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ORIGINAL RESEARCH

Surveillance of Antibiotic Resistance and Molecular Epidemiology of *Staphylococcus aureus* in Baotou, Inner Mongolia, China

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Objective: To investigate the distribution, drug resistance patterns, multi-locus sequence typing (MLST), and virulence gene carriage of *Staphylococcus aureus* in the Baotou region of Inner Mongolia, China.

Methods: From January 2018 to December 2020, clinical isolates of *S. aureus* were collected from 11 hospitals in Baotou participating in the China Antimicrobial Resistance Surveillance System (CARSS). Data were analyzed using WHONET 5.6 and SPSS 26.0. Ninety methicillin-resistant *S. aureus* (MRSA) strains from 2020 were typed by MLST, with results analyzed via the eBURST program. Ninety randomly selected methicillin-sensitive *S. aureus* (MSSA) strains from 2020, along with the 90 MRSA strains, underwent polymerase chain reaction to detect 20 virulence genes, including *clfa, clfb, fnbB, scn, chp, sak, coa, nuc, ebps, eno, cna*, and *bbp*.

Results: A total of 2453 *S. aureus* strains, including 309 MRSA, were isolated. Secretions were the main source for *S. aureus* (38.0%), and sputum for MRSA (16.7%). MRSA showed higher resistance to most antimicrobials than MSSA. Among eight identified sequence types, ST59 dominated (60%), mainly linked to hospital-acquired surgical infections (66.7%, 36/54). All strains carried *hla, hld, nuc, clfa*, and *clfb* genes, and over 90% carried *sak, scn*, and *coa* genes. The *fnb*B gene detection rate was significantly higher in MSSA (83%) than in MRSA (15%). Detection rates of *chp, pvl*, and *Luk*ED genes varied significantly among sequence types. The detection rate of the *pvl* gene was 67% in ST22 strains, which was higher than that observed in the ST59, ST398, and ST25 strains.

Conclusion: The isolation rates of *S. aureus* and MRSA in Baotou are lower than the national averages, with no strains resistant to vancomycin, linezolid, or teicoplanin. The predominant MRSA strain in this region is ST59. The *S. aureus* strains in this region carry a large number of virulence genes, indicating high virulence.

Keywords: antibiotic resistance, methicillin-resistant *Staphylococcus aureus*, multilocus sequence typing, virulence gene

Introduction

Staphylococcus aureus was first discovered in the 1880s by Ogston in pus and isolated shortly thereafter by Rosenbach. Over time, through adaptation to the human host and medical environments, *S. aureus* has evolved into an important opportunistic pathogen for humans. It can asymptomatically colonize various body sites, such as the pharynx, nasal cavity, axillae, and perineum, while also serving as a major cause of skin infections, endocarditis, and bacteremia.[1,2] Bacteria have developed complex mechanisms of antibiotic resistance to ensure their survival within the host. The increasing incidence of *S. aureus* infections has been accompanied by the prevalence of antibiotic-resistant strains, particularly those resistant to methicillin. In the United States, the proportion of infections caused by methicillin-resistant

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Staphylococcus aureus (MRSA) has steadily increased, now accounting for 60% to 70% of cases. The death toll from MRSA infections has far surpassed the combined deaths caused by acquired immunodeficiency syndrome, tuberculosis, and viral hepatitis.[3] In most cases, *S. aureus* develops antibiotic resistance by acquiring resistance genes. MRSA strains, however, exhibit intrinsic methicillin resistance and increased resistance to conventional antibiotics due to genetic mutations. Given the rapid emergence of antibiotic-resistant *S. aureus* strains and the lack of new antibiotics in development, there is an urgent need for alternative strategies to combat resistant *S. aureus*.

Multilocus sequence typing (MLST) technology allows for sequencing analysis of seven housekeeping genes to determine the corresponding sequence types (STs). STs have distinct geographic distribution characteristics; for example, ST5 and ST8 are common in the United States and Japan, while ST80 is widely prevalent in the Middle East.[4] MLST in combination with staphylococcal cassette chromosome *mec* typing has become a critical approach for investigating the global epidemiology of various MRSA clones. A key feature of the pathogenicity of *S. aureus* is the production of a large number of virulence factors, including secreted proteins, enzymes, and toxins, that are used to establish and sustain infections.[5] These virulence factors enable *S. aureus* to evade host defenses and damage host cells. In the clinical treatment of infectious diseases, MRSA has shown high virulence and resistance to therapy, primarily due to the presence of these virulence factors. Therefore, in-depth studies of virulence factors and their pathogenic mechanisms hold significant potential for advancing the clinical treatment of *S. aureus* infections.

This study analyzed the distribution and resistance patterns of *S. aureus* collected from 11 tertiary hospitals in Baotou, Inner Mongolia, China, that participated in the China Antimicrobial Resistance Surveillance System (CARSS) from January 2018 to December 2020. Using MLST to investigate the epidemiological characteristics of all 90 MRSA strains isolated in 2020 from The First Affiliated Hospital of Baotou Medical College, the study aimed to understand the epidemiological and resistance characteristics of *S. aureus* in this region. Additionally, it examined the presence of 20 virulence genes, including *scn, chp, sak, coa, nuc, ebps, eno, cna, tst*, and *bbp*, and their associations with virulence.

Materials and Methods

Materials

Bacterial Strains

Staphylococcus aureus isolates were collected from 11 CARSS member hospitals in Baotou between January 2018 and December 2020. All samples were derived from different patients, resulting in a total of 2453 *S. aureus* strains. The quality control strain, *S. aureus* ATCC 25923, was purchased by the National Center for Clinical Laboratories. All 90 MRSA strains isolated in 2020 from The First Affiliated Hospital of Baotou Medical College were selected, and 90 MSSA strains from The First Affiliated Hospital of Baotou Medical College were randomly chosen as controls.

