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Supplementary Materials for

Identity and Function of a Large Gene Network Underlying Mutagenic Repair of DNA Breaks

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Materials and Methods

Strains and materials

Strains and plasmids used in this study are given in Table S4. Strains were constructed using standard techniques (*29, 30*). To construct strains for selection of I-*Sce*I-induced nalidixic-acid resistance (Nal^R) forward base-substitution mutations in the *gyrA* gene, we introduced I-*Sce*I-cut site-*cat*FRT or I-SceI-cut site-KanFRT 273 bp downstream of the *gyrA* gene (insertion/deletion site: 2334541-233503) by recombineering (*30*) using PCR products from plasmids pKD3 and pKD4, respectively, and primers: P531 = 5' GGC GTT GCA ATA TGC TGG CAG TGG GCC AGA AAA AAC GTT G **GTG TAG GCT GGA GCT GCT TC** 3' and P533 = 5' CTC AGA GAG TCG CTG TCG AGA AAG AGG TCC GGC TCA TTG A *ATT ACC CTG TTA TCC CTA* **CAT ATG AAT ATC CTC CTT AG** 3' (normal font, *gyrA* downstream homology; bold, pKD3/pKD4 homology; Italics, I-*Sce*I-cut site). Sequencing was performed by SeqWright DNA technology services (Houston). M9 minimal medium (*29*) had carbon sources added at 0.1% and thiamine (vitamin B1) at 10 μg/ml. Minimal A medium (*29*); LBH medium (*31*). Antibiotic and other additives (Sigma) were used at the following final concentrations: ampicillin (100 μg/ml), chloramphenicol (25 μg/ml), kanamycin (50 μg/ml), tetracycline (10 μg/ml), rifampicin (100 μg/ml), 5-bromo-4-chloro-3-indolyl- β-D-galactoside (X-gal, 10 μg/ml), phenyl-β-D-galactoside (P-Gal, 500 μg/ml) and sodium citrate (20 mM). Exonuclease I, Taq DNA polymerase and dNTPs were from New England Biolabs, Inc.

Qualitative colony-color papillation assay for stress-induced reversion of a *lac* frameshift allele

We developed a qualitative papillation assay for DSB-dependent stress-induced mutagenesis (SIM) (*8*), based on reversion of a conjugative-plasmid (F')-borne *lacI-lacZ* +1 bp frameshift allele to Lac^+ during prolonged starvation (*32*), an assay in which nearly all of the mutagenesis under experimental starvation conditions is DSB-dependent SIM (*3*). This assay was conducted as previously (*8*) for the first 40,000 mutants screened (Fig. S1, screen 1), or modified as follows for improved efficiency of detection of SIM-down mutants, based on Lac-reversion screens of Miller (*29*), for the next 19,000 screened (Fig. S1, screen 2). Cultures were spotted into grids on papillation plates: minimal A medium containing 0.035% glycerol, 500 μg/ml of P-Gal (a non-inducing lactose analogue that serves as a carbon source for Lac^+ revertants) and 10 μg/ml of the β-galactosidase color indicator X-Gal. Plates were incubated at 37°C for 8-10 days. After exhausting the limiting glycerol carbon source, the colony is starved for carbon and stops growing. However, any Lac^+ revertant cells that arise within the Lac^- colonies during the prolonged starvation use the P-Gal as a carbon source, and produce Lac^+ mutant microcolonies (papillae) within the larger growth-arrested Lac^- colony. The Lac^+ papillae are stained blue by X-Gal (Fig. 1B), which is hydrolyzed to an insoluble blue dye by β-galactosidase (encoded by the $lacZ^+$ gene). The number of blue Lac^+ papillae

reflects DSB-dependent stress-induced Lac reversion-proficiency with "wild-type" (WT) showing abundant papillae and known DSB-dependent-SIM-defective mutants, such as *rpoS* (SMR6541), *recA* (SMR5535), *recB* (SMR593), *lexA*Ind$^-$ (SOS "off") (SMR820), or *dinB* (SMR5830) showing fewer (Fig. 1B).

For the semi-quantitative version of the improved version of this assay (data in Table S1), cells were streaked on papillation medium and grown for 8-10 days at 37°C. Papillae were counted for at least for four colonies. Fold decrease is the number of papillae of the WT (control) divided by the number of papillae of the mutant assayed in parallel then averaged for 3 independent experiments.

Transposon-insertion mutagenesis and screen for SIM-deficient (SIM-down) mutants, secondary screens and mutation sequencing

We isolated mutants defective in DSB-dependent SIM by screening libraries of random transposon-insertion mutants for reduced mutagenesis using two qualitative colony papillation assays [(*8*), and as improved above, **Qualitative colony-color papillation assay**].

Two successive libraries of mini-Tn*10* (Tn*10*dCam) transposon-insertion mutants were constructed [per (*9*)], first, in Lac-reversion-assay strain FC40 or identical strain SMR4562, from which 40,000 mutants were screened (*8*) (Fig. S1, screen 1), then for the second screen of 19,000 mutants, in their F$^-$ parent strain FC36, a chromosomal *lac* deletion strain (Fig. S1, screen 2). Although DSB-dependent SIM is detected very sensitively in SMR4562, which carries the revertible *lacI-Z* +1bp frameshift allele in an F′ plasmid, most transposon insertions target the F′, and require secondary screening for chromosomal insertions (*8*). Therefore, the second library, from which a total of 19,000 insertions were screened, was made in FC36 (F$^-$) to avoid insertions into the F′ plasmid, and the F′ was moved in afterward. After transfection with phage bearing the transposon, cells were plated onto minimal proline with 25 μg/ml chloramphenicol (Cam) plates to select Cam-resistant (Cam^R) transposon-insertion mutants. Cam^R colonies were screened for SIM-deficient phenotypes by gridding onto Lac-assay papillation plates (**Qualitative colony-color papillation assay**, above) seeded with the F′ donor strain SMR6269, to transfer the F′ with revertible *lacIZ* allele by conjugation. Neither auxotrophic parent, and so only conjugants, can grow on these plates, on which the ability to mutate during starvation is assayed and scored visually as described above (**Qualitative colony-color papillation assay**, Figs. 1B, S1AB).

Colonies with fewer papillae than the "WT" SMR4562 (Fig. 1B), are candidate SIM-deficient or "SIM-down" mutants. These were purified and further screened *via* a variety of secondary screens outlined in Fig. S1, including heritability of the papillation phenotype, linkage of the SIM-down phenotype with the transposon, determined by papillation assay after transduction of the Tn*10*dCam into a "clean" non-mutagenized strain, and quantitative SIM assays performed with either the original transposon-insertion allele after transduction into a new strain or with deletion alleles of non-essential genes after gene identification determined by sequencing. The SIM rates from these quantitative assays for all new SIM network genes/mutants identified are given in Table S3, summarized Table S1).

The genes mutated were identified by sequencing as follows. The Tn*10*dCam insertion site was amplified by a two-stage single-primer PCR, followed by sequence analysis by SeqWright (Houston). First, to 20 μl of the reaction volume, we added 100 ng of genomic DNA as template, 10 pmol of primer P1 (5′ GCCGCTGGCGATTCAGGTTC 3′; specific to the *cat* gene in the mini-Tn*10*dCam), and the DNA was subjected to linear amplification in PCR buffer (10 mM Tris-HCl pH 8.3, 50 mM KCl, 1.5 mM $MgCl_2$, 0.2 mM each of dATP, dTTP, dCTP and dGTP, and 2.5 units of Taq DNA polymerase) as follows. Cycle 1: 94° for 4 min; cycles 2-20: 94° for 30 sec; 55° for 30 sec; 72° for 2 min. This product was then used in the second stage of PCR with the same primer P1 and annealing performed at low annealing temperature. At low annealing temperature the primer not only anneals at the end of the transposon sequence, but will occasionally also bind nonspecifically to sequences downstream of the transposon setting the stage for further amplification geometrically. To 50 μl reaction volume, we added 5 μl of the stage 1 product, 20 pmol of primer P1, and the DNA was subjected to amplification in PCR buffer as follows. Cycle 1: 94° for 2 min; cycles 2-10: 94° for 5 sec; 35° for 5 sec; 72° for 2 min; cycles 11-35: 94° for 5 sec; 55° for 5 sec; 72° for 2 min; cycle 36: 72° for 5 min. This PCR product was then digested with 1 unit of exonuclease I (37° for 1h) to remove any excess ssDNA present in the PCR products, purified with PCR purification Kit (Qiagen), and used as template for sequencing with primer P2 (5′ TACAACAGTACTGCGATGAG 3′), which binds downstream of primer P1 in the transposon sequence. To validate this experimental approach for determining sequence into which transposons were inserted in the SIM-down mutants, the site of insertion of a previously defined *rpoE* promoter mutant (*8*) was determined. After sequencing 200 mutants from screen 2 (Fig. S1C), we found that they represented 93 genes (many were "hit" in two or more independent mutants). An additional 3 genes not found in screen 2 were identified in screen 1, along with 10 genes found also in screen 2.

We verified the mutants identified using *bona fide* null alleles of each of the non-essential genes identified using the Keio deletion collection (*33*), and the quantitative stress-induced Lac reversion assay (*34*) as described below. For polycystronic genes that were not last in the operon, the Kan gene that disrupts each Keio deletion allele was excised at its flanking *FRT* sites to make nonpolar deletion alleles, per (*33*). For essential genes, the quantitative assays and subsequent studies were done with the non-null transposon-insertion alleles that we had isolated after transduction into "clean" non-mutagenized cells. Results of the quantitative Lac assay are given in Table S3, summarized Table S1.

Quantitative SIM assays with and without enhancement by I-*Sce*I-induced double-strand-breaks

Standard (no I-*Sce*I) assays were performed per (*34*). For a more sensitive assay with higher SIM rates, and thus greater range for measuring the magnitude of SIM defects of SIM-down mutants, and double mutants in epistasis (double-mutant) analyses, we used I-*Sce*I-endonuclease induced cleavage near *lac*, which increases DSB-dependent SIM dramatically over rates without local I-*Sce*I cleavage (*1*). Bacterial strains carrying the inducible double-strand-break (DSB) system consisting of a chromosomally encoded I-

*Sce*I endonuclease gene controlled by the glucose-repressible P_{BAD} promoter and an F′ cut-site near *lac* (*1*) were maintained on glucose medium to repress I-*SceI* synthesis and experiments performed per (*1, 35*). Lac^+ colonies originate either by a -1-bp frameshift "point" mutation (*36, 37*) or by amplification of the leaky *lac* allele to multiple copies (*38, 39*). Because amplification accounts for 5% or fewer of the Lac^+ colonies arising on day 5 or earlier (*38*), data were not corrected to exclude amplification. Relative viability was determined per (*34*) and wherever necessary the mutation rates were normalized for relative viability per (*1*).

Reconstruction experiments: demonstration that electron-transfer mutants are mutagenesis-, not growth-defective

We used reconstruction experiments to determine whether the electron transfer mutants were defective in mutagenesis, or only in their speed of formation of Lac^+ colonies under selection conditions [reviewed, (*40*)]. Slow colony growth could give the appearance of fewer stress-induced mutants, when actually the mutants formed but failed to produce colonies rapidly enough to be scored (*40*). We constructed strains carrying mutations for major electron-transfer genes and the Lac^+ allele and determined their speed of colony formation on lactose medium in the presence of neighbor cells, under exact reconstructions of quantitative Lac assay conditions (results shown in Table S13). The electron-transfer mutant alleles were transduced into three Lac^+ day-5 stress-induced mutants (SMR3856, SMR3858 and SMR3859) that had been characterized with respect to colony forming ability on lactose medium (*41*). Days required for colony formation under conditions identical to those of quantitative SIM experiments, including the addition of the same number of *lac*-deleted scavenger cells, are shown (Table S13).

DSB-dependent chromosomal stress-induced Tet frameshift-mutation assays

Description

The Tet mutation assay (*2*) measures reversion of a chromosomal *tet* +1-bp frameshift allele, placed 8.5 kb from an I-*Sce*I double-strand endonuclease cutsite (I-site A, *tet2*) engineered into plasmid-free cells with a chromosomal, regulatable I-*Sce*I gene (*2*). The cells are starved in liquid with no tetracycline (no selection for Tet function) then rescued from starvation and plated for tetracycline-resistant (Tet^R) mutant and total cfu and mutant frequencies quantified (*2*). The mutation pathway requires starvation stress, which induces the σ^S response, *or* artificial upregulation of the σ^S stress response in unstarved cells (*2*), and so is stress-induced mutagenesis. Mutagenesis also requires the SOS response, DinB/Pol IV error-prone DNA polymerase, and homologous recombinational DSB-repair enzymes including RecB which is specific for DSBs, and requires DSBs made either by I-*Sce*I, or spontaneously (*2*). These DNA breaks are repaired by homologous recombination (HR) with either an uncleaved sister chromosome [sisters are present in ~40% of starving *E. coli* (*42*)] or an uncleaved spontaneous tandem DNA duplication [duplications present in about 10^{-3} of cells, reviewed, (*7*)]. The protein requirements for σ^S and SOS responses, DSB repair proteins and DinB define this as a similar or the same DSB-dependent stress-induced mutagenesis mechanism operative in the Lac assay starvation conditions (*2*).

Rationale:
This assay addresses two concerns about mutagenesis studied in the Lac assay: that it might have been F'-plasmid dependent; and that it might have required selection for the gene undergoing mutagenesis and thus possible selection of growth of cells with multiple copies of that gene that then mutate at rates difficult to determine (per gene copy or base pair). This assay employs plasmid-free cells and the mutations accumulate dependently on time in starvation under no selection for *tet* gene function.

Procedure
Briefly, per (*2*), cells were streaked on M9 glucose vitamin B1 (B1) medium supplemented with proline (20 μg/ml) and incubated for ~22 hours at 37°C. Single colonies were inoculated into 5 ml of M9 glucose B1 proline broth and grown for 12 hours with shaking (200-250 rpm). These were diluted 1:100 into the same medium and grown 8-10 hours, diluted further by 1:100 and grown for 84 hours with shaking (200-250 rpm). Three-independent cultures per strain were used for each experiment. Mutant frequencies are the titers of cfu on LBH glucose tetracycline (Tet^R mutant cfu) divided by those from LBH glucose plates (total cfu), and mean ± SEM for ≥3 independent experiments are displayed (Fig 2, Table S5).

DSB-dependent chromosomal stress-induced Nal base-substitution forward-mutation assays

The Nal mutation assay measures chromosomal forward mutations in the *gyrA* gene that confer resistance to the topoisomerase inhibitor nalidixic acid [*e.g.*, (*2*)]. These are base-substitution mutations, predominantly C/G to T/A and A/T to G/C transitions with some G/C to T/A and A/T to C/G transversions also observed (*2, 43*). Assays were performed as for the Tet assay, but in cells with their I-*Sce*I site 273 bp downstream of *gyrA*, and with the final selection step on nalidixic acid-containing medium. Cells were starved in liquid with no nalidixic acid then rescued from starvation and plated for nalidixic acid-resistant (Nal^R) mutant and total cfu and mutant frequencies quantified (*2*). 24h-grown colonies from M9 glucose B1 proline plates were inoculated into 5 ml of M9 glucose B1 proline broth, grown for 12 hours with shaking (200-250 rpm), diluted 1:100 into the same medium and grown 8-10 hours, diluted further by 1:100 and grown for 84 hours with shaking (200-250 rpm). Three-independent cultures per strain were used for each experiment. The 84h grown cultures were plated on LBH glucose 30 μg/ml nalidixic acid solid medium to score Nal^R mutant colonies, and LBH glucose for total cfu. Mutant frequencies were determined by dividing titers of Nal^R cfu by the total cfu, and mean ± SEM for ≥3 independent experiments are displayed (Fig 2, Table S5).

Identification of network mutants deficient in activating the RpoS (σ^S) stress response: catalase and fluorescence assays

Catalase assay:
We first screened SIM-down mutants for those that fail to activate the σ^S response, using a simple colony assay for catalase activity in stationary phase (Table S7). σ^S

transcriptionally upregulates *katE*, encoding catalase, in stationary phase (*44*). Catalase metabolizes hydrogen peroxide (H_2O_2) (*45*) such that cells defective for σ^S activity are catalase defective (*44*). Colonies of the mutants were grown on M9 glycerol medium for 3 days at 37°C. 5 μl of H_2O_2 was dropped onto each colony, and the time elapsed before bubbles appeared was measured. SMR4562 (WT) and *rpoS* mutant (SMR6541) strains used as controls take 0 and 10-15 sec. to form bubbles, respectively. Mutants with >2 sec. delay in bubbling were scored as catalase (σ^S-activity) defective (Table S7).

Flow cytometric σ^S-dependent transcription assay:
We next quantified transcription from the σ^S-dependent *yiaG* promoter by flow cytometry of strains with a chromosomal *yiaG-yfp* fusion in our mutation-assay strain into which we moved each catalase-negative SIM-down mutation. Strains were cultured for 48h with aeration at 37°C in M9 B1 glycerol. A "yellow" gate was set as the window below which control *rpoS* mutant cells fall (vertical line in *rpoS* plot in Fig. S4A). Cells falling below the gate are inferred to be σ^S-activity deficient. $\geq 10^5$ cells per culture and ≥ 3 cultures per strain were analyzed per experiment. All catalase-deficient mutants were confirmed in this assay as σ^S-response deficient (Figs. 4A, S4A,B).

Generic transcription assay:
For all mutants that were σ^S-response deficient, to control for possible mutations that might decrease transcription generally, not specifically to the σ^S response, we measured transcription from the σ^S-independent IPTG-inducible P_{lac} promoter using a chromosomal P_{lac}*cfp* fusion gene and fluorescence (Figs. 4B,S4C). Cells were grown for 48h with aeration at 37°C in M9 B1 glycerol with or without 1 mM IPTG. 0.1 ml of 48h culture was used to determine the fluorescence intensity at 480 nm, and OD for cells was taken at 600 nm using a 386 well microplate (Greiner Bio One) and Multi-mode Microplate Reader (Model-Infinite M200 Pro, Tecan). None of the σ^S-response-deficient mutations caused reduced transcription of P_{lac}*cfp* (Figs. 4B, S4C), and thus all were confirmed as conferring specifically σ^S-response deficiency.

Growth rate control:
We established growth curves for all σ^S-response-deficient mutants to rule out the possibility that their failure to elicit the response could be caused by delayed entry into stationary phase, when the σ^S-response is induced. Overnight cultures in LB medium were diluted 1/100 with M9 medium (with 10 μg/ml thiamine and 0.1% glycerol) and 0.1 ml used in a 386 well microplate. The OD_{600} was read by a Multi-mode Microplate Reader (Model-Synergy 2, BioTek) at 30-min intervals for 48-h. Data presented (Figs. 4C,S5) are the means of ≥3 experiments of 3 cultures per strain.

Identification of network genes that function in activation of the SOS DNA-damage response: UV-sensitivity and flow-cytometric assays

We screened SIM-down mutants for those that fail to activate the SOS-response first by sensitivity to UV light. Mutants were grown overnight in liquid LB, diluted 1/10 with LB and then streaked on LB solid medium and exposed to UV at 50 and 60 joules/m^2.

recB (SMR593) and *recG* (SMR1982) mutant strains were used for strongly and weakly UV-sensitive controls, respectively. Mutants with the same or greater sensitivity as *recB* were scored as "strongly" UV-sensitive, and those more sensitive than *recG* but less than *recB,* and less than or equal to *recG* but more than WT, as "moderately" and "weakly" UV-sensitive, respectively.

Twenty-six of the SIM network mutant genes confer sensitivity to UV irradiation (Tables S1, S7). To determine which are defective in activating the SOS DNA-damage response, required for SIM, we determined the percentage of cells spontaneously SOS induced *via* GFP expression from the chromosomal SOS-dependent P_{sulA}*gfp-mut2* construct per (*21*), shown in Fig. 4K. Whereas normally just under 1% of cells are SOS induced due to spontaneous DNA damage (*21*), these mutants are deficient in activation of SOS and have fewer SOS-induced green cells. The SMR15962 (*pgi*), SMR15980 (*uvrY*), SMR16024 (*recB*), SMR17043 (*recC*), SMR6039 (WT; positive control) and SMR6178 [(*lexA3* (Ind^-); negative control] strains (Table S4) were used in Fig. 4K.

Identification of network genes that function in activation of the σ^E response: SDS-EDTA-sensitivity and β-galactosidase assays

To identify mutants defective in activating the RpoE (σ^E) membrane-protein stress response, SIM-down mutants were tested for SDS-EDTA sensitivity (Fig. S9A and Tables S1, S7) (*11*). 10^2-10^3 cells from LBH saturated cultures were spotted onto M9 B1 glycerol plates with and without 0.01% SDS and 0.25 mM EDTA, grown for 48h at 30°C, and compared with WT SMR4562 and SMR5236 cells carrying the stress-response defective (but essential-function-proficient) *rpoE2072*::Tn*10*dCam allele (*8*).

Mutant alleles from SDS-EDTA-sensitive mutants (Fig. S9A and Table S1, S7) were transduced into strain CAG45114, containing the σ^E-dependent *rpoH*P3-*lacZ* reporter (*20*) and tested for *lacZ* expression from this σ^E-response-dependent promoter per Table S11. β-galactosidase assays were performed as described (*29, 46*) except that saturated LBH cultures were used, rather than log-phase. We reasoned that the starvation induction of the σ^E response is more likely under our stationary-phase-stress experimental conditions, and so to reflect that, we measured σ^E activity in saturated stationary-phase cultures measured as expression of the σ^E-response-dependent *rpoH*P3-*lacZ* fusion gene (*20*) in cultures grown to saturation.

Statistical methods

Except where indicated otherwise, statistical analyses were performed using the Statplus software package. *P* values were determined by two-tailed Student's *t* test for strains compared in parallel in the same experiments.

Supplementary Text

Text S1. Identification of *crp* by candidate-gene approach

We identified *crp* as a gene in the stress-induced mutagenesis network by candidate-gene approach based on our having found its partners *barA* and *uvrY* (*47*) in our screen. Based on their identities, we constructed a *crp* null-mutant derivative of the Lac-assay strain (Table S4) and demonstrated its moderate 3.1-fold SIM defect (Table S3).

Text S2. Calculation of saturation of the screens

We found that out of 93 genes identified in screen 2, 80 mutant genes (10 of which overlapped with genes identified in screen 1, Fig. S1C) could be confirmed by quantitative SIM assay (Fig. S1C, Table S3). Screen 1 produced an additional 3 genes not found in screen 2. With identification of 80 genes from 187 mutants (from the second screen) + 3 additional new mutants obtained from the first screen, the screen is 94% saturated, calculated using Poisson distribution as follows.

Calculating saturation from screen 2:
Number of mutants isolated for SIM = 187
Number of genes identified = 80
Mean hits per gene (m) = number of mutants isolated for SIM/number of genes identified
= 187/80 = 2.3375
Poisson distribution $P_0 = e^{-m} = e^{-2.3375} = 0.0966$, *i.e.*, 90.34% saturation achieved in screen 2.
Total number of genes to be found: 80/0.9034 = 88.55.
Including screen 1 with screen 2, we found 83 of 88.55 = 93.8% saturation.

Estimations of SIM network genes not yet discovered. Note that although we have identified more than the predicted 89 genes in the SIM network (we have a total network of 93 genes, Fig. 1, Table S2) some of the genes identified previously by candidate gene approaches could not have been obtained in this screen for reasons outlined in Table S2. Therefore, our data and calculation imply that there are still SIM genes that remain to be discovered. The number remaining can be roughly projected in two ways.

First, the number of genes remaining that this screen could in principle detect can be estimated to be about 5, as follows. The number remaining could be 6 genes predicted (we found 83 of a predicted 89) – 1 gene from candidate gene approaches that could in principle have been found in our screen but was not: *recF* (see Table S2) = a predicted 5 genes that remain to be discovered.

An alternative projection leads to a possible ~20 genes not yet discovered, as follows. The fact that some known genes cannot be obtained in this screen (see Table S2) suggests that there could be other network genes that might not be detected for similar reasons. Of those undetectable, seven can be excluded from consideration: LexA, is not positively required for mutation—it represses the SOS regulon, so it should not be counted (Table S2 legend); Pols II and V play minor roles (see Table S2); the *ruvABC* genes produce an unusual phenotype (Table S2 legend) that we did screen for, so others

of this type are likely to have been detected; and DinB/PolIV though important is present in two copies (mero-diploid) in the strain, and so could not be obtained (Fig S1, legend). Because a very small fraction of the genome is mero-diploid, we can exclude *dinB* as non-representative. That leaves 2 of the 9 genes not obtained in the screen that could be representative of other important SIM genes that the screen might not detect (*nusA,* an essential gene for which only a temperature-sensitive mutant is known to be viable, and *recF*). Conceivably, if the screens missed 2 representative targets and found 7 (or 78%) among previously known genes, then the 83 found by the screens represent 78% of the total network. That leaves 23 genes (minus *crp*, which we added here by candidate gene approach, *nusA*, and *recF*) = a possible 20 network genes not yet identified. This number is based on very small numbers (2 or so and 7) and so could vary considerably in either direction.

Text S3. Estimated number of essential gene mutants not found in the screen

The essential genes that we identified in the SIM-down mutant screen are *cysD* (essential for growth in M9 medium), *hemL* (*33*), *groES* (*48*), *ligA* (*49*), *rpoE* (*11*), *ubiA* (*33*), *ubiD* (*33*) and *yigP* (*33*). We estimate that an additional two are likely to have been missed as follows.

Number of hits for essential gene mutants for SIM = 12
Number of essential genes identified = 8
Mean hits per gene (m) = number of essential gene mutants for SIM/number of essential genes identified

$$= 12/8 = 1.5$$

Poisson distribution $P_0 = e^{-m} = e^{-1.5} = 0.223$, *i.e.*, 78% saturation achieved
Total number of essential genes to be found: 8/0.78 = 10
Essential genes remaining to be found = number of essential genes to be found -number of essential genes found

$$= 10\text{-}8 = 2$$

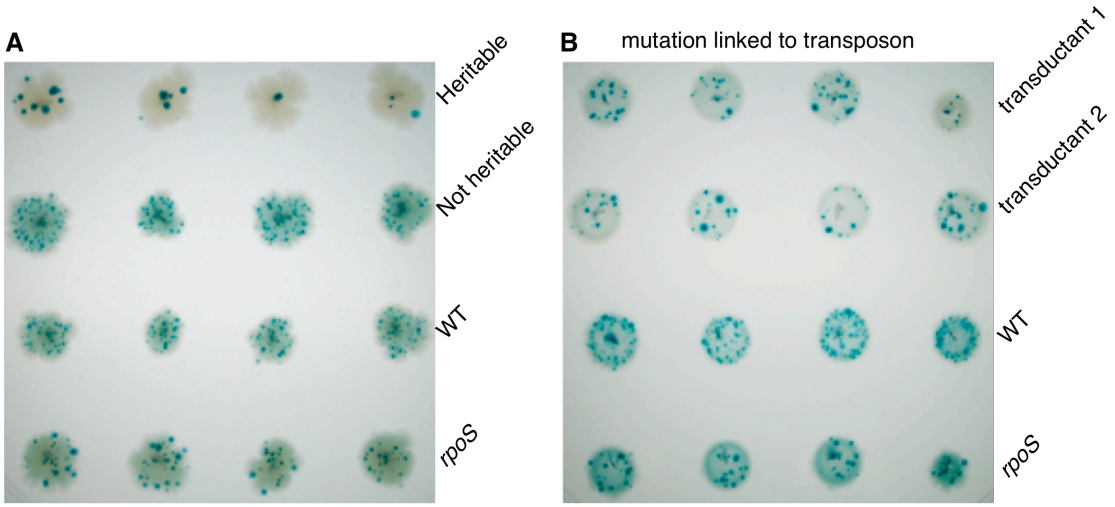
Text S4. Model for role of the electron transfer chain in activation of the σ^S response during starvation and stress-induced mutagenesis

In Fig. 4L, we suggest that during starvation some amount of the ETC product, ATP, is required for mutagenesis. Our data suggest that only specific energy sources malate and succinate are used to generate that ATP, during either an internal autophagy-like recycling process or external cannibalism at the brink of starvation. The ETC acts partly upstream of ArcBA/RssB (suppressor analyses), which control σ^S protein levels (*19*), and partly downstream (epistasis analyses) possibly by activating σ^S-controlled genes or other components of mutagenesis (Fig. 4L). The data show a previously unknown specific σ^S activation route during starvation, from consumption of malate and succinate, through a particular ETC branch to σ^S-response activation and mutagenesis (Fig. 4L). Activation of the σ^S response is a critical hub in the network, supported by a large allocation of

network genes (Fig. 3A, green) demonstrating its importance to mutagenesis. Generalization of the roles of the σ^S response (*7*) and supporting network proteins in the chromosomal Tet and Nal assays (Fig. 2, table S5) indicates that this large network segment and hub is generally important to *E. coli* mutagenesis.

