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Evolution and Convergence of CA-MRSA and HA-MRSA: Is PVL Still a Discriminating Factor?

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ABSTRACT: Methicillin-resistant *Staphylococcus aureus* (MRSA) remains a significant global pathogen responsible for both healthcare-associated and community-associated infections. Historically, the presence of Panton–Valentine leukocidin (PVL) served as a key molecular marker distinguishing community-associated MRSA (CA-MRSA) from hospital-associated MRSA (HA-MRSA). However, increasing evidence of genetic convergence between these lineages challenges the reliability of PVL as a discriminating factor. Over time, the distinctions between community-associated (CA-MRSA) and hospital-associated (HA-MRSA) strains have become increasingly blurred. This is largely due to the ongoing exchange of genetic material, clonal expansion, and the dynamic nature of antibiotic resistance. In this review, we take a closer look at the molecular epidemiology of both CA-MRSA and HA-MRSA, with particular attention to the structure and pathogenic role of Panton–Valentine leukocidin (PVL). We also examine how the presence of PVL varies among different MRSA strains and discuss whether it still holds value as a marker in surveillance efforts. Given the rapid evolution of bacterial genomes, we reflect on the clinical implications of relying on PVL for classification. The review concludes by outlining future research priorities, especially the need for integrated genomic monitoring and more comprehensive typing strategies to enhance MRSA management and improve patient care.

KEYWORDS: *Staphylococcus aureus*; PVL; CA-MRSA; HA-MRSA

1. Introduction

Staphylococcus aureus is an exceptionally versatile and resilient pathogen, capable of causing a wide spectrum of diseases. Its clinical impact ranges from mild skin and soft tissue infections (SSTIs) to life-threatening conditions like bacteremia, pneumonia, and endocarditis (Silva-Santana, 2025). The appearance of methicillin-resistant *S. aureus* (MRSA) in the early 1960s, remarkably soon after methicillin was first introduced,signalled the start of a global struggle against antibiotic resistance (Larsen *et al*., 2022). This resistance is primarily driven by the *mecA* gene, located on a mobile genetic element known as the staphylococcal cassette chromosome *mec* (SCCmec). The *mecA* gene encodes an alternative penicillin-binding protein (PBP2a), which renders β-lactam antibiotics ineffective (Mohammed *et al*., 2025).

Historically, MRSA strains were divided into two main categories based on their epidemiology: hospital-associated (HA-MRSA) and community-associated (CA-MRSA). HA-MRSA was largely restricted to hospital environments, where it affected immunocompromised patients and typically carried large SCCmec elements (types I–III), conferring resistance to multiple drug classes. CA-MRSA, on the other hand, was identified in otherwise healthy individuals with no prior contact with healthcare facilities. These strains often carried smaller SCCmec types (IV or V), displayed enhanced virulence, and tended to remain more susceptible to non–β-lactam antibiotics (Yamaguchi *et al*., 2020). By the 1990s, HA-MRSA had become endemic in hospitals around the world, prompting infection control strategies to focus primarily on this phenotype (Uhlemann *et al*., 2014).

A distinguishing molecular feature of CA-MRSA has traditionally been the presence of Panton–Valentine leukocidin (PVL), a cytotoxin linked to the development of SSTIs (Al-Halaq and Utba, 2023) and, in some cases, necrotising pneumonia—especially in young and otherwise healthy individuals. For a time, detecting PVL was considered a strong marker of community-associated MRSA origin (Mediavilla *et al*., 2012). Outbreaks were reported in schools, prisons, military barracks, and sports teams, highlighting the pathogen's ability to thrive outside hospital settings. Several prominent CA-MRSA clones—most notably those belonging to lineages such as ST8 (USA300), ST30, and ST80—have been found to carry genes encoding Panton–Valentine leukocidin (PVL), a two-component toxin associated with the destruction of neutrophils and severe tissue damage. (Coombs *et al*., 2020).

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However, over the past two decades, the boundaries between CA-MRSA and HA-MRSA have become increasingly porous. Clonal expansion, horizontal gene transfer (HGT), and global dissemination of hybrid strains have contributed to the convergence of genetic, phenotypic, and ecological features across MRSA lineages. Notably, PVL-positive MRSA strains are now frequently identified in hospital environments, while CA-MRSA clones have acquired multidrug resistance and established a foothold in nosocomial settings (Otto, 2013).

This review aims to critically examine the current relevance of PVL as a molecular marker in the classification and surveillance of MRSA. We explore the molecular epidemiology of CA- and HA-MRSA, the structure and pathogenic role of PVL, and the genetic mechanisms driving convergence. In light of increasing genomic plasticity, we assess whether PVL remains a meaningful discriminator in the modern clinical and epidemiological landscape.

