Supplementary Figure S1: Platelets and Cox1 are required for neutrophil recruitment and pathogen control during pulmonary *K. pneumoniae* infections. Glycol- and busulfan-treated wildtype as well as Cox1−/− mice were injected intratracheally with viable *K. pneumoniae*. (a) Neutrophil recruitment into the alveoli and the CFU count in the BAL (b), lung tissue (c) and the spleen (d) were analyzed after 24 hours (n=4-5). Mean±SEM, ANOVA plus Bonferroni testing, * p <0.05
Supplementary Figure S2: **Busulfan treatment does not alter neutrophil function.** Isotype antibody- and platelet-depleting antibody-treated (R300) wildtype mice were injected intratracheally with viable *E. coli*. (a) Neutrophil recruitment into the alveoli and the CFU count in the BAL (b), lung tissue (c) and the spleen (d) were analyzed after 24 hours (n=4-5). Neutrophils were isolated from glycol- or busulfan-treated mice and (e) ICAM1-binding (n=4), (f) transmigration (n=9) and (g) phagocytosis of pHrodo *E. coli* particles were analyzed (n=6). Mean±SEM, 2-tailed t test in S2a-d and S2f, ANOVA plus Bonferroni testing in S2e and S2g, * p <0.05.
Supplementary Figure S3: Hematopoietic Cox1 is indispensable to control bacterial spread. WT mice, Cox1<sup>-/-</sup> and Cox2<sup>−/−</sup> mice were injected intratracheally with viable *E. coli*. (a) Neutrophil recruitment into the alveoli and the CFU count in the BAL (b), lung tissue (c) and the spleen (d) were analyzed after 24 hours (n=4). Bone marrow chimeric mice (WT<->WT, Cox1<sup>−/−</sup><->WT, WT<->Cox1<sup>−/−</sup>, Cox1<sup>−/−</sup><->Cox1<sup>−/−</sup>) were generated by irradiation and bone marrow transplantation and (e) neutrophil recruitment into the alveoli and the CFU count in the BAL (f), lung tissue (g) and the spleen (h) were analyzed after 24 hours (n=4). (i) Number of accumulated neutrophils per field of view in lung IVM (n=3). (j) Neutrophils interacting with platelets in the lung capillaries in vivo (n=3). (k) Survival 24 hours after instillation of 8x10<sup>6</sup> viable *E. coli* (n=8-15). Mean±SEM, ANOVA plus Bonferroni testing in S3a-k, log rank test in S3k, * p <0.05
Supplementary Figure S4: Lung intravital microscopy after *E. coli* instillation. Mice were intratracheally instilled with *E. coli* or saline as a control. Neutrophils were labeled with Gr1-Alexa488 (green) and platelets were labeled with CD41-PE (red). Exemplary micrographs from WT mice treated with saline (a), WT mice instilled with *E. coli* (b) and Cox1<sup>−/−</sup> mice instilled with *E. coli* (c). Arrows indicate platelet-neutrophil interactions. Scale bar equals 50 µm.
Supplementary Figure S5: Platelet P-selectin is required for neutrophil recruitment and pathogen control during pulmonary *E. coli* infections. Bone marrow from P-selectin deficient (Selp⁻/⁻) or WT donor mice was transplanted into lethally irradiated WT recipient mice. 6 weeks after transplantation mice were injected intratracheally with saline or viable *E. coli*. (a) Neutrophil recruitment into the alveoli and the CFU count in the BAL (b), lung tissue (c) and the spleen (d) were analyzed 24 hours thereafter (n=4-5). (e) Circulating platelets were isolated from blood of saline- and *E. coli*-treated mice and P-selectin expression was analyzed by flow cytometry after 4, 12 and 24 hours (n=4). Mean±SEM, ANOVA plus Bonferroni testing, * p <0.05.
Supplementary Figure S6: **GPIbα on EVs is necessary for host defense.** Wildtype mice pretreated with the blocking antibody against GPIbα (clone Xia.B2, 50µg/mouse) were injected intratracheally with viable *E. coli* and reconstituted with either untreated isolated EVs or with isolated EVs that had been preincubated with the blocking antibody against GPIbα (clone Xia.B2) *in vitro*. Neutrophil recruitment into the alveoli (a) and the CFU count in the BAL (b), lung tissue (c) and the spleen (d) were analyzed after 24 hours (n=4). Mean±SEM, 2-tailed t test, * p <0.05
Supplementary Figure S7: Characterization of neutrophil-derived EVs. (a) Mac-1, CD11a, CD18, PSGL-1, and CD63 in supernatant and EV fraction from control and stimulated samples was detected by western blot (exemplary blot from 3 experiments). (b) PMNs and platelets were isolated from WT mice and treated with a blocking antibody against GPIbα. Isolated neutrophil-derived EVs were reconstituted at the designated concentration and TxB₂ production in control and ADP/fMLP-stimulated samples was analyzed (n=3). Mean±SEM, ANOVA plus Bonferroni testing, * p <0.05
Supplementary Figure S8: Uncut Western Blot gels. (a) Blots corresponding to Figure 5a. Left blot: Caveolin-1. Right blot: Clathrin. (b) Blots corresponding to Figure 5d. (c) Blots corresponding to Figure 5g. Left blot: Mac-1. Right blot: Rap1. (d) Blots corresponding to Supplementary Figure S7. Blots in following order (from top to bottom): Mac-1, CD11a, CD18, PSGL-1, CD63.