

Supporting Information

TITLE

Functional identification of serine hydroxymethyltransferase as a key gene involved in lysostaphin resistance and virulence potential of *Staphylococcus aureus* strains

RUNNING TITLE

Role of *shmT* in lysostaphin resistance and virulence

AUTHORS

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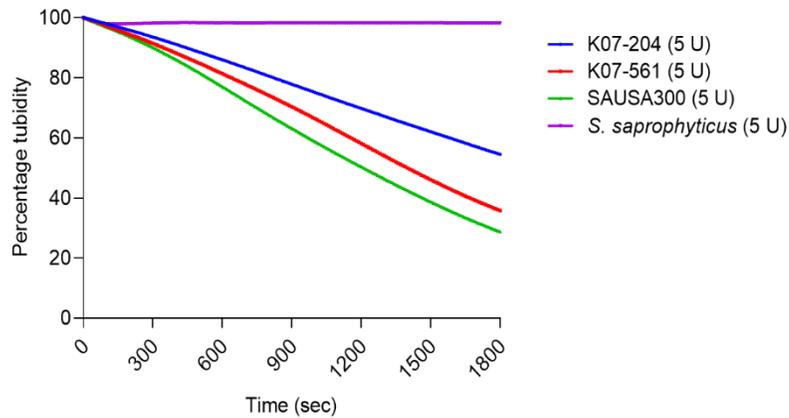
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Supporting Fig. S1



Supporting Fig. S1. Lysostaphin killing kinetics of human isolates of ST72. (A)

Lysostaphin mediated killing efficiency using turbidity reduction among *lys*^r (K07-204) and *lys*^s (K07-561) in comparison to *S. aureus* USA300. Lysostaphin resistant *lys*^r K07-204 showed 37% percent turbidity reduction as compared to $\geq 60\%$ turbidity reduction for K07-561 and *S. aureus* USA300 within 30 min of lysostaphin treatment. *S. saprophyticus* displayed resistance to lysostaphin treatment.

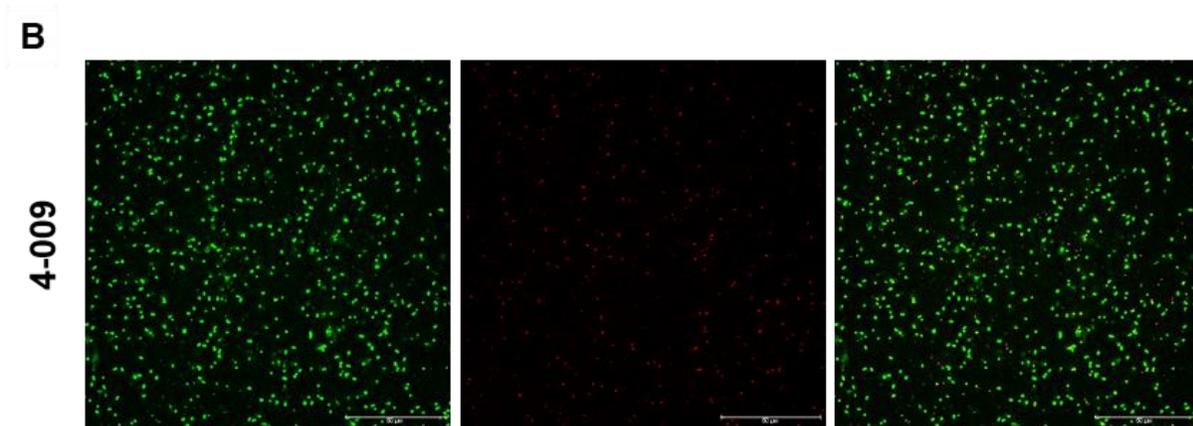
Supporting Fig. S2

A



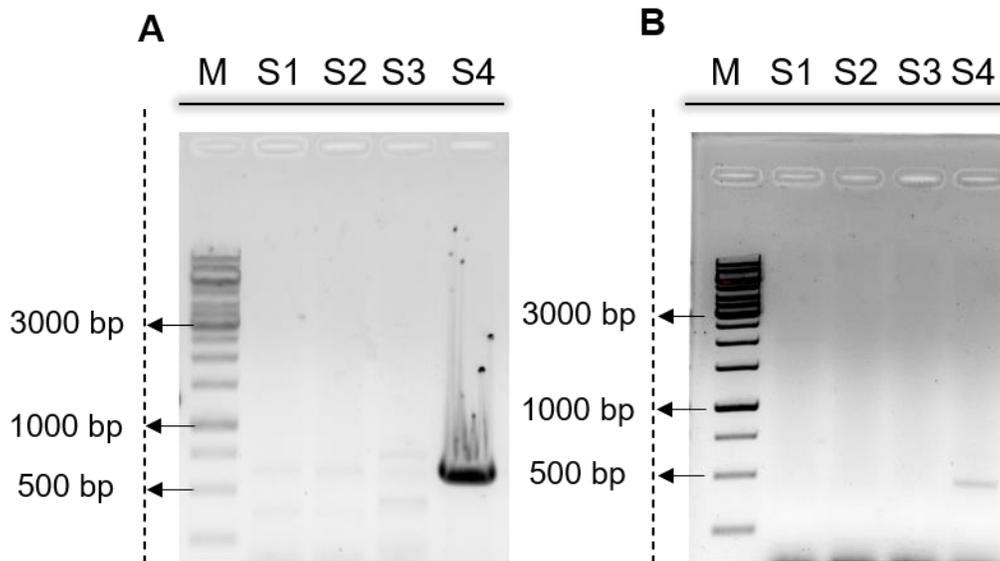
A'





