

SUPPLEMENTAL FIGURES

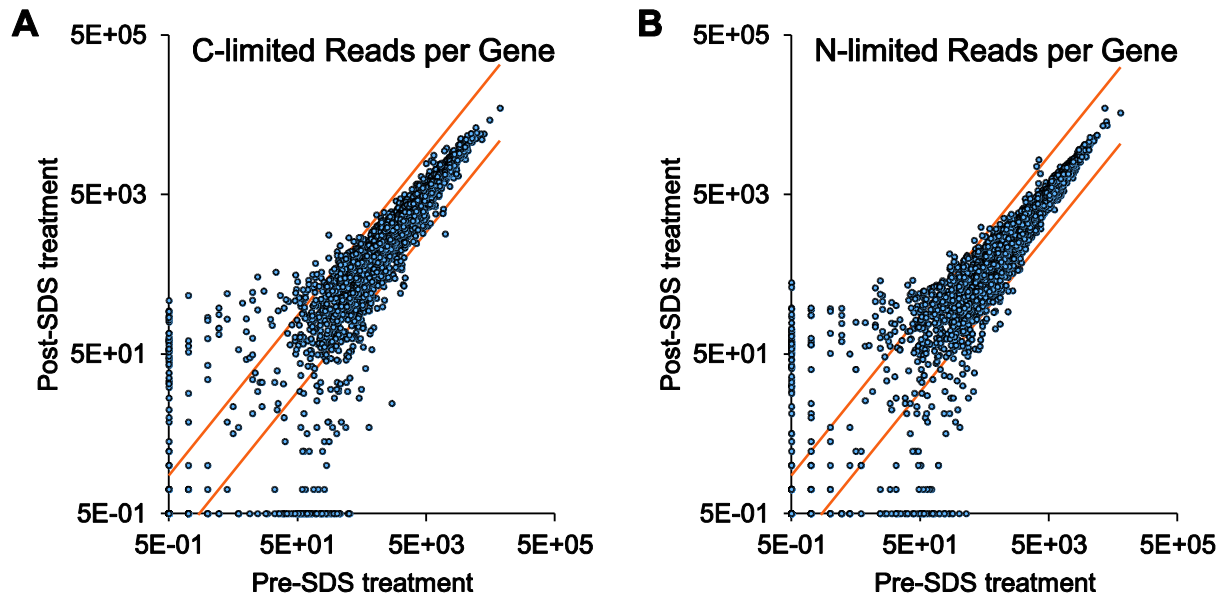


Figure S1. Comparison of sequencing reads per gene in pre- and post-treatment samples. The comparison of reads per gene in pre- and post-treatment for all genes is shown for carbon-limited samples (A) and for nitrogen-limited samples (B). Each data point represents a single gene. Orange lines represent a 3-fold change between pre- and post-treatment samples. For genes with zero reads, the value was replaced with 0.5 to allow display on a logarithmic scale.

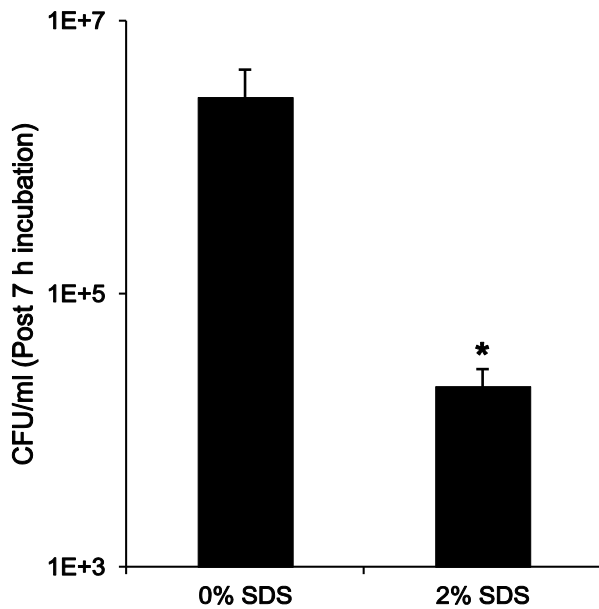


Figure S2. *wecE* mutant cells are SDS sensitive in exponential phase. Actively growing cells with a deletion of *wecE* were treated with SDS. Viability was assessed after 7 hours of treatment. * $p < 0.05$ compared to untreated sample