2. Molecular Epidemiology of CA-MRSA vs. HA-MRSA

The distinction between CA-MRSA and HA-MRSA has traditionally gone beyond clinical settings, resting heavily on unique molecular signatures. These genetic markers have long informed diagnostic approaches and epidemiological tracking. However, advances in genomic sequencing and surveillance have revealed a growing overlap between these strains, complicating their classical classification (Deurenberg and Stobberingh, 2008).

2.1 SCCmec Typing and Genetic Background

Historically, HA-MRSA strains predominantly carry larger SCCmec types, namely I, II, or III, which frequently harbour additional genes conferring resistance to multiple antibiotic classes, resulting in multidrug-resistant (MDR) phenotypes. These strains are linked to well-known hospital-associated clones, including ST239, ST22, and ST5 (Yamaguchi *et al*., 2020).

In contrast, CA-MRSA strains typically harbour smaller SCCmec elements—types IV or V—which generally do not carry the extensive array of resistance genes found in HA-MRSA. As a result, these strains tend to remain more responsive to non–β-lactam antibiotics. Notable and well-documented CA-MRSA lineages include ST8 (USA300) in the United States, ST80 across Europe and the Middle East, and ST30, which is prevalent throughout the Asia-Pacific region (Otto, 2013).

2.2 Clonal Complexes and Geographic Variability

The global distribution of MRSA clones shows significant regional variation, shaped by local evolutionary pressures and patterns of transmission. In the Middle East, the ST80-IV clone is the predominant CA-MRSA strain, commonly linked to skin and soft tissue infections and frequently harboring the PVL genes (Albrecht, 2013). Meanwhile, in North America, the USA300 clone (ST8-IV) has emerged as the dominant CA-MRSA lineage, gradually replacing other strains. Notably, it has also made inroads into hospital settings, highlighting its remarkable virulence and adaptability (Planet *et al*., 2015).

Interestingly, hospital-associated clones such as ST239-III and ST22-IV (EMRSA-15), which were once largely restricted to healthcare environments, are now increasingly detected in community-acquired infections. This shift points to a changing ecological landscape and further complicates the distinction between HA- and CA-MRSA strains (Otter and French, 2010).

2.3 Mobile Genetic Elements and Horizontal Gene Transfer

Recent advances in genomic analysis have highlighted the central role of HGT in shaping the evolution of MRSA. Mobile genetic elements, such as plasmids, pathogenicity islands, transposons, and bacteriophages, act as key vehicles for the dissemination of both resistance and virulence genes (Emamalipour *et al*., 2020). These elements often carry important determinants, including those for PVL, enterotoxins, immune evasion mechanisms, and antibiotic resistance, allowing *S. aureus* to adapt rapidly to changing environments (Malachowa and DeLeo, 2010).

This high degree of genetic mobility has led to increasing overlap between the genotypic and phenotypic profiles of CA-MRSA and HA-MRSA. For instance, some CA-MRSA strains have acquired multidrug resistance plasmids, enabling them to survive in hospital environments. At the same time, certain HA-MRSA strains have incorporated virulence factors like PVL, enhancing their ability to cause infections in community settings (Otto, 2013).

2.4 Evidence of Convergence

Increasingly, reports worldwide—and particularly in the Middle East—describe the presence of PVL-positive HA-MRSA and multidrug-resistant CA-MRSA in both hospital and outpatient environments. For instance, PVL-positive USA300 strains have been isolated from intensive care units and surgical wards, areas traditionally dominated by HA-MRSA (Planet *et al*., 2015). HA

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clones such as ST239 have occasionally been found to carry PVL genes, especially in Asia and the Middle East, regions with high MRSA diversity (Mohamad Farook *et al*., 2022). In Iraq, Ibrahim and Al-Mathkhury (Ibrahim and Al-Mathkhury, 2018) study identified PVL-positive MRSA strains isolated from hospitalised patients, challenging the long-held notion that PVL is exclusive to CA-MRSA.

These observations highlight an ongoing convergence in both genetic makeup and ecological distribution between CA- and HA-MRSA. Consequently, classical classification systems based solely on PVL status or SCCmec type are becoming less reliable, potentially leading to misclassification, obscured epidemiological trends, and challenges in infection prevention and control.

3. Panton–Valentine Leukocidin

3.1 Structure and Mechanism of Action

PVL is a bicompartmental pore-forming exotoxin produced by some strains of *Staphylococcus aureus*. It is composed of two distinct protein subunits, LukS-PV and LukF-PV, which are encoded by the *lukS-PV* and *lukF-PV* genes, respectively. These genes are carried on a temperate bacteriophage (ΦSa2) that integrates into the *S. aureus* genome, allowing horizontal transmission among strains (Boakes *et al*., 2011).