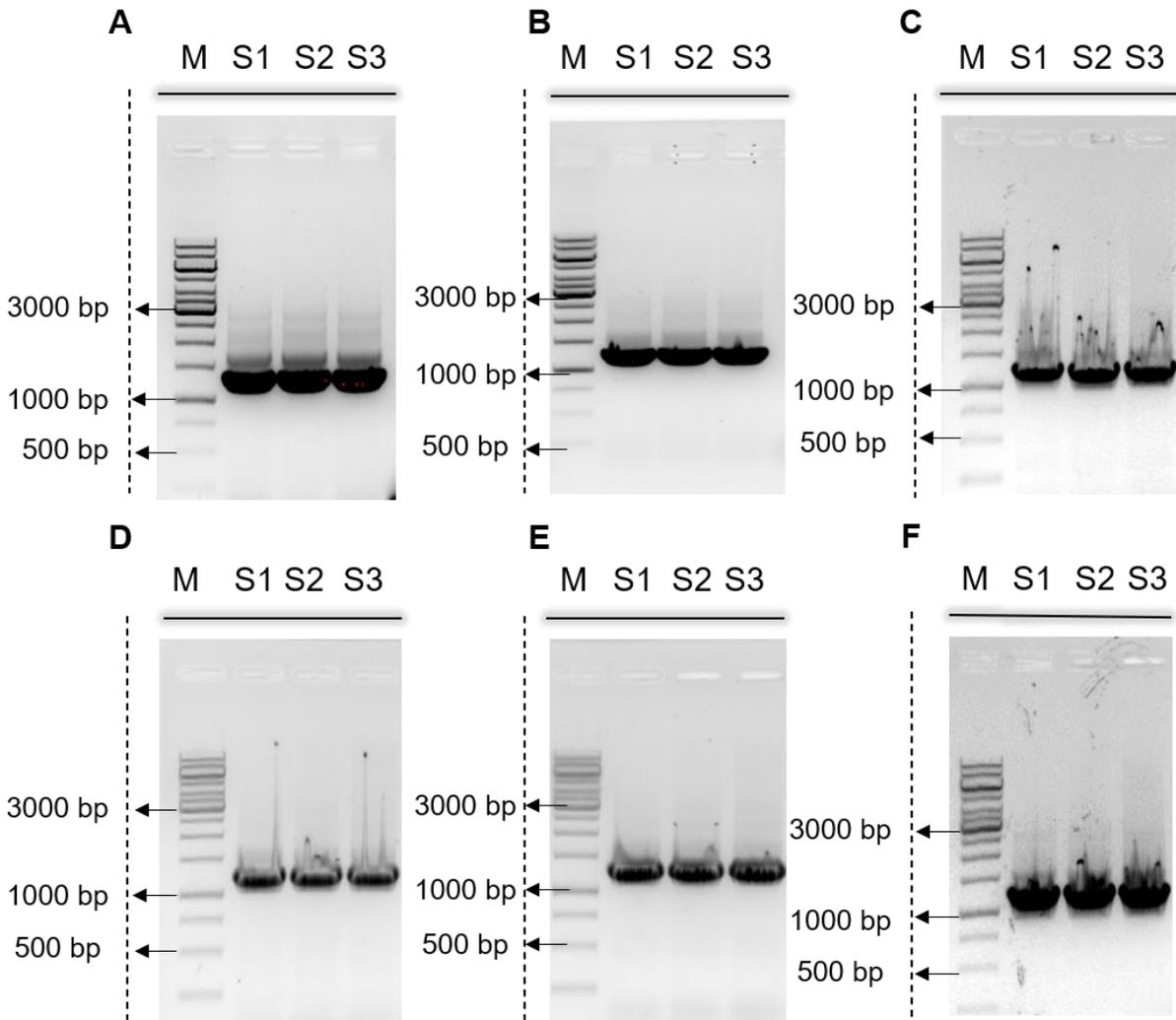
Supporting Fig. S2. Scanning electron and confocal microscopy of ST72 resistant soil isolate, 4-009 to assess lysostaphin-mediated alteration in cell morphology and live/dead staining. (A-A') SEM photomicrograph to assess the lysostaphin-mediated alteration in the cell morphology of lysostaphin resistant (*lys^r*) ST72 soil isolate 4-009 before (A) and after lysostaphin treatment (A') and displayed no alterations post lysostaphin treatment (A'); (B) Live/dead images of *S. aureus* lysostaphin resistant (*lys^r*) ST72 isolate, 4-009 after lysostaphin treatment (4 U) using SYTO9/PI for 5 min. The total number of 4-009 cells were stained with SYTO9 stain (SYTO channel; green fluorescent cells) while a smaller proportion of cells were stained with PI (PI channel; red fluorescent cells) showing dead cells.