Legend to Figure 4L Model: ETC-mediated stress-sensing from starvation to mutation.

Genes identified in screens, red; black and blue lines, biochemical transformations; gray lines, regulation. During nutrient availability reduced ubiquinones (UQH_2) allow autophosphorylation of ArcB sensor-kinase (*19*), which phosphorylates ArcA, which then transcriptionally represses *rpoS* (encoding σ^S) and its positive regulator (*19*) reducing σ^S and turning mutation "Off". Upon starvation, oxidized UQ prevents ArcB autophosphorylation, ending ArcA repression (*19*), thus allowing σ^S accumulation; so mutation turns "On". UQ inhibition of repression of σ^S is, we suggest, the component of ETC function suppressed by Δ*arcB* and Δ*arcA* **(D-F).** The component not suppressed could be reduction in levels of ATP (blue arrow). HemL, biosynthesis of hemes [prosthetic groups of SdhBD and CyoAD (*14*)]. HscB, biogenesis of Fe-S cluster, a cofactor. e^-, electron; Ⓟ, phosphate.



C

Screen	Screen 1	Screen 2
Primary screen:		
Number of Tn*10*dCam insertion mutants screened in colony-papillation screen	40,000	19,000
Number of candidate SIM-down mutants identified	427	1043
Secondary screens:		
Number that show SIM-down papillation phenotype repeatably / heritably	130	256
Number of chromosomal insertion mutations that show SIM-down papillation phenotype after transducing the Tn insertion into a new strain and re-testing	27	200 (187 that pass quantitative test below)
Number of genes shown by sequencing to have been mutated (many were identified in multiple independent isolates)	13	93
Number of genes verified in quantitative Lac mutagenesis assays (results Table S3)	13 (3 unique + 10 also found in screen 2)	80
Total genes indentified	83	
Number not known from previous work	76	

Fig. S1. Representative example of SIM-down mutant in papillation secondary screens and summary of results from screens

(A) Example of results from secondary screen for repeatability (heritability) of mutation-defective phenotype of mutants identified in the primary screen. Colonies with few Lac^+ mutant blue papillae in the primary screen 2 were streak-purified and re-tested, in four spots each, for a heritable reduced-papillation phenotype with positive, *rpoS* or *recA* mutant, and negative, WT, control strains in parallel. **(B)** Test for mutations linked with the transposon. Heritable mutations were moved by phage P1-mediated transduction to a clean, non-mutagenized Lac-assay strain and two independent transductants of each were retested alongside positive, *rpoS*, and negative, WT, control strains. Shown are two transductants of a single mutation that tested positive in screen 2. **(C)** Summary of results of screens 1 (*8*) and 2 and the secondary screens that identified *bona fide* SIM network genes. In screen 2, of 1043 candidates identified in the primary screen, 256

passed the heritability test (per A), and 200 passed the transduction test (per B) and were winnowed down to 187, representing 80 genes, by the quantitative test after sequencing.

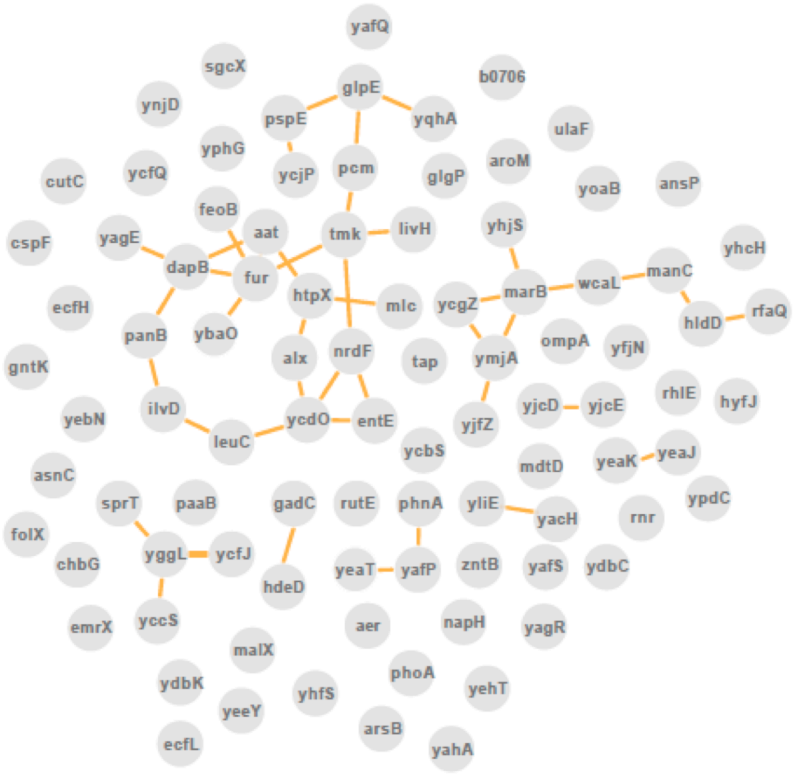


Fig. S2. A group of 93 randomly chosen genes from *E. coli* do not form a network of protein-protein interactions.

This control group was chosen by random selection from a total of 4233 protein-encoding genes in *E. coli.* All network links between those control genes were taken from STRING (Search Tool for Interacting Genes) 9.0 (*12*). The network layout was then computed, similarly to the SIM network shown in Fig. 3A, with Cytoscape 2.8.3 software using the "*unweighted force-directed layout*" algorithm.

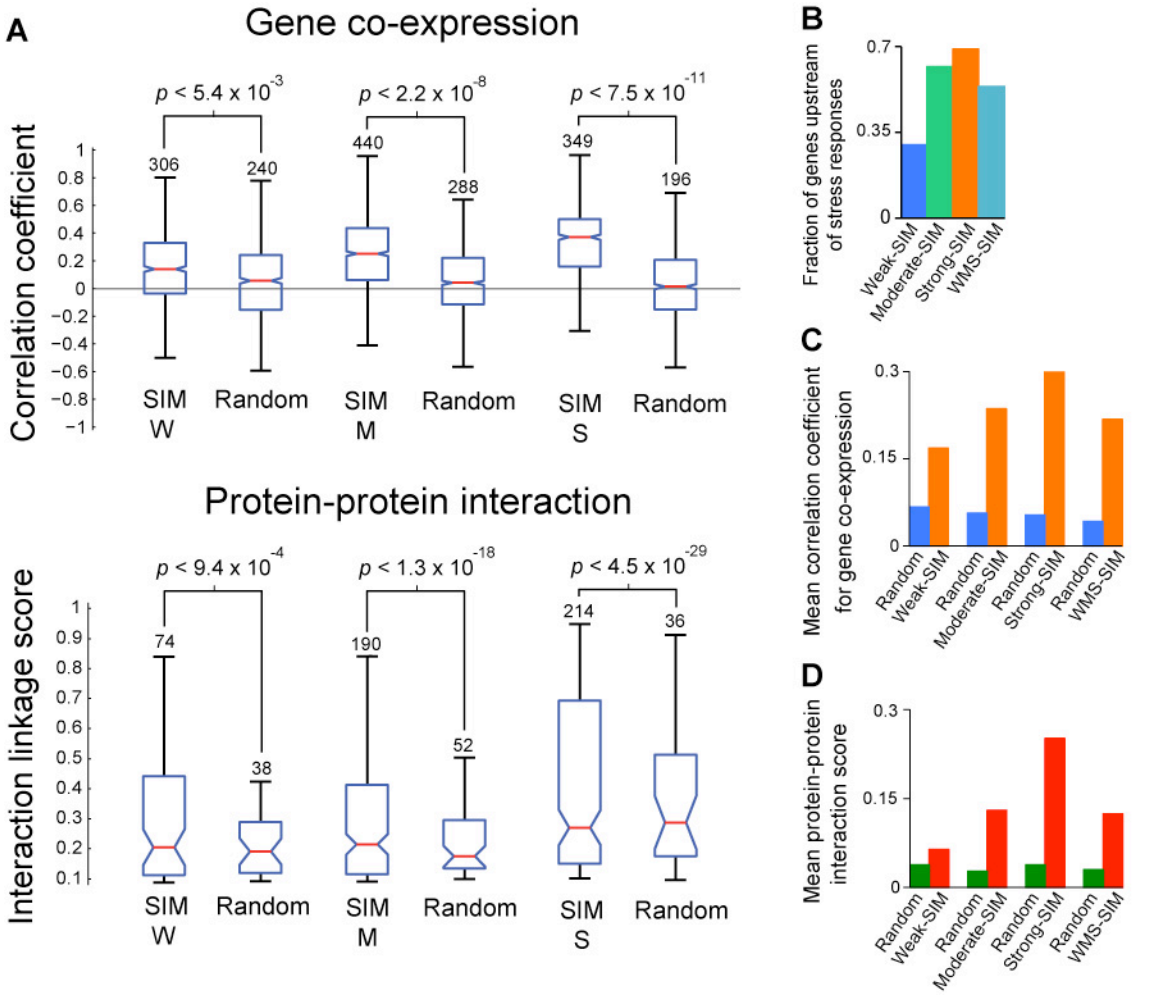


Fig. S3. Protein-protein interaction and gene co-expression analyses for network genes of strong, moderate and weak SIM defects and stress-response activators in each class

(A) The statistical significance for both gene co-expression and protein-protein interaction are in the following order: strong (S) > moderate (M) > weak (W) SIM defects defined as >9-fold, 3.1-9-fold, and 1.5-3-fold, respectively (Table S3). For gene co-expression (top panel), the number of pairs with positive co-expression Pearson correlation coefficients, for the groups weak, moderate and strong, were 306 pairs out of 435 [(30×29)/2], 440 pairs out of 561 [(34×33)/2] and 349 pairs out of 406 [(29×28)/2]. For protein-protein interaction (bottom panel), the number of pairs with positive interaction scores for the groups weak, moderate and strong were 74 pairs out of 435 [(30×29)/2], 190 pairs out of 561 [(34×33)/2] and 214 pairs out of 406 [(29×28)/2] that have positive protein-protein interaction linkage scores. Co-expression correlation coefficients were determined from 907 genome-wide microarray experiments over 4297 genes deposited in the Many Microbe Microarrays Database (*13*), and protein-protein interaction linkage scores were taken from STRING 9.0 database (*12*) resulting in 439486 interaction pairs. Random controls were produced by selecting a random group of equal size among all *E. coli* genes. *P* values were calculated with a sign-test giving the probability of the null-hypothesis "*number of tested positive pairs equals the random*

control number of positive pairs". **(B)** Genes upstream of the σ^S, σ^E and SOS stress responses are a greater fraction of strong than moderate, and moderate than weak SIM genes. The number of mutants with functions upstream of the three stress responses for the weak, moderate and strong groups are 9, 21 and 20, respectively (Tables S1, S3). The fraction of the upstream mutants in each group was determined by dividing the number of mutants upstream of stress responses for each groups with total number SIM mutants for the respective groups, and plotted. WMS, all SIM genes for weak, moderate and strong groups. **(C and D)** Both co-expression and protein-protein interaction increase with increasing strength of the effect of the mutations in SIM. The mean co-expression coefficient **(C)** and mean interaction linkage score **(D)** for the weak (30 genes), moderate (34 genes) and strong (29 genes) groups are plotted. For each of the groups, the values for random pairs were used as controls.

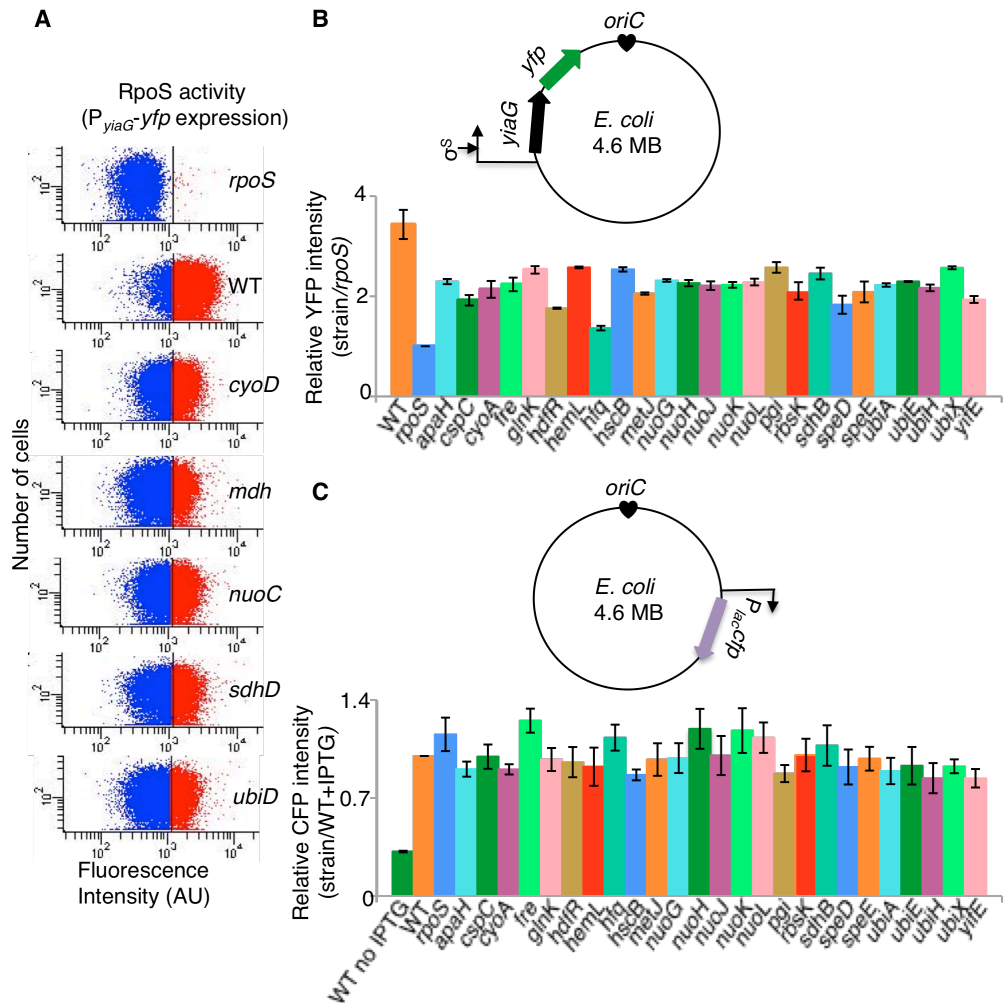


Fig. S4. SIM network genes that function in activation of the σ^S stress response

Thirty-one SIM-down mutations (here and Fig. 4A) were identified as catalase- (σ^S-activity-) defective and show reduced transcription from the σ^S-dependent P_{yiaG} promoter but not the σ^S-independent P_{lac} promoter (Methods, assays for σ^S-response-deficiency). **(A)** Reduced transcription from *yiaG-yfp*, representative example of flow cytometric data. Representative dot plots of fluorescence of five electron-transfer SIM-down mutants showing reduced σ^S activity at *yiaG-yfp* compared with the wild-type control. Data points indicate flow-cytometry events (cells) and are colored red to the yellow side of the yellow gate and blue on the non-yellow side of the yellow gate (gate set to define *rpoS* null mutant cells as "non-yellow", Methods). Strains *cyoD* (SMR13035), *mdh* (SMR13058), *nuoC* (SMR12655), *sdhD* (SMR13033) and *ubiD* (SMR13098), WT (SMR10582) and *rpoS* (SMR12661). **(B)** Quantification from multiple experiments of reduced σ^S-dependent transcription in catalase-defective SIM-down mutants. Data are the means ± SEM (≥ 3 experiments each) of the ratio of the mean-fluorescence intensity of each mutant divided by the mean-fluorescence intensity of Δ*rpoS* control cells assayed in parallel. Strains are those in (A) and *apaH* (SMR12830), *cspC* (SMR14263), *cyoA* (SMR14283), *fre* (SMR14267), *glnK* (SMR14281), *hdfR* (SMR14261), *hemL* (SMR14431), *hfq* (SMR14295), *hscB* (SMR14275), *metJ* (SMR14269), *nuoG*

(SMR14420), *nuoH* (SMR14287), *nuoJ* (SMR14289), *nuoK* (SMR14285), *nuoL* (SMR14273), *pgi* (SMR13295), *rbsK* (SMR13312), *sdhB* (SMR14297), *speD* (SMR12828), *speE* (SMR13310), *ubiA* (SMR14447), *ubiE* (SMR12657), *ubiH* (SMR12686), *ubiX* (SMR12699) and *yifE* (SMR14259). All are significantly lower than WT (*P* values, and all numerical data and *P* values, Table S8). **(C)** The σ^S-activity-deficient mutants are not generally transcription-deficient as seen by their robust transcription of the σ^S-independent, IPTG-inducible *lac* promoter. Cultures were grown with and without IPTG and fluorescence determined by plate reader. Values are for the IPTG-induced transcription (both IPTG-induced and uninduced shown for WT). As for the WT, CFP expression was also 2.2-4.8-fold induced in the presence of IPTG compared with no IPTG for the mutants. Fluorescence intensity for cells grown in the presence of IPTG was normalized to their OD_{600} and this value normalized to that of WT + IPTG fluorescence/WT OD_{600} and plotted. Mean ± SEM for 3 experiments of 3 cultures per strain. The strains used for CFP expression analysis in Fig. 4B and in panel C of this Fig. are listed in Table S4 (SMR14471 to SMR15109).

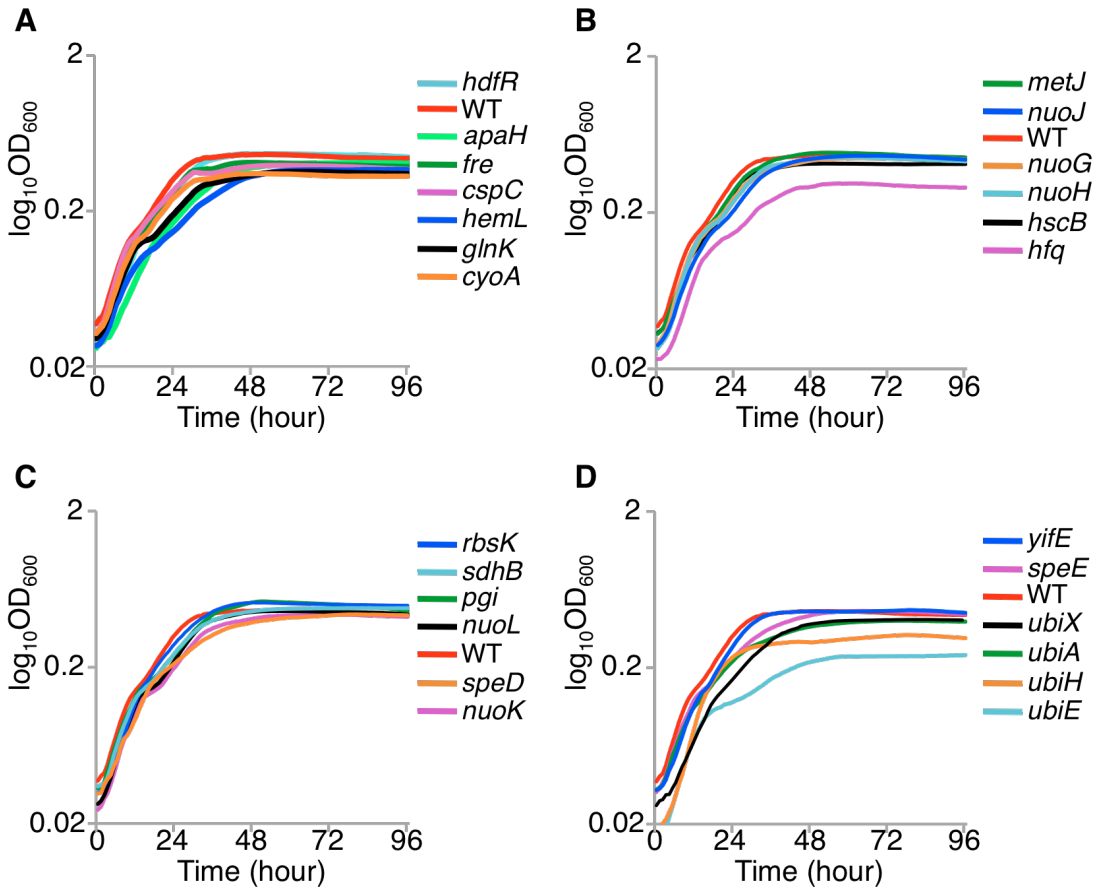


Fig. S5. Stationary phase is not delayed in SIM network mutants defective in σ^S activity, indicating σ^S-response defects

Mutants defective in activation of σ^S-controlled genes could be defective in inducing the σ^S response, or might in principle be slow growers that are delayed in entry into stationary phase, when the σ^S response is induced. The growth curves of the mutants show that none has delayed entry into stationary phase. Therefore, these mutants are σ^S-response defective, not defective in entering stationary phase in which the σ^S response is induced. Means of $\geq$ 3 experiments of three cultures per strain. Strains: WT (SMR10582) **(A)** *apaH* (SMR12830), *cspC* (SMR14263), *cyoA* (SMR14283), *fre* (SMR14267), *glnK* (SMR14281), *hdfR* (SMR14261) and *hemL* (SMR14431); **(B)** *hfq* (SMR14295) *hscB* (SMR14275), *metJ* (SMR14269), *nuoG* (SMR14420), *nuoH* (SMR14287) and *nuoJ* (SMR14289); **(C)** *nuoK* (SMR14285), *nuoL* (SMR14273), *pgi* (SMR13295), *rbsK* (SMR13312), *sdhB* (SMR14297) and *speD* (SMR12828); **(D)** *speE* (SMR13310), *ubiA* (SMR14447), *ubiE* (SMR12657), *ubiH* (SMR12686), *ubiX* (SMR12699) and *yifE* (SMR14259).

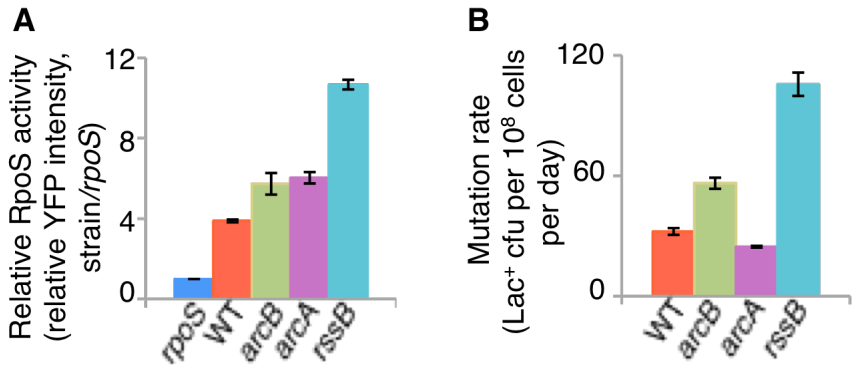


Fig. S6. Upregulating σ^S by *arcB* and *rssB* mutations is correlated with increased stress-induced mutagenesis

(A) Increase in σ^S activity for *arcB* (SMR12673), *arcA* (SMR12672) and *rssB* (SMR12566) mutants measured with the *yiaG-yfp* fusion per Fig. S4B and Methods. **(B)** Increased SIM in *arcB* (SMR12684) and *rssB* (SMR12692) mutants. Tables S8 and S9 show values for the increased σ^S activity and mutation rates for *arcB*, *arcA* and *rssB* mutants compared with controls.

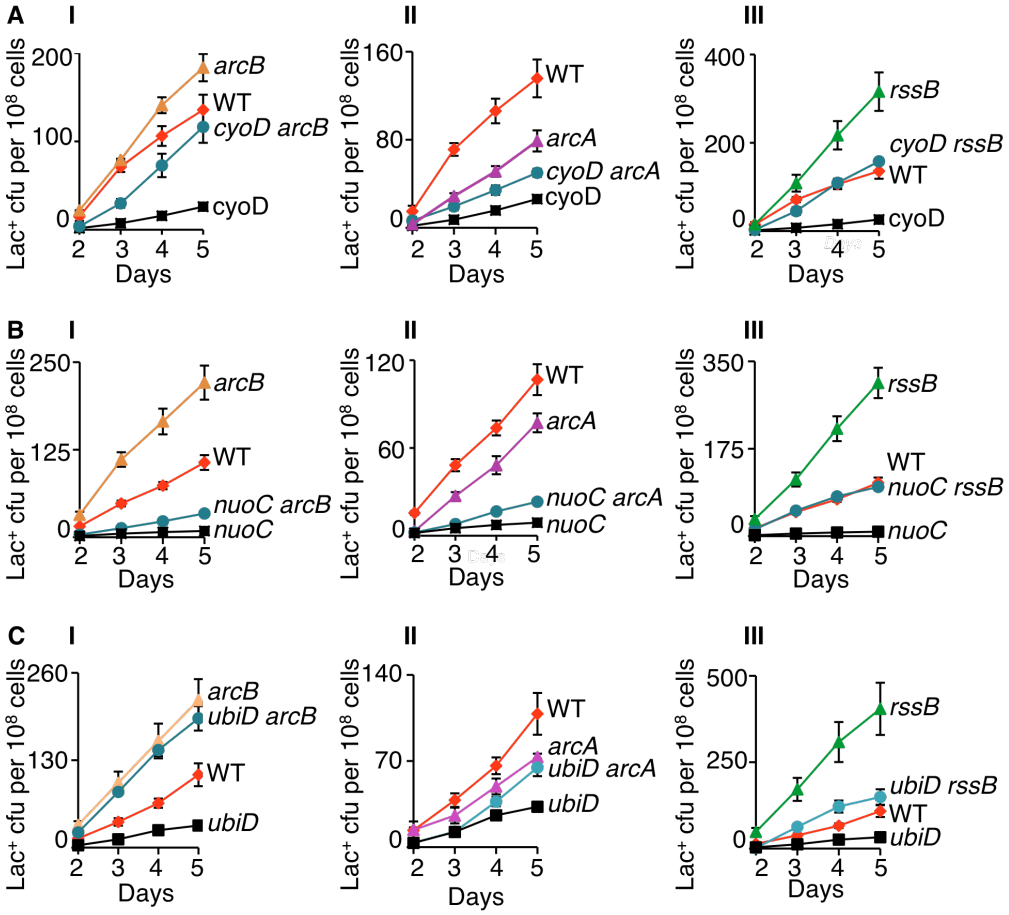


Fig. S7. σ^S-expression-activating *arcB*, *arcA*, and *rssB* mutations restore or partially restore mutagenesis to electron-transfer mutants

Representative examples of quantitative SIM assays showing restoration of SIM in *cyoD* **(A)**, *nuoC* **(B),** and *ubiD* **(C)** electron-transfer mutants by *arcB* **(I)**, *arcA* **(II)** or *rssB* **(III)** mutation. Quantification from multiple experiments shown in Fig. 4D-F and Table S9. Mutagenesis is restored significantly ($P \leq 0.05$) by introduction of *arcB*, *arcA* or *rssB* mutation in the electron-transfer mutants c*yoD* **(A-I-III)**, *nuoC* **(B-I-III)** and *ubiD* (**C-I-III**) (Table S9). For proteins for which restoration is complete or nearly so, such as UbiD suppressed by *arcB* or *arcA* mutations, we infer that the primary role of the protein (UbiD) in mutagenesis is upstream of ArcBA-inhibited transcriptional activation of *rpoS*. For proteins for which restoration is partial, NuoC and CyoD, these proteins may promote mutagenesis partially *via* their upregulation of σ^S, and partially *via* other mechanism(s). We note that *rssB* mutation could not be expected to suppress any of these mutations completely because RssB acts post-translationally, to target σ^S protein for degradation (*50, 51*), whereas the electron transfer proteins are likely to promote *rpoS* gene transcription and translation (Fig. 4L). That is, failure to degrade σ^S protein in the *rssB* mutant cannot fully compensate for mutations in which there is insufficient synthesis and translation of *rpoS* mRNA and thus σ^S protein.