PVL targets human polymorphonuclear leukocytes, particularly neutrophils, which are key effectors in the innate immune response. The LukS-PV subunit initially binds to the C5a receptor on the surface of polymorphonuclear neutrophils (PMNs), where it becomes phosphorylated by host protein kinases A or C. This phosphorylation is followed by the induction of calcium ion channels, suggesting activation of intracellular signal transduction pathways that may trigger the production of interleukins and other inflammatory mediators. Subsequently, LukS-PV recruits and oligomerizes with LukF-PV to form a transmembrane heptameric pore (Hussain *et al*., 2022). This pore formation leads to osmotic lysis of neutrophils, the release of cytotoxic granules, reactive oxygen species, and inflammatory mediators, and ultimately tissue necrosis and pus formation (Boyle-Vavra and Daum, 2007, Hussain *et al*., 2022) (Figure 1). This mechanism is believed to enhance *S. aureus* survival by evading phagocytic clearance and promoting inflammation (McGuinness *et al*., 2016).

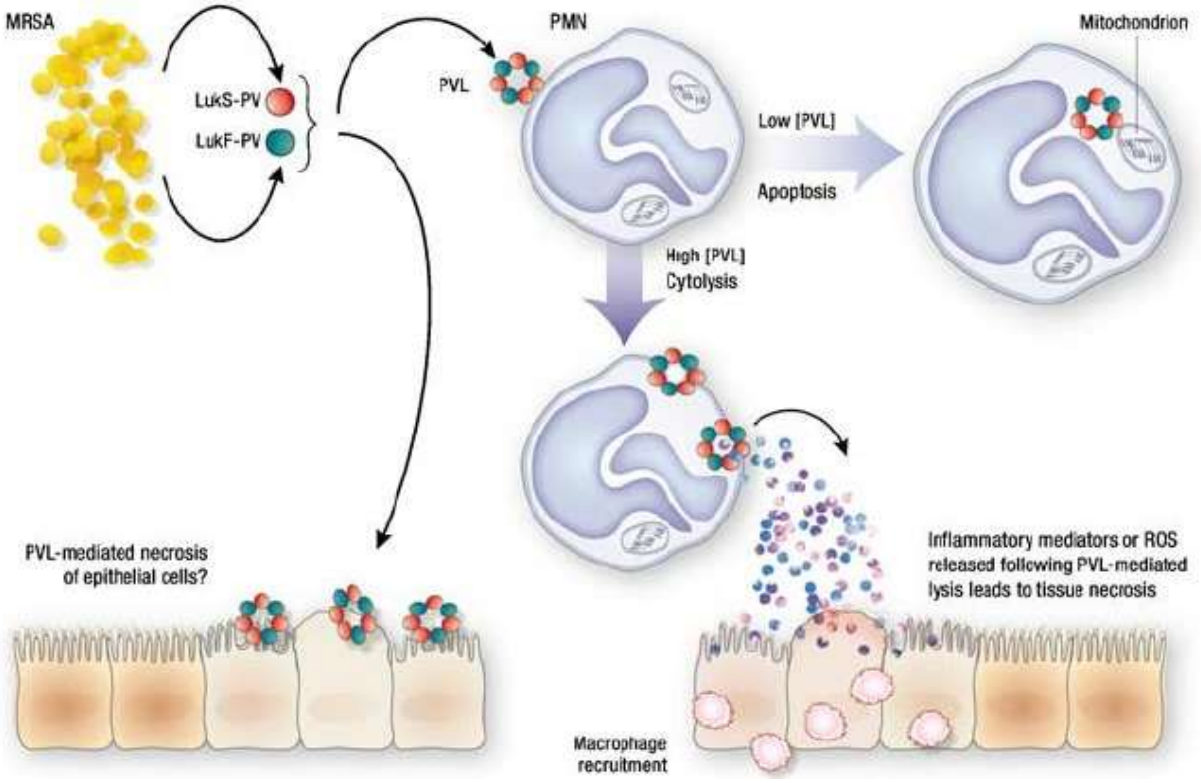

Figure 1: Molecular pathogenesis of PVL-SA infection, PVL pore formation on cell membrane, leukocytes and epithelial cells adopted from Leistner *et al*. (2022)

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Although PVL is not essential for all *S. aureus* strains, its presence has been repeatedly associated with enhanced virulence, particularly in CA-MRSA strains that cause aggressive, purulent skin infections and necrotising pneumonia (Voyich *et al*., 2006).

3.2 Historical Association with Skin and Soft Tissue Infections

PVL first gained attention in the early 20th century when it was linked to furunculosis and severe cutaneous infections. However, it was not until the rise of CA-MRSA in the 1990s and early 2000s that its clinical importance was firmly established. Studies from that era reported that the majority of CA-MRSA isolates involved in SSTIs were PVL-positive, and PVL was proposed as a major driver of the aggressive and necrotic nature of these infections (Mediavilla *et al*., 2012).