Supporting Fig. S3



Supporting Fig. S3. PCR based screening of presence/absence of *epr* and *lss* genes responsible for lysostaphin resistance in human isolates of ST72. (A) Agarose gel showing the presence/absence of endopeptidase gene (*epr*) screened by PCR amplification in ST72 isolates, K07-204 (S1), K07-561 (S2) as compared to *S. aureus* USA300 (negative control, S3) and *S. simulans* (positive control, S4) wherein the *epr* gene was amplified only in *S. simulans* on agarose gel conferring lysostaphin protection. (B) Agarose gel showing the PCR amplified lysostaphin gene (*lss*) in ST72 isolates, K07-204 (S1), K07-561 (S2) as compared to *S. aureus* USA300 (negative control, S3) and *S. simulans* (positive control, S4) wherein the *lss* gene was amplified only in *S. simulans* conferring lysostaphin production. (M denotes the 1kb DNA marker ranging from 250 bp to 10 kb). These results indicate that the ST72 isolates, K07-204 (*lys*^r) and K07-561 (*lys*^r) are not the lysostaphin producers.

Supporting Fig. S4



Supporting Fig. S4. PCR amplification, cloning, sequencing, and screening of associated mutation(s) in other key genes known for lysostaphin resistance. (A-F) Agarose gel showing the amplification of *femA* (A) *femB* (B) *femX* (C) *fmhC* (D) *lyrA* (E) and *shmT* (F) in ST72 isolates, K07-204 (S1), K07-561 (S2) and *S. aureus* USA300 (S3). These genes were amplified to clone in pCRTPO2.1 cloning vector. Clones were sequenced to assess the mutation(s), if any, known to be responsible for lysostaphin resistance. M denotes the 1kb DNA marker ranging from 250 bp to 10 kb.

Supporting Fig. S5

A

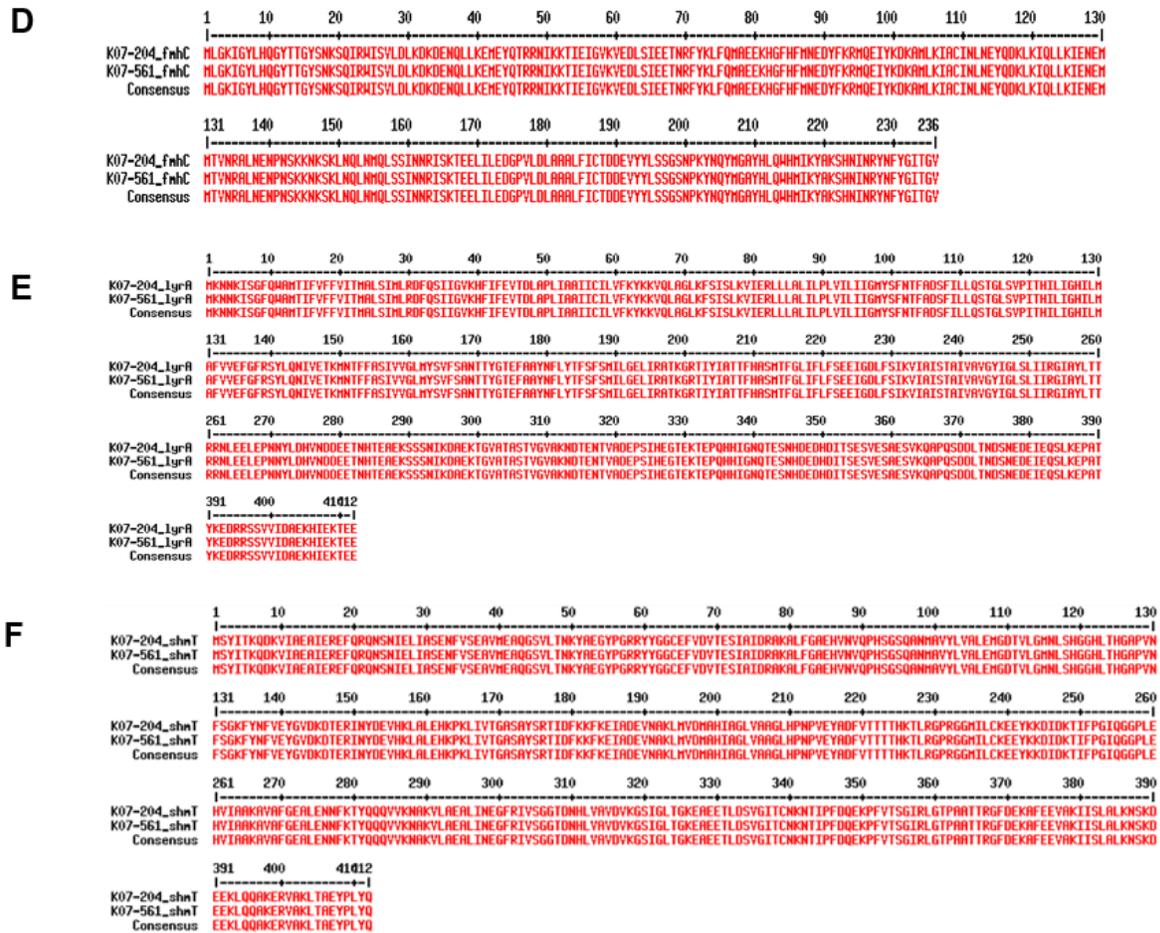
	1	10	20	30	40	50	60	70	80	90	100	110	120	130
K07-204_fenA	MKLNERIQGYMINKFTNLTAKEFGAFDTSHPYSHFQTQVGHYELKLAGGYETHLVGKNNNNEVIARCLLTAVPVHKVFKYFYSNRGPIIDYENQELVHFFNELSKYKKAHRCLYLHIDPVPYQYLN													
K07-561_fenA	MKLNERIQGYMINKFTNLTAKEFGAFDTSHPYSHFQTQVGHYELKLAGGYETHLVGKNNNNEVIARCLLTAVPVHKVFKYFYSNRGPIIDYENQELVHFFNELSKYKKAHRCLYLHIDPVPYQYLN													
Consensus	MKLNERIQGYMINKFTNLTAKEFGAFDTSHPYSHFQTQVGHYELKLAGGYETHLVGKNNNNEVIARCLLTAVPVHKVFKYFYSNRGPIIDYENQELVHFFNELSKYKKAHRCLYLHIDPVPYQYLN													
	131	140	150	160	170	180	190	200	210	220	230	240	250	260
K07-204_fenA	HDEITGNAGNDHFFDKMSNLGFEHTGFHKGFDPVLQIRYHSVLDLKKTTADDIIKNNHGLRKRNTKKVKNKGVKVFLEEELPIFRSFHEDTSESKAFADRODKFYNNRLKYYKDRVLYPLAYINPDE													
K07-561_fenA	HDEITGNAGNDHFFDKMSNLGFEHTGFHKGFDPVLQIRYHSVLDLKKTTADDIIKNNHGLRKRNTKKVKNKGVKVFLEEELPIFRSFHEDTSESKAFADRODKFYNNRLKYYKDRVLYPLAYINPDE													
Consensus	HDEITGNAGNDHFFDKMSNLGFEHTGFHKGFDPVLQIRYHSVLDLKKTTADDIIKNNHGLRKRNTKKVKNKGVKVFLEEELPIFRSFHEDTSESKAFADRODKFYNNRLKYYKDRVLYPLAYINPDE													
	261	270	280	290	300	310	320	330	340	350	360	370	380	390
K07-204_fenA	YIKELMEERDILNKDLNKALKDIEKRPENKKAHNRKMLQQQLDANEQKTEEGKRLQEEHGNELPTSRGFFINPFEVYVYAGGTSNRFHFAGSYAVQHEINIALNHGIDRYNFYVGSYSGKFTEDREDA													
K07-561_fenA	YIKELMEERDILNKDLNKALKDIEKRPENKKAHNRKMLQQQLDANEQKTEEGKRLQEEHGNELPTSRGFFINPFEVYVYAGGTSNRFHFAGSYAVQHEINIALNHGIDRYNFYVGSYSGKFTEDREDA													
Consensus	YIKELMEERDILNKDLNKALKDIEKRPENKKAHNRKMLQQQLDANEQKTEEGKRLQEEHGNELPTSRGFFINPFEVYVYAGGTSNRFHFAGSYAVQHEINIALNHGIDRYNFYVGSYSGKFTEDREDA													
	391	400	410	420	430	433								
K07-204_fenA	GVYFKKGYNAEIIIEYVGFIPKINPKYARYTALKKVKORIF													
K07-561_fenA	GVYFKKGYNAEIIIEYVGFIPKINPKYARYTALKKVKORIF													
Consensus	GVYFKKGYNAEIIIEYVGFIPKINPKYARYTALKKVKORIF													