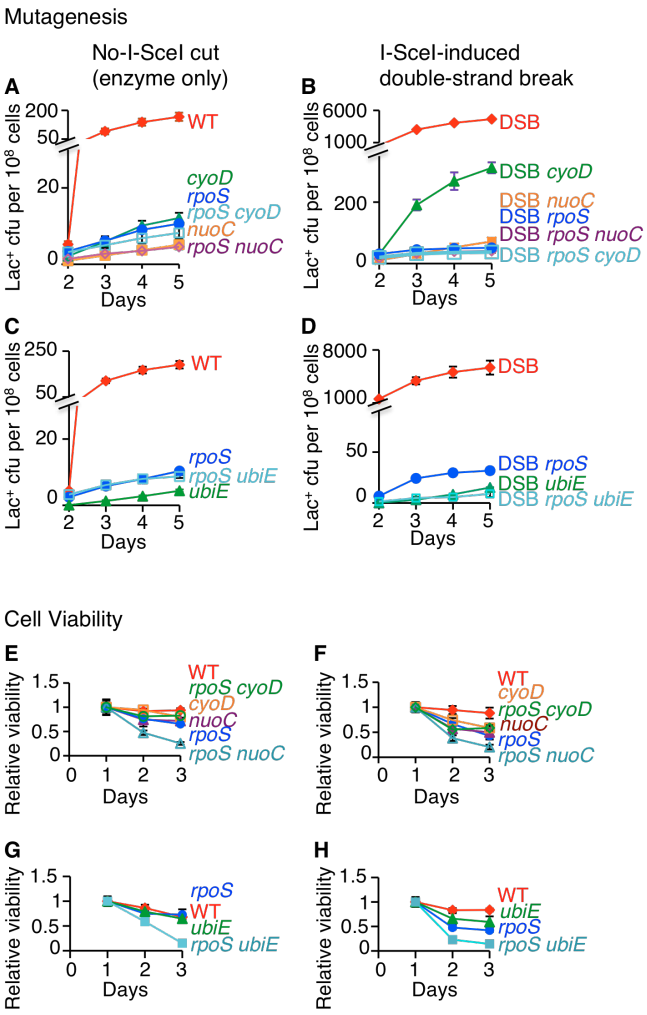


Fig. S8. *rpoS* is epistatic to (acts in the same pathway as) *cyoD*, *nuoC*, and *ubiE* electron-transfer mutations in DSB-dependent SIM

Representative examples. Fig. 4G-I and Table S10 for quantification of multiple experiments with many electron transfer mutants. **(A, C)** There is no greater mutagenesis defect in the each of the following double mutants than the *rpoS* single mutant control indicating that RpoS is epistatic to (acts in the same pathway as) these genes, in the standard Lac SIM assay ("enzyme-only") (**A**) *rpoS cyoD* and *rpoS nuoC* mutants; **(C)** *rpoS ubiE*. **(B, D)** Using a sensitized, Lac SIM assay with mutagenesis enhanced by I-*Sce*I-endonuclease cleavage near *lac* per (*35*), for maximal detection of any possible additive defects in SIM of the double mutants compared with the *rpoS* single mutants, there is still no greater mutagenesis defect in the following double mutants than the *rpoS* single-mutant control indicating that RpoS is epistatic to (acts in the same pathway as) these genes (**B**) *rpoS cyoD* and *rpoS nuoC* mutants; **(D)** *rpoS ubiE*. **(E-H)** Relative viabilities of the *lac*$^-$ cells during SIM [per (*34*)]. Only the *nuoC rpoS* and *ubiE rpoS* mutants without or with I-*Sce*I-induced DSBs show slightly decreased viability compared with the *rpoS* single mutant alone by about 2-fold. For these strains, the Lac$^+$ cfu shown are normalized for Fig. 4, part of this Fig. and Table S10 for viability loss as described (*1*).

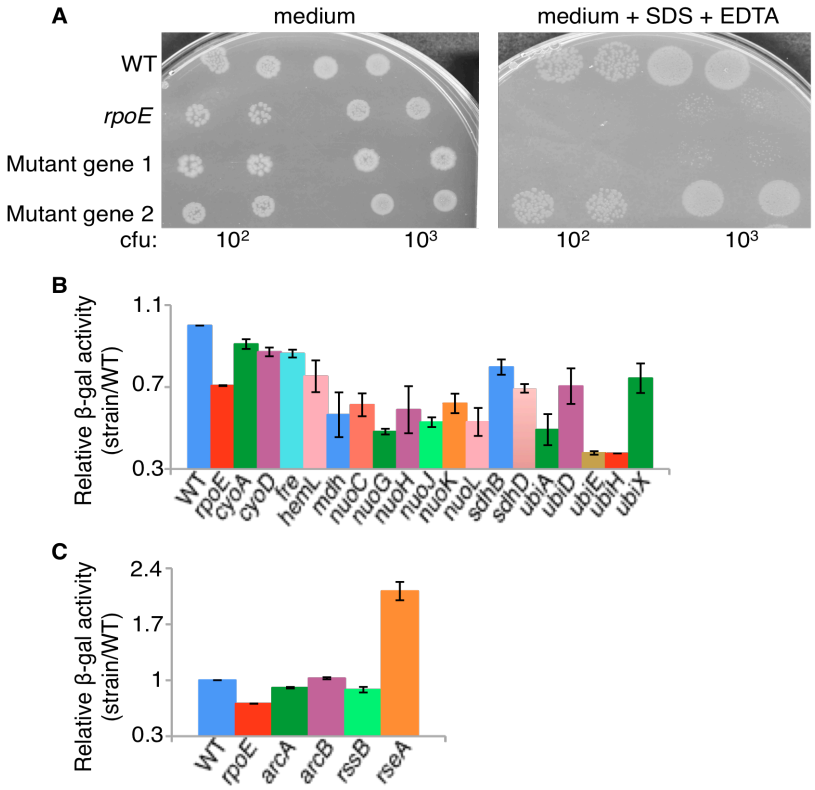


Fig. S9. Identification SIM network mutants deficient in activation of the σ^E response

(A) SDS-EDTA-sensitivity test for candidate σ^E-response-deficient mutants (Methods). The *rpoE2072*::Tn*10*dCam (*8*) positive control and one of the network mutants fail to grow on medium with SDS and EDTA (right) but grow on the same medium without SDS-EDTA (left). Of 93 SIM-down mutants, we find that 44 were sensitive to SDS-EDTA (Table S7) indicating membrane stress, and potential σ^E-response deficiency (*20*). Of those, 33 also showed reduced transcription from the σ^E-dependent *rpoH*P3 promoter, confirming their roles in σ^E-response induction (B, and Tables S7, S11, Fig. 4J). However failure to activate this specific promoter under the particular assay conditions used does not rule out a role in σ^E-response induction during mutagenesis. **(B)** Confirmation of σ^E response induction-deficiency of the SDS-EDTA-sensitive mutants by β-galactosidase assay for σ^E-induced transcription from *rpoH*P3-*lacZ*. Per (*20*) β-galactosidase activity was assayed using the reporter strain CAG45114 [MG1655(λ *rpoH*P3-*lacZ*)] (Methods, Table S11). Electron-transfer mutants for *cyoA* (SMR15330), *cyoD* (SMR15293), *fre* (SMR15362), *hemL* (SMR15319), *mdh* (SMR15356), *nuoC* (SMR15277), *nuoG* (SMR16038), *nuoH* (SMR15249), *nuoJ* (SMR15358), *nuoK* (SMR15366), *nuoL* (SMR15354), *sdhB* (SMR15261), *sdhD* (SMR15167), *ubiA* (SMR15321), *ubiD* (SMR15323), *ubiE* (SMR15257), *ubiH* (SMR15295) and *ubiX* (SMR15317) show significantly reduced σ^E activity at *rpoH*P3, as do mutants for 15 other SIM network genes (*P* values Table S11, Fig 4J, remaining mutants tested). **(C)** *arcB* (SMR12673), *arcA* (SMR12672), or *rssB* (SMR12566) mutations, which cause upregulation of σ^S (*50, 51*) and suppress the σ^S response-defects of electron-transfer mutants (Fig. 4D-F, Table S9), do not affect σ^E activity. Therefore, the electron-transfer

proteins activate the σ^E response independently of their effects on the *arcB*, *arcA* and *rssB* regulatory systems**.** WT (CAG45114), *rpoE* (SMR15311) and *rseA* (SMR15754, σ^E-response constitutive). Table S11 for numerical values and detailed description of assay. The *rpoH*-P3-*lacZ* reporter strains used in Fig. 4J follow: *arcA* (SMR15360), *apaH* (SMR15328), *fruR* (SMR15253), *lipB* (SMR15259), *lon* (SMR15273), *lrp* (SMR15265), *pstB* (SMR15301), *pstC* (SMR15287), *rppH* (SMR15315), *trxB* (SMR15283), *ygfA* (SMR15326), *yigP* (SMR15309), *znuC* (SMR15291), and *rpoE* (SMR15311) (Table S4).

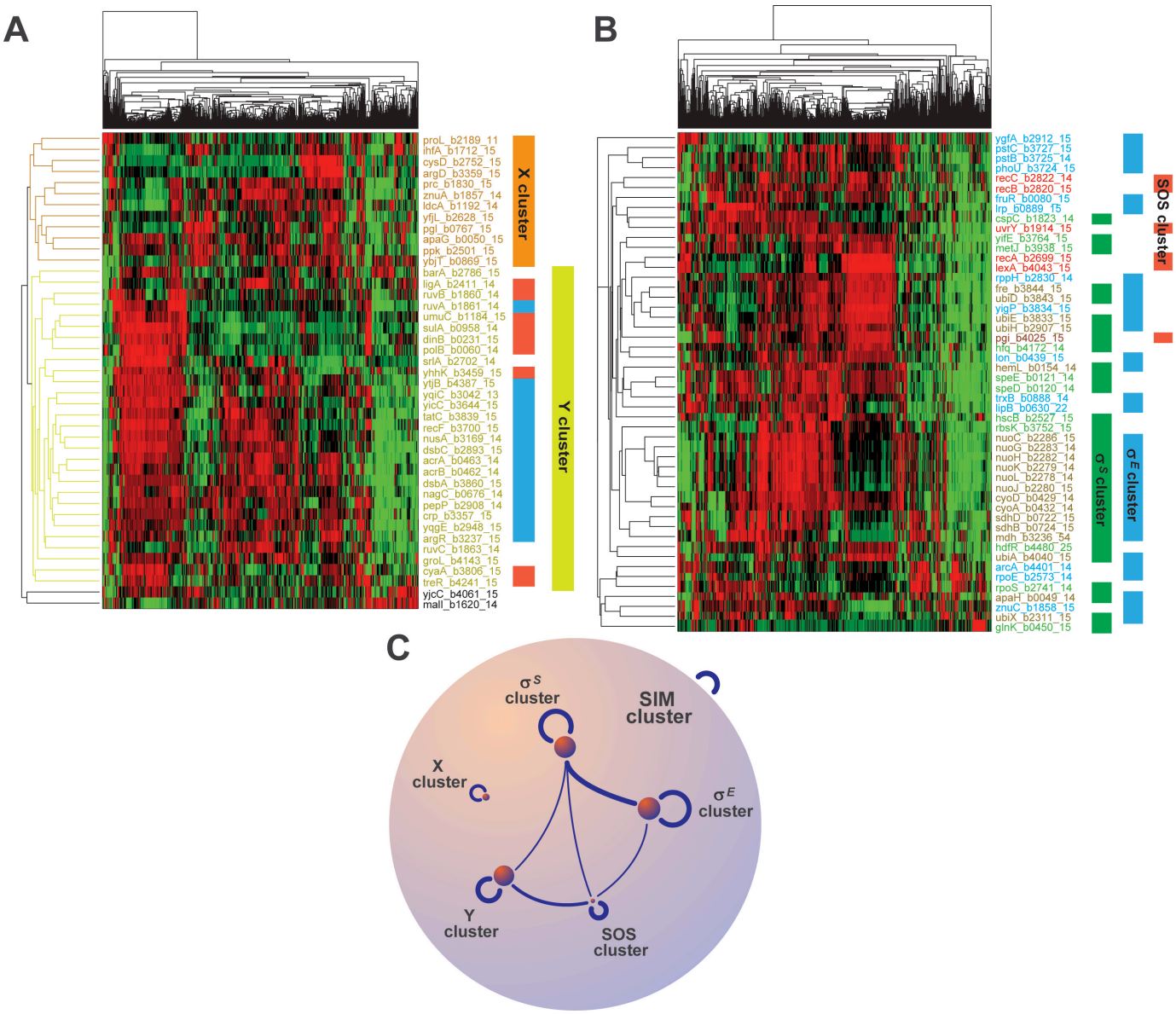
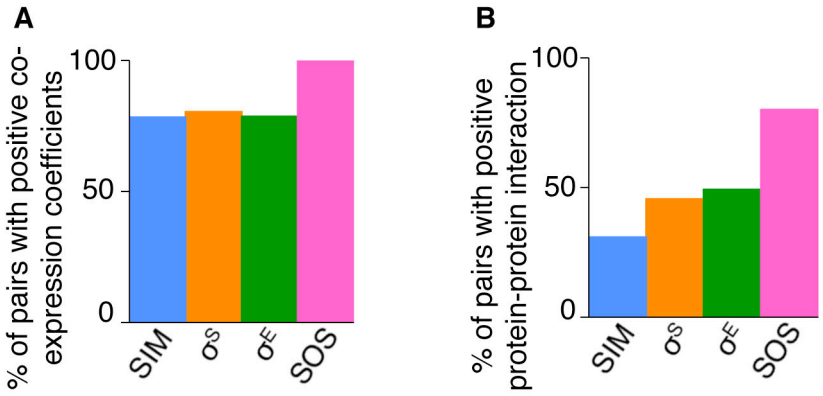


Fig. S10. Cluster analysis with large-scale microarray data idenifies additional sub-networks

(A) Among 43 of the total 93 SIM genes that were not classified by functional studies as activators of the σ^E, σ^S, or SOS pathways, indicated in blue, green, and red, respectively, hierarchical clustering of gene expression data from the Many Microbial Microarrays Databank (*13*) idenitfied two additional clusters (cluster X and cluster Y). **(B)** The σ^E, σ^S, or SOS clusters shown respectively, in blue, green, and red. **(C)** Calculating average gene expression between clusters resulted in a cluster coexpression network. In this network, statistically significant coexpression levels are indicated by blue lines with their thickness proportional to the integer number of standard deviations from a level of coexpression expected by chance; the radius of the network nodes is proportional to the number of genes within that cluster. This analysis suggests functional divergence of

cluster X, which does not yield any significant coexpression with any of the others; in contrast, cluster Y is significantly coexpressed with both the SOS (red in panel A) and with the σ^S cluster (blue in panel A), thus suggesting some functional similarity. Cluster analysis was performed with Matlab computer software (Mathworks Inc., Natick, MA, USA) version 7.13.0.64 (R2011B) by using the clustergram command in the Matlab Bioinformatics Toolbox. Non-parametric statistical significance of a network link was established by standardizing through mean and standard deviation from a sampling of one million random clusters.



C

Analysis	Gene class	Total number of gene pairs analyzed	Number of gene pairs with positive co-expression coefficient or PPI score	% of pairs with positive co-expression coefficient or PPI score
Co-expression	SIM	93 × 92/2=4278	3350	78
	σ^S	31 × 30/2=465	374	80
	σ^E	33 × 32/2=528	415	79
	SOS	6 × 5/2=15	15	100
Protein-protein interaction (PPI)	SIM	93 × 92/2=4278	1320	31
	σ^S	31 × 30/2=465	212	46
	σ^E	33 × 32/2=528	260	49
	SOS	6 × 5/2=15	12	80

Fig. S11. Positive gene co-expression coefficients and protein-protein interaction scores for the mutant genes upstream of σ^S, σ^E and SOS stress responses.

Gene co-expression coefficients and protein-protein interaction (PPI) scores were determined for all SIM mutants and for the genes upstream of each of the three stress responses as described in Fig. S3. The percents of the pairs of genes compared for upstream of σ^S, σ^E or SOS responses that have positive correlation coefficients **(A)** or protein-protein interaction scores **(B)** were determined as follows: (number of gene pairs with positive co-expression coefficient or protein-protein interaction score/total number of gene pairs analyzed) × 100. **(C)** The same data in tabular form show the number of pairs with positive correlation out of the total 4278 pairs compared.

Table S1. Summary of stress-induced-mutagenesis network genetic analyses.

Four mutation assays. Fold-defects of the SIM network mutants are relative to the mutation rates in the wild-type strain. *P* values differ significantly from the WT (≤0.05) in all four mutation assays except for nine mutants the names of which are annotated with[13]. This confirms 43 of 52 tested in the Tet assay, and 41 of 50 tested in the Nal mutation assay. Of the nine not confirmed, all but two, *ldcA* and *yqiC*, are SDS-EDTA sensitive, indicating membrane stress and possible membrane-stress (σ^E)-response defects. The roles of these proteins in SIM might be *via* activating the σ^E response, which promotes mutagenesis by promoting spontaneous DNA breaks, and so is superfluous when DSBs are provided by I-*Sce*I (*8*), as in the Tet (*2*) and Nal assays (Methods). Of the two remaining, *yqiC* encodes a putative membrane protein (Table S2) and *ldcA* encodes L,D-carboxypeptidase which has an essential function in murein (peptidoglycan) recycling (*52*), and so might also be defective in inducing the σ^E-membrane-protein stress response under the conditions of our SIM assay. NT, not tested; Q, tested in qualitative papillation assay only.

Summary of stress-response-activation pathways data. See the tables and figures cited below for descriptions of the quantitative data summarized qualitatively here. Suppressor and epistasis analyses were conducted on one mutant of each of the following protein machines or pathways, and the data for the gene tested are given: *cyoD* tested for CyoAD; *nuoC* tested for NuoC, NuoG, NuoH, NuoJ, NuoK, and NuoL components of NADH oxidoreductase; *ubiD* or *ubiE* tested for UbiA, UbiD, UbiE, UbiH, and UbiX components of ubiquinone biosynthesis; *sdhD* for SdhBD. The genes not tested in each protein machine or pathway group are expected to have similar phenotypes, which we represent by showing for each the data for the gene tested marked with *.

Mutant gene	**Papillation Assay:** Mean fold decrease in number of papillae (WT/mutant) ± SEM [1]	**Quantitative Lac Assay:** Mean fold decrease mutation rate (WT/mutant) ± SEM [2]	**Tet Assay:** Mean fold decrease in Tet^R mutant frequency (WT/mutant) ± SEM [3]	**Nal Assay:** Mean fold decrease in Nal^R mutant frequency (WT/mutant) ± SEM [4]	σ^S upstream [5]	Suppressor tests, % mutation restored by *arcB, arcA, rssB* respectively [6]	Epistatic w. *rpoS* [7]	σ^E upstream probable (SDS/EDTA^S) [8]	σ^E upstream confirmed [9]	UV sensitivity [10]	SOS upstream [11]
srlA	1.6 ± 0.2	1.5 ± 0.04	NT	NT	-			-		R	
ybjT	1.8 ± 0.2	1.5 ± 0.2	NT	3.2 ± 0.6	-			-		R	
treR	1.9 ± 0.1	1.5 ± 0.1	NT	2.8 ± 1	-			-		R	
yjcC	3.1 ± 0.3	1.5 ± 0.06	22 ± 3	3.4 ± 1.1	-			-		S	-
yicC	3 ± 0.1	1.5 ± 0.1	2.6 ± 0.5	3.7 ± 1.4	-			-		R	
ytjB	3 ± 0.1	1.6 ± 0.1	16 ± 8	7.9 ± 3.3	-			-		R	
arcA	2.9 ± 0.1	1.7 ± 0.1	30 ± 17	2.4 ± 0.6	-			+	+	R	
ligA	1.7 ± 0.1	1.7 ± 0.07	NT	NT	-			-		S	-
yqgE	2.2 ± 0.3	1.7 ± 0.06	8 ± 2	5 ± 1.8	-			-		S	-
argD	2.2 ± 0.1	1.8 ± 0.2	NT	NT	-			-		R	
apaG	2.1 ± 0.01	1.8 ± 0.1	NT	NT	-			-		R	
yfjL	3 ± 0.2	1.8 ± 0.05	NT	5.2 ± 1.5	-			-		R	
acrB	3.6 ± 0.7	1.8 ± 0.1	13 ± 3	11.8 ± 6.6	-			+	-	R	
malI	2.4 ± 0.3	2.0 ± 0.2	6.3 ± 3.7	4.5 ± 1.7	-			-		R	
lrp	2.1 ± 0.2	2.0 ± 0.4	2.6 ± 0.8	2.9 ± 1	-			+	+	R	
acrA	3.2 ± 0.6	2.3 ± 0.8	7 ± 3	13 ± 4.2	-			+	-	R	
uvrY	1.9 ± 0.1	2.3 ± 0.5	NT	NT	-			-		S	+
trxB[13]	3.5 ± 0.1	2.5 ± 0.3	1.3 ± 0.3	1.4 ± 0.4	-			+	+	R	

fre	11 ± 0.7	2.5 ± 0.2	8 ± 2	10.5 ± 2.3	+		+	+	+	R	
cspC	2 ± 0.05	2.6 ± 0.2	5.7 ± 0.6	7.7 ± 2.3	+			-		R	
barA	3 ± 0.4	2.6 ± 0.7	5 ± 1	6.2 ± 2.3	-			-		R	
prc	1.7 ± 0.04	2.7 ± 0.5	NT	3.4 ± 0.9	-			-		R	
pepP	4.5 ± 0.3	2.7 ± 0.2	NT	NT	-			-		R	
pstB	7.2 ± 1.6	2.8 ± 0.9	2.4 ± 0.3	3 ± 1	-			+	+	S	-
ubiX	2.3 ± 0.2	2.8 ± 0.4	NT	NT	+	*86, 87, 51	*+	+	+	R	
znuC	1.7 ± 0.08	2.9 ± 0.6	5 ± 1	4.7 ± 1.8	-			+	+	R	
crp	NT	3.1± 0.3	NT	NT	-			-		R	
ubiD	3.5 ± 0.3	3.1 ± 0.6	NT	NT	+	86, 87, 51	*+	+	+	R	
yhhK	9.8 ± 3.2	3.2 ± 0.3	NT	NT	-			-		R	
metJ	2 ± 0.2	3.3 ± 0.6	2.4 ± 0.7	2.2 ± 0.2	+			-		R	
phoU	5.6 ± 1.1	3.5 ± 0.2	2.8 ± 0.7	NT	-			+	+	R	
lipB	7.7 ± 0.4	3.7 ± 0.2	4 ± 2	NT	-			+	+	R	
proL	2.4 ± 0.07	3.8 ± 0.2	5 ± 1	4.2 ± 1.2	-			-		R	
speD	3.6 ± 0.1	3.9 ± 0.5	12 ± 6	3.5 ± 0.9	+			-		R	
cysD[13]	3.3 ± 0.2	3.9 ± 1.9	1.9 ± 0.2	2.6 ± 1.1	-			+	-	R	
sdhD	3.1 ± 0.2	4.2 ± 1.6	NT	NT	+		+	+	+	R	
speE	3 ± 0.2	4.2 ± 0.5	12 ± 2	3.8 ± 1	+			+	-	R	
fruR[13]	3.4 ± 0.9	4.2 ± 0.5	0.7 ± 0.1	1.6 ± 0.3	-			+	+	R	
argR	3.1 ± 0.6	4.3 ± 0.3	3.8 ± 0.8	5.1 ± 1	-			-		R	
pgl	2.3 ± 0.3	4.3 ± 0.5	NT	NT	-			-		S	-
znuA	1.7 ± 0.1	4.6 ± 1.4	4.1 ± 0.8	NT	-			+	-	R	
pstC	6 ± 1.2	4.7 ± 0.2	2.4 ± 0.4	NT	-			+	+	R	
dsbC	5 ± 0.1	5 ± 0.7	NT	NT	-			-		R	
cyoD	3.9 ± 0.2	5.3 ± 1.8	NT	NT	+	56, 56, 39	+	+	+	R	
hscB	4.3 ± 0.3	5.2 ± 0.5	7 ± 1	2.6 ± 1.1	+		+	-		S	-
cyoA	4.2 ± 0.3	5.5 ± 0.5	2.5 ± 0.8	4.6 ± 1.2	+	*56, 56, 39		+	+	R	
apaH	8.4 ± 1.1	5.6 ± 1.1	2.8 ± 0.6	2.2 ± 0.3	+			+	+	R	
yqiC[13]	3.3 ± 0.4	5.7 ± 0.7	0.8 ± 0.2	1.4 ± 0.2	-			-		R	
sdhB	3 ± 0.3	5.7 ± 0.6	1.8 ± 0.3	3.9 ± 0.8	+		*+	+	+	R	
glnK	2.2 ± 0.4	6 ± 3	7 ± 3	3.9 ± 1.7	+			-		R	
tatC[13]	2.3 ± 0.2	6.5 ± 2.2	0.5 ± 0.1	2 ± 1	-			+	-	R	
cyaA	26 ± 7	7.3 ± 0.6	NT	NT	-			-		R	
mdh	4.3 ± 0.3	7.6 ± 1.7	64 ± 16	8 ± 4	+		+	+	+	R	
yigP	11 ± 2.5	7.6 ± 1.3	4 ± 2	4.5 ± 0.9	-			+	+	R	
ygfA	4.6 ± 0.03	7.7 ± 2.1	9 ± 3	5.4 ± 2.1	-			+	+	R	
pgi	8.6 ± 1.8	8.1 ± 1.1	2.6 ± 0.6	2.6 ± 0.7	+		+	-		S	+
nuoK	3.9 ± 0.5	8.7 ± 0.2	NT	NT	+	*24, 31, 18	*+	+	+	R	
hdfR	6.3 ± 0.4	9.2 ± 3.1	6 ± 2	6.2 ± 2.9	+			-		S	-
hemL	7.8 ± 1.2	9.7 ± 1.3	5 ± 1	NT	+		+	+	+	S	-
rbsK	4.2 ± 0.4	10.1 ± 0.5	9 ± 3	3.9 ± 0.2	+			-		R	
nuoG	3 ± 0.3	10.2 ± 0.2	NT	NT	+	*24, 31, 18	*+	+	+	R	
ldcA[13]	34 ± 5	10.4 ± 2.7	0.36 ± 0.07	2.6 ± 1	-			-		S	-
nagC	4.2 ± 0.3	11 ± 1.4	NT	NT	-			-		R	
lon	3 ± 0.2	11.4 ± 1.4	5 ± 2	11.2 ± 7.3	-			+	+	S	-
rpoE[12]	13 ± 2.4	11 ± 3 (*8*)	NT	NT	-			+	+	R	
rppH[13]	9.3 ± 0.3	11.7 ± 1.9	0.9 ± 0.1	2.5 ± 0.8	-			+	+	R	
yifE	6.3 ± 0.3	12.1 ± 1.4	4 ± 1	9.6 ± 5.3	+			-		S	-
nuoH	3.3 ± 0.4	12.5 ± 2.1	NT	NT	+	*24, 31, 18	*+	+	+	R	
dsbA	3.6 ± 0.4	13 ± 1.8	NT	3.2 ± 1	-			-		R	
ubiH	29 ± 4	13.6 ± 0.2	11 ± 4	NT	+	*86, 87, 51	*+	+	+	R	
nuoL	4.3 ± 0.6	14.7 ± 2.8	2.7 ± 0.8	NT	+	*24, 31, 18	*+	+	+	R	
nuoC	4.9 ± 0.4	16 ± 0.5	6 ± 2	4.8 ± 1.5	+	24, 31, 18	+	+	+	R	
nuoJ	3.4 ± 0.4	16 ± 0.3	NT	NT	+	*24, 31, 18	*+	+	+	R	