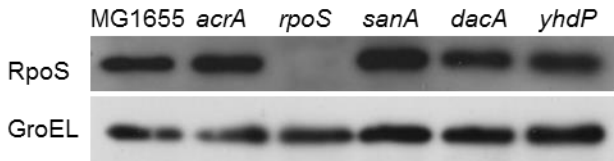


Figure S3. RpoS levels are not affected by *acrA*, *sanA*, *yhdP*, or *dacA* deletion. Protein levels of RpoS were determined by immunoblot analysis for the indicated strains in carbon-limited conditions as has been previously described with minor modifications (1): cells were lysed in sample loading buffer without protein precipitation and GroEL (Sigma G-6532, 1:10 000 dilution) was included as a loading control.

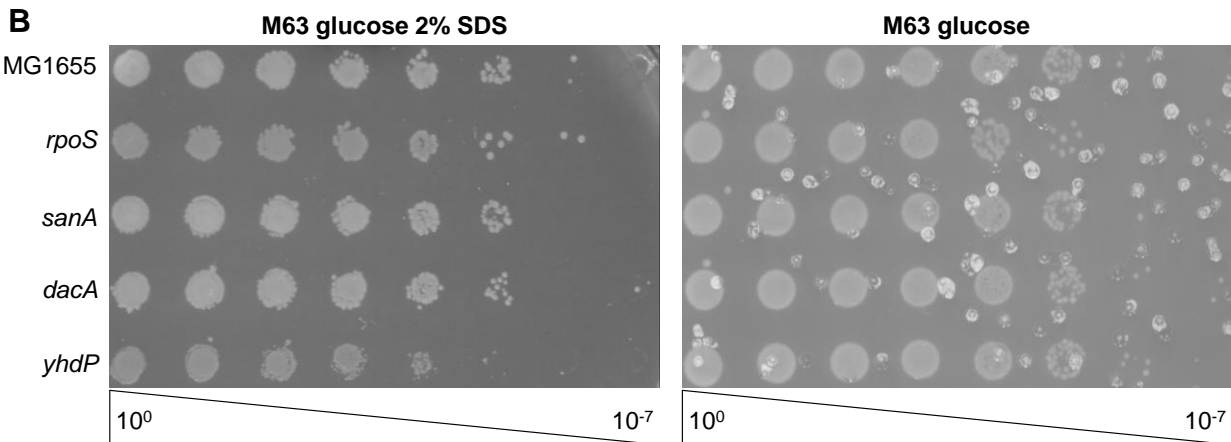
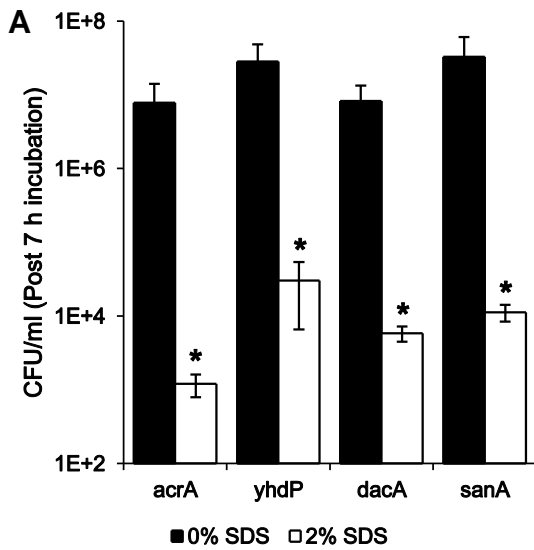


Figure S4. Genes identified in RpoS-dependent mechanism of SDS resistance have a small effect on SDS resistance in exponential phase. **A.** Actively growing cells with deletions of either *acrA*, *yhdP*, *dacA*, or *sanA* were treated with SDS and viability was assayed 7 h post-treatment. * $p < 0.05$ against untreated sample. **B.** LB overnight cultures were serially diluted and plated on M63 glucose media with or without 2% SDS for an efficiency of plating assay.

