Table S1. ***Pseudomonas aeruginosa*** **strains used in this study.**

Strain	Laboratory/Clinical	Source	Motility	Flagellin	Reference
PAK	Laboratory	Unknown	+	a-type	S1
PAO1	Laboratory	Wound	+	b-type	S2
PAK/fliC^{-}	Laboratory	Unknown	—	N/A	S3
PAO1/NanPs^{-}	Laboratory	Wound	+	b-type	S4
PA50241	Clinical	Pneumonia[a]	+	b-type	S5
PA50255	Clinical	Pneumonia[a]	+	a-type	S5
PA50296	Clinical	Pneumonia[a]	+	b-type	S5
PA50312	Clinical	Pneumonia[a]	+	a-type	S5
PA50327	Clinical	Pneumonia[a]	+	a-type	S5

[a]The diagnosis of pneumonia was made based on the presence of pulmonary infiltrates on chest x-ray and/or computerized axial tomography and the presence of Gram-negative bacilli in sputum samples that cultured positive for Pa.

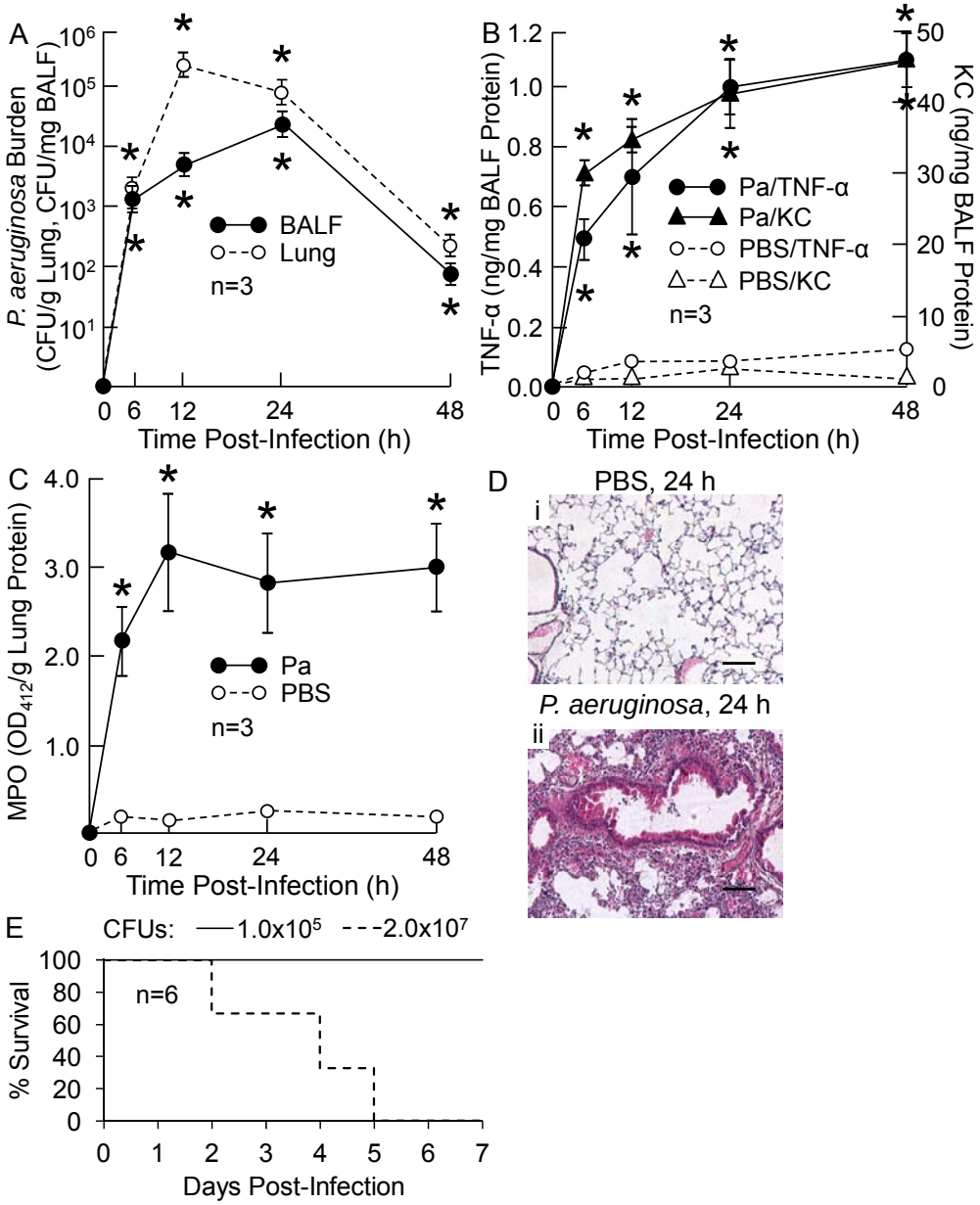

FIGURE S1. **A mouse model of Pa pneumonia.** (A-D) BALB/c mice were administered i.n. with 1.0 x 10^5 CFUs/mouse of PAK or the PBS vehicle. At increasing times post-infection, BALF and lung tissues were collected. (A) Pa CFUs in BALF and lung tissues were quantified on agar plates and normalized to lung or BALF protein. (B) TNF-α and KC levels in BALF were measured by ELISA and normalized to BALF protein. (C) MPO activity in lung tissues as a marker of recruited neutrophils was measured and normalized to lung protein. (A-C) Bars represent mean ± SE Pa CFUs, TNF-α or KC levels, or MPO activity (n = 3). *, increased Pa CFUs, TNF-α or KC levels, or MPO activity compared with (A) preinfection or (B, C) uninfected controls at $p<0.05$. (D) At 24 h post-infection, lung sections from mice administered with PBS (panel i) or Pa (panel ii) were stained with hematoxylin and eosin, and examined by microscopy. Each section was photographed at 400X. Scale bar, 50 μM. Each photomicrograph is representative of three mouse lung sections. (E) BALB/c mice were infected i.n. with 1.0 x 10^5 or 2.0 x 10^7 CFUs of PAK, and mouse survival was measured daily by Kaplan-Meier analysis (n = 6/group). The results are representative of three independent experiments.

