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Supplemental Information

Braun's Lipoprotein Facilitates OmpA Interaction with the *Escherichia coli* Cell Wall

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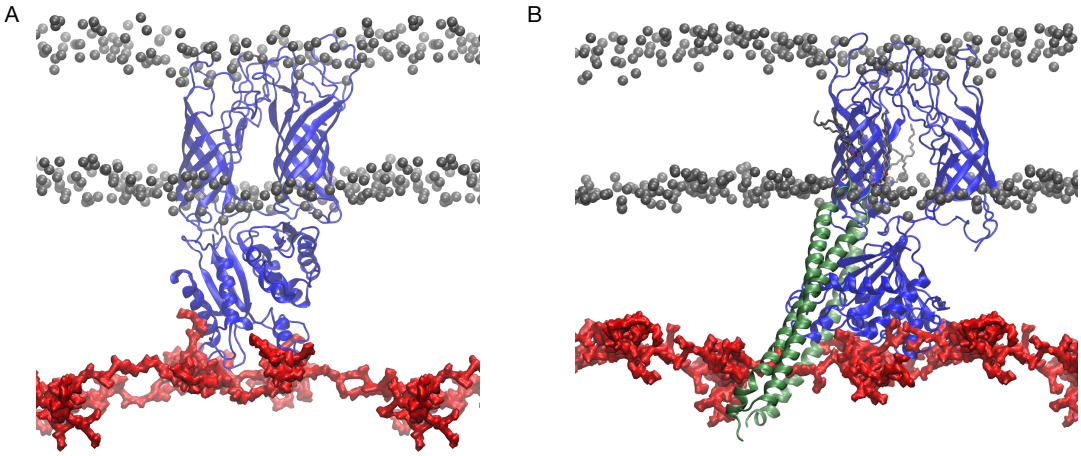

Figure S1, related to Figure 1: A snapshot at the end of a 500 ns simulation of systems containing (A) OmpA dimer without BLP, and (B) OmpA dimer with BLP. The OmpA dimer and BLP are in cartoon representation in blue and green, respectively. The PGN network is in surface representation in red, while the phosphorus atoms of the OM are shown as grey spheres.

