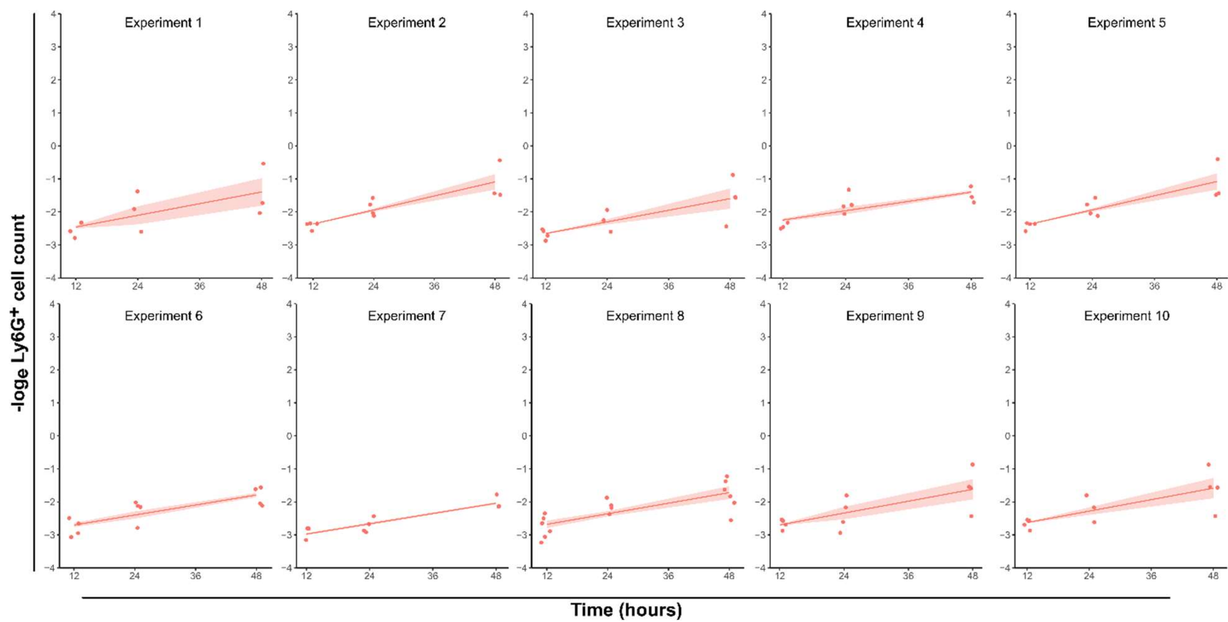


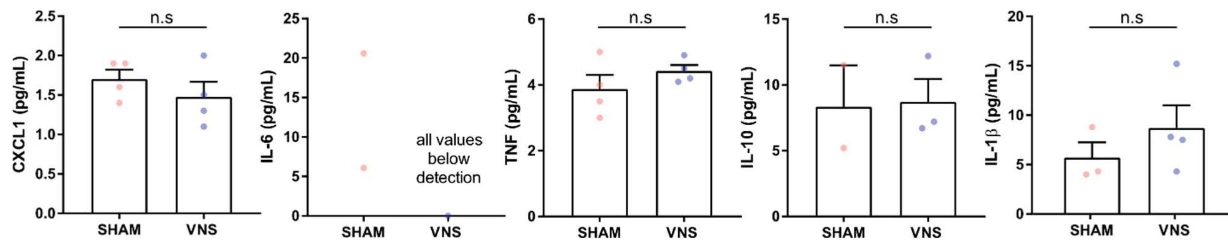
Supplementary Figure 1. Gating strategy for identification of neutrophil populations and efferocytosis by flow cytometry. (A) Side scatter area (SSC-A) versus forward scatter area (FSC-A) contour plot (left). Side scatter height (SSC-H) versus SSC-A dot plot to identify singlets (right). (B) Fluorescence minus one (FMO) controls for PE (F4/80) and Pacific Blue (Ly6G) for identification of neutrophils (Ly6G$^+$ cells). (C) Representative plots for F4/80 versus Ly6G dot plot to identify the Ly6G$^+$ neutrophil population (left, sham; right, VNS). (D) Fluorescence minus one (FMO) controls for PE (F4/80) and Pacific Blue (Ly6G) for identification of efferocytosis (F4/80$^+$Ly6G$^+$ cells). (E) Representative control plot of extracellular PE (F4/80) and Pacific Blue (Ly6G) cells. (F) Representative plots for extracellular PE (F4/80) versus intracellular Pacific Blue (Ly6G) dot plot to identify F4/80+Ly6G+ efferocytosis gate (left, sham; right, VNS).