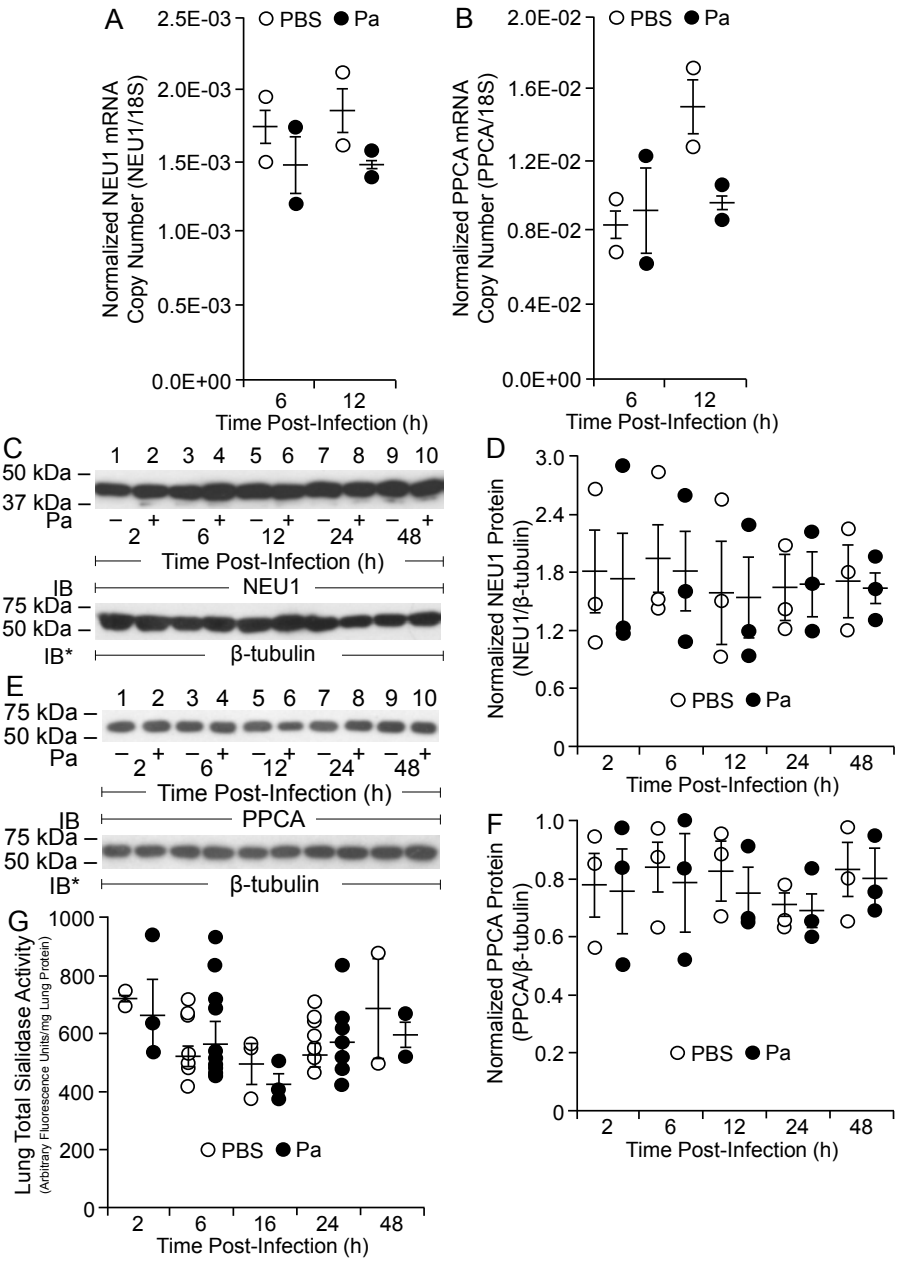

FIGURE S2. **Pa lung infection fails to upregulate NEU1 or PPCA expression or sialidase catalytic activity.** (A-F) BALB/c mice were administered i.n. 1.0 x 10^5 CFUs/mouse of PAK or the PBS vehicle. At increasing times post-infection, BALF and lung tissues were collected. (A, B) qRT-PCR analyses of (A) NEU1 and (B) PPCA transcripts. NEU1/PPCA mRNA levels were normalized to the 18S rRNA internal control. Bars represent mean ± SE normalized mRNA levels. (C-F) Lysates of lung tissues were processed for quantitative NEU1 (C, D) and PPCA (E, F) immunoblotting. To control for protein loading and transfer, blots were stripped and reprobed for β-tubulin. Molecular weights in kDa are indicated on the left. IB, immunoblot; IB*, immunoblot after stripping. (D, F) Densitometric analyses of the blots in C and E, respectively. Bars represent mean ± SE NEU1/PPCA signal normalized to β-tubulin signal in the same lane on the same stripped and reprobed blot. (G) Lung total sialidase activity for the 4-MU-NANA substrate was determined and normalized to lung protein. Bars represent mean ± SE sialidase activity expressed as arbitrary fluorescence units/mg lung protein. The results are representative of three independent experiments.