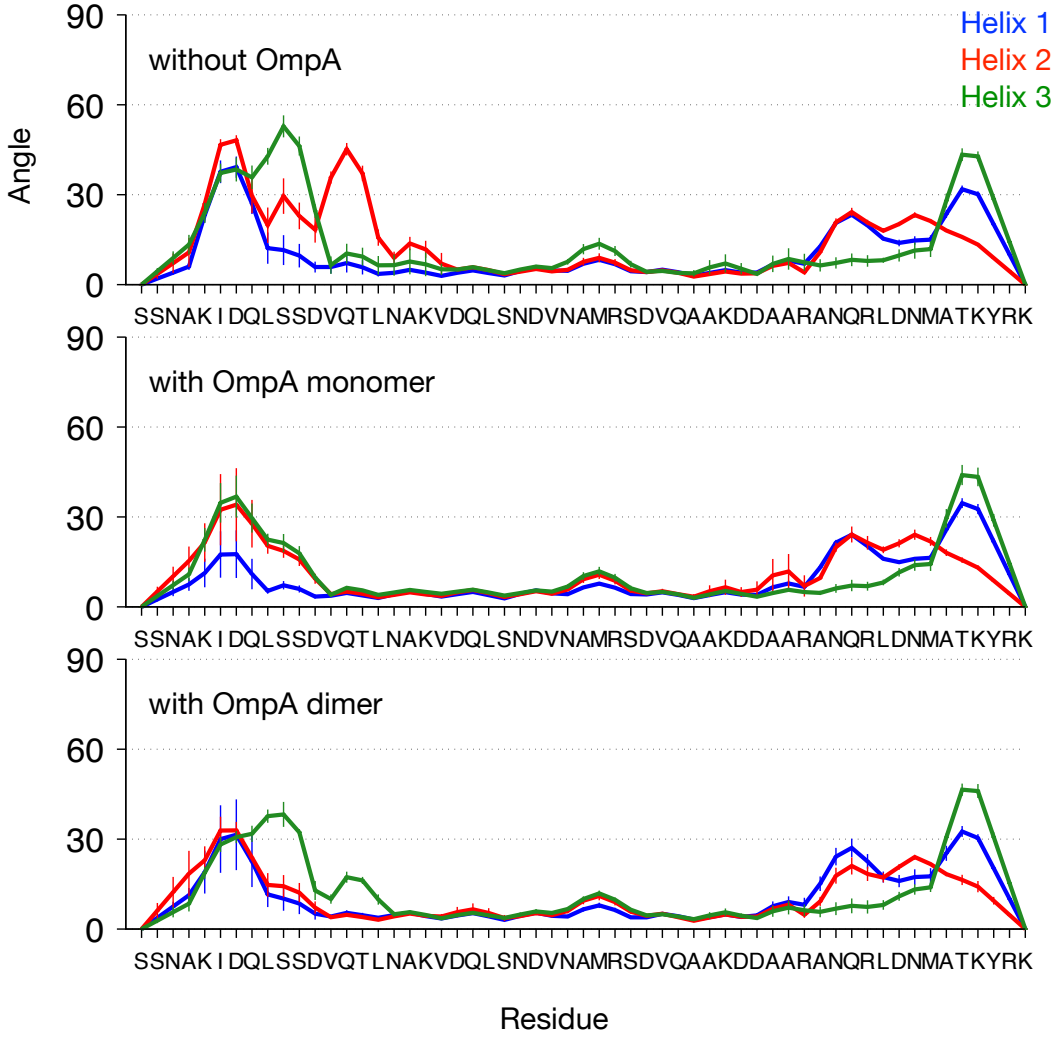

Figure S2, related to Figure 2: Helix kink angle for each residue in each helix of the BLP trimer, measured using Bendix (Dahl et al., 2012). This analysis is performed separately for systems without OmpA (top), with OmpA monomer (middle), and with OmpA dimer (bottom). The angle value is averaged over all independent repeats of the simulations for each system and the error bars represent standard deviation.

