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Genetic characterization of Panton–Valentine leukocidin-producing methicillin-resistant *Staphylococcus aureus* in Western Austria

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Genetische Charakterisierung von Panton-Valentine Leukozidin produzierenden Methicillin-resistenten *Staphylococcus aureus* in Westösterreich

Zusammenfassung

Grundlagen Community-acquired methicillin-resistente *Staphylococcus aureus* (caMRSA) stellen ein zunehmendes gesundheitsökonomisches Problem dar. Sie können speziell bei ansonsten gesunden Patienten schwerwiegende Infektionen auslösen, was vermutlich auf die Fähigkeit einiger Stämme, Panton-Valentine Leukozidin (PVL) zu produzieren, zurückzuführen ist. Das Ziel dieser Studie war es, die Prävalenz von PVL-positiven (PVL$^+$)-MRSA in Westösterreich im Zeitraum von Dezember 2005 bis Mai 2010 zu erheben und identifizierte PVL$^+$-MRSA genetisch zu charakterisieren.

Methodik Sechshundertfünfzig MRSA-Stämme der Medizinischen Universität Innsbruck, von Bezirkskrankenhäusern und von niedergelassenen Ärzten in Westösterreich wurden auf das Vorhandensein des *lukS-lukF* Gens (welches für PVL kodiert) untersucht. Identifizierte PVL$^+$-MRSA wurden auf Antibiotika-Resistenz getestet. Weiteres wurden der SCC*mec*-, *agr*-, MLST- und *spa*-Typ bestimmt sowie auf das Vorhandensein des *arcA*-Gens untersucht.

Ergebnisse Von 650 MRSA, welche von verschiedenen Körperregionen sowohl von hospitalisierten wie auch von nicht-hospitalisierten Patienten isoliert wurden, wurden 31 (4,8 %) als PVL$^+$-MRSA identifiziert. Der in unserer Untersuchung am häufigsten vorkommende *agr*-Typ war *agr*-1 ($n=18$, 58,1 %), die häufigsten SCC*mec*-Typen waren SCC*mec* IV oder die Varianten IVa und IVc ($n=27$, 87,1 %). Alle untersuchten Stämme waren empfindlich auf Vancomycin und Rifampicin. Es zeigten sich jedoch Resistenzen gegen Co-trimoxazol (6,4 %), Clindamycin (9,7 %), Gentamicin (9,7 %), Fusidinsäure (12,9 %), Levofloxacin (12,9 %) und Erythromycin (61,3 %). Die meisten *lukS-lukF*-positiven MRSA in unserer Studie zeigten ST8, t008 und waren positiv auf *arcA*.

Schlussfolgerungen Der Hauptteil der in unserer Untersuchung identifizierten *lukS-lukF*-positiven MRSA-Stämme zeigte ST8, t008 und war *arcA*-positiv. Ein Phänotyp, welcher hauptsächlich aus den USA beschrieben ist. ST80-Stämme wurden in unserer Untersuchung nicht so häufig wie in anderen europäischen Ländern identifiziert.

Schlüsselwörter: PVL-positive MRSA, Auftreten, Charakterisierung, Tirol, Westösterreich

Summary

Background Community-acquired methicillin-resistant *Staphylococcus aureus* (caMRSA) is an emerging pathogen which causes potentially severe infections in young and healthy individuals due to the ability of most strains to produce Panton–Valentine leukocidin (PVL).

The aim of this study was to evaluate the prevalence of PVL-positive (PVL$^+$)-MRSA strains in Western Austria in the period from December 2005 to May 2010 and to characterize the identified PVL$^+$-MRSA strains.

Methods Six hundred and fifty MRSA strains from Innsbruck Medical University hospital, district hospitals, and general practitioners were investigated for the presence of *lukS-lukF* gene (encoding for PVL). Antimicrobial resistance testing, SCC*mec*-, *agr*-, MLST- and *spa*-typing, as well as *arcA* determination were performed on PVL$^+$-MRSA.

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Results Among 650 MRSA strains collected from various body sites from hospitalized patients and outpatients, 31 strains (4.8 %) were positive for *lukS-lukF* and thus identified as PVL⁺-MRSA.