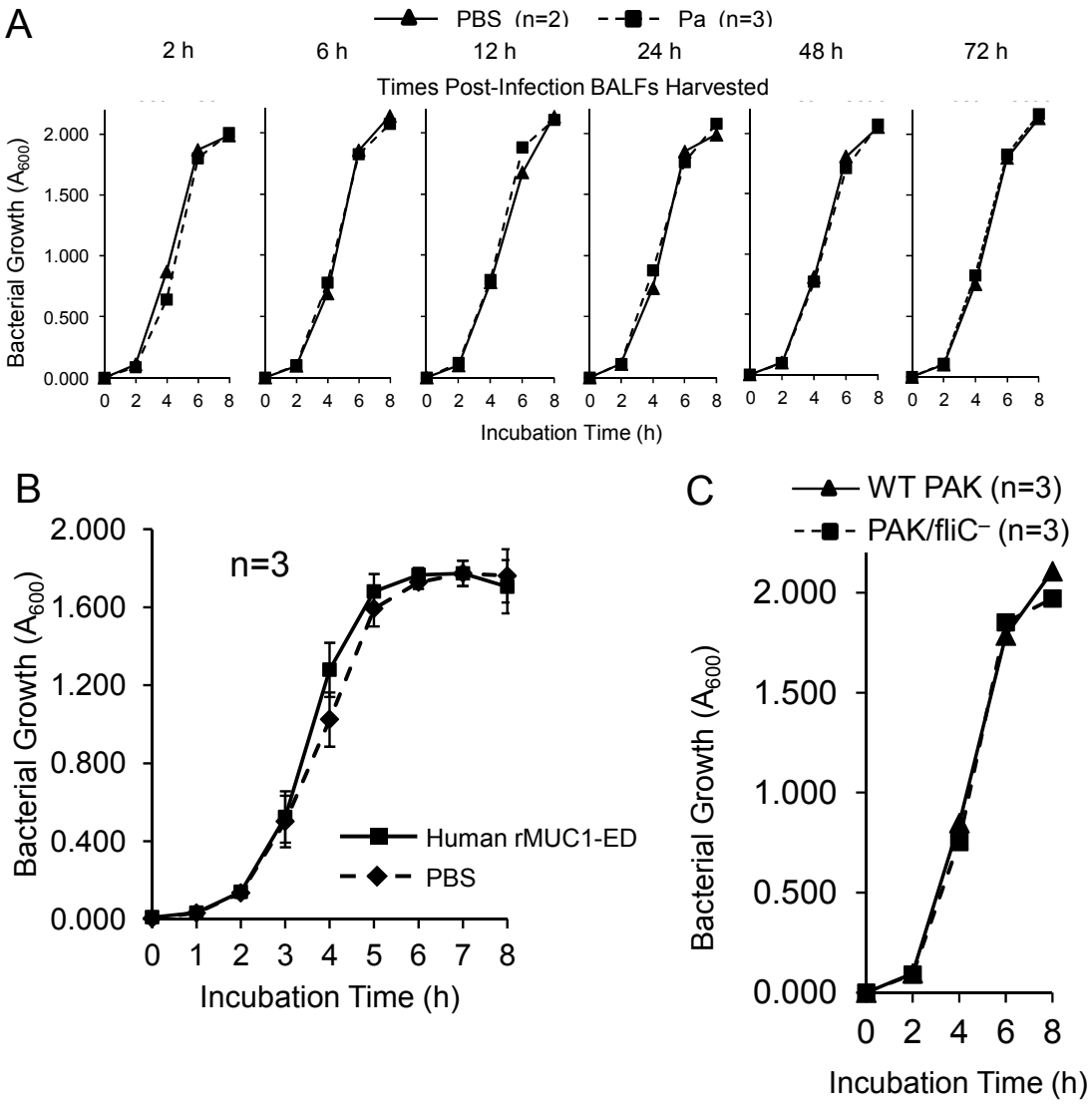

FIGURE S3. **BALF from Pa-infected mice, human rMUC1-ED, and lack of flagellin expression all fail to inhibit Pa growth.** (A) BALB/c mice were administered i.n. with 1.0 x 10^5 CFUs/mouse of PAK or the PBS vehicle. At increasing times post-administration, BALF was collected. PAK was incubated for 30 min at 4°C with BALF from uninfected or Pa-infected mice and cultured over time for bacterial growth in LB broth. (B) PAK were cultured with 100 μM of human rMUC1-ED or the PBS vehicle and cultured over time for bacterial growth in LB broth. (C) WT PAK and the flagellin-deficient PAK/fliC$^-$ isogenic mutant were cultured over time for bacterial growth in LB broth. Bars represent mean ± SE A_{600} values (n = 2-3). The results are representative of three independent experiments.