Instruments and Reagents

The bacterial identification and drug susceptibility tests were conducted using the VITEK 2 Compact system (bioMérieux, Craponne, France) or the BD Phoenix-100 system (BD, Mississauga, ON, Canada) with corresponding reagents. MH agar, MH agar containing 5% defibrinated sheep blood, and Columbia blood agar plates were provided by Autobio (Zhengzhou, China). Antimicrobial susceptibility test disks were purchased from Oxoid (Hampshire, UK); E-test strips for penicillin, cefoxitin, linezolid, and vancomycin were obtained from Autobio; and LB broth was obtained from Aobox (Beijing, China).

The bacterial genomic DNA extraction kit was purchased from Tiangen Biotech (Beijing, China). Polymerase chain reaction (PCR) reagents included 2× Taq PCR MasterMix (GreatOcean Biotech, Beijing, China), agarose (Solarbio, Beijing, China), 50× TAE buffer (Solarbio), ethidium bromide staining solution (10 mg/mL; Solarbio), and a 100 bp DNA marker (Solarbio).

Methods

Cultivation and Identification of S. aureus

Isolated bacterial strains were streaked onto Columbia blood agar plates and incubated at 35°C for 24 hours. Bacterial identification was conducted using the VITEK 2 Compact or BD Phoenix-100 system.

Antibiotic Susceptibility Testing

Antibiotic susceptibility tests were performed using the VITEK 2 Compact or BD Phoenix-100 system. For antibiotics not covered by these systems, susceptibility tests were conducted using the Kirby-Bauer disk diffusion method or E-test per Clinical and Laboratory Standards Institute (CLSI) guidelines. The results were interpreted following the 2023 CLSI standards.[6]

Identification of MRSA

MRSA identification was performed using the cefoxitin disk diffusion method in accordance with CLSI standards.[6] Strains with an inhibition zone diameter of ≥22 mm were classified as methicillin-sensitive and those with a diameter of ≤21 mm as methicillin-resistant. The strains confirmed to harbour the *mecA* gene by PCR.[7]

Bacterial Genomic DNA Extraction

A single bacterial colony was inoculated into 5 mL of LB broth and incubated at 37°C and 150 rpm for 18 hours. Three milliliters of the bacterial culture were centrifuged at 10,000 rpm (~11,500 ×*g*) for 1 minute. The pellet was treated with 120 μL of lysostaphin (100 mg/mL) at 37°C for 40 minutes. The specific procedure followed the instructions for the genomic DNA extraction kit, and the extracted DNA was stored in aliquots at 4°C for later use.

MLST

DNA sequences of seven housekeeping genes (*arcc, aroe, glpf, gmk, pta, tpi*, and *yqil*) were analyzed according to the MLST website (www.mlst.net). A 25 μL PCR mixture was prepared according to the following composition: 2×TaqPCR MasterMix (12.5 μL), DNA (1 μL), Upstream primer (2 μL), downstream primer (2 μL), ddH2O (7.5 μL), and the PCR cycling conditions were set as step one: pre denaturation (95°C, 5 min, 1 cycle), step two: denaturation (95°C, 30 s), annealing (55°C, 33 s), extension (72°C, 1 min), 35 cycles, step three: final extension (72°C, 5 min), 1 cycles. The PCR products were sent to Great Ocean Biotech (Beijing, China) for bidirectional sequencing. STs were identified using the *S. aureus* MLST database (https://pubmlst.org/organisms/staphylococcus-aureus).

PCR Detection of Virulence Genes

The primer sequences for the 20 virulence genes were *scn, chp, sak, coa, nuc, ebps, eno, cna, bbp, fnbB, clfa, clfb, seb, seh, tst, eta, pvl, LukED, hla*, and *hld*, as previously described.[6] The names and functions of each virulence gene are shown in Table 1. Sequence of virulence gene primers are shown in Table 2. A 25 μL PCR mixture was prepared

Table 1 Name and Function of Virulence Genes

Genes	Virulence Factors	Functions
clfA	Clumping factor	ClfA and ClfB bind to different sites in fibrinogen. ClfA binds to the γ-chain whereas ClfB binds to the α-chain. ClfA through its fibrinogen-binding function is a mediator of S. aureus-induced platelet aggregation.
clfB	Clumping factor	ClfA and ClfB bind to different sites in fibrinogen. ClfA binds to the γ-chain whereas ClfB binds to the α-chain.
cna	CAN (Collagen binding protein)	Not generally expressed by the majority of strains. Mediates bacterial adherence to collagen substrates and collagenous tissues, strongly associated with pathogenesis of osteomyelitis and septic arthritis.
ebp	EbpS (Elastin-binding protein)	Promotes bacterial colonization to facilitate pathogenesis.
fnbB	FnBPs (Fibronectin binding proteins)	Facilitates attachment of the staphylococcus to host cells. May function as invasin to invade non-professional phagocytes.
hly/hla	α-hemolysin	Forms pores in cell membrane to kill or limit the ability of neutrophils. Inducing cellular damage that triggers cytokine production.
hld	δ-hemolysin	Capable of lysing erythrocytes and other mammalian cells, as well as subcellular structures.
lukD	LukED (Leukotoxin)	The leukotoxins lyse cells of the leukotocytic lineage and also kill neutrophils, LukED have demonstrated lytic activity against red blood cells
lukE	LukED (Leukotoxin)	The leukotoxins lyse cells of the leukotocytic lineage and also kill neutrophils, LukED have demonstrated lytic activity against red blood cells
lukS-PV *lukF-PV* *lukM*	PVL (Panton-Valentine leukocidin)	Induce cell activation linked to a Ca2+ influx, and pore formation as two consecutive and independently inhibitable events.