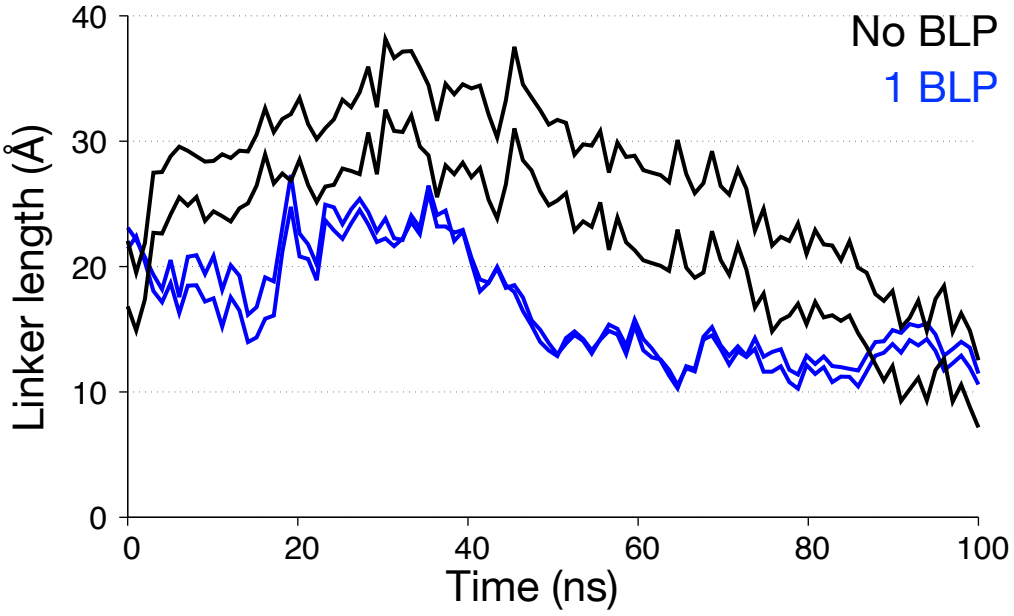

Figure S3, related to Figure 5: The length of the linker between OmpA NTD and CTD from one dimer simulation of a system with one BLP attached to the PGN (blue) and a system without any BLP present (black).

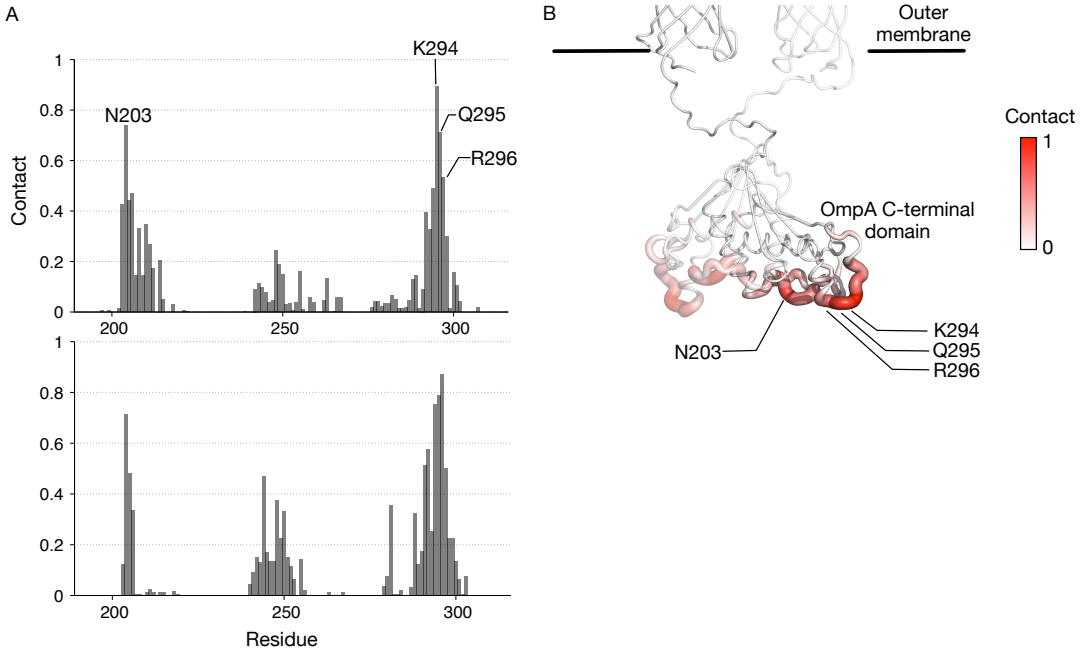

Figure S4, related to Figure 5: Residues involved in PGN initial binding. (A) Contact analysis performed for each residue of the OmpA CTD averaged over all simulations for the OmpA dimer system (top) and simulations with BLP for the OmpA monomer system (bottom). A score of 1 indicates contact throughout the entire 100 ns simulation. A distance cut-off of 4 Å was used for the analysis. (B) The results of the contact analysis mapped onto the structure of OmpA dimer to highlight the positions of residues involved.

