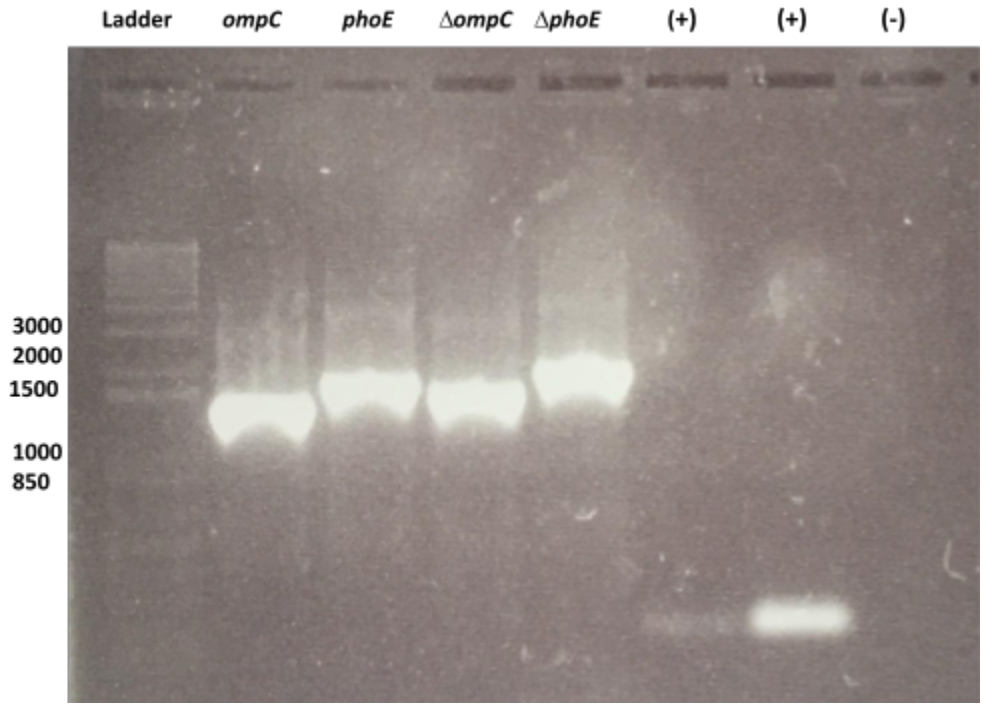

FIG S1. Genotype validation of wildtype, Δ*ompC* and Δ*phoE* strains. Visualization of the *ompC* and *phoE* genes within the wild type strain, and kanamycin resistance cassettes within each mutant strain on a 1% agarose gel. Amplification of puC19 was used as a positive control.

Table S1. Primers for genotype validation used in this study. Taken from Boen *et al.* (11)

Primer	**Primer Sequence 5' → 3'**
ompC forward	G AGA ATG GAC TTG CCG ACT GAT TAA TGA G
ompC reverse	CAC GCC AGA AGG TAC CCA TAG TTT TG
phoE forward	GA TAT CAA ACG AAC GTT TTA GCA GGA CTG TCG TCG GTT G
phoE reverse	GA GCT GGA AGC GCA GGA ATC CCG TTT TAC
Control forward	GCA AAT AAA GGC ATA TAA CAG AGG GTT AAT AAC ATG
Control reverse	C AGG CCC TTT GTT CGA TAT CAA TCG AGA TTA