The link between PVL and necrotising pneumonia was also highlighted in several outbreaks and case series, where young, previously healthy individuals developed rapidly progressive and often fatal pneumonia caused by PVL-positive *S. aureus* strains (Costa *et al*., 2025). These findings elevated PVL to the status of a virulence hallmark of epidemic CA-MRSA clones such as USA300 (ST8-IV) and ST80-IV.

Nevertheless, the precise role of PVL in virulence has since become a topic of debate. Animal models and human studies have produced inconsistent results, some confirming its cytotoxic effects, while others suggest PVL is neither necessary nor sufficient for severe infection in all contexts (Voyich *et al*., 2006). As a result, PVL is now considered a contributor to virulence, particularly in skin infections, but not the sole determinant.

3.3 Global Prevalence and Distribution Among MRSA Strains

The prevalence of PVL varies widely across geographic regions and clonal types. In North America, the PVL-positive USA300 clone is the predominant cause of CA-MRSA infections, especially SSTIs. Similarly, in Europe and the Middle East, ST80-IV, another PVL-positive clone, is frequently associated with community outbreaks (Otto, 2013, Mediavilla *et al*., 2012, Mohamad Farook *et al*., 2022). Locally 4% of PVL-positive MRSA has been implicated in SSTIs (Al-Halaq and Utba, 2023).

In Asia, however, PVL-positive strains are less common overall, with CA-MRSA infections often linked to PVL-negative clones such as ST59. That said, PVL genes are increasingly being detected in traditionally hospital-associated clones, such as ST239 and ST22, especially in regions like the Middle East, South America, and parts of Asia (Zhao *et al*., 2023, Goudarzi *et al*., 2020).

In Iraq, PVL-positive MRSA strains isolated from hospitalised patients, providing compelling evidence of PVL's spread into HA-MRSA backgrounds, and further eroding the traditional molecular boundary between CA- and HA-MRSA (Ibrahim and Al-Mathkhury, 2018, Jawad *et al*., 2022, Hussein and Mohammed Saleh, 2024).

These observations suggest that PVL is no longer exclusive to community settings and that mobile genetic elements may be facilitating its horizontal dissemination across *S. aureus* lineages globally. Consequently, the presence of PVL alone can no longer be relied upon to infer strain origin or predict clinical severity.

4. The Clinical and Epidemiological Relevance of PVL Today

Over the past two decades, PVL has transitioned from a niche virulence factor to a prominent feature in discussions of *Staphylococcus aureus* pathogenicity, particularly among CA-MRSA clones. However, the clinical and epidemiological relevance of PVL has become increasingly complex and, at times, controversial.

4.1 Shifting Distribution and the Collapse of the CA/HA Dichotomy

Historically, PVL served as a molecular marker that helped distinguish CA-MRSA from HA-MRSA. Its high prevalence among early CA-MRSA clones like USA300 and ST80, and near absence in hospital-associated strains, supported its use in molecular epidemiology and outbreak investigations (Otter and French, 2012). However, this binary classification has steadily eroded (Otter and French, 2010, Zhao *et al*., 2022).

Recent genomic studies and surveillance reports indicate that PVL genes are increasingly detected in hospital-associated MRSA strains, especially in regions such as the Middle East, Latin America, and parts of Asia (Monecke *et al*., 2012). The horizontal transfer of PVL-carrying phages into hospital clones such as ST239 and ST22 has introduced PVL into settings previously dominated by PVL-negative lineages, thus complicating the use of PVL as a lineage discriminator (Hsu *et al*., 2015).

In parallel, multidrug-resistant CA-MRSA strains, some still PVL-positive, have been isolated from healthcare environments, indicating that CA clones have not only gained resistance traits but have also successfully adapted to nosocomial niches (Mediavilla *et al*., 2012).

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4.2 Correlation with Disease Severity: Conflicting Evidence

While PVL is strongly associated with skin and SSTIs and necrotising pneumonia, its causal role in disease severity remains a subject of debate. Several studies have suggested that PVL-positive strains are more likely to cause abscesses and necrosis in SSTIs, particularly in otherwise healthy individuals (Loffler *et al*., 2010, Gillet *et al*., 2002). In these contexts, PVL-mediated neutrophil lysis may amplify local inflammation and tissue destruction.

However, animal models and clinical cohort studies have presented mixed results. Some murine and rabbit models showed minimal impact of PVL on mortality or systemic infection severity (Voyich *et al*., 2006), while others highlighted PVL's role in lung damage and leukocyte recruitment. Human studies, too, have failed to consistently correlate PVL status with worse outcomes in pneumonia or bacteremia (Shallcross *et al*., 2013). These discrepancies likely reflect strain background, host factors, and co-expressed virulence genes.