(*Continued*)

Table 1 (Continued).

Genes	Virulence Factors	Functions
seb	SE (Staphylococcal enterotoxin)	Responsible for the symptoms of food poisoning.
seh	SE (Staphylococcal enterotoxin)	Responsible for the symptoms of food poisoning.
tst	TSST-1 (Toxic shock syndrome toxin-1)	Responsible for the symptoms of toxic shock syndrome.
coa	Staphylocoagulase	Promotes the pathogenesis of S. aureus abscess formation and lethal bacteremia.
sak	Staphylokinase	Contributes to spread of the bacteria. Activates plasminogen (not in a controlled way) to dissolve clots, also destroys the ECM and fibrin fibers that hold cells together, thus allowing the bacteria escape from abscesses.
nuc	Thermonuclease nuc	
chp	CHIPS (Chemotaxis inhibitory protein of Staphylococcus)	Evasion from innate immunity.
scn	SCIN (Staphylococcal complement inhibitor)	Evasion from innate immunity.

Table 2 Sequence of Virulence Gene Primers

Genes	Primers	bp
scn	F: ATACTTGCGGGAACTTTAGCAA R: TTTTAGTGCTTCGTCAATTTCG	320
chp	F: TTTTTAACGGCAGGAATCAGTA R: TGCATATTCATTAGTTTTTCCAGG	404
sak	F: TGAGGTAAGTGCATCAAGTTCA R: CCTTTGTAATTAAGTTGAATCCAGG	403
coa	F: CCGCTTCAACTTCAGCCTAC R: TTAGGTGCTACAGGGGCAAT	204
nuc	F: AGTTCAGCAAATGCATCACA R: TAGCCAAGCCTTGACGAACT	400
ebps	F: CATCCAGAACCAATCGAAGAC R: AGTTACATCATCATGTTTATCTTTTG	188
eno	F: ACGTGCAGCAGCTGACT R: CAACAGCATCTTCAGTACCTTC	301
cna	F: AAAGCGTTGCCTAGTGGAGA R: AGTGCCTTCCCAAACCTTTT	192
bbp	F: AACTACATCTAGTACTCAACAACAG R: ATGTGCTTGAATAACACCATCATCT	574
fnbB	F: GTAACAGCTAATGGTCGAATTGATACT R: CAAGTTCGATAGGAGTACTATGTTC	524
clfa	F: ATTGGCGTGGCTTCAGTGCT R: CGTTTCTTCCGTAGTTGCATTTG	292
clfb	F: GCAGCATTTACTACCGGTTC R: CTACAACAGAGCCAGCTTCA	301
seb	F: ATTCTATTAAGGACACTAAGTTAGGGA R: ATCCCGTTTCATAAGGCGAGT	404
seh	F: CAATCACATCATATGCGAAAGCAG R: CATCTACCCAAACATTAGCACC	376
tst	F: TTCACTATTTGTAAAAGTGTCAGACCCACT R: TACTAATGAATTTTTTTATCGTAAGCCCTT	180
eta	F: ACTGTAGGAGCTAGTGCATTTGT R: TGGATACTTTTGTCTATCTTTTTCATCAAC	190
pvl	F: ATCATTAGGTAAAATGTCTGGACATGATCCA R: GCATCAASTGTATTGGATAGCAAAAGC	433

(*Continued*)

Table 2 (Continued).

Genes	Primers	bp
LukED	F: TGAAAAAGGTTCAAAGTTGATACGAG R: TGTATTCGATAGCAAAAGCAGTGCA	269
hla	F: CTGATTACTATCCAAGAAATTCGATTG R: CTTTCCAGCCTACTTTTTTATCAGT	209
hld	F: AAGAATTTTTATCTTAATTAAGGAAGGAGTG R: TTAGTGAATTTGTTCACTGTGTCGA	111

Table 3 PCR Cycle Conditions of Virulence Genes

Genes	Step One		Step Two				Step Three	
	Pre Denaturation	Cycle	Denaturation	Annealing	Extension	Cycle	Final Extension	Cycle
hla, hld, pvl, lukED, seh, seb, eta, tsst, scn, chp, sak,	95°C, 5min	1×	94°C, 1min	55°C, 1min	72°C, 1min	35×	72°C, 10min	1×
ebps, eno, cna, bbp, fnbB, clfa, clfb	94°C, 5min	1×	94°C, 1min	55°C, 1min	72°C, 1min	25×	72°C, 10min	1×
coa, nuc	94°C, 5min	1×	94°C, 1min	56°C, 1min	68°C, 1min	30×	72°C, 7min	1×

according to the following composition: 2×TaqPCR MasterMix (12.5 μL), DNA (1 μL), Upstream primer (2 μL), downstream primer (2 μL), ddH2O (7.5 μL), and the PCR cycling conditions were set as Table 3.

Statistical Methods

Data analysis was conducted using WHONET 5.6 and SPSS 26.0 software. Categorical data were expressed as counts or percentages, and the χ^2 test was performed at a significance level of $P<0.05$.

Results

Isolation of S. aureus and MRSA

From 2018 to 2020, a total of 24,297 bacterial strains were isolated from 11 CARSS member hospitals in Baotou, including 2453 *S. aureus* strains, at an isolation rate of 10.1%. These isolates were collected from patients who had cough, fever and other clinical symptoms related to infection and whose peripheral white blood cell and/or neutrophil counts were elevated. The annual isolation rates of *S. aureus* from 2018 to 2020 were 11%, 10.2%, and 8.9%, respectively. Over the three years, 309 MRSA strains were identified at an isolation rate of 12.6%. The annual isolation rates of MRSA from 2018 to 2020 were 11.7%, 13.7%, and 12.4%, respectively, with the highest rate observed in 2019 and the lowest in 2018 (Table 4). The decrease in SAU separation rate from 2019 to 2020 is statistically significant (P=0.004), indicating a significant decrease in SAU prevalence. The isolation rate of MRSA has significantly increased from 2018 to 2019 (P=0.036).