hfq	7 ± 0.3	16.4 ± 2	45 ± 16	3.1 ± 1	+			+	-	S	-
ubiE	24 ± 1.9	18.2 ± 2.6	13 ± 6	22 ± 7	+	*86, 87, 51	+	+	+	R	
ubiA	30 ± 3	30.7 ± 2.1	NT	NT	+	*86, 87, 51	*+	+	+	S	-
ihfA	36 ± 3.2	175 ± 33	23 ± 9	5.5 ± 2.3	-			+	-	R	
ppk[12, 13]	Q	2.9 (*53*)	0.9 ± 0.4	2.5 ± 0.8	-			+	-	R	
groES[12, 13]	Q	10 (*54*)	1.2 ± 0.1	2.6 ± 1	-			+	-	R	
dinB[12]	4 ± 0.2	3.3 ± 0.5 (*35*)	5.5 ± 0.75 (*2*)	4.4 ± 1.3	-			-		R	
nusA[12]	NT	470 (*55*)			-			+	-	S	-
umuC	NT	1.2 ± 0.2 (*56*)	1.7 ± 0.25 (*2*)	5.6 ± 1.3	-			-		S	-
lexA3[12]	3.8 ± 0.5	3.3 ± 0.4 (*57*)	5.2 ± 0.54 (*2*)	20 ± 5	-			-		S	+
recA[12]	37 ± 9	100 ± 30 (*57*)	16 ± 3.6 (*2*)	35 ± 15	-			-		S	+
recB[12]	5.5 ± 0.4	36 ± 7 (*58*)	220 ± 130 (*2*)	32 ± 10	-			-		S	+
recC[12]	4.8 ± 0.8		NT	NT	-			-		S	+
recF[12]	NT	3.1 (*57*)	NT	NT						S	-
rpoS[12]	6.5 ± 1	9.3 ± 1.2 (*59*)	25 ± 12 (*2*)	4.5 ± 1.7	+			-		S	-
ruvA[12]	NT	18 ± 6 (*57*)	NT	NT	-			-		S	-
ruvB[12]	NT	16 ± 5 (*57*)	NT	NT	-			-		S	-
ruvC[12]	NT	20 ± 8 (*57*)	5.8 ± 0.5 (*2*)	121 ± 52	-			-		S	-
sulA[12]	Q	1.4 (*56*)	NT	NT	-			-		R	

Data taken from the following:

[1] Quantification from the Lac-papillation colony-screen assay (Methods).

[2] Table S3

[3] Table S5 (and Fig. 2)

[4] Table S5 (and Fig. 2)

[5] Transcription from σ^S-controlled chromosomal *yiaG-yfp* (+, partially or fully required; -, not required). Figs. 4A, S4, Tables S7, S8

[6] Figs. 4D-F, S7, Table S9

[7] Figs. 4G-I, S8, Table S10

[8] SDS/EDTA-sensitivity (+, sensitive; -, resistant), Tables S7, S11

[9] Transcription from σ^E-controlled *rpoH*P3-*lacZ* (+, partially or fully required; -, not required). Figs. 4J, S8, Table S11

[10] Table S7. S, sensitive; R, resistant

[11] Transcription from the SOS-controlled chromosomal Δ*attλ-P*$_{sulA}$*-gfp* (+, partially or fully required; -, not), Fig. 4K

[12] For these mutants, quantitative Lac reversion data and, for some, Tet reversion data, are taken from the papers cited after the data.

[13] These mutants show insignificant SIM deficiency ($P > 0.05$) in the Tet and Nal assays.

Table S2. Genes of the stress-induced-mutagenesis network

List of the Tn-insertion mutant genes that function in SIM, isolated by papillation screen, and confirmed by quantitative Lac mutation assay (Table S3), plus previously identified SIM genes, plus *crp*, identified by candidate-gene approach here, and their descriptions. Genes annotated "F", for found, under "Previously Known" were previously identified as required for SIM and also identified in our screens (references listed, those that showed the requirement). Some genes previously known to be required for SIM (per the references cited) were not found by the papillation screen (annotated "N", for not found, under "Previously Known"), most for reasons we understand: *dinB* is present in two copies in Lac assay strains used in the primary screen, one in the chromosome and one in an F' plasmid, and both copies must be mutated to observe SIM-deficiency (*60*), which is very unlikely in a transposon mutagenesis and screen. Special *lexA* mutations called *lexA*Ind, which encode an uncleavable LexA transcriptional repressor protein, cause SIM defect (*32, 56*), and these cannot be created by Tn insertion. *nusA* is an essential gene the defect of which is studied with temperature-sensitive alleles (*61, 62*), and an appropriate conditional mutation may not have been obtainable by Tn insertion. *polB* accounts for ~15% of stress-induced frameshift reversion and shows its SIM-defective phenotype only in cells lacking *dinB* (*35*), which accounts for the other 85%, and so would not be expected to be identified in our screen. *umuCD*, encoding DNA polymerase V, is required for some of the DSB-dependent stress-induced loss-of-function mutations in the *ampD* gene (*63*) and some DSB-dependent stress-induced reversion of a *tet* frameshift allele (*2*) but not reversion of the *lac* allele used in our primary screen (*32, 56*), so could not have been found in this screen. Mutant alleles of the *ruvA*, *ruvB*, and *ruvC* mutations render the primary screen ineffective because all cause the entire colony to turn blue obscuring observation of blue mutant papillae.

Classification	Gene	Gene-product description	Reference(s)	SIM defect strength[1]	Previously known (F/N)
Regulators	*arcA*	Dual transcriptional regulator for anoxic redox control and a response regulator of ArcBA two component regulatory system	(*64, 65*)	W	
	argR	DNA-binding transcriptional dual regulator	(*66, 67*)	M	
	barA	Sensor kinase of BarA-UvrY two component regulatory system	(*68*)	W	
	crp	Transcriptional dual regulator	(*69*)	M	
	cyaA	Adenylate cyclase synthesizes c-AMP; c-AMP-CRP complex is a DNA binding transcriptional dual regulator	(*70*)	M	
	fruR	Dual transcriptional regulator involved in carbon metabolism	(*71*)	M	
	glnK	Nitrogen assimilation regulatory protein	(*72*)	M	
	hdfR	Dual transcriptional regulator	(*73, 74*)	S	
	ihfA	Integration host factor, α subunit; DNA-binding transcriptional dual regulator	(*75*)	S	

	lexA	Transcriptional repressor of the SOS DNA-damage response genes	(*32, 56*)	M	N
	lrp	DNA-binding transcriptional dual regulator	(*76*)	W	
	malI	Transcriptional repressor	(*77*)	W	
	metJ	DNA binding transcriptional repressor	(*78*)	M	
	nagC	Transcriptional dual regulator; regulates phosphotransferase system	(*79*)	S	
	nusA	Transcription/antitermination factor, essential gene	(*55*)	S	N
	phoU	Negative regulator of PhoR/PhoB two-component regulatory system	(*80*)	M	
	ppk	Polyphosphate kinase, involved in stress responses in some bacteria	(*53*)	W	F
	rpoE	Encodes σ^E transcription factor; transcriptional activator of the membrane protein stress response, dual transcriptional regulator	(*8*)	S	
	rpoS	*Stationary-phase sigma factor σ^S, transcriptional activator of the general/starvation-stress response*	(*59, 81*)	S	F
	treR	DNA-binding transcriptional repressor	(*82*)	W	
	uvrY	Cognate response regulator of BarA-UvrY two-component regulatory system	(*83*)	W	
Chaperones and proteases					
	cspC	Binds RNA and ssDNA	(*84*)	W	
	groES	Molecular chaperone	(*54*)	S	F
	hfq	Binds RNA; involved in post-transcriptional regulation	(*85*)	S	
	ldcA	LD carboxypeptidase A; recycling of peptidoglycan A	(*52*)	S	
	lon	DNA binding ATP-dependent protease	(*86*)	S	
	pepP	Proline aminopeptidase II	(*87*)	W	
	prc	Carboxy-terminal protease for penicillin-binding protein	(*88*)	W	
DNA replication and repair-related					
	dinB	DNA polymerase IV	(*60*)	M	N
	ligA	DNA ligase	(*89*)	W	
	polB	DNA polymerase II	(*35*)		N
	recA	*Recombination and SOS-response activation*	(*32, 90*)	S	F
	recB	*Exonuclease V- RecBCD complex, DNA double-strand end (DSE)-repair recombination and SOS response activation by DSEs*	(*90*)	S	F
	recC	*Exonuclease V- RecBCD complex, DSE-repair recombination and SOS response activation by DSEs*	(*90*)		F
	recF	ssDNA and dsDNA binding recombinational repair protein	(*56, 57*)	M	N
	ruvA	Branch migration of Holliday junctions	(*34, 58*)	S	N
	ruvB	Branch migration of Holliday junctions	(*34, 58*)	S	N

	ruvC	Holiday junction endonuclease	(*34, 58*)	S	N
	sulA	Cell division inhibition during SOS response	(*56*)	W	F
	umuC D	DNA polymerase V	(*2, 63*)	W	N
Electron transfer					
	cyoA	Cytochrome bo′ terminal oxidase subunit I	(*91*)	M	
	cyoD	Cytochrome bo′ terminal oxidase subunit IV	(*92*)	M	
	fre	NAD(P)H:flavin oxidoreductase	(*93*)	W	
	hemL	glutamate-1-semialdehyde aminotransferase; biosynthesis of hemes	(*94*)	S	
	hscB	Co-chaperone of HscA ATPase; involved in biogenesis of Fe-S cluster	(*95, 96*)	M	
	mdh	*Malate dehydrogenase; transfer of NADH to NADH:ubiquinone oxidoreductase*	(*97, 98*)	M	
	nuoC	NADH ubiquinone oxidoreductase subunit C; belongs to connecting fragment	(*17, 99*)	S	
	nuoG	NADH ubiquinone oxidoreductase subunit G; belongs to water-soluble dehydogenase fragment	(*17, 99*)	S	
	nuoH	NADH ubiquinone oxidoreductase subunit H; belongs to membrane fragment	(*17, 99*)	S	
	nuoJ	NADH ubiquinone oxidoreductase subunit J; belongs to membrane fragment	(*17, 99*)	S	
	nuoK	NADH ubiquinone oxidoreductase subunit K; belongs to membrane fragment	(*17, 99*)	M	
	nuoL	NADH ubiquinone oxidoreductase subunit L; belongs to membrane fragment	(*17, 99*)	S	
	pgi	Phosphoglucose isomerase	(*100*)	M	
	sdhB	Subunit B of succinate ubiquinone oxidoreductase	(*101*)	M	
	sdhD	Subunit D of succinate ubiquinone oxidoreductase	(*101*)	M	
	ubiA	4-Hydroxybenzoate octaprenyltransferase*; ubiquinone biosynthesis*	(*18*)	S	
	ubiD	Decarboxylase; ubiquinone biosynthesis	(*18*)	M	
	ubiE	Methyltransferase; ubiquinone biosynthesis	(*18*)	S	
	ubiH	2-octaprenyl-6-methoxyphenol hydroxylase; ubiquinone biosynthesis	(*18*)	S	
	ubiX	Decarboxylase; ubiquinone biosynthesis	(*102*)	W	
Metabolism					
	apaH	Diadenosine tetraphosphatase	(*103*)	M	
	cysD	ATP sulfurylase	(*104*)	M	
	argD	Arginine biosynthesis	(*105*)	W	
	lipB	Lipoyl-protein ligase; lipoate biosynthesis	(*106, 107*)	M	
	pgl	6-phosphogluconolactonase	(*108*)	M	
	rbsK	Ribokinase	(*109*)	S	
	speD	S-adenosylmethionine decarboxylase; polyamine biosynthesis	(*110*)	M	
	speE	Spermidine synthase; polyamine biosynthesis	(*110*)	M	
Cellular processes					
	acrA	Multidrug efflux system	(*111*)	W	

	acrB	Multidrug-efflux system	(*111*)	W	
	dsbA	Periplasmic disulfide-introducing catalyst	(*112*)	S	
	dsbC	Periplasmic protein disulfide isomerase	(*113*)	M	
	proL	Proline tRNA	(*114*)	M	
	pstB	ATP-binding component of the phosphate ABC transporter	(*115*)	W	
	pstC	ATP-binding component of the phosphate ABC transporter	(*115*)	M	
	rppH	RNA pyrophosphohydrolase	(*116*)	S	
	srlA	Subunit of glucitol/sorbitol phosphotransferase system	(*117*)	W	
	tatC	Subunit of the twin-arginine translocation complex	(*118*)	M	
	trxB	Thioredoxin reductase	(*119*)	W	
	znuA	Periplasmic zinc-binding component of the zinc transporter	(*120*)	M	
	znuC	ATPase subunit of the zinc transporter	(*120*)	W	
Unknown function					
	apaG	Unknown protein, gene mutation is polar on *apaH*	(*121*)	W	
	ybjT	Conserved protein with NAD(P)-binding Rossmann-fold domain	(*122*)	W	
	yfjL	Predicted CP4-57 cryptic prophage protein	(*122*)	W	
	ygfA	Putative 5-formyl-tetrahydrofolate cyclo-ligase; highly conserved from prokaryotes to human	(*122*)	M	
	yhhK	Predicted acetyl transferase predicted to be involved in pantothenate metabolism	(*123*)	M	
	yjcC	Predicted signal-transduction protein	(*122*)	W	
	yjcC	Conserved protein	(*122*)	W	
	yifE	Conserved protein	(*122*)	S	
	yigP	Conserved protein	(*122*)	M	
	yqgE	Predicted protein	(*122*)	W	
	yqiC	Conserved protein, Salmonella homologue a membrane protein	(*122, 124*)	M	
	ytjB	A membrane protein with a unique signal sequence	(*125*)	W	

[1]Weak (W), moderate (M), and strong (S), SIM defect strengths are defined in Table S3 as 1.5-3- (W), 3.1-9- (M), and >9-fold (S) stress-induced mutation rate defects caused by the mutations in these genes.

Table S3. Stress-induced mutation rates of previously unpublished SIM network mutants in quantitative assay

Mutation rates are the change in mutant frequencies between days 3 and 5 for three to four independent cultures [**Quantitative SIM assay**, Methods, per (*59*)], and are grouped by strength of phenotype: weak (W, 1.5-3-fold), moderate (M, 3.1-9-fold), and strong (S, >9-fold) mutagenesis defects, compared with the WT control. Fold decrease in mutation rate is the mutation rate of the WT (control) divided by the rate from the mutant assayed in parallel then averaged for 3 independent experiments. *P* values are for differences from the WT control strain per Methods.

Strain	**Mutant gene**	**Mutation rate (Lac^+ cfu per 10^8 cells per day)**			**Fold decrease in mutation rate of mutants (WT/mutant) ± SEM**			**Mean fold decrease in mutation rate of mutants (WT/mutant) ± SEM**	***P* value**	**SIM defect strength**
		Exp 1	Exp 2	Exp 3	Exp 1	Exp 2	Exp 3			
SMR4562	WT	32.5	33	37.5						
SMR12233	*srlA*	22.3	23.6	24.2	1.5	1.4	1.5	1.5 ± 0.04	0.003	W
SMR4562	WT	*32.5	34.5	*37.5						W
SMR12221	*ybjT*	25.2	24.6	19.6	1.3	1.4	1.9	1.5 ± 0.2	0.007	W
SMR4562	WT	37	*33	28						W
SMR12001	*treR*	21.4	26.4	17.2	1.7	1.3	1.6	1.5 ± 0.1	0.042	W
SMR4562	WT	*32.5	*34.5	31.2						W
SMR12056	*yjcC*	19.7	23.6	20.6	1.6	1.5	1.5	1.5 ± 0.06	0.002	W
SMR4562	WT	*32.5	33.5	30						W
SMR11978	*yicC*	24.9	23.2	17.6	1.3	1.4	1.7	1.5 ± 0.1	0.014	W
SMR4562	WT	33.2	29.9	39						W
SMR11981	*ytjB*	21.7	19.8	21.7	1.5	1.5	1.8	1.6 ± 0.1	0.009	W
SMR4562	WT	37.3	*33.5	35.7						W
SMR12023	*arcA*	23	21.4	18.5	1.6	1.6	1.9	1.7 ± 0.1	0.001	W
SMR4562	WT	29.3	30.1	34.2						W
SMR11973	*ligA*	18.4	17	18.8	1.6	1.8	1.8	1.7 ± 0.07	0.001	W
SMR12050	*yqgE*	18.7	17.9	19.3	1.6	1.7	1.8	1.7 ± 0.06	0.001	W
SMR4562	WT	*37.3	*33	*28						W
SMR12005	*argD*	24.6	20.9	12.6	1.5	1.6	2.2	1.8 ± 0.2	0.04	W
SMR4562	WT	*32.5	29.8	35.2						W
SMR12241	*apaG*	16.7	18.4	20.6	1.9	1.6	1.7	1.8 ± 0.1	0.002	W
SMR4562	WT	*29.3	*30.1	*34.2						W
SMR11979	*yfjL*	16.8	17.2	18	1.7	1.8	1.9	1.8 ± 0.05	0.001	W
SMR4562	WT	*37	30.6	36.1						W
SMR11984	*acrB*	22.6	17.5	17.8	1.6	1.7	2	1.8 ± 0.1	0.004	W
SMR4562	WT	*28.5	36	*37.5						W
SMR11992	*malI*	17.9	17.4	15.7	1.6	2.1	2.4	2.0 ± 0.2	0.004	W
SMR4562	WT	44.6	143	139						W
PJH1276	*lrp*	26	96	51	1.7	1.5	2.7	2.0 ± 0.4	0.25	W
SMR4562	WT	*34.5	*34.2	*31.2						W
SMR12220	*acrA*	26.3	8.8	17.4	1.3	3.9	1.8	2.3 ± 0.8	0.038	W
SMR4562	WT	*28.5	*30	35.2						W
SMR12314	*uvrY*	9.31	20.7	13.9	3.1	1.4	2.5	2.3 ± 0.5	0.013	W

SMR4562	WT	*28.5	*29.8	*35.2						W
SMR11969	*trxB*	9.74	14.8	13.7	2.9	2	2.6	2.5 ± 0.3	0.002	W
SMR4562	WT	30.9	34.4	35.5						W
SMR12007	*fre*	13.9	14.1	12.9	2.2	2.4	2.7	2.5 ± 0.2	0.0002	W
SMR4562	WT	*29.3	*30.1	*34.2						W
SMR12148	*cspC*	12.5	11.9	11.3	2.3	2.5	3	2.6 ± 0.2	0.0002	W
SMR4562	WT	*37.3	*35.2	*30						W
SMR12253	*barA*	9.4	17.8	16.3	4	2	1.8	2.6 ± 0.7	0.004	W
SMR4562	WT	*37.3	*33	*37.5						W
SMR12251	*prc*	10.5	11.8	22.5	3.6	2.8	1.7	2.7 ± 0.5	0.007	W
SMR4562	WT	26.9	32.6	*36.1						W
SMR11970	*pepP*	10	14.5	11.7	2.7	2.2	3.1	2.7 ± 0.2	0.003	W
SMR4562	WT	*32.5	*33.5	*35.2						W
SMR12232	*pstB*	7.15	17.1	20	4.5	2	1.8	2.8 ± 0.9	0.009	W
SMR4562	WT	*37	28.3	*28						W
SMR11967	*ubiX*	11.9	13.9	8.8	3.1	2	3.2	2.8 ± 0.4	0.004	W
SMR4562	WT	*28.5	*31.2	*36						W
SMR12211	*znuC*	6.9	13.4	15.3	4.1	2.3	2.4	2.9 ± 0.6	0.004	W
SMR4562	WT	18	24.7	22.5						M
SMR17290	*crp*	5.1	8	8.8	3.5	3.1	2.6	3.1± 0.3	0.003	M
SMR4562	WT	29	*28.3	*35.2						M
SMR12313	*ubiD*	6.9	11.1	14.6	4.2	2.5	2.4	3.1 ± 0.6	0.003	M
SMR4562[1]	WT	38.7	56	65						M
SMR12134[1]	*yhhK*	10.8	16	25	3.6	3.5	2.6	3.2 ± 0.3	0.025	M
SMR4562	WT	37	29.8	27.9						M
SMR11997	*metJ*	8.3	12.8	9.1	4.4	2.3	3.1	3.3 ± 0.6	0.002	M
SMR4562	WT	*37	*28.5	*30.6						M
SMR13473	*phoU*	9.9	8.8	9.1	3.7	3.2	3.4	3.5 ± 0.2	0.011	M
SMR4562	WT	*29.3	*30.1	*34.2						M
SMR12219	*lipB*	8.9	7.3	9.4	3.3	4.1	3.7	3.7 ± 0.2	0.0002	M
SMR4562	WT	30.87	34.4	35.45						M
SMR11983	*proL*	8.6	9.3	8.5	3.6	3.7	4.1	3.8 ± 0.2	6×10^{-5}	M
SMR4562	WT	*30.9	*36	27.1						M
SMR11962	*speD*	9.6	10.1	5.5	3.2	3.6	4.9	3.9 ± 0.5	0.001	M
SMR4562	WT	*37.3	*28.3	28.1						M
SMR12302	*cysD*	4.86	15.9	13.4	7.7	1.8	2.1	3.9 ± 1.9	0.012	M
SMR4562	WT	42.4	32.4	*37.3						M
SMR12252	*sdhD*	5.7	16.5	11.2	7.4	2	3.3	4.2 ± 1.6	0.003	M
SMR4562	WT	*30.9	*34.4	*35.5						M
SMR12240	*speE*	6.8	10.5	7.3	4.6	3.3	4.8	4.2 ± 0.5	0.0002	M
SMR12002	*fruR*	8.9	8.4	7	3.5	4.1	5.1	4.2 ± 0.5	7×10^{-5}	M
SMR11994	*argR*	8	7	8.8	3.9	4.9	4	4.3 ± 0.3	7×10^{-5}	M
SMR4562	WT	*33.5	*28.1	*36.1						M
SMR12257	*pgl*	7.2	8.6	7.34	4.7	3.3	4.9	4.3 ± 0.5	0.0005	M
SMR4562	WT	37.3	35.2	30						M
SMR12236	*znuA*	5	9.5	11.1	7.5	3.7	2.7	4.6 ± 1.4	0.0008	M
SMR4562	WT	37	30.6	36.1						M
SMR12071	*pstC*	7.77	7.25	7.16	4.8	4.2	5	4.7 ± 0.2	0.005	M
SMR4562	WT	*33.4	26	34						M
SMR12069	*dsbC*	6.4	7.1	5.7	5.2	3.7	6	5 ± 0.7	0.0007	M
SMR4562	WT	35.7	*29	*33.2						M