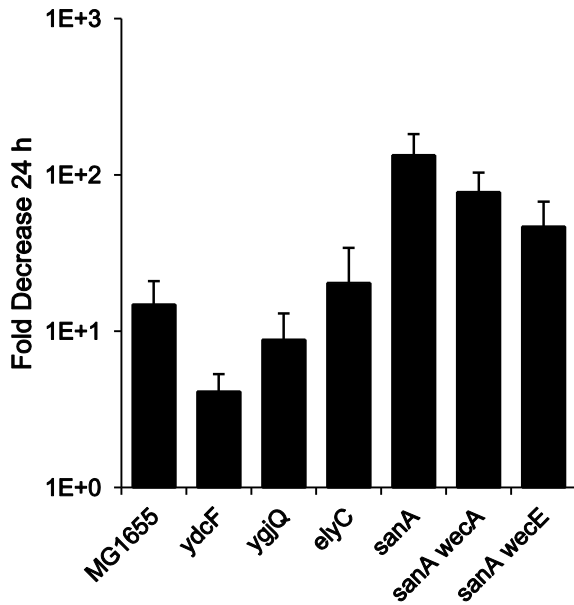


Figure S5. *sanA* acts in a different manner than its homologs, including *elyC*. Carbon-limited cells with the indicated genotype were treated with 2% SDS and incubated for 24 h before viability was assayed. The fold decrease of the SDS treated sample compared to the untreated sample is shown. No significant changes were observed between the parent (MG1655 for *ydcF*, *ygjQ*, and *elyC*, *sanA* for *sanA wecA* and *sanA wecE*) and mutant strains.

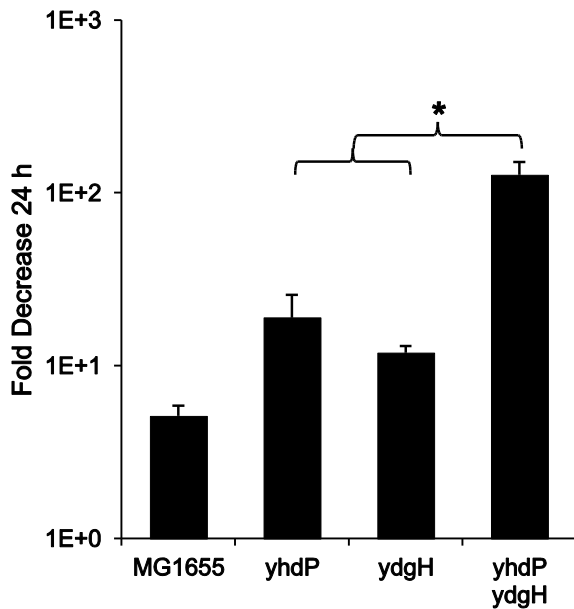


Figure S6. *yhdP* and *ydgH* are not epistatic. Carbon-limited cells from the indicated strains were treated with 2% SDS for 24 h and then viability was assayed. The fold decrease of the SDS treated sample compared to the untreated sample is shown. * $p < 0.05$ against indicated samples

