Acta Scientific Microbiology (ISSN: 2581-3226)

Volume 8 Issue 7 July 2025

Short Communication

Antibiotic Resistance and Pathogenicity of *Staphylococcus aureus*

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Received: May 15, 2025

Published: June 16, 2025

Abstract

Staphylococcus aureus can cause a wide range of infections ranging from minor skin abscesses to more serious invasive diseases. The development and spread of bacterial strains that are resistant to antibacterial drugs has emerged as a global problem. The appearance of antibiotic resistant bacteria over the past decades has been regarded as an inevitable genetic response to the strong selective pressure imposed by antimicrobial chemotherapy, which plays a crucial role in the evolution of antibiotic resistant bacteria.

Keywords: *Staphylococcus aureus*; Antibiotic Resistant

Staphylococcus aureus, a major cause of potentially life-threatening infections acquired in healthcare and community settings, has developed resistance to most classes of antimicrobial agents. Penicillin was the first choice of antibiotics to treat staphylococcal infection. In 1944, by destroying the penicillin by *penicillinase* (*beta-lactamase*), *S. aureus* become resistant to penicillin. Most of the *S. aureus* strains (≥90%) are resistant to penicillin [1]. Methicillin, a semisynthetic penicillins was used to treat penicillin resistant *Staphylococcus aureus* but resistance finally emerge in 1962 [2]. Methicillin-resistance in *S. aureus* is mediated by the presence of penicillin-binding protein 2a (PBP-2a) which is expressed by an exogenous gene, *mecA*. This gene is carried by a genetic element, designated as staphylococcal cassette chromosome *mec (SCCmec)*, which is inserted near the chromosomal origin of replication. High prevalence of methicillin resistant *Staphylococcus aureus* (MRSA) in hospitals has been reported from many states of India. Methicillin resistance among *S. aureus* isolates has reached phenomenal proportions in Indian hospitals, with some cities reporting 70% of the strains to be resistant to methicillin [3]. Vancomycin continues to be an important antimicrobial agent for treatment of MRSA infections but resistance finally emerges. In 1996, a *S. aureus* strain with intermediate resistance to vancomycin (VISA) (vancomycin MIC= 8 µg/ml) was first isolated from a patient in Japan. Shortly afterward, VISA strains were isolated in USA, Europe and other Asian countries, arousing considerable concern regarding the emergence of *S. aureus* strains for which there will be no effective therapy. Characterization of these VISA strains indicates that the mechanisms of resistance are complex and involve changes in cell wall content and composition [4].

In June 2002, the world's first reported clinical infection due to *S. aureus* with high resistance to vancomycin (VRSA) (vancomycin MIC>128 µg/ml) was diagnosed in a patient in the USA. This isolate contain the *vanA* genes from enterococci and the methicillin-resistance gene *mecA*. The possible emergence and dissemination of VRSA strains is a serious health threat and makes it absolutely necessary to optimize prevention strategies and fast detection methods [5]. Till today only six VRSA have been found all over the world, first in USA in 2002, second in Michigun in 2002, third in Pennsylvania in 2002, fourth in New York in 2004, fifth in New York in 2005, and the sixth in India in 2005 [6].

Staphylococcus aureus expresses a wide array of secreted and cell surface associated virulence factors to help evade immune responses. *S. aureus* were able to survive within phagocytic cells both in polymorphonuclear leukocytes (PMN) and monocytes. In order to survive and induce infection, pathogenic bacteria have to cope with their changing environment, as well as continuous attacks of the host anti-microbial defense system [7].

The generation and release of toxic reactive oxygen species by phagocytic cells is thought to be an important component of the host's immunity against bacterial infection. Reactive oxygen intermediates are part of the oxygen dependent bactericidal mechanisms that the phagocytic cell employs. After engulfment of bacteria by professional phagocytes the induction of highly microbicidal reactive oxygen metabolites during the oxidative burst occurs, resulting in killing. Monocyte derived macrophages produce a large amount of hydrogen peroxide (H_2O_2) in response to heat killed *S. aureus*. Catalase has been suggested to protect *S. aureus* [8]. However, the exact mechanism by which bacteria combat oxidative burst during phagocytosis to enable intracellular survival remains unclear. Other studies have indicated that production of catalase correlates to virulence of *S. aureus* and other microorganism. It was proposed that Cu-Zn SOD could offer an important advantage in survival within host cells to bacteria expressing high levels of these enzymes. The interaction of *S. aureus* with murine macrophages and the contribution of catalase and SOD in intracellular persistence of *S. aureus* within murine macrophages during in vitro infection was reported [6]. Previous studies have shown that both bovine and mammary epithelial cells and human endothelial cells internalize *S. aureus* and subsequently undergo apoptosis. Further studies are needed to understand the molecular mechanisms by which *S. aureus* replicates intracellularly and induces apoptosis. Hence, the investigation of *S. aureus* induced oxidative damage in lymphocytes may achieve importance to reflect the immune response during staphylococcal infection.

Declaration of Interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of this paper.

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