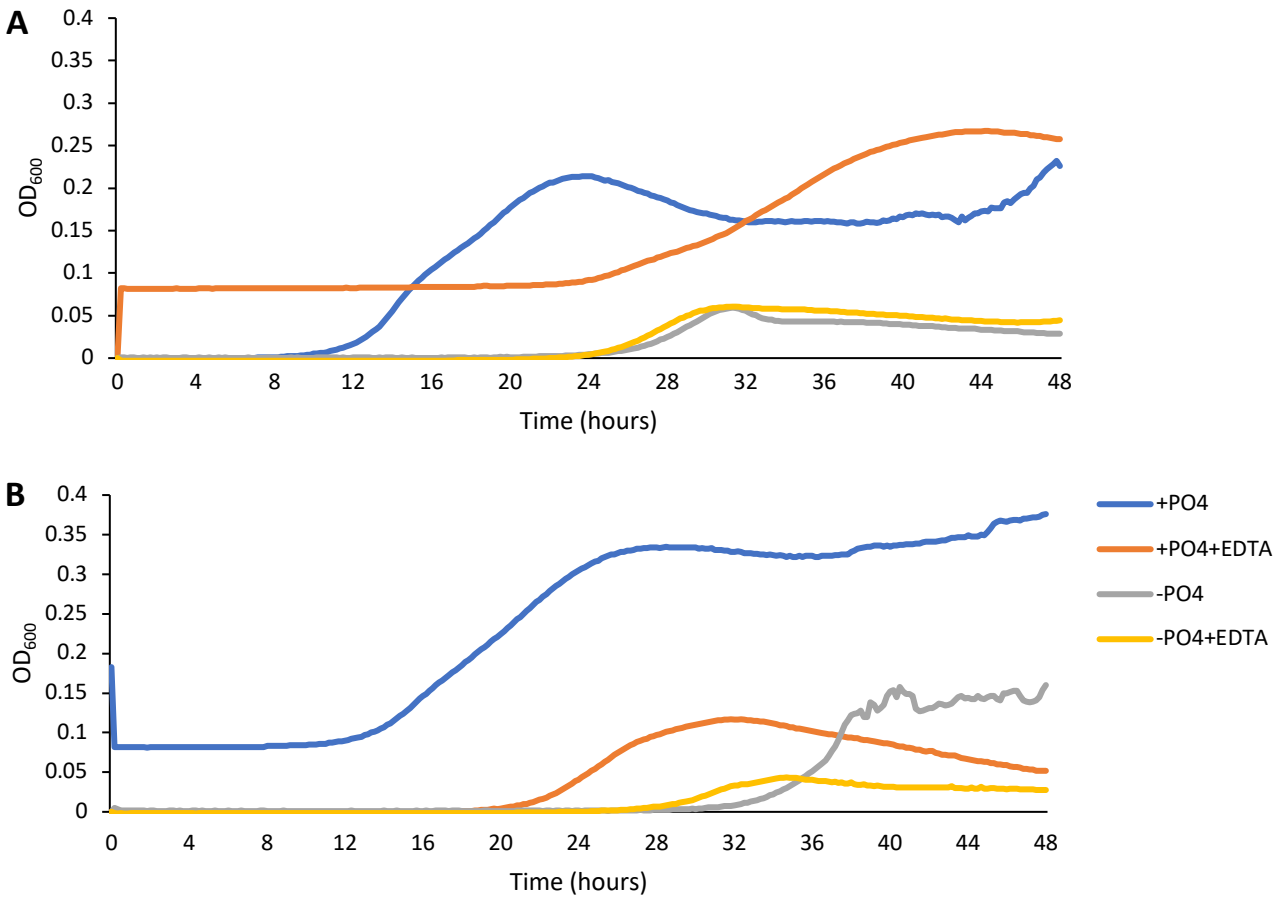

FIG S2. SDS-EDTA growth curves of *E.coli* (A) wild type and (B) Δ*ompC* strains in phosphate sufficient and deficient media with 0.05% SDS and 0.30 mM EDTA.

1x10^{5}cells from 48 hour starter cultures were added to a 96-well plate and incubated for 48 hours at 37°C. OD_{600} was measured every 10 min for 48 hours (n=3).