SMR12574	*cyoD*	11.8	3.28	8.5	3	8.8	3.9	5.3 ± 1.8	0.0014	M
SMR4562	WT	*37	*36	*27.1						M
SMR12307	*hscB*	6	7.3	6.2	6.2	4.9	4.4	5.2 ± 0.5	0.012	M
SMR4562	WT	*33.2	*29.9	*39						M
SMR12570	*cyoA*	5.7	6.7	6.3	5.8	4.4	6.2	5.5 ± 0.5	0.0005	M
SMR4562	WT	*37	*36	*37.5						M
SMR11990	*apaH*	4.93	6.3	10.4	7.5	5.7	3.6	5.6 ± 1.1	6×10^{-5}	M
SMR4562	WT	*30.9	*34.4	*35.5						M
SMR12216	*yqiC*	6.3	4.9	6.8	4.9	7	5.2	5.7 ± 0.7	5×10^{-5}	M
SMR4562	WT	*33.2	*29.9	*39						M
SMR12332	*sdhB*	6.1	6.2	5.6	5.5	4.8	7	5.7 ± 0.6	0.0005	M
SMR4562	WT	*37.3	*29.8	*35.2						M
SMR12227	*glnK*	3.2	14.9	8.1	11.8	2	4.3	6 ± 3	0.003	M
SMR4562	WT	*29.8	*31.2	*36.1						M
SMR12008	*tatC*	12.6	3.1	5	2.4	10.1	7.2	6.5 ± 2.2	0.002	M
SMR4562	WT	*30.9	*34.4	*35.5						M
SMR11998	*cyaA*	4.5	4.1	5.4	6.9	8.4	6.6	7.3 ± 0.6	4×10^{-5}	M
SMR4562	WT	*42.4	*32.4	*37.3						M
SMR12060	*mdh*	3.86	6.3	5.5	11	5.1	6.8	7.6 ± 1.7	0.0004	M
SMR4562	WT	38.7	*29	*32.6						M
SMR11980	*yigP*	7.1	3.9	3.3	5.5	7.5	9.9	7.6 ± 1.3	0.004	M
SMR4562	WT	*37.3	*33.5	*35.2						M
SMR12316	*ygfA*	3.14	5.9	6.4	11.9	5.7	5.5	7.7 ± 2.1	4×10^{-5}	M
SMR4562	WT	*36	*37.5	*36.1						M
SMR11995	*pgi*	6	4.4	3.74	6	8.5	9.7	8.1 ± 1.1	3×10^{-6}	M
SMR4562	WT	*38.7	*29	*32.6						M
SMR13653	*nuoK*	4.7	3.3	3.6	8.2	8.8	9	8.7 ± 0.2	0.0005	M
SMR4562	WT	*37.3	*36	*27.1						M
SMR12235	*hdfR*	2.45	6.9	3.8	15.2	5.2	7.1	9.2 ± 3.1	0.001	S
SMR4562	WT	*33.2	*29.9	*39						S
SMR11982	*hemL*	2.7	3.8	4.3	12.2	7.9	9	9.7 ± 1.3	0.0004	S
SMR4562	WT	*29.3	*30.1	*34.2						S
SMR12584	*rbsK*	2.7	3.2	3.4	11	9.3	10.1	10.1 ± 0.5	5×10^{-5}	S
SMR4562	WT	*38.7	*29	*32.6						S
SMR13640	*nuoG*	4	2.8	3.1	9.8	10.3	10.4	10.2 ± 0.2	0.0004	S
SMR4562	WT	*37	*30.6	*36.1						S
SMR11964	*ldcA*	2.35	4.4	4.3	15.8	7	8.5	10.4 ± 2.7	0.0001	S
SMR4562	WT	*30.9	*34.4	*35.5						S
SMR11966	*nagC*	3.2	2.5	3.8	9.6	13.8	9.5	11 ± 1.4	3×10^{-5}	S
SMR12254	*lon*	3.5	2.5	3.1	8.9	13.7	11.5	11.4 ± 1.4	3×10^{-5}	S
SMR4562	WT	18.8	16.9	29.2						S
SMR5236	*rpoE*	2.8	1.6	1.7	6.7	10.6	17.2	11.5 ± 3	0.007	S
SMR4562	WT	*33.4	*26	*34						S
SMR12210	*rppH*	3.3	2.7	2.2	10.1	9.5	15.5	11.7 ± 1.9	0.0004	S
SMR12238	*yifE*	3.6	2	2.4	9.3	13.2	13.9	12.1 ± 1.4	0.0004	S
SMR4562	WT	*33.2	*29.9	*39						S
SMR13654	*nuoH*	2.7	3.3	2.4	12.3	9	16.4	12.5 ± 2.1	0.0003	S
SMR4562	WT	*28.3	*35.2	*30.6						S
SMR12309	*dsbA*	1.8	3.7	2.2	15.7	9.6	13.8	13 ± 1.8	0.0002	S
SMR4562	WT	40.2	*30.6	*36.1						S
SMR12260	*ubiH*	3	2.3	2.6	13.4	13.4	14.1	13.6 ± 0.2	0.0003	S

SMR4562	WT	*28.3	*27.1	*36.1						S
SMR12239	*nuoL*	2.9	1.4	2.4	9.8	19.4	15.1	14.7 ± 2.8	0.0006	S
SMR4562	WT	*29.3	*28.3	34.1						S
SMR12223	*nuoC*	1.9	1.8	2	15.5	15.7	17.1	16 ± 0.5	0.0001	S
SMR4562	WT	*38.7	*29	*32.6						S
SMR13652	*nuoJ*	2.5	1.8	2	15.5	16.5	16.1	16 ± 0.3	0.0004	S
SMR12261	*hfq*	3	1.8	1.6	13.1	16.1	20	16.4 ± 2	0.0004	S
SMR4562	WT	*28.3	*33.2	*36.1						S
SMR12242	*ubiE*	1.6	1.44	2.6	17.7	23.1	14	18.2 ± 2.6	0.0002	S
SMR4562	WT	*29.3	*30.1	*34.2						S
SMR12311	*ubiA*	0.84	1.02	1.23	34.7	29.4	27.9	30.7 ± 2.1	4×10^{-5}	S
SMR4562	WT	*33.4	*26	*34						
SMR11971	*ihfA*	0.14	0.19	0.23	241	137	148	175 ± 33	0.0003	S

* Data indicated are repeats (in the table) from experiments with many different mutants run in parallel with each WT control measurement.

[1] The mutant and control WT were grown in LBH liquid medium due to growth defect of the *yhhK* mutant in liquid M9 medium and the mutation rates were determined as per Methods.

Table S4. *Escherichia coli* strains and plasmid used

(P) indicates a transposon insertion in the promoter region. CGSC, *E. coli* Genetic Stock Center (Yale).

Strain	Relevant genotype*	Reference and/or source
CAG45114	MG1655 λ *rpoH*P3-*lacZ*	(*20*)
FC29	Δ(*lac-proB*) $_{XIII}$ *ara thi* [F' Δ(*lacIZ*) *proAB*$^{+}$]	(*32*)
FC36	Δ(*lac-proB*)$_{XIII}$ *ara thi* Rif^{R}	(*32*)
FC40	FC36 [F' *lacI33*Ω*lacZ proAB*$^{+}$]	(*32*)
JW0048	BW25113 Δ*apaH761*::FRTKanFRT	CGSC (*33*)
JW0049	BW25113 Δ*apaG762*::FRTKanFRT	″
JW0078	BW25113 Δ*fruR786*::FRTKanFRT	″
JW0116	BW25113 Δ*speD738*::FRTKanFRT	″
JW0117	BW25113 Δ*speE739*::FRTKanFRT	″
JW0419	BW25113 Δ*cyoD786*::FRTKanFRT	″
JW0422	BW25113 Δ*cyoA789*::FRTKanFRT	″
JW0429	BW25113 Δ*lon725*::FRTKanFRT	″
JW0440	BW25113 Δ*glnK736*::FRTKanFRT	″
JW0451	BW25113 Δ*acrB747*::FRTKanFRT	″
JW0452	BW25113 Δ*acrA748*::FRTKanFRT	″
JW0662	BW25113 Δ*nagC725*::FRTKanFRT	″
JW0712	BW25113 Δ*sdhD772*::FRTKanFRT	″
JW0714	BW25113 Δ*sdhB774*::FRTKanFRT	″
JW0750	BW25113 Δ*pgl739*::FRTKanFRT	″
JW0871	BW25113 Δ*trxB786*::FRTKanFRT	″
JW0872	BW25113 Δ*lrp787*::FRTKanFRT	″
JW1181	BW25113 Δ*ldcA782*::FRTKanFRT	″
JW1612	BW25113 Δ*malI768*::FRTKanFRT	″
JW1702	BW25113 Δ*ihfA786*::FRTKanFRT	″
JW1812	BW25113 Δ*cspC747*::FRTKanFRT	″
JW1819	BW25113 Δ*prc755*::FRTKanFRT	″
JW1847	BW25113 Δ*znuC783*::FRTKanFRT	″
JW1852	BW25113 Δ*ruvC789*::FRTKanFRT	″
JW1899	BW25113 Δ*uvrY760*::FRTKanFRT	″
JW2273	BW25113 Δ*nuoL760*::FRTKanFRT	″
JW2274	BW25113 Δ*nuoK761*::FRTKanFRT	″
JW2275	BW25113 Δ*nuoJ762*::FRTKanFRT	″
JW2277	BW25113 Δ*nuoH764*::FRTKanFRT	″
JW2308	BW25113 Δ*ubiX732*::FRTKanFRT	″
JW2511	BW25113 Δ*hscB773*::FRTKanFRT	″
JW2556	BW25113 Δ*rseA*::FRTKanFRT	(*33*)
JW2757	BW25113 Δ*barA784*::FRTKanFRT	CGSC (*33*)
JW2788	BW25113 Δ*recB745*::FRTKanFRT	″
JW2798	BW25113 Δ*rppH754*::FRTKanFRT	″
JW2861	BW25113 Δ*dsbC744*::FRTKanFRT	″
JW2875	BW25113 Δ*ubiH758*::FRTKanFRT	″
JW2876	BW25113 Δ*pepP759*::FRTKanFRT	″
JW2879	BW25113 Δ*ygfA763*::FRTKanFRT	″
JW2915	BW25113 Δ*yqgE723*::FRTKanFRT	″
JW3205	BW25113 Δ*mdh761*::FRTKanFRT	″
JW3206	BW25113 Δ*argR762*::FRTKanFRT	″
JW3320	BW25113 Δ*crp765*::FRTKanFRT	″
JW3322	BW25113 Δ*argD767*::FRTKanFRT	″
JW3619	BW25113 Δ*yicC750*::FRTKanFRT	″
JW3702	BW25113 Δ*phoU755*::FRTKanFRT	″
JW3703	BW25113 Δ*pstB756*::FRTKanFRT	″
JW3705	BW25113 Δ*pstC758*::FRTKanFRT	″

JW3731	BW25113 Δ*rbsK781*::FRTKanFRT	″
JW3737	BW25113 Δ*yifE786*::FRTKanFRT	″
JW3778	BW25113 Δ*cyaA751*::FRTKanFRT	″
JW3815	BW25113 Δ*tatC781*::FRTKanFRT	″
JW3820	BW25113 Δ*fre784*::FRTKanFRT	″
JW3832	BW25113 Δ*dsbA723*::FRTKanFRT	″
JW3909	BW25113 Δ*metJ725*::FRTKanFRT	″
JW3985	BW25113 Δ*pgi721*::FRTKanFRT	″
JW4022	BW25113 Δ*yjcC755*::FRTKanFRT	″
JW4130	BW25113 Δ*hfq722*::FRTKanFRT	″
JW4200	BW25113 Δ*treR788*::FRTKanFRT	″
JW4364	BW25113 Δ*arcA726*::FRTKanFRT	″
JW5089	BW25113 Δ*lipB769*::FRTKanFRT	″
JW5116	BW25113 Δ*ybjT770*::FRTKanFRT	″
JW5375	BW25113 Δ*nuoC768*::FRTKanFRT	″
JW5429	BW25113 Δ*srlA777*::FRTKanFRT	″
JW5437	BW25113 Δ*rpoS746*::FRTKanFRT	″
JW5505	BW25113 Δ*yqiC737*::FRTKanFRT	″
JW5536	BW25113 Δ*arcB738*::FRTKanFRT	″
JW5581	BW25113 Δ*ubiE778*::FRTKanFRT	″
JW5607	BW25113 Δ*hdfR785*::FRTKanFRT	″
JW5831	BW25113 Δ*znuA782*::FRTKanFRT	″
MG22	*galK*::P_{lac}*cfp* Amp	(*16*)
MG1655	*rph-1* λ^- F^-	(*126*)
MV1161	*supE44*	(*127*)
NEC252	MG1655 *yiaG-yfp* FRT*cat*FRT	This work
PJH1242	SMR4562 Δ*lrp787*::FRTKanFRT	SMR4562 X P1 (JW0872)
PJH1276	SMR4562 Δ*lrp787*::FRT	PJH1242 X pCP20
RTC0013	MG1655 Δ*recB*::Kan	(*6*)
SG12066	*rssB*::Tet	Susan Gottesman (NIH)
SMR593	FC40 *recB21*	(*90*)
SMR820	FC40 *lexA3*(Ind^-) *malB*::*Tn*9	(*56*)
SMR1982	FC40 *recG258*::Tn*10* miniKan	(*34*)
SMR3525	FC40 Δ*umuDC*::*cat*	(*56*)
SMR3856	FC40 Lac^+ day 5 isolate	(*128*)
SMR3858	FC40 Lac^+ day 5 isolate	(*128*)
SMR3859	FC40 Lac^+ day 5 isolate	(*128*)
SMR4562	Independent construction of FC40	(*56*)
SMR4610	FC40 *recA*::Tn*10*dCam	SIM down-mutant
SMR5236	SMR4562 *rpoE2072*::Tn*10*dCam	(*8*)
SMR5535	SMR4562 Δ(*srlR-recA*)::Tn*10*	Laboratory collection
SMR5830	SMR4562 *dinB10* [F' *dinB10*]	(*60*)
SMR5833	SMR4562 [pKD46]	(*129*)
SMR5889	FC36 Δ*dinB50*::FRTKanFRT [F' *dinB50*::FRT]	(*129*)
SMR6039	FC40 Δ*attλ*::P_{sulA}*gfp-mut2*	(*130*)
SMR6178	SMR6039 *lexA3*(Ind^-) *malB* ::Tn*9*	SMR6039 X P1 (SMR820)
SMR6269	SMR4562 *leu*::Tn*10*	(*131*)
SMR6276	SMR4562 Δara_{BAD}*567* Δ*attλ*::P_{BAD}I-*Sce*I	(*1*)
SMR6308	SMR6276 [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR9*]	(*1*)
SMR6541	SMR4562 *rpoS*::Tn*10*	(*59*)
SMR7530	SMR4562 *rssB*::Tet	SMR4562 X P1 (SG12066)
SMR8097	SMR4562 Δ*attλ*::P_{sulA}*gfp-mut2* Δ*recF1804*::FRTKanFRT	(*132*)
SMR10356	FC36 *rpoS*::Tn*10*	Laboratory collection
SMR10582	SMR4562 *yiaG-yfp* FRT*cat*FRT	SMR4562 X P1 (NEC252)
SMR10772	FC36 Δ*araBAD*567	(*2*)
SMR10774	FC36 Δara_{BAD}567 Δ*attλ*::P_{BAD}I-*Sce*I	(*2*)
SMR10799	FC36 Δ*araBAD*567 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	(*2*)
SMR10800	FC36 Δ*araBAD*567 Δ*attλ*::P_{BAD}I-*Sce*I	(*2*)

	Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	
SMR11351	FC36 *speD*::Tn*10*dCam	SIM down-mutation recipient strain
SMR11353	FC36 *hfq*::Tn*10*dCam	″
SMR11369	FC36 *ldcA*::Tn*10*dCam	″
SMR11375	FC36 *dsbA*::Tn*10*dCam	″
SMR11379	FC36 *nagC*::Tn*10*dCam	″
SMR11385	FC36 *ubiX*::Tn*10*dCam	″
SMR11387	FC36 *glnK*::Tn*10*dCam	″
SMR11392	FC36 *trxB*::Tn*10*dCam	″
SMR11398	FC36 *pepP*::Tn*10*dCam	″
SMR11404	FC36 *ihfA*::Tn*10*dCam	″
SMR11408	FC36 *prc*::Tn*10*dCam	″
SMR11410	FC36 *arcA*(P)::Tn*10*dCam	″
SMR11412	FC36 *ligA*(P)::Tn*10*dCam	″
SMR11431	FC36 *rppH*::Tn*10*dCam	″
SMR11437	SMR4562 Δ*araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	(*35*)
SMR11462	FC36 *hdfR*::Tn*10*dCam	SIM down-mutation recipient strain
SMR11463	FC36 *yqiC*::Tn*10*dCam	″
SMR11464	FC36 *yifE*::Tn*10*dCam	″
SMR11465	FC36 *yicC*::Tn*10*dCam	″
SMR11472	FC36 *yfjL*(P)::Tn*10*dCam	″
SMR11475	FC36 *yigP*::Tn*10*dCam	″
SMR11476	FC36 *ytjB*(P)::Tn*10*dCam	″
SMR11478	FC36 *hemL*::Tn*10*dCam	″
SMR11479	FC36 *proL*::Tn*10*dCam	″
SMR11482	FC36 *yhhK*::Tn*10*dCam	″
SMR11522	FC36 *acrB*::Tn*10*dCam	″
SMR11523	FC36 *acrA*::Tn*10*dCam	″
SMR11524	FC36 *barA*::Tn*10*dCam	″
SMR11527	FC36 *uvrY*::Tn*10*dCam	″
SMR11529	FC36 *nuoL*::Tn*10*dCam	″
SMR11530	FC36 *sdhD*::Tn*10*dCam	″
SMR11531	FC36 *apaH*::Tn*10*dCam	″
SMR11532	FC36 *pgl*::Tn*10*dCam	″
SMR11533	FC36 *mall*::Tn*10*dCam	″
SMR11534	FC36 *hscB*::Tn*10*dCam	″
SMR11815	FC36 *argR*::Tn*10*dCam	″
SMR11818	FC36 *pgi*::Tn*10*dCam	″
SMR11820	FC36 *ubiH*::Tn*10*dCam	″
SMR11821	FC36 *metJ*::Tn*10*dCam	″
SMR11822	FC36 *cyaA*::Tn*10*dCam	″
SMR11851	FC36 *nuoH*::Tn*10*dCam	″
SMR11854	FC36 *ybjT*::Tn*10*dCam	″
SMR11865	FC36 *treR*::Tn*10*dCam	″
SMR11872	FC36 *fruR*::Tn*10*dCam	″
SMR11873	FC36 *lipB*::Tn*10*dCam	″
SMR11880	FC36 *znuC*::Tn*10*dCam	″
SMR11881	FC36 *argD*::Tn*10*dCam	″
SMR11883	FC36 *pstB*::Tn*10*dCam	″
SMR11885	FC36 *fre*::Tn*10*dCam	″
SMR11886	FC36 *tatC*::Tn*10*dCam	″
SMR11923	FC36 *ygfA*::Tn*10*dCam	″
SMR11924	FC36 *yjcC*::Tn*10*dCam	″
SMR11925	FC36 *cysD*(P)::Tn*10*dCam	″
SMR11926	FC36 *dsbC*::Tn*10*dCam	″
SMR11927	FC36 *apaG*::Tn*10*dCam	″
SMR11929	FC36 *cyoA*::Tn*10*dCam	″
SMR11930	FC36 *cyoD*::Tn*10*dCam	″

SMR11932	FC36 *recB*::Tn*10*dCam	″
SMR11934	FC36 *groES*(P)::Tn*10*dCam	″
SMR11935	FC36 *lrp*::Tn*10*dCam	″
SMR11936	FC36 *phoU*::Tn*10*dCam	″
SMR11937	FC36 *rpoE*(P)::Tn*10*dCam	″
SMR11938	FC36 *rpoS*::Tn*10*dCam	″
SMR11939	FC36 *sulA*::Tn*10*dCam	″
SMR11940	FC36 *lon*::Tn*10*dCam	″
SMR11941	FC36 *recC*::Tn*10*dCam	″
SMR11942	FC36 *mdh*::Tn*10*dCam	″
SMR11943	FC36 *nuoC*::Tn*10*dCam	″
SMR11944	FC36 *ubiA*::Tn*10*dCam	″
SMR11945	FC36 *ubiD*::Tn*10*dCam	″
SMR11946	FC36 *speE*::Tn*10*dCam	″
SMR11947	FC36 *pstC*::Tn*10*dCam	″
SMR11948	FC36 *srlA*::Tn*10*dCam	″
SMR11949	FC36 *yqgE*::Tn*10*dCam	″
SMR11950	FC36 *znuA*::Tn*10*dCam	″
SMR11951	FC36 *sdhB*::Tn*10*dCam	″
SMR11952	FC36 *ubiE*::Tn*10*dCam	″
SMR11953	FC36 *nuoJ*::Tn*10*dCam	″
SMR11954	FC36 *nuoK*::Tn*10*dCam	″
SMR11955	FC36 *ppk*::Tn*10*dCam	″
SMR11956	FC36 *nuoG*::Tn*10*dCam	″
SMR11957	FC36 *arcA*(P)::Tn*10*dCam	″
SMR11958	FC36 *cysD*(P)::Tn*10*dCam	″
SMR11959	FC40 *rbsK*::Tn*10*dCam	SIM down-mutant
SMR11960	FC40 *cspC*::Tn*10*dCam	“
SMR11962	SMR4562 Δ*speD738*::FRTKanFRT	SMR4562 X P1 (JW0116)
SMR11963	SMR4562 Δ*hfq722*::FRTKanFRT	SMR4562 X P1 (JW4130)
SMR11964	SMR4562 Δ*ldcA782*::FRTKanFRT	SMR4562 X P1 (JW1181)
SMR11965	SMR4562 Δ*dsbA723*::FRTKanFRT	SMR4562 X P1 (JW3832)
SMR11966	SMR4562 Δ*nagC725*::FRTKanFRT	SMR4562 X P1 (JW0662)
SMR11967	SMR4562 Δ*ubiX732*::FRTKanFRT	SMR4562 X P1 (JW2308)
SMR11968	SMR4562 Δ*glnK736*::FRTKanFRT	SMR4562 X P1 (JW0440)
SMR11969	SMR4562 Δ*trxB786*::FRTKanFRT	SMR4562 X P1 (JW0871)
SMR11970	SMR4562 Δ*pepP759*::FRTKanFRT	SMR4562 X P1 (JW2876)
SMR11971	SMR4562 Δ*ihfA786*::FRTKanFRT	SMR4562 X P1 (JW1702)
SMR11972	SMR4562 Δ*prc755*::FRTKanFRT	SMR4562 X P1 (JW1819)
SMR11973	SMR4562 *ligA*(P)::Tn*10*dCam	SMR4562 X P1 (SMR11412)
SMR11974	SMR4562 Δ*rppH754*::FRTKanFRT	SMR4562 X P1 (JW2798)
SMR11975	SMR4562 Δ*hdfR785*::FRTKanFRT	SMR4562 X P1 (JW5607)
SMR11976	SMR4562 Δ*yqiC737*::FRTKanFRT	SMR4562 X P1 (JW5505)
SMR11977	SMR4562 Δ*yifE786*::FRTKanFRT	SMR4562 X P1 (JW3737)
SMR11978	SMR4562 Δ*yicC750*::FRTKanFRT	SMR4562 X P1 (JW3619)
SMR11979	SMR4562 *yfjL*(P)::Tn*10*dCam	SMR4562 X P1 (SMR11472)
SMR11980	SMR4562 *yigP*::Tn*10*dCam	SMR4562 X P1 (SMR11475)
SMR11981	SMR4562 *ytjB*(P)::Tn*10*dCam	SMR4562 X P1 (SMR11476)
SMR11982	SMR4562 *hemL*::Tn*10*dCam	SMR4562 X P1 (SMR11478)
SMR11983	SMR4562 *proL*::Tn*10*dCam	SMR4562 X P1 (SMR11479)
SMR11984	SMR4562 Δ*acrB747*::FRTKanFRT	SMR4562 X P1 (JW0451)
SMR11985	SMR4562 Δ*acrA748*::FRTKanFRT	SMR4562 X P1 (JW0452)
SMR11986	SMR4562 Δ*barA784*::FRTKanFRT	SMR4562 X P1 (JW2757)
SMR11987	SMR4562 Δ*uvrY760*::FRTKanFRT	SMR4562 X P1 (JW1899)
SMR11988	SMR4562 Δ*nuoL760*::FRTKanFRT	SMR4562 X P1 (JW2273)
SMR11989	SMR4562 Δ*sdhD772*::FRTKanFRT	SMR4562 X P1 (JW0712)
SMR11990	SMR4562 Δ*apaH761*::FRTKanFRT	SMR4562 X P1 (JW0048)
SMR11991	SMR4562 Δ*pgl739*::FRTKanFRT	SMR4562 X P1 (JW0750)
SMR11992	SMR4562 Δ*mall768*::FRTKanFRT	SMR4562 X P1 (JW1612)
SMR11993	SMR4562 Δ*hscB773*::FRTKanFRT	SMR4562 X P1 (JW2511)

SMR11994	SMR4562 Δ*argR762*::FRTKanFRT	SMR4562 X P1 (JW3206)
SMR11995	SMR4562 Δ*pgi721*::FRTKanFRT	SMR4562 X P1 (JW3985)
SMR11996	SMR4562 Δ*ubiH758*::FRTKanFRT	SMR4562 X P1 (JW2875)
SMR11997	SMR4562 Δ*metJ725*::FRTKanFRT	SMR4562 X P1 (JW3909)
SMR11998	SMR4562 Δ*cyaA751*::FRTKanFRT	SMR4562 X P1 (JW3778)
SMR11999	SMR4562 Δ*nuoH764*::FRTKanFRT	SMR4562 X P1 (JW2277)
SMR12000	SMR4562 Δ*ybjT770*::FRTKanFRT	SMR4562 X P1 (JW5116)
SMR12001	SMR4562 Δ*treR788*::FRTKanFRT	SMR4562 X P1 (JW4200)
SMR12002	SMR4562 Δ*fruR786*::FRTKanFRT	SMR4562 X P1 (JW0078)
SMR12003	SMR4562 Δ*lipB769*::FRTKanFRT	SMR4562 X P1 (JW5089)
SMR12004	SMR4562 Δ*znuC783*::FRTKanFRT	SMR4562 X P1 (JW1847)
SMR12005	SMR4562 Δ*argD767*::FRTKanFRT	SMR4562 X P1 (JW3322)
SMR12006	SMR4562 Δ*pstB756*::FRTKanFRT	SMR4562 X P1 (JW3703)
SMR12007	SMR4562 Δ*fre784*::FRTKanFRT	SMR4562 X P1 (JW3820)
SMR12008	SMR4562 Δ*tatC781*::FRTKanFRT	SMR4562 X P1 (JW3815)
SMR12023	SMR4562 *arcA*(P)::Tn*10*dCam	SMR4562 X P1 (SMR11410)
SMR12050	SMR4562 Δ*yqgE723*::FRTKanFRT	SMR4562 X P1 (JW2915)
SMR12056	SMR4562 Δ*yjcC755*::FRTKanFRT	SMR4562 X P1 (JW4022)
SMR12058	SMR4562 Δ*speE739*::FRTKanFRT	SMR4562 X P1 (JW0117)
SMR12060	SMR4562 Δ*mdh761*::FRTKanFRT	SMR4562 X P1 (JW3205)
SMR12062	SMR4562 Δ*nuoK761*::FRTKanFRT	SMR4562 X P1 (JW2274)
SMR12065	SMR4562 Δ*ubiE778*::FRTKanFRT	SMR4562 X P1 (JW5581)
SMR12067	SMR4562 Δ*apaG762*::FRTKanFRT	SMR4562 X P1 (JW0049)
SMR12069	SMR4562 Δ*dsbC744*::FRTKanFRT	SMR4562 X P1 (JW2861)
SMR12071	SMR4562 Δ*pstC758*::FRTKanFRT	SMR4562 X P1 (JW3705)
SMR12075	SMR4562 Δ*lon725*::FRTKanFRT	SMR4562 X P1 (JW0429)
SMR12081	SMR4562 Δ*znuA782*::FRTKanFRT	SMR4562 X P1 (JW5831)
SMR12130	SMR4562 Δ*nuoJ762*::FRTKanFRT	SMR4562 X P1 (JW2275)
SMR12134	SMR4562 *yhhK*::Tn*10*dCam	SMR4562 X P1 (SMR11482)
SMR12140	SMR4562 Δ*nuoC768*::FRTKanFRT	SMR4562 X P1 (JW5375)
SMR12148	SMR4562 Δ*cspC747*::FRTKanFRT	SMR4562 X P1 (JW1812)
SMR12205	SMR4562 Δ*srlA777*::FRTKanFRT	SMR4562 X P1 (JW5429)
SMR12210	SMR4562 Δ*rppH754*::FRT	SMR11974 X pCP20
SMR12211	SMR4562 Δ*znuC783*::FRT	SMR12004 X pCP20
SMR12216	SMR4562 Δ*yqiC737*::FRT	SMR11976 X pCP20
SMR12219	SMR4562 Δ*lipB769*::FRT	SMR12003 X pCP20
SMR12220	SMR4562 Δ*acrA748*::FRT	SMR11985 X pCP20
SMR12221	SMR4562 Δ*ybjT770*::FRT	SMR12000 X pCP20
SMR12223	SMR4562 Δ*nuoC768*::FRT	SMR12140 X pCP20
SMR12227	SMR4562 Δ*glnK736*::FRT	SMR11968 X pCP20
SMR12232	SMR4562 Δ*pstB756*::FRT	SMR12006 X pCP20
SMR12233	SMR4562 Δ*srlA777*::FRT	SMR12205 X pCP20
SMR12235	SMR4562Δ *hdfR785*::FRT	SMR11975 X pCP20
SMR12236	SMR4562 Δ*znuA782*::FRT	SMR12081 X pCP20
SMR12238	SMR4562 Δ*yifE786*::FRT	SMR11977 X pCP20
SMR12239	SMR4562 Δ*nuoL760*::FRT	SMR11988 X pCP20
SMR12240	SMR4562 Δ*speE739*::FRT	SMR12058 X pCP20
SMR12241	SMR4562 Δ*apaG762*::FRT	SMR12067 X pCP20
SMR12242	SMR4562 Δ*ubiE778*::FRT	SMR12065 X pCP20
SMR12251	SMR4562 Δ*prc755*::FRT	SMR11972 X pCP20
SMR12252	SMR4562 Δ*sdhD772*::FRT	SMR11989 X pCP20
SMR12253	SMR4562 Δ*barA784*::FRT	SMR11986 X pCP20
SMR12254	SMR4562 Δ*lon725*::FRT	SMR12075 X pCP20
SMR12257	SMR4562 Δ*pgl739*::FRT	SMR11991 X pCP20
SMR12260	SMR4562 Δ*ubiH758*::FRT	SMR11996 X pCP20
SMR12261	SMR4562 Δ*hfq722*::FRT	SMR11963 X pCP20
SMR12302	SMR4562 *cysD*(P)::Tn*10*dCam	SMR4562 X P1 (SMR11925)
SMR12307	SMR4562 Δ*hscB773*::FRT	SMR11993 X pCP20