Agr-1 was the most common *agr*-type ($n=18$, 58.1 %) and SCC*mec*-IV or variants IVa and IVc were the most common SCCmec types ($n=27$, 87.1 %). All tested strains showed in-vitro susceptibility to vancomycin and rifampicin, but resistance against cotrimoxazol (6.4 %), clindamycin (9.7 %), gentamicin (9.7 %), fusidic acid (12.9 %), levofloxacin (12.9 %), and erythromycin (61.3 %) was found.

Most *lukS-lukF*-positive MRSA detected in our survey shared ST8 and t008 and were positive for *arcA*.

Conclusions The major *lukS-lukF*-positive MRSA lineage found in our population was ST8, t008 and positive for *arcA* which is mainly found in the USA. In contrast, ST80 strains were not found as frequently in our region as in many other European countries.

Keywords: PVL-positive MRSA, Emergence, Characterization, Tyrol, Western Austria

Introduction

Formerly methicillin-resistant *Staphylococcus aureus* (MRSA) infections were mostly acquired in health care facilities by hospitalized patients [1] and therefore these strains were called hospital-acquired MRSA.

However, in the last years, MRSA also have become an important pathogen in the out-clinic environment [2]. These strains, called community-acquired MRSA (caMRSA), tend to cause infections of the skin and soft tissue and may also be responsible for a severe necrotizing pneumonia [3, 4]. This may be due to the expression of Panton–Valentine leukocidin (PVL, coded by the *lukS-lukF*-gene), which is able to cause severe necrotic skin lesions in rabbits and lung necrosis in mice [5–8]. PVL-production has also been observed in MRSA associated with hospital infection and in some cases caMRSA without *lukS-lukF*-gene have been reported [9–11]. Thus, production of PVL is a common feature of caMRSA, but not a defining criterion. However, the Center of Disease Control and Prevention (CDC) describes caMRSA only by anamnestic characteristics (http://www.cdc.gov/mrsa/diagnosis). It has been shown that caMRSA usually harbor SCC*mec* element type IV and sometimes type V, both smaller SCC*mec* variants, which carry less resistance information, making caMRSA more susceptible against a number of antimicrobial drugs [6, 12–15]. It has also been shown by several authors that caMRSA strains reported in Europe carry *agr*-3 in many cases [5, 16, 17], whereas strains presenting *agr*-1 are found predominantly in the USA [5, 18]. As PVL-producing (PVL⁺) MRSA are able to cause such severe conditions, it is important to know the epidemiology and prevalence of these bacteria as well as their genetic features. Hence, the aim of the present study was to investigate the prevalence of PVL⁺-MRSA strains in Western Austria and to characterize them.

Materials and methods

Isolates

All *Staphylococcus aureus* isolates diagnosed in routine laboratory at the Division of Hygiene and Medical Microbiology, Innsbruck Medical University between December 2005 and May 2010 were included in the study. Those, which showed resistance to cefoxitin in agar-diffusion test according to CLSI 2009 guidelines, underwent confirmation using cefoxitin screen-test by the VITEK2 system (bioMérieux, Lyon, France). If positive, these strains were characterized as MRSA. Isolates were stored until further investigation at −20 °C. A total of 650 MRSA isolates was identified, originating from Innsbruck Medical University hospital (51 %, $n=332$), from district hospitals (38.2 %, $n=248$), and from general practitioners (10.8 %, $n=70$) in Western Austria. The MRSA strains were gained from various swabs (skin, nose, throat, ear, and wound), aspirates (joint, ascites), and from blood, sputum, tracheal fluid, and urine. Only one isolate per patient was included in the study.

Presence of the mecA-gene and the lukS-lukF-gene

Presence of the *mecA*-gene was determined in all investigated strains to verify their classification as MRSA and *lukS-lukF* was determined to evaluate PVL expression. Both the genes were determined by polymerase chain reaction (PCR) using the GenoType Staphylococcus VER 1.0 kit from Hain Lifesciences (Hain Lifesciences, Nehren, Germany) according to the manufacturer's instructions [19].

Classification of the SCCmec-element and the agr-gene

SCC*mec* elements of types I–V were identified by using a combination of different PCRs according to www.staphylococcus.net. The *agr*-gene of all the *lukS-lukF*-positive strains was determined using multiplex PCR as described elsewhere [20].