Thus, while PVL may contribute to localised immune evasion and tissue pathology, it should not be regarded as the sole determinant of clinical severity.

4.3 Implications for Infection Control and Surveillance

Given its shifting prevalence and ambiguous role in virulence, PVL is increasingly seen as an imperfect epidemiological marker. Relying on PVL presence alone for CA-MRSA identification or outbreak tracing risks misclassification, particularly in regions with high rates of PVL-positive HA clones.

Moreover, molecular diagnostics that target PVL genes without contextual genomic data may fail to capture the dynamic nature of MRSA evolution. Whole-genome sequencing (WGS), which integrates SCCmec typing, core genome phylogeny, virulence factor profiling, and resistance gene detection, is now considered the gold standard for strain classification and epidemiological analysis (Uhlemann *et al*., 2014).

Nevertheless, PVL detection remains clinically relevant, especially in the context of severe SSTIs and necrotising pneumonia, where its presence may prompt clinicians to anticipate aggressive disease progression and adjust management strategies accordingly (Otto, 2013). Furthermore, in resource-limited settings, PVL detection by PCR still serves as a useful, if limited, surrogate marker for highly virulent CA-MRSA strains.

5. Is PVL Still a Discriminating Factor in MRSA Surveillance?

For much of the early 2000s, PVL was regarded as a hallmark of CA-MRSA. Its strong correlation with severe skin infections, distinctive molecular carriage on ΦSa2 phages, and frequent detection in emerging CA-MRSA clones made it an attractive epidemiological marker for tracking strain origin and transmission (Boakes *et al*., 2011, Otto, 2013). However, as the epidemiological and genetic landscapes of MRSA have evolved, the reliability of PVL as a discriminating factor has come into question (Lakhundi and Zhang, 2018).

5.1 Molecular Convergence and Genetic Plasticity

One of the primary challenges undermining PVL's utility as a lineage marker is the increasing convergence between CA-MRSA and HA-MRSA strains. The HGT of PVL-encoding phages into traditionally hospital-associated clones, such as ST239 in Asia and the Middle East, has introduced PVL into healthcare settings (Monecke *et al*., 2012). Simultaneously, PVL-positive CA-MRSA clones like USA300 and ST80 have acquired multidrug resistance and now frequently cause nosocomial infections (Uhlemann *et al*., 2014). These shifts blur the once-clear molecular and clinical boundaries between community and hospital strains.

Furthermore, PVL carriage is not clonal; it is phage-mediated, mobile, and subject to loss or gain depending on selective pressures. Strains may lose PVL phages without fundamentally altering their virulence potential or acquire them without significantly changing pathogenic behaviour. This genomic plasticity means that PVL presence does not equate to functional virulence or predict clinical outcomes with certainty (Otto, 2013, Shallcross *et al*., 2013).

5.2 Diagnostic and Surveillance Limitations

Despite its early promise, the use of PVL as a diagnostic marker has clear limitations. PCR-based assays for *lukF-PV/lukS-PV* genes can identify PVL-positive strains, but without additional genomic context, such as clonal lineage, SCCmec type, and resistance profile, the results are insufficient for epidemiological classification. In high-burden regions where PVL-positive HA-

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MRSA strains are common, such as Iraq and other parts of the Middle East, PVL detection alone may misclassify isolates, undermining infection control efforts (Ibrahim and Al-Mathkhury, 2018, Jawad *et al*., 2022, El Aila *et al*., 2023).

Modern surveillance increasingly relies on WGS, which allows for high-resolution typing, outbreak tracking, and comprehensive virulence/resistance profiling (Uhlemann *et al*., 2014). WGS can accurately identify hybrid strains, trace transmission events, and clarify the role of PVL within a broader genomic and epidemiological context.

5.3 Clinical Relevance and Contextual Value

Although PVL's utility as a molecular discriminator is diminishing, it remains clinically relevant, particularly in severe SSTIs and necrotising pneumonia. The detection of PVL may alert clinicians to increased risk of abscess formation or rapid tissue necrosis, prompting more aggressive management strategies (Otto, 2013). In resource-limited settings, PVL testing via PCR remains a practical tool, not for definitive classification, but as part of a composite assessment that includes clinical, demographic, and phenotypic data.

Ultimately, PVL should be regarded as a context-dependent virulence factor, not a categorical marker. Its presence may support certain clinical decisions or reinforce epidemiological trends, but it cannot stand alone as a reliable discriminator between CA- and HA-MRSA in today's interconnected genomic landscape.

6. Conclusion and Future Directions

The historical division between CA-MRSA and HA-MRSA once offered a practical framework for clinicians and microbiologists alike. This dichotomy, long supported by differences in genetic makeup, antimicrobial susceptibility, and carriage of virulence factors such as PVL, has grown increasingly porous. Mounting evidence of genomic convergence between CA- and HA-MRSA lineages, through HGT, clonal expansion, and ecological adaptation, has challenged the utility of PVL as a reliable molecular marker.