B

	1	10	20	30	40	50	60	70	80	90	100	110	120	130
K07-204_fenB	MKFTLTYTEFDMFYQNPSESHYFQVKEIIVTRENDFEVYLLGKDDNNKVIARSLFSKIPITGSGYVYYSNRGPIYDFSDGLVDYVYKELDKYLQHQCLYKLDYPLAYLHIDPVPYQYLN													
K07-561_fenB	MKFTLTYTEFDMFYQNPSESHYFQVKEIIVTRENDFEVYLLGKDDNNKVIARSLFSKIPITGSGYVYYSNRGPIYDFSDGLVDYVYKELDKYLQHQCLYKLDYPLAYLHIDPVPYQYLN													
Consensus	MKFTLTYTEFDMFYQNPSESHYFQVKEIIVTRENDFEVYLLGKDDNNKVIARSLFSKIPITGSGYVYYSNRGPIYDFSDGLVDYVYKELDKYLQHQCLYKLDYPLAYLHIDPVPYQYLN													
	131	140	150	160	170	180	190	200	210	220	230	240	250	260
K07-204_fenB	ALVNLKFSHGIEHGFTEYDTSQVYRHGVLNLEGGTPELTKKTFDSQRRKINIKAIYGVKVFLEERDFNLDLYRETEERAGFVSKTDQYFYNIIDTYGDKVLYPLAYLHIDPVPYQYLN													
K07-561_fenB	ALVNLKFSHGIEHGFTEYDTSQVYRHGVLNLEGGTPELTKKTFDSQRRKINIKAIYGVKVFLEERDFNLDLYRETEERAGFVSKTDQYFYNIIDTYGDKVLYPLAYLHIDPVPYQYLN													
Consensus	ALVNLKFSHGIEHGFTEYDTSQVYRHGVLNLEGGTPELTKKTFDSQRRKINIKAIYGVKVFLEERDFNLDLYRETEERAGFVSKTDQYFYNIIDTYGDKVLYPLAYLHIDPVPYQYLN													
	261	270	280	290	300	310	320	330	340	350	360	370	380	390
K07-204_fenB	ENRRDQMAKKEKSKQKQKIKRELKQIDHDQHELLNASELSKTDGPTLNLASGYVYFANRYEYNYVSGGSEKYNQFNGPYNHAFHINCYFDNGYDRYNYGLSGDFTESEDEYGYRFKRGFVYQIEE													
K07-561_fenB	ENRRDQMAKKEKSKQKQKIKRELKQIDHDQHELLNASELSKTDGPTLNLASGYVYFANRYEYNYVSGGSEKYNQFNGPYNHAFHINCYFDNGYDRYNYGLSGDFTESEDEYGYRFKRGFVYQIEE													
Consensus	ENRRDQMAKKEKSKQKQKIKRELKQIDHDQHELLNASELSKTDGPTLNLASGYVYFANRYEYNYVSGGSEKYNQFNGPYNHAFHINCYFDNGYDRYNYGLSGDFTESEDEYGYRFKRGFVYQIEE													
	391	400	410	419										
K07-204_fenB	LIGDFYKPIHKVYALFTTLKLRKLRK													
K07-561_fenB	LIGDFYKPIHKVYALFTTLKLRKLRK													
Consensus	LIGDFYKPIHKVYALFTTLKLRKLRK													