Antimicrobial susceptibility testing

Resistance pattern (clindamycin, cotrimoxazol, erythromycin, fusidic acid, gentamicin, levofloxacin, rifampicin) was evaluated by agar-diffusion testing performed on Müller-Hinton-Agar (BBL; Becton Dickinson, Cockeysville, MD, USA) and using epsilometertest (E-Test®;

AB Biodisk, Solna, Sweden) for vancomycin. Plates were incubated for 24 hours at 37 °C. Resistance was interpreted according to the guidelines of the Clinical and Laboratory Standards Institute (Performance Standards for Antimicrobial Susceptibility Testing; Nineteenth Informational Supplement; CLSI, 2009). Concerning fusidic acid, breakpoints published by Olsson-Liljequist et al. [21] were used.

Spa-typing, multilocus-sequence typing (MLST), and arcA-gene

Spa typing was performed according to the Ridom Staph type standard protocol (www.ridom.com) and by the use of the Ridom Staph type software package for assigning *spa*-types [22].

By the application of the BURP algorithm, *spa*-types were clustered into different groups, as described by Strommenger et al. [23].

Selected isolates, which were grouped ambiguously by *spa*-typing, were investigated via multilocus sequence typing (MLST) according to Enright et al. [24] with the exception of an alternative forward primer for the *tpi* amplimer [25].

All *lukS-lukF*-positive isolates exhibiting *spa*-type t008 and sequence type (ST)8 or clonal complex (CC)8 were subjected to PCR for *arcA*, according to Strommenger et al. [23]. *ArcA* is indicative for the ACME-element, which has been shown to be present in caMRSA "USA300".

Results and discussion

Six hundred and fifty MRSA strains (332 from Innsbruck Medical University hospital, 248 from district hospitals, and 70 from general practitioners) were characterized. Presently, this represents the largest PVL$^+$-MRSA survey for Western Austria.

Presence of the mecA-gene and prevalence of the lukS-lukF-gene

All investigated strains carried the *mecA*-gene (data not shown), confirming their identities as MRSA. Thirty-one of the 650 strains (4.8 %) also harbored the *lukS-lukF*-gene.

The annual prevalence varied from 0 % in 2005 (only 1 month included in this year) to 8.3 % in 2007. During the study period no marked trend of the prevalence of PVL$^+$-MRSA among MRSA was detectable.

In Innsbruck University hospital, 5.4 % out of 332 isolates ($n=18$) were *lukS-lukF* positive. In district hospitals, 3.2 % out of 248 isolates ($n=8$) harbored the *lukS-lukF* gene. Twenty-three of the 26 (88.4 %) hospitalized patients were admitted due to (mainly skin and soft tissue) infection with PVL$^+$-MRSA and only one patient acquired the MRSA strain during hospitalization. In the remaining two cases, information about the mode of acquisition of PVL$^+$-MRSA was not available. Among the isolates from general practitioners, 7.1 % out of 70 isolates ($n=5$) carried the *lukS-lukF* gene (Table 1). The overall prevalence of PVL$^+$-MRSA infections in community-acquired skin and soft-tissue infections with *Staphylococcus aureus* was 0.3 % in samples admitted to our department during the study period.

Prevalence of the *lukS-lukF*-positive strains in Europe varies. A Belgian study has investigated 41 MRSA isolates between 2002 and 2004 and has found a *lukS-lukF* prevalence of 40 % among their isolates. At the first glance, this seems to be extremely high, however, Denis et al. [16] have investigated only MRSA strains originating from community-associated infection. Witte's research group has found a prevalence of only 0.4 % in Germany in 2003. The reason for this rather low *lukS-lukF* prevalence may be that the investigated strain collection also contained strains from 221 hospitals [26].

Compared with other data from Austria, we found a rather low prevalence of PVL$^+$-MRSA among our investigated MRSA.

In a survey performed at Vienna General Hospital in 2009, Bauer et al. [27] have found 16 (39 %) out of 41 tested *mecA*-positive MRSA also harboring *lukS-lukF*. This rather high prevalence, however, can in part be attributed to a preselection by phenotypic criteria (resistance pattern). Concerning the southeast region of Austria, Grisold et al. [28] have identified 70 caMRSA (13.1 %) among 534 MRSA strains between 2002 and 2007, however, in their survey Grisold et al. observed a tremendous increase of *lukS-lukF*-positive MRSA from 3.0 % (2002) to 24.3 % (2007) during their study period.