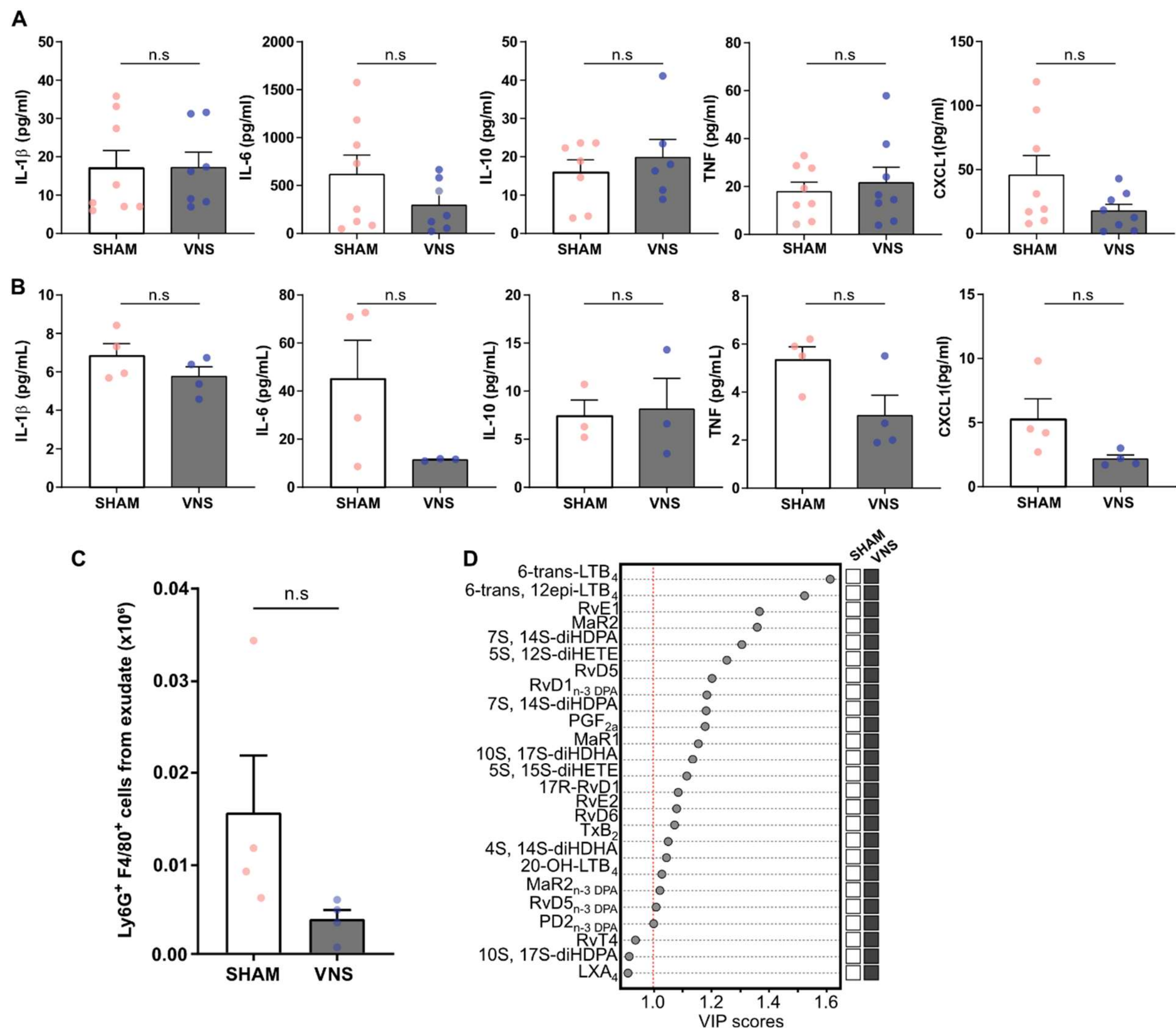
Supplementary Figure 2. Zymosan-induced peritonitis in wild type mice. (A) Mice were injected intraperitoneally with 0.1, 0.5, or 1 mg of zymosan. Peritoneal exudates were collected 4 h after injection and analyzed for Ly6G$^+$ cells by flow cytometry. Results are shown as mean ± SEM. (B) Mice were subjected to left cervical vagus nerve stimulation 1 h before initiation of zymosan-induced peritonitis. Peritoneal exudates were collected at 4, 12, 24, and 48 h after zymosan challenge. Peritoneal exudates were analyzed by flow cytometry. Resolution indices were determined as previously defined (28), including Ψ_{max} (maximal neutrophil infiltration), T_{max} (time point when neutrophils reach Ψ_{max}), T_{50} (time point corresponding to 50% reduction from peak neutrophil infiltration), and R_i (resolution interval, the time interval from Ψ_{max} to 50% reduction point, Ψ_{50}). (C) Negative control of immunofluorescence staining. Peritoneal lavage was collected at 12 h after zymosan-induced peritonitis. Cells were plated *in vitro* and stained using a rabbit polyclonal antibody, followed by anti-rat and anti-rabbit fluorescently labelled secondary antibodies. Nuclei were stained with DAPI (blue). Fluorescence was visualized using a Nikon confocal microscope.



