Binding From both sides: TolR and full-length OmpA bind and maintain the local structure of the *E. coli* cell wall.

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SUPPLEMENTAL INFORMATION
Figure S1, Related to Figure 1: Thickness of one, two, or three layers of PGN cell wall during 100 ns simulations, measured as the distance along the z-axis between the lowest and highest points in the molecule.
Figure S2, Related to Figure 1: All atom root means square deviation (RMSD) of (A) the N-terminal beta barrels (residue 1-172) of OmpA, (B) the C-terminal periplasmic domain (residue 189-316) of OmpA, (C) the N-terminal transmembrane domains (residue 1-36) of TolR, and (D) the C-terminal periplasmic domain (residue 62-141) of TolR. Data from three independent simulations and the RMSD of each protomer plotted separately. (E) Secondary structure preservation for the periplasmic domain of one TolR protomer during one of the simulations. (F) Snapshots of TolR periplasmic domain in ribbon representation throughout one of the simulations to illustrate the flexibility of the C-termini.
Figure S3, Related to Figure 2: Minimum distance between the carboxyl group on Pro141 (from both protomers) to the PGN cell wall in the first 10 ns of all three simulations.
Figure S4, Related to Figure 2: The center of mass motion along the z-axis for OmpA C-terminal domain, PGN cell wall, and ToIR periplasmic domain (as described in Figure 2) for two extended simulations.
Figure S5, Related to Figure 3: (Top) Binding energy of one of TolR protomers to the cell wall from one simulation decomposed into its Coulombic and Lennard-Jones components. (Middle and bottom) The number of hydrogen bonds and salt bridges formed between the TolR protomer and the cell wall during this simulation. The shaded region highlights a portion of the simulation whereby the highest number of hydrogen bonds were formed, which corresponds to the highest (most negative) binding energy.
Figure S6, Related to Figure 3: Electrostatic surface map of the whole length TolR (left) and the periplasmic domain as observed from the periplasm (right) for the (A) open and (B) closed states. The positions of key residues in cell wall binding (as highlighted in Figure 2C) are labelled. Examples of solvent exposed charged residues in the closed state are also labelled in B.
Figure S7, Related to Figure 3: Steered MD of TolR and OmpA periplasmic domains. (A) Force profile whereby the periplasmic domain of TolR was pulled away from the cell wall. Data show average of three independent steered MD simulations and the error bars indicate standard deviation. Snapshots at the beginning and at the end of one of the simulations are shown. (B) Similar to (A) for steered MD simulations whereby the periplasmic domain of OmpA was pulled away from the cell wall.
Figure S8, Related to Figure 4: Simulation of TolR with BLP and full-length OmpA. (A) A snapshot at the end of a 200 ns simulation. (B) The center of mass motion along the z-axis of OmpA C-terminal domain, PGN cell wall and TolR periplasmic domain as described in Figure 2. (C) BLP tilt angle from three independent simulations as measured in Figure 4.