Krziwanek et al. [29] have performed their evaluation in five federal provinces of Austria (Upper Austria, Lower Austria, Salzburg, Carinthia, and Vienna). Among 1,150 investigated MRSA strains, they have found 8.2 % ($n=94$) positive for *lukS-lukF*. Also, Krziwanek et al. experienced an increase in their PVL$^+$-MRSA prevalence from 3.7 % in 2005 to 7.7 % in 2006.

Sex distribution in our survey was nearly 50:50: Sixteen (51.6 %) of the *lukS-lukF*-positive isolates originated from male patients (Table 1).

The average age of patients suffering from PVL$^+$-MRSA was 42.2 years, ranging from 7 years to 93 years. This average age fits well to the data published by others. Jung et al. [30] have reported 42.8 years in their investigation. Estivariz et al. [31] have found a lower median age with only 32 years.

The average age of patients suffering from PVL-negative MRSA in our survey was much higher (62.7 years, ranging from 1 month to 101 years) than in our and the published PVL-positive cohorts [30, 31].

The majority of the *lukS-lukF*-positive MRSA strains isolated in our laboratory were found in infections of the skin and soft tissue (80.6 %, $n=25$). This finding confirms that MRSA strains, which carry the PVL, are likely to cause infections of the skin and soft tissue like already postulated before [32]. Also, the dermatonecrotic poten-

Table 1. Characteristics of *lukS-lukF*-positive MRSA strains

Isolate	Sex	Year[a]	Age (years)	Outpatient	Origin of sample	ST, CC	Resistance phenotype	*Agr*	SCC*mec*	*Spa*	*ArcA*
1	Female	2006	22	No	*n.d.*	ST8		1	IVc	t008	Negative
2	Female	2006	24	No	Wound	ST8	ERY	1	IVa	t008	Positive
3	Female	2006	67	No	Wound	CC22	GEN	1	IVa	t2816	*n.d.*
4	Male	2006	48	No	Wound	ST8	CLI, ERY	4	IV	t024	*n.d.*
5	Male	2006	78	Yes	Tracheal fluid	CC8	CLI, GEN	2	I	t024	*n.d.*
6	Female	2007	17	No	Wound	ST80	FUS	3	IV	t1201	*n.d.*
7	Male	2007	53	No	Wound	ST8	ERY	1	IVa	t008	Positive
8	Male	2007	27	No	*n.d.*	CC5	CLI, ERY	1	*n.d.*	t437	*n.d.*
9	Female	2007	64	No	Wound	ST80		3	IV	t455	*n.d.*
10	Male	2007	45	Yes	Nose	CC8		1	IV	t121	*n.d.*
11	Female	2007	35	No	Blood	ST8	ERY	1	IVa	t008	Positive
12	Female	2007	93	No	Wound	ST8	ERY	1	IVa	t008	Positive
13	Male	2007	32	No	Wound	ST8	LEV, ERY	1	IVa	t008	Positive
14	Female	2007	82	No	Wound	ST8	LEV, ERY	1	IVa	t008	Positive
15	Male	2007	24	No	Wound	ST8	ERY	*n.d.*	IVa	t008	Positive
16	Female	2007	40	No	Wound	ST8		1	IVa	t008	Positive
17	Male	2007	26	Yes	Wound	ST8		1	IVa	t008	Positive
18	Female	2008	21	No	Abscess	ST80	FUS, ERY	3	IV	t044	*n.d.*
19	Male	2008	69	No	Wound	CC30		3	IV	t019	*n.d.*
20	Male	2008	31	No	Wound	ST8	ERY	1	IVa	t008	Positive
21	Female	2008	78	No	Wound	ST8	LEV, ERY	1	IVa	t008	Positive
22	Male	2008	22	No	Wound	CC30		3	IV	t019	*n.d.*
23	Male	2008	27	No	Wound	ST8	ERY	1	IVc	t008	Negative
24	Male	2008	12	Yes	Wound	ST8	FUS	3	IV	t044	*n.d.*
25	Female	2008	87	Yes	Ear	CC8	COT, FUS	*n.d.*	*n.d.*	t008	Negative
26	Male	2009	67	No	Furuncle	ST8	ERY	1	IVa	t008	Positive
27	Male	2009	42	No	Abscess	ST8	LEV, ERY	1	IVa	t008	Positive
28	Female	2009	7	No	Nail	ST88	ERY	3	IV	t692	*n.d.*
29	Female	2010	17	No	Breast	ST88	ERY	3	IV	t692	*n.d.*
30	Male	2010	24	No	Furuncle	CC8	ERY	1	*n.d.*	t008	Negative
31	Female	2010	29	No	Abscess	CC1	COT, GEN, ERY	2	IV	t657	*n.d.*