While PVL remains epidemiologically associated with severe skin and soft tissue infections, and its detection may have clinical implications in specific settings, its role as a standalone discriminator between CA and HA origins is no longer tenable. Strains previously considered "community-associated" now frequently exhibit hospital-adapted resistance profiles, and vice versa. These changes reflect the fluidity of MRSA evolution, shaped by selective pressures within both healthcare and community environments.

Future efforts should prioritize routine use of WGS in surveillance to track MRSA evolution, transmission, and mobile genetic elements (Larsen *et al*., 2022). Particular attention should be paid to emerging hybrid strains and clonal shifts that challenge traditional classification.

Beyond SCCmec and PVL status, improved typing systems incorporating both core genome and accessory gene content are needed to enhance epidemiological resolution (Lakhundi and Zhang, 2018).

Given the variable link between PVL and disease severity, further studies should clarify its role across diverse strains and infection models. Other virulence factors, such as alpha-toxin, phenol-soluble modulins, and immune evasion genes, also warrant investigation (Otto, 2013, Voyich *et al*., 2006).

In low-resource settings, PVL may still offer diagnostic value, but its interpretation must be guided by local epidemiology, especially the spread of PVL-positive HA-MRSA.

As MRSA lineages increasingly converge, coordinated strategies across hospital and community settings are essential. Integrated surveillance, stewardship, and education will be key to curbing transmission.

Ultimately, while PVL remains of interest, no single gene defines MRSA. Genomic tools must be used to navigate its growing complexity.

REFERENCES

1. Al-Halaq, A. a. I. & Utba, N. M. (2023). Prevalence of Methicillin-resistant Staphylococcus aureus Carrying lukS-lukF Gene in Iraqi Patients with Furunculosis. Iraqi Journal of Science, 64**:** 4223-4229.
2. Albrecht, C. (2013). Molecular characterization of community-associated Staphylococcus aureus isolates from the Middle East. . Journal of Clinical Microbiology, 51**:** 145–150.

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DOI: 10.47191/ijcsrr/V9-i3-38, Impact Factor: 8.048
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www.ijcsrr.org