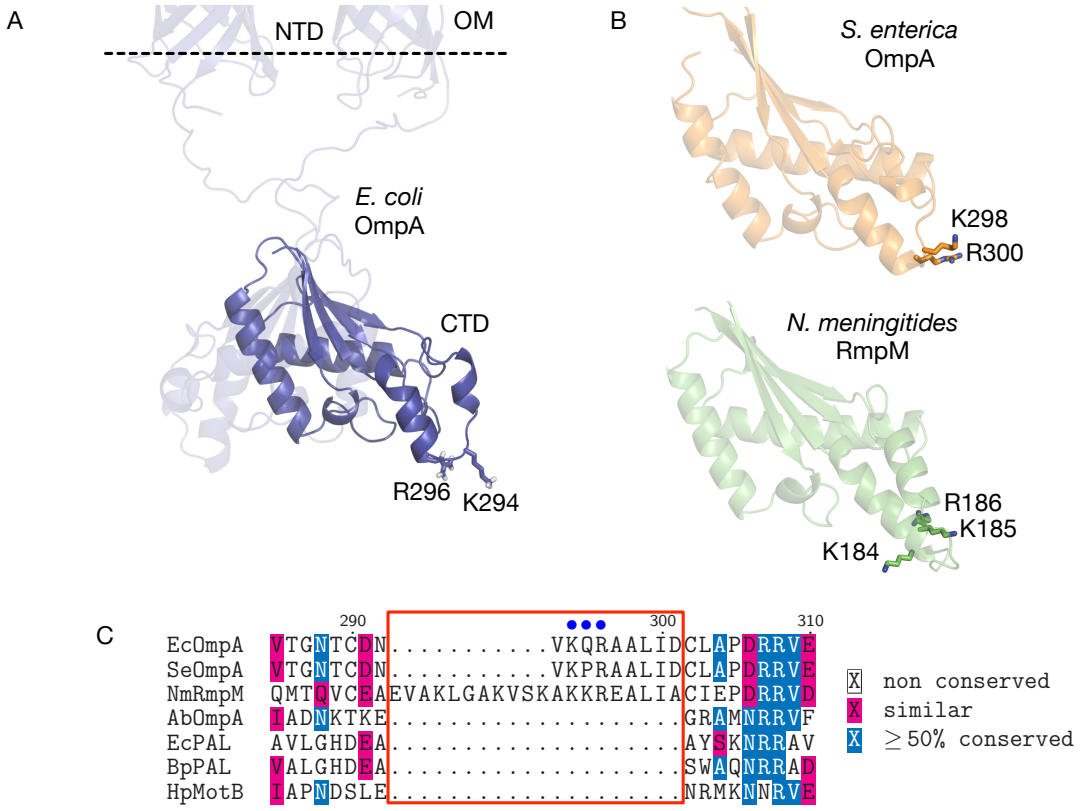

Figure S5, related to Figure 5: Key residues for PGN initial binding are conserved in two other species. (A) The positions of K294 and R296 in full length *E. coli* OmpA dimer as mapped onto a model from Marcoux et. al. (Marcoux et al., 2014). (B) The positions of equivalent residues in homologs, *S. enterica* OmpA (PDB: 4RHA) and *N. meningitides* RmpM (PDB: 1R1M) (Grizot and Buchanan, 2004). (C) Sequence alignment of *E.coli* OmpA with six PGN binding proteins, i.e. *S. enterica* OmpA (SeOmpA), *N. meningitides* RmpM (NmRmpM), *A. baumannii* OmpA (AbOmpA) (PDB: 3TD5), *E. coli* PAL (EcPAL) (PDB: 1OAP), *B. pseudomallei* PAL (BpPAL) (PDB: 4B5C), and *H. pylori* MotB (HpMotB) (PDB: 3CYP). The large insert present only in *E.coli*, *S. enterica*, and *N. meningitides* is shown in the red box. Three key residues for

initial binding with PGN are highlighted by the blue circles. Conserved and similar residues are marked in light blue and magenta, respectively.