SMR12309	SMR4562 Δ*dsbA723*::FRT	SMR11965 X pCP20
SMR12311	SMR4562 *ubiA*::Tn*10*dCam	SMR4562 X P1 (SMR11944)
SMR12313	SMR4562 *ubiD*::Tn*10*dCam	SMR4562 X P1 (SMR11945)
SMR12314	SMR4562 Δ*uvrY760*::FRT	SMR11987 X pCP20
SMR12316	SMR4562 Δ*ygfA763*::FRTKanFRT	SMR4562 X P1 (JW2879)
SMR12332	SMR4562 Δ*sdhB774*::FRTKanFRT	SMR4562 X P1 (JW0714)
SMR12338	SMR4562 Δ*cyoA789*::FRTKanFRT	SMR4562 X P1 (JW0422)
SMR12340	SMR4562 Δ*cyoD786*::FRTKanFRT	SMR4562 X P1 (JW0419)
SMR12562	SMR4562 Δ*nuoC768*::FRT *rssB*::Tet	SMR12223 X P1 (SMR7530)
SMR12566	SMR4562 *rssB*::Tet	SMR4562 X P1 (SMR7530)
SMR12570	SMR4562 Δ*cyoA789*::FRT	SMR12338 X pCP20
SMR12574	SMR4562 Δ*cyoD786*::FRT	SMR12340 X pCP20
SMR12580	SMR4562 Δ*arcA726*::FRTKanFRT	SMR4562 X P1 (JW4364)
SMR12582	SMR4562 Δ*arcB738*::FRTKanFRT	SMR4562 X P1 (JW5536)
SMR12584	SMR4562 Δ*rbsK781*::FRTKanFRT	SMR4562 X P1 (JW3731)
SMR12590	SMR4562 Δ*nuoC768*::FRT Δ*arcA726*::FRTKanFRT	SMR12223 X P1 (JW4364)
SMR12594	SMR4562 Δ*nuoC768*::FRT Δ*arcB738*::FRTKanFRT	SMR12223 X P1 (JW5536)
SMR12596	SMR4562 *ubiD*::Tn*10*dCam Δ*arcA726*::FRTKanFRT	SMR12313 X P1 (JW4364)
SMR12598	SMR4562 *ubiD*::Tn*10*dCam Δ*arcB738*::FRTKanFRT	SMR12313 X P1 (JW5536)
SMR12613	SMR4562 *ubiD*::Tn*10*dCam *rssB*::Tet	SMR12313 X P1 (SMR7530)
SMR12655	SMR4562 Δ*nuoC768*::FRT *yiaG-yfp* FRT*cat*FRT	SMR12223 X P1 (SMR10582)
SMR12657	SMR4562 Δ*ubiE778*::FRT *yiaG-yfp* FRT*cat*FRT	SMR12242 X P1 (SMR10582)
SMR12661	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*rpoS746*::FRTKanFRT	SMR10582 X P1 (JW5437)
SMR12672	SMR4562 Δ*arcA726*::FRT	SMR12580 X pCP20
SMR12673	SMR4562 Δ*arcB738*::FRT	SMR12582 X pCP20
SMR12683	SMR4562 Δ*arcA726*::FRT *yiaG-yfp* FRT*cat*FRT	SMR12672 X P1 (SMR10582)
SMR12684	SMR4562 Δ*arcB738*::FRT *yiaG-yfp* FRT*cat*FRT	SMR12673 X P1 (SMR10582)
SMR12686	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*ubiH758*::FRTKanFRT	SMR10582 X P1 (JW2875)
SMR12692	SMR4562 *yiaG-yfp* FRT*cat*FRT *rssB*::Tet	SMR10582 X P1 (SMR7530)
SMR12699	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*ubiX732*::FRTKanFRT	SMR10582 X P1 (JW2308)
SMR12828	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*speD738*::FRTKanFRT	SMR10582 X P1 (JW0116)
SMR12830	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*apaH761*::FRTKanFRT	SMR10582 X P1 (JW0048)
SMR13033	SMR4562 *yiaG-yfp* FRT*cat*FRT *sdhD772*::FRTKanFRT	SMR11989 X P1 (SMR10582)
SMR13035`	SMR4562 Δ*cyoD786*::FRTKanFRT *yiaG-yfp* FRT*cat*FRT	SMR12340 X P1 (SMR10582)
SMR13048	SMR4562 Δ*arcA726*::FRT Δ*cyoD786*::FRTKanFRT	SMR12672 X P1 (JW0419)
SMR13050	SMR4562 Δ*arcB738*::FRT Δ*cyoD786*::FRTKanFRT	SMR12673 X P1 (JW0419)
SMR13058	SMR4562 Δ*mdh761*::FRTKanFRT *yiaG-yfp* FRT*cat*FRT	SMR12060 X P1 (SMR10582)
SMR13084	SMR4562 Δ*cyoD786*::FRT *rssB*::Tet	SMR12574 X P1 (SMR7530)
SMR13096	SMR4562 *yiaG-yfp* FRT	SMR10582 X pCP20
SMR13098	SMR4562 *yiaG-yfp* FRT *ubiD*::Tn*10*dCam	SMR13096 X P1 (SMR11945)
SMR13274	FC40 day 5 Lac$^+$ isolate Δ*cyoD786*::FRTKanFRT	SMR3856 X P1 (JW0419)
SMR13275	FC40 day5 Lac$^+$ isolate Δ*cyoD786*::FRTKanFRT	SMR3858 X P1 (JW0419)
SMR13276	FC40 day 5 Lac$^+$ isolate Δ*cyoD786*::FRTKanFRT	SMR3859 X P1 (JW0419)
SMR13280	FC40 day 5 Lac$^+$ isolate Δ*ubiE778*::FRTKanFRT	SMR3856 X P1 (JW5581)
SMR13281	FC40 day 5 Lac$^+$ isolate Δ*ubiE778*::FRTKanFRT	SMR3858 X P1 (JW5581)
SMR13282	FC40 day 5 Lac$^+$ isolate Δ*ubiE778*::FRTKanFRT	SMR3859 X P1 (JW5581)
SMR13283	FC40 day 5 Lac$^+$ isolate *ubiD*::Tn*10*dCam	SMR3856 X P1 (SMR11945)
SMR13284	FC40 day 5 Lac$^+$ isolate *ubiD*::Tn*10*dCam	SMR3858 X P1 (SMR11945)
SMR13285	FC40 day 5 Lac$^+$ isolate *ubiD*::Tn*10*dCam	SMR3859 X P1 (SMR11945)
SMR13289	FC40 day 5 Lac$^+$ isolate Δ*mdh761*::FRTKanFRT	SMR3856 X P1 (JW3205)
SMR13290	FC40 day 5 Lac$^+$ isolate Δ*mdh761*::FRTKanFRT	SMR3858 X P1 (JW3205)
SMR13291	FC40 day 5 Lac$^+$ isolate Δ*mdh761*::FRTKanFRT	SMR3859 X P1 (JW3205)
SMR13292	FC40 day 5 Lac$^+$ isolate Δ*sdhD772*::FRTKanFRT	SMR3856 X P1 (JW0712)
SMR13293	FC40 day 5 Lac$^+$ isolate Δ*sdhD772*::FRTKanFRT	SMR3858 X P1 (JW0712)
SMR13294	FC40 day 5 Lac$^+$ isolate Δ*sdhD772*::FRTKanFRT	SMR3859 X P1 (JW0712)
SMR13295	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*pgi721*::FRTKanFRT	SMR10582 X P1 (JW3985)
SMR13301	SMR4562 day 5 Lac$^+$ isolate Δ*nuoC768*::FRTKanFRT	SMR3856 X P1 (JW5375)
SMR13302	SMR4562 day 5 Lac$^+$ isolate Δ*nuoC768*::FRTKanFRT	SMR3858 X P1 (JW5375)

SMR13303	SMR4562 day 5 Lac$^+$ isolate Δ*nuoC768*::FRTKanFRT	SMR3859 X P1 (JW5375)
SMR13310	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*speE739*::FRTKanFRT	SMR10582 X P1 (JW0117)
SMR13312	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*rbsK781*::FRTKanFRT	SMR10582 X P1 (JW3731)
SMR13448	SMR4562 Δ*araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10*	SMR6276 X P1 (SMR6541)
SMR13473	SMR4562 Δ*phoU755*::FRTKanFRT	SMR4562 X P1 (JW3702)
SMR13484	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*nuoC768*::FRTKanFRT	SMR6276 X P1 (JW5375)
SMR13487	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*cyoD786*::FRTKanFRT	SMR6276 X P1 (JW0419)
SMR13489	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*nuoC768*::FRTKanFRT	SMR13448 X P1 (JW5375)
SMR13491	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*cyoD786*::FRTKanFRT	SMR13448 X P1 (JW0419)
SMR13493	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*nuoC768*::FRT	SMR13484 X pCP20
SMR13494	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*cyoD786*::FRT	SMR13487 X pCP20
SMR13495	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*cyoD786*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR13494 X P1 (SMR6308)
SMR13497	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*nuoC768*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR13493 X P1 (SMR6308)
SMR13499	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* *nuoC768*::FRT	SMR13489 X pCP20
SMR13500	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*cyoD786*::FRT	SMR13491 X pCP20
SMR13510	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*mdh761*::FRTKanFRT	SMR6276 X P1 (JW3205)
SMR13630	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*nuoC768*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR13499 X P1 (SMR6308)
SMR13633	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*cyoD786*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR13500 X P1 (SMR6308)
SMR13635	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*mdh761*::FRT	SMR13510 X pCP20
SMR13640	SMR4562 *nuoG*::Tn*10*dCam	SMR4562 X P1 (SMR11956)
SMR13652	SMR4562 Δ*nuoJ762*::FRT	SMR12130 X pCP20
SMR13653	SMR4562 Δ*nuoK761*::FRT	SMR12062 X pCP20
SMR13654	SMR4562 Δ*nuoH764*::FRT	SMR11999 X pCP20
SMR13655	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*ubiE778*::FRTKanFRT	SMR13448 X P1 (JW5581)
SMR13657	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ubiE778*::FRTKanFRT	SMR6276 X P1 (JW5581)
SMR13659	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*ubiE778*::FRT	SMR13655 X pCP20
SMR13661	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ubiE778*::FRT	SM13657 X pCP20
SMR13663	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*ubiE778*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR13659 X P1 (SMR6308)
SMR13669	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ubiE778*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR13661 X P1 (SMR6308)
SMR14259	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*yifE786*::FRTKanFRT	SMR10582 X P1 (JW3737)
SMR14261	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*hdfR785*::FRTKanFRT	SMR10582 X P1 (JW5607)

SMR14263	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*cspC747*::FRTKanFRT	SMR10582 X P1 (JW1812)
SMR14267	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*fre784*::FRTKanFRT	SMR10582 X P1 (JW3820)
SMR14269	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*metJ725*::FRTKanFRT	SMR10582 X P1 (JW3909)
SMR14273	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*nuoL760*::FRTKanFRT	SMR10582 X P1 (JW2273)
SMR14275	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*hscB773*::FRTKanFRT	SMR10582 X P1 (JW2511)
SMR14281	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*glnK736*::FRTKanFRT	SMR10582 X P1 (JW0440)
SMR14283	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*cyoA789*::FRTKanFRT	SMR10582 X P1 (JW0422)
SMR14285	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*nuoK761*::FRTKanFRT	SMR10582 X P1 (JW2274)
SMR14287	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*nuoH764*::FRTKanFRT	SMR10582 X P1 (JW2277)
SMR14289	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*nuoJ762*::FRTKanFRT	SMR10582 X P1 (JW2275)
SMR14295	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*hfq722*::FRTKanFRT	SMR10582 X P1 (JW4130)
SMR14297	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*sdhB774*::FRTKanFRT	SMR10582 X P1 (JW0714)
SMR14420	SMR4562 *yiaG-yfp* FRT *nuoG*::Tn*10*dCam	SMR13096 X P1 (SMR11956)
SMR14431	SMR4562 *yiaG-yfp* FRT *hemL*::Tn*10*dCam	SMR13096 X P1 (SMR11478)
SMR14447	SMR4562 *yiaG-yfp* FRT *ubiA*::Tn*10*dCam	SMR13096 X P1 (SMR11944)
SMR14455	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*mdh761*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR13635 X P1 (SMR6308)
SMR14461	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*mdh761*::FRT *rpoS*::Tn*10*	SMR13635 X P1 (SMR6541)
SMR14467	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*mdh761*::FRT *rpoS*::Tn*10* [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR14461 X P1 (SMR6308)
SMR14471	MG1655 *galK*::P_{lac}*cfp* Amp	MG1655 X P1 (MG22)
SMR15080	MG1655 *galK*::P_{lac}*cfp* Amp Δ*cyoA789*::FRTKanFRT	SMR14471 X P1 (JW0422)
SMR15081	MG1655 *galK*::P_{lac}*cfp* Amp Δ*cyoD786*::FRTKanFRT	SMR14471 X P1 (JW0419)
SMR15082	MG1655 *galK*::P_{lac}*cfp* Amp Δ*cspC747*:FRTKanFRT	SMR14471 X P1 (JW1812)
SMR15083	MG1655 *galK*::P_{lac}*cfp* Amp Δ*fre784*::FRTKanFRT	SMR14471 X P1 (JW3820)
SMR15084	MG1655 *galK*::P_{lac}*cfp* Amp Δ*glnK736*::FRTKanFRT	SMR14471 X P1 (JW0440)
SMR15085	MG1655 *galK*::P_{lac}*cfp* Amp Δ*hdfR785*::FRTKanFRT	SMR14471 X P1 (JW5607)
SMR15086	MG1655 *galK*::P_{lac}*cfp* Amp Δ*hscB773*::FRTKanFRT	SMR14471 X P1 (JW2511)
SMR15087	MG1655 *galK*::P_{lac}*cfp* Amp Δ*mdh761*::FRTKanFRT	SMR14471 X P1 (JW3205)
SMR15088	MG1655 *galK*::P_{lac}*cfp* Amp Δ*metJ725*::FRTKanFRT	SMR14471 X P1 (JW3909)
SMR15089	MG1655 *galK*::P_{lac}*cfp* Amp Δ*nuoC768*::FRTKanFRT	SMR14471 X P1 (JW5375)
SMR15090	MG1655 *galK*::P_{lac}*cfp* Amp *nuoG*::Tn*10*dCam	SMR14471 X P1 (SMR11956)
SMR15091	MG1655 *galK*::P_{lac}*cfp* Amp Δ*nuoH764*::FRTKanFRT	SMR14471 X P1 (JW2277)
SMR15092	MG1655 *galK*::P_{lac}*cfp* Amp Δ*nuoJ762*::FRTKanFRT	SMR14471 X P1 (JW2275)
SMR15093	MG1655 *galK*::P_{lac}*cfp* Amp Δ*nuoK761*::FRTKanFRT	SMR14471 X P1 (JW2274)
SMR15094	MG1655 *galK*::P_{lac}*cfp* Amp Δ*nuoL760*::FRTKanFRT	SMR14471 X P1 (JW2273)
SMR15095	MG1655 *galK*::P_{lac}*cfp* Amp Δ*pgi721*::FRTKanFRT	SMR14471 X P1 (JW3985)
SMR15096	MG1655 *galK*::P_{lac}*cfp* Amp Δ*rbsK781*::FRTKanFRT	SMR14471 X P1 (JW3731)
SMR15097	MG1655 *galK*::P_{lac}*cfp* Amp Δ*rpoS746*::FRTKanFRT	SMR14471 X P1 (JW5437)
SMR15098	MG1655 *galK*::P_{lac}*cfp* Amp Δ*sdhB774*::FRTKanFRT	SMR14471 X P1 (JW0714)
SMR15099	MG1655 *galK*::P_{lac}*cfp* Amp Δ*sdhD772*::FRTKanFRT	SMR14471 X P1 (JW0712)
SMR15100	MG1655 *galK*::P_{lac}*cfp* Amp Δ*speD738*::FRTKanFRT	SMR14471 X P1 (JW0116)
SMR15101	MG1655 *galK*::P_{lac}*cfp* Amp Δ*speE739*::FRTKanFRT	SMR14471 X P1 (JW0117)
SMR15102	MG1655 *galK*::P_{lac}*cfp* Amp *ubiA*::Tn*10*dCam	SMR14471 X P1 (SMR11944)
SMR15103	MG1655 *galK*::P_{lac}*cfp* Amp *ubiD*::Tn*10*dCam	SMR14471 X P1 (SMR11945)
SMR15104	MG1655 *galK*::P_{lac}*cfp* Amp Δ*ubiE778*::FRTKanFRT	SMR14471 X P1 (JW5581)
SMR15105	MG1655 *galK*::P_{lac}*cfp* Amp Δ*ubiH758*::FRTKanFRT	SMR14471 X P1 (JW2875)
SMR15106	MG1655 *galK*::P_{lac}*cfp* Amp Δ*ubiX732*::FRTKanFRT	SMR14471 X P1 (JW2308)
SMR15107	MG1655 *galK*::P_{lac}*cfp* Amp Δ*yifE786*::FRTKanFRT	SMR14471 X P1 (JW3737)
SMR15108	MG1655 *galK*::P_{lac}*cfp* Amp *hemL*::Tn*10*dCam	SMR14471 X P1 (SMR11478)
SMR15109	MG1655 *galK*::P_{lac}*cfp* Amp Δ*hfq722*::FRTKanFRT	SMR14471 X P1 (JW4130)
SMR15249	CAG45114 Δ*nuoH764*::FRTKanFRT	CAG45114 X P1 (JW2277)
SMR15253	CAG45114 Δ*fruR786*::FRTKanFRT	CAG45114 X P1 (JW0078)
SMR15257	CAG45114 Δ*ubiE778*::FRTKanFRT	CAG45114 X P1 (JW5581)
SMR15259	CAG45114 Δ*lipB769*::FRTKanFRT	CAG45114 X P1 (JW5089)

SMR15261	CAG45114 Δ*sdhB774*::FRTKanFRT	CAG45114 X P1 (JW0714)
SMR15265	CAG45114 Δ*lrp787*::FRTKanFRT	CAG45114 X P1 (JW0872)
SMR15267	CAG45114 Δ*sdhD772*::FRTKanFRT	CAG45114 X P1 (JW0712)
SMR15271	CAG45114 *rssB*::Tet	CAG45114 X P1 (SMR7530)
SMR15273	CAG45114 Δ*lon725*::FRTKanFRT	CAG45114 X P1 (JW0429)
SMR15277	CAG45114 Δ*nuoC768*::RTKanFRT	CAG45114 X P1 (JW5375)
SMR15283	CAG45114 Δ*trxB786*::FRTKanFRT	CAG45114 X P1 (JW0871)
SMR15287	CAG45114 Δ*pstC758*::FRTKanFRT	CAG45114 X P1 (JW3705)
SMR15289	CAG45114 Δ*phoU755*::FRTKanFRT	CAG45114 X P1 (JW3702)
SMR15291	CAG45114 Δ*znuC783*::FRTKanFRT	CAG45114 X P1 (JW1847)
SMR15293	CAG45114 Δ*cyoD786*::FRTKanFRT	CAG45114 X P1 (JW0419)
SMR15295	CAG45114 Δ*ubiH758*::FRTKanFRT	CAG45114 X P1 (JW2875)
SMR15301	CAG45114 Δ*pstB756*::FRTKanFRT	CAG45114 X P1 (JW3703)
SMR15309	CAG45114 *yigP*::Tn*10*dCam	CAG45114 X P1 (SMR11475)
SMR15311	CAG45114 *rpoE2072*::Tn*10*dCam	CAG45114 X P1 (SMR5236)
SMR15315	CAG45114 Δ*rppH754*::FRTKanFRT	CAG45114 X P1 (JW2798)
SMR15317	CAG45114 Δ*ubiX732*::FRTKanFRT	CAG45114 X P1 (JW2308)
SMR15319	CAG45114 *hemL*::Tn*10*dCam	CAG45114 X P1 (SMR11478)
SMR15321	CAG45114 *ubiA*::Tn*10*dCam	CAG45114 X P1 (SMR11944)
SMR15323	CAG45114 *ubiD*::Tn*10*dCam	CAG45114 X P1 (SMR11945)
SMR15326	CAG45114 Δ*ygfA763*::FRTKanFRT	CAG45114 X P1 (JW2879)
SMR15328	CAG45114 Δ*apaH761*::FRTKanFRT	CAG45114 X P1 (JW0048)
SMR15330	CAG45114 Δ*cyoA789*::FRTKanFRT	CAG45114 X P1 (JW0422)
SMR15354	CAG45114 Δ*nuoL760*::FRTKanFRT	CAG45114 X P1 (JW2273)
SMR15356	CAG45114 Δ*mdh761*::FRTKanFRT	CAG45114 X P1 (JW3205)
SMR15358	CAG45114 Δ*nuoJ762*::FRTKanFRT	CAG45114 X P1 (JW2275)
SMR15360	CAG45114 Δ*arcA726*::FRTKanFRT	CAG45114 X P1 (JW4364)
SMR15362	CAG45114 Δ*fre784*::FRTKanFRT	CAG45114 X P1 (JW3820)
SMR15364	CAG45114 Δ*arcB738*::FRTKanFRT	CAG45114 X P1 (JW5536)
SMR15366	CAG45114 Δ*nuoK761*::FRTKanFRT	CAG45114 X P1 (JW2274)
SMR15754	CAG45114 Δ*rseA*::FRTKanFRT	CAG45114 X P1 (JW2556)
SMR15962	SMR6039 Δ*pgi721*::FRTKanFRT	SMR6039 X P1 (JW3985)
SMR15980	SMR6039 Δ*uvrY760*::FRTKanFRT	SMR6039 X P1 (JW1899)
SMR16024	SMR6039 Δ*recB*::Kan	SMR6039 X P1 (RTC0013)
SMR16032	MG1655 *galK*::P_{lac}*cfp* Amp Δ*apaH761*::FRTKanFRT	SMR14471 X P1 (JW0048)
SMR16038	CAG45114 *nuoG*::Tn*10*dCam	CAG45114 X P1 (SMR11956)
SMR16093	SMR6039 Δ*recF1804*::FRTKanFRT	SMR6039 X P1 (SMR8097)
SMR17043	SMR6039 *recC*::Tn*10*dCam	SMR6039 X P1 (SMR11941)
SMR17173	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*speE739*::FRTKanFRT	SMR10774 X P1 (JW0117)
SMR17174	SMR17173 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17173 X P1 (SMR10800)
SMR17175	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*fruR786*::FRTKanFRT	SMR10774 X P1 (JW0078)
SMR17176	SMR17175 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17175 X P1 (SMR10800)
SMR17177	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yifE786*::FRTKanFRT	SMR10774 X P1 (JW3737)
SMR17178	SMR17177 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17177 X P1 (SMR10800)
SMR17179	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yqgE723*::FRTKanFRT	SMR10774 X P1 (JW2915)
SMR17180	SMR17179 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17179 X P1 (SMR10800)
SMR17181	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yqiC737*::FRTKanFRT	SMR10774 X P1 (JW5505)
SMR17182	SMR17181 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17181 X P1 (SMR10800)

SMR17183	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*speD738*::FRTKanFRT	SMR10774 X P1 (JW0116)
SMR17184	SMR17183 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17183 X P1 (SMR10800)
SMR17185	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*nuoL760*::FRTKanFRT	SMR10774 X P1 (JW2273)
SMR17186	SMR17185 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17185 X P1 (SMR10800)
SMR17187	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*rbsK781*::FRTKanFRT	SMR10774 X P1 (JW3731)
SMR17188	SMR17187 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17187 X P1 (SMR10800)
SMR17189	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*pgi721*::FRTKanFRT	SMR10774 X P1 (JW3985)
SMR17190	SMR17189 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17189 X P1 (SMR10800)
SMR17191	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*cspC747*::FRTKanFRT	SMR10774 X P1 (JW1812)
SMR17192	SMR17191 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17191 X P1 (SMR10800)
SMR17193	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*apaH761*::FRTKanFRT	SMR10774 X P1 (JW0048)
SMR17194	SMR17193 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17193 X P1 (SMR10800)
SMR17195	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*metJ725*::FRTKanFRT	SMR10774 X P1 (JW3909)
SMR17196	SMR17195 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17195 X P1 (SMR10800)
SMR17197	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ubiH758*::FRTKanFRT	SMR10774 X P1 (JW2875)
SMR17198	SMR17197 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17197 X P1 (SMR10800)
SMR17199	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*rppH754*::FRTKanFRT	SMR10774 X P1 (JW2798)
SMR17200	SMR17199 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17199 X P1 (SMR10800)
SMR17201	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hscB773*::FRTKanFRT	SMR10774 X P1 (JW2511)
SMR17202	SMR17201 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17201 X P1 (SMR10800)
SMR17205	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yjcC755*::FRTKanFRT	SMR10774 X P1 (JW4022)
SMR17206	SMR17205 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17205 X P1 (SMR10800)
SMR17207	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*barA784*::FRTKanFRT	SMR10774 X P1 (JW2757)
SMR17208	SMR17207 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17207X P1 (SMR10800)
SMR17209	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*trxB786*::FRTKanFRT	SMR10774 X P1 (JW0871)
SMR17210	SMR17209 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17209X P1 (SMR10800)
SMR17211	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*lon725*::FRTKanFRT	SMR10774 X P1 (JW0429)
SMR17212	SMR17211 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17211X P1 (SMR10800)
SMR17213	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*argR762*::FRTKanFRT	SMR10774 X P1 (JW3206)
SMR17214	SMR17213Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17213X P1 (SMR10800)
SMR17215	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I	SMR10774 X P1 (JW4130)