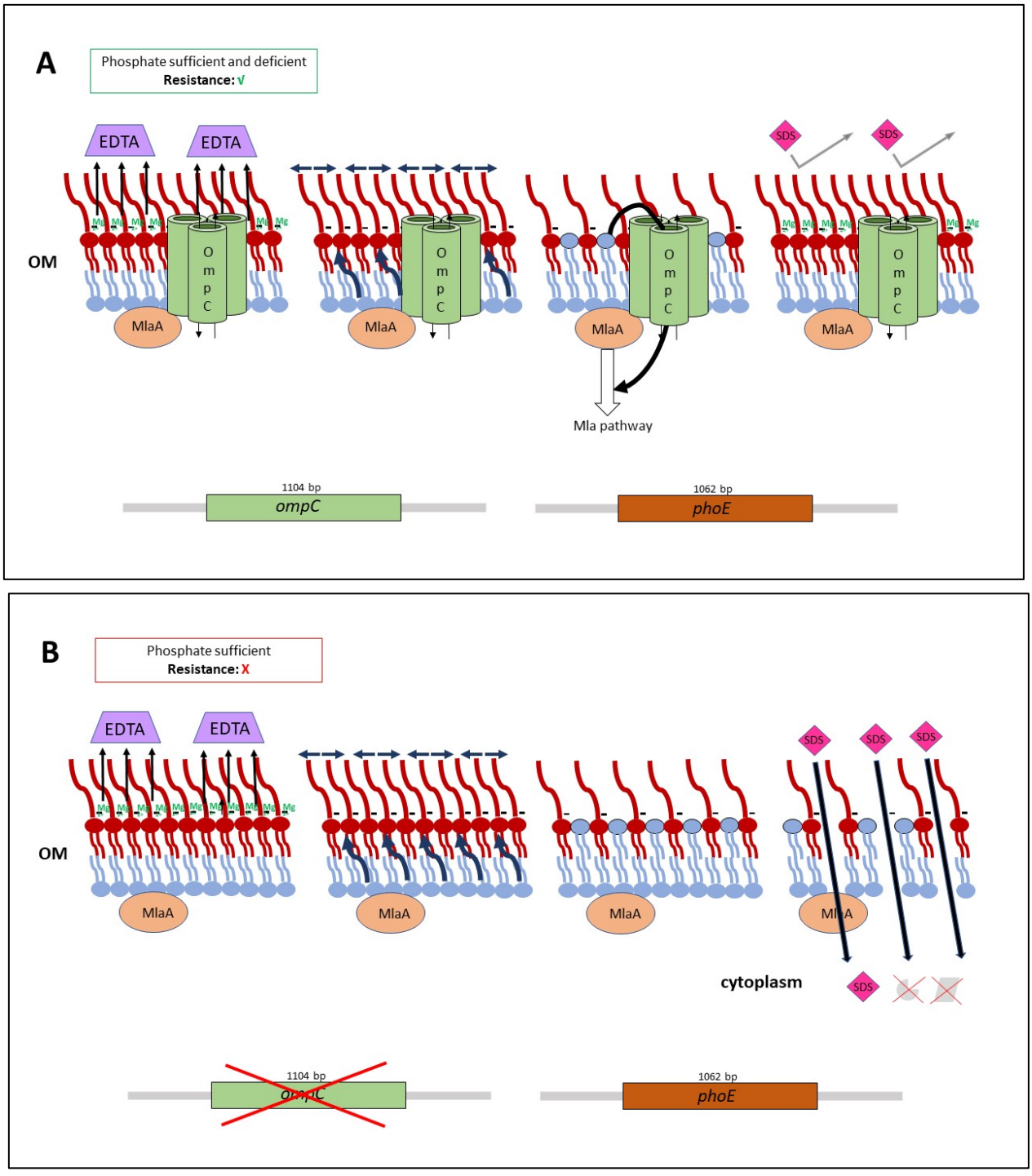

Fig S3. Possible mechanism for maintaining OM asymmetry in *Escherichia coli* via osmoporin OmpC in phosphate sufficient and phosphate deficient media and

the effect on the OM in the absence of *ompC*. When *ompC* is constitutively expressed, resistance to SDS-EDTA is conferred, because OM asymmetry is maintained in wild type cells via OmpC in both phosphate sufficient and deficient media. *Not shown: *phoE* would be expressed in phosphate deficient media in wild type (A). Resistance to SDS-EDTA is not conferred, as OM asymmetry is compromised due to the absence of *ompC* in phosphate sufficient media (B).