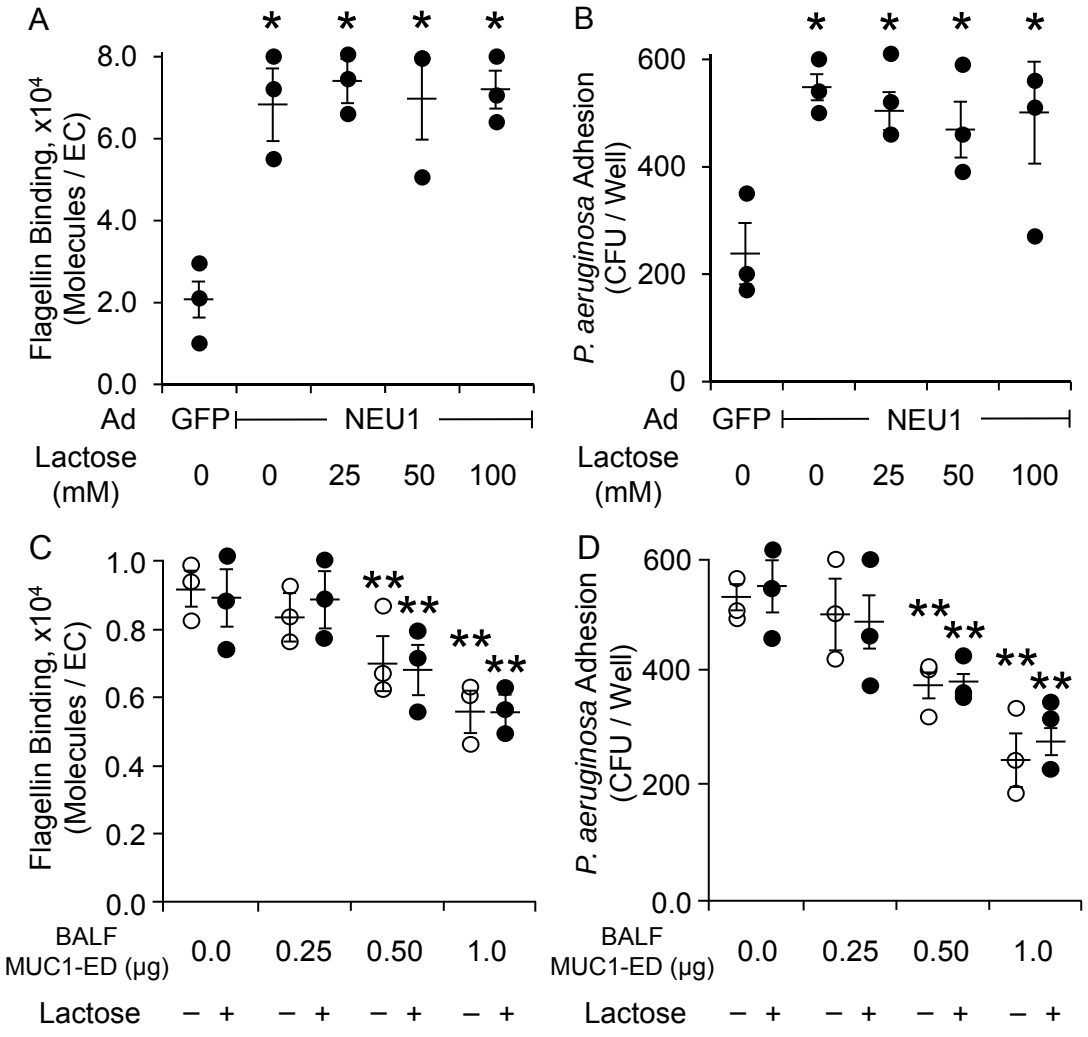

FIGURE S4. **Lactose fails to inhibit the flagellin-MUC1-ED interaction.** (A, B) A549 cells were infected with Ad-GFP or Ad-NEU1 (MOI = 100) and cultured for 48 h. (A) Alexa Fluor 594-labeled flagellin was preincubated for 30 min at 4°C with increasing concentrations of lactose or the PBS vehicle and assayed for binding to Ad-GFP- or Ad-NEU1-infected A549 cells. (B) PAK were preincubated for 30 min at 4°C with increasing concentrations of lactose or the PBS vehicle, and assayed for adhesion to Ad-GFP or Ad-NEU1-infected A549 cells. (C, D) BALB/c mice were administered i.n. 1.0 x 10^5 CFUs/mouse of PAK or the PBS vehicle and BALF was collected at 48 h post-administration. (C) Alexa Fluor 594-labeled flagellin was preincubated for 30 min at 4°C with BALF from uninfected or PAK-infected mice containing increasing concentrations of MUC1-ED, as measured by ELISA, in the absence or presence of 100 mM lactose and assayed for binding to Ad-NEU1-infected A549 cells (MOI = 100). (D) PAK was preincubated for 30 min at 4°C with BALF from uninfected or PAK-infected mice containing increasing concentrations of MUC1-ED, as measured by ELISA, in the absence or presence of 100 mM lactose and assayed for adhesion to Ad-NEU1-infected A549 cells. Bars represent mean ± SE flagellin binding or Pa adhesion. *, increased flagellin binding