	Δ*hfq722*::FRTKanFRT	
SMR17216	SMR17215 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17215X P1 (SMR10800)
SMR17217	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*fre784*::FRTKanFRT	SMR10774 X P1 (JW3820)
SMR17218	SMR17217 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17217X P1 (SMR10800)
SMR17219	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yicC750*::FRTKanFRT	SMR10774 X P1 (JW3619)
SMR17220	SMR17219 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17219X P1 (SMR10800)
SMR17221	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ubiE778*::FRTKanFRT	SMR10774 X P1 (JW5581)
SMR17222	SMR17221 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17221X P1 (SMR10800)
SMR17225	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*lipB769*::FRTKanFRT	SMR10774 X P1 (JW5089)
SMR17226	SMR17225 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17225X P1 (SMR10800)
SMR17227	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ldcA782*::FRTKanFRT	SMR10774 X P1 (JW1181)
SMR17228	SMR17227 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17227X P1 (SMR10800)
SMR17229	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*tatC781*::FRTKanFRT	SMR10774 X P1 (JW3815)
SMR17230	SMR17229 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17229X P1 (SMR10800)
SMR17233	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*malI768*::FRTKanFRT	SMR10774 X P1 (JW1612)
SMR17235	FC36 Δ*araBAD*567 Δ*attλ*::P_{BAD}I-*Sce*I Δ*ybjT770*::FRTKanFRT	SMR10774 X P1 (JW5116)
SMR17234	SMR17233 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17233X P1 (SMR10800)
SMR17237	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*mdh761*::FRTKanFRT	SMR10774 X P1 (JW3205)
SMR17238	SMR17237 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17237X P1 (SMR10800)
SMR17239	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ygfA763*::FRTKanFRT	SMR10774 X P1 (JW2879)
SMR17240	SMR17239 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17239X P1 (SMR10800)
SMR17241	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hdfR785*::FRTKanFRT	SMR10774 X P1 (JW5607)
SMR17242	SMR17241 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17241X P1 (SMR10800)
SMR17243	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*glnK736*::FRTKanFRT	SMR10774 X P1 (JW0440)
SMR17244	SMR17243 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17243 X P1 (SMR10800)
SMR17245	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*nuoC768*::FRTKanFRT	SMR10774 X P1 (JW5375)
SMR17246	SMR17245 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17245 X P1 (SMR10800)
SMR17247	FC36 Δ*araBAD*567 Δ*attλ*::P_{BAD}I-*Sce*I *yfjL*(P)::Tn*10*dCam	SMR10774 X P1 (SMR11472)
SMR17249	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *yigP*::Tn*10*dCam	SMR10774 X P1 (SMR11475)
SMR17250	SMR17249 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17249X P1 (SMR10800)
SMR17253	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *ytjB*(P)::Tn*10*dCam	SMR10774 X P1 (SMR11476)
SMR17254	SMR17253 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17253 X P1 (SMR10800)

SMR17255	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *hemL*::Tn*10*dCam	SMR10774 X P1 (SMR11478)
SMR17256	SMR17255 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17255 X P1 (SMR10800)
SMR17257	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *proL*::Tn*10*dCam	SMR10774 X P1 (SMR11479)
SMR17258	SMR17257 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17257 X P1 (SMR10800)
SMR17259	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*pgi721*::FRTKanFRT	SMR6276 X P1 (JW3985)
SMR17261	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*fre784*::FRTKanFRT	SMR6276 X P1 (JW3820)
SMR17262	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*pgi721*::FRTKanFRT	SMR13448 X P1 (JW3985)
SMR17264	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*fre784*::FRTKanFRT	SMR13448 X P1 (JW3820)
SMR17265	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*barA784*::FRTKanFRT	SMR10582 X P1 (JW2757)
SMR17267	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*uvrY760*::FRTKanFRT	SMR10582 X P1 (JW1899)
SMR17270	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hscB773*::FRTKanFRT	SMR6276 X P1 (JW2511)
SMR17272	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*hscB773*::FRTKanFRT	SMR13448 X P1 (JW2511)
SMR17274	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*pgi721*::FRT	SMR17259 X pCP20
SMR17275	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*pgi721*::FRT	SMR17262 X pCP20
SMR17276	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*fre784*::FRT	SMR17261 X pCP20
SMR17277	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*fre784*::FRT	SMR17264 X pCP20
SMR17282	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *recA*::Tn*10*dCam	SMR6276 X P1 (SMR4610)
SMR17286	SMR4562 *yiaG-yfp* FRT*cat*FRT Δ*crp765*::FRTKanFRT	SMR0582 X P1 (JW3320)
SMR17290	SMR4562 Δ*crp765*::FRTKanFRT	SMR4562 X P1 (JW3320)
SMR17291	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*] *hemL*::Tn*10*dCam	SMR6038 X P1 (SMR11478)
SMR17293	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*] *hemL*::Tn*10*dCam	SMR11437 X P1 (SMR11478)
SMR17295	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hscB773*::FRT	SMR17270 X pCP20
SMR17296	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*hscB773*::FRT	SMR17272 X pCP20
SMR17297	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*fre784*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR17276 X P1 (SMR6308)
SMR17299	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*fre784*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR17277 X P1 (SMR6308)
SMR17301	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *sdhD*::Tn*10*dCam [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR6308 X P1 (SMR11530)
SMR17370	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*pgi721*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR17274 X P1 (SMR6308)
SMR17372	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*pgi721*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR17275 X P1 (SMR6308)
SMR17374	SMR4562 Δ*ara araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hscB773*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*]	SMR17295 X P1 (SMR6308)
SMR17376	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*hscB773*::FRT [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site)	SMR17296 X P1 (SMR6308)

	Δ*traI*::*dhfR*]	
SMR17385	SMR4562 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* [F' *codA21*::mini-Tn*7*Kan(I-*Sce*I site) Δ*traI*::*dhfR*] *sdhD*::Tn*10*dCam	SMR11437 X P1 (SMR11530)
SMR17506	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *ppk*::Tn*10*dCam	SMR10774 X P1 (SMR11955)
SMR17507	SMR17506 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17506 X P1 (SMR10800)
SMR17508	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *groES*(P)::Tn*10*dCam	SMR10774 X P1 (SMR11934)
SMR17509	SMR17508 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17508 X P1 (SMR10800)
SMR17520	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ihfA786*::FRTKanFRT	SMR10774 X P1 (JW1702)
SMR17521	SMR17520 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17520 X P1 (SMR10800)
SMR17526	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*acrA748*::FRTKanFRT	SMR10774 X P1 (JW0452)
SMR17527	SMR17526 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17526 X P1 (SMR10800)
SMR17528	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*acrB747*::FRTKanFRT	SMR10774 X P1 (JW0451)
SMR17529	SMR17528 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17528 X P1 (SMR10800)
SMR17534	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*arcA726*::FRTKanFRT	SMR10774 X P1 (JW4364)
SMR17535	SMR17534 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17534 X P1 (SMR10800)
SMR17536	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*lrp787*::FRTKanFRT	SMR10774 X P1 (JW0872)
SMR17537	SMR17536 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17536 X P1 (SMR10800)
SMR17538	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*phoU755*::FRTKanFRT	SMR10774 X P1 (JW3702)
SMR17539	SMR17538 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17538 X P1 (SMR10800)
SMR17540	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*pstB756*::FRTKanFRT	SMR10774 X P1 (JW3703)
SMR17541	SMR17540 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17540 X P1 (SMR10800)
SMR17542	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*pstC758*::FRTKanFRT	SMR10774 X P1 (JW3705)
SMR17543	SMR17542 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17542 X P1 (SMR10800)
SMR17544	FC36 Δ *araBAD567*Δ*attλ*::P_{BAD}I-*Sce*I Δ*znuC783*::FRTKanFRT	SMR10774 X P1 (JW1847)
SMR17545	SMR17544 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17544 X P1 (SMR10800)
SMR17546	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *cysD*(P)::Tn*10*dCam	SMR10774 X P1 (SMR11925)
SMR17547	SMR17546 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17546 X P1 (SMR10800)
SMR17551	FC36 Δ *araBAD567*Δ*attλ*::P_{BAD}I-*Sce*I Δ*sdhB774*::FRTKanFRT	SMR10774 X P1 (JW0714)
SMR17552	SMR17551 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17551 X P1 (SMR10800)
SMR17554	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*znuA782*::FRTKanFRT	SMR10774 X P1 (JW5831)
SMR17555	SMR17554 Δ*zie3913.1*::*tetRtetA+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17554 X P1 (SMR10800)
SMR17562	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*treR788*::FRTKanFRT	SMR10774 X P1 (JW4200)

SMR17568	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*prc755*::FRTKanFRT	SMR10774 X P1 (JW1819)
SMR17578	FC36 Δ *araBAD567*Δ*attλ*::P_{BAD}I-*Sce*I Δ*dsbA723*::FRTKanFRT	SMR10774 X P1 (JW3832)
SMR17599	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*cyoA789*::FRTKanFRT	SMR10774 X P1 (JW0422)
SMR17601	SMR17599 Δ*zie3913.1*::*tetR*tet*A+1*FRT*cat*FRT Δ*zie3920.5*::3ChiKanI*Sce*Isite	SMR17599 X P1 (SMR10800)
SMR18004	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *lexA3*(Ind$^-$) *malB*::*Tn*9	SMR10774 X P1 (SMR820)
SMR18010	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*dinB50*::FRTKanFRT	SMR10774 X P1 (SMR5889)
SMR18013	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10*	SMR10774 X P1 (SMR6541)
SMR18018	SMR5833 Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR5833 x Short homology from pKD3 using primer P531 and P533
SMR18020	SMR5833 Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR5833 x Short homology from pKD4 using primer P531 and P533)
SMR18022	SMR10772 Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR10772 X P1 (SMR18020)
SMR18024	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR10774 X P1 (SMR18020)
SMR18025	SMR10772 Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR10772 X P1 (SMR18018)
SMR18026	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR10774 X P1 (SMR18018)
SMR18034	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*umuDC*::*cat*	SMR10774 X P1 (SMR3525)
SMR18038	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*cyoA789*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17599 X P1 (SMR18018)
SMR18040	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*pgi721*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17189 X P1 (SMR18018)
SMR18042	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*lrp787*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17536 X P1 (SMR18018)
SMR18043	FC36 Δ *araBAD567*Δ*attλ*::P_{BAD}I-*Sce*I Δ*znuC783*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17544 X P1 (SMR18018)
SMR18044	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*metJ725*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17195 X P1 (SMR18018)
SMR18045	FC36 Δ *araBAD567*Δ*attλ*::P_{BAD}I-*Sce*I Δ*sdhB774*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17551 X P1 (SMR18018)
SMR18046	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*prc755*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17568 X P1 (SMR18018)
SMR18053	FC36 Δ *araBAD567*Δ*attλ*::P_{BAD}I-*Sce*I Δ*dsbA723*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17578 X P1 (SMR18018)
SMR18054	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*arcA726*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17534 X P1 (SMR18018)
SMR18055	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*treR788*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17562 X P1 (SMR18018)
SMR18056	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ihfA786*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17520 X P1 (SMR18018)
SMR18061	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hscB773*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17201 X P1 (SMR18018)
SMR18062	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*rppH754*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17199 X P1 (SMR18018)

SMR18064	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*trxB786*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17209 X P1 (SMR18018)
SMR18065	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*speE739*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17173X P1 (SMR18018)
SMR18066	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hfq722*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17215 X P1 (SMR18018)
SMR18067	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yicC750*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17219 X P1 (SMR18018)
SMR18068	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*nuoC768*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17245 X P1 (SMR18018)
SMR18069	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*barA784*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17207 X P1 (SMR18018)
SMR18070	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*hdfR785*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17241 X P1 (SMR18018)
SMR18071	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*glnK736*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17243 X P1 (SMR18018)
SMR18072	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yjcC755*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17205 X P1 (SMR18018)
SMR18073	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yqiC737*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17181 X P1 (SMR18018)
SMR18074	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*cspC747*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17191 X P1 (SMR18018)
SMR18075	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*argR762*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17213 X P1 (SMR18018)
SMR18076	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*speD738*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17183 X P1 (SMR18018)
SMR18077	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*yifE786*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17177 X P1 (SMR18018)
SMR18078	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*rbsK781*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17187 X P1 (SMR18018)
SMR18079	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *yigP*::Tn*10*dCam Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR17249 X P1 (SMR18020)
SMR18080	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ldcA782*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17227 X P1 (SMR18018)
SMR18081	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*malI768*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17233 X P1 (SMR18108)
SMR18082	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*ygfA763*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17239 X P1 (SMR18108)
SMR18083	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*lon725*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17211 X P1 (SMR18108)
SMR18084	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*tatC781*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17229 X P1 (SMR18108)
SMR18085	FC36 Δ*araBAD*567 Δ*attλ*::P_{BAD}I-*Sce*I Δ*ybjT770*::FRTKanFRT	SMR17235 X P1 (SMR18018)

	Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	
SMR18087	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*fre784*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17217 X P1 (SMR18018)
SMR18088	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*mdh761*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17237 X P1 (SMR18018)
SMR18089	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*apaH761*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR17193 X P1 (SMR18018)
SMR18092	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*dinB50*::FRTKanFRT Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR18010 X P1 (SMR18018)
SMR18093	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *ytjB*(P)::Tn*10*dCam Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR17253 X P1 (SMR18020)
SMR18094	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*umuDC*::*cat* Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR18034 X P1 (SMR18020)
SMR18095	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *lexA3*(Ind$^-$) *malB*::*Tn*9 Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR18004 X P1 (SMR18020)
SMR18096	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *groES*(P)::Tn*10*dCam Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR17508 X P1 (SMR18020)
SMR18097	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *proL*::Tn*10*dCam Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR17257 X P1 (SMR18020)
SMR18098	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *cysD*(P)::Tn*10*dCam Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR17546 X P1 (SMR18020)
SMR18100	FC36 Δ*araBAD*567 Δ*attλ*::P_{BAD}I-*Sce*I *yfjL*(P)::Tn*10*dCam Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite	SMR17247 X P1 (SMR18020)
SMR18102	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite *recA*::Tn*10*dCam	SMR18024 X P1 (SMR4610)
SMR18103	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I *rpoS*::Tn*10* Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite	SMR18013 X P1 (SMR18018)
SMR18105	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRTKanFRTI*Sce*Isite *ppk*::Tn*10*dCam	SMR18024 X P1 (SMR11955)
SMR18108	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*ubiE778*::FRTKanFRT	SMR18026 X P1 (JW5581)
SMR18109	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*recB745*::FRTKanFRT	SMR18026 X P1 (JW2788)
SMR18110	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*yqgE763*::FRTKanFRT	SMR18026 X P1 (JW2915)
SMR18111	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*fruR786*::FRTKanFRT	SMR18026 X P1 (JW0078)
SMR18113	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*acrB747*::FRTKanFRT	SMR18026 X P1 (JW0451)
SMR18114	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*lrp787*::FRTKanFRT	SMR18026 X P1 (JW0872)
SMR18115	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*acrA748*::FRTKanFRT	SMR18026 X P1 (JW0452)
SMR18116	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*pstB756*::FRTKanFRT	SMR18026 X P1 (JW3703)
SMR18117	FC36 Δ *araBAD567* Δ*attλ*::P_{BAD}I-*Sce*I Δ*zfa2334.5*::FRT*cat*FRTI*Sce*Isite Δ*ruvC789*::FRTKanFRT	SMR18026 X P1 (JW1852)
ZK1268	*rpoS*::Tn*10*	(*133*)
Plasmid		
pCP20	Yeast Flp recombinase on a temperature-sensitive replicon λ*cIts857*	(*30*)

pKD3	Source of FRT*cat*FRT	(*30*)
pKD4	Source of FRTKanFRT	(*30*)
pKD46	*ori101 repA101ts* P_{BAD}*-gam-bet-exo AmpR*	(*30*)

Table S5. Validation of stress-induced mutagenesis defects of SIM network mutants in chromosomal Tet frameshift and Nal^R base-substitution forward-mutation assays

Mutant frequencies are Tet^R cfu / total cfu or Nal^R cfu / total cfu, with three independent cultures per experiment [Methods, per (*2*)], and the means are for ≥3 independent experiments. Fold decrease in mutant frequency is the mutant frequency of the WT-DSB (control) divided by the frequency from the mutant DSB strain assayed in parallel, then averaged for ≥3 independent experiments. *P* values are for differences from the WT DSB control strain per Methods. Most mutants identified in the Lac assay primary screen were confirmed in Tet (43 of 52 tested) and Nal (41 of 50) assays in which their defects are quantitatively similar (Tables S1, S6). Nine mutants, the names of which are annotated*, were not confirmed for both assays. All but two of these 9, *yqiC* and *ldcA*, are SDS-EDTA sensitive (Table S7), indicating membrane stress and possible membrane-stress (σ^E)-response defects, suggesting that these proteins promote SIM by promoting the σ^E response, which promotes mutagenesis by promoting spontaneous DNA breaks and is superfluous when DSBs are provided by I-*Sce*I (*8*), as is the case in the Tet assay (*2*). Also, *yqiC* encodes a putative membrane protein (Table S2) and *ldcA* encodes L,D-carboxypeptidase that has an essential function in murein (peptidoglycan) recycling (*52*), and so might also be defective in inducing the σ^E- membrane-protein stress response under the conditions of our SIM assay. Thus, proteins that promote mutation *via* activation of the σ^E response are expected to be superfluous in this assay. Thus, the overwhelming majority of proteins tested are confirmed. Seven mutants of genes previously known to be central to SIM (*3*) were used as controls, and also show decrease in stress-induced Nal^R base-substitution mutation in compared with WT. NT, not tested.

Mutant Gene	Strain	Mean mutant frequency (Tet^R cfu/10^8 cfu) ± SEM	Mean fold decrease in mutant frequency of mutants (WT/mutant) ± SEM	*P* value	Strain	Mean mutant frequency (Nal^R cfu/10^8 cfu) ± SEM	Mean fold decrease in mutant frequency of mutants (WT/mutant) ± SEM	*P* value
No DSB	SMR10799	0.26 ± 0.07	50 ± 20	1 X 10^{-9}	SMR18025	0.22 ± 0.02	16 ± 3.5	0.007
DSB	SMR10800	10 ± 2			SMR18026	3.6 ± 0.9		
Regulators								
arcA	SMR17535	0.4 ± 0.1	30 ± 17	0.004	SMR18054	0.98 ± 0.2	2.4 ± 0.6	0.046
argR	SMR17214	3.4 ± 0.5	3.8 ± 0.8	0.053	SMR18075	0.65 ± 0.2	5.1 ± 1	0.033
barA	SMR17208	2.8 ± 0.8	5 ± 1	0.029	SMR18069	0.54 ± 0.3	6.2 ± 2.9	0.028
*fruR**	SMR17176	17 ± 3	0.7 ± 0.1	0.325	SMR18111	2.5 ± 0.4	1.6 ± 0.3	0.307
glnK	SMR17244	2.2 ± 0.6	7 ± 3	0.034	SMR18071	0.7 ± 0.2	3.9 ± 1.7	0.034
hdfR	SMR17242	2.5 ± 0.6	6 ± 2	0.037	SMR18070	0.9 ± 0.5	6 ± 2	0.042
ihfA	SMR17521	1 ± 0.7	23 ± 9	0.005	SMR18056	0.6 ± 0.05	5.5 ± 2.3	0.032
lrp	SMR17537	6 ± 3	2.6 ± 0.8	0.027	SMR18042	1.1 ± 0.03	2.9 ± 1	0.05
malI	SMR17234	3 ± 1	6.3 ± 3.7	0.05	SMR18081	0.94 ± 0.2	4.5 ± 1.7	0.044

metJ	SMR17196	5.9 ± 0.9	2.4 ± 0.7	0.02	SMR18044	0.97 ± 0.04	2.2 ± 0.2	0.047
phoU	SMR17539	4.1 ± 0.9	2.8 ± 0.7	0.029	NT			
*ppk**	SMR17507	14 ± 5	0.9 ± 0.4	0.342	SMR18105	1.4 ± 0.7	2.5 ± 0.8	0.127
treR	NT				SMR18055	0.9 ± 0.2	2.8 ± 1	0.043
Proteases and chaperones								
cspC	SMR17192	1.9 ± 0.3	5.7 ± 0.6	0.017	SMR18074	0.76 ± 0.5	7.7 ± 2.3	0.04
*groES**	SMR17509	8 ± 1	1.2 ± 0.1	0.539	SMR18096	1.7 ± 0.6	2.6 ± 0.9	0.141
hfq	SMR17216	0.4 ± 0.2	45 ± 16	0.006	SMR18066	1 ± 0.06	3.1 ± 1	0.049
*ldcA**	SMR17228	26 ± 0.1	0.36 ± 0.07	0.0006	SMR18080	1.9 ± 0.3	2.6 ± 0.9	0.625
lon	SMR17212	2.6 ± 0.4	5 ± 2	0.024	SMR18083	0.45 ± 0.1	11.2 ± 7.3	0.027
prc	NT				SMR18046	0.94 ± 0.1	3.4 ± 0.9	0.045
Electron transfer								
cyoA	SMR17601	5.5 ± 1	2.5 ± 0.8	0.017	SMR18038	0.92 ± 0.5	4.6 ± 1.2	0.045
fre	SMR17218	1.5 ± 0.1	8 ± 2	0.01	SMR18087	0.33 ± 0.1	10.5 ± 2.3	0.024
hemL	SMR17256	1.9 ± 0.3	5 ± 1	0.009	NT			
hscB	SMR17202	2 ± 1	7 ± 1	0.022	SMR18061	1 ± 0.3	2.6 ± 1.1	0.049
mdh	SMR17238	0.2 ± 0.1	64 ± 16	0.006	SMR18088	0.98 ± 0.5	8.1 ± 4	0.046
nuoC	SMR17246	2.1 ± 0.5	6 ± 2	0.013	SMR18068	0.7 ± 0.1	4.8 ± 1.5	0.034
nuoL	SMR17186	4.1 ± 0.5	2.7 ± 0.8	0.05	NT			
pgi	SMR17190	6 ± 0.8	2.6 ± 0.6	0.039	SMR18040	0.9 ± 0.2	2.6 ± 0.7	0.043
sdhB	SMR17552	5 ± 1	1.8 ± 0.3	0.038	SMR18045	0.89 ± 0.2	3.9 ± 0.8	0.042
ubiE	SMR17222	0.9 ± 0.2	13 ± 6	0.005	SMR18108	0.2 ± 0.05	22 ± 7	0.021
ubiH	SMR17198	1.1 ± 0.4	11 ± 4	0.013	NT			
Metabolism								
apaH	SMR17194	3.9 ± 0.3	2.8 ± 0.6	0.045	SMR18089	1 ± 0.1	2.2 ± 0.6	0.048
*cysD**	SMR17547	5 ± 1	1.9 ± 0.2	0.069	SMR18098	1.8 ± 0.4	2.6 ± 1.1	0.198
lipB	SMR17226	3 ± 1	4 ± 2	0.008	NT			
rbsK	SMR17188	0.85 ± 0.3	9 ± 3	0.039	SMR18078	0.85 ± 0.3	3.9 ± 0.2	0.039
speD	SMR17184	0.84 ± 0.3	12 ± 6	0.046	SMR18076	0.84 ± 0.3	3.5 ± 0.9	0.046
speE	SMR17174	0.7 ± 0.2	12 ± 2	0.033	SMR18065	0.7 ± 0.2	3.8 ± 1	0.033
Cellular processes								
acrA	SMR17527	2.2 ± 0.6	7 ± 3	0.002	SMR18115	0.3 ± 0.1	13 ± 4.2	0.023
acrB	SMR17529	1 ± 0.2	13 ± 3	0.001	SMR18113	0.4 ± 0.08	11.8 ± 6.6	0.025
dsbA	NT				SMR18053	1 ± 0.06	3.2 ± 1	0.049
proL	SMR17258	2.9 ± 0.8	5 ± 1	0.044	SMR18097	1.1 ± 0.2	4.2 ± 1.2	0.05

pstB	SMR17541	4 ± 1	2.4 ± 0.3	0.048	SMR18116	1.1 ± 0.01	3 ± 1.1	0.05
pstC	SMR17543	4.1 ± 0.8	2.4 ± 0.4	0.04	NT			
*rppH**	SMR17200	14 ± 3	0.9 ± 0.1	0.749	SMR18062	1.8 ± 0.6	2.5 ± 0.8	0.176
*tatC**	SMR17230	20 ± 0.1	0.5 ± 0.1	0.003	SMR18084	3.3 ± 1.4	2.1 ± 1	0.879
*trxB**	SMR17210	7 ± 2	1.3 ± 0.3	0.332	SMR18064	2.1 ± 0.1	1.4 ± 0.4	0.18
znuA	SMR17555	2.4 ± 0.4	4.1 ± 0.8	0.012	NT			
znuC	SMR17545	2.2 ± 0.5	5 ± 1	0.011	SMR18043	0.98 ± 0.3	4.7 ± 1.8	0.046
Unknown function								
ybjT	NT				SMR18085	1 ± 0.2	3.2 ± 0.2	0.047
yfjL	NT				SMR18100	1 ± 0.3	5.2 ± 1.5	0.046
ygfA	SMR17240	1.6 ± 0.2	9 ± 3	0.027	SMR18082	1 ± 0.2	5.4 ± 2.1	0.046
yicC	SMR17220	4.4 ± 0.4	2.6 ± 0.5	0.039	SMR18067	0.8 ± 0.4	3.7 ± 1.4	0.038
yjcC	SMR17206	0.5 ± 0.1	22 ± 3	0.009	SMR18072	0.8 ± 0.3	3.4 ± 1.1	0.036
yifE	SMR17178	4 ± 1	4 ± 1	0.044	SMR18077	0.45 ± 0.1	9.6 ± 5.3	0.027
yigP	SMR17250	2.8 ± 0.8	4 ± 2	0.019	SMR18079	0.71 ± 0.1	4.5 ± 0.9	0.035
yqgE	SMR17180	1.6 ± 0.5	8 ± 2	0.016	SMR18110	0.6 ± 0.04	5 ± 1.8	0.033
*yqiC**	SMR17182	16 ± 4	0.8 ± 0.2	0.506	SMR18073	2.3 ± 0.9	1.4 ± 0.2	0.359
ytjB	SMR17254	1 ± 0.3	16 ± 8	0.008	SMR18093	0.6 ± 0.2	7.9 ± 3.3	0.029
Previously Known								
dinB					SMR18092	1 ± 0.3	4.4 ± 1.3	0.05
lexA					SMR18095	0.2 ± 0.1	20 ± 5	0.021
recA					SMR18102	0.13 ± 0.05	35 ± 15	0.02
recB					SMR18109	0.1 ± 0.04	32 ± 10	0.02
rpoS					SMR18103	0.9 ± 0.3	4.5 ± 1.7	0.043
ruvC					SMR18117	0.04 ± 0.01	121 ± 52	0.019
umuC					SMR18094	0.9 ± 0.4	5.6 ± 1.3	0.041

Table S6. Correlations of the magnitudes of SIM-deficiency in four stress-induced mutation assays

Correlation analysis for the fold difference between the wild-type (WT) control and each SIM-down mutant among the four mutation assays employed (summarized, Table S1), for genes assayed that displayed mutagenesis-down phenotypes in Tet and Nal^R assays, in addition to their defects in the primary screen (Lac) assays. The four assays compared here are Lac^+ papillation (Pap), quantitative Lac^- to Lac^+ frameshift reversion (Lac), I-*Sce*I-stimulated Tet^S to Tet^R frameshift reversion (Tet), and I-*Sce*I-stimulated Nal^S to Nal^R forward, base-substitution mutagenesis (Nal). Pearson correlation coefficients are given along with their *P* values, which indicate the probability that there is no correlation, and were computed with the *Matlab* computer program (software version 7.13; corrcoef command) by transforming the correlation to a *t*-statistic with *N*-2 degrees of freedom. Note that although the correlations of both Lac assays (Lac and Pap) with Tet are not significant ($P > 0.05$), Tet is significantly correlated with Nal, which is significantly correlated with both Lac and Pap ($P < 0.05$).