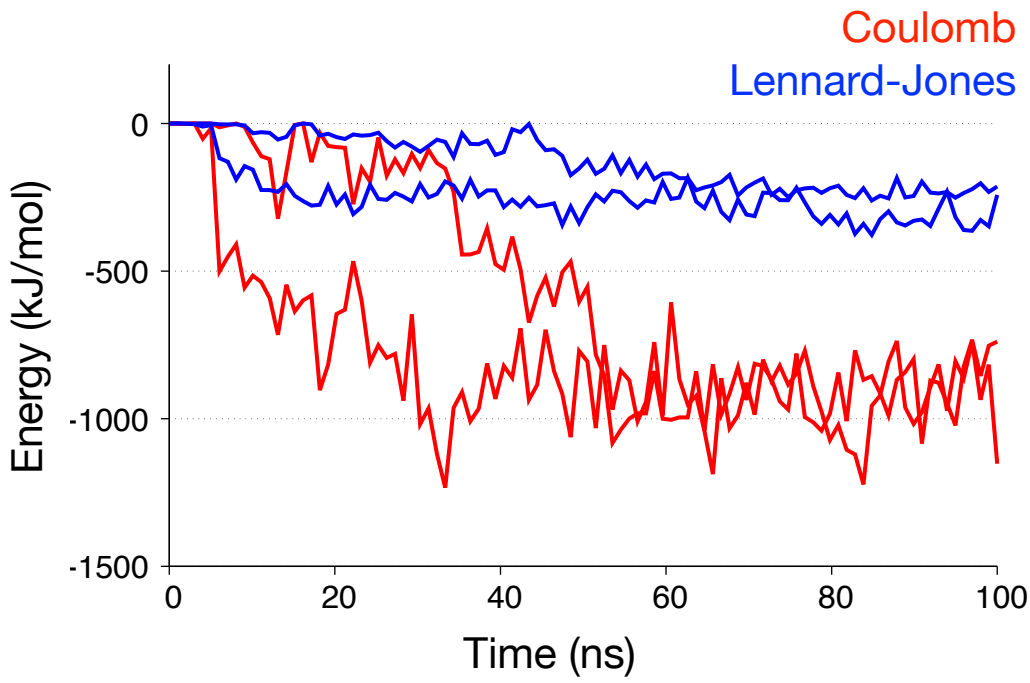

Figure S6, related to Figure 5: Energy decomposition of OmpA and PGN interactions for the two independent simulations of a system without BLP at 310 K. The non-bonded energies are decomposed into their Coulombic (red) and Lennard-Jones (blue) components.

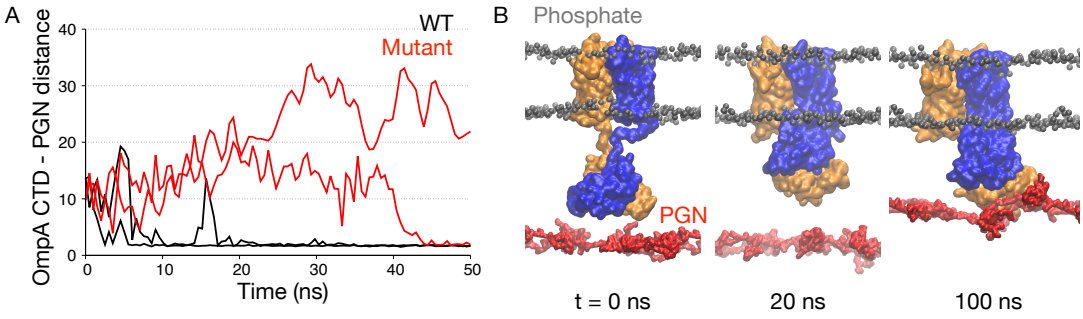

Figure S7, related to Figure 5: Mutation of key residues impeded binding to PGN. (A) Minimum distance between the OmpA CTD and the PGN network for two independent simulations of wild type OmpA (black) and mutant (red), where residues N202, K294, Q295, and R296 were mutated to alanine. Only the first 50 ns of the 100 ns simulation are shown for clarity. (B) Snapshots of a simulation with OmpA mutant at three different time points. The two subunits of OmpA dimer were coloured in blue and orange, to highlight that only one subunit interacted with the PGN.