ST sequence type, *CC* clonal complex, *CLI* clindamycin, *COT* cotrimoxazol, *GEN* gentamicin, *FUS* fusidic acid, *ERY* erythromycin, *LEV* levofloxacin, *n.d.* not determined

[a]Year of strain isolation

tial of PVL is known since a long time [33]. The remaining *lukS-lukF*-positive isolates originated from blood culture and ear swab (both from patients with infection) and from tracheal fluid and nose swab (both from colonized patients), two samples could not be determined (Table 1).

Classification of the SCCmec-element and the agr-gene

The SCC*mec* type of all the *lukS-lukF*-positive isolates was determined. The vast majority (87.1 %) of our tested isolates carried SCC*mec* IV ($n=11$) or variants IVa ($n=14$) or IVc ($n=2$), one isolate was typed SCC*mec* I and the SCC*mec* types of three isolates were not determinable (Table 1). The dominance of SCC*mec* type IV among our strains fits very well to the data reported by other groups. Denis et al. [16] have investigated the SCC*mec* of 16 *lukS-lukF*-positive MRSA strains. Among them they have found all strains carrying SCC*mec* type IV. Another group has found 16 (72.7 %) out of 22 *lukS-lukF*-positive MRSA isolates carrying SCC*mec* IV [17]. Baggett et al. [34] have tested five *lukS-lukF*-positive MRSA strains and reported that all of them carried SCC*mec* type IV. These data show that *lukS-lukF*-positive MRSA strains carry the SCC*mec* element type IV in most cases, which is also confirmed by our findings.

Investigation of the *agr*-gene subtype revealed a majority of *agr*-1 (58.1 %, $n=18$). *Agr* type 3 was found in eight strains (25.8 %). Two strains (6.5 %) were classified *agr*-2 and one (3.2 %) showed *agr*-4. In two strains, it was not possible to identify a certain *agr*-subtype (Table 1).

In contrast to other surveillances in Europe, we have found a very high rate of *agr* type 1 and only a rather low rate of *agr* type 3. In Belgium, 13 (81.3 %) of 16 tested *lukS-lukF*-positive strains have presented *agr* type 3, only 3 strains (18.7 %) have harbored *agr*-1 [16]. Naas et al. [17] in France also have found the majority of their tested strains presenting *agr*-3 (72.7 %, $n=16$). Vandenesch et al. [5], who have investigated MRSA strains from three continents, found all *lukS-lukF*-positive MRSA strains which originated from Europe (France and Switzerland) carrying *agr*-3, this also holds true for the strains they have received from Australia and Oceania. However, all the *lukS-lukF*-positive strains in Vandenesch's study, which have shown *agr*-1 ($n=4$) have originated from the USA, nevertheless, also among the US-derived strains the majority carried *agr*-3 (87.8 %, $n=29$). Tsuji et al. [18] have reported a dominance of *agr* type 1 among their tested caMRSA strains in the USA.

These differences may be due to the fact that our strains might be a mixture of different genetic backgrounds, likely due to the role of the Tyrol as a touristic hotspot in Western Austria.

Antimicrobial susceptibility

Resistance patterns of all the *lukS-lukF*-positive strains revealed that none of the tested strains was resistant against vancomycin and rifampicin. Two strains (6.4 %) were resistant against cotrimoxazol (Table 1).

