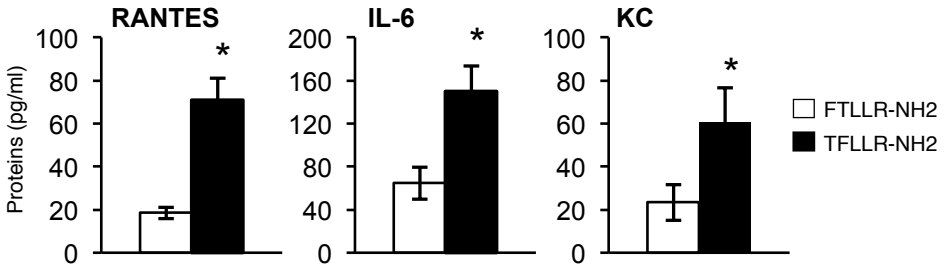
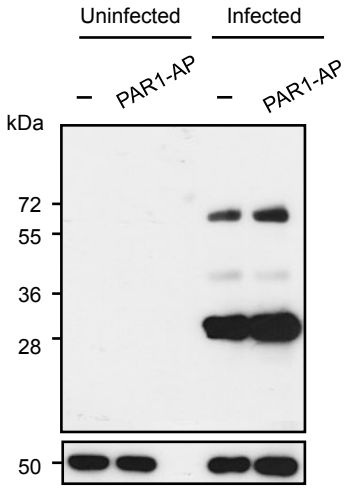
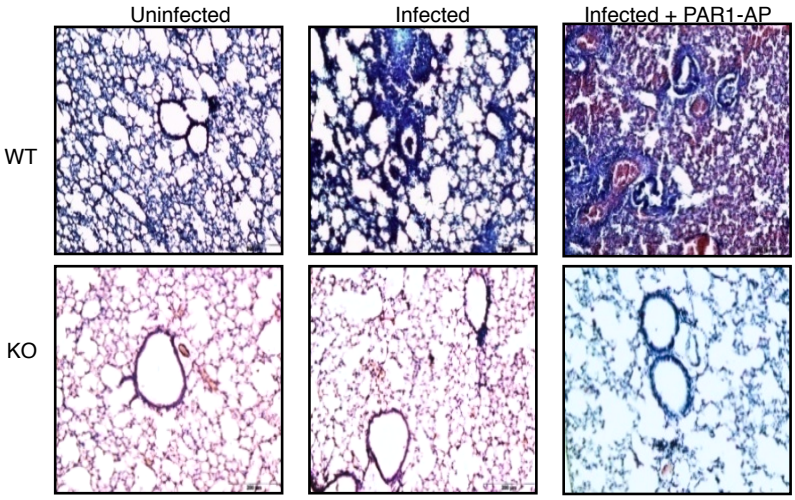


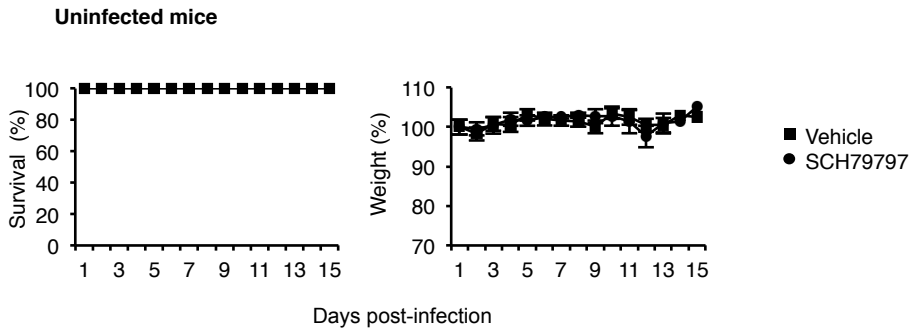
Supplemental data S1: PAR1 agonist specifically increases inflammation in IAV-infected mice. Cytokines in the BAL of infected mice (50PFU) treated with PAR1-AP (TFLLR-NH2) or control peptides (FTLLR-NH2) were measured by ELISA assay 24 hours post-infection. Histograms represents the mean values ± standard deviation from 6 individual animals per groups from 2 independent experiments.



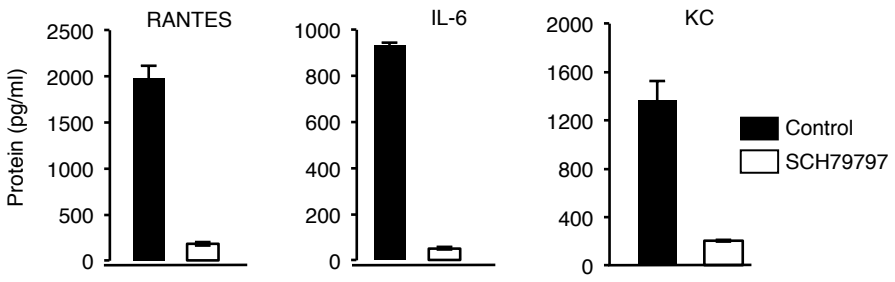
Supplemental data S2: PAR1 agonist does not affect virus entry into cells. A549 cells were infected with A/PR/8/34 virus (MOI 20) in absence (-) or presence of PAR1-AP for one hour and accumulation of viral proteins from incoming virus was analyzed by western blot using a polyclonal anti-A/PR/8/34 antibody. PAR1-AP did not have an effect on accumulation of viral proteins in infected cells. kDa : apparent molecular weight.



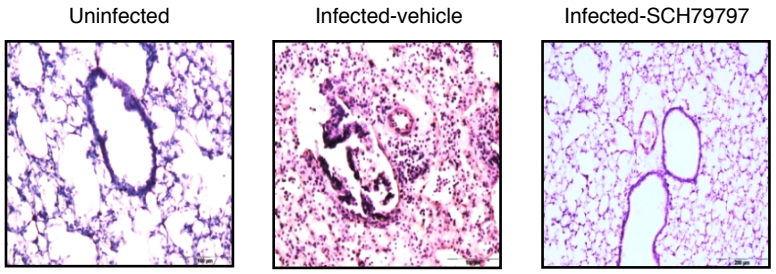
Supplemental data S3: Histopathological examination of lungs from infected mice treated or not with PAR1-AP. Infiltration of inflammatory cells in the lungs of uninfected or infected mice (50 PFU) treated or not with PAR1-AP (50μM) at day three post-inoculation. PAR1-mediated inflammation was abolished in PLG KO mice (KO).



Supplemental data S4: Survival and body weight of mice after administration of SCH79797. Survival and weight loss was assessed on uninfected mice, treated or not with 50 µM SCH79797 (n= 7 mice per group).



Supplemental data S5: Cytokines levels in the BAL of infected mice treated or not with SCH79797. Cytokines levels were measured by ELISA in the BAL of infected mice 5 days post inoculation. Results show the average values ± standard deviations from 5 individual mice per group.



Supplemental data S6: Histopathological examination of lungs from infected mice treated or not with SCH79797. Infiltration of inflammatory cells in the lungs of uninfected or infected mice (500 PFU) treated or not with SCH79797 (50μM) at day three post-inoculation. SCH79797 prevented PAR1-mediated inflammation.