or Pa adhesion compared with Ad-GFP-infected cells at $p<0.05$. **, decreased flagellin binding or Pa adhesion following preincubation with MUC1-ED-containing BALFs from Pa-infected mice compared with uninfected controls at $p<0.05$. The results are representative of three independent experiments.

References

S1. Minamishima, Y., Takeya, K., Ohnishi, Y., and Amako, K. (1968) Physicochemical and biological properties of fibrous *Pseudomonas* bacteriophages. *J. Virol.* **2,** 208-213

S2. Holloway, B. W. (1955) Genetic recombination in *Pseudomonas aeruginosa*. *J. Gen. Microbiol.* **13,** 572-581

S3. Feldman, M., Bryan, R., Rajan, S., Scheffler, L., Brunnert, S., Tang, H., and Prince, A. (1998) Role of flagella in pathogenesis of *Pseudomonas aeruginosa* pulmonary infection. *Infect. Immun.* **66,** 43-51

S4. Soong, G., Muir, A., Gomez, M. I., Waks, J., Reddy, B., Planet, P., Singh, P. K., Kaneko, Y., Wolfgang, M. C., Hsiao, Y. S., Tong, L., and Prince, A. (2006) Bacterial neuraminidase facilitates mucosal infection by participating in biofilm production. *J. Clin. Invest.* **116,** 2297-305

S5. Shanks, K. K., Guang, W., Kim, K. C., and Lillehoj, E. P. (2010) Interleukin-8 production by human airway epithelial cells in response to *Pseudomonas aeruginosa* clinical isolates expressing type a or type b flagellins. *Clin. Vaccine Immunol.* **17,** 1196-1202