3. Boakes, E., Kearns, A. M., Ganner, M., Perry, C., Hill, R. L. & Ellington, M. J. (2011). Distinct bacteriophages encoding Panton-Valentine leukocidin (PVL) among international methicillin-resistant Staphylococcus aureus clones harboring PVL. Journal of Clininical of Microbiology, 49: 684-92.
4. Boyle-Vavra, S. & Daum, R. S. (2007). Community-acquired methicillin-resistant Staphylococcus aureus: the role of Panton-Valentine leukocidin. Laboratory Investigation, 87: 3-9.
5. Coombs, G. W., Baines, S. L., Howden, B. P., Swenson, K. M. & O'brien, F. G. (2020). Diversity of bacteriophages encoding Panton-Valentine leukocidin in temporally and geographically related Staphylococcus aureus. PLoS One, 15: e0228676.
6. Costa, C. A., Inácio, H., Trevas, S. & Simas, Â. (2025). Panton-Valentine Leukocidin in Necrotizing Pneumonia: A Case Report. Cureus, 17: e78774.
7. Deurenberg, R. H. & Stobberingh, E. E. (2008). The evolution of Staphylococcus aureus. Infection, Genetics and Evolution, 8: 747-763.
8. El Aila, N. A., Al Laham, N. A. & Naas, T. (2023). Prevalence of mecA and Panton-Valentine Leukocidin Genes in Staphylococcus aureus Clinical Isolates from Gaza Strip Hospitals. Microorganisms, 11.
9. Emamalipour, M., Seidi, K., Zununi Vahed, S., Jahanban-Esfahlan, A., Jaymand, M., Majdi, H., Amoozgar, Z., Chitkushev, L. T., Javaheri, T., Jahanban-Esfahlan, R. & Zare, P. (2020). Horizontal Gene Transfer: From Evolutionary Flexibility to Disease Progression. Frontiers in Cell and Developmental Biology, 8: 229.
10. Gillet, Y., Issartel, B., Vanhems, P., Fournet, J. C., Lina, G., Bes, M., Vandenesch, F., Piémont, Y., Brousse, N., Floret, D. & Etienne, J. (2002). Association between Staphylococcus aureus strains carrying gene for Panton-Valentine leukocidin and highly lethal necrotising pneumonia in young immunocompetent patients. Lancet, 359: 753-9.
11. Goudarzi, H., Goudarzi, M., Sabzehali, F., Fazeli, M. & Salimi Chirani, A. (2020). Genetic analysis of methicillin-susceptible Staphylococcus aureus clinical isolates: High prevalence of multidrug-resistant ST239 with strong biofilm-production ability. Journal of Clinical Laboratory Analysis, 34: e23494.
12. Hsu, L.-Y., Harris, S. R., Chlebowicz, M. A., Lindsay, J. A., Koh, T.-H., Krishnan, P., Tan, T.-Y., Hon, P.-Y., Grubb, W. B., Bentley, S. D., Parkhill, J., Peacock, S. J. & Holden, M. T. G. 2015. Evolutionary dynamics of methicillin-resistant Staphylococcus aureus within a healthcare system. *Genome biology* [Online], 16. Available: http://europepmc.org/abstract/MED/25903077
13. https://doi.org/10.1186/s13059-015-0643-z
14. https://europepmc.org/articles/PMC4407387
15. https://europepmc.org/articles/PMC4407387?pdf=render [Accessed 2015/04//].
16. Hussain, K., Bandyopadhyay, A., Roberts, N., Mughal, N., Moore, L. S. P. & Fuller, L. C. (2022). Panton-Valentine leucocidin-producing Staphylococcus aureus: a clinical review. Clinical and Experimental Dermatology, 47: 2150-2158.
17. Hussein, S. Z. & Mohammed Saleh, G. (2024). Molecular Detection of Virulence Factors Genes for Staphylococcus aureus in Diabetic Foot Ulcers in Iraq. Ibn AL-Haitham Journal For Pure and Applied Sciences, 37: 98–105.
18. Ibrahim, H. & Al-Mathkhury, H. (2018). pvl-carried methicillin resistant Staphylococcus aureus isolated from hospitalized patients in Baghdad, Iraq. Iraqi Journal of Science, 59: 1967-1972.
19. Jawad, A. A., Mohammed, A. A. & Al-Hashimi, A. (2022). Epidemiological Study on the Prevalence of Staphylococcus aureus PVL Gene Among Healthy Community in Al- Karkh and Al -Rusaffa Districts Baghdad, Iraq. Iraqi Journal of Science, 63: 441-448.
20. Lakhundi, S. & Zhang, K. (2018). Methicillin-Resistant Staphylococcus aureus: Molecular Characterization, Evolution, and Epidemiology. Clinical Microbiolgy Reviews, 31.
21. Larsen, J., Raisen, C. L., Ba, X., Sadgrove, N. J., Padilla-González, G. F., Simmonds, M. S. J., Loncaric, I., Kerschner, H., Apfalter, P., Hartl, R., Deplano, A., Vandendriessche, S., Černá Bolfíková, B., Hulva, P., Arendrup, M. C., Hare, R. K., Barnadas, C., Stegger, M., Sieber, R. N., Skov, R. L., Petersen, A., Angen, Ø., Rasmussen, S. L., Espinosa-Gongora, C., Aarestrup, F. M., Lindholm, L. J., Nykäsenoja, S. M., Laurent, F., Becker, K., Walther, B., Kehrenberg, C., Cuny, C., Layer, F., Werner, G., Witte, W., Stamm, I., Moroni, P., Jørgensen, H. J., De Lencastre, H., Cercenado, E., García-Garrote, F., Börjesson, S., Hæggman, S., Perreten, V., Teale, C. J., Waller, A. S., Pichon, B., Curran, M. D., Ellington, M. J., Welch, J.

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J., Peacock, S. J., Seilly, D. J., Morgan, F. J. E., Parkhill, J., Hadjirin, N. F., Lindsay, J. A., Holden, M. T. G., Edwards, G. F., Foster, G., Paterson, G. K., Didelot, X., Holmes, M. A., Harrison, E. M. & Larsen, A. R. (2022). Emergence of methicillin resistance predates the clinical use of antibiotics. Nature, 602: 135-141.

22. Leistner, R., Hanitsch, L. G., Kruger, R., Lindner, A. K., Stegemann, M. S. & Nurjadi, D. (2022). Skin Infections Due to Panton-Valentine Leukocidin-Producing S. Aureus. Deutsches Ärzteblatt International 119: 775-784.
23. Loffler, B., Hussain, M., Grundmeier, M., Bruck, M., Holzinger, D., Varga, G., Roth, J., Kahl, B. C., Proctor, R. A. & Peters, G. (2010). Staphylococcus aureus panton-valentine leukocidin is a very potent cytotoxic factor for human neutrophils. PLoS Pathogens, 6: e1000715.
24. Malachowa, N. & Deleo, F. R. (2010). Mobile genetic elements of Staphylococcus aureus. Cellular and Molecular Life Sciences, 67: 3057-3071.
25. Mcguinness, W. A., Kobayashi, S. D. & Deleo, F. R. (2016). Evasion of Neutrophil Killing by Staphylococcus aureus. Pathogens, 5.
26. Mediavilla, J. R., Chen, L., Mathema, B. & Kreiswirth, B. N. (2012). Global epidemiology of community-associated methicillin resistant Staphylococcus aureus (CA-MRSA). Current Opinion in Microbiology, 15: 588-595.
27. Mohamad Farook, N. A., Argimón, S., Abdul Samat, M. N., Salleh, S. A., Sulaiman, S., Tan, T. L., Periyasamy, P., Lau, C. L., Ismail, Z., Muhammad Azami, N. A., Ang, M. Y. & Neoh, H.-M. (2022). Diversity and Dissemination of Methicillin-Resistant Staphylococcus aureus (MRSA) Genotypes in Southeast Asia. Tropical Medicine and Infectious Disease, 7: 438.
28. Mohammed, M. M., Isa, M. A., Abubakar, M. B., Dikwa, A. S. B. & Kappo, A. P. (2025). Molecular detection of mecA gene from methicillin-resistant Staphylococcus aureus isolated from clinical and environmental samples and its potential inhibition by phytochemicals using in vitro and in silico approach. In Silico Pharmacology, 13: 26.
29. Monecke, S., Skakni, L., Hasan, R., Ruppelt, A., Ghazal, S. S., Hakawi, A., Slickers, P. & Ehricht, R. (2012). Characterisation of MRSA strains isolated from patients in a hospital in Riyadh, Kingdom of Saudi Arabia. BMC Microbiology, 12: 146.
30. Otter, J. A. & French, G. L. (2010). Molecular epidemiology of community-associated meticillin-resistant Staphylococcus aureus in Europe. The Lancet Infectious Diseases, 10: 227-239.
31. Otter, J. A. & French, G. L. (2012). Community-associated meticillin-resistant Staphylococcus aureus: the case for a genotypic definition. Journal of Hospital Infection, 81: 143-148.
32. Otto, M. (2013). Community-associated MRSA: what makes them special? Intenational Journal of Medical Microbiologiy, 303: 324-30.
33. Planet, P. J., Diaz, L., Kolokotronis, S.-O., Narechania, A., Reyes, J., Xing, G., Rincon, S., Smith, H., Panesso, D., Ryan, C., Smith, D. P., Guzman, M., Zurita, J., Sebra, R., Deikus, G., Nolan, R. L., Tenover, F. C., Weinstock, G. M., Robinson, D. A. & Arias, C. A. (2015). Parallel Epidemics of Community-Associated Methicillin-Resistant Staphylococcus aureus USA300 Infection in North and South America. The Journal of infectious diseases, 212: 1874-1882.
34. Shallcross, L. J., Fragaszy, E., Johnson, A. M. & Hayward, A. C. (2013). The role of the Panton-Valentine leucocidin toxin in staphylococcal disease: a systematic review and meta-analysis. The Lancet Infectious Diseases, 13: 43-54.
35. Silva-Santana, G. (2025). Staphylococcus aureus: Dynamics of pathogenicity and antimicrobial-resistance in hospital and community environments - Comprehensive overview. Research in Microbiology, 176: 104267.
36. Uhlemann, A. C., Otto, M., Lowy, F. D. & Deleo, F. R. (2014). Evolution of community- and healthcare-associated methicillin-resistant Staphylococcus aureus. Infection, Genetics and Evolution, 21: 563-74.
37. Voyich, Jovanka m., Otto, M., Mathema, B., Braughton, Kevin r., Whitney, Adeline r., Welty, D., Long, R. D., Dorward, David w., Gardner, Donald j., Lina, G., Kreiswirth, Barry n. & Deleo, Frank r. (2006). Is Panton-Valentine Leukocidin the Major Virulence Determinant in Community-Associated Methicillin-Resistant Staphylococcus aureus Disease? The Journal of Infectious Diseases, 194: 1761-1770.
38. Yamaguchi, T., Ono, D. & Sato, A. (2020). Staphylococcal Cassette Chromosome mec (SCCmec) Analysis of MRSA. Methods in Molecular Biology, 2069: 59-78.

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39. Zhao, H., Wu, X., Wang, B., Shen, L., Rao, L., Wang, X., Zhang, J., Xiao, Y., Xu, Y., Yu, J., Guo, Y., Zhou, Y., Wan, B., Wu, C., Chen, L. & Yu, F. (2023). Phenotypic and genomic analysis of the hypervirulent ST22 methicillin-resistant Staphylococcus aureus in China. mSystems, 8: e0124222.
40. Zhao, R., Wang, X., Wang, X., Du, B., Xu, K., Zhang, F., Jiang, C., Zhao, Y. & Zhu, Y. (2022). Molecular characterization and virulence gene profiling of methicillin-resistant Staphylococcus aureus associated with bloodstream infections in southern China. Frontiers in Microbiology, 13: 1008052.

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