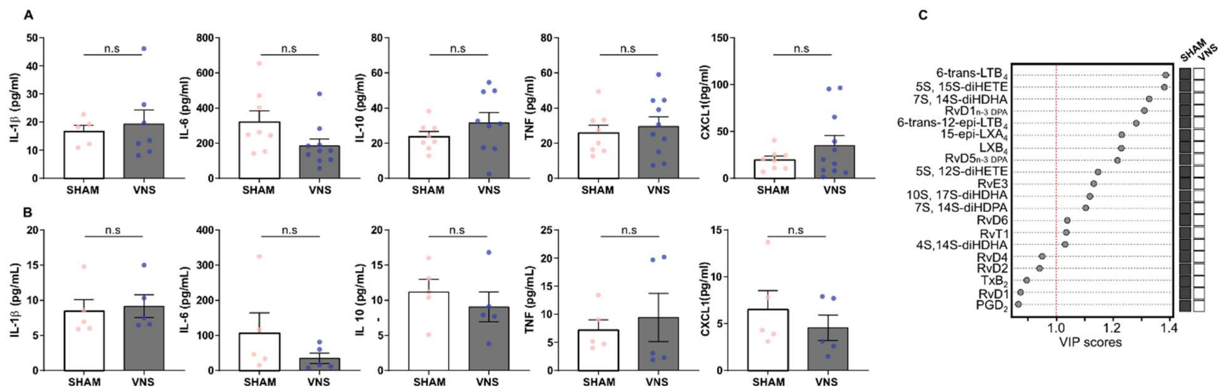
Supplementary Figure 3. Neutrophil counts in peritoneal exudates during inflammation decay. Data from previously published studies in which wild-type mice were subjected to zymosan-induced peritonitis (Arnardottir HH et al., J Immunol 2014; Arnardottir H et al., Mucosal Immunol 2016) were analyzed. $\log_e$ cell counts (dots) over time from these datasets were plotted and linear regression analysis performed (lines), with shaded areas indicating the mean squared error. Results are from n=10 separate experiments.



Supplementary Figure 4. Cytokine concentrations in peritoneal exudates from wild-type mice 24 h after induction of zymosan-induced peritonitis. Wild-type mice were subjected to either left cervical vagus nerve stimulation (blue dots) or sham surgery (red dots) followed by intraperitoneal injection of zymosan (0.1 mg/mouse) after 1h. Peritoneal exudates were collected at 24 h and levels of CXCL1, IL-6, TNF, IL-10, and IL-1β were measured using the MSD multiplex assay. Mean ± SEM are plotted. n=2 experiments.



Supplementary Figure 5. Cytokine concentrations and lipid mediator analysis in peritoneal exudates from Alox15-deficient mice. Alox15-deficient mice were subjected to vagus nerve stimulation or sham-surgery, injected with zymosan (0.1 mg/mouse) intraperitoneally, and peritoneal exudates collected 12 h after zymosan challenge. (A) Concentrations of IL-1β, IL-6, IL-10, TNF, and CXCL1 in peritoneal exudates collected at 12 h were measured by MSD multiplex assay. Mean ± SEM are plotted. n=3 experiments. (B) Levels of IL-1β, IL-6, IL-10, TNF, and CXCL1 in peritoneal exudates collected at 24 h. n=3 experiments. Mean ± SEM are plotted. (C) Efferocytosis plotted as absolute numbers of $F4/80^+Ly6G^+$ cells measured by flow cytometry following fixation and intracellular Ly6G staining of exudates collected at 12 h. n=2 experiments. (D) Lipid mediators were measured using LC-MS/MS. VIP score plot of 25 lipid mediators in peritoneal exudates from VNS- and sham-treated mice with the greatest contribution to the separation of the groups. White squares indicate lower levels, and black squares higher levels in the sham or VNS-treated group. n=2 experiments.



Supplementary Figure 6. Cytokine concentrations and lipid mediator analysis in exudates from α7nAChR-deficient mice. α7nAChR-deficient mice were subjected to either left cervical vagus nerve stimulation or sham surgery followed by intraperitoneal injection of zymosan. Peritoneal exudates were collected 12 h after zymosan injection. Concentrations of IL-1β, IL-6, IL-10, TNF, and CXCL1 in peritoneal exudates from VNS- (blue dots, grey bars) and sham (blue dots, open bars)-treated mice were measured by MSD multiplex assay at (A) 12 h and (B) 24 h after zymosan injection (0.1 mg/mouse). Mean ± SEM are plotted. n=3 (12 h) or n=2 (24 h) experiments. (C) Concentrations of lipid mediators in peritoneal exudates collected 12 h after intraperitoneal zymosan injection were measured using LC-MS/MS. VIP score plot of 25 lipid mediators in peritoneal exudates from VNS- and sham-treated mice with the greatest contribution to the separation of the groups. White squares indicate lower levels, and black squares higher levels in the sham or VNS-treated group. n=2 experiments.