Table 4 Isolation of *Staphylococcus aureus* and Methicillin-Resistant *Staphylococcus aureus* Strains Between 2018 and 2020

Year	Total Pathogens (Strains), n	SAU (Strains), n	SAU Isolation Rate, %	MRSA (Strains), n	MRSA Isolation Rate, %
2018	7773	866	11.1	101	11.7
2019	8430	864	10.2	118	13.7**
2020	8094	723	8.9*	90	12.4
Total	24,297	2453	10.1	309	12.6

Notes: This note applies to the tables that follow.*: 2019 vs 2020, P=0.004.**: 2018 vs 2019, P=0.036.
Abbreviation: SAU, *Staphylococcus aureus*.

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Specimen Types of S. aureus Infections and MRSA Isolation

The 2453 strains of *S. aureus* were isolated from various sources, with the majority of strains from wound secretions (932, 38.0%), followed by sputum (462, 18.8%), pus (210, 8.6%), and blood (164, 6.7%). The isolation rates of *S. aureus* from different specimen types showed statistically significant differences ($P<0.05$). The highest isolation rate of MRSA was observed in sputum (16.7%) and the lowest in pus (8.6%). The MRSA isolation rates also differed significantly among specimen types ($P<0.05$). The results are shown in Table 5.

Antibiotic Resistance of MRSA and MSSA to Common Antimicrobial Agents

The resistance rates of MRSA to penicillin, erythromycin, and clindamycin were all greater than 70%. MSSA exhibited high resistance rates to penicillin (90.1%) and erythromycin (70.1%). MRSA demonstrated significantly higher resistance rates to penicillin, clindamycin, tetracycline, and ciprofloxacin than MSSA ($P<0.05$). No strains resistant to vancomycin, linezolid, or teicoplanin were detected (Table 6).

Table 5 Specimen Types of *Staphylococcus aureus* Infections and Isolation of Methicillin-Resistant *Staphylococcus aureus* Strains

Specimen Type	Total Strains, n	Constituent Ratio, %	MRSA (Strains), n	MRSA Isolation Rate, %
Wound Secretions	932	38.0	109	11.7
Sputum	462	18.8	77	16.7
Pus	210	8.6	18	8.6
Blood	164	6.7	20	12.2
Other	685	27.9	85	12.4
χ^2	–	1067.181	–	10.775
P	–	0.000	–	0.029

Notes: "-" indicates not applicable or not analyzed. This note applies to the tables that follow.

Table 6 Resistance of Methicillin-Resistant and Methicillin-Sensitive *Staphylococcus aureus* to Common Antimicrobial Agents

Antibiotic	MRSA (n=309)		MSSA (n=2144)		χ^2	*P*
	R	S	R	S		
Penicillin G	100	0	90.1	9.8	33.445	0.000
Erythromycin	84.6	14.4	70.1	29.2	27.585	0.000
Clindamycin	73.1	26.3	53.2	46.5	43.441	0.000
Tetracycline	44.4	55.2	6.8	93.0	372.666	0.000
Ciprofloxacin	32.5	63.0	9.2	87.2	136.295	0.000
Levofloxacin	27.0	70.1	8.0	90.4	102.897	0.000
Moxifloxacin	21.9	73.3	6.0	92.1	93.488	0.000
Gentamicin	20.9	76.1	17.1	79.7	2.857	0.091
Trimethoprim/sulfamethoxazole	17.0	83.0	24.5	75.5	8.067	0.005
Rifampin	13.7	85.8	0.7	99.2	188.486	0.000
Teicoplanin	0	100	0	100	–	–
Linezolid	0	100	0	100	–	–
Vancomycin	0	100	0	100	–	–

Note: This note applies to the tables that follow.
Abbreviations: R, resistance rate; S, sensitivity rate; MSSA, methicillin-sensitive *Staphylococcus aureus*.

Table 7 Resistance of Methicillin-Resistant *Staphylococcus aureus* to Common Antimicrobial Agents in Different Types of Specimens

Antibiotic	Secretions (n=109)		Sputum (n=77)		Pus (n=18)		Blood (n=20)		Other (n=85)		χ^2	*P*
	R	S	R	S	R	S	R	S	R	S		
Penicillin G	100	0	100	0	100	0	100	0	100	0	–	–
Erythromycin	80.7	19.3	81.8	16.9	94.4	5.6	90.0	5.0	89.6	7.8	5.047	0.282
Clindamycin	75.0	24.0	73.5	25.0	90.0	10.0	75.0	25.0	84.0	16.0	4.058	0.398
Tetracycline	44.6	55.4	42.9	57.1	33.3	66.7	61.1	38.9	33.9	66.1	5.798	0.215
Ciprofloxacin	24.7	72.7	41.7	53.3	4.5	86.4	43.8	50.0	27.1	66.1	13.986	0.007
Gentamicin	18.7	79.4	32.5	63.6	25.8	74.2	30.0	65.0	14.1	80.8	9.935	0.042
Levofloxacin	18.0	77.0	47.6	52.4	4.8	95.2	40.0	60.0	22.2	75.0	27.931	0.000
Moxifloxacin	15.4	80.2	41.5	49.1	14.5	85.5	25.0	75.0	14.9	79.1	21.863	0.000
Trimethoprim/sulfamethoxazole	14.4	85.6	18.9	81.1	5.6	94.4	35.0	65.0	17.6	82.4	6.840	0.145
Rifampin	11.8	87.3	21.1	78.9	5.6	94.4	20.0	78.4	5.5	94.5	9.997	0.040
Vancomycin	0	100	0	100	0	100	0	100	0	100	–	–
Linezolid	0	100	0	100	0	100	0	100	0	100	–	–
Teicoplanin	0	100	0	100	0	100	0	100	0	100	–	–

Antibiotic Resistance of MRSA in Different Specimen Types

In all specimen types, MRSA demonstrated resistance rates exceeding 70% to penicillin, erythromycin, and clindamycin. Resistance rates to rifampin were below 22% across specimen types. Ciprofloxacin resistance was highest in strains isolated from blood specimens (43.8%) and lowest in strains from pus (4.5%). Gentamicin resistance was highest in strains from sputum (32.5%) and lowest in strains from secretions (18.7%). The resistance rates of MRSA to ciprofloxacin, gentamicin, levofloxacin, moxifloxacin, and rifampin varied significantly among specimen types ($P<0.05$). The results are shown in Table 7.