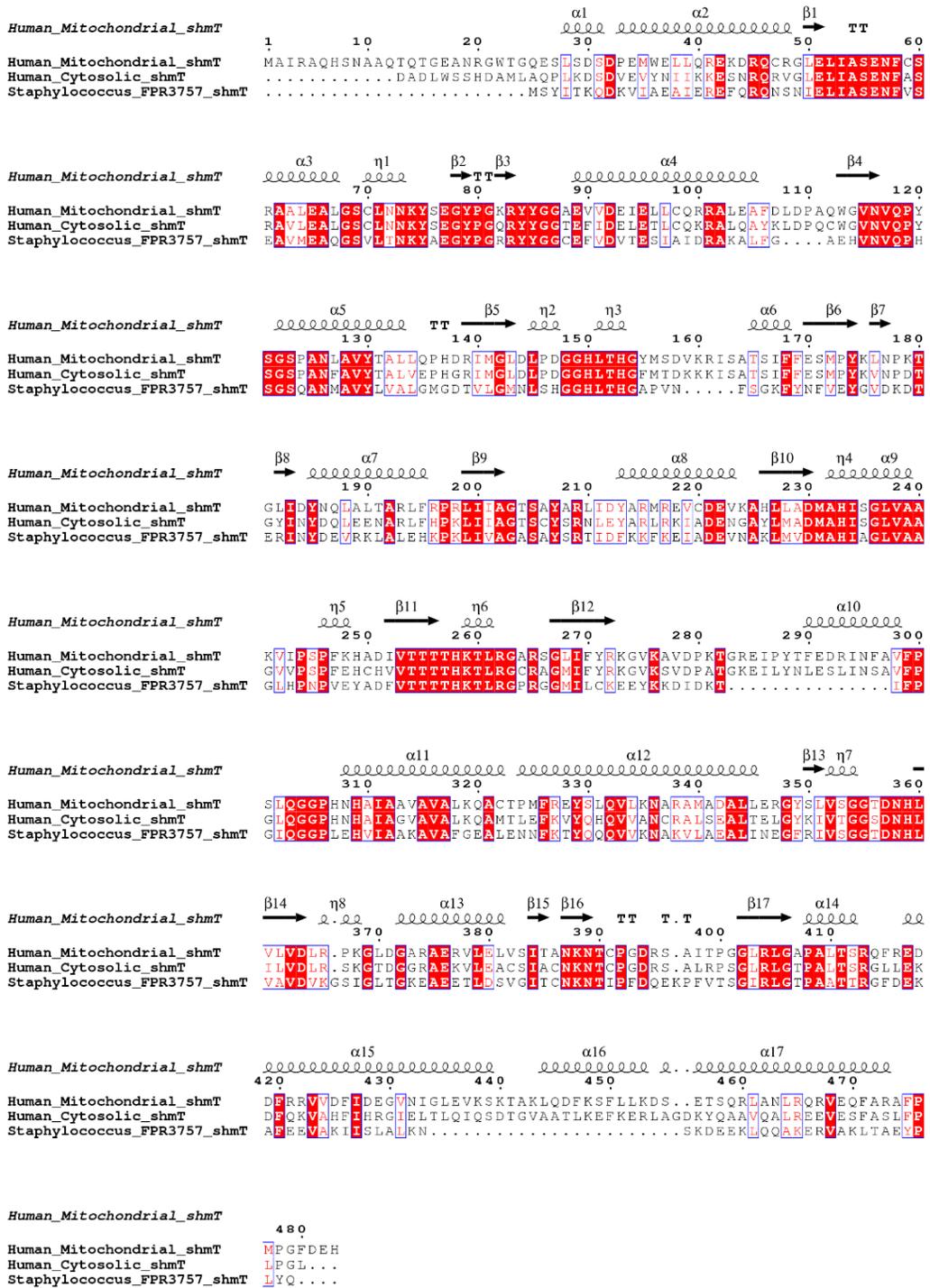
C

	1	10	20	30	40	50	60	70	80	90	100	110	120	130
K07-204_fenX	HEKMHITNQEHDFAFKSHIPNGOLLQTKARETKKLTGYHARRIYVGRDGEVQVQALLFKKVPKLPYTLCTYSRGFVYDYSNKEALNALLDSARAKTAKREKRYAIKIDPQVEVDKGTDLQNLKALGFKH													
K07-561_fenX	HEKMHITNQEHDFAFKSHIPNGOLLQTKARETKKLTGYHARRIYVGRDGEVQVQALLFKKVPKLPYTLCTYSRGFVYDYSNKEALNALLDSARAKTAKREKRYAIKIDPQVEVDKGTDLQNLKALGFKH													
Consensus	HEKMHITNQEHDFAFKSHIPNGOLLQTKARETKKLTGYHARRIYVGRDGEVQVQALLFKKVPKLPYTLCTYSRGFVYDYSNKEALNALLDSARAKTAKREKRYAIKIDPQVEVDKGTDLQNLKALGFKH													
	131	140	150	160	170	180	190	200	210	220	230	240	250	260
K07-204_fenX	KGFKEGLSKDYIQPRNTHITPIDKNDDELLNSFERNRKSKVRLALKRGTTVERSOREGLKTFRELAKITGERDGLTRDTSYFENIYDALHEDGARELFLVKLPKENTAKYNOELNELHAEITKQKQKH													
K07-561_fenX	KGFKEGLSKDYIQPRNTHITPIDKNDDELLNSFERNRKSKVRLALKRGTTVERSOREGLKTFRELAKITGERDGLTRDTSYFENIYDALHEDGARELFLVKLPKENTAKYNOELNELHAEITKQKQKH													
Consensus	KGFKEGLSKDYIQPRNTHITPIDKNDDELLNSFERNRKSKVRLALKRGTTVERSOREGLKTFRELAKITGERDGLTRDTSYFENIYDALHEDGARELFLVKLPKENTAKYNOELNELHAEITKQKQKH													
	261	270	280	290	300	310	320	330	340	350	360	370	380	390
K07-204_fenX	ETSEKQAKKQMHNDQAQKIAKNEOLKRDLEALEKHEPEGTYL SGALLHFAFGSKSYLYGASSNEFRDFLPNHQHQTTHMKYAREHGATTYDFGGTNDPDKSEHYGLHAFKKYVGTLYSEKIGEFDY													