Our data are similar to the data from other research groups who investigated the resistance pattern of the *lukS-lukF*-positive strains. Also, in Germany [26], in France [17], and in the USA [35], they found all their strains susceptible against vancomycin and rifampicin. However, they reported their strains also susceptible to cotrimoxazol. Concerning gentamicin and clindamycin, we found three (9.7 %) of our strains resistant, which is higher compared with other data from Europe and the USA. Witte et al. [26] in Germany and Naas et al. [17] in France have reported that all their tested strains were susceptible to gentamicin and clindamycin. Fey et al. [35] in the USA have also found none of their tested strains resistant against gentamicin and a moderate resistance rate (6.3 %) for clindamycin. In our surveillance, four strains (12.9 %) were resistant against levofloxacin, seven (22.6 %) showed intermediate resistance. In another survey, Tsuji et al. [18] found 12 % of their tested caMRSA to be resistant and 88 % sensitive to levofloxacin. Concerning fusidic acid we found 27 strains susceptible and four strains (12.9 %) resistant. This is a sharp contrast to other data from Europe. Witte et al. [26] in Germany and Naas et al. [17] in France have described all their investigated isolates as resistant against fusidic acid, however, Witte investigated only ST80 strains. Denis in Belgium has identified 56 % of his isolates as resistant against fusidic acid [16]. In our surveillance, we found 19 strains (61.3 %) resistant against erythromycin and two strains (6.5 %) were detected as intermediate susceptible. In literature, the data dealing with the resistance against erythromycin are quite inconsistent: In the USA, Tsuji et al. [18] have reported 92 % of their investigated caMRSA resistant against erythromycin. Naas in France and Denis in Belgium have found erythromycin resistance rates lower than 20 % among their PVL-positive MRSA: Naas et al. [17]: 19 % , Denis et al. [16]: 6.25 % . Interestingly, Witte et al. [26] found none of their investigated strains resistant against erythromycin. The comparison of our data with the data from other groups shows that there are rather big differences in the resistance rate concerning fusidic acid and erythromycin which may occur because in different nations other antimicrobial drugs might be used and also strains with other genetic background may be predominant.

Spa-typing, multilocus sequence typing (MLST), and arcA-gene

Evaluation of the *spa*-type and sequence type by MLST method revealed 11 different *spa*-types (t008, t019, t024, t044, t121, t437, t455, t657, t692, t1201, and t2816) and eight different sequence types or clonal complexes (CC1,

CC5, CC8, ST8, CC22, CC30, ST80, and ST88) with ST8 or CC8 (67.7 %, $n=21$) and t008 (54.8 %, $n=17$) being the most prevalent (Table 1). Thus, there seems to be one large clonal group with 17 isolates containing ten isolates from Innsbruck University Hospital, five isolates from district hospitals, and two isolates from general practitioners. All these strains share the following genetic features: ST8 or CC8, t008, *agr*-1 (except for two cases where *agr* could not be determined), and SCC*mec* IVa ($n=13$) or IVc ($n=2$; except for two cases, where SCC*mec* could not be determined). Determination of *arcA* among t008 strains revealed 13 of 17 (76.5 %) tested strains *arcA* positive (Table 1). These features give hint to a "USA300" genetic background. USA300 is the most frequent caMRSA strain in the USA but has also spread to Europe [25] and to Asia [36], recently. Furthermore, our population showed two smaller clonal groups consisting of two isolates each: One group shared ST88, t692, *agr*-3, and SCC*mec* IV, both isolates originated from Innsbruck University Hospital. The second complex had the following similarities in common: CC30, t019, *agr*-3, and SCC*mec* IV. One of these two isolates was found in Innsbruck University Hospital, the other in a district hospital. The remaining ten strains showed no common genetic background and seem to be diverse.

Krziwanek et al. [29] have investigated 94 *lukS-lukF*-positive MRSA isolates in Austria and have found a majority of them belonging to sequence type ST8 (31.9 %, $n=30$). However, they have found 16 strains belonging to sequence type ST152. We found no ST152-strains in our population. On the other hand, Krziwanek et al. [29] have reported 14 strains in their collection belonging to ST80. ST80 also is the second most frequent sequence type in our survey, however with only three isolates. Other data from Belgium showed only three out of 16 strains representing sequence type ST8 and *spa*-type t008, but eight strains belonging to ST80 and t044 [16]. This shows that also in Europe there is a big variability in the genetic background of the occurring *lukS-lukF*-positive MRSA strains.

In conclusion, the prevalence of PVL^+-strains among MRSA isolates collected in hospitals and general practitioners in Western Austria is 4.8 %. Comparison with other data from Europe is difficult due to unequal criteria defining the study population. Resistance pattern and SCC*mec* type (mainly IV) was similar to other *lukS-lukF*-positive MRSA reported from Europe. However, the predominance of *agr*-1 is special for our population.

The major PVL-positive MRSA lineage found in our population was ST8, t008 which is mainly found in the USA. In contrast, ST80 strains are not found as frequently in Western Austria as in many other European countries.

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Conflict of interest
The authors declare that there is no conflict of interest to disclose.

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