MLST of MRSA

MLST analysis of 90 MRSA strains identified eight STs. The predominant STs were ST59 (54, 60%), ST398 (11, 12.2%), ST239 (7, 7.8%), ST22 (7, 7.8%), ST25 (5,5.6%), ST5 (2,2.2%), ST188 (2,2.2%), ST7 (2,2.2%).

MLST of MRSA Associated with Different Infection Types

When the STs of the 90 MRSA strains were grouped by infection types, ST59 was mainly associated with surgical infections (66.7%, 36/54), followed by respiratory infections (12.5%, 7/54) and other infections (12.5%, 7/54). ST59 strains predominantly originated from orthopedic hand, foot, and ankle surgeries; ostomy wound clinics; and trauma departments. Two ST5 strains were linked to respiratory infections, while two ST188 strains and two ST7 strains were associated with other infections (Table 8).

Table 8 Multilocus Sequence Typing of Methicillin-Resistant *Staphylococcus aureus* with Different Infection Types

Sequence Type	Infection Type (Strains)*			
	Surgical Infections (Strains), n	Respiratory Infections (Strains), n	Bloodstream Infections (Strains), n	Other Infections (Strains), n
ST59	36	6	5	6
ST398	7	2	–	2
ST239	5	2	–	–
ST22	5	–	2	–

(*Continued*)

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Table 8 (Continued).

Sequence Type	Infection Type (Strains)*			
	Surgical Infections (Strains), n	Respiratory Infections (Strains), n	Bloodstream Infections (Strains), n	Other Infections (Strains), n
ST25	3	–	2	–
ST5	–	2	–	–
ST188	–	–	–	2
ST7	–	–	–	2

Notes: *: There is a highly significant correlation ($P<0.001$) between ST type and infection type, with ST59 dominating surgical infections, ST5 specifically associated with respiratory infections, and ST188/ST7 specifically associated with other infections.

Detection of Virulence Genes in MRSA and MSSA

Among the 20 virulence genes tested in 90 MRSA and 90 MSSA strains, all strains carried the *hla, hld, nuc, clfa*, and *clfb* genes. Over 90% of the strains carried the *sak, scn*, and *coa* genes, among which the *coa* gene showed a 100% detection rate in MSSA strains. Only 12 strains carried the *seh* gene, seven carried the *bbp* gene, four carried the *tst* gene, and two MRSA strains carried the *eta* gene. Two MRSA strains each carried 17 virulence genes, while five MSSA strains each carried 14 virulence genes. The detection rate of the *fnbB* gene was significantly ($P<0.05$) higher in MSSA (83%) than in MRSA (15%). The detection rate of the *ebps* gene was 6% in both MRSA and MSSA (Figure 1).

Detection of Virulence Genes in Different Types of S. aureus Infections

The virulence genes of 180 strains of *Staphylococcus aureus* were grouped according to infection type. The *bbp, seh*, and *LukED* genes were only present in strains isolated from surgical and bloodstream infections, the *tst* gene was only present in strains isolated from surgical and respiratory infections, and the *eta* gene was only detected in strains isolated from other infections. The detection rate of the *chp* gene in surgical infections (82%) was higher than that in bloodstream infections (50%), $P<0.05$. The detection rate of the *PVL* gene in bloodstream infections (50%) was higher than that in surgical infections (18%), $P<0.05$. The detection rate of the *LukED* gene in surgical infections (45%) and bloodstream infections (40%) was higher than that in respiratory infections (0%), $P<0.05$. See Table 9.

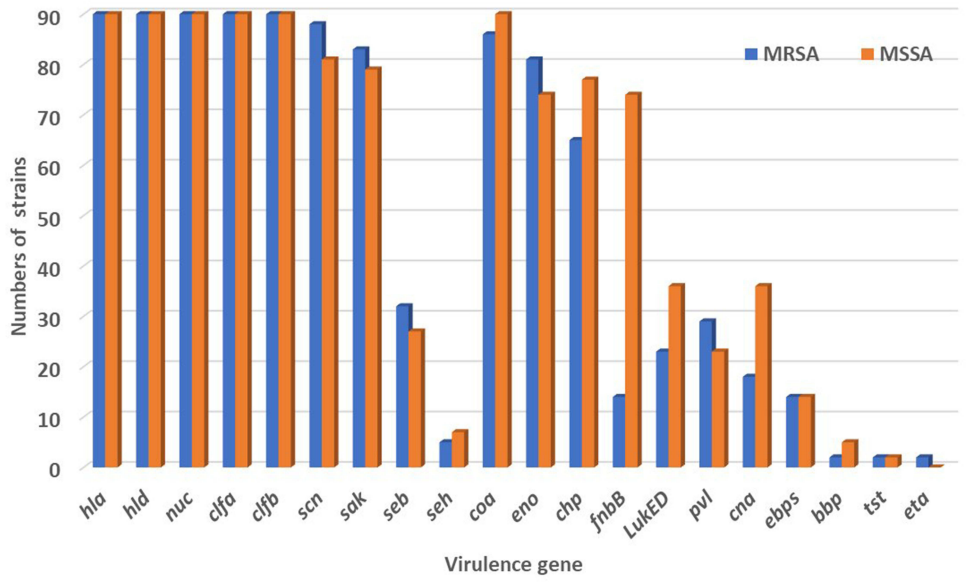

Figure 1 Virulence Genes in Methicillin-Resistant and Methicillin-Sensitive *Staphylococcus aureus*.