Mutation assays compared	Pearson correlation coefficient (*r*)	*P* value	Number of mutant pairs compared (*N*)
Lac *vs*. Pap	0.87	5×10^{-14}	40
Lac *vs.* Tet	0.11	0.52	40
Lac *vs.* Nal	0.34	0.032	40
Pap *vs.* Tet	0.04	0.82	40
Pap *vs.* Nal	0.53	4×10^{-4}	40
Tet *vs.* Nal	0.55	2.6×10^{-4}	40

Table S7. Results of screens for mutants defective in activation of σ^S, SOS and σ^E responses

All 93 SIM mutants, separated here into those with weak (1.5-3-fold), moderate (3.1 to 9-fold) and strong (>9-fold) SIM-deficiency in the quantitative Lac mutation assay (Table S3) were screened for possible defects in activating the σ^S, SOS, and σ^E stress responses. We used catalase defect of colonies to screen for mutants lacking σ^S activity in stationary phase (**Catalase colony assay**, Methods). All of the catalase-defective mutants were confirmed as *bona fide* σ^S-response-defective by their failure to activate the σ^S-dependent P*yiaG* promoter measured using of a *yiaG-yfp* fusion gene, while maintaining proficiency at transcription from a non-σ^S promoter, indicating no general transcription defect, and also showing normal growth curves, indicating that delayed entry into stationary phase did not cause reduced σ^S activity (Figs. 4A-C, S4,S5). We used UV-sensitivity (Methods) as an initial screen to identify candidate mutants that might be defective in inducing the SOS response. A subset of these was confirmed as SOS-induction-defective by failure at spontaneous induction of the SOS-dependent P_{sulA} promoter controlling *gfp* [Methods, (*21*) Fig. 4K]. We used sensitivity to SDS-EDTA (Methods, Fig. S9) as an initial screen for inability to induce the σ^E response. Out of 44, 33 of the SDS-EDTA-sensitive mutants, were confirmed as σ^E-response-deficient by their significant decrease in activity of the σ^E-dependent *rpoH*P3 promoter fused to *lacZ* (per Table S11, Figs. 4J, S9). The remaining 11 showed reductions in *rpoH*P3-*lacZ* activity that were not statistically significant, and though not demonstrated to be σ^E-response-deficient are not confirmed as σ^E-response-proficient (legend Table S11). Some genes were both σ^E-response-deficient and σ^S-response-deficient, or σ^S-response-deficient and deficient in spontaneous induction of the SOS response, and are listed under more than one phenotype.

SIM phenotype group	**Mutant genes**	**Phenotypes**
Weak (30 mutants)	*acrA*, *acrB, apaG*, *arcA*, *argD*, *barA*, *cspC*, *fre*, *ligA*, *lrp*, *malI, pepP*, *polB*, *ppk*, *prc, pstB*, *srlA*, *sulA*, *treR*, *trxB*, *ubiX*, *umuC*, *uvrY*, *ybjT*, *yicC*, *yjcC*, *yfjL*, *yqgE*, *ytjB*, *znuC*	**σ^S-response-deficient:** *cspC, fre, ubiX* **UV sensitive:** *ligA*, *pstB, umuC, uvrY, yjcC*, *yqgE* **Confirmed spontaneous SOS-induction defective:** *uvrY* **SDS-EDTA sensitive:** *acrA, acrB, arcA, fre, lrp, ppk, pstB, trxB, ubiX, znuC* **Confirmed σ^E-response-deficient:** *arcA, fre, lrp, pstB, trxB, ubiX, znuC*
Moderate (34 mutants)	*apaH, argR, crp, cyaA, cyoA, cyoD, cysD, dinB, dsbC, fruR, glnK, hscB, lexA, lipB, mdh, metJ, nuoK*, *pgi, pgl, phoU,*	**σ^S-response-deficient:** *apaH, cyoA, cyoD, glnK, hscB, mdh, metJ, nuoK*, *pgi, sdhB, sdhD, speD, speE, ubiD* **UV sensitive:** *hscB, lexA, pgi, pgl, recF*

	proL, pstC, recF, sdhB, sdhD, speD, speE, tatC, ubiD, ygfA, yhhK, yigP, yqiC, znuA	**Confirmed spontaneous SOS-induction defective:** *lexA, pgi* **SDS-EDTA sensitive:** *apaH, cyoA, cyoD, cysD, fruR, lipB, mdh, nuoK, phoU, pstC, sdhB, sdhD, speE, tatC, ubiD, ygfA, yigP, znuA* **Confirmed σ^E-response-deficient:** *apaH, cyoA, cyoD, fruR, lipB, mdh, nuoK, phoU, pstC, sdhB, sdhD, ubiD, ygfA, yigP*
Strong (29 mutants)	*dsbA, hdfR, hemL, groES, hfq, ihfA, ldcA, lon, nagC, nuoC, nuoG, nuoH, nuoJ, nuoL, nusA, rbsK, recA, recB, recC, rpoE, rppH, rpoS, ruvA, ruvB, ruvC, ubiA, ubiE, ubiH, yifE*	**σ^S-response-deficient:** *hdfR, hemL, hfq, nuoC, nuoG, nuoH, nuoJ, nuoL, rbsK, rpoS, ubiA, ubiE, ubiH, yifE* **UV sensitive:** *hdfR, hemL, hfq, ldcA, lon, nusA, recA, recB, recC, rpoS, ruvA, ruvB, ruvC, ubiA, yifE* **Confirmed spontaneous SOS-induction defective:** *recA, recB, recC* **SDS-EDTA sensitive:** *hemL, groES, hfq, ihfA, lon, nuoC, nuoG, nuoH, nuoJ, nuoL, nusA, rpoE, rppH, ubiA, ubiE, ubiH* **Confirmed σ^E-response-deficient:** *hemL, lon, nuoC, nuoG, nuoH, nuoJ, nuoL, rpoE, rppH, ubiA, ubiE, ubiH*

Table S8. σ^S activity of catalase-defective mutants

σ^S activity was assayed as YFP expression from σ^S-dependent promoter P_{yiaG} (*15*) from a *yiaG-yfp* fusion measured by flow cytometry (Fig. S4 legend). Relative σ^S activity is YFP mean fluorescence intensity of the tested strain divided by that of the *rpoS* mutant. Means ± SEM of ≥3 experiments, 3 cultures per strain. % decrease in σ^S activity= [Relative σ^S (WT – mutant)/WT) ×100] averaged from ≥3 experiments with mutant and control strains tested in parallel. *P* values per Methods. Fold increase in σ^S activity is the relative σ^S activity of the mutants divided by that of the WT control tested in parallel.

Strain	Relevant genotype	Relative σ^S activity	% decrease in σ^S activity relative to WT (mean ±SEM)	*P* value
SMR10582	WT	3.4 ± 0.3		
SMR12661	*rpoS*	1 ± 0	71 ± 2	0.0007
SMR12830	*apaH*	2.29 ± 0.05	38 ± 3	0.009
SMR14263	*cspC*	1.9 ± 0.1	44 ± 5	0.002
SMR14283	*cyoA*	2.1 ± 0.2	38 ± 5	0.005
SMR13035	*cyoD*	2.27 ± 0.09	33 ± 5	0.002
SMR14267	*fre*	2.2 ± 0.1	34 ± 6	0.007
SMR14281	*glnK*	2.53 ± 0.08	26 ± 5	0.02
SMR14261	*hdfR*	1.76 ± 0.01	48 ± 4	0.0004
SMR14431	*hemL*	2.58 ± 0.02	24 ± 6	0.009
SMR14295	*hfq*	1.36 ± 0.05	60 ± 4	0.0001
SMR14275	*hscB*	2.54 ± 0.04	25 ± 7	0.007
SMR13058	*mdh*	2.4 ± 0.1	30 ± 9	0.003
SMR14269	*metJ*	2.1 ± 0.02	40 ± 5	0.001
SMR12655	*nuoC*	2.03 ± 0.03	41 ± 4	0.002
SMR14420	*nuoG*	2.31 ± 0.03	32 ± 6	0.003
SMR14287	*nuoH*	2.26 ± 0.06	33 ± 6	0.006
SMR14289	*nuoJ*	2.21 ± 0.09	35 ± 6	0.005
SMR14285	*nuoK*	2.23 ± 0.06	34 ± 6	0.002
SMR14273	*nuoL*	2.28 ± 0.07	33 ± 6	0.02
SMR13295	*pgi*	2.6 ± 0.1	14 ± 4	0.002
SMR13312	*rbsK*	2.1 ± 0.2	30 ± 7	0.0023
SMR14297	*sdhB*	2.5 ± 0.1	28 ± 6	0.02
SMR13033	*sdhD*	2.63 ± 0.07	22 ± 7	0.01
SMR12828	*speD*	1.8 ± 0.2	39 ± 6	0.001
SMR13310	*speE*	2.1 ± 0.2	30 ± 7	0.003
SMR14447	*ubiA*	2.22 ± 0.04	35 ± 5	0.002
SMR13098	*ubiD*	2.57 ± 0.03	25 ± 5	0.008
SMR12657	*ubiE*	2.1 ± 0.2	37 ± 6	0.002
SMR12686	*ubiH*	2.17 ± 0.07	36 ± 4	0.004
SMR12699	*ubiX*	2.6 ± 0.1	24 ± 5	0.008
SMR14259	*yifE*	1.94 ± 0.07	43 ± 5	0.002
			Fold-increase in σ^S activity (mean±SEM)	*P* value
SMR10582	WT	3.9 ± 0.07		
SMR12684	*arcB*	5.7 ± 0.5	1.5 ± 0.1	0.003
SMR12683	*arcA*	6.0 ± 0.3	1.6 ± 0.08	5×10^{-5}
SMR12692	*rssB*	10.7 ± 0.2	2.8 ± 0.1	2×10^{-8}

Table S9. Upregulating σ^S *via arcB*, *arcA* or *rssB* mutations partially restores SIM-proficiency to electron transfer mutants

SIM is restored or partially restored in *cyoD*, *nuoC* or *ubiD* mutant strains by increasing σ^S by introduction of *arcB*, *arcA* or *rssB* mutations. Mutation rates taken from days 3 to 5 per (*59*). Rates and mean fold increases calculated from 3 experiments with strains tested in parallel, per (*57*). Fold difference of ratios of mutation rates for double mutants to control are, *e.g.*, the ratios of mutation rates for *cyoD arcB*, *cyoD arcA* or *cyoD rssB* to single mutant *cyoD* divided by the respective controls, *i.e.*, the ratios of *arcB*, *arcA* or *rssB* to WT. Values greater than one indicate specific suppression of the electron-transfer mutant's SIM-down phenotype by *arcB*, *arcA*, or *rssB* mutations that upregulate σ^S, and reflect the fraction of the SIM-down phenotypes of the electron transfer mutants attributable to their failure to activate the σ^S response. Values averaged from three experiments with the strains compared tested in parallel. % of mutagenesis restored was determined by multiplying the ratios of mutation rates for double mutants to single mutant by 100, such as, mutation rates (*cyoD arcB*/*arcB*) × 100, and indicate the fraction of the electron-transfer mutant's SIM deficiency that was restored by σ^S upregulation, and thus what fraction of their SIM deficiency relates to their σ^S-activation defects. *P* values for mutation rates from three experiments for strains tested in parallel, per Methods. *P* values for the ratios of mutation rates for double to single mutant (*e.g.*, *cyoD arcB*/*cyoD*) to the ratios of mutation rates for *arcB*, *arcA* or *rssB* to WT were determined by two-tailed Student's *t* test tested in parallel.

Strain	**Relevant genotype**	**Mean mutation rate (Lac^+ cfu/ 10^8 cells/day) ± SEM**	**Mean fold-increase in mutation rate over WT ± SEM**			***P* value (mutation rates)**
SMR4562	WT	32 ± 2				
SMR12673	*arcB*	56 ± 3	1.8 ± 0.07			0.002
SMR12672	*arcA*	24.6 ± 0.4	0.77 ± 0.05			0.012
SMR12566	*rssB*	106 ± 6	3.3 ± 0.03			0.0003
			Mutation rate: double mutants / single mutant ± SEM	**Mean fold-difference of ratios of mutation rate for double mutants relative to control ± SEM**	**Percent of mutagenesis restored (mean % ± SEM)**	***P* value (ratios of mutation rates for double : single mutants)**
SMR4562	WT	32 ± 2				
SMR12574	*cyoD*	4.9 ± 0.9				
SMR13050	*cyoD arcB*	31 ± 7	6.3 ± 0.4	3.6 ± 0.4	56 ± 15	0.0004
SMR13048	*cyoD arcA*	14 ± 1	2.9 ± 0.4	3.8 ± 0.3	56 ± 4	0.004
SMR13084	*cyoD rssB*	42 ± 9	8.4 ± 0.5	2.6 ± 0.2	39 ± 8	0.007
SMR4562	WT	31 ± 2				
SMR12223	*nuoC*	1.8 ± 0.1				
SMR12594	*nuoC arcB*	13.2 ± 0.05	7.3 ± 0.2	4.2 ± 0.1	24 ± 1	0.0005
SMR12590	*nuoC arcA*	7.6 ± 0.2	4.2 ± 0.1	5.5 ± 0.3	31 ± 1	0.0006
SMR12562	*nuoC rssB*	19 ± 3	10 ± 1	3.1 ± 0.3	18 ± 2	0.004
SMR4562	WT	30 ± 3				
SMR12313	*ubiD*	9 ± 1				

SMR12598	*ubiD arcB*	48 ± 4	5.4 ± 0.8	3.1 ± 0.4	86 ± 11	0.05
SMR12596	*ubiD arcA*	21 ± 2	2.4 ± 0.3	3 ± 0.2	87 ± 9	0.003
MR12613	*ubiD rssB*	55 ± 11	5.8 ± 0.5	1.8 ± 0.1	51 ± 8	0.008

Table S10. RpoS acts in the same pathway (is epistatic with) electron transfer mutations in DSB-dependent SIM

Mutation rates are the change in mutant frequency between days 3 and 5 for 3-5 independent experiments with 3-4 independent cultures per strain per experiment. Enzyme-only strains carry the inducible I-*Sce*I gene but no cutsite, and so do not induce DSBs near *lac*. Fold decrease of mutation rate for three independent experiments is the mean mutation rate for the WT-enzyme-only or WT-DSB (control) divided by that of the mutant. *P* values for mutation rates from three experiments for strains tested in parallel, and *P* values for mutation rates for double mutants (*e.g.*, *cyoD rpoS*) to single *rpoS* mutant tested in parallel, were determined by two-tailed Student's *t* test.

Strain	Relevant genotype	Mean mutation rate (Lac^+ cfu per 10^8 cells per day ± SEM)	Mean fold decrease in mutation rate (± SEM)	*P* value (mutation rates)	*P* value (rates for double : single mutants tested in parallel)
SMR6276	enzyme only WT	50 ± 8			
SMR13487	enzyme only *cyoD*	5.7 ± 0.8	9.1 ± 1	0.00002	
SMR13484	enzyme only *nuoC*	2.5 ± 0.7	21 ± 3	0.00001	
SMR13661	enzyme only *ubiE*	2.4 ± 0.4	19 ± 2	0.001	
SMR13448	enzyme only *rpoS*	2.9 ± 0.6	18 ± 1	0.00001	
SMR13491	enzyme only *rpoS cyoD*	2.5 ± 0.6	24 ± 3	0.0049	0.318
SMR13489	enzyme only *rpoS nuoC*	2.3 ± 0.3	25 ± 2	0.0049	0.161
SMR13659	enzyme only *rpoS ubiE*	1.9 ± 0.3	24 ± 2	0.001	0.247
SMR6308	DSB WT	682 ± 86			
SMR11437	DSB *rpoS*	6.4 ± 2	132 ± 42	0.0043	
SMR13495	DSB *cyoD*	91 ± 20	9 ± 2	0.000006	
SMR13497	DSB *nuoC*	25 ± 5	31 ± 5	0.000002	
SMR13669	DSB *ubiE*	4.4 ± 0.8	210 ± 30	0.00004	
SMR13633	DSB *rpoS cyoD*	4.3 ± 0.6	210 ± 16	0.0003	0.666
SMR13630	DSB *rpoS nuoC*	2.9 ± 0.4	200 ± 20	0.0003	0.794
SMR13663	DSB *rpoS ubiE*	3.4 ± 0.2	261 ± 14	0.00004	0.081
SMR17297	DSB *fre*	62 ± 10	9.9 ± 1.1	0.001	
SMR17291	DSB *hemL*	20 ± 7.8	35 ± 7.8	0.0006	
SMR17374	DSB *hscB*	34 ± 4.7	18.6 ± 4.8	0.0007	
SMR14455	DSB *mdh*	5.3 ± 1.4	127 ± 31	0.0006	
SMR17370	DSB *pgi*	34 ± 9.7	22 ± 8	0.0007	
SMR17301	DSB *sdhD*	39 ± 4.2	16 ± 1.2	0.00001	
SMR17299	DSB *rpoS fre*	6.6 ± 1.6	104 ± 9	0.00001	0.091
SMR17293	DSB *rpoS hemL*	4.5 ± 1.8	170 ± 50	0.0006	0.286
SMR17376	DSB *rpoS hscB*	6.1 ± 1.9	129 ± 25	0.00001	0.201
SMR14467	DSB *rpoS mdh*	4.5 ± 0.4	131 ± 2.7	0.0006	0.449
SMR17372	DSB *rpoS pgi*	5.4 ± 1.5	127 ± 30	0.0006	0.396
SMR17385	DSB *rpoS sdhD*	3.7 ± 1	176 ± 36	0.0006	0.872
SMR17282	DSB *recA*	1 ± 0.09	619 ± 56	0.0005	

Table S11. σ^E activity of SDS-EDTA-sensitive mutants

The σ^E membrane-protein stress response can be induced either by peptide fragments of membrane proteins in log-phase cells, or by an alternative starvation-induced pathway in stationary-phase cells (*134, 135*). We reasoned that the starvation induction of the σ^E response is more likely under our stationary-phase-stress experimental conditions, and so to reflect that, we measured σ^E activity in saturated stationary-phase cultures (Methods) measured as expression of the σ^E-response-dependent *rpoH*P3-*lacZ* fusion gene (*20*) in cultures grown to saturation. β-galactosidase activity in Miller units of cultures grown to saturation were taken for each strain and divided by that of the WT control strain assayed in parallel to obtain the relative σ^E activity. The table shows those mutants that showed significantly decreased σ^E activity, indicating that these genes encode proteins that promote/allow activating the σ^E response, which is required for SIM (*8*). Means ± SEM of ≥3 experiments, two cultures per strain. *P* values (per Methods) were calculated based on values normalized to WT tested in parallel for the ≥3 experiments. % reduction in σ^E activity= [Relative σ^E (WT – mutant)/WT) ×100] averaged from ≥3 experiments with the mutant and control strains tested in parallel.

Strain	Relevant genotype	Relative σ^E activity (mean ± SEM)	% reduction in σ^E activity relative to WT (mean ±SEM)	*P* value
CAG45114	WT	1 ± 0		
SMR15311	*rpoE2072*::Tn*10*dCam	0.71 ± 0.002	29.3 ± 0.2	0.00005
SMR15328	*apaH*	0.71 ± 0.01	29.2 ± 1.3	0.000001
SMR15360	*arcA*	0.91 ± 0.01	9.4 ± 0.94	0.00002
SMR15330	*cyoA*	0.91 ± 0.01	9 ± 1	0.0001
SMR15293	*cyoD*	0.87 ± 0.02	12.9 ± 2.1	0.0008
SMR15362	*fre*	0.86 ± 0.02	13.7 ± 2	0.001
SMR15253	*fruR*	0.78 ± 0.02	22.2 ± 2.3	0.00008
SMR15319	*hemL*	0.75 ± 0.08	24.8 ± 7.8	0.01
SMR15259	*lipB*	0.79 ± 0.05	20.6 ± 4.8	0.004
SMR15273	*lon*	0.89 ± 0.01	10.9 ± 1.3	0.0002
SMR15265	*lrp*	0.86 ± 0.03	14.2 ± 3.5	0.0003
SMR15356	*mdh*	0.56 ± 0.11	43.5 ± 11	0.005
SMR15277	*nuoC*	0.61 ± 0.06	38.7 ± 5.6	0.0004
SMR16038	*nuoG*	0.48 ± 0.01	51.8 ± 1.4	0.0007
SMR15249	*nuoH*	0.59 ± 0.11	41.1 ± 11.5	0.03
SMR15358	*nuoJ*	0.53 ± 0.02	47.2 ± 2.4	0.000002
SMR15366	*nuoK*	0.62 ± 0.05	38 ± 4.8	0.0002
SMR15354	*nuoL*	0.53 ± 0.07	47 ± 6.8	0.0004
SMR15289	*phoU*	0.85 ± 0.03	14.9 ± 3.6	0.001
SMR15301	*pstB*	0.88 ± 0.02	12.4 ± 2.7	0.001
SMR15287	*pstC*	0.92 ± 0.01	7.7 ± 1.3	0.001
SMR15315	*rppH*	0.84 ± 0.03	16.2 ± 2.8	0.003
SMR15261	*sdhB*	0.8 ± 0.04	20.3 ± 3.7	0.001
SMR15267	*sdhD*	0.69 ± 0.02	30.6 ± 2.1	0.00001
SMR15283	*trxB*	0.91± 0.02	9.1 ± 1.8	0.02
SMR15321	*ubiA*	0.49 ± 0.08	50.8 ± 7.6	0.004
SMR15323	*ubiD*	0.7 ± 0.09	29.6 ± 8.7	0.004
SMR15257	*ubiE*	0.38 ± 0.01	62.2 ± 0.85	$2X10^{-10}$
SMR15295	*ubiH*	0.38 ± 0.01	62.4 ± 0.73	$1X10^{-10}$

SMR15317	*ubiX*	0.74 ± 0.07	26.2 ± 8.8	0.02
SMR15326	*ygfA*	0.88 ± 0.02	11.7 ± 1.8	0.0006
SMR15309	*yigP*	0.55 ± 0.05	45.4 ± 5.4	0.0007
SMR15291	*znuC*	0.89 ± 0.02	11.2 ± 2	0.001
			Fold-increase in σ^E activity relative to WT (mean ±SEM)	***P* value**
CAG45114	WT	1 ± 0	1 ± 0	
SMR15364	*arcB*	1.02 ± 0.01	1.02 ± 0.01	0.08
SMR15360	*arcA*	0.91 ± 0.01	0.91 ± 0.01	0.00002
SMR15271	*rssB*	0.88 ± 0.03	0.88 ± 0.03	0.009
SMR15754	*rseA*	2.1 ± 0.11	2.1 ± 0.11	0.0004

Table S12. Transposon insertion position for promoter-insertion mutations

Transposon insertion sites in promoter region for the SIM-down mutants. Insertion sites are bp upstream of the start codon.

Mutant gene	Isolate (strain)	Transposon insertion site (bp)
arcA	1 (SMR11410) 2 (SMR11957)	-248 -252
cysD	1 (SMR11925) 2 (SMR11958)	-79 -88
groES	1 (SMR11934)	-93
ligA	1 (SMR11412)	-27
rpoE	1 (SMR5236) 2 (SMR11937)	-90 -80
yfjL	1 (SMR11472)	-73
ytjB	1 (SMR11476)	-57

Table S13. Reconstruction experiments: speed of colony formation of electron-transfer mutants does not account for their SIM-down phenotypes

Average days to form colonies shows the means of three independent reconstruction experiments of 3-4 independent cultures each under exact reconstruction of Lac$^+$ selection conditions (**Reconstruction experiments**, Methods). Day = 24h interval. Percent of viable cells of Lac$^+$ colonies are the cfu on M9 B1 lactose *versus* M9 B1 glycerol plates, and would indicate any reduction in the number of colonies formed on lactose medium. The results indicate that the electron-transfer mutants are not slow colony formers on lactose medium, so the absence of stress-induced mutants in these strains is not the result of slow growth of mutants, but rather of decreased mutagenesis.

Strain	Relevant Genotype	Average days to form colonies (± range)	% viable cells of Lac$^+$ colonies (± range)
SMR3856	Lac$^+$	2.05 ± 0.03	99 ± 7
SMR3858	Lac$^+$	2.06 ± 0.02	100 ± 7
SMR3859	Lac$^+$	2.06 ± 0.04	98 ± 1
SMR13301	*nuoC* Lac$^+$	2.11 ± 0.01	94 ± 13
SMR13302	*nuoC* Lac$^+$	2.13 ± 0.04	88 ± 7
SMR13303	*nuoC* Lac$^+$	2.1 ± 0.02	86 ± 21
SMR13274	*cyoD* Lac$^+$	2.12 ± 0.04	93 ± 6
SMR13275	*cyoD* Lac$^+$	2.13 ± 0.06	95 ± 3
SMR13276	*cyoD* Lac$^+$	2.11 ± 0.07	84 ± 2
SMR13283	*ubiD* Lac$^+$	2.16 ± 0.01	97 ± 1
SMR13284	*ubiD* Lac$^+$	2.2 ± 0.3	93 ± 7
SMR13285	*ubiD* Lac$^+$	2.12 ± 0.02	92 ± 3
SMR13292	*sdhD* Lac$^+$	2.1 ± 0.03	94 ± 3
SMR13293	*sdhD* Lac$^+$	2.1 ± 0.1	94 ± 5
SMR13294	*sdhD* Lac$^+$	2.09 ± 0.03	92 ± 5
SMR13289	*mdh* Lac$^+$	2.44 ± 0.03	86 ± 0.4
SMR13290	*mdh* Lac$^+$	2.38 ± 0.04	87 ± 2
SMR13291	*mdh* Lac$^+$	2.34 ± 0.02	80 ± 3
SMR13280	*ubiE* Lac$^+$	2.66 ± 0.03	89 ± 6
SMR13281	*ubiE* Lac$^+$	2.6 ± 0.1	83 ± 17
SMR13282	*ubiE* Lac$^+$	2.4 ± 0.2	88 ± 15

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