K07-561_fenX	ETSEKQAKKQMHNDQAQKIAKNEOLKRDLEALEKHEPEGTYL SGALLHFAFGSKSYLYGASSNEFRDFLPNHQHQTTHMKYAREHGATTYDFGGTNDPDKSEHYGLHAFKKYVGTLYSEKIGEFDY													
Consensus	ETSEKQAKKQMHNDQAQKIAKNEOLKRDLEALEKHEPEGTYL SGALLHFAFGSKSYLYGASSNEFRDFLPNHQHQTTHMKYAREHGATTYDFGGTNDPDKSEHYGLHAFKKYVGTLYSEKIGEFDY													
	391	400	410	421										
K07-204_fenX	VLNQLYQLIEQVKPRLTKAKIKISRLLKRL													
K07-561_fenX	VLNQLYQLIEQVKPRLTKAKIKISRLLKRL													
Consensus	VLNQLYQLIEQVKPRLTKAKIKISRLLKRL													



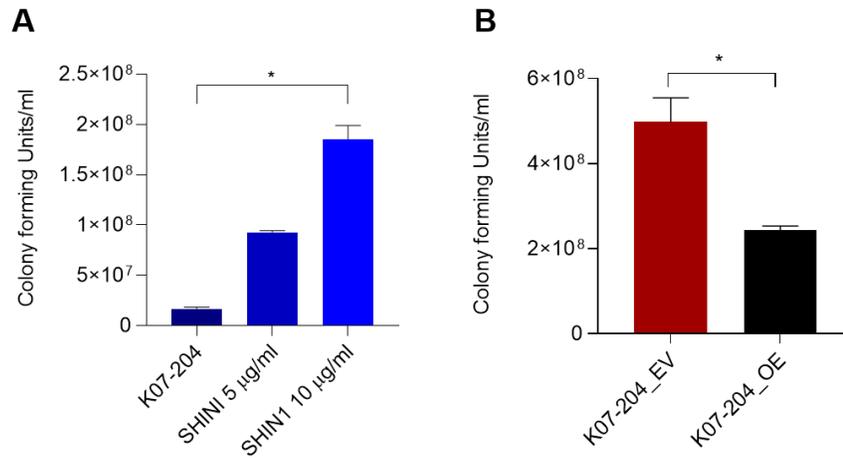
Supplementary Fig. S5. Cloning, sequencing, multiple sequence alignment to assess the mutation(s) upon translated DNA sequences. (A-F) Cloning of (A) *femA*, (B) *femB*, (C) *femX*, (D) *fmhC*, (E) *lyrA* (F) and *shmT* into pCR2.1TOPO cloning vector followed by DNA sequencing of multiple clones to assess mutation, if any, responsible for differential lysostaphin resistance between human isolates of ST72 K07-204 (*lys^r*) and K07-561 (*lys^s*). The sequenced DNA were translated *in-silico* to get amino acid sequences. The amino acid sequences of lysostaphin resistant (*lys^r*) K07-204 and lysostaphin susceptible (*lys^s*) K07-561 showed 100% identity. These results indicated that no known mechanism exists to explain the differential lysostaphin resistance between the lysostaphin resistant (*lys^r*) K07-204 and lysostaphin susceptible (*lys^s*) K07-561, human isolates of ST72.