Table 9 Detection of Virulence Genes of *Staphylococcus aureus* in Different Infection Types

Genes	Infection Types			Surgical vs Respiratory		Surgical vs Bloodstream		Respiratory vs Bloodstream	
	Surgical Infection	Respiratory Infection	Bloodstream Infection	χ^2	*P*	χ^2	*P*	χ^2	*P*
clfa	38 (100%)	9 (100%)	10 (100%)	–	–	–	–	–	–
clfb	38 (100%)	9 (100%)	10 (100%)	–	–	–	–	–	–
fnbB	17 (45%)	3 (33%)	5 (50%)	0.387	0.534	0.088	0.766	0.540	0.463
eno	34 (89%)	7 (77%)	8 (80%)	0.894	0.344	0.650	0.420	0.014	0, 906
ebps	7 (18%)	1 (11%)	2 (20%)	0.275	0.600	0.013	0.909	0.281	0.596
scn	36 (95%)	8 (89%)	10 (100%)	0.416	0.519	0.549	0.459	1.173	0.279
coa	37 (97%)	9 (100%)	10 (100%)	0.242	0.623	0.269	0.604	–	–
chp	31 (82%)	7 (78%)	5 (50%)	0.068	0.794	4.211	0.040	1.571	0.210
sak	35 (92%)	9 (100%)	8 (80%)	0.759	0.384	1.243	0.265	2.012	0.156
nuc	38 (100%)	9 (100%)	10 (100%)	–	–	–	–	–	–
cna	13 (34%)	2 (22%)	5 (50%)	0.481	0.488	0.842	0.359	1.571	0.210
bbp	2 (5%)	–	1 (10%)	–	–	0.303	0.582	0.950	0.330
pvl	7 (18%)	3 (33%)	5 (50%)	0.966	0.326	4.211	0.040	0.540	0.463
seb	10 (26%)	5 (56%)	3 (30%)	2.863	0.091	0.054	0.816	1.269	0.260
tst	1 (3%)	1 (11%)	–	1.284	0.257	0.269	0.604	–	–
hla	38 (100%)	9 (100%)	10 (100%)	–	–	–	–	–	–
hld	38 (100%)	9 (100%)	10 (100%)	–	–	–	–	–	–
seh	3(8%)	–	2 (20%)	0.759	0.384	1.243	0.265	2.012	0.156
LukED	17 (45%)	–	4 (40%)	6.308	0.012	0.072	0.788	4.560	0.033
eta	–	–	–	–	–	–	–	–	–

Detection of Virulence Genes in Different ST Types of MRSA

The virulence genes of 90 MRSA strains were grouped according to ST type. The *ebps, bbp*, and *tst* genes were only detected in ST59 type, with detection rates of 25%, 4%, and 4%, respectively. ST398 did not detect *seh*, and the *seb* detection rate was only 20%. The detection rate of the *PVL* gene in ST59 type was 33%, and the detection rate in ST22 type was 67%. ST59 and ST398 MRSA carried at least 10 virulence genes, while ST239 MRSA carried at least 11 virulence genes. See Table 10.

Table 10 Detection of MRSA Virulence Genes in Different ST Types

Genes	ST59	ST398	ST239	ST22	ST25	ST5	ST188	ST7
hla, hld, clfa, clfb, nuc	54(100%)	11(100%)	7(100%)	7(100%)	5(100%)	2(100%)	2(100%)	2(100%)
scn	54(100%)	9(82%)	7(100%)	7(100%)	5(100%)	2(100%)	2(100%)	2(100%)
sak	52(96%)	9(82%)	5(71%)	7(100%)	5(100%)	2(100%)	2(100%)	2(100%)
seb	21(39%)	4(36%)	7(100%)	–	–	–	2(100%)	–
seh	–	–	2(29%)	–	–	–	–	2(100%)
coa	50(93%)	11(100%)	7(100%)	7(100%)	5(100%)	2(100%)	2(100%)	2(100%)
eno	52(96%)	7(64%)	7 (100%)	7(100%)	5(100%)	–	2(100%)	2(100%)
chp	36(67%)	9(82%)	5(71%)	7(100%)	5(100%)	2(100%)	2(100%)	–
cna	7(13%)	4(36%)	2(29%)	2(29%)	–	–	2(100%)	–

(*Continued*)

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Table 10 (Continued).

Genes	ST59	ST398	ST239	ST22	ST25	ST5	ST188	ST7
ebps	14(26%)	–	–	–	–	–	–	–
bbp	2(4%)	–	–	–	–	–	–	–
tst	2(4%)	–	–	–	–	–	–	–
eta	–	2(18%)	–	–	–	–	–	–
fnbB	2(4%)	4(36%)	2(29%)	–	3(60%)	2(100%)	–	–
LukED	14(26%)	4(36%)	–	2(29%)	–	–	–	2(100%)
pvl	18(33%)	2(18%)	–	5(71%)	3(60%)	–	2(100%)	–