SUPPLEMENTAL TABLES

Table S1. Strains used in this study

Strain Name	Genotype
MG1655	K-12 F ⁻ λ ⁻ <i>rph-1</i> (Coli Genetic Stock Center 6300)
AM102	MG1655 Δ <i>acrA</i>
AM103	MG1655 Δ <i>acrB</i>
AM144.5	MG1655 <i>sprE</i> :Tn10
AM145	MG1655 Δ <i>acrA sprE</i> :Tn10
AM104	MG1655 Δ <i>rpoS</i>
AM146	MG1655 Δ <i>rpoS sprE</i> :Tn10
AM136	MG1655 Δ <i>acrA ΔrpoS</i>
AM137	MG1655 Δ <i>acrB ΔrpoS</i>
AM163	MG1655 <i>ydgH</i> ::Kan
AM172	MG1655 <i>bssR</i> ::Kan
AM173	MG1655 <i>tatB</i> ::Kan
AM174	MG1655 <i>wecA</i> ::Kan
AM175	MG1655 <i>ompA</i> ::Kan
AM176	MG1655 <i>dacA</i> ::Kan
AM144	MG1655 <i>sanA</i> ::Kan
AM177	MG1655 <i>yftK</i> ::Kan
AM179	MG1655 <i>wecE</i> ::Kan
AM180	MG1655 <i>rfaH</i> ::Kan
AM178	MG1655 <i>yhdP</i> ::Kan
AM187	MG1655 Δ <i>rpoS yhdP</i> ::Kan
AM190	MG1655 Δ <i>rpoS dacA</i> ::Kan
AM149	MG1655 Δ <i>rpoS sanA</i> ::Kan
AM184	MG1655 Δ <i>rpoS ydgH</i> ::Kan
AM193	MG1655 Δ <i>rpoS rfaH</i> ::Kan
AM186	MG1655 Δ <i>acrA yhdP</i> ::Kan
AM189	MG1655 Δ <i>acrA dacA</i> ::Kan
AM148	MG1655 Δ <i>acrA sanA</i> ::Kan
AM183	MG1655 Δ <i>acrA ydgH</i> ::Kan
AM192	MG1655 Δ <i>acrA rfaH</i> ::Kan
AM182	MG1655 Δ <i>yhdP</i>
AM197	MG1655 Δ <i>dacA</i>
AM196	MG1655 Δ <i>yhdP sanA</i> ::Kan
AM200	MG1655 Δ <i>yhdP ΔdacA</i>
AM199	MG1655 Δ <i>dacA sanA</i> ::Kan
AM201	MG1655 Δ <i>yhdP ΔdacA sanA</i> ::Kan
AM198	MG1655 Δ <i>yhdP ydgH</i> ::Kan
AM215	MG1655 <i>ycfF</i> ::Kan
AM216	MG1655 <i>ygjQ</i> ::Kan
AM217	MG1655 <i>elyC</i> ::Kan
AM158	MG1655 Δ <i>sanA</i>
AM221	MG1655 Δ <i>sanA wecA</i> ::Kan
AM222	MG1655 Δ <i>sanA wecE</i> ::Kan

Table S2. Tn-Seq reads per gene

Data Set	Reads per gene	
	Average	Median
Carbon-limited pre-treatment	3017	1740
Carbon-limited post-treatment	3077	1813
Nitrogen-limited pre-treatment	2673	1443
Nitrogen-limited post-treatment	2713	1439

Table S3. Descriptive statistics for Tn-Seq data

Statistic	Log2 Fold Post/Pre treatment	
	Carbon-limited	Nitrogen-limited
Average	-0.04	0.06
Median	0	0
Standard Deviation	1.43	1.35
Max	8.87	9.61
Min	-9.35	-9.01

Table S4. Non-envelope related Tn-Seq hits causing SDS sensitivity only in carbon-limited cells

Gene ^a	Carbon-limited Reads per Kbp ^b		Log2 Fold Post/Pre treatment ^c	
	Pre-treatment	Post-treatment	Carbon-limited	Nitrogen-limited
<i>dam</i>	1338	174	-2.2	0.9
<i>paaD</i>	1739	231	-2.2	0.3
<i>ccmC</i>	4352	735	-1.9	0.2
<i>bioA</i>	1552	274	-1.8	0.2
<i>argH</i>	1539	298	-1.7	1.1
<i>aceA</i>	6995	1386	-1.6	-0.1
<i>xerC</i>	1216	241	-1.6	-0.2
<i>rluC</i>	1385	282	-1.6	0.8

^a Genes with at least 700 sequence reads, decreasing at least 3-fold during treatment in carbon-limiting conditions, and changing less than 3-fold in nitrogen limiting conditions

^b The number of sequence reads for each gene was normalized to the length of the gene

^c The fold change in read number before and after SDS treatment in either carbon- or nitrogen-limiting conditions

SUPPLEMENTAL REFERENCES

1. Mandel, M. J., and T. J. Silhavy. 2005. Starvation for different nutrients in *Escherichia coli* results in differential modulation of RpoS levels and stability. *Journal of Bacteriology* 187:434-442.