Supporting Fig. S6



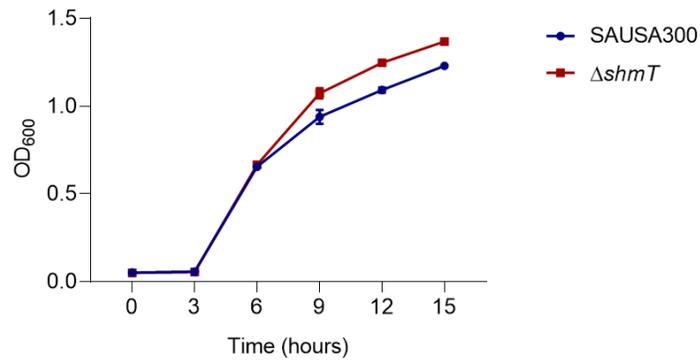
Supporting Fig. S6. Alignment of SHMT from *S. aureus* USA300 with human SHMTs to assess the overall similarity and identity. Alignment results showed a significantly high identity with two human SHMTs, human cytosolic (UniProtKB - P34896) and mitochondrial SHMT (UniProtKB - P34897) with SHMT of *S. aureus* USA300 FPR3757 ([CP000255.1](#)). The human cytosolic and mitochondrial SHMT displayed 63.45% identity, while the human cytosolic and mitochondrial SHMT displayed 45.5% and 42% identity with SHMT of *S. aureus* USA300 FPR3757, respectively

Supporting Fig. S7



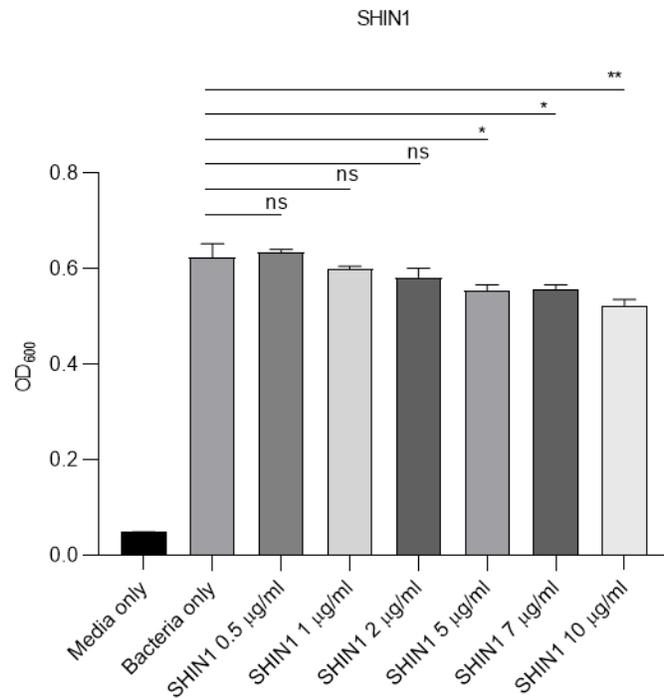
Supporting Fig S7. Role of SHMT in lysostaphin resistance of K07-204 human isolate of ST72. (A) The phenotypic assessment of lysostaphin resistance/susceptibility of K07-204 upon SHIN1-mediated inhibition of SHMT wherein the inhibition of SHMT marginally enhanced the resistance of K07-204, while (B) the overexpression of *shmT* (K07-204 with pRMC2_ *shmT*) showed reduced lysostaphin resistance of K07-204. The lysostaphin killing assay was performed by using 5 units for 10 min incubation.

Supporting Fig. S8



Supporting Fig. S8. The role of *shmT* on the fitness of *S. aureus* USA300. The role of *shmT* on the fitness of SAUSA300 was assessed by comparing the growth of wild type SAUSA300 and $\Delta shmT$ knockout in TSB media for 16h. The growth of the $\Delta shmT$ knockout and wild type SAUSA300 was found to be comparable.

Supporting Fig. S9



Supporting Fig. S9. Serine hydroxymethyltransferase inhibitor 1 (SHIN1) toxicity to *S. aureus* USA300 cells at varying concentrations. The SHIN1 showed insignificant inhibition of bacterial growth up to 2 µg/mL while a mild inhibition in the cell division was observed beyond 2 to 10 µg/mL, measured by estimating the inhibition of cell density at OD_{600 nm}.

Table S1. *S. aureus* ST72 isolates

Sequence type 72	MRSA/MSSA	Source of Isolation	Reference
K01-140	MRSA	Human	[1]
K07-204	MRSA	Human	
K07-322	MRSA	Human	
K01-799	MSSA	Human	
K07-006	MSSA	Human	
K07-561	MSSA	Human	
05-B-52	MRSA	Animal	
05-B-60	MRSA	Animal	
08-B-93	MRSA	Animal	
08-P-236	MRSA	Animal	
4-009	MRSA	Soil	

Table S2. Primers used in the study

Purpose	Name	Sequence (5'-3')	Reference/Source	Amplicon size (bp)
PCR	<i>lss_fwd</i>	GCTATTGGACTGAGTACATTTGCC	This study	Not amplified
	<i>lss_rev</i>	CTGCGGCATGCTTCTAAATGGACCAGTC		
	<i>epr_fwd</i>	CTAYWCACATMGMGGTCCWGTCATRRAC	This study	Not amplified
	<i>epr_rev</i>	TTAGAATTAGGGTTTTCTTTTAAT		
	<i>fmhC_fwd_KpnI</i>	AACATAGGTACCATGAAATTTTCAACTTTAAGTG	This study	1245
	<i>fmhC_rev_EcoRI</i>	AAGATAGAATTCTCAAACCTTATAAATAAGTTTTGC		
	<i>femA_fwd_KpnI</i>	AACATAGGTACCTTGCAGAGGGGAAATAGAAAACTG	This study	1338
	<i>femA_rev_EcoRI</i>	C AAGATAGAATTCCTAAAAAATTCTGTCTTTAACTTTTT		
	<i>femB_fwd_KpnI</i>	AACATAGGTACCATGAAATTTACAGAGTTAACTG	This study	1260
	<i>femB_rev_EcoRI</i>	AAGATAGAATTCCTATTTCTTTAATTTTTTACGT		
	<i>femX_fwd_KpnI</i>	AACATAGGTACCATGGAAAAGATGCATATCACTAATC	This study	1266
	<i>femX_rev_EcoRI</i>	AAGATAGAATTCCTATTTTCGTTTTAATTTACGAG		
	<i>lyrA_fwd_KpnI</i>	AACATAGGTACCATGAAGAACAATAAAATTTCTG	This study	1260
	<i>lyrA_rev_EcoRI</i>	AAGATAGAATTCCTATTTGTTTTATCTGAAGATTG		
	<i>shmT_fwd_KpnI</i>	AACATAGGTACCATGTCTTATATCACCAAGCAAG	This study	1239
	<i>shmT_rev_EcoRI</i>	AAGATAGAATTCCTATTGATATAGAGGATATTCAGC		
qRT-PCR	<i>gyrA_fwd</i>	CGTCAACGTATTGTTGTCAC	This study	180
	<i>gyrA_rev</i>	ACACTAGCATTTCATCCTT		
	<i>shmT_fwd</i>	TCGGAAGCGGTTATGGAA	This study	196
	<i>shmT_rev</i>	CAGCCATGTTGCTTGTG		

Table S3. Staphylococcal strains/isolates and plasmid used in the study

Strains	Organisms	Descriptions	Reference/Source
Strains	<i>Escherichia coli</i> DH5 α	<i>F- endA1 glnV44 thi-1 recA1 relA1 gyrA96 deoRnupG Φ80dlacZΔM15 Δ(lacZYA-argF) U169, hsdR17(rK- mK+), λ-</i>	Invitrogen, USA
	<i>E. coli</i> DH5 α _pRMC2	For amplification of pRMC2 vector, amp ^r	This study
	WT USA300 FPR3757	JE2, wild-type epidemic community-associated methicillin-resistant <i>S. aureus</i> isolate USA300 LAC	NARSA
	RN4220	Restriction-deficient strain of NCTC8325	[2]
	WT <i>Staphylococcus simulans</i>	Lysostaphin synthesizing (<i>lss</i>) and resistance gene (<i>epr</i>)	Lab collection
	WT <i>Staphylococcus saprophyticus</i>	Lysostaphin resistant strain	KCTC3345
	RN4220_pRMC2	For amplification of pRMC2 vector in <i>S. aureus</i> RN4220 as cloning intermediate, Cm ^r	This study
	RN4220_pRMC2_ <i>shmT</i>	For amplification of pRMC2_ <i>shmT</i> in <i>S. aureus</i> RN4220 as cloning intermediate, Cm ^r	
	Δ <i>shmT</i>	Knock out of <i>shmT</i> gene, Em ^r	Nebraska library
	SAUSA300_pRMC2	SAUSA300_EV, Cm ^r	This study
	Δ <i>shmT</i> _pRMC2	Δ <i>shmT</i> _EV, Cm ^r	This study
	Δ <i>shmT</i> _pRMC2_ <i>shmT</i>	Δ <i>shmT</i> _Comp, Cm ^r	This study
	SAUSA300_pRMC2_ <i>shmT</i>	SAUSA300_OE, Cm ^r	This study
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_ <i>K07-561_fmhC</i>	P _{lac} , <i>K07-561_fmhC</i> , Km ^r , Amp ^r	This study
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_ <i>K07-561_femA</i>	P _{lac} , <i>K07-561_femA</i> , Km ^r , Amp ^r	This study
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_ <i>K07-561_femB</i>	P _{lac} , <i>K07-561_femB</i> , Km ^r , Amp ^r	This study
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_ <i>K07-561_femX</i>	P _{lac} , <i>K07-561_femX</i> , Km ^r , Amp ^r	This study
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_ <i>K07-561_lyrA</i>	P _{lac} , <i>K07-561_lyrA</i> , Km ^r , Amp ^r	This study
	pCR2.1 TOPO_ <i>K07-561_shmT</i>	P _{lac} , <i>K07-561_shmT</i> , Km ^r , Amp ^r	This study

	<i>E. coli</i> DH5 α _pCR2.1 TOPO_K07-204_fmhC	P _{lac} , K07-204_fmhC, Km ^r , Amp ^r	[3]
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_K07-204_femA	P _{lac} , K07-204_femA, Km ^r , Amp ^r	Invitrogen
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_K07-204_femB	P _{lac} , K07-204_femB, Km ^r , Amp ^r	
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_K07-204_femX	P _{lac} , K07-204_femX, Km ^r , Amp ^r	
	<i>E. coli</i> DH5 α _pCR2.1 TOPO_K07-204_lyrA	P _{lac} , K07-204_lyrA, Km ^r , Amp ^r	
	pCR2.1 TOPO_K07-204_shmT	P _{lac} , K07-204_shmT, Km ^r , Amp ^r	
Native	pRMC2	Expression vector under control of tetracycline	
Plasmids		inducible P _{xyl/tetO} , Amp ^r , Cm ^r	
	pCR2.1 TOPO	Expression vector under control of lactose	
		inducible promoter P _{lac} , Km ^r , Amp ^r	

References

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