Discussion

Staphylococcus aureus can be carried by patients both prior to and following hospital admission. As a common commensal organism in humans and animals, it is capable of colonizing the nares, axillae, perineum, skin, and numerous other bodily sites. Approximately 15% of the general population is estimated to be persistent carriers of *S. aureus* in the anterior nares. Patients may already be carriers of *S. aureus* upon hospital entry; furthermore, they can acquire the bacterium during their inpatient stay, particularly when undergoing surgical interventions or other invasive procedures. Methicillin-resistant *Staphylococcus aureus* (MRSA) is widely recognized as a dominant nosocomial pathogen, responsible for significant morbidity and mortality on a global scale. MRSA colonization not only elevates the risk of subsequent infection but also contributes to healthcare-associated transmission.[2,8]

The China Antimicrobial Resistance Surveillance System (CARSS) was established in 2005 and is responsible for monitoring the sensitivity and resistance rates of common clinical pathogens in China to various antibiotics, as well as continuously monitoring the changes in bacterial resistance. A total of 11 tertiary hospitals in Baotou, Inner Mongolia participated in CARSS. We analyzed the data uploaded to CARSS from 2018 to 2020, a total of 2453 *S. aureus* strains were isolated in Baotou at an isolation rate of 10.1%, significantly lower than the 32.9% observed in the 2020 China Antimicrobial Surveillance Network.[9] The isolation rate of *S. aureus* in Baotou decreased from 11% in 2018 to 8.9% in 2020, as did that in southern China (9.9–9.5%)[10] but not that in northwest China, which showed a significant increase (3.7–9.7%).[11] The decline in *S. aureus* isolation rate in 2020 may be related to the COVID-19 pandemic, which led to a reduction in both inpatient and outpatient cases compared to 2019 and consequently fewer specimens, particularly from outpatients.

The incidence of MRSA in Asia is much higher than in other parts of the world,[12] with isolation rates in Asia ranging from 28% to 70%.[13] In comparison, the average isolation rates of MRSA in Ireland and Canada are 2.2% and 11.8%, respectively.[14] In our study, the isolation rates of MRSA from 2018 to 2020 were 11.7%, 13.7%, and 12.4%, respectively, showing no obvious trend, but lower than the results reported by the previous team in 2019.[15] The average isolation rate of MRSA was 12.6%, slightly higher than that in Canada but significantly lower than that in Asia (46.9%) and other regions of China (31.6%).[16,17] This rate is below the national average reported by the CARSS but close to the average in the Inner Mongolia Autonomous Region (CARSS 2020 National Antimicrobial Resistance Surveillance Report, http://www.carss.cn/Report/Details?aId=808). These findings indicate that the isolation rates of *S. aureus* and MRSA in Baotou are relatively low.

In the previous study by our team, only the drug resistance analysis of the isolated strains was conducted,[15] and the sample sources were not distinguished. In this study, a detailed subdivision was carried out. The 2453 strains of *S. aureus* in this experiment were derived from secretions, sputum, pus, and blood. Among them, the proportion of *Staphylococcus aureus* in wound secretions was 38.0%, which was higher than that in other specimen types ($P<0.05$), consistent with the conclusion of He et al.[18] The highest isolation rate of MRSA was observed in sputum (16.7%), which was significantly ($P<0.05$) higher than that in blood (12.2%), in which the second-highest isolation rate was observed. As these results

suggest that surgical operations are the primary source of *S. aureus* infections, MRSA should be considered in the diagnosis of clinical pulmonary and bloodstream infections, especially as severe cases of bloodstream MRSA infections can be life-threatening.

Antibiotic susceptibility analysis of MRSA and MSSA revealed that MRSA had a resistance rate of 73.1% to clindamycin, higher than the results reported by Weiner et al (56.2%)[19] and the 2020 CHINET surveillance of bacterial resistance (58.6%). MRSA showed higher resistance rates to penicillin and clindamycin compared to MSSA. These results suggest that although the MRSA isolation rate in Baotou is low, resistance rates to certain antibiotics exceed the national averages. Therefore, clinicians should use antibiotics judiciously, ensuring that empirical treatment aligns with local surveillance data to guide the appropriate selection of antimicrobial agents.

The resistance rates of MRSA to ciprofloxacin, gentamicin, levofloxacin, moxifloxacin, and rifampin varied significantly among the specimen types. Notably, MRSA isolates from blood samples exhibited the highest resistance rate to ciprofloxacin, while those from pus samples demonstrated the lowest. Similarly, gentamicin resistance rate was highest in MRSA strains from sputum and lowest in those from wound secretions. These variations reflect the heterogeneity of *S. aureus* in antibiotic resistance and virulence systems, which are closely linked to host conditions, treatment regimes, and infection severity. Therefore, clinical treatment strategies should be tailored based on specimen type and resistance profiles to avoid the overuse of antimicrobial agents, which could lead to the proliferation of resistant strains.

In previous studies by our team, only drug resistance monitoring was conducted, without molecular epidemiological and virulence mechanism studies on the isolated *S. aureus*.[15] Based on the previous findings, our team has carried out further research. The MLST analysis of 90 MRSA strains identified eight STs, including ST59, ST398, ST239, ST22, ST25, ST5, ST188, and ST7. Among them, ST59 was the predominant type, representing 60.0% of the isolates, consistent with the findings of Cui et al (63.7%).[20] ST59 was first reported in Taiwan in 2004 and has since evolved into one of the most prevalent STs in East Asia. Although ST59 has also been reported in Europe and North America, it remains uncommon in those regions. In Taiwan, ST59 accounts for 56% of CA-MRSA infections in children, and ST59-related sepsis has been observed in nearly 70% of pediatric CA-MRSA infections. Furthermore, 66.7% of ST59 infections in our study were linked to surgical operations, primarily in orthopedic hand, foot, and ankle surgeries; ostomy wound clinics; and trauma departments, indicating that skin and soft tissue remain the primary sites of ST59 infections.

The virulence factors of *S. aureus* are crucial during pathogenesis.[21,22] Consistent with most studies from mainland China,[23,24] nearly all strains in our study carried the *hla, hld, nuc, clfa* and *clfb* genes, confirming these as the most common virulence factors in *S. aureus* and indicating no regional variation in their distribution. Over 90% of the strains carried the *sak, scn*, and *coa* genes. Only 12 strains carried the *seh* gene, seven carried the *bbp* gene, four carried the *tst* gene, and two MRSA strains carried the *eta* gene. The detection rate is lower than that reported by Li et al.[25] Two MRSA strains each carried 17 virulence genes, while five MSSA strains each carried 14 virulence genes.

The detection rate of the *fnbB* gene was significantly higher in MSSA (83%) than in MRSA (15%). The fibronectin-binding proteins expressed by *fnbA* and *fnbB* act as mediators for cell signaling and actin cytoskeleton rearrangement and facilitate the invasion of *S. aureus* into tissues. The *fnbB* gene is frequently detected in strains from keratitis, osteomyelitis, medical device surfaces, and orthopedic infections. In the current study, the detection rate of *fnbB* was 49%, which is consistent with the 43.6% detection rate reported by Soltani et al[26] but higher than the 29.5% detection rate reported in a study on bloodstream infections.[27] These findings indicate that *fnbB* exhibits heterogeneity in detection rates across specimen types. Contrary to previous studies showing higher biofilm production and *fnbA*/*fnbB* expression in MRSA strains compared to MSSA,[28] we found a significantly higher *fnbB* detection rate of 83% in MSSA than 15% in MRSA ($P<0.05$). Further investigation is needed to determine whether this difference is related to variable expression of biofilm-associated genes and regulatory elements in MRSA genomes.

The detection rate of the *bbp* gene in the current study was only 4%, consistent with the findings of Kot et al.[29] Previous studies have shown that *S. aureus* strains carrying the *bbp* gene have a greater ability to produce biofilms than those with other biofilm-related genes, although its low prevalence limits further research. The *bbp* gene encodes a protein with high affinity for bone sialoprotein and has been linked to hematogenous osteomyelitis and arthritis. These

factors explain its detection in three patients in the present study, including two with lumbar spinal stenosis and one with acute osteomyelitis.

Panton-Valentine leukocidin is an endogenous factor in clinical infections that exerts significant cytotoxic effects on neutrophils. As the reported positivity rate of the *pvl* gene ranges from 2% to 35%, the 29% positivity rate in the present study is close to the upper limit. Elevated *pvl* expression increases MRSA virulence, causing necrotizing pneumonia with mortality rates as high as 75%.[30] Our study found that the detection rate of *pvl* was significantly higher ($P<0.05$) in *S. aureus* strains from bloodstream infections (50%) than those from surgical infections (18%), suggesting that the *pvl* gene is frequently associated with deep infections. Additionally, the *pvl* gene was slightly more prevalent in MRSA (33%) than in MSSA (25%). Early studies suggested that the *pvl* gene was a genetic marker for MRSA identification. However, Motamedi et al[31] found no significant association between *pvl* and *mecA* in *S. aureus*, and MSSA may secrete a relatively high level of toxins, particularly Panton-Valentine leukocidin.[32] Leukocidin ED induces the death of neutrophils and lymphocytes by forming oligomeric pore-like structures.

The *chp, sak*, and *scn* genes are specific to and commonly found in *S. aureus* strains derived from humans.[33] In our study, the prevalence rates of *chp, sak*, and *scn* were 79%, 90%, and 94%, respectively, which supports this conclusion. Additionally, the detection rate of *chp* was significantly higher ($P<0.05$) in *S. aureus* strains from surgical infections (82%) than those from bloodstream infections (50%).

Conclusions

This study reveals distinct epidemiological and molecular characteristics of S. aureus and MRSA in the region. While isolation rates are lower than national averages, elevated resistance to key antibiotics underscores the need for tailored antimicrobial stewardship. Clinical management should account for source-specific resistance patterns, particularly given MRSA's higher resistance across most antibiotics and its predominance in respiratory specimens. The dominance of the ST59 lineage—strongly associated with healthcare-associated skin and soft tissue infections in high-risk surgical settings (orthopedics, trauma, wound care)—demands intensified infection control in these units, including strict antisepsis protocols, environmental decontamination, and preoperative MRSA screening.

Notably, the near-universal carriage of virulence genes (*hla, hld, nuc, clfa, clfb, sak, scn, coa*) among isolates indicates significant pathogenic potential, amplifying concerns for severe surgical site infections or outbreaks. Public health efforts must prioritize surveillance of these hypervirulent strains, especially in vulnerable surgical populations. The stark contrast in *fnbB* gene prevalence between MSSA (83%) and MRSA (15%) warrants investigation into its role in adhesion or immune evasion. Future studies should: (1) expand regional surveillance to track ST59 spread and virulence gene distribution; (2) correlate ST59 infections with clinical outcomes (eg, treatment failure, mortality); and (3) evaluate targeted decolonization protocols in high-risk surgical cohorts to mitigate ST59-associated infections.

Abbreviations

CA, community-acquired; CARSS, China Antimicrobial Resistance Surveillance System; CC, clonal complex; CLSI, Clinical and Laboratory Standards Institute; CNA, collagen-binding adhesin; HA, hospital-acquired; MLST, Multilocus sequence typing; MRSA, methicillin-resistant *Staphylococcus aureus*; MSSA, methicillin-sensitive *Staphylococcus aureus*; PCR, Polymerase chain reaction; SAU, *Staphylococcus aureus*; ST, sequence type.

Data Sharing Statement

Experimental data related to this study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of The First Affiliated Hospital of Baotou Medical College, Inner Mongolia University of Science & Technology (2025-1-20/K018-01). The data are anonymous, and the requirement for informed consent was therefore waived.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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