Acta Scientific Microbiology (ISSN: 2581-3226)

Volume 9 Issue 10 October 2026

Mini Review

Alleviating Host Defense System with Emerging Antibiotic Resistance of *Staphylococcus aureus*

Subhankari Prasad Chakraborty*

Assistant Professor, Department of Physiology, Ramananda College, Bishnupur, Bankura-722 122, West Bengal, India

***Corresponding Author:** Subhankari Prasad Chakraborty, Assistant Professor, Department of Physiology, Ramananda College, Bishnupur, Bankura-722 122, West Bengal, India.

DOI: 10.31080/ASMI.2026.09.1629

Received: August 14, 2026

Published: September 22, 2026

Abstract

Staphylococcus aureus is responsible for various infections ranging from minor skin abscesses to invasive infections. The development of strains of bacteria that are resistant to antibacterial drugs is now seen as a worldwide phenomenon. The emergence of resistant bacteria against the use of antibiotics in recent decades has been viewed as a natural genetic consequence of the tremendous selection pressure applied on bacteria due to antimicrobial chemotherapy.

Keywords: *Staphylococcus aureus*; Penicillin; Methicillin

Staphylococcus aureus, a leading bacterial pathogen associated with fatal infections that may arise in healthcare as well as community settings, is resistant to most antimicrobial classes [1]. Penicillin was the earliest class of antibiotics to be used in the treatment of staphylococcal infections. The emergence of penicillin resistance in *S. aureus* by 1944 as a result of destruction of penicillin by *penicillinase* (*beta-lactamase*) was documented. Penicillin resistance occurs in nearly all of the strains of *S. aureus* (≥90%). Methicillin, a semisynthetic penicillin antibiotic was employed in the treatment of penicillin-resistant *S. aureus* but resistance emerged as early as 1962 [2]. Methicillin resistance in *S. aureus* is through the presence of penicillin binding protein 2a (PBP-2a) which is encoded by the *mecA* gene. This gene is carried by a genetic element referred to as staphylococcal cassette chromosome mec (SCCmec) which is inserted at the vicinity of the chromosomal origin of replication. Prevalence of MRSA is high among the hospitalized patients in many states of India. Methicillin resistance in *S. aureus* strains is common in Indian hospitals to the extent of 70% being methicillin-resistant [3]. Vancomycin remains a significant anti-infective agent used to treat MRSA infection cases; however, eventually resistance is developed. The first *S. aureus* isolate with intermediate resistance to vancomycin (Vancomycin-intermediate resistant *Staphylococcus aureus* or VISA) with vancomycin minimum inhibitory concentration of 8µg/ml was reported in 1996 from a patient in Japan. Soon after, strains with intermediate vancomycin resistance were isolated in USA, Europe and other Asian nations, leading to much concern about the emergence of *S. aureus* strains resistant to all forms of therapy. Studies on such VISA strains have revealed that the mechanisms of resistance are complicated and involve alterations in cell wall composition [4].

First clinical case of VRSA (vancomycin MIC>128 µg/ml) in the world was diagnosed in June 2002 in USA in the patient infected by *S. aureus* with increased vancomycin resistance. Isolate harbors the *vanA* genes from enterococci and the methicillin resistance genes

mecA. Emergence and spread of VRSA strains is a very dangerous issue for public health and thus requires optimization of prevention measures and rapid detection techniques [5]. Up till now, there have been six cases of VRSA found throughout 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].

S. aureus produces many secreted or cell-surface bound virulence factors that aid it in its efforts to escape immune response. The *S. aureus* survived inside phagocytic cells in PMN and monocytes. To be able to survive and cause disease, bacterial pathogens need to be able to adapt to changes in their environment, along with attacks from the host's antimicrobial defense mechanism [7]. Production and secretion of reactive oxygen species through the phagocytic cells is believed to be one of the important elements of host defense mechanism against the infection. Reactive oxygen species are included in oxygen-dependent bactericidal mechanism used by phagocytic cells. Following ingestion of the bacteria by the professional phagocytes, the generation of highly microbicidal reactive oxygen species in oxidative bursts leads to killing of the bacteria. A large amount of H_2O_2 is produced in monocyte-derived macrophages in response to heat-killed *S. aureus*. *Catalase* is supposed to be one of the protective mechanisms of *S. aureus* [8]. However, the detailed mechanism of action by which bacteria neutralize the effect of oxidative bursts during phagocytosis and facilitate their survival is unknown. There are evidences that catalase production is associated with the virulence of *S. aureus* and other microorganisms. Cu-Zn SOD is supposed to provide an important survival factor for bacteria having high amounts of these enzymes. Interaction between *S. aureus* and murine macrophages and role of *catalase* and SOD in intracellular survival of *S. aureus* in murine macrophages in vitro was described [6]. Earlier findings have revealed that *S. aureus* is taken up by bovine and mammary epithelial cells as well as human endothelial cells and then undergoes cell death by apoptosis. There is need for further investigations on the molecular basis by which *S. aureus* replicates in cells and causes cell death through apoptosis. Therefore, the study of oxidative damage in lymphocytes caused by *S. aureus* may become significant.

Acknowledgement

Author express gratefulness to Ramananda College, Bishnupur, Bankura, West Bengal, India for providing the space and facilities to execute this work. Author is highly obliged to the Principal, Ramananda College, Bishnupur, Bankura, West Bengal, India